SOURCES, EFFECTS AND RISKS OF IONIZING RADIATION

United Nations Scientific Committee on the Effects of Atomic Radiation

UNSCEAR 2019
Report to the General Assembly, with Scientific Annexes

UNITED NATIONS
New York, 2020

NOTE

The report of the Committee without its annexes appears as *Official Records of the General Assembly*, Seventy-fourth Session, Supplement No. 46 (A/74/46).

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UNITED NATIONS PUBLICATION
Sales No. E.20.IX.5
ISBN: 978-92-1-139184-8
eISBN: 978-92-1-005136-1

This publication has not been formally edited.

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ANNEX B

LUNG CANCER FROM EXPOSURE TO RADON

CONTENTS

The attachment "Models of excess relative risk with modifying effects" is electronically available for download from http://www.unscear.org/unscear/en/publications/2019.html

I. INTRODUCTION

1. The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) has previously considered the issue of sources and effects of exposure to radon (^{222}Rn) (including thoron (^{220}Rn)) with regard to workers and the public in two comprehensive reports in 2000 and 2006 [U3, U6]. The Committee concluded that inhaled radon and its progeny are established carcinogens for the lung (doses to other organs and tissues were at least an order of magnitude smaller than the doses to the lung). The Committee also recognized that the annual per caput dose from inhalation of radon gas and its progeny represented typically about half of the effective dose*[1] (as defined by the International Commission on Radiological Protection (ICRP) in its publication 60 [I4]) received by members of the public from all natural sources of ionizing radiation. For certain occupations, radon and its progeny also dominate occupational radiation exposure. For example, in the nuclear fuel cycle, radon inhaled following its release from uranium mines makes a substantial contribution to the workers doses.

2. There are two approaches for deriving the effective dose per unit exposure from radon and its progeny. These are hereafter referred to as "dosimetric" and "epidemiological", the latter approach is also referred to as the dose conversion convention [I6]. The dosimetric approach derives the effective dose from a given exposure using a dosimetry model of the respiratory tract and applying radiation and tissue weighting factors. It is worth mentioning that the tissue weighting factors are determined mainly from epidemiological studies of Japanese atomic bombing survivors exposed largely to gamma radiation. The epidemiological approach compares the radiation detriment* associated with unit radon progeny exposure and unit effective dose. Thus, the lifetime risk* of lung cancer per unit exposure to radon progeny is divided by the detriment per unit effective dose. The former was determined from miner epidemiology and the latter determined mainly from epidemiological studies of Japanese atomic bombing survivors exposed largely to gamma radiation. Based on a review of available dosimetric models by an expert group of the Nuclear Energy Agency [N4], the Committee adopted in its 1982 Report an effective dose coefficient for indoor exposure of 8.7 nSv per h Bq m^{-3} of equilibrium equivalent concentration (EEC)* of ^{222}Rn [U1]. This value was retained and rounded to 9 nSv per h Bq m^{-3} EEC of ^{222}Rn for indoor and outdoor exposure in the UNSCEAR 1988, 2000 and 2006 Reports [U2, U3, U6]. In its UNSCEAR 2006 Report, the Committee recognized that more recent calculations with new dosimetric models resulted in higher values of the dose conversion factor. However, because of the lower value calculated with the epidemiological approach of ICRP Publication 65 [I6], the Committee concluded that the previous value of 9 nSv per h Bq m^{-3} EEC of ^{222}Rn is within the range of possible dose conversion factors. Consequently, the Committee recommended that this value should continue to be used in UNSCEAR dose assessments [U6]. The value of 9 nSv per h Bq m^{-3} EEC of ^{222}Rn corresponds to 1.6 mSv per mJ h m^{-3} or 5.7 mSv per working level month (WLM)*.

3. ICRP in its Publication 115 [I10], reviewed epidemiological data of radon-induced lung cancers, focusing on low levels of exposure and exposure rates in mines [N10, T11]. As a result, for radiation protection purposes, a detriment adjusted nominal risk coefficient of 1.4×10^{-4} per mJ h m^{-3} equivalent to 5×10^{-4} WLM^{-1} was recommended for a mixed adult population of smokers and non-smokers. This is approximately twice the value given previously in ICRP Publication 65 [I6].

[1] Technical terms are explained in the glossary and are marked with an asterisk (*) the first time that they appear.

4. The Committee has not studied issues concerning exposure and effects from radon (and thoron) since its UNSCEAR 2006 Report [U6]. However, between 2006 and 2017 there have been several hundred scientific publications concerning radon exposure and lung cancer, including those related to epidemiological studies of radon-induced lung cancer in exposed populations as well as those with relevant dosimetry. The present annex, in agreement with the Committee's mandate from the General Assembly of the United Nations for estimating effects and risk from radiation exposure, aims to thoroughly assess the newly available literature.

II. DOSIMETRY

A. Introduction

5. The risk of lung cancer associated with radon arises from the inhalation of the airborne radon progeny and the resulting dose to the lung, mainly from alpha radiation. In general, lung doses from inhaling the airborne radon progeny are about two orders of magnitude higher than the corresponding lung dose from inhaled radon gas. Although lung cancers are caused by the inhaled short-lived radon progeny and not by the radon gas, it is the radon gas which is commonly measured and not its progeny. The International Commission on Radiation Units and Measurements (ICRU) in its Report 88 [I14] provided detailed information on how to measure radon, covering measurement techniques of radon in air and water. The only measurable parameters which allow the determining of lung doses are the radon and radon progeny activity concentrations and related particle size distributions. The highest doses are received by the bronchial and bronchiolar regions of the lung consistent with the observation that most of the lung carcinomas associated with radon exposure originate in the bronchi [I14, S1, U3, U6, W14].

6. Calculations of the absorbed dose* to the target tissues of the lung per unit exposure to radon progeny can be carried out with dosimetric models of the human respiratory tract. Such calculations require information on the activity size distribution of the radon progeny aerosol, the unattached fraction, the breathing rate, the fractional deposition in the airways, mucous clearance rate, transport into blood and the location of the target cells in the airways. The calculations are subject to all of the variability and uncertainty associated with the model parameter values as well as the assumptions built into the particular computational model [H3, I14, M1, M2, U3, W12, Z5]. A detailed description of dosimetric models and choice of parameter values is given in appendix A.

7. In its UNSCEAR 2000 Report [U4], the Committee reported that the dosimetric evaluation of the absorbed dose to the basal cells of the bronchial epithelium per unit exposure of EEC of ^{222}Rn were in the range of 5 to 25 nGy $(\text{h Bq m}^{-3})^{-1}$. A central value of 9 nGy $(\text{h Bq m}^{-3})^{-1}$ of EEC of ^{222}Rn was estimated for average indoor conditions. This dosimetry evaluation was reproduced in the UNSCEAR 2006 Report [U6]. By applying an apportioned tissue weighting factor of 0.08 for the bronchial (BB) and bronchiolar (bb) regions [U4] and a radiation weighting factor of 20 for alpha particles [I9], the central value in terms of effective dose per unit EEC of ^{222}Rn exposure became 15 nSv $(\text{h Bq m}^{-3})^{-1}$ with a range of 8–40 nSv $(\text{h Bq m}^{-3})^{-1}$ [U4, U6].

B. Aim and objectives

8. This section focuses on updating the UNSCEAR 2006 Report [U6] with regard to the following aspects: dosimetric models, dosimetry of radon, dose conversion factor, and uncertainties in dose conversion factors. Specific objectives are to address the following questions:

- What is the current status of the development of dosimetric models and model parameters and associated impact on the assessment of radon dose to human lung? What are the uncertainties and variations in the dosimetric assessment?
- What is the current status of dosimetric assessment for smokers and non-smokers?
- What is the current status of dosimetric assessment for different working environments/conditions?

C. Scope and literature search

9. The scope of the review includes results of primary peer-reviewed studies published in the scientific literature and publications of relevant international organizations. The focus is on dosimetric assessments of radon exposure to the lung. Additional background information about different dosimetric models and choice of parameters is found in appendix A.

10. A comprehensive literature review of scientific journal articles published since the UNSCEAR 2006 Report [U6] was conducted to identify studies providing new information on dosimetric assessment of radon exposure in homes, in indoor workplaces and in mines. Due to the physical nature of radon dosimetry, the search was performed with the database of Scopus. The literature search was conducted with query string “Search (((((“lung” or “radon” [Title/Abstract]) AND “dosimetr*” [Title/Abstract])) AND (""01/01/2006-""[Date - Publication] : ""31/12/2017""[Date - Publication]))).

11. In total, 98 references were identified for the radon dosimetric assessment. All articles were reviewed with respect to authors’ potential conflicts, and by using screening and quality questions. The selection criteria developed by the Committee in its UNSCEAR 2017 Report [U8] were applied for the dosimetric review.

12. Identified publications that did not meet the screening questions were excluded from the review. This included non-original publications, studies in non-human populations, and studies on measurement techniques that were not relevant to dosimetric assessment. Based on the quality questions, reviewers made the final decision on whether to include/exclude studies in the evaluation. Study item reviews, final inclusion/exclusion decisions, and reasons for the decisions were recorded, along with summaries of findings and appraisals for included studies.

13. After an initial literature search additional publications were identified that had been published since 1998 and had not been considered in the UNSCEAR 2006 Report [U6]. In total, 94 publications were reviewed in detail, 50 of them developed/assessed human lung dosimetric models or provided information on human lung dosimetry parameters. A detailed review of the results from these publications are summarized in section D.

D. Review of dosimetric assessments

1. Overview of dosimetric assessments

14. The review results of more recent dosimetric calculations of absorbed dose to regions of the lung and the corresponding dose conversion factor for unit radon progeny exposure in homes, indoor workplaces and mines are summarized in tables 1 to 3, respectively. Information on model type, breathing rates, activity size distribution of the inhaled aerosol, the unattached fraction, the assumed value for the hygroscopic growth factor (*hgf*) or the attached progeny are given whenever available in the literature. Special quantities and units that characterize the concentration and exposure of radon progeny in air, which are used in the summary tables are described in appendix A. The choice and range of model parameter values based on published experimental data are discussed in appendix A. Further, the dose conversion factor approximates the effective dose per unit exposure and is calculated as described in appendix A.

15. The dosimetric calculations have been carried out with deterministic airway generation models (DAGM) [P6, W12, Y2, Y3], stochastic airway generation models (SAGM) [H9, W13] and the ICRP human respiratory tract model (HRTM) [I7, I11]. For more details see also section II in appendix A.

16. In total, 16 assessments are summarized in table 1 for radon exposure in homes under various conditions. The assessed dose conversion factor in terms of effective doses per unit exposure of EEC of ^{222}Rn varied from 7 to 34 nSv $(\text{h Bq m}^{-3})^{-1}$ with an arithmetic mean of 18 nSv $(\text{h Bq m}^{-3})^{-1}$, and a geometric mean of 16 nSv $(\text{h Bq m}^{-3})^{-1}$. The corresponding doses per WLM range from 4 to 21 mSv WLM^{-1} with an arithmetic mean of 11 mSv WLM^{-1} and a geometric mean of 10 mSv WLM^{-1}.

17. Dose conversion factors were calculated with the HRTM for different age groups of males and females for home exposure to radon progeny [B13]. The calculated values were 20 mSv WLM^{-1} for 3-month infants, 21 mSv WLM^{-1} for 1 year olds, 18 mSv WLM^{-1} for 5 year olds, 20 mSv WLM^{-1} for 10 year olds, 18 mSv WLM^{-1} for 15-year-old males, 16 mSv WLM^{-1} for 15-year-old females, 18 mSv WLM^{-1} for adult males and 17 mSv WLM^{-1} for adult females. Calculations by Marsh et al. [M3] for different age groups with the HRTM gave lower values of 9.6 to 12.9 mSv WLM^{-1}. The reason why the dose per WLM is relatively insensitive to age is that competing effects tend to cancel each other out. For example, children have lower breathing rates, so this decreases the intakes and dose per unit exposure. However, this is partly compensated by the smaller target tissue masses, which in turn increases the dose. Children have smaller airways which increase deposition by diffusion, but this is compensated in part by smaller residence times (i.e. higher respiratory rates) that decrease deposition by diffusion [M1, N9].

18. Marsh et al. [M3] calculated effective doses per unit exposure to radon progeny for adults living in houses with and without smokers. Values for houses with smokers were calculated assuming that the subjects were in a living room in which cigarettes are smoked except while they were asleep in a smoke-free bedroom. Aerosol parameter values assumed were based on measurement data published in the literature [M2, N9, P5]. Effective dose coefficients for houses with and without smokers were 9.9 mSv WLM^{-1} and 13 mSv WLM^{-1}, respectively. The lower value for homes with smokers was obtained because the unattached fraction was estimated to be lower and because no nucleation mode was assumed in the presence of cigarette smoke. James et al. [J3] calculated higher values of 18 and 21 mSv WLM^{-1} for houses with and without smokers, respectively, using the HRTM. These values are higher because of the different activity size distribution and unattached fraction assumed by the authors, which were based on the measurements of Hopke et al. [H12, N10]. In contrast, for active smoking where the aerosol is dominated by cigarette smoke and the unattached fraction is negligible, Porstendörfer [P6]

calculated a dose conversion factor of 4.2 mSv WLM^{-1} using the DAGM. The value assuming no additional aerosol sources was 8.0 mSv WLM^{-1} [P6]. The DAGM developed by Zock et al. [Z5] gives correspondingly lower values for attached progeny compared with the HRTM.

19. ICRP has recently updated its Publication 66 HRTM [I7] in Publication 130 [I12]. Changes mainly relate to particle transport from the nasal passages, bronchial tree and alveolar region. However, the lung dose arising from exposure to radon progeny is not very sensitive to the bronchial clearance rates as the progeny are short-lived compared to the half-times [M1]. Therefore, lung doses calculated with the revised model are not significantly different for radon progeny. The effective dose per unit exposure to radon progeny calculated with the this model for home exposure was about 13 mSv WLM^{-1} [M5].

20. The morphometric model used to model aerosol deposition adopted by ICRP [I7] is the one suggested by James [J2]. This model is based on average airway dimensions of three models published by Weibel [W4, W5, W6], Yeh and Schum [Y1] and Phalen et al. [P2] and adjusted to a standard functional residual capacity of 3.3×10^{-3} m^3. With a DAGM, Yu et al. [Y3] compared dosimetric calculations using the morphometric model of James [J2] with that of Yeh and Schum [Y1]. Both models were scaled to the functional residual capacity of the ICRP Reference Man [I8]. The airway generation models were used to determine the equilibrium surface activities per unit exposure in each airway (a cylindrical tube). For the dosimetry calculations, the target cell layers containing basal and/or secretory cells as defined by the HRTM [I7] were assumed. The effective doses calculated with the James model and Yeh-Schum model were respectively 12 and 8 mSv WLM^{-1} for home exposure. Thus, the HRTM predicts higher values than the Yeh-Schum model. The higher dose for the James model arises from the differences in the surface areas of the trachea-bronchial tree (T-B) between the two models [N5]. The James model has a lower surface area, which gives higher surface activities (Bq cm^{-2}) resulting in higher doses compared with the Yeh-Schum model. In other words, lower surface areas of the T-B give lower masses of the assumed target cell regions leading to higher doses. In contrast, Winkler-Heil and Hofmann [W12] also compared the morphometric models of James [J2] with that of Yeh and Schum [Y1] (normalized to the same lung volume). The two models gave similar dose conversion factors.

21. Yu et al. [Y2] carried out dose assessment for Caucasian males, Chinese males and Chinese females using the Yeh-Schum morphometric model [Y1]. Scaling factors based on functional residual capacity and mass were used to scale the airway dimensions for Caucasian males to give those for Chinese males and females. The ventilation rates for Caucasian males were also scaled to give lower values for Chinese males and females. For the dosimetry calculations, the target cell layers containing basal and/or secretory cells as defined by the HRTM [I7] were assumed. The dose conversion factors for a typical home environment for Caucasian males, Chinese males and Chinese females were 9.2, 7.4 and 6.5 mSv WLM^{-1}, respectively. Thus, lower dose conversion factors are obtained for Chinese males and females compared with that for Caucasian males.

22. Nikezic et al. [N6] carried out dose assessments following inhalation of radon progeny with the HRTM [I7] using a micro-dosimetric approach to investigate the effects of calculating cellular doses rather than the approach used by ICRP, where the absorbed doses to target cell layers are calculated. In their calculations the nuclei of basal cells were approximated as cones while those for the secretory cells as ellipsoids instead of spherical cell nuclei as assumed by Hofmann et al. [H8]. The abundance and depth of basal and secretory cells were taken from Mercer et al. [M8]. For indoor exposure, the effective dose calculated was 13 mSv WLM^{-1} with the cellular dose approach compared with 15 mSv WLM^{-1} obtained assuming target cell layers. If spherical target cells are assumed, then the effective dose is 13.5 mSv WLM^{-1}. The authors noted that changes in the geometry of secretory-cell nuclei have only a small effect on their absorbed doses, whereas changes from spherical to conical basal-cell nuclei reduced their absorbed doses from ~4 to ~3 mGy WLM^{-1}.

23. Five studies are summarized in table 2 for radon exposure in indoor workplaces under various indoor conditions. The assessed effective doses per unit exposure of EEC of ^{222}Rn varied from 9 to 38 nSv (h Bq m^{-3})$^{-1}$ with an arithmetic mean of 25 nSv (h Bq m^{-3})$^{-1}$, and a geometric mean of 23 nSv (h Bq m^{-3})$^{-1}$. The corresponding doses per WLM, range from 6 to 24 mSv WLM^{-1} with an arithmetic mean of 16 mSv WLM^{-1} and a geometric mean of 15 mSv WLM^{-1}.

24. Tokonami et al. [T6] carried out aerosol measurements of particle size and unattached fraction in a computer room, where environmental conditions such as ventilation rate, air-conditioning and the operation of an air cleaner were varied. Using the data and the HRTM with a reference breathing rate of 1.2 m^3 h^{-1} for occupational exposure [I7], the calculated effective dose varied from 20 to 24 mSv WLM^{-1} with changes in ventilation and air-conditioning (table 2). Lower values of 14 to 17 mSv WLM^{-1} were calculated assuming a lower breathing rate of 0.78 m^3 h^{-1}. With the air-cleaner in operation, the unattached fraction, f_p increased significantly from about 0.12 to 0.56 resulting in a higher effective dose of 56 mSv WLM^{-1} for occupational exposure.

25. The effective doses per unit radon progeny exposure for indoor workplaces are on average higher than the corresponding values for home exposure. This is mainly because the assumed breathing rate is higher for a worker than for an adult at home. For example, with the revised HRTM of ICRP Publication 130 [I12], the effective doses per WLM are 13 and 20 mSv WLM^{-1} for homes and indoor workplaces, respectively [I13, M5]. ICRP assumes a reference breathing rate for a standard worker of 1.2 m^3 h^{-1}, where one third of the time is spent sitting and two thirds of the time is spent undertaking light exercise [I7]. For a male adult at home, the assumed breathing rate is lower: 0.78 m^3 h^{-1} (55% sleep, 15% sitting and 30% light exercise) [I7]. However, it is acknowledged that the assessed dose conversion factors for indoor workplaces and homes overlap.

26. Solomon et al. [S14] carried out activity size distribution measurements of radon progeny at the Olympic Dam underground uranium mine, Roxby Downs, South Australia in December 2013. Effective dose coefficients were calculated with the HRTM and the aerosol measurement data. For work locations with high ventilation and low levels of PAEC (i.e. at the underground workshop sites), the calculated effective dose was about 23 mSv WLM^{-1} (6.5 mSv per mJ h m^{-3}). The high fraction of the nucleation mode and the high unattached fraction accounted for the relatively high dose coefficient (table 3). For the operational areas of the mine with higher levels of potential alpha energy concentration (PAEC)*, the average effective dose was about 16 mSv WLM^{-1} (4.5 mSv per mJ h m^{-3}) (table 3). This is the most recent dose assessment to date for a mine using modern mining practices.

Table 1. Summary of dosimetric assessments for exposure to radon progeny in homes

GSD = geometric standard deviation; hgf = hygroscopic growth factor

Model type	*Aerosol source or action*	*Breathing rate ($m^3 h^{-1}$)*	*Unattached aerosol characteristics*			*Attached aerosol characteristics*					*Absorbed dose per unit exposure*[h] *($mGy\ WLM^{-1}$)*	*Dose conversion factor*		*Year*	*Reference*
			f_p[a]	*Particle size*[b] *(nm)*	*GSD*	*Mode, i*[d]	f_{pi}[e]	*Particle size*[b f] *(nm)*	*GSD*	*hgf*		*mSv per WLM*	*nSv per h Bq m^{-3} of EEC*		
HRTM [I7]	Typical home environment	0.78	0.08	0.9	1.3	n, a, c	f_{pn}=0.28, f_{pa}=0.7, f_{pc}=0.02	50[f], 250[f], 1 500[f]	2.0, 2.0, 1.5	1.5	BB: 8.9 bb: 9.2 AI: 0.33	15	23	2000	[M1]
DAGM [Z5]	A: no sources B: with sources (active smoking)	0.75	A: 0.05 (0.02-0.14) B: 0.005 (0.0008-0.02)	0.8	1.2	A: n, a B: a	A: f_{pn}=0.3, f_{pa}=0.7 B: f_{pa}=1	A: 20-40[f], 210 (120-350)[f], B: 300[f]	A: 1.7-2.2 (1.6-3.0) B: 2.0	A: 2.0 B: 1.0		A: 8.0[g] B: 4.2[g]	A: 13[g] B: 7[g]	2001	[P6]
DAGM [Y1, Y2]	Typical home environment		0.08	1.1				200[b]				Chinese male: 7.4 Chinese female: 6.5 Caucasian male: 9.2	Chinese male: 12 Chinese female: 10 Caucasian male: 14	2001	[Y2]
Morphometry model: A: [J1]; B: [Y1]			0.08	1.1				200[b]	2.35			A: 12 B: 8.1	A: 19 B: 13	2001	[Y3]
DAGM [W12]		0.78	0.08	0.9	1.3	n, a, c	f_{pn}=0.28, f_{pa}=0.7, f_{pc}=0.02	50[f], 250[f], 1 500[f]	2.0, 2.0, 1.5	1.5	BB: 5.45 bb: 5.50	8.8	14	2002	[W12]
HRTM [I7]	A: non-smoking B: smoking	0.78	A: 0.15 B: 0.08							1	A: 8.9[i] B: 7.5[i]	A: 21 B: 18	A: 34 B: 28	2004	[J3]

Model type	Aerosol source or action	Breathing rate ($m^3 h^{-1}$)	Unattached aerosol characteristics			Attached aerosol characteristics					Absorbed dose per unit exposure[h] ($mGy\ WLM^{-1}$)	Dose conversion factor		Year	Reference
			f_p[a]	Particle size[b] (nm)	GSD	Mode, i[d]	f_{pi}[e]	Particle size[b f] (nm)	GSD	hgf		mSv per WLM	nSv per h Bq m^{-3} of EEC		
HRTM [I7]	A: typical home without smokers B: typical home with smokers	0.75	A: 0.1 B: 0.03	0.9	1.3	A: n, a, c B: a	A: f_{pn}=0.15, f_{pa}=0.83, f_{pc}=0.02 B: f_{pa}=1.0	A: 50[f], 230[f], 2 500[f] B: 348[f]	A: 2.0, 2.1, 1.5 B: 2.0	A: 2.0 B: 1.0		A: 13 B: 9.9	A: 20 B: 16	2005	[M3]
HRTM [I7]		0.78	0.066	0.9	1.3	n, a, c	f_{pn}=0.28, f_{pa}=0.7, f_{pc}=0.02	50[f], 250[f], 1 500[f]	2.0, 2.0, 1.5	1.5	BB: 8.42 bb: 10.4	15.1	24	2006	[L5]
HRTM [I7] with cellular micro-dosimetry [N6]		0.78	0.066	0.9	1.3	n, a, c	f_{pn}=0.28, f_{pa}=0.7, f_{pc}=0.02	50[f], 250[f], 1 500[f]	2.0, 2.0, 1.5	1.5	BB: 6.4 bb: 10	13	20	2006	[N6]
HRTM [I7]		0.78	0.1	1.0		n, a, c	f_{pn}=0.15, f_{pa}=0.83, f_{pc}=0.02	50[f], 230[f], 2 500[f]				15	24	2011	[A2]
SAGM (IDEAL-DOSE)[c] [H9, H10]		0.78	0.1	0.8	1.3	n, a	f_{pn}=0.2, f_{pa}=0.8	40[f], 230[f]	2.0, 2.1	2	BB: 4.17 bb: 2.51	5.3	8	2011	[H10]
Revised HRTM [I12]		0.78	0.1	0.9	1.3	n, a	f_{pn}=0.2, f_{pa}=0.8	40[f], 230[f]	2.0, 2.0	2		13	20	2013	[M5]
HRTM [I7]		Adults A: male 0.78 B: female 0.61	0.1	1.0	1.0	n, a, c	f_{pn}=0.14, f_{pa}=0.75, f_{pc}=0.02	50[f], 230[f], 2 500[f]	1.45, 2.26, 2.26	1.0		A: 18 B: 17	A: 28 B: 27	2014	[B13]

Model type	Aerosol source or action	Breathing rate ($m^3 h^{-1}$)	Unattached aerosol characteristics			Attached aerosol characteristics					Absorbed dose per unit exposure[h] ($mGy\ WLM^{-1}$)	Dose conversion factor		Year	Reference
			f_p[a]	Particle size[b] (nm)	GSD	Mode, i[d]	f_{pi}[e]	Particle size[b f] (nm)	GSD	hgf		mSv per WLM	nSv per h Bq m^{-3} of EEC		
SAGM (IDEAL-DOSE)[c] [H9]	Indoors	0.78	0.08	1.2	1.1	a		150[b]	2.0		T-B[j]: 6.2 AI: 0.17	10	16	2014	[W11]
SAGM (IDEAL-DOSE)[c] [H9, H10]	Home	0.78	0.08	1.0	1.3	a		300[b]	2.1	1.0	T-B[j]: 4.62 AI: 0.15	7.5	12	2015	[W15]
SAGM (IDEAL-DOSE)[c] [H9]	Indoors	0.54 (sitting)	0.08	1.0	1.3	a		300[b]	2.1	1.0	BB: 5.53 bb: 2.34	6.3	10	2015	[H11]
Arithmetic mean Geometric mean Range												11 10 4 to 21	18 16 7 to 34		

[a] f_p is the unattached fraction, in terms of PAEC.

[b] AMTD = activity median thermodynamic diameter.

[c] IDEAL-DOSE code comprises three stochastic models based on Monte Carlo methods, a stochastic particle deposition model, a stochastic bronchial clearance model, and a stochastic cellular dosimetry model [H9, W13].

[d] The attached size distribution may consist of three modes, these are the nucleation mode (n), the accumulation mode (a) and the coarse mode (c).

[e] f_{pi} is the fraction of the attached PAEC associated with mode i. The indices, n, a, and c represent the nucleation mode, the accumulation mode and the coarse mode, respectively.

[f] AMAD = activity median aerodynamic diameter.

[g] Apportionment factors of (A_{BB}=0.80, A_{bb}=0.15, A_{AI}=0.05) were assumed [J1].

[h] The absorbed dose per WLM to regions of the lung. BB = bronchial; bb = bronchiolar; AI = alveolar interstitial.

[i] Absorbed dose per WLM averaged for all target cell nuclei in the lung regions (BB, bb and AI).

[j] Absorbed dose per WLM averaged for all target cell nuclei in the trachea-bronchial tree (T-B).

27. Table 3 summarizes 12 dosimetric assessments for radon exposure in mines under various working conditions. Under mining conditions, the assessments were made for exposure to radon progeny in the historical units of WLM, and for the most recent assessment [S14] in units of mJ h m^{-3}. The assessed dose factors varied from 4 to 23 mSv WLM^{-1} [1.2 to 6.6 mSv per mJ h m^{-3}; 7 to 37 nSv (h Bq m^{-3})$^{-1}$ of EEC] with an arithmetic mean of 12 mSv WLM^{-1} [3.3 mSv per mJ h m^{-3}; 18 nSv (h Bq m^{-3})$^{-1}$ of EEC], and a geometric mean of 11 mSv WLM^{-1} [3.0 mSv per mJ h m^{-3}; 17 nSv (h Bq m^{-3})$^{-1}$ of EEC].

28. Solomon et al. [S14] also made a comparison of the measurement results of December 2013 with the measurement analysis carried out in 1992 [S15]. The 1992 measurement results were interpreted in terms of "diesel" and "non-diesel" areas of the mine and the assessed average effective doses were about 16 mSv WLM^{-1} and 27 mSv WLM^{-1}, respectively with the HRTM model. A re-evaluation of the 1992 measurement results determined sites "away from the actual working mine areas". For these sites the effective dose was calculated as 24 mSv WLM^{-1} similar to the dose calculated for the workshops based on the 2013 measurements. Sites "close to the operating areas of the mine" with higher PAEC values were also identified from the 1992 data. Again, these gave similar doses (15 mSv WLM^{-1}) to the assessed doses based on the 2013 aerosol measurements of operational sites [S14].

29. Winkler-Heil et al. [W13] compared predicted effective doses for radon progeny inhalation obtained using the HRTM, DAGM and SAGM models. The same input parameter values were assumed for mine conditions. The three models yielded similar results: 8.3 mSv WLM^{-1} for the DAGM, 8.9 mSv WLM^{-1} for the SAGM and 11.8 mSv WLM^{-1} for the HRTM. Thus, the HRTM gives a higher value compared with the airway generation models by about 25 to 30%. The authors noted that one of the important issues affecting the comparison is the averaging procedure for the cellular doses calculated in airway generation models. For this comparison, cellular doses were averaged with equal weights over all airway generations, which is the equivalent to calculation of the arithmetic mean. It was also noted that the regional lung deposition fractions for the attached progeny were similar in the BB and bb regions for all three models. However, the HRTM predicted higher deposition fractions in the bb region for the unattached progeny.

30. Target basal and secretory cell nuclei receive a wide range of doses among human bronchial and bronchiolar airways. With the SAGM, distributions of weighted epithelial doses for each airway generation have been calculated taking account of the intra- and inter-subject variability in the morphology of the respiratory tract. These dose distributions are very wide with geometric standard deviations ranging from about 2 to 7 depending on airway generation [H9, H10]. The weighted epithelial doses were obtained by weighting the doses to the basal and secretory cells by their relative volumetric densities. Taking the median of these distributions to compute an average lung dose resulted in effective dose coefficients of 4 mSv WLM^{-1} (7 nSv per h Bq m^{-3} of EEC) for mine exposure [H9, H10]. However, taking the arithmetic mean of all the cellular doses gives rise to a higher effective dose coefficient of 9 mSv WLM^{-1} for mine exposure (14 nSv per h Bq m^{-3} of EEC) [W13, W15].

31. Smoking may change the morphological and physiological parameters of the lung. Lung dosimetry for inhaled radon progeny in smokers was studied by Baias et al. [B1, B2] using the SAGM for four different categories of smokers. These were: *(a)* light, short-term smokers; *(b)* light, long-term smokers; *(c)* heavy, short-term smokers; and *(d)* heavy, long-term smokers. Calculations were performed for a typical mine atmosphere, and for never smokers a dose conversion factor of 7.2 mSv WLM^{-1} was estimated [B2]. Dose conversion factors for light, short-term smokers hardly differed (<1%) from those for never smokers because only small changes in morphological and physiological parameters have been reported. For light, long-term smokers and heavy, short-term smokers, lung doses were reduced due to the thickening of the mucous layer. The majority of the studies reviewed by the authors indicated an average thickening of the mucous layer between 20 to 40%. Assuming a thickening of the mucous layer of 28%, reduced the dose conversion factor by about 10% compared with never smokers. For heavy, long-term smokers, the determining factors

modelled by the authors were: thicker mucous layer, impaired mucociliary clearance, higher breathing frequencies and reduced lung volume due to obstructive lung diseases. The combined effect of these factors increased the dose conversion factor by approximately a factor of two relative to a never smoker. Histological data show that heavy long-term smoking can damage and destroy cilia, and consequently the authors assumed a significant decrease in mucociliary clearance [B2] (see also section III.F, appendix A). It is noted that physiological and morphological data of the influence of smoking vary widely among different sources, so the physiological effects of smoking categorized by the authors represent only rough approximations [I14]. However, it can be concluded that the results of the dosimetric calculations alone cannot account for the large difference in risk between smokers and never smokers due to the sub-multiplicative or multiplicative interaction* between smoking and radon. For example, assuming a multiplicative interaction between smoking and radon, the absolute risk of lung cancer per unit increase in radon is about 25 times greater in current smokers (of 15–24 cigarettes d^{-1}) compared with lifelong non-smokers [D3].

Table 2. Summary of dosimetric assessments for exposure to radon progeny in indoor workplaces

GSD = geometric standard deviation; hgf = hygroscopic growth factor

Model type	*Aerosol source or action*	*Breathing rate ($m^3 h^{-1}$)*	*Unattached aerosol characteristics*			*Attached aerosol characteristics*					*Absorbed dose per unit exposure*[g] *($mGy WLM^{-1}$)*	*Dose conversion factor*		*Year*	*Reference*
			f_p[a]	*Particle size*[b] *(nm)*	*GSD*	*Mode, i*[c]	f_{pi}[d]	*Particle size*[b,e] *(nm)*	*GSD*	*hgf*		*mSv per WLM*	*nSv per h Bq m^{-3} of EEC*		
DAGM [Z5]	With cigarette smoking	1.2	0.005	0.8	1.2	a	1.0	300[e]	2.0	1.0		5.7	9	1999	[P5]
DAGM [Z5]	A: no sources B: with sources & coarse mode	1.2	A: 0.05 (0.02-0.14) B: 0.01 (0.007-0.02)	0.8	1.2	A: n, a B: n, a, c	A: 0.3, 0.7 B: 0.3, 0.6, 0.1	A: 15-40[e], 300[e] (150–450) B: 15–40[e], 300[e], 5 000[e] (3 000–8 000)	A: 1.6–2.2, 2.5 (1.8–4.0) B: 1.6–2.2, 2.5, 1.8 (1.1–2.8)	A: 2.0 B: 1.0		A: 11.5[f] B: 11.2[f]	A: 18[f] B: 18[f]	2001	[P6]
HRTM [I7]	A: A/C on, Vent. off B: A/C off, Vent. off C: A/C on, Vent. On	1.2	A: 0.13 B: 0.10 C: 0.13					A: 196 B: 210 C: 193	A: 1.6 B: 1.6 C: 1.7			A: 22.3 B: 19.9 C: 24.2	A: 35 B: 31 C: 38	2003	[T6]
HRTM [I7]	Mineral treatment industry: Office building	1.2		1.0	1.3	a	1.0	470[e]	2.2	1.0		13	20	2012	[K4]

Model type	*Aerosol source or action*	*Breathing rate ($m^3\ h^{-1}$)*	*Unattached aerosol characteristics*			*Attached aerosol characteristics*					*Absorbed dose per unit exposure*[g] *($mGy\ WLM^{-1}$)*	*Dose conversion factor*		*Year*	*Reference*
			f_p[a]	*Particle size*[b] *(nm)*	*GSD*	*Mode, i*[c]	f_{pi}[d]	*Particle size*[b,e] *(nm)*	*GSD*	*hgf*		*mSv per WLM*	*nSv per h Bq m^{-3} of EEC*		
Revised HRTM [I12]		1.2	0.08	1.0	1.3	n a	0.2 0.8	30[b] 250[b]	2.0 2.0	2 2	BB: 11[h] bb: 11[h] AI: 0.5[h]	20	31	2017	[I13]
Arithmetic mean Geometric mean Range												16 15 6 to 24	25 23 9 to 38		

[a] f_p is the unattached fraction, in terms of PAEC.

[b] AMTD = activity median thermodynamic diameter.

[c] The attached size distribution may consist of three modes, these are the nucleation mode (n), the accumulation mode (a) and the coarse mode (c).

[d] f_{pi} is the fraction of the attached PAEC associated with mode i. The indices, n, a, and c represent the nucleation mode, the accumulation mode and the coarse mode, respectively.

[e] AMAD = activity median aerodynamic diameter.

[f] Apportionment factors of (A_{BB}=0.80, A_{bb}=0.15, A_{AI}=0.05) were assumed [J1].

[g] The absorbed dose per WLM to regions of the lung. BB = bronchial; bb = bronchiolar; AI = alveolar interstitial.

[h] These regional doses averaged between males and females were used to calculate effective doses per WLM for ICRP Publication 137 [I13, M7].

Table 3. Summary of dosimetric assessments for exposure to radon progeny in mines

GSD = geometric standard deviation; hgf = hygroscopic growth factor

Model type	*Aerosol source or action*	*Breathing rate ($m^3\ h^{-1}$)*	*Unattached aerosol characteristics*			*Attached aerosol characteristics*					*Absorbed dose per unit exposure*[k] *($mGy\ WLM^{-1}$)*	*Dose conversion factor*		*Year*	*Reference*
			f_p[a]	*Particle size*[b] *(nm)*	*GSD*	*Mode, i*[d]	f_{pi}[e]	*Particle size*[b f] *(nm)*	*GSD*	*hgf*		*mSv per WLM*	*nSv per h Bq m^{-3} of EEC*		
DAGM [Z5]	A: working + diesel B: without working	A: 1.7[g] B: 1.2	A: 0.006 B: 0.02	0.8		a		A: 200[f] B: 350[f]		1		A: 9.0 B: 6.7	A: 14 B: 11	1999	[P5]
HRTM [I7]		1.2	0.028							1	8.7[h]	21	33	2004	[J3]
HRTM [I7]	Typical mine	1.2	0.01	0.8	1.3	a		250[b]	2.4	2		12.5	20	2005	[M3]
A: HRTM [I7] B: DAGM [W12] C: SAGM (IDEAL-DOSE)[c] [W13]	Uranium mines	1.2	0.01	0.8	1.3	a		250[f]	2.39	2		A: 11.8 B: 8.3 C: 8.9	A: 19 B: 13 C: 14	2007	[W13]
SAGM (IDEAL-DOSE)[c] [W13]	A: never-smoker B: light, short-term smoker C: light, long-term smoker[g] D: heavy, short-term smoker[g] E: heavy, long-term smoker	1.2	0.01	0.8	1.3	a		250[f]	2.39	2		A: 7.20 B: 7.25 C: 6.4[i] D: 6.4[i] E: 13.34	A: 11 B: 11 C: 10[i] D: 10[i] E: 21	2010	[B2]

Model type	*Aerosol source or action*	*Breathing rate ($m^3\ h^{-1}$)*	*Unattached aerosol characteristics*			*Attached aerosol characteristics*					*Absorbed dose per unit exposure*[k] *($mGy\ WLM^{-1}$)*	*Dose conversion factor*		*Year*	*Reference*
			f_p[a]	*Particle size*[b] *(nm)*	*GSD*	*Mode, i*[d]	f_{pi}[e]	*Particle size*[b f] *(nm)*	*GSD*	*hgf*		*mSv per WLM*	*nSv per h Bq m^{-3} of EEC*		
SAGM (IDEAL-DOSE)[c] [H9]	Typical mining condition	1.2	0.01	0.8	1.3	a		250[b]	2.39	1.5	BB: 3.18 bb: 2.25	4.3	7	2010	[H9]
SAGM (IDEAL-DOSE)[c] [H9, H10]		1.2	0.01	0.8	1.3	a		250[b]	2.39	2	BB: 4.5 bb: 2.05	5.2	8.2	2011	[H10]
HRTM [I7]	Uranium mines wet drilling + medium ventilation (no diesel)	1.2	0.01	0.8	1.3	a		250[f]	2.2	1.5	BB: 6.7 bb: 7.0 AI: 0.4	11	18	2012	[M4]
Revised HRTM [I12]	Diesel-powered mine with medium-to-good ventilation	1.2	0.01	0.9	1.3	a		250[b]	2.0	1.0		10.6	17	2013	[M5]
SAGM (IDEAL-DOSE)[c] [H9, H10]	Uranium mine	1.2	0.01	1.0	1.3	a		500[b]	2.4	1.0	T-B[j]: 5.54 AI: 0.09	8.9	14	2015	[W15]
Revised HRTM [I12]	Diesel-powered mine with medium-to-good ventilation	1.2	0.01	1.0	1.3	a		250[b]	2.0	1.0	BB: 4.6[l] bb: 8.3[l] AI: 0.4[l]	11	17	2017	[I13]

Model type	Aerosol source or action	Breathing rate ($m^3 h^{-1}$)	Unattached aerosol characteristics			Attached aerosol characteristics					Absorbed dose per unit exposure[k] ($mGy\ WLM^{-1}$)	Dose conversion factor		Year	Reference
			f_p[a]	Particle size[b] (nm)	GSD	Mode, i[d]	f_{pi}[e]	Particle size[b f] (nm)	GSD	hgf		mSv per WLM	nSv per h Bq m^{-3} of EEC		
Revised HRTM [I12]	A: 420 Drill workshop	1.2	A: 0	B: 0.6	B: 1.1	A: n, a	A: 0.46, 0.53	A: 28[b], 267[b]	A: 1.2, 1.3	1.0		A: 23	A: 36	2018	[S14]
	B: 420 Auto workshop		B: 0.28			B: a	B: 1.0	B: 160[b]	B: 2.0			B: 23	B: 37		
	C: 49MB52 Exploration		C: 0			C: n, a	C: 0.1, 0.9	C: 27[b], 186[b]	C: 1.2, 1.2			C: 14	C: 21		
	D: 39Blue 102, drilling site		D: 0			D: n, a	D: 0.33, 0.67	D: 42[b], 182[b]	D: 1.4, 1.4			D: 17.5	D: 27		
	E:36Yellow 419, drilling site		E: 0			E: n, a	E: 0.33, 0.68	E: 38[b], 179[b]	E: 1.4, 1.7			E: 17	E: 27		
	F: Whenan crusher		F: 0			F: a	F: 1.0	F: 114[b]	F: 1.6			F: 15	F: 24		
Arithmetic mean												12	18		
Geometric mean												11	17		
Range												4 to 23	7 to 37		

[a] f_p is the unattached fraction, in terms of PAEC.

[b] AMTD = activity median thermodynamic diameter.

[c] IDEAL-DOSE code comprises three stochastic models based on Monte Carlo methods, a stochastic particle deposition model, a stochastic bronchial clearance model, and a stochastic cellular dosimetry model [H9, W13].

[d] The attached size distribution may consist of three modes, these are the nucleation mode (n), the accumulation mode (a) and the coarse mode (c).

[e] f_{pi} is the fraction of the attached PAEC associated with mode i. The indices, n, a, and c represent the nucleation mode, the accumulation mode and the coarse mode, respectively.

[f] AMAD = activity median aerodynamic diameter.

[g] At the higher breathing rate of 1.7 $m^3 h^{-1}$, Porstendörfer and Reineking [P5] assumed the miner breathed through the mouth, whereas miners with a breathing rate of 1.2 $m^3 h^{-1}$ were assumed to be nose breathers.

[h] Absorbed dose per WLM averaged for all target cell nuclei in the lung regions (BB, bb and AI).

[i] For light, long-term smoker and the heavy, short-term smoker it is assumed that the mucous thickness increases by 128±5%. However, some publications argued that the increase in mucous thickness is much greater (500±200%), and assuming these values would give an effective dose coefficient of 1.74 mSv WLM^{-1} (3 nSv per h Bq m^{-3} of EEC) [B2].

[j] Absorbed dose per WLM averaged for all target cell nuclei in the trachea-bronchial (T-B) tree.

[k] The absorbed dose per WLM to regions of the lung. BB = bronchial; bb = bronchiolar; AI = alveolar interstitial.

[l] These regional doses averaged between males and females were used to calculate effective doses per WLM for ICRP Publication 137 [I13, M7].

2. Uncertainties and variations in dosimetric assessment

32. Uncertainties in the calculated doses occur due to the uncertainty and variability of model parameter values and due to uncertainties associated with the assumptions built into the particular model. Such uncertainties may arise because the structure of the model provides an over-simplification of the known processes; because the calculations cannot account for unknown processes; or because part of the model structure is based on mathematical convenience rather than the actual processes [I12].

33. Several researchers have carried out uncertainty analyses of the lung dose per unit exposure to radon progeny [B8, H3, H9, I14, M2, W12, Z5]. The main sources of uncertainty considered include:

- The activity size distribution of the radon progeny aerosol including the unattached fraction;
- The breathing rates;
- The model used to predict aerosol deposition in the respiratory tract;
- Lung clearance including mucociliary clearance and absorption into blood;
- The identification of the target cells and their location within the bronchial and the bronchiolar epithelium;
- The inter-subject variation in the morphology of the lung;
- The relative sensitivity of different cell types to radiation;
- The regional differences in the radiation sensitivity of the lung.

34. The range of variations and uncertainties associated with the model parameters were recently assessed by Harley [H3]. There are five major parameters that must be evaluated to model and calculate bronchial airway dose from inhaled radon and its progeny. These are the breathing rate, inhaled activity particle size, airway particle deposition, airway particle clearance, and target cell depth within the bronchial epithelium. Overall, the models and the parameters that define a model showed that dose factors are variable ranging 20 to 50% from a mean value [H3].

35. Marsh and Birchall [M1] performed a sensitivity analysis to identify those HRTM parameters that significantly affect the equivalent dose* to lung (H_{lung}) per WLM under typical conditions found in houses. However, the ICRP alpha radiation factor, w_R was not included in the sensitivity analysis. Using reference parameter values or "best estimate values" proposed by the authors, the effective dose was calculated to be 15 mSv WLM^{-1}. The sensitivity of H_{lung} per WLM to each of the HRTM parameters was examined by varying the values of one while keeping all other parameters equal to their reference values. The parameters most affecting H_{lung} per WLM were the unattached fraction, the unattached aerosol size, the nucleation fraction, the nucleation aerosol size, the breathing rate, the basal cell weighting factor, and the target cell layer depth and thickness of the target cell layer. Table 4 shows the sensitivity of the dose conversion factor (mSv WLM^{-1}) to these parameters, quantified by the reliability factor. The reliability factor is defined as $(B/A)^{½}$, where A and B are the minimum and maximum values of the dose conversion factor per WLM within the given parameter value range, respectively. Thus, all values between A and B are within the reliability factor of the geometric mean of A and B. For the unattached fraction (f_p), ranging from 0.03 to 0.15, the effective dose varies from 13 to 18 mSv WLM^{-1}, an increase of 38%. Without a nucleation mode, the effective dose would decrease from 15 to 11 mSv WLM^{-1}, a decrease of 30%.

Table 4. Sensitivity of the dose conversion factor to HRTM parameters

Best estimate values for aerosol parameters are those proposed by the authors for home conditions [M1]

Description of parameter	*Best estimate or reference value*	*Parameter value range*	*Range of dose conversion factors*[a] *(mSv WLM^{-1})*	*Reliability factor*[b]
Breathing rate	0.78 m^3 h^{-1}	0.45–1.5 m^3 h^{-1} [c]	10–25	1.58
Nucleation aerosol size (AMAD)	50 nm	10–80 nm	12–24	1.36
Unattached fraction (f_p) [d]	0.08	0.03–0.20	13–20	1.26
Nucleation fraction [e]	0.28	0–0.4	11–16	1.21
Basal cell weighting factor [f]	0.5	0–1	12–17	1.19
Target cell layer depth and thickness factor [g]	1	0.5–1	15–21	1.18
Unattached aerosol size (AMTD)	0.9 nm	0.5–3.5 nm	13–18	1.18
Fraction breathed through nose (F_n)	1.0	0.4 [h]–1	15–19	1.13

[a] The dose conversion factor was calculated as $w_{lung}\times H_{lung}=0.12\times H_{lung}$, which approximates the effective dose, whereas w_{lung} is the ICRP lung tissue weighting factor.

[b] The reliability factor is defined as $(B/A)^{½}$, where A and B are the minimum and maximum values of the dose conversion factor per WLM within the given parameter value range, respectively.

[c] The ICRP reference ventilation rates for sleep and light exercise are 0.45 and 1.5 m^3 h^{-1}, respectively [I7].

[d] Expressed as a fraction of the total PAEC.

[e] Expressed as a fraction of the attached PAEC.

[f] HRTM assumes that the basal and secretory cell populations in the bronchial epithelium are equally sensitive to dose [I7]. Thus $H_{BB}=0.5H_{bas}+0.5H_{sec}$. A basal cell weighting factor of 1.0 means that $H_{BB}=H_{bas,}$ whereas a basal cell weighting factor of zero means that $H_{BB}=H_{sec}$.

[g] This parameter is a multiplying factor by which the target cell layer depth and thickness of the target cell layer are multiplied.

[h] ICRP Publication 66 recommends an F_n value of 0.4 for a mouth breather performing light exercise [I7].

36. The effective dose is very sensitive to particle sizes of the unattached mode (0.5 to 5 nm) and the nucleation mode (10 to 100 nm). However, it is less sensitive to particle sizes in the region of 150 to 900 nm corresponding to the accumulation mode [I13, I14, J3, P6].

37. Marsh et al. [M2] carried out a parameter uncertainty analysis with the HRTM to calculate the probability distribution of the effective dose per unit exposure to radon progeny in the home. Parameters considered included: *(a)* aerosol parameters; *(b)* subject related parameters such as breathing rate, fraction breathed through the nose, and particle transport rates; *(c)* target cell parameters such as depth of basal and secretory cell layer; and *(d)* absorption rates of attached and unattached radon progeny. The resulting distribution was approximately log-normal with a geometric mean of 14 mSv WLM^{-1} (95% confidence interval* corresponded to 6.3–31 mSv WLM^{-1}) and a geometric standard deviation of 1.5. Birchall and James [B8] also carried out a parameter uncertainty analysis with the HRTM to calculate the distribution of effective doses per unit exposure to radon progeny in a mine and obtained similar results for the statistical parameters of the dose distribution.

38. Hofmann et al. [H9] carried out an uncertainty analysis with the SAGM to derive the frequency* distribution of bronchial doses per WLM. The inter- and intra-subject variability of the airway

dimensions, the respiratory parameters, the mucociliary clearance rates and the depth of the target cells were all considered, while aerosol parameter values were fixed for a "typical" mine atmosphere. In the model, the depth of the target cells is assumed to be related to the airway diameters. Resulting bronchial dose distributions were approximated by log-normal distributions; BB: median=3.2 mGy WLM^{-1}, σ_g=2.3; and bb: median=2.3 mGy WLM^{-1}, σ_g=4. The median values correspond to an effective dose coefficient of 4 mSv WLM^{-1} (7 nSv per h Bq m^{-3} of EEC). The geometric standard deviations, σ_g of the bronchial dose distributions for a given airway generation ranged from about 2 to 3 in airway generations 1–9 and from 3 to 7 in airway generations 10–20. Thus, the inter-subject variations were significantly higher in the peripheral bronchial airways than in the larger bronchial airways. The results also show that a small fraction of cells in each airway generation may receive doses that are roughly an order of magnitude higher than the median doses, particularly for the peripheral airways. The parameters that most affected the dose distribution were the asymmetry and variability of the airway dimensions, the filtering efficiency of nasal passages, and the depth of the target cells. The dose distributions are much wider than the distributions obtained with the HRTM [M2, M4] indicating that inter-subject variability in the morphology of the respiratory tract is an important source of uncertainty in the dose calculations.

39. Hofmann and Winkler-Heil [H11] investigated the effects of different weighting procedures for combining target cell doses to calculate an average lung dose with the SAGM. These included equal weighting for basal and secretory cells and weighting factors based on: *(a)* relative frequencies of basal and secretory cells; *(b)* number of progenitor cells; *(c)* contribution of dose enhancement at airway bifurcations; and *(d)* promotional effect of cigarette smoking. Calculations were performed assuming aerosol parameters for indoor exposure with a breathing rate for sitting (0.54 $m^3\ h^{-1}$). Assuming the ICRP apportionment factors of (A_{BB}=0.333, A_{bb}=0.333, A_{AI}=0.333) [I7], the detriment-weighted absorbed dose to lung range from 2.1 to 3.4 mGy WLM^{-1}; (corresponding to dose conversion factors between 6.3 and 8.2 mSv WLM^{-1}). This represents a variation of a factor 1.6. However, assuming (A_{BB}=0.80, A_{bb}=0.15, A_{AI}=0.05) [J1], the variation in the lung doses is greater with dose conversion factors ranging from 6.0 to 21 mSv WLM^{-1}, which represents a variation of a factor 3.5.

E. Summary of dosimetric assessments

40. Published results of dosimetric calculations carried out with the HRTM, DAGM or SAGM models are summarized in tables 1 to 3 for radon progeny exposure in homes, indoor workplaces and mines. The absorbed doses to regions of the lung and the corresponding effective dose per unit radon exposure are given. Due to uncertainties associated with the model parameter values and with the assumptions built into particular models, the variation of the assessed effective dose coefficients is large. The mean values of effective dose coefficients calculated with dosimetric models are similar for exposure in homes and in underground mines (18 nSv per h Bq m^{-3} of EEC of ^{222}Rn; tables 1 and 3). However, the assessed mean effective dose coefficient is higher for indoor workplaces (25 nSv per h Bq m^{-3} of EEC of ^{222}Rn) compared with that for home exposure. This is mainly because the assumed breathing rate is higher for a worker compared with an adult at home.

41. For home exposure, the range of the assessed effective doses per unit of exposure of EEC of ^{222}Rn are from 7 to 34 nSv per h Bq m^{-3} with an arithmetic mean of 18 nSv per h Bq m^{-3}, and a geometric mean of 16 nSv per h Bq m^{-3}. This is similar to the central value of 15 nSv per h Bq m^{-3} previously estimated by the Committee in the range of 8–40 nSv per h Bq m^{-3} of EEC of ^{222}Rn for average indoor conditions based on dosimetric evaluations [U4, U6].

III. EPIDEMIOLOGY

A. Introduction

42. Radon (^{222}Rn) is widely regarded as the second leading cause of lung cancer among tobacco smokers and the leading cause among non-smokers. Already in 1988, the International Agency for Research on Cancer (IARC) classified radon as a proven human carcinogen and placed radon in the same IARC carcinogen group as tobacco smoke, asbestos and benzene [I1]. Accordingly, there is great interest in considering the exposure to radon as a public health issue and in estimating the lung cancer risk from exposure to radon in both occupational and residential settings. The World Health Organization (WHO) provided several guidelines and recommendations to the management of health risk related to radon as an indoor air pollutant [W7, W8, W9]. A recent analysis of 66 countries conducted by Gaskin et al. [G1] concluded that 16.5% of lung cancer cases were attributable to residential radon exposure.

43. The Committee has previously considered the issue of effects from radon exposure (including ^{220}Rn commonly known as thoron) with regard to workers and the public in two comprehensive reports in 2000 and 2006 [U3, U6]. The Committee also concluded that inhaled radon and its progeny are established carcinogens for the lung, however, doses to other organs and tissues were at least an order of magnitude smaller than the doses to the lung. In the UNSCEAR 2006 Report, studies of miners occupationally exposed to radon were noted to provide a direct basis for assessing their lung cancer risk [U6]. The sixth report from the United States National Research Council's Committee on Health Risks of Exposure to Radon in the study series Biological Effects of Ionizing Radiation (BEIR VI) had previously estimated an excess relative risk (ERR)* of 0.012 (95% CI: 0.002, 0.025) per WLM for miners with radon exposure below 50 WLM [N10]. The Committee reviewed in its UNSCEAR 2006 Report [U6] cohort studies of miner cohorts that had been updated since BEIR VI and found the general patterns of risk with dose and time since exposure to be consistent with the BEIR VI findings. Because of high exposure levels in the early days of the mining industry, studies of miners were identified as providing a strong basis for investigating the effects of potential modifiers of the dose-response relationship, such as age at exposure, attained age*, radon exposure rate and time since exposure.

44. Prior to the UNSCEAR 2006 Report, radon concentrations in mines were routinely extrapolated down to those typical in homes to provide an indirect basis for assessing the lung cancer risk from residential exposure to radon [U6]. However, by the time of the report, direct risk estimates were available from over 20 published epidemiological studies of residential exposure. The Committee evaluated these studies and identified the pooled analyses of European, North American, and Chinese residential case-control studies as the primary sources of synthesized residential risk estimates. Based on the available evidence, it was concluded that there was a small but detectable increased frequency of lung cancer from residential exposure that increased with increasing exposure. In particular, the Committee recommended adoption of the uncertainty-corrected excess relative risk estimate of 0.16 (95% CI: 0.05, 0.31) per 100 Bq m^{-3} from a pooled analysis of the European studies [D3]. Furthermore, smokers were inferred to account for nearly 90% of the population average risk from residential radon due to a synergistic interaction between the effects of radon exposure and inhalation of tobacco smoke.

45. Although there are major uncertainties in extrapolating lung cancer effects of radon exposure in mines to the residential setting, good agreement was observed between risk estimates derived from miner studies and those derived from residential case-control studies. Nevertheless, the Committee concluded in the UNSCEAR 2006 Report that more work is needed to better understand and account for the influence of modifying and confounding factors, especially tobacco smoking [U6].

B. Aim and objectives

46. The main aim of the present literature review is to update the UNSCEAR 2006 Report [U6] with the best and most up-to-date knowledge concerning lung cancer risk due to residential and occupational radon exposure, and uncertainties in exposure assessment. Specific objectives are to address the following research questions:

- What is the current status of evidence regarding increased frequency of lung cancer for smokers and non-smokers, for subgroups of different age and sex, due to exposure to radon?
- What is the confidence in any inferred risk estimates?

C. Scope and literature search

47. The scope of the review is primary peer-reviewed studies published in the scientific literature and publications of relevant international organizations. The focus is on epidemiological studies of lung cancer risk due to radon exposure.

48. A comprehensive literature review was conducted of scientific journal articles published since the UNSCEAR 2006 Report [U6] to identify epidemiological study results providing new information on the lung cancer risk associated with radon. The selection criteria developed by the Committee in its UNSCEAR 2017 Report [U8] were applied for the review. A literature search in PubMed, Scopus, and Web of Science was performed with the query string “Search (((((“lung” or “pulmonary” [Title/Abstract]) AND “cancer”, “carcinoma”, or “neoplasm” [Title/Abstract])) AND (""01/01/2006-""[Date - Publication] : ""31/12/2017""[Date - Publication]))) AND radon [Title/Abstract]” resulted in 261 entries. The results from the literature searches were merged, where duplicates were discarded. The articles were reviewed with respect to authors’ potential conflicts, publication type, and screening and quality questions. Screening questions were discussed and agreed prior to the review.

49. Epidemiological studies that did not meet the screening questions were excluded from the review. This included non-original publications, studies in non-human populations, and risk assessments in diseases other than lung cancer. Publications meeting the requisite screening questions were further assessed with respect to their quality. Based on the quality questions, a final decision on whether to include/exclude studies in the evaluation were made. Study item reviews, final inclusion/exclusion decisions, and reasons for the decisions were recorded in a master file, along with summaries of findings and appraisals for included studies. A selected subsample of 35 publications underwent a secondary review to identify potential differences in the application of review criteria. Differences were discussed and reconciled among the experts, and prior reviews were updated accordingly.

D. Review of epidemiological studies for occupational exposure

1. Overview of cohort studies

50. The first papers studying occupational health risks from radiation exposure among uranium miners were published in the 1960s and early 1970s [A7, L13, M10, S7, W1]. A first comprehensive study using the same methodology was published in the BEIR IV report [N8], which included cohort studies of

Colorado Plateau, Ontario, Eldorado, and Malmberget miners (table 6). The report's final model of relative risk included modifying factors of time since exposure and age. In 1999, the BEIR IV report was substantially extended to include 11 miner studies resulting in the BEIR VI report [N10] (table 5). The BEIR VI model of relative risk included modifying factors of time since exposure (5–14, 15–24, 25+ exposure windows), age (four categories), and exposure rate (six categories). Since the BEIR VI report, several studies were extended and in Germany a new study (Wismut) was established. These studies were described and evaluated in detail in the UNSCEAR 2006 Report [U6] (table 7).

Table 5. Summary information of studies included in the 1999 BEIR VI report [N10]

Study	*Period of follow-up*	*Number of cohort members*[a]	*Number of person-years*	*Number of lung cancers*	*Mean cumulative exposure (WLM)*	*ERR per 100 WLM*[b]
Beaverlodge[c]	1950–1980	6 895	67 080	56	21.2	2.33
China	1976–1987	13 649	134 842	936	286.0	0.17
Colorado	1950–1990	3 347	79 556	334	578.6	0.44
Czechoslovakia[d]	1952–1990	4 320	102 650	701	196.8	0.67
France	1948–1986	1 769	29 172	45	59.4	0.51
New Mexico	1943–1985	3 457	46 800	68	110.9	1.58
Newfoundland	1950–1984	1 751	33 795	112	388.4	0.82
Ontario	1955–1986	21 346	300 608	285	31.0	0.82
Port Radium[a]	1950–1980	1 420	30 454	39	243.0	0.24
Radium Hill	1948–1987	1 457	24 138	31	7.6	2.75
Sweden	1951–1991	1 294	32 452	79	80.6	1.04
Total[e]		**60 606**	**888 906**	**2 674**		**0.76**

[a] Excluding miners with zero exposure.

[b] Two-stage statistical modelling approach.

[c] Subsequently included in Eldorado study.

[d] The former Czechoslovak study included data of Czech and Slovak miners until 1993. Afterwards it is known as Czech study.

[e] Totals adjusted for miners and lung cancers that were included in both the Colorado and New Mexico studies.

Table 6. Summary information of studies included in the 1988 BEIR IV report [N8]

Study	*Period of follow-up*	*Number of cohort members*[a]	*Number of person-years*	*Number of lung cancers*	*ERR per 100 WLM (95% CI)*
Colorado	1951–1982	3 347	73 642	256	0.6 (0.3, 1.3)
Eldorado	1950–1980	6 847	114 170	65	2.6 (1.3, 6.0)
Malmberget	1951–1976	1 292	27 397	51	1.4 (0.3, 8.9)
Ontario	1955–1981	11 076	217 810	87	1.4 (0.6, 3.3)
Total		**22 562**	**433 019**	**459**	**1.3 (0.8, 2.3)**

[a] Including miners with zero exposure.

Table 7. Summary information of studies of miners included in the UNSCEAR 2006 Report [U6]

Study	*Period of follow-up*	*Number of cohort members*[a]	*Number of person-years*	*Number of lung cancer*	*Mean cumulative exposure (WLM)*	*ERR per 100 WLM (95% CI)*
Beaverlodge[b]	1950–1999	10 050	285 964	279	23.2	0.96 (0.56, 1.56)
China	1976–1987	13 639	135 357	936	277.4	0.16 (0.1, 0.2)
Colorado	1950–1990	3 347	75 032	327	807.2	0.42 (0.3, 0.7)
Czech Republic	1952–1999	9 941	261 428	915	70	1.6 (1.2, 2.2)
France	1946–1994	5 098	133 521	125	36.5	0.8 (0.3, 1.4)
Newfoundland	1950–2001	1 751	70 894	206	378	0.47 (0.28, 0.65)
Ontario	1955–1986	21 346	319 701	282	30.8	0.89 (0.5, 1.5)
Port Radium[b]	1950–1999	3 300	111 222	230	174.2	0.37 (0.23, 0.59)
Sweden	1951–1991	1 294	32 452	79	80.6	0.95 (0.1, 4.1)
Wismut	1946–1998	59 001	1 801 626	2 328	242	0.21 (0.18, 0.24)
Total[c]		**69 766**	**1 425 571**	**3 379**		**0.59 (0.35, 1.0)**

[a] Including miners with zero exposure.

[b] Subsequently included in Eldorado study.

[c] Totals and combined ERR estimate exclude the Wismut cohort, which was under development at the time of the report [U6].

51. Table 8 summarizes the occupational studies published in the period of 2006–2017 and reviewed in this section. Included among these are further extensions of eight existing studies and new results from the Wismut study. Recently published numbers are provided for the cohort sizes, person-years of follow-up, observed lung cancers, and estimated average excess relative risk. Overall, the extended data from the German, Czech, French, Canadian, and United States' studies represent 1,110,287 additional person-years of follow-up and 2,874 new lung cancers accumulated since the UNSCEAR 2006 Report [U6]. Descriptive summaries of the updated cohort studies are presented in this section.

(a) *Colorado Plateau uranium miners*

52. Studies of uranium miners in the Colorado Plateau area were started in 1949 by the United States Public Health Service, in cooperation with the United States Atomic Energy Commission and the state health departments of Arizona, Colorado, New Mexico and Utah. From 1950 to 1960, a cohort of 4,137 males who had worked in a uranium mine in the area for at least one month was established. The cohort was followed through 2005 and linked to national databases to ascertain vital statuses and causes of death [S5].

53. Radon exposure assessment relied on estimates from the early 1970s. Exposure information consisted of the dates on which each miner's exposure to radon progeny reached 1 of 9 cumulative exposure levels (0, 60, 120, 240, 360, 600, 840, 1,800, and 3,720 WLM). Daily exposure for each were then interpolated between the reported dates. Exposure to radon progeny from prior non-uranium mining was included as occurring on the day before uranium mining employment began. Smoking status was available for the miners from surveys conducted up until 1985.

54. In an update of the Colorado Plateau cohort covering the period of 1991–2005, Schubauer-Berigan et al. [S5] calculated a standardized mortality ratio (SMR) for lung cancer of 3.99 (95% CI: 3.43, 4.62) for white miners and 3.27 (95% CI: 2.19, 4.73) for American Indian miners. Keil et al. [K1] subsequently used G-estimation of structural nested models to adjust for healthy worker survivor bias* in estimating the time ratio (relative change in median survival time) associated with radon exposure. In their statistical analysis, the authors estimated that median lung cancer survival time decreased by a factor of 1.168 (95% CI: 1.152, 1.174) per 100 WLM.

(b) *Czech uranium miners*

55. The cohort was established in 1969 using a database of approximately 100,000 Czech and Slovak uranium miners. The selected cohort included 4,364 miners first employed underground since 1948 in the former Czechoslovak uranium industry for at least four years (S subcohort). First analyses based on 244 lung cancers were reported in 1971 [S7]. By 1970, the cohort was extended by 5,625 miners firstly employed since 1969 for at least one year (N subcohort), first report on this study was in 1997. The study population of the cohort (after excluding duplicates) now consists of a total of 9,978 miners. The mean duration of exposure in the entire cohort was seven years. Information on vital status and dates of death were obtained from the Czech Population Registry at the Ministry of the Interior. Before 1982, the causes of death were obtained from local death registries, and since 1982 from the Institute of Health Information and Statistics of the Czech Republic.

56. Details of exposure estimation have been described elsewhere [S7, T9]. Briefly, exposure estimates in the S subcohort were derived from extensive measurements of radon that had already commenced in 1949. On average, there were about 200 measurements of radon gas per each shaft and year. The concentrations of radon gas were converted to concentrations of radon progeny using measurements of equilibrium factors* in mines also when ventilation was not operated for one month. Each miner's annual exposure to radon progeny was estimated, combining measurement data with registered employment details, including duration of underground work at different shafts and job category. In the N subcohort the exposure estimates were based on personal dosimetric records, based on several thousand measurements per year and shaft. Exposure of some miners in both cohorts involved work in non-uranium mines. Their exposure (substantially lower than those in uranium mines) were derived from radon measurement in non-uranium mines [S6].

57. Results from the Czech study were reported in 1971 [S7], more detailed analyses in 1988 [S8], and further summarized in 1990s publications [T9, T10]. The last report [T15] included 1,141 lung cancers observed in the cohort by 2010. The relative risk models included all modifying factors like attained age, age at exposure, time since exposure, and exposure rate. Smoking was assessed in a nested case control study within the cohort [T16]. This study resulted in an evaluation of the risks from radon and smoking based on 1,029 lung cancer cases and 2,648 controls matched by year of birth, age, and subcohort. Data on smoking in the study were collected from subjects by personal interview, from medical records, and from relatives. The combined effect from radon and smoking was analysed in terms of geometric mixture models of which the additive and multiplicative models are special cases. The resulting model is relatively close to the additive interaction (mixing parameter of 0.2).

(c) *Eldorado miners*

58. The previous analyses of the Beaverlodge mine workers were based on 10,945 male workers employed at the mine between 1948 and 1980 during 1950–1980 [H13, H15]. The cohort was updated to include 305 workers who joined the Beaverlodge mine between the cut off of the original study and the

final closure of the mine in June 1982 [L3]. Improvements in the quality and quantity of identifying data in the nominal cohort and work histories resulted in a final updated cohort of 11,788 workers.

59. The previous analysis of the Port Radium uranium mine workers included all male workers employed at the mine between 1942 and 1960 and known to be alive at the start of follow-up in 1950 and followed up for mortality during 1950–1980 [H14]. The original nominal cohort included 2,696 workers, many of whom were excluded from linkage and analysis because of missing date of birth information [H14]. After an extensive search and review of original records, the updated nominal cohort was expanded to include 4,079 workers.

60. Mortality follow-up of Beaverlodge and Port Radium cohorts was extended from 1980 to the end of 1999 and, for the first time, incidence analysis was conducted for cancers occurring during 1969–1999. For updated follow-up, these two cohorts were combined and analysed together with workers of the Port Hope radium and uranium refinery and processing facility as the Eldorado cohort study [L3]. In total, the Eldorado cohorts includes 17,660 workers. Analyses were restricted to 16,236 (91.9% of the cohort) male workers with a mean cumulative radon exposure of 100 WLM (117 WLM, weighted by person–years, among those with non-zero exposure). Lung cancer was the only cancer site with significantly elevated death (SMR=1.31; 95% CI: 1.21, 1.42) and incidence rates (SIR=1.23; 95% CI: 1.14, 1.33) compared to the general Canadian male population. The excess relative risk per 100 WLM of lung cancer mortality (n=618; ERR per 100 WLM=0.55; 95% CI: 0.37, 0.78; p<0.01) and incidence (n=626; ERR per 100 WLM=0.55; 95% CI: 0.37, 0.81; p<0.001) increased linearly with increasing radon exposure. Adjustment for effect modification by time since exposure, exposure rate and age at risk resulted in comparable estimates of risk of lung cancer. All models were adjusted for age at risk, calendar year at risk and duration of employment by stratification [L3].

61. Recently, Lane et al. [L4] assessed the risk of lung cancer mortality at low radon exposure (<100 WLM), among a joint cohort of Czech, French and Beaverlodge miners employed when quality radon exposure measurements were available (1953, 1956, and 1965, respectively) and radiation protection programmes were in place. For Beaverlodge, an ERR per 100 WLM estimate of 2.4 (95% CI: 0.9, 4.7) was obtained with a Poisson regression model for grouped survival data, with background stratification by age at risk categories. Similar to studies with smoking information [B9, H7, L7], two sensitivity analyses found that smoking was not a strong confounding factor in the joint cohort. A statistically significant decrease in risk with time since exposure was observed. Exposure rate was not an effect modifier, consistent with other studies with quality radon measurements, low exposure and low exposure rates [K6, N10, T4, T12]. The models were adjusted for age at risk [L4].

62. A separate analysis of Port Hope radium and uranium refining and processing workers with no mining experience was published after the update of the Eldorado cohort study [Z2]. This analysis was based on a slightly different cohort with more detailed exposure information, including exposure to radium and uranium, than was included in the Eldorado cohort [L3] and excluded all workers with any mining experience. This analysis also used different analytical methods than in the Eldorado cohort to analyse and compare mortality and cancer incidence in the cohort with special attention to cancers of the lung and bronchi, leukaemia and lymphoma, bone, liver and kidney cancers, as well as non-malignant respiratory, renal and cardiovascular diseases.

63. The Port Hope radium and uranium refinery and processing plant became operational in 1932 and continues to operate today as Cameco Corporation Port Hope Conversion Facility. Port Hope workers were exposed to a wide variety of chemicals and radiation types. In addition to gamma radiation and radon decay product radiation, they were exposed to relatively concentrated forms of uranium (sulphates and nitrates during the refining years and UO_2, UO_3 and uranium fluorides more recently) through inhalation and ingestion. Workers were also exposed to radium, which tends to naturally concentrate in

the bones, potentially exposing the surrounding tissues, including bone marrow, to ionizing radiation. There were no early radon measurements taken at Port Hope at the time of the start-up in 1932. In the 1930s to 1950s, radon estimates were based on the quantities of radium present in the plant in ore and at various stages of refinement, measured radon emanation rates from various radium-bearing materials, building air volumes and estimates of air exchange rates. In the early 1970s, radon measurements were conducted in the yellowcake warehouses, but occupancy was generally low, and no exposure estimates were made. The individual annual exposure in working level months were calculated from working level (WL)* estimates for each type of workplace, the proportion of employees in each occupation and the proportion of time spent in each type of workplace by employees in each occupation. Gamma radiation was the primary type of radiation exposure at Port Hope. There were no measurements at the time of start-up. Film badges were used on some individuals in the late 1940s and were worn by most radium workers and a sampling of others from mid-1947 to early 1953. Full individual external dosimetry (100% coverage) was in place by about 1970 and individual records were kept. Individual gamma doses were calculated from the average dose rates and time on the job and expressed in millisieverts for everyone who had not been wearing a personal dosimeter. All gamma doses were whole-body effective doses.

64. The Port Hope cohort includes about 3,000 workers who were employed between the age of 15 and 75 years sometime between 1932 and 1980, had their last contact after 1940 and never worked in underground or open-pit mines [Z2]. All workers were included regardless of the duration of employment. In analyses restricted to males (n=2,645), the person-year weighted mean cumulative radon exposure was 15.9 WLM and the mean cumulative whole-body gamma radiation dose was 134.4 mSv. Risks of lung cancer mortality and incidence were comparable with the general Canadian male population (SMR=1.08; 95% CI: 0.88, 1.32 and SIR=1.08; 95% CI: 0.89, 1.30, respectively). Small, non-statistically significant increases in radiation risks of mortality and incidence of lung cancer due to radon exposure (n=99; ERR per 100 WLM=0.21; 95% CI: <−0.45, 1.59 and n=108; ERR per 100 WLM=0.77; 95% CI: <−0.19, 3.39, mortality and incidence, respectively), were reported, with similar risks for those exposed to radium and uranium [Z2]. All other causes of mortality and cancer incidence were not significantly associated with radon exposure or gamma radiation doses or a combination of both.

(d) *French uranium miners*

65. Vacquier et al. [V1] described the results conducted on the updated French uranium miner cohort after the extension of the follow-up until 1999. The cohort included 5,086 miners employed at least one year by the CEA-COGEMA group between 1946 and 1990 and followed up during 30.1 years in average. Among the 1,411 deaths, 159 were from lung cancer. A significant excess of mortality from lung cancer was found again in the cohort (SMR=1.43; 95% CI: 1.22, 1.68) and this excess was significantly associated with cumulative radon exposure (ERR per 100 WLM=0.58; 95% CI: 0.20, 1.17, on the base of a Poisson regression model with internally defined baseline risk, or ERR per 100 WLM=0.71; 95% CI: 0.29, 1.35, on the base of a Poisson regression model with baseline risk assumed proportional to an external reference population).

66. Radon levels* in French uranium mines decreased sharply in 1956 with the setting up of the radioprotection measures and the introduction of the forced ventilation in mines; mean annual radon exposure was 21.3 WLM before 1956 and 1.7 WLM after 1956. A sensitivity analysis was conducted according to the period of exposure and showed a higher ERR per 100 WLM of 2.0 (95% CI: 0.91, 3.65) after 1956 compared to an ERR per 100 WLM of 0.20 (95% CI: NA, 0.70) before 1956.

67. The modifying effect of time-dependent factors and other non-temporal factors on the relationship between cumulative radon exposure and lung cancer risk has been studied among French uranium miners [V2]. The method of windows of cumulative exposure defined according to the modifying factors was used

and the ERR assessment was based on internal Poisson regression. Period of exposure appeared as a major modifying factor with an ERR per WLM ten times higher for exposure received after 1956 compared to those received before 1956 (ERR per 100 WLM=2.09 (standard error 0.68) and ERR per 100 WLM=0.21 (standard error 0.22)). The ERR per WLM decreased significantly with the time since exposure. A possible inverse exposure rate effect was suggested due to a decrease in the ERR per WLM especially for exposure rate greater than 1 WL per year. Mine type and physical activity appeared to be also strong modifying factors: ERR per WLM was significantly higher for underground miners (compared to open pit miners) and for miners with high physical activity (compared to low/intermediate activity).

68. The systematic measurements of the exposure to radon, long-lived radionuclides, and external gamma radiation began in 1956 in the French uranium mines. The influence of these multiple ionizing radiation exposure was analysed among the 3,377 French uranium miners hired after 1955 in the post-55 subcohort by Vacquier et al. [V3]. A significant excess of mortality from lung cancer was still observed (n=66; SMR=1.30 [1.01–1.65]). The ERR for lung cancer death was significantly associated with cumulative radon exposure (ERR per 100 WLM=2.12; 95% CI: 0.53, 5.28), and also with the other exposure. But methodological limits due to the strong correlation between exposure and a lack of statistical power did not allow to take into account multicollinearity and the estimated effect of each separate ionizing radiation exposure.

69. The absorbed dose to lung has been calculated in the French post-55 subcohort by Rage et al. [M4, R2]. The lung dose results from the chronic exposure* to alpha particle emitters, i.e. radon gas, radon short-lived progenies, and long-lived radionuclides, and to low-linear energy transfer radiations, i.e. gamma radiation. Radon short-lived progenies contributed to 97% of the absorbed lung dose caused by alpha particle emitters, which are the main contributor with 58% of the absorbed lung dose. Consequently, the dose-risk relationship was driven primarily by radon short-lived progenies exposure. When considering the value of relative biological effectiveness (RBE)*=20 for alpha particles, generally accepted for alpha emitters, the ERR per Sv associated with the total dose was equal to 0.22 (95% CI: 0.06, 0.53) and with the radon short-lived progenies was equal to 0.23 (95% CI: 0.07, 0.56).

70. The effect of chest X-ray screening examinations on the exposure-risk relationship was studied by Laborde-Castérot et al. [L1]. Chest X-ray exposure received by French uranium miners during their occupational medical surveillance was assessed from different scenarios based on the type of procedure, period, frequency, and the lung dose delivered. No evidence of a confounding effect of X-ray exposure was observed. However, X-ray exposure was not associated with lung cancer mortality and did not confound the dose-risk relation between radon exposure and lung cancer mortality.

71. In the last update of the French cohort, an additional eight years have been added [R3]. At the end of 2007, the duration of follow-up was 35.1 years in average and a total of 1,935 deaths were counted including 211 from lung cancer. Mortality analysis confirmed the significant increased risk for lung cancer death with a SMR=1.34; 95% CI: 1.16, 1.53. Among the post-55 subcohort, the excess of mortality from lung cancer remained borderline (SMR=1.18; 95% CI: 0.96, 1.45). Excess relative risk remained significantly associated with cumulative radon exposure both in the French cohort and the post-55 subcohort (ERR per 100 WLM=0.71; 95% CI: 0.31, 1.30 and ERR per 100 WLM=2.42; 95% CI: 0.90, 5.14, respectively).

(e) *New Mexico uranium miners and millers*

72. The cohort of New Mexico uranium miners has been previously presented in the BEIR VI and UNSCEAR 2006 Reports [N10, U6]. It included 3,457 underground uranium miners with at least one year of mining experience [S2]. Another retrospective cohort study consisted of 2,745 workers engaged

in uranium mining and milling activities near Grants, New Mexico, during 1955–1990. Some of the Grants underground miners were likely included in the previous cohort of miners in New Mexico published by Samet et al. [S2]. Assignment of exposure potential to uranium ore or processing was based on job titles. Workers vital status and causes of death were determined from various linkages with national databases. Smoking histories were not known. Boice et al. [B10] reported disease-specific mortality for the 63,395 person-years of follow-up accumulated during 1979–2005. Overall, statistically significant increased numbers of deaths were found for lung cancer (SMR=1.65; 95% CI: 1.36, 1.97; *n*=117). In stratified analyses, an excess number of lung cancer deaths was observed for workers with underground mining experience (SMR=2.17; 95% CI: 1.75, 2.65; *n*=95) but not for those without such experience (SMR=0.85; 95% CI: 0.52, 1.29; *n*=21). No statistically significant elevation in any cause of death was seen among the 904 non-miners employed at the Grants uranium mill.

(f) *Newfoundland fluorspar miners*

73. The Newfoundland fluorspar cohort was updated in 2007 with an 11-years extension of follow-up to 2001 [V10]. The cohort included 1,742 underground miners with a mean exposure of 378 WLM and 328 unexposed surface miners. Underground miners were exposed to radon from water running through the mine, which eliminated the possibility of confounding from gamma radiation or thoron. The SMR for lung cancer in the underground miners was 3.09 (95% CI: 2.66, 3.56) based on 191 lung cancer deaths. The ERR per 100 WLM among underground miners adjusted for age and calendar period with a 5-year lag was 0.43 (95% CI: 0.23, 0.62). The ERR per WLM decreased with attained age and time since last exposure and an inverse dose-rate effect was also observed. Smoking history was available for 53% of the full cohort. The ERR per WLM for ever smokers did not differ significantly from never smokers but did increase with cigarettes smoked per day.

(g) *Ontario uranium miners*

74. The Ontario uranium miners' cohort was updated with 21 additional years of mortality follow-up (through 2007), as well as cancer incidence follow-up through 2005 [N1]. The cohort was also expanded through broader inclusion criteria and by identifying additional previous miners through Canada's National Dose Registry, which was also used to improve the assessment of radon exposure. The cohort now includes 28,959 miners who worked for at least one week between 1954 and 1996, when mining operations ceased. Analyses were restricted to 28,546 male miners with a mean cumulative radon exposure of 21.0 WLM and increased lung cancer mortality (SMR=1.34; 95% CI: 1.27, 1.42). Based on 1,230 lung cancer deaths, the ERR per 100 WLM was 0.66 (95% CI: 0.44, 0.87). Results were similar for lung cancer incidence. All models were adjusted for the calendar period and attained age. A subsequent paper assessed uncertainty in radon exposure assessment due to natural variations in radon concentration, estimation of working time, precision of the radon measurement method, unintended errors during sampling, and record keeping and transcription of exposure data using the root sum square method [N2]. Exposure uncertainty was found to be 53 to 67% prior to 1968 and 31 to 39% for the period 1968 to 1996 with natural variations being the most important source of uncertainty.

(h) *Swedish iron miners*

75. The Swedish cohort among iron underground miners exposed to radon is one of the oldest studies [A8, D1, E1, J5, R1, S13]. The cohort participated in joint studies of BEIR IV and BEIR VI. The iron ore mine was established in 1890. In the early 1900s, this mine was mainly open cast but by 1920 about 80%

of mining took place underground. Originally the mine only had natural ventilation, forced ventilation was gradually introduced during 1950s and 1960s. Radon measurements began in 1969 and were improved in 1972–1973. The exposure matrix was specified over time for different ore bodies being constant from 1974 until 1998. Data from 1969–1972 were considered as representative of earlier periods; the level in 1910 was set to 380 Bq m^{-3} followed by linear increase from 1910 until 1925.

76. Jonsson et al. [J4] focused on a cohort of surface and underground workers who had worked at the mine between 1923 and 1996. Employment data were collected from manual work records in years 1923–1981 and from computerized register since 1982. Follow-up relied on the Swedish personal registry. Combining the two sources resulted in a cohort of 5,588 males. Mortality data in the period 1952–2000 were obtained by record linkage to the national cause of death register and the national population register. After excluding males not found in the registries, the cohort consisted of 5,486 males. Since lung cancer morbidity was available only for years 1958–2000, the cohort was further restricted to 5,449 miners alive at 1958. Data on smoking were available for 2,310 workers from health examinations.

77. By the end of employment, the mean cumulative exposure in the cohort was 65 WLM, with a maximum of 665 WLM [J4]. The overall excess relative risk per WLM was 0.022, and an excess lung cancer risk was found for small cell and squamous cell but not for adenocarcinoma histological types. Risk from radon was also evaluated in relation to cumulative quartz exposure. When radon excess relative risk was adjusted for cumulative quartz, the estimate decreased from 0.022 to 0.015 per WLM [J4].

78. Another study among iron ore miners was conducted in the combined Malmberget and Kiruna cohort [B7]. Because of the lower radon exposure levels than in the Malmberget study, the lung cancer risk was evaluated only in relation to quartz and diesel exhaust exposure. The main implication of the results is for interpretation of the dose-response curve for radon and lung cancer in underground iron ore miners. Since exposure to radon and quartz is often correlated, quartz exposure can be a confounder.

(i) *German Wismut uranium miners*

79. The German Wismut cohort includes approximately 59,000 miners employed for at least six months between 1946 and 1989 at the Wismut uranium mining company in Eastern Germany. Grosche et al. [G3] described the first results covering the follow-up period 1946–1998. The cohort included 59,001 former employees, with a mean duration of follow-up of 30.5 years, 1,801,630 person-years at risk and 2,388 lung cancer deaths. The average ERR per 100 WLM was 0.21 (95% CI: 0.18, 0.24). Time since exposure, attained age and exposure rate were strong modifiers of the ERR per WLM based on the BEIR VI exposure-age-concentration model.

80. The Wismut uranium mining company operated from 1946 to 1989. The early period of mining (1946–1955) was characterized by high radon exposure due to natural ventilation. In 1955, ambient air measurements of radon gas were initiated in the different mines, and between 1955 and 1958 the radon concentrations sharply decreased due to introduction of several ventilation measures in the different mines. A comprehensive job-exposure matrix was used for the cohort to retrospectively estimate the exposure to radon progeny in WLM for each calendar year, place of work (surface, underground, milling, open pit), mining facility/mine shaft and job type. For the period 1946–1954, radon concentrations were estimated by an expert rating based on measurements from 1955, considering, for example the ventilation rate, vein space and uranium content. The mean cumulative radon level among exposed cohort members was 280 WLM, with a maximum of 3,224 WLM [G3, K7].

81. Lung cancer risk was doubled in the cohort compared to external exposure rates (SMR=2.03; 95% CI: 1.96, 2.10) based on 3,016 lung cancer deaths with end of follow-up 2008 [W3]. Application of an

internal parametric Poisson regression model with exponential effect modifiers, demonstrated that time since exposure, age at exposure and exposure rate were strong effect modifiers of the ERR per WLM. Adjustment for potential confounders such as external gamma radiation, long-lived radionuclides or silica dust did not lead to major changes in ERR per WLM, indicating little confounding.

82. Kreuzer et al. found an average ERR per 100 WLM of 0.19 (95% CI: 0.17, 0.22) based on the extended follow-up period (1946–2013) with 3,942 lung cancer deaths and 2,332,008 person-years at risk [K7]. The ERR per WLM significantly decreased with increasing time since exposure, age at exposure and exposure rate. The ERR per 100 WLM was 0.75 (95% CI: 0.59, 0.98) for age at median exposure of 30 years, time since median exposure of 20 years and a mean exposure rate of 3 WL. The method for determining radon exposure did not have an important influence on risk estimation, indicating no major differences in the quality of exposure assessment in the early or later years (i.e. before 1955 and from 1955 onwards).

83. In order to determine specifically the risk at low exposure or exposure rates within the Wismut cohort and to investigate as far as possible effect modification by time and age, several strategies were used. Person-years at risk were restricted to <0.5 WLM, <50 WLM, <100 WLM [K7] and two subcohorts with low exposure and exposure rates were considered. The 1960+ subcohort with miners first hired in 1960 or later with low-radon concentrations all based on measurements (average exposure rate 0.2 WL) [K6, K7] and the subgroup of millers [K5].

84. The miller subcohort consists of 4,054 males of the Wismut cohort study, who worked between 1946 and 1989 in milling facilities, but never underground or in open pit mines [K5]. Mortality follow-up was from 1946 to 2008, accumulating 158,383 person-years at risk and 159 lung cancer deaths. Mean radon exposure was very low with 8 WLM and a maximum of 127 WLM. An increased, however, imprecise, radon-related lung cancer risk was found (ERR per 100 WLM=3.39; 95% CI: −0.01, 6.78).

85. The 1960+ subcohort included 26,677 miners and 334 or 495 lung cancer deaths in the follow-up period from 1960 to 2008 [K6] or to 2013 [K7], respectively. Mean radon exposure among exposed miners was 17 WLM. There was a clear exposure-response-relationship (ERR per 100 WLM=1.1; 95% CI: 0.6, 1.7). Smoking information was available for 56% cohort members. Most of the miners had been smokers and smoking turned out to be no major confounder of the ERR per WLM. No statistically significant effect modification of the ERR per WLM by age or time since exposure was observed. However, the cohort members were relatively young and only 19% were deceased [K7].

86. Restricting the full Wismut cohort to person-years at risk with low exposure (<50 WLM or <100 WLM) or exposure rates (<0.5 WL) showed a clearly increased radon-related risk of lung cancer (ERR per 100 WLM=0.6) that is modified by time since exposure and to some extent by age at exposure [K7]. In the cohort with restriction to <100 WLM, the ERR per 100 WLM was 1.6 (95% CI: 0.8, 2.8) at an age at exposure of 30 years and time since exposure of 20 years.

Table 8. Summary of occupational radon studies of miners published since the UNSCEAR 2006 Report [U6]

Study	*Period of follow-up*	*Number of cohort members*	*Number of person-years*	*Number of lung cancers*	*Mean cumulative exposure (WLM)*	*ERR per 100 WLM (95% CI)*	*Reference*
Colorado	1950–2005	4 137	120 437	612	794	NA	[S5]
Czech	1952–2010	9 978	308 910	1 141	73	0.97 (0.74, 1.27)[a]	[T15]
Czech and French	1946–1995	10 100	248 782	574	46.8	1.6 (1.0, 2.4)	[T12]
Eldorado	1950–1999	17 660	508 673	618	100.2	0.55 (0.37, 0.81)	[L3]
Beaverlodge	1950–1999	10 050	285 846	279	84.8	0.96 (0.56, 1.56)	[L3]
	1965-1999	NA	134 113	123	32.3	2.4 (0.9, 4.7)	[L4]
Port Hope	1950–1999	2 645	82 999	99	15.9	0.21 (–0.45, 1.59)	[Z2]
Port Radium	1950–1999	3 300	111 297	230	180.1	0.37 (0.23, 0.59)	[L3]
French	1956–1999	3 377	89 405	66	17.8	0.22/Sv[b] (0.06, 0.53)	[R2]
	1946–2007	5 086	179 995	211	36.6	0.71 (0.31, 1.30)	[R3]
	1946–1999	5 086	153 063	159	36.6	0.58 (0.20, 1.17)	[V1]
	1946–1999	5 086	153 063	159	36.6	0.58 (0.18, 0.98)	[V2]
	1956–1999	3 377	89 405	66	17.8	2.12 (0.53, 5.28)	[V3]
	1946–1999	5 086	153 063	159	36.6	0.53[c] (0.19, 1.07)	[L1]
New Mexico	1979–2005	2 745	63 395	117	NA	NA	[B10]
Newfoundland	1950–2001	1 742	59 797	191	378	0.43 (0.23, 0.62)	[V10]
Ontario	1954–2007	28 546	805 650	1 230	21.0	0.64 (0.43, 0.85)	[N1]
Swedish	1958–2000	5 486	170 204	122	65	2.20 (0.73, 3.77)	[J4]

Study	*Period of follow-up*	*Number of cohort members*	*Number of person-years*	*Number of lung cancers*	*Mean cumulative exposure (WLM)*	*ERR per 100 WLM (95% CI)*	*Reference*
Wismut	1946–1998	59 001	1 801 630	2 388	241	0.21 (0.18, 0.24)	[G3]
	1960–2008	26 766	846 809	334	17	1.30 (0.7, 2.1)	[K6]
	1946–2008	4 054	158 383	159	8	3.39 (−0.01, 6.78)	[K5]
	1946–2003	58 987	1 996 880	3 016	218	0.19 (0.16, 0.22)	[W2, W3]
	1946–2013	58 974	2 332 008	3 942	241	0.19 (0.17, 0.22)	[K7]
			1 620 190	1 254	18.4	0.6 (0.3, 1.0)[d]	
			956 776	495	16.8	1.1(0.6, 1.7)[e]	

[a] 90% CI.

[b] Related to equivalent lung dose from radon gas and short-lived progenies, long-lived radionuclides and gamma radiation.

[c] In a scenario that was based on chest X-rays using the lowest doses (~0.1 mGy) that were theoretically achievable during occupational medical surveillance.

[d] Analysis restricted to <100 WLM person-years at risk.

[e] Analysis restricted to 1960+ cohort.

2. Overall evaluation of miner studies

87. In order to compare the most current updates of occupational radon studies in miners with results reported in the previous UNSCEAR 2006 Report [U6], only those studies (unadjusted for modifying factors and covering the whole cohort) are considered as listed in table 9. Based on a random effects meta-analysis model with inverse-variance weighting* [D6], the combined ERR estimate from the entire cohorts of recently published studies is 0.60 (95% CI: 0.34, 0.87) per 100 WLM. This new estimate is in close agreement with the previous combined estimate of 0.59 (95% CI: 0.35, 1.0) per 100 WLM. Also summarized in the table are risk estimates for recent periods of work were provided, or for low exposure to radon otherwise. These include estimates for miners in the Czech study who began working during 1968–1974 (mean 7.5 WLM), cumulative exposure below 100 WLM in the Eldorado study (mean 32.3 WLM), workers hired after 1955 (mean 17.8 WLM) in the French study, Newfoundland miners hired in 1960 or later, miners in the Ontario study with exposure rates below 5 WLM per year, the cohort of the Swedish iron ore miners which had relatively low rates of radon exposure (mean 65 WLM), and Wismut workers hired in 1960 or later (mean 17 WLM). Altogether, the combined ERR for low radon exposure was estimated as 1.53 (95% CI: 1.11, 1.94) per 100 WLM.

Table 9. Estimates of ERR per 100 WLM in studies of miners published since the UNSCEAR 2006 Report [U6]

Study	*Entire cohort*		*Recent work periods/low radon exposure*	
	ERR per 100 WLM (95% CI)	*Reference*	*ERR per 100 WLM (95% CI)*	*Reference*
Czech	0.97 (0.74, 1.27)[a]	[T15]	2.0 (0.1, 5.3)[a]	[T15]
Eldorado	0.55 (0.37, 0.81)	[L3]	2.4 (0.90, 4.7)	[L4]
French	0.71 (0.31, 1.30)	[R2]	2.42 (0.90, 5.14)	[R3]
Newfoundland	0.43 (0.23, 0.62)	[V10]	2.85 (–2.58, 8.27)	[V10]
Ontario	0.64 (0.43, 0.85)	[N1]	3.08 (0.56, 5.60)[b] 1.81 (0.88, 2.73)[b]	[C23]
Swedish	2.20 (0.73, 3.77)	[J4]	2.20 (0.73, 3.77)	[J4]
Wismut	0.19 (0.17, 0.22)	[K7]	1.1 (0.6, 1.7)	[K7]
Combined[c]	**0.60 (0.34, 0.87)**		**1.53 (1.11, 1.94)**	

[a] 90% CI.

[b] Estimates for separate low-exposure rate categories of <2 and 2 to <5 WLM per year, respectively.

[c] Combined estimate from a random-effects meta-analysis model with inverse-variance weighting.

3. Modifying factors in relative risk models

88. The first studies of miners reported the risk in terms of risk coefficients (ERR per WLM or EAR per 10^4 PY WLM) for different subgroups defined by attained age, age at first exposure and time since first exposure. Later results included risk coefficients for low cumulated exposure (<100 WLM). Models of BEIR IV [N8] were developed using Poisson regression, where the regression coefficient represented

ERR per WLM. The model included the modifying effect of time since exposure (in two "time since exposure" windows: 5–14, ≥15 years) and attained age (in three categories: ≤54, 55–64, ≥65 years).

$$RR = 1 + (b_{5–14}\, W_{5–14} + b_{15+}\, W_{15+})\, a(j) \qquad j: \leq 54,\ 55–64,\ \geq 65$$

where cumulative radon exposure W is partitioned into temporal exposure windows, with $W_{5–14}$ and W_{15+} denoting exposure incurred 5–14 and ≥15 years prior to the current age; $b_{5–14}$ and b_{15+} represent the relative contributions to risk from the corresponding exposure; and a(j) is the modifying effect of attained age.

89. Models reported in BEIR VI [N10] were extended to three time since exposure windows (5–14, 15–24, ≥25) and four attained age categories (≤54, 55–64, 65–74, ≥75). The main difference to earlier results was the inclusion of exposure rate modifying factor (<0.5 WL, 0.5–1 WL, 1–3 WL, 3–5 WL, 5–15 WL, ≥15 WL for the exposure-age-concentration model).

$$RR = 1 + (b_{5–14}\, W_{5–14} + b_{15–24}\, W_{15–24} + b_{25+}\, W_{25+})\, a(j)\, c(k)$$

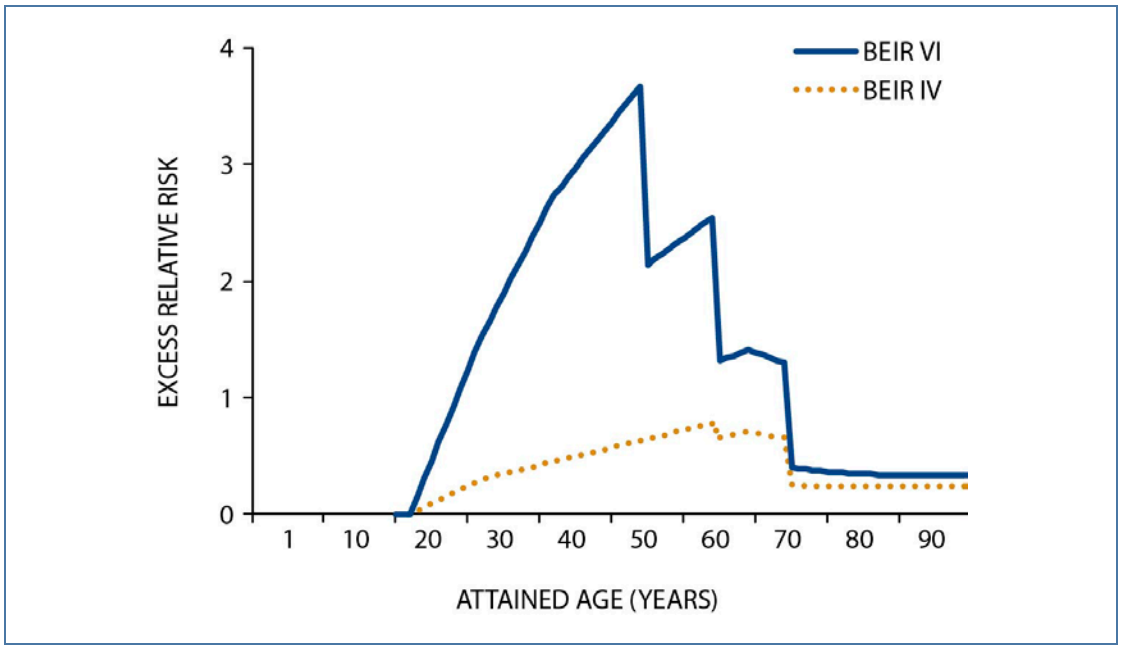
$$\text{(attained age categories) } j = \leq 54,\ 55–64,\ 65–74,\ \geq 75$$

$$\text{(exposure rate categories) } k = <0.5,\ 0.5–1,\ 1–3,\ 3–5,\ 5–15,\ \geq 15 \text{ WL}$$

where the W and a(j) model terms are defined as before; c(k) is an additional modifying effect of exposure rate; and WL stands for exposure rate levels defined as the ratio of cumulated WLM exposure to cumulated duration of exposure in months (170 hours).

90. The modifying effect of time since exposure and attained age was similar to the risk model of BEIR IV. The exposure rate modifying effect, however, substantially influenced the predicted risk (figure I). The difference between the first and last categories of exposure rate was approximately a factor of nine. The predicted risks in radiation protection reflect present levels in mines (mostly below 0.5 WL).

Figure I. Predicted excess relative risks (ERR) from annual exposure of 2 WLM at age 18–64 years according to BEIR IV and BEIR VI models



91. The strong exposure rate effect has several possible explanations:

- Random or systematic errors in estimates of annual exposure;
- Non-linearity between exposure and dose to target cells at high exposure rates;
- Cell killing at high exposure rates [S8];
- Genuine inverse dose-rate effect at cellular level [C5];
- Effect due to the multistage nature of carcinogenesis [B12, T1].

92. As noted in the NIOSH-NIEHS Joint Monograph No. 1 [L13], proportionally more measurements were made in areas of mines that experienced high radon levels which tended to yield radon-progeny values greater than would have been obtained by sampling all work areas with equal frequency. As demonstrated in risk models with an exposure rate modifying factor, the exposure assessment is the most important issue in occupational epidemiological studies. The approach used in BEIR VI which is based on average exposure rate, however, is sometimes misleading because the mean exposure rate for a long exposure duration combines different levels of exposure rates. Considering the hypothetical example given in table 10 of a 15-year total exposure of 80 WLM, the exposure would be wholly classified as a mean exposure rate of less than 0.5 WL, a 50 WLM (1961–1963), however, the majority is at a higher exposure rate category of 1–2 WL and only a 30 WLM minority belongs to the below 0.5 WL category.

Table 10. Example of cumulative exposure and exposure rates for a hypothetical 15-year period in which the rate is relatively low overall (0.48 WL) but high (1.52 WL) over the first three years where the majority of cumulative exposure occurs

Year/Period	*Period-specific cumulative exposure (WLM)*	*Period-specific mean exposure rate (WL)*
1961	20	1.82
1962	15	1.36
1963	15	1.36
1964	4	0.36
1965	4	0.36
1966	4	0.36
1967	3	0.27
1968	3	0.27
1969	3	0.27
1970	2	0.18
1971	2	0.18
1972	2	0.18
1973	1	0.09
1974	1	0.09
1975	1	0.09
1961–1975	80	0.48
1961–1963	50	1.52
1964–1975	30	0.23

93. Because of the above differences in the assessment of exposure rate, another approach was used in analyses from a French and Czech combined study [T11]. In addition to the aforementioned reason, cumulated exposures were separated according to the background of annual estimates (measured versus estimated). In this approach, the cumulated exposure was considered in two exposure windows (measured and estimated). In addition to estimated levels in non-uranium Czech mines, the French study made the clear distinction between exposure before and after 1956 in their assessment. Indeed, the risk coefficient related to pre-1956 exposure was substantially lower (even if adjusted for age and time since exposure). A similar approach in the risk models was applied in analyses of three European studies within the Alpha-Risk project [T14]. The estimated ERR per WLM in the joint study were virtually equal when related to exposure after 1953, 1956 and 1964 in the Czech, French and German studies, respectively.

94. A recent paper on the Czech uranium miners [T15] found no differences in exposure rate categories below 7 WL. The ERR per 100 WLM adjusted for time since exposure was 3.6 (95% CI: 2.6, 5.1) below 7 WL and 1.0 (95% CI: 0.3, 2.0) above 7 WL. The risk related to higher exposure rates probably reflects reasons other than systematic errors in estimates of annual exposure. It should be noted that extensive measurements of radon levels were conducted since the first years of mining.

95. The results on exposure rate effect point out the importance to report on the reliability of exposure estimates. This is not always addressed in scientific reports. It is crucial to report numbers of measurements of radon and its progeny. Often, radon levels were estimated or extrapolated in early years of mining, resulting in uncertainty and over estimation. As the number and quality of radon exposure increased and radiation protection was in place, radon exposure measurements became more reliable. For these reasons, models should take account of this issue.

96. The results published in 2006–2017 in nine epidemiological studies (table 8) are mostly given in terms of a simple linear excess relative risk model for lung cancer and other cancers, with the exception of the Colorado and New Mexico studies that only reported SMRs in different categories of exposure. In some studies, this model was also fitted for gamma radiation (France, Ontario, Wismut and Eldorado) and long-lived alpha exposure (France, Germany). Effects of other exposure (silica, quartz) were investigated in the German and Swedish studies. Modifying effects of single factors (age, time since exposure, age at exposure) were investigated in the Newfoundland study. Only four studies (Czech-French, Wismut, Czech and Eldorado) reported the combined effect of all modifying factors (age, time since exposure, exposure rate) using the same or similar model as in the BEIR VI report.

97. The same model as BEIR VI was fitted in the Eldorado, Wismut, and Czech studies. Age-specific estimates for the Eldorado study were similar to the BEIR VI studies. In the Wismut study, the parameters of modifying factors were very close to those of BEIR VI, but the age-specific ERR estimates were substantially lower giving about a third of the risk (table 11 and figure II). In the Czech study, the BEIR VI model was evaluated in a slightly different way using exposure windows of time since exposure, age at exposure and exposure rate. Exposure rate and age were the most influential modifying effects in the risk models. Risk decreased 6–9 times for exposure rates 15+ versus <0.5 WL and 3–11 times for ages 75+ versus <55. In the Czech study the inverse exposure rate effect was not observed, suggesting that exposure rate reflects the overestimation of exposure in early periods of mining. This effect is studied in categories of exposure rate defined as mean exposure rate for the entire exposure history. For this reason, mean exposure rates over 3 WL were not observed. However, the annual variability in exposure rates within each category of exposure rate is quite large. For this reason, a new model of exposure rate effect is suggested and discussed in the electronic attachment of this annex. The modifying effect of time since exposure in all four studies is similar. The modifying effect of attained age is generally similar. Statistical comparisons of the Eldorado, Wismut, and Czech age and time since exposure-specific rates were only significantly different for age <55 (p=0.014).

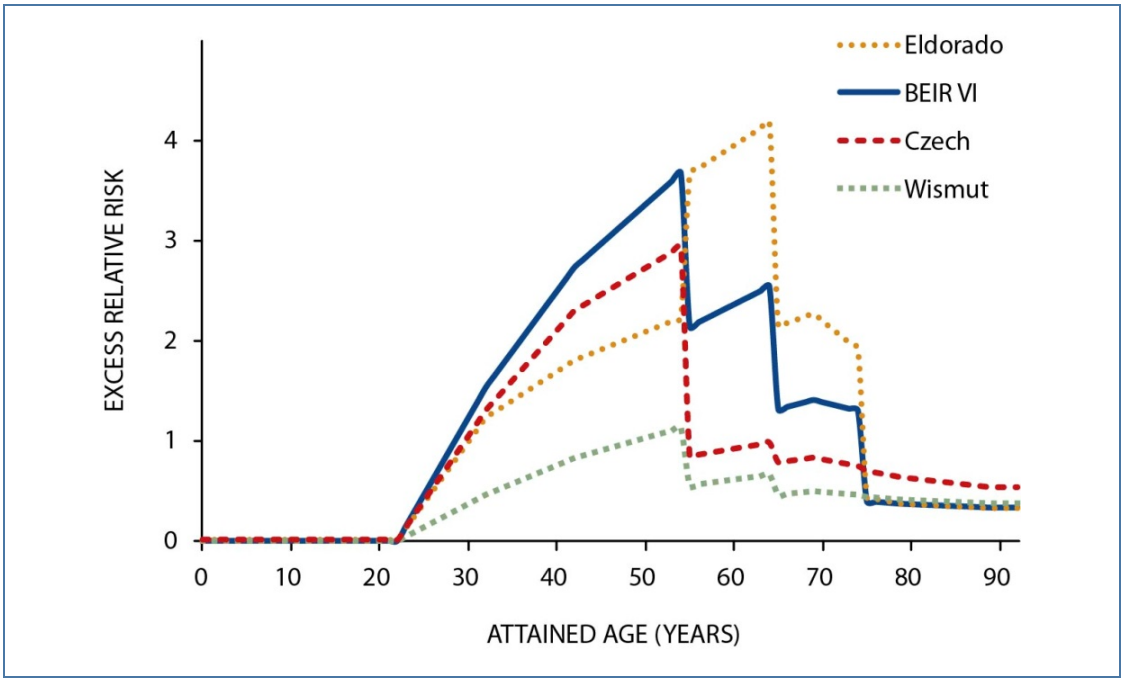
Table 11. Estimates of ERR per 100 WLM from exposure-age-concentration model employed in the BEIR VI report and individually applied to the Eldorado and Wismut and Czech studies

Age (years) [a]	*ERR per 100 WLM (standard error)*				*p-value* [b]
	BEIR VI	*Eldorado*	*Wismut*	*Czech*	
<55	7.68	6.11 (3.11)	2.31 (0.71)	6.47 (1.31)	0.014
55–64	4.38	9.90 (5.55)	1.11 (0.36)	1.81 (0.37)	0.125
65–74	2.23	5.01 (3.04)	0.76 (0.26)	1.42 (0.47)	0.191
75+	0.69	1.16 (4.92)	0.74 (0.28)	1.36 (1.06)	0.850
Time since exposure (years)					
5–14 (reference)	1	1	1	1	
15–24	0.78	0.47 (0.54)	0.79 (0.21)	0.77 (0.12)	0.855
25+	0.51	0.29 (0.59)	0.54 (0.14)	0.41 (0.07)	0.687
Exposure rate (WL)					
<0.5 (reference)	1	1	1	1	
0.5–1	0.49	1.05 (0.55)	0.61 (0.17)	0.63 (0.20)	
1–3	0.37	0.47 (0.23)	0.44 (0.10)	1.29 (0.27)	
3–5	0.32	0.34 (0.19)	0.39 (0.09)		
5–15	0.17	0.31 (0.15)	0.34 (0.08)		
15+	0.11	0.16 (0.08)	0.16 (0.06)		

[a] Estimates adjusted to 5–14 years since exposure and exposure rate <0.5 WL.

[b] Difference between Eldorado, Wismut and Czech study estimates.

Figure II. Projection of the risk in the BEIR VI, Eldorado, Czech and Wismut cohorts from annual exposure of 2 WLM at age 18–64 years



4. Lifetime risk calculation

98. The concept of lifetime risk is generally used in ICRP recommendations [I4, I5, I6, I9]. The idea is to extrapolate risk from cohorts with incomplete follow-up to complete lifespan. The risk models for cohorts in this extrapolation should therefore include temporal and age modifying factors that enable risk predictions over follow-up periods.

99. The approach for estimating lifetime risk related to occupational radon exposure was introduced in ICRP Publication 65 [I6]. The ICRP excess relative risk model was based on a meta-analysis of seven cohort studies: Colorado, New Mexico, Ontario, Beaverlodge, Czech, French and Swedish. The risk projection model involved modifying effects of time since exposure and age at exposure (estimated from the Czech cohort), but not exposure rate. The background lung cancer rate was taken as the average of five countries: China, Japan, Puerto Rico, United Kingdom and United States. Calculations were based on combined male and female rates, assuming that the ERR model is not different between male and female populations. This assumption, however, was not appropriate because the ERR per Sv in females is about three times higher than in males as estimated from the life span study [I9]. The lifetime risk, i.e. the probability of death from a specific cause at age a (during remaining life) is calculated as follows:

$$LR(a) = \Sigma_{t \geq a}\, r(t)\, S(t|a)$$

where $r(t)$ is the cause specific mortality at age t, $S(t|a)$ is the probability of survival from age a to t

$$S(t|a) = \prod_{a \leq u \leq t} (1-q(u))$$

and $q(u)$ is the annual mortality from all causes at age u. The probabilities of survival can be obtained from life tables routinely published by statistical offices.

100. The lifetime risks were calculated for ages a=18 to t=95 using background age specific lung cancer rates $r_0(t)$ in a "non-exposed" population and with rates $r_E(t)$ for an exposed population. The lung cancer mortality rates for exposed and non-exposed populations are related as follows:

$$r_E(t) = r_0(t)\, RR(t, E)$$

where $RR(t, E)$ is the relative risk at exposure E and age t according to the projection model.

101. Lifetime excess absolute risk (LEAR) is calculated from a background rate and from a radon risk model with modifying effects of attained age, time since exposure and exposure rate. The calculation makes use of the lung cancer mortality in age categories and all causes mortality in the whole lifespan from ages 18 to 95 years. Therefore, the risk is projected to the lifetime of adult populations.

102. As indicated in the UNSCEAR 2006 Report [U5], the lifetime risk estimates are sensitive to variations in underlying rates. Issues of uncertainty in lifetime risk estimates are discussed in more detail in NCRP Report No. 126 [N3] and in the BEIR VII report [N11].

103. For a constant exposure scenario of 2 WLM per year from 18 to 64 years of age, the LEAR was calculated for the ICRP Asia and Euro-American male population [I9] from age 18 up to 95 years of age. For comparison with ICRP 65 [I6], lifetime risks were calculated for combined male and female populations. Calculations were performed using the BEIR VI exposure-age-concentration model [N10]. The model was applied individually to the Czech, Wismut and Eldorado cohort miner studies as well as to the joint analysis of the 11 cohort studies from the BEIR VI report. Results are given in table 12. The LEAR per WLM estimates were 2.4×10^{-4} for the newly published large Wismut study, 3.9×10^{-4} for the updated Czech study and 7.5×10^{-4} for the updated Eldorado study. For the BEIR VI studies, the LEAR per WLM was 5.5×10^{-4}. LEARs were also calculated for male populations only (table 12).

Table 12. Lifetime excess absolute risks according to the BEIR VI exposure-age-concentration model for a constant exposure scenario of 2 WLM per year from 18 to 64 years of age

Cohort	*BEIR VI (combined 11 studies)*	*Czech*	*Wismut*	*Eldorado*
ICRP male and female rates LEAR per 10^4 persons per WLM (standard error)	5.5 (1.3)	3.9 (1.0)	2.4 (0.6)	7.5 (3.0)
ICRP male rates LEAR per 10^4 persons per WLM (standard error)	7.0 (1.6)	5.0 (1.3)	3.0 (0.7)	9.6 (3.8)

5. Smoking-radon interaction in occupational studies

104. The earlier analyses of smoking-radon interaction in occupational studies were given in the BEIR VI report [N10]. Smoking data were available from six cohorts (China, Colorado, Newfoundland, Sweden, New Mexico and Radium Hill). The estimates of ERR per 100 WLM in never smokers (1.02; 95% CI: 0.15, 7.18; 64 cases) were higher in comparison to ever-smokers (0.48; 95% CI: 0.18, 1.27;

1,292 cases). In addition to these analyses, BEIR VI suggested analyses based on fitting the so-called geometric mixture models of additive and multiplicative interactions:

$$RR = ((1 + c\,S)\,(1 + b\,W))^{\theta}\,(1 + c\,S + b\,W)^{1-\theta} \qquad \text{(Model G1)}$$

where *S* is smoking indicator (never-, ex-, and current-smoker), *W* is cumulated exposure to radon and *c* and *b* are estimated risk coefficients. The mixture model has as special cases models with multiplicative (θ=1) and additive (θ=0) effects of smoking, as well as models that are intermediate ($0 < \theta < 1$), sub-additive ($\theta < 0$), sub-multiplicative ($\theta < 1$), and super-multiplicative ($\theta > 1$). Lower mixing parameters are in favour of additive interaction. BEIR VI reported estimates of θ obtained with maximum likelihood methods applied to data from each of the cohort studies. Estimates ranged from −0.3 to 0.7 with a mean value of 0.5.

105. Smoking-radon interaction in the period 2006–2017 was investigated in three European studies as well as the Colorado, Newfoundland, and Wismut studies. The numbers of cases were similar to the numbers in original cohorts. The design of European studies was based on case-control studies nested within cohorts. The analyses in these studies depended mostly on numbers of non-smoking cases. Basic information is given in table 13. In studies, where numbers of non-smoking cases are large, the risk coefficients in never-smokers are substantially higher than in ever-smokers. The geometric mixture models resulted in sub-multiplicative interaction with mixing parameters ranging from 0.5 in the three European studies (BEIR VI) to 0.2 in the Czech study.

Table 13. Summary information on smoking and radon in underground miner studies

Study	*Number of lung cancers*	*ERR per 100 WLM (95% CI)*			*Reference*
		Overall	*Non-smokers*	*Smokers*	
BEIR VI	1 356	0.53 (0.20, 1.38)	1.02 (64) (0.15, 7.18)	0.48 (1 292) (0.18, 1.27)	[N10]
Colorado	612		3.0 (76) [a]	0.3 (536) [a]	[S5]
Czech	1 028	1.4 (0.9, 2.3)	4.9 (80) (1.0, 17.9)	1.0 (948) (0.6, 1.7)	[T16]
French	62	0.98 (0.18, 3.08)			[L6]
Wismut	421	0.23 (0.11, 0.46)			[S4]
	246		2.0 (28) [b] (–1.6, 5.7)	1.2 (218) [b] (0.4, 2.1)	[K7]
Three European studies	1 046	0.8 (0.4, 1.4)	1.2 (197) [c] (0.5, 2.6)	0.7 (849) [c] (0.3, 1.3)	[L7]
	1 046	0.8 (0.4, 1.4)	1.1 (64) [d] (0.3, 4.0)	0.8 (982) [d] (0.3, 1.3)	[H17]
Newfoundland	140	0.47 (0.28, 0.65)	0.42 (8) (<0, 0.85)	0.48 (132) (0.29, 0.67)	[V10]

[a] Calculated from the SMR in Schubauer-Berigan et al. [S5].

[b] Smokers defined as persons who ever smoked in the last 20 years.

[c] Smokers defined as current or ex-smokers who had quit within 10 years.

[d] Smokers defined as current smokers and non-smokers as ex- or never-smokers.

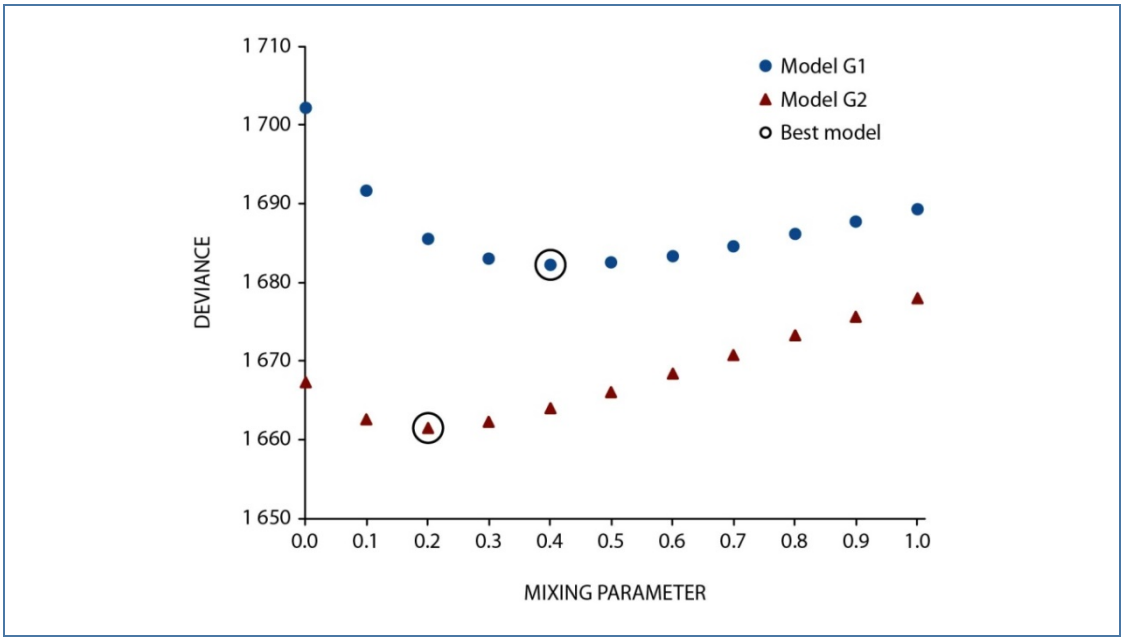
106. In the 1960+ German subcohort, the ERR per WLM for the group of non/light smokers (ERR per 100 WLM=2.0; 95% CI: −1.6, 5.7 based on 28 lung cancer deaths) was slightly higher compared to that of moderate/heavy smokers (ERR per 100 WLM=1.2; 95% CI: 0.4, 2.1; *n*=218) pointing to a sub-multiplicative interaction between smoking and radon. Also, geometric mixture models were fitted to investigate the nature of interaction between smoking and radon. They indicated a supra-multiplicative interaction. Due to small numbers of deaths in the respective smoking categories and thus low statistical power, the findings are consistent with both a supra-multiplicative and a sub-multiplicative interaction.

107. The geometric mixture model was fitted in the Czech nested case-control study using model G1 and further with the following model:

$$RR = ((1 + c\,S)\,(1 + b_1\,W_1 + b_2\,W_2))^{\theta}\,(1 + c\,S + b_1\,W_1 + b_2\,W_2)^{1-\theta} \qquad \text{(Model G2)}$$

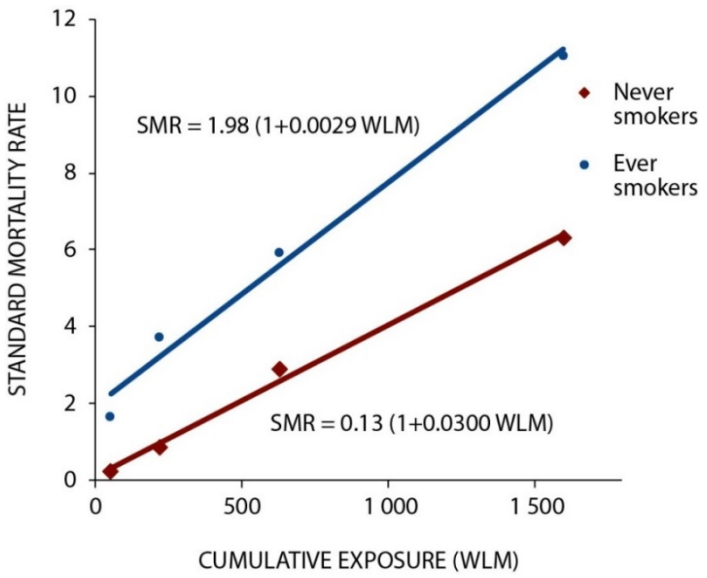
where W_1 and W_2 are cumulated exposures in time since exposure windows (5–19, 20+). As indicated by the lower model deviances shown in figure III, the best fitting model was G2 with a mixing parameter close to additive interaction. In statistical comparisons, model G2 was found to provide a significantly better fit to the data than model G1 (p<0.001).

Figure III. Model fit deviances over ranges of mixing parameter values for geometric mixture models G1 (cumulated exposure) and G2 (time since exposure) fit in the Czech study [T16]



108. Smoking was recently investigated in the Colorado study [S5]. Results were given in terms of SMR classified by smoking categories and cumulated exposure. The dependence of risks (SMR) on smoking and radon exposure is shown graphically in figure IV. From the fitted lines, it can be derived that ERR per 100 WLM=3.0 in never-smokers and 0.3 in ever-smokers. The relative risk of smoking at zero exposure is RR=15.2. Effects of radon by smoking status were also estimated in a joint analysis of three European nested case-control studies of uranium miners. As reported by Leuraud et al. [L7], ERR per 100 WLM was found to be 1.2 (95% CI: 0.5, 2.6) in never- and long-term ex-smokers and 0.7 (95% CI: 0.3, 1.3) in short-term ex- and current smokers. Hunter et al. [H17] considered other classifications of smoking status for this study and reported ERR of 1.0 (95% CI: 0.3, 4.0) for never-smokers, 1.2 (95% CI: 0.5, 3.4) for long-term ex-smokers, and 0.7 (95% CI: 0.3, 1.3) for current smokers.

Figure IV. Lung cancer risk (SMR) in dependence on cumulated exposure (WLM) and smoking in the Colorado study (adapted from [S5])



6. Summary of occupational studies

109. Reviewed occupational evidence of lung cancer risk due to radon included published extensions of eight pre-existing studies (Colorado, Czech Republic, Eldorado, France, New Mexico, Newfoundland, Ontario, Sweden) and new results from the Wismut study. Substantial variability in overall ERR estimates was observed, with values ranging from 0.19 to 3.4 per 100 WLM. Likewise, mean cumulative exposure to underground radon ranged from 17 WLM in low-exposed Wismut miners to 794 WLM in Colorado miners. A combined ERR estimate of 0.60 (95% CI: 0.34, 0.87) per 100 WLM was calculated based on results for the entire cohorts in each study. A higher combined ERR estimate of 1.53 (95% CI: 1.11, 1.94) was calculated when considering subcohort results for more recent work periods and lower exposure.

110. Growing evidence of the modifying effects of exposure rates, age, and times since exposure raised doubts about the appropriateness of a combined estimate of unadjusted risk from studies that are heterogeneous with respect to exposure. The BEIR VI risk model was summarized for its role in past risk estimation approaches to account for the modifying effects of exposure rate as well as time since exposure and attained age. The BEIR VI model estimates were calculated with current data available from the Czech, Eldorado, and Wismut studies. Resulting central estimates at 5–14 years since exposure, attained age <55, and exposure rate <0.5 WL ranged from 2.31–6.47 ERR per 100 WLM as compared to the BEIR VI published estimate of 7.68. Exposure rate level and attained age were found to be strong modifying factors with risks decreased by 6–9 times at rates of 15+ versus <0.5 WL and by 3–11 times for ages 75+ versus <55 years. Modifying effects of time since exposure decreased by factors of 2–3 for ages 25+ versus 5–14 years. The BEIR VI approach is based on average exposure rate which combines different levels of exposure rates.

111. Smoking-radon interaction tended to be sub-multiplicative in the three European studies, as well as in Colorado and in Newfoundland studies. Excess relative risk per unit exposure estimates were higher among non-smokers in all, apart from the Newfoundland study, where the confidence intervals were wide due to relatively small numbers of lung cancer cases.

112. Since the previous pooled study of 11 underground miner cohorts in BEIR VI, the cohorts have been extended, their follow-up has been updated and new cohorts have been set up. Miners working in more recent periods have been exposed to lower levels of radon and the assessment of their exposure estimates were more accurate. As a result, the increased statistical power reached with this hindsight allows for improved risk estimation of cancer and non-cancer diseases, in particular at low levels of radon exposure. The collaborative Pooled Uranium Miners Analysis (PUMA) is an ongoing international initiative set up to gather the most informative cohorts of uranium miners and to conduct statistical analyses with a greater ability to study the risks associated with radon exposure. The PUMA cohort includes seven cohorts of uranium miners: three European cohorts (Czech, French and Wismut), two Canadian cohorts (Eldorado and Ontario) and two American cohorts (Colorado Plateau and New Mexico). This combined cohort includes a total of 124,507 uranium miners (twice the size of the BEIR VI cohort) among which there are 7,825 cases of death from lung cancer (three times more than in the BEIR VI cohort). Specific objectives dealing with radon-related lung cancer risk are focused on low exposure and low exposure rate effects, on the time-dependent modifying factor effects, on the risk among non-smokers and on the impact of the other radiological exposure present in the atmosphere of uranium mines. Future findings from the PUMA study should strengthen our understanding of radon and radon progeny-related lung cancer risks.

E. Review of epidemiological studies for residential exposure

113. The UNSCEAR 2006 Report [U6] identified the main studies of residential radon to be the pooled analyses of European [D2, D3], North American [K8, K10] and Chinese [L10] residential case-control studies. Summarized in table 14 are the lung cancer risk estimates from the three studies.

Table 14. Summary of lung cancer excess relative risk estimates per 100 Bq m^{-3} (95% CI) from major residential studies included in the UNSCEAR 2006 Report [U6]

Study	*Primary analysis*	*Restricted analysis*	*Exposure adjusted for uncertainty*
European [D2, D3]	0.084 (0.03, 0.158)	0.094[a] (0.034, 0.175)	0.16[b] (0.05, 0.31)
North American [K8, K10]	0.11 (0.00, 0.28)	0.18[a] (0.02, 0.43)	
Chinese [L10]	0.133 (0.01, 0.36)	0.319[c] (0.07, 0.91)	
Combined	**0.093 (0.04, 0.15)**	**0.11 (0.05, 0.19)**	

[a] Restricted to individuals who had lived in at most two residences during the previous 30 years.

[b] Statistical correction for measurement error uncertainty.

[c] Restricted to individual who had lived in only one residence with complete radon coverage.

114. The UNSCEAR 2006 Report concluded that these study results indicate a significant association between residential radon exposure and lung cancer risk. Moreover, the studies were noted to have found no significant heterogeneity in relative risk estimates from radon for individuals with different smoking habits. Based on the evidence available at the time, the Committee deemed it reasonable to adopt the

uncertainty adjusted estimate from the European study of 0.16 (95% CI: 0.05, 0.31) per 100 Bq m^{-3} excess relative risk from residential radon. A review of the literature was conducted for the present report to identify new evidence concerning the lung cancer risk posed by radon [U6]. Review findings are presented here for residential studies with direct measurements of radon exposure, indirect measurements, and studies combined in meta-analyses.

1. Studies with direct radon measurements

(a) *North American pooling*

115. Two case-control studies *(a)* New Jersey, United States and *(b)* Connecticut, Utah and Southern Idaho, United States included in the Northern American pooling had their main findings published after the UNSCEAR 2006 Report. Since the main findings from these studies were not available for the previous report, their study designs and results are briefly summarized here.

116. The New Jersey study involved 651 and 740 sex, race, age, and smoking-matched cases and controls enrolled during 1989–1992 [W10]. Cases had primary, histologically confirmed lung cancer. Radon concentrations were measured with alpha-track detectors placed in the living areas of homes for one-year periods. An attempt was made to measure homes previously lived in for a minimum of two years over an exposure time window of 5–30 years prior to study enrolment. Radon exposure was calculated as a time-weighted average over the exposure time window with equal weights given to all years and as a BEIR VI weighted average with decreasing weights of 1.0, 0.8, and 0.3 for exposure time window years 5–14, 15–24, and 25+, respectively. Subjects were 46.7% males. Cases and controls were 6.1 and 16% never smokers and had mean time/BEIR VI weighted average radon exposures of 46.0/36.2 and 46.4/36.4 Bq m^{-3}. Multivariable regression was used to estimate ERR due to radon while adjusting for sex, age, smoking, number of residences and years of radon monitoring. Overall, the ERR was estimated to be 0.05 (95% CI: −0.14, 0.56) per 100 Bq m^{-3} for time weighted exposures. In subset analysis, estimates were −0.13 (95% CI: −0.30, 0.44) for males and 0.29 (95% CI: −0.12, 1.70) for females. Risk estimates were also computed separately by age and smoking categories but not found to be significantly different.

117. The Connecticut, Utah and Southern Idaho study enrolled 1,474 and 1,811 eligible cases and controls between 1989–1993 [S3]. Random recruitment was employed to maximize the proportion of non-smoking cases and to select a control group that was similar with respect to age, sex and smoking. Eligible subjects were 40–79 years of age, residents of their respective states, and residents of Utah and Southern Idaho for at least 50% of the time from age 25 to five years prior to enrolment or residents their entire adult lives (after age 25) in Connecticut. In addition, cases had a pathologically confirmed primary lung cancer. Radon concentrations were measured with 12-month alpha-track detectors placed on multiple levels of current and past homes occupied by study participants. Radon exposure was calculated as the average of concentrations on each level of the homes lived in five to 25 years prior to enrolment, weighted by the proportion of time spent in each location. Subjects were 57.1% males. Cases and controls were 8.7 and 14.1% never smokers and had mean estimated radon exposures of 40.3 and 44.8 Bq m^{-3}. Multivariable regression was used to estimate ERR due to radon while adjusting for state, age, sex, study phase, altitude, population density, smoking, residential stability, working hours, and education level. Overall, the ERR was estimated to be 0.002 (95% CI: −0.21, 0.21) per 100 Bq m^{-3}. In subset analyses, estimates were 0.134 (95% CI: −0.23, 0.50) for Connecticut; −0.112 (95% CI: −0.34, 0.11) for Utah and Southern Idaho; −0.006 (95% CI: −0.29, 0.27) for males; 0.061 (95% CI: −0.30, 0.43) for females; and 0.141 (95% CI: −0.47, 0.75) for non-smokers.

(b) *Massachusetts, United States*

118. Thompson et al. [T2] conducted a case-control study of residential radon exposure and lung cancer in Worcester County, Massachusetts. The study was carried out between 1990–1999 with 200 cases and 397 age and sex-matched controls all enrolled from the same health maintenance organization. Subjects were eligible to participate if they were at least 40 years of age, had the permission of their primary care physician, had lived in a radon-testable residence for at least 10 years, and were not cigar or pipe smokers. In addition, cases had histologically or cytologically confirmed primary lung cancers. Radon concentrations were measured with Radtrack etch-track detectors (Tech/Ops Landauer, Inc.) placed for a period of one year in the living area most often used, the present bedroom, any former bedroom, and any other level of the house that had been used on average for one or more hours per week. Exposure rate was calculated as a weighted average of detector measurements and time spent in the locations in which they were placed. Subjects were 58% males and had a mean age of 67.3 years and home residency of 30 years. Cases and controls were 7.5 and 40.8% never smokers and had mean estimated radon exposures of 67.5 and 66.3 Bq m^{-3}. Multivariable regression techniques were used to estimate the lung cancer risk associated with radon. Covariates were included to adjust for potential confounding effects of smoking, residency, job exposure, income and education. Based on a linear model, excess lung cancer odds were estimated to be 0.04 (95% CI: −0.20, 0.35) per 100 Bq m^{-3}. Conditional logistic regression analysis produced estimated odds ratios* of 1.00 (reference), 0.53 (95% CI: 0.24, 1.13), 0.31 (95% CI: 0.13, 0.73), 0.47 (95% CI: 0.20, 1.10), 0.22 (95% CI: 0.04, 1.13), and 2.50 (95% CI: 0.47, 13.46) for radon categorized as <25, 25–49, 50–74, 75–149, 150–249, and ≥250 Bq m^{-3}, respectively. Risk estimates stratified by age, sex, or smoking were not provided for this study.

(c) *Galicia, Spain*

119. Barros-Dios et al. [B6] reported on the findings from a hospital-based case-control study of residential radon exposure and lung cancer in Galicia, Spain. The study was conducted between 2004 and 2008 with 349 cases and 513 sex-matched controls from two hospitals. Subjects were eligible to participate if at least 30 years of age and resided in their radon-measured home for at least five years. Cases had a pathologically confirmed lung cancer; whereas, controls were hospital patients receiving non-tobacco-related surgeries. Radon measurements were obtained with alpha-track detectors (Radosys Inc.) placed away from doors, windows, and electric devices for a period of three to six months. Measurements were seasonally adjusted. Subjects were 87% males and had lived in their homes for a median of 30 years. They were distributed 9, 57, and 34% across age categories <50, 51–70 and >70 years; and 32, 35, 16 and 17% across radon exposure categories <50, 50–100, 101–147 and >147 Bq m^{-3}. Cases and controls were 15.3 and 45.5% never smokers. Lung cancer risk was estimated with multivariable logistic regression adjusted for age, sex and tobacco consumption. Estimates of linear excess risk were not provided. Rather, odds ratios were reported as 1.00 (reference), 1.87 (95% CI: 1.21, 2.88), 2.25 (95% CI: 1.32, 3.84) and 2.21 (95% CI: 1.33, 3.69) for the four radon exposure categories. Statistical evidence of an interaction between radon and smoking indicated an additive (p-value<0.001) and not a multiplicative (p-value=0.19) effect. Risk estimates stratified by smoking categories were also obtained as shown in table 15. Estimates of linear excess risk were not provided.

Table 15. Risk estimates stratified by smoking categories [B6]

Radon exposure (Bq m^{-3})	*Odds ratios by tobacco consumption (pack-years[a])*			
	Never smokers	*1–33*	*34–66*	*>66*
0–50 (reference)	1.00	1.00	1.00	1.00
51–100	0.89	3.92	2.22	1.66
101–147	1.39	4.12	5.15	1.05
>147	1.16	6.09	1.85	2.57

[a] Packs of (20) cigarettes smoked per day multiplied by the number of years smoked.

120. Torres-Durán et al. [T18] conducted another hospital-based case-control study in the Northwest of Spain (Galicia and Asturias) in which 521 never smokers (192 cases and 329 controls) were enrolled between 2011–2013. Subjects were eligible to participate if they were over 30 years of age. Cases had pathologically confirmed lung cancer. Controls were selected from ambulatory individuals undergoing minor, non-oncological surgery and frequency matched to cases according to age and sex. Radon measurements were obtained with alpha-track detectors placed in participants' bedrooms for a minimum period of three months. Measurements were seasonally adjusted. Subjects were 21% males and had a median age of 70 years. The median number of years lived in measured homes was 30 years for cases and 36 years for controls. Radon exposures were distributed 24, 19, 20 and 37% across defined categories of ≤100, 101–147, 148–199 and ≥200 Bq m^{-3}. Multivariable logistic regression was used to estimate lung cancer risk for radon as a categorical effect and included adjustment variables for age, sex and environmental tobacco smoke exposure. When compared to the reference exposure of ≤100 Bq m^{-3}, odds ratios were 0.80 (95% CI: 0.43, 1.50) for 101–147 Bq m^{-3}, 1.16 (95% CI: 0.64, 2.11) for 148–199 Bq m^{-3} and 2.42 (95% CI: 1.45, 4.06) for ≥200 Bq m^{-3}. Statistical significance was observed for the highest exposure category, as indicated by the confidence interval. Odds ratios were additionally estimated in a sub-analysis of female participants to be 0.87 (95% CI: 0.43, 1.75), 1.00 (95% CI: 0.52, 1.95) and 2.84 (95% CI: 1.58, 5.09). Estimates of linear excess risk were not provided.

121. In a secondary analysis of 216 and 329 never-smoking cases and controls from the aforementioned study, Torres-Durán et al. [T19] estimated lung cancer odds ratios for radon exposure (≥200 versus <200 Bq m^{-3}) by histological types to be 2.14 (95% CI: 1.45, 3.17), 2.39 (95% CI: 0.89, 6.42), 2.19 (95% CI: 1.44, 3.33) and 2.05 (95% CI: 0.85, 4.95) for all histologies, squamous, adenocarcinoma and other types. Estimates were adjusted for age, sex and environmental tobacco smoke. Risk from radon was not significantly different across histological types (p-value=0.16). Radon risk was also assessed by age at diagnosis and found to be non-significantly higher among younger cases (p-value=0.28).

122. In a study by Barbosa-Lorenzo et al. [B5], a cohort of 2,127 disease-free participants were enrolled between 2002–2009 from the Galician radon map study and one of the prior case-control studies. Twenty-four lung cancers were observed during the follow-up period ending 31 December 2012. Radon was measured with alpha-track detectors placed for a period of three to six months in the rooms in which participants spent the majority of their time. Measurements were seasonally adjusted. Subjects were 57.4% males, 50% aged 51 or older at the time of enrolment, 54% never smokers, and 53.1% residents of their current home for over 25 years. Radon was categorized as <50 and ≥50 Bq m^{-3} for risk analysis with 64% of all subjects falling in the higher category. Relative risk for the high versus low radon categories was estimated with multivariable Cox regression models adjusted for sex, age, and tobacco use to be 1.2 (95% CI: 0.5, 2.8) overall and 1.4 (95% CI: 0.5, 3.6) for males. Excess relative risk estimates were not provided, nor were smoking-specific risks.

(d) *Czech study*

123. A nested case-control study of residential radon and lung cancer was conducted in the Czech study [T16]. Controls were matched to cases on birth, age, sex and cohort. Subjects were selected from two cohorts. The first was a Mid-Bohemia Pluton cohort study designed in 1989 that contributed 289 cases and 1,156 controls to the present study. The second was a hospital-based case-control study (Bulovka) in Prague and adjacent regions in Central Bohemia that contributed 81 cases and 243 controls. Subjects were included if they had resided in their current home for at least 30 years. Radon progeny was measured with passive track detectors placed for a period of one year in the two most occupied rooms of each home. Risk estimates were reported in terms of radon gas derived from radon progeny measurements and simultaneous placement of electrets and CR−39 detectors. Mean radon exposures were 87, 423 and 361 Bq m^{-3} among the Bulovka, Pluton and combined study subjects, respectively. ERR was estimated with conditional logistic regression models. In the primary analysis, smoking was included in the models as a multiplicative effect. An ERR estimate of 0.14 (90% CI: 0.03, 0.39) per 100 Bq m^{-3} was obtained in the analysis of all subjects combined. In subset analyses of never and ever smokers, the ERRs were 0.73 (90% CI: 0.02, 1.90) and 0.14 (90% CI: 0.02, 0.30), respectively. A geometric mixture model, which is a weighted combination of multiplicative and additive models for smoking, was additionally fitted and placed more weight (60% versus 30%) on the additive model. Risk estimates stratified by age or sex were not provided.

2. Studies with modelled radon exposure estimates

(a) *Danish study*

124. Bräuner et al. [B11] assessed long-term association between radon and lung cancer risk in a prospective cohort of 57,053 Denmark residents recruited during 1993–1997. Subjects had to be between 50 and 64 years old and cancer-free at the time of enrolment and were followed for occurrence of any cancer up to 27 June 2006 at which time 589 primary lung cancers were observed. Thirty-five years of home address information from 1971–2006 was obtained for each participant. Radon was not measured directly in this study. Instead, radon concentrations for each address were predicted from a validated regression model that used nine explanatory variables for geographic location, soil type and dwelling characteristics. Time-weighted average radon concentrations were then calculated for cohort member's addresses occupied during the study follow-up period. Subjects were 47.5% males and 63.7% non-smokers with a median radon exposure of 39.5 Bq m^{-3}. Multivariable Cox proportional hazards regression was used to estimate incidence rate ratios with radon exposure included as a time-dependent variable and covariate adjustment for age, sex, body mass index, length of school attendance, socio-economic status, smoking status, environmental tobacco smoke, fruit intake, alcohol, residence type, employment in an industry or job associated with higher risk for lung cancer, and traffic-related NOx exposure. Reported estimates of incidence rate ratios (95% CI) per 100 Bq m^{-3} increase in exposure for the entire cohort as well as for subgroups as summarized in table 16. Incidence rates in this study are noted to have been estimated as exponential, and not linear functions, of radon exposure.

Table 16. Estimates of incidence rate ratios (95% CI) per 100 Bq m^{-3} increase in exposure for the entire cohort, non-smokers and smokers [B11]

Sex	*Smoking status*		
	All	*Non-smokers*	*Smokers*
Males	1.01 (0.62, 1.64)	1.35 (0.44, 4.11)	0.95 (0.55, 1.63)
Females	1.06 (0.64, 1.77)	2.02 (0.70, 5.76)	0.89 (0.49, 1.61)
Both sexes	1.04 (0.69, 1.56)	1.67 (0.69, 4.04)	0.92 (0.58, 1.46)

(b) *American Cancer Society study*

125. Turner et al. [T21] examined the association between residential radon exposure and lung cancer mortality in a cohort of 811,961 participants enrolled into the American Cancer Society Cancer Prevention Study-II in 1982. At the time of enrolment, participants were at least 30 years of age and had at least one family member aged 45 or older. Since cigarette smoking information was only collected at enrolment, follow-up for the present study was restricted to the first 6 years of the study, at the end of which 3,493 lung cancer deaths were observed. Indirect estimates of radon exposure were obtained by linking participant addresses to county-level radon concentrations derived from non-random, screening measurement obtained by the University of Pittsburgh [P3], and other state-level sources. Participants were 44.7% males and 46.2% never smokers, with the mean radon concentration being 53.5 Bq m^{-3}. Multivariable Cox regression was used to estimate the risk due to radon while adjusting for education; marital status; body mass index; cigarette smoking; vegetable, fruit, fibre, and fat intakes; occupational exposure to asbestos, formaldehyde, coal or stone dusts, coal tar/pitch/asphalt, and diesel engine exhaust; and other occupational pollutants. An estimated 0.15 (95% CI: 0.01, 0.31) increased lung cancer risk was reported per 100 Bq m^{-3} increase in radon.

(c) *Irish longitudinal study*

126. The relationship between radon and lung cancer prevalence was examined using 5,590 subjects enrolled during 2009–2011 into the Irish longitudinal study on aging [D4]. Study participants were 50 years or older and had to have lived in their homes for at least five years to be included in the analysis. During the study follow-up, 35 lung cancers were observed. Indirect estimates of radon exposure were obtained by linking participant addresses to a radon risk map of Ireland published by the Irish Environmental Protection Agency. The map provides the percentage of homes in each 10 km grid square predicted to have radon concentrations above the national reference level of 200 Bq m^{-3} based on results of the Irish National Radon Survey conducted between 1992–1999. Participants were 45.7% males and 45.7% never smokers with an average age of 63.1 years. The distribution across radon risk categories of <1, 1–5, 5–10, 10–20 and >20% of households above 200 Bq m^{-3} was 25.9, 27.4, 21.0, 12.9 and 12.9%. Risk was estimated with multivariable logistic regression and adjusted for smoking, age, sex, education and population density. Resulting odds ratio estimates for the upper four radon categories relative to <1% were 1.12, 0.87, 2.85 and 0.65, respectively. The *p*-values for tests of these odds ratios did not achieve statistical significance at the 5% level.

(d) *Canada National Enhanced Cancer Surveillance System*

127. Hystad et al. [H18] linked ecologic radon exposure to subjects from a case-control study of 2,390 lung cancer cases and 3,507 controls enrolled between 1994–1997 from eight Canadian provinces. Cancer cases were drawn from provincial cancer registries to which population controls were sex and age matched. Complete 20-year geocoded residential histories were required for participants to be included in the analysis. Two radon risk maps were created from: *(a)* a residential radon survey of three-month radon concentration measurements from approximately 14,000 households; and *(b)* a radon risk map grouped into three zones (guarded, moderate or high) of equal landmass according to geology, soil uranium geochemistry and aerial gamma spectrometry. Subjects were 51.3% males with a mean age of 60.8 years. Cases and controls were 6% and 38% never smokers and had average health region radon concentrations of 81.3 and 78.6 Bq m^{-3}. Mixed effects multivariable logistic regression was used to estimate the lung cancer odds associated with radon. Included in the models were random effects for census divisions and covariate adjustments for age, sex, study province, smoking exposure, education, income, dietary intake, occupational exposure and geographic covariates. Odds ratios were estimated to increase by 0.14 (95% CI: −0.12, 0.46) for every 100 Bq m^{-3} increase in regional radon concentrations and by 0.11 (95% CI: 0.01, 0.23) for every 10 years lived in a high radon potential geological region.

3. Meta-analyses

128. Zhang et al. [Z4] performed a meta-analysis of published residential radon studies. Studies were eligible for inclusion if they met the following criteria: *(a)* case-control study design; *(b)* residential radon as the primary exposure, measured with alpha-track detectors, and expressed as a time-weighted mean; *(c)* lung cancer incidence as the primary outcome; and *(d)* odds ratios reported or calculable from reported data. Twenty-two primary studies published between 1990–2008 were identified from a literature review to be included in the meta-analysis. By region, there were 11 European, 8 North American, 2 Chinese and 1 Japanese study. The total numbers of cases and controls were 13,380 and 21,102. A dose-response analysis was performed with studies reporting three or more exposure levels and providing the data for categories of median exposure levels, numbers of cases and controls, and adjusted logarithm of the OR with its standard error. Five of the 22 studies were excluded for not having reported all data needed for this analysis. With the remaining 17 studies, the ERR was estimated to be 0.07 (95% CI: 0.04, 0.10) per 100 Bq m^{-3}. This result is not statistically different from those of the three pooled analyses reported in table 14.

129. An unconventional Bayesian meta-analysis of case-control, cohort and ecological studies of lung cancer risk associated with residential radon exposure was conducted by Dobrzyński et al. [D7]. Studies had to meet the following criteria for inclusion: cases and controls were subject to well-defined criteria; residential histories were given for all periods considered; periods of measurements were provided along with clear indicators of smoking habits, sex and mean age, and information about the methods of group selection. Adjusted relative risks or odds ratios and their 95% CIs were obtained from each study. Thirty-two independent studies published between 1990–2014 were identified and covered approximately 22,000 cases and 900,000 controls. Most of the controls, about 812,000, as well as 3,500 cases come from the cohort study of Turner et al. [T21] which used ecological indicators of residential radon. The meta-analysis combined estimates of lung cancer incidence as well as mortality. Linear effects of radon on OR/RR were estimated over four ranges of annual equivalent (mSv per year) radon doses to lungs. The ranges in terms of radon concentrations were 0–84, 0–168, 0–391 and 0–838 Bq m^{-3} with corresponding linear slope estimates of 0.09 (95% CI: −0.39, 0.57), 0.07 (95% CI: −0.04, 0.18), 0.07 (95% CI: 0.04, 0.11) and 0.00 (95% CI: −0.02, 0.02) per 100 Bq m^{-3}. The authors assessed the relative fits of models with constant and quadratic effects for radon, and ultimately concluded that the constant

model with no dose-response effect of radon was most consistent statistically with the meta-analysis data. The results of these meta-analyses are quite different from the results of the three pooled analyses reported in table 14 and from the previously described meta-analysis conducted by Zhang et al. [Z4].

4. Summary of residential studies

130. Table 17 summarizes the main findings of reviewed residential studies in which risk estimates were reported for radon exposure individually linked to lung cancer outcomes. Information is provided on the study region, design, number of cases, numbers of controls for case-control (C-C) studies and total number of subjects for cohort studies, type of risk measure reported and corresponding risk estimates. Of the three United States' studies, the Connecticut, Utah and Southern Idaho study, and the New Jersey study were included in the 2006 North American pooling and published individually afterwards; whereas, the Massachusetts study represents a new independent finding. Likewise, the three Spanish and one Czech study represent new independent findings. The two meta-analyses provide an aggregate synthesis of case-control studies published since 1990. Of the new studies, quantitative excess relative risks of 0.04 (95% CI: −0.20, 0.35) and 0.14 (95% CI: 0.03, 0.39) per 100 Bq m^{-3} are provided by the Massachusetts and Czech studies, respectively. The Spanish studies provide categorized risk estimates. In particular, categories whose midpoints are ~100 Bq m^{-3} larger than the references provide overall odds and hazard ratio* estimates of 2.25 (95% CI: 1.32, 3.84) for 101–147 versus <50 Bq m^{-3} and 1.2 (95% CI: 0.5, 2.8) for ≥50 versus <50. Risk estimates are provided for different smoking habits (never and ever-smokers) in the Spanish and Czech studies. The former reported reduced risk for never smokers, whereas the latter reported increased risk. However, the confidence intervals were wide and no statistically significant evidence of differential risk by smoking was noted in the reviewed studies. To a greater extent, currently reviewed and past epidemiological studies have not been able to assess exposure in childhood and adolescence. Thus, the impact of radon exposure at young ages on the induction of lung cancer is unclear.

Table 17. Summary of main study findings from epidemiological studies of residential radon published since the UNSCEAR 2006 Report [U6]

Region	*Design/subset*	*Cases*	*Controls/ cohort*	*Risk*[a]		*Reference*
				Measure	*Estimate (95% CI)*	
United States						
Connecticut and Utah	Case-control	1 474	1 811	ERR	0.002 (–0.21, 0.21)	[S3]
	Males	846	1 029	ERR	–0.006 (–0.29, 0.27)	
	Females	628	782	ERR	0.061 (–0.30, 0.43)	
	Never smokers	128	255	ERR	0.141 (–0.47, 0.75)	
New Jersey	Case-control	651	740	ERR	0.05 (–0.14, 0.56)	[W10]
	Males	302	348	ERR	–0.13 (–0.30, 0.44)	
	Females	349	392	ERR	0.29 (–0.12, 1.70)	
Massachusetts	Case-control	200	397	ERR	0.04 (–0.20, 0.35)	[T2]
Spain	Case-control	349	513	OR	<50: 1.00 (reference) 50–100: 1.87 (1.21, 2.88) 101–147: 2.25 (1.32, 3.84) >147: 2.21 (1.33, 3.69)	[B6]
	Case-control Never smokers	192	329	OR	≤100: 1.00 (reference) 101–147: 0.80 (0.43, 1.50) 148–199: 1.16 (0.64, 2.11) ≥200: 2.42 (1.45, 4.06)	[T18]
	Females	153	329	OR	≤100: 1.00 (reference) 101–147: 0.87 (0.43, 1.75) 148–199: 1.00 (0.52, 1.95) ≥200: 2.84 (1.58, 5.09)	
	Cohort	24	2 127	HR	<50: 1.0 (reference) ≥50: 1.2 (0.5, 2.8)	[B5]
	Males	20	1 220	HR	<50: 1.0 (reference) ≥50: 1.4 (0.5, 3.6)	

Region	*Design/subset*	*Cases*	*Controls/ cohort*	*Risk*[a]		*Reference*
				Measure	*Estimate (95% CI)*	
Czech Republic	Nested case-control	370	1 399	ERR	0.14 (0.03, 0.39)[b]	[T16]
	Never smokers	58	670	ERR	0.73 (0.02, 1.90)[b]	
	Ever smokers	312	NA	ERR	0.14 (0.02, 0.30)[b]	
Meta-analyses	Case-control	13 380	21 102	ERR	0.07 (0.04, 0.10)	[Z4]
	Mixed (case-control, cohort, ecological)	22 000	900 000	Stratified ERR	0–84: 0.09 (–0.39, 0.57) 0–168: 0.07 (–0.04, 0.18) 0–391: 0.07 (0.04, 0.11) 0–838: 0.00 (–0.02, 0.02)	[D7]

[a] Excess relative risk (ERR) estimates are for an increase in radon concentrations of 100 Bq m^{-3}; odds ratio (OR) and hazard ratio (HR) estimates are for the indicated categories of Bq m^{-3} concentrations.

[b] 90% CI.

F. Uncertainties in epidemiological studies

131. Measurement error* in assessment of residential radon exposure stems from several sources, but the fundamental issue is the need to cover long-term residential history, with dwellings at least during the period 5–20 years preceding the diagnosis, practically always with retrospective assessment. Further, none of the residential studies have evaluated radon exposure outside the home. For the miner studies, major uncertainty is related to the early period, typically before the 1960s, with few or no radon measurements [P7]. Also, during the later periods, the measurements did not cover all sites within the mining area and additional uncertainty stems from inaccurate data on working locations. During the past decades, personal dosimetry has strongly reduced these uncertainties.

132. As outlined in the UNSCEAR 2012 Report [U7], uncertainties in epidemiological studies include those related to outcome data (health effect information), and those concerning exposure assessment. Dosimetric models are always used for estimating radiation dose from radon from measured activity concentrations. Parameter values used in the models always involve uncertainties. Such uncertainties can affect the dose response (shape and steepness of the gradient) and the precision (confidence interval width). The bias from shared or differential errors in dosimetry can introduce major bias in risk estimates. Non-differential unshared errors tend to attenuate any real effect, and assignment (Berkson-type) error would not bias the result but only decrease precision [H5]. In addition, overly simplified models ignoring important influences can involve conceptual uncertainty besides uncertainty from imprecise parameter values.

133. The key components of uncertainty in exposure assessment for residential studies arise from the variability in exposure, which is difficult to cover completely, i.e. to construct complete and accurate cumulative exposure estimates over time from concurrent measurements of radon concentrations. First, the alpha track detectors used for measuring radon concentrations have a relatively small level of uncertainty. More importantly, radon concentrations vary within a building at a given time between rooms (spatial variation within the home), and over time. This includes short-term variation depending on changes in heating and ventilation, seasonal differences and possible structural changes that affect in- and outflow of air including insulation (particularly of windows), renovation or remodelling and radon remediation measures [K9]. Studies with repeated measurements in the same dwellings have also shown yearly variation reflecting influence of behavioural factors and weather [H2, S16]. Exposure estimation also relies on assumptions concerning time spent in the dwelling (occupancy), which is often presumed to be similar for a group of subjects. Nearly all studies of lung cancer risk from residential radon have been case-control studies, in which exposure assessment is always retrospective. Using concurrent radon concentration as a proxy indicator of past radon level adds to uncertainty. If the behaviours that affect the radon levels are not similar for the current residents of the dwelling and the study subjects, additional error results. Radon exposures outside the home, e.g. workplace and any secondary residence have not been covered in epidemiological studies. Radon measurements involve additional uncertainty related to the fact that only radon gas and not its progeny (or thoron) is measured, and the assumption that activity concentration is a valid indicator of the radiation dose to the lung.

134. Empirical studies of radon risk nearly always have missing data, and imputation* of missing values with credible estimates adds to uncertainty (it should be noted that ignoring the missing data can increase uncertainty even more). Reconstruction of complete residential history with valid measurements in all dwellings during the aetiologically relevant period is very challenging and some studies have focused on persons with stable residence. Often, 20–30% of the intended residential period has not been covered by radon measurements. Measurements after the residential period may be distorted by behavioural factors such as ventilation and heating habits, as well as any structural changes that can influence radon levels.

Some additional uncertainty may stem from imperfect accounting for different effects of exposures incurred during various time periods, including time since exposure and age at exposure (i.e. not being able to incorporate correct weighting for exposures during different time windows prior to cancer occurrence). Residential radon exposure starts in childhood, unlike occupational exposure in mining setting. The role of radon exposure in childhood on lung cancer risk has not been examined.

135. Several analyses of lung cancer risk from residential radon have employed statistical techniques to account for both classical and Berkson type measurement error in exposure assessment [F1, H4, H5, R5]. Uncertainties in exposure assessment tend to attenuate the dose response (bias the results toward the null) if non-differential, but the effect can be difficult to predict across exposure categories [D8]. In the large European pooled analysis of residential studies, the risk estimate per unit exposure was doubled after correction for uncertainties in radon measurements [D2].

136. Due to the highly complex combination of factors (related to both building and soil characteristics) strongly influencing radon levels in a dwelling, it has been exceedingly difficult to create prediction models that could provide accurate estimates without performing radon measurements. Therefore, studies without empirical exposure estimates were not included in this annex.

137. Thoron is present everywhere together with radon. Therefore, both radon and thoron can be detected in varying concentrations. In some epidemiological studies, no distinction is made between exposure to radon and its progeny and exposure to thoron and its progeny [U6]. Since thoron and its progeny can be an important component of the total exposure in some situations (workplaces or dwellings), it can thus be a source of error in radon studies that do not distinguish radon and thoron contributions to the total exposure [C20, S10, T3, T5, T7]. More details on thoron interference with radon measurements and risk assessment are given in section III.H.

138. In occupational studies, uncertainty in exposure assessment was highest during the early periods with the highest exposure levels with substantial improvement over time. Especially for older time periods, radon measurements were not comprehensive in terms of all mining locations, and also performed infrequently. Data on the workers' locations within the mines also have inaccuracies. Assigning exposure levels for workers in various parts of the mines at different times from a very limited set of measurements involves substantial uncertainty. Therefore, misclassification of exposure is likely to impact on risk estimates from miner studies even more than residential studies. Dosimetric models also incorporate assumptions regarding equilibrium factors for radon and its progeny, unattached fraction, and breathing rate. Regression calibration, Bayesian methods and other approaches have been used to deal with the effects of measurement error [A5, A6, H7, S19]. The impact of the correction for uncertainty in dosimetry has ranged from negligible to 50–60% of the effect size, and it tends to amplify the dose response (as is expected when classical measurement error diluting an effect is involved). Yet, the error correction methods also involve assumptions or priors, and the results may depend on methodological approaches applied.

139. In addition to uncertainty from exposure assessment, epidemiological studies assessing the health risk of exposure to radon also involve systematic errors such as selection bias, information bias* and confounding. In the residential case-control studies, selection bias could arise from differential participation of controls recruited for the studies. In particular, participation proportions among controls is a concern, as commonly 20–50% of the invited controls have refused. If for instance highly educated people are more likely to participate as is often the case [C24, R9], radon exposure in the population free of lung cancer is likely overestimated (as a high level of education is associated with residence in houses that have higher radon levels than flats), which would result in underestimation of risks. In occupational studies such as underground miners, healthy worker bias may result in underestimation of risk or distorted dose responses if an employed cohort is compared with the general population (standardized mortality

ratios). Information bias could occur if better radon exposure data are available for controls than cases (the former are likely to be alive and hence similar ventilation behaviour could be expected than for cases likely to have succumbed to their disease).

140. Smoking is a major potential confounder in indoor radon studies and as socio-economic factors are associated with both smoking habits and type of dwelling (houses have higher radon potential than flats), substantial negative confounding has been reported in studies of indoor radon (i.e. lack of adequate adjustment tends to decrease or mask the true effect of radon). In case-control studies, information bias may occur, as smoking history has to be obtained from proxy respondent for deceased cases, with more complete and precise data for controls with fewer deceased subjects. Matching for vital status has been employed in some studies to minimize this potential bias. To fully account for the effect of smoking, information should be obtained for intensity and duration of smoking, age at start and time since quitting, as well as types of tobacco products and exposure to environmental tobacco smoke. Any imprecision in the confounder data will reduce the effectiveness of control of confounding and result in residual confounding despite efforts to adjust for such effects. In the European pooled study of residential radon, smoking masked the effect of radon nearly completely in the preliminary analysis without adjustment for the effect of smoking [D2]. In miner studies, confounding by smoking does not appear to be very strong [H17, L6, L7, R8, S4, T13].

141. Besides being a confounder, smoking also modifies the effect of radon on lung cancer risk. In residential studies, the relative risk per unit exposure has been similar among smokers and non-smokers (no modification in the context of the multiplicative model, suggesting the findings are consistent with a multiplicative joint effect or combined effect) [D3]. In miner studies, on the other hand, a sub-multiplicative (but supra-additive) interaction has been reported [L7, S5, T13], although some results are also consistent with an additive interaction. A sub-multiplicative joint effect implies larger risks per unit exposure for non-smokers (on the multiplicative scale). A possible explanation for the discrepancy is the lack of sufficient power in residential studies with substantially lower exposure levels and effect sizes for radon than in miner studies. Converting relative risks into absolute risks, in both cases (i.e. in residential and miner studies) the absolute lung cancer risk from radon exposure would be much higher for a smoker than for a non-smoker, due to the much higher lung cancer risk from smoking.

142. Other known risk factors for lung cancer are also potential confounders, including exposure to asbestos, chromium, nickel, silica dust and diesel exhaust gas [I2, I3]. Of these, silica dust and diesel exhaust gas occur also in mining environment and may correlate with radon exposure. In such cases, positive confounding can occur, with incomplete control of other exposures exaggerating the effect of radon. The possible role of uranium dust in miner studies has not been examined, though its contribution to lung dose is smaller than that from external gamma radiation and from inhaled radon progeny [M4, R2]. Other risk factors for lung cancer that should ideally be also evaluated in both residential and miner studies cancer include benign lung disease and dietary factors [D5, V9].

G. Alternative models for lung cancer risk from radon exposure

143. Lung cancer risk after occupational radon exposure depends in a complicated way on the temporal modifiers attained age, age at exposure and time since exposure, and on exposure rate. Furthermore, the temporal modifiers are interrelated and cannot be easily separated. Therefore, alternative models to the BEIR VI model have been developed and applied to various cohorts. The parametric ERR models are structurally similar to the BEIR VI model but use continuous functions for the temporal modifiers. The biologically-based models of carcinogenesis try to quantify the effects of radon exposure on lung cancer development and to infer risk from these processes.

1. Parametric ERR models

144. Parametric ERR models were developed based on the observation that the relative risk consistently decreases with increasing attained age (or increasing age at exposure), increasing time since exposure and increasing exposure rate. Whereas the relative risk in the BEIR VI model is given for each modifier by categories, and each category by a separate parameter, parametric models usually assume an exponential dependence of the excess relative risk with the modifiers, with one parameter for each modifier. These models therefore have a smaller number of parameters related to radiation than used for description of the BEIR VI categories and avoid discontinuous changes of risk at the category limits. The smaller number of parameters should have better statistical support from the data and might be more robust among different cohorts.

145. Tomášek et al. [T12] applied an ERR model with exponential modifying terms on time since exposure and age at exposure. In both cohorts, the French and Czech uranium miners, similar decreasing modifying effects of time since exposure and age at exposure were found, and the relative effects were significantly different in the joint study.

146. The Wismut miners were analysed both with parametric ERR models and the BEIR VI model by Walsh et al. [W3]. Among various tested parametric models, two alternative formulations were found to describe best the data. The first model is linear in radon exposure with exponential effect modifiers that depend on age at median exposure, time since median exposure, and radon exposure rate. The second model is similar to the first one but includes attained age as covariable instead of the age at median exposure. Since age at median exposure, time since median exposure and attained age are linearly related, the two different formulations are identical in terms of quality of fit.

2. Mechanistic models of carcinogenesis

147. Biologically-based mechanistic models of carcinogenesis describe the development from early cellular changes towards malignant cells. In an effective way, the complex process is reduced to a few time-limiting steps. Essential elements of the model include mutational processes between different stages, clonal expansion of pre-malignant cells, and the transition from pre-malignant to malignant cells. Malignant cells turn into a detectable cancer after a certain lag period. In principle, radiation can act on any stage in the carcinogenic process. More details on this class of models and its applications to radio-epidemiological cohorts can be found in Rühm et al. [R11]. Several studies applied these models to miners exposed by radon and analysed the effect of radon on the different stages and the consequences for radiation risk. Most often the two-stage clonal expansion model was used that includes two stages from healthy stem cells towards malignant cells. The end point of all studies was lung cancer mortality.

148. The two-stage clonal expansion model was applied to the Colorado miners by Luebeck et al. [L12]. The effect of radon on clonal expansion was highly significant. An additional effect on the initiation step was observed but was not significant. It was shown that the inverse dose rate effect* is a consequence of radon exposure acting on clonal expansion. Smoking information was available, and the risk of smoking and radiation was between additive and multiplicative. This analysis was later extended to three stages by Little et al. [L9].

149. European miners from the Czech Republic, France and Germany were analysed with the two-stage clonal expansion model by Heidenreich et al. [H6]. Different possible actions of radon on the model parameters were tested. All three cohorts indicated a highly significant action of radon exposure on clonal expansion with a linear-levelling form. An additional action on initiation was only minor and not

significant in all cohorts, and an action on transformation was not found to be significant. The analysis included a correction for radon measurement errors.

150. The Eldorado cohort was analysed by Eidemüller et al. [E2] with the two-stage clonal expansion model. Again, the action of radiation on the clonal expansion rate was highly significant. The clonal expansion rate increased in a non-linear way, with a steep increase until exposure rates of about 20 WLM per year, and a smaller increase above. The authors mentioned that such an exposure response might be related to a bystander effect. In addition, the data were analysed by a parametric ERR model and the BEIR VI model, and the risk predictions from the different models were compared.

151. The Wismut workers were analysed by van Dillen et al. [V4] with a two-mutation carcinogenesis model that is structurally similar to the two-stage clonal expansion model and includes two stages and a net clonal expansion of intermediate cells. Assuming that radiation could only affect initiation or transformation, but not the net clonal expansion rate, the authors found a significant cell killing contribution in the second mutation rate and a large value of the lag time of 13–14 years from a malignant cell to an observable tumour. They further consider that there is low biological plausibility of a strong dose-dependent net-clonal expansion rate.

152. An updated data set of the Wismut workers with a longer follow-up and more workers and lung cancer deaths was studied by Zaballa and Eidemüller [Z1] with the two-stage clonal expansion model. Among a variety of models, the best model describing the data included an exposure-dependent clonal expansion rate. At lower exposure rates, it increased linearly with annual exposure. At rates around 100 WLM per year, the exposure response saturated. No indication for an additional radiation effect on the initiation rate or on the transformation rate was found. Applying the model for typical domestic exposure with a total of 11 WLM during 25 years of exposure, the model predicted a decrease in ERR per dose with attained age of about 25% per decade.

153. The mechanistic studies of lung cancer after radon exposure indicate a very consistent exposure response. All studies in which a radiation action on clonal expansion was tested showed a strong preference for radiation modifying clonal expansion. A possible additional effect of radon on initiation was minor and not observed in all studies, and almost no effect on transformation was found. Furthermore, the clonal expansion rate increased with exposure rate in a non-linear way, with a steeper increase at lower exposure rates, and a smaller increase or saturation at higher exposure rates. Noteworthy, such an exposure response is different to low-LET studies, where radiation usually acts either on initiation or on clonal expansion in a linear way [R11]. In the mechanistic model framework, the distinctive dependence of risk on the temporal modifiers as well as the inverse exposure rate effect results from the specific non-linear dependence of the clonal expansion on exposure rate. With a given model, risk predictions for different exposure scenarios are obtained from the annual exposure rates without explicit parametrization of the temporal modifiers.

154. The mechanistic studies were all applied to miner cohorts including also higher levels of radon exposure and exposure rate. Therefore, the suggested mechanisms from these studies are strongly influenced by the high exposure part of the cohorts. It is unknown whether the inferred mechanisms are similar to those operating at low levels of exposure and exposure rate. A better understanding of the biological mechanisms of radon carcinogenesis at different exposure levels, together with improved mechanistic description, can help to improve future risk inference* at lower exposure levels.

3. Discussion of alternative models

155. Since the temporal modifiers are interrelated, it is possible to obtain a similar description of the radio-epidemiological cohorts with different sets of modifiers, e.g. by using attained age and time since exposure, or by using age at exposure and time since exposure. Due to the complex model structure with many correlated parameters, the transfer of risk to other populations with different exposures involves a substantial uncertainty that relates to the type of selected model. A risk comparison of the BEIR VI model, the parametric ERR model and the two-stage clonal expansion model was conducted by Eidemüller et al. [E2] for different exposure scenarios with 50 and 500 WLM, and for 2 and 5 years of exposure duration. The resulting uncertainty from the choice of model was found to be larger than the statistical uncertainty.

156. Compared to the BEIR VI model, the parametric ERR model assumes a specific functional form of the temporal modifiers with a smaller number of parameters. The mechanistic model structure is fundamentally different and based on the long-term cancer development. The model parameter values can change during radiation exposure depending on the annual exposure rate. Similar predictions from different types of models for specific exposure scenarios, as well as consistent parameter values for a particular model type across various cohorts, can strengthen the confidence in the risk estimates. Better understanding of the underlying mechanisms can help to guide the risk transfer to other populations.

H. Thoron interference with radon measurements and risk assessment

157. Thoron (^{220}Rn) is an isotope of radon (^{222}Rn), 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.

158. In the past, exposures to thoron and its progeny were often ignored due to usually low concentration of thoron and its progeny in indoor environments. Since thoron ($t_{1/2}$=55 s) has a much shorter half-life than radon ($t_{1/2}$=3.83 days), the distance it can travel before undergoing radioactive decay is very much shorter than the distance radon can travel in the same medium. Thoron in the soil gas beneath a building cannot survive long enough, in most situations, to enter a building and thereby accumulate to a significant level in indoor air. Indoor thoron is generally due to the exhalation of thoron from thorium that may be present in the materials of the internal surfaces of the building. Because of its short half-life, thoron gas migration from the source is generally considered to be very limited in the indoor environment as well. This is true when the ventilation rate is low or poor [C11].

159. In most epidemiological studies, no distinction is made between exposure to radon and its progeny and exposure to thoron and its progeny [U6]. However, it has become increasingly clear that exposure to thoron and its progeny cannot be ignored in some specific environments (both workplaces and homes) as it contributes to the risks otherwise assigned solely to exposure to radon and its progeny.

160. A number of authors reported that the contribution of thoron and its progeny can be a significant component of the total exposure (radon and thoron). For example, Evans and Goodman [E3] measured the thoron content in the air of workplaces and found that some locations in a thorium mantle plant had thoron gas concentrations ranging from 8,510 to 148,000 Bq m^{-3} (230 to 4,000 pCi/L) which were greater than the average radon gas concentration measured in the Schneeberg and Jachymov mines. Data for two Elliot Lake mines showed thoron decay product levels ranging from 0.1 to 0.3 WL, with parallel radon decay product levels ranging from 0.2 to 0.5 WL [C4]. Radon and thoron levels were investigated in old

uranium mines in Dolina Bialego, Tatra Mountains, Poland [K3], where the average ratio of thoron to radon concentration was 0.017. Stegnar et al. [S17] assessed radiation exposure at selected former uranium mining and processing sites in the Central Asian countries of Kazakhstan, Kyrgyzstan, Uzbekistan and Tajikistan. While radon measurements were performed at all sites, thoron measurements were limited. The available results showed that outdoor radon and thoron concentrations varied from 16 to 1,500 Bq m^{-3} and from 8 to 500 Bq m^{-3} in Digmai; while indoor radon and thoron concentrations varied from 43 to 470 Bq m^{-3} and from 5 to 810 Bq m^{-3} in Kadji-Sai, respectively.

161. Since thoron is present everywhere together with radon, both radon and thoron can be detected in residential homes in varying concentration. The ratios of thoron to radon gas concentration varied from 0.023 to 0.29 with an average of 0.11 in 3,215 Canadian homes [C10], 0.82 to 3.1 with an average of 1.7 in high background radiation areas in 747 Indian homes [M9], and 1.6 to 11 with an average of 4.5 in Chinese traditional residential dwellings constructed with loam bricks or soil walls (n=164) [S9]. Average ratios between thoron and radon gas concentrations were also determined as 0.51 in the Balkan region (n=300) [S18], 0.65 in South Korea (n=1,550) [K2], 0.8 in the high background radiation areas of India (n=200) [C22] and 2.1 in Sokobanja municipality of Serbia (n=40) [Z7].

162. Simultaneous long-term measurements of thoron and its progeny were carried out in 248 Canadian homes [C7, C9]. While thoron gas was not detectable in 54% of the homes, thoron progeny was measured in all homes tested. This means that one of thoron's progeny, ^{212}Pb with a relatively long half-life of 10.6 hours can migrate away from its source and enter the immediate environment of human beings before undergoing radioactive decay. The results showed that thoron progeny may have enough time to escape from the point, where it is formed and migrate a considerable distance in an indoor environment while thoron gas concentration may fall off rapidly with distance from its source, especially with poor ventilation.

163. Measurements of radon and radon progeny together with thoron and thoron progeny were carried out in 141 dwellings in a thorium abundant beach environment of south west India [V11]. The average radon gas concentration was 24 Bq m^{-3} while the average thoron gas concentration was 37 Bq m^{-3}. The average equilibrium equivalent progeny concentrations were found to be 11 Bq m^{-3} and 1.6 Bq m^{-3} for radon and thoron, respectively. On average, thoron progeny concentration was much lower than radon progeny concentration while thoron gas concentration was higher than radon gas concentration. Based on progeny concentrations, it was estimated that thoron contributes 35% to the total inhalation dose from exposure to radon and thoron.

164. A large-scale study on radon gas concentrations and thoron progeny concentrations (EETC) in 2,500 dwellings was conducted in the Netherlands [S12] leading to an overall average radon gas concentration of 15.6 Bq m^{-3} and an average thoron-progeny concentration of 0.64 Bq m^{-3}. Applying an equilibrium factor of 0.4 for radon and a dose conversion factor of 9 nSv (h Bq m^{-3})$^{-1}$ EEC for radon and a 40 nSv (h Bq m^{-3})$^{-1}$ EETC for thoron, the thoron progeny in Dutch dwellings contribution is about 30% of the total effective dose due to radon and thoron and their progeny.

165. Since thoron and its progeny can be a significant component of the total exposure in some specific situations (workplaces or dwellings), it can thus be a source of error in radon studies that do not distinguish radon and thoron contributions to the total exposure [C20, T3, T5]. For example, with radon/thoron discriminative alpha-track detectors, Shang et al. [S10] measured radon and thoron gas concentrations in Gansu, China, where a radon study with a popular radon gas detector (Radtrak) was conducted jointly by the US National Cancer Institute and the China Ministry of Health. The average radon and thoron gas concentrations were 120±61 Bq m^{-3} and 430±210 Bq m^{-3}, respectively. Based on the re-investigation results, the actual level of indoor radon gas was about 3 times lower than that reported by Lubin et al. [L11] which was heavily affected by high concentrations of thoron gas.

166. A study conducted by Tokonami et al. [T7] in 202 cave dwellings in Shanxi and Shaanxi provinces of China found that indoor radon gas concentrations ranged from 19 to 195 Bq m^{-3} with a geometric mean of 57 Bq m^{-3} while indoor thoron gas concentrations ranged from 10 to 865 Bq m^{-3} with a geometric mean of 153 Bq m^{-3}. The study revealed that thoron is not negligible for accurate radon measurements and thus that special attention should be paid to thoron and its progeny for dose assessment in such specific environment.

167. Lung cancer risk due to residential radon has been revised based on more recent epidemiological data analyses in Europe [D2, D3] and North America [K8, K10]. In these large-scale surveys, passive radon gas detectors were used for exposure assessment. Although these kinds of detector are well designed so as to detect radon effectively, it is however also true that, in general, thoron (^{220}Rn) can also be detected. Some popular passive radon detectors were tested for their thoron sensitivity [C6, T8]. The relative sensitivity (RS) between thoron and radon was very low for some detectors, such as RS=0.05 for NRPB/SSI detectors used in radon studies in the United Kingdom, Ireland and Sweden and RS=0.02 for detectors used in the Winnipeg case-control study. However, for some detectors, the thoron and radon sensitivities were comparable, such as RS=0.68 for the Radtrak detector used in radon studies in the United States and RS=0.78 for the KfK detector used in radon studies in Germany [U9].

168. Five types of radon gas detectors commonly available to Canadian home owners were tested for their thoron sensitivity [C8]; the thoron sensitivity varied widely from 0.012 to 0.741. For example, a linear fitting of the average reported radon concentrations and the actual thoron concentrations at three different levels revealed a thoron sensitivity of 7.5% for radon gas detectors used by Health Canada's National Radon Laboratory. This means that a thoron gas concentration of 100 Bq m^{-3} could be reported as a false radon gas concentration of 7.5 Bq m^{-3} added to the true radon indoor concentration.

169. When a radon gas detector has a sensitivity to thoron, the reported radon result will always be the sum of the "true" radon concentration and a portion of the thoron concentration. The latter will be falsely treated as additional radon concentration, i.e. it will give a "false" radon concentration. If radon and thoron had the same potential alpha energy, this false reading would not be a big issue. In this case, the radon measurement from a detector equally sensitive to radon and thoron could then serve as an indicator of radiation dose to the lung. However, as indicated in the Committee's reports [U3, U6], at the same gas concentration, the annual effective dose of radon is 4.5 times the annual effective dose of thoron. This means the thoron gas concentration must be 4.5 times higher than the radon gas concentration in order to deliver the same amount of radiation dose to the lung. In other words, at a given concentration, thoron could be 4.5 times less effective than radon in producing damage to the lung.

170. Radon case-control studies have been performed to estimate the risk of radon-induced lung cancer. Radon gas detectors highly sensitive to thoron gas were used in some such studies. This suggests that in some studies radon exposure could have been overestimated, i.e. radon measurements may have been contaminated by the presence of thoron. Thoron contamination in radon measurements could have a significant impact on the assessment of lung cancer risk following radon exposure, because the risk associated with the radon exposure could, in fact, be underestimated. For observed lung cancer incidents, an overestimation in radon exposure will result in an underestimation in the risk. For more details see also appendix B.

I. Summary of epidemiological studies reviews

171. New epidemiological information on the lung cancer risk due to radon and radon progeny was found in the form of updated occupational studies, new results from the Wismut study, and new residential studies conducted in Massachusetts, United States, Spain and Czech Republic. Combined risk estimates from the updated underground miner studies were derived from published results reported for the entire cohorts as well as for recent periods of work and/or low radon progeny exposure. The combined ERR estimate of 0.60 (95% CI: 0.34, 0.87) per 100 WLM for the entire cohorts was similar to the UNSCEAR 2006 Report's estimate 0.59 (95% CI: 0.35, 1.0) [U6]. A higher combined ERR estimate of 1.53 (95% CI: 1.11, 1.94) was obtained when considering more recent work periods and lower exposure. Risk estimates from the Spanish residential studies were higher than the 0.16 (95% CI: 0.05, 0.31) ERR per 100 Bq m^{-3} adopted in the previous UNSCEAR 2006 Report [U6]. However, the Spanish study is much smaller (349 cases and 512 controls compared to 7,148 cases and 14,208 controls for the European pooled study), and the Spanish study did not provide estimates of linear excess relative risk. Estimates from the other residential studies reviewed were closer to and had wider confidence intervals that included the adopted ERR. There still exists a degree of variability in residential risk estimates, for example with regard to smoking habits and sex and uncertainty of exposure in dependence of duration of measurements.

172. Residential and occupational risk estimates can be compared by converting a residential radon concentration into a cumulative exposure. To do so, assumptions must be made about home occupancy, the equilibrium of radon with its progeny, and time-period of exposure. Applying 7,000 hours indoor occupancy in a year an equilibrium factor of 0.4 and the European pooling exposure period of 30 years [D2], the relationship between radon concentration and cumulative radon progeny exposure is that 100 Bq m^{-3}=13.2 WLM [I6]. Accordingly, the UNSCEAR 2006 Report [U6] residential ERR estimate can be expressed as 1.21 (95% CI: 0.38, 2.35) per 100 WLM, which is between the occupational estimates for entire cohorts and subcohorts of more recent work periods, closer to the occupational estimate of 1.53 (95% CI: 1.11, 1.94) per 100 WLM at low exposure.

IV. FUTURE RESEARCH NEEDS

173. Even though extensive research has been conducted on dosimetric and epidemiological evaluations related to lung cancer from exposure to radon and progeny, large uncertainties were reported. The main uncertainties in the assessment of dose by using the dosimetric approach are primarily due to the uncertainty and variability of model parameter values and uncertainties associated with the assumptions built into the particular model, including over-simplification of the underlying processes. The current literature review has highlighted several areas, where future research is required in order to improve estimates for dose conversion factor. Estimates of dose conversion factors still vary widely irrespective of the chosen approach and future research should aim at reducing the uncertainties inherent in both dosimetric and epidemiological studies where possible.

174. Further research studying the influence of smoking versus non-smoking on incidence of lung cancer attributable to radon exposure is necessary for subgroups of different age and sex as well as for different working environments/conditions. Limitations in evaluation of differences in risk across subgroups of the population include low precision due to small numbers of lung cancer cases among non-smokers, women, younger age groups as well as the lack of studies conducted on radon exposure during childhood. Besides the risk of lung cancer, further studies need to be conducted on risks of cancer other than lung and non-cancer diseases. Ongoing projects, such as the PUMA study would be able to provide important new information to improve knowledge on radon risks.

175. For existing residential studies, further combined analyses should be conducted in order to take modifying factors, such as age and sex, into consideration, while for studies of uranium miners, further analyses are needed to address exposure rate effect. Exposure estimates should also be described in more detail, including exposure uncertainties, method of measurements (radon gas or radon progeny measurement), and number of radon measurements in calendar periods.

176. In workplaces, other possible confounding factors such as asbestos, crystalline silica, diesel engine exhaust, nickel, chromium, lead and other ionizing radiations such as external gamma radiation or long-lived radionuclides also merit further research to study the extent to which they can alter or enhance the effects of exposure to radon.

177. Further research studying effect modifiers such as childhood exposure, exposure rate, time since exposure and attained age is also important, as well as more research detailing uncertainty analyses when calculating dose conversion factors.

178. In addition, further research is needed to assess the impact of low dose and low dose-rate exposures to radon. In order to reduce the impact of misclassification of exposure, this research should focus on studies, or time periods within studies, with the best exposure assessment available.

179. The current literature review has also highlighted the need for further research investigating the effects of thoron. Since thoron and its progeny can be a significant component of the total exposure in some specific situations (workplaces or dwellings), it can be an additional source of error in radon studies that do not distinguish radon and thoron contributions to the total exposure. Thoron contamination in radon measurements could have an impact on the assessment of lung cancer risk following radon exposure. In a similar manner, further research regarding the possible influence of external radiation on risk assessment for lung cancer is also identified as a future research need.

V. GENERAL CONCLUSIONS

180. The Committee has previously considered the issue of sources and effects of exposure to radon (^{222}Rn) and thoron (^{220}Rn) and their progeny with regard to workers and the public in several evaluations. The Committee confirmed that inhalation of radon and its progeny is an established carcinogen for the lung. Since its last evaluation in the UNSCEAR 2006 Report [U6], many new articles have been published concerning radon exposure and lung cancer, including epidemiological studies, as well as relevant information on radon dosimetry. The present work, in agreement with the Committee's mandate from the United Nations General Assembly for estimating effects and risk from radiation exposure, has thoroughly assessed the newly available literature, and presents the Committee's conclusions here.

181. The Committee reviewed 33 dosimetric assessments in homes, indoor workplaces and mines. For home exposures, the range of the assessed effective doses per unit of exposure of EEC of ^{222}Rn are from 7 to 34 nSv per h Bq m^{-3} with an arithmetic mean of 18 nSv per h Bq m^{-3}, and a geometric mean of 16 nSv per h Bq m^{-3}. These values are consistent with the central value of 15 nSv per h Bq m^{-3} with a range of 8–40 nSv per h Bq m^{-3} of EEC of ^{222}Rn previously estimated by the Committee for average indoor conditions based on dosimetric evaluations.

182. The Committee identified 26 articles reporting on epidemiological studies (five residential and 21 occupational) of lung cancer risk from radon exposure published since 2006. Among these new studies, the ERR estimates from residential studies varied from −0.13 to 0.73 per 100 Bq m^{-3} for exposure to radon gas concentration, with the mean ERR of 0.13 per 100 Bq m^{-3}. It is noted that no new pooled

analysis has been carried out after the three major pooled residential radon studies reported in the UNSCEAR 2006 Report [U6]. However, a recent analysis of radon exposures for 66 countries concluded that 16.5% of lung cancer cases were attributable to residential radon in 2012 [G1]. Currently reviewed epidemiological studies did not assess exposure in childhood or adolescence. Thus, the impact of radon exposure at young ages on the induction of lung cancer remains unclear.

183. Occupational studies of miners published since the UNSCEAR 2006 Report [U6] were based on extended follow-up of earlier cohort studies and the new Wismut study. Substantial variability in ERR estimates was observed in updated occupational studies, with values ranging from 0.19 to 3.4 per 100 WLM without adjustment for modifying factors. Based on a random-effects meta-analysis with inverse-variance weighting, the combined ERR estimated from the entire study cohorts is 0.60 (95% CI: 0.34, 0.87) per 100 WLM, in close agreement with the previous combined estimate of 0.59 (95% CI: 0.35, 1.0) per 100 WLM [U6]. A higher combined ERR estimate of 1.53 (95% CI: 1.11, 1.94) per 100 WLM was obtained when restricting the analysis to more recent work periods and lower exposure. Preference is given to the latter estimate due to improved radon exposure assessments in more recent periods and to these radon exposures being more reflective of current mining conditions. However, this estimate is less precise due to smaller sample sizes.

184. Residential and occupational risk estimates can be compared by converting a residential radon concentration into a cumulative radon progeny exposure with 7,000 hours indoor occupancy in a year, using an equilibrium factor of 0.4 and cumulative exposure period of 30 years. The residential ERR estimate of 0.16 per 100 Bq m^{-3}, adjusted for exposure uncertainty, can thus be expressed as 1.21 (95% CI: 0.38, 2.35) per 100 WLM, which is between the occupational combined risk estimate of 0.60 for the combined entire miner cohorts and 1.53 for combined subcohorts for more recent work periods. Although similar, residential and miner risks are not expected to be the same due to the different conditions under which the two populations are exposed.

185. A notable limitation of the combined estimates is that they ignore the modifying effect of exposure rate and attained age across studies, which reduces the comparability of individual results. In studies that did report on effect modifiers, risk was observed to decrease with increasing attained age, with increasing time since exposure, and with increasing exposure rate, albeit only for cumulative exposure above 50 to 100 WLM. Modifying effects of sex could not be investigated by the occupational studies. It is concluded that models including effect modifiers are preferred for better comparability across studies and improved risk transfer to specific combinations of modifying factors.

186. Lifetime risk up to age 95 from constant exposure to 2 WLM per year from age 18 to 65 years was estimated by applying the BEIR VI exposure-age-concentration model to the Czech, Wismut and Eldorado miner studies and the combined 11 studies used in the BEIR VI report. The estimates of lifetime excess absolute risk were 2.4 per 10,000 persons WLM^{-1} for the newly published large Wismut study, 3.9 for the updated Czech study and 7.5 for the updated Eldorado study. For the BEIR VI studies, the estimated lifetime risk was 5.5 per 10,000 persons WLM^{-1}. The totality of this evidence is compatible with previous UNSCEAR assessments of lung cancer risk due to radon.

187. The joint effect of smoking and radon exposure was assessed in several epidemiological studies. Smoking-radon interaction was studied occupationally in a combined analysis of three European case-control studies as well as individually in each of the Colorado, Newfoundland and Wismut cohort studies. Evidence of sub-multiplicative interaction was found in the combined analysis, whereas sub- to supra-multiplicative interaction was indicated by the Wismut analysis. Reported risk estimates per unit exposure were higher among non-smokers in all but the Newfoundland study, although confidence intervals in general were wide due to relatively small numbers of lung cancer cases among non-smokers. In residential studies, no obvious deviation from a multiplicative effect has been consistently observed,

although point estimates have been reported as higher for non-smokers than for smokers in both European and North American pooled analyses. The difference in results may be due to lower statistical power in residential studies, a reflection of substantially lower exposure levels and smaller risks attributable to radon. However, as in the occupational analyses, large uncertainties in the estimates were reflected by wide confidence intervals and lack of statistically significant differences across smoking status.

188. Neither the new nor the previous residential studies show a significant difference in risk per unit exposure from radon for smokers and non-smokers. Analyses of miner studies show largely sub-multiplicative joint effect of radon and smoking. Assuming a synergistic effect of smoking and radon means that the lifetime absolute risk from radon depends on the prevalence of smoking in the population—when the prevalence decreases, the risk decreases.

189. Even though extensive research has been conducted on dosimetric and epidemiological evaluations, uncertainties remain large. The main uncertainties in the assessment of dose arise from dosimetric considerations, which are primarily due to the uncertainty and variability of model parameter values and uncertainties associated with the assumptions built into the particular model including over-simplification of the underlying processes. Both the miner studies and the residential studies of lung cancer risk from radon are subject to limitations arising mainly from uncertainties in estimates of radon exposure, particularly in early mining periods, and confounding by other exposures such as smoking. Limitations in evaluation of differences in risk across subgroups of the population include low precision due to small numbers of cases among non-smokers, women and younger age groups. As outlined in the UNSCEAR 2012 Report [U7] on uncertainties in risk estimates for radiation-induced cancer, uncertainties are likely to underestimate ERR estimates in studies of residential radon by 50–100%. Since thoron and its progeny can be a significant component of the total exposure in some specific situations (workplaces or dwellings), it can be an additional source of error in radon studies that do not distinguish radon and thoron contributions to the total exposure. Thoron contamination in radon measurements could have an impact on the assessment of lung cancer risk following radon exposure.

190. Given that the uncertainties from both dosimetric and epidemiological aspects of studies give rise to a broad range of risk estimates, and that values from the current dosimetric and epidemiological reviews are consistent with those used in previous UNSCEAR reports, the Committee further recommends, when estimating the impact of radon exposure levels in a population, the use of the general dose conversion factor of 9 nSv (h Bq m^{-3})$^{-1}$ EEC of ^{222}Rn, which corresponds to 1.6 mSv (mJ h m^{-3})$^{-1}$ (5.7 mSv WLM^{-1}). Applying the Committee's default equilibrium factors for the indoor and outdoor environment of 0.4 and 0.6 results in a dose conversion factor in terms of ^{222}Rn gas exposure of 3.6 nSv (h Bq m^{-3})$^{-1}$ indoors and 5.4 nSv (h Bq m^{-3})$^{-1}$ outdoors.

191. The evidence reviewed by the Committee is compatible with the previous UNSCEAR assessment of lung cancer risk due to radon. Therefore, the Committee concluded that there is no reason to change the established dose conversion factor for its assessments. The Committee continues with its general review of population exposure to radon, with a focus on the consequent risk of lung cancer.

ACKNOWLEDGEMENTS

The Committee acknowledges with gratitude the work of the members of the Expert Group involved in preparing this annex, in particular, P. Strand (Norway) who chaired the Expert Group and J. Chen (Canada) who acted as Senior Technical Adviser. The Committee would like also to thank M. Tirmarche (France), M. Kreuzer (Germany) and N. Harley (United States) for critically reviewing the manuscript. M. Eidemüller (Germany) kindly contributed to the section on alternative risk modelling. The views expressed in the annex remain those of the Committee and do not necessarily represent the views of the United Nations or its Member States.

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APPENDIX A. RADON DOSIMETRY

A1. This appendix describes the special quantities and units used to characterize the concentration of the radon and radon progeny in air, and the resulting inhalation exposure. Further, dosimetric models of the human respiratory tract are used for calculations of the absorbed dose to the target tissues of the lung per unit exposure to radon progeny. These models together with various influencing parameters are described in this appendix.

I. SPECIAL QUANTITIES AND UNITS

A2. The radon activity concentration is the activity per unit volume of the gas, expressed in units of Bq m^{-3}. The dose to the lung mainly arises from the inhalation of airborne radon progeny and the alpha particles emitted during their decay and that of their short-lived progeny. Consequently, the quantity "potential alpha energy concentration (PAEC)" of the radon progeny mixture was historically used as a measure of concentration that was an indicator of dose and risk. The PAEC is the total alpha energy ultimately emitted by the decay of the short-lived progeny in unit volume of air. The historical unit of PAEC was the working level (WL), where a concentration of 1 WL is defined as any combination of the short-lived radon progeny in 1 m^3 of air that will result in the emission of 1.300×10^8 MeV of alpha energy [I6, I13, U1]:

$$1 \text{ MeV} = 1.602 \times 10^{-13} \text{ J}$$

$$1 \text{ WL} = 1.300 \times 10^{8} \text{ MeV m}^{-3} \text{ or } 2.08 \times 10^{-5} \text{ J m}^{-3}$$

A3. The equilibrium equivalent concentration (EEC) is defined as the activity concentration of radon gas in equilibrium with its short-lived progeny which would have the same potential alpha energy concentration as the existing non-equilibrium mixture. One WL equals approximately 3,750 Bq m^{-3} of EEC of ^{222}Rn (radon gas) or approximately 275 Bq m^{-3} of EEC of ^{220}Rn (thoron gas) [I13]. The EEC is therefore a measure of the radon progeny concentration or more precisely the PAEC. The EEC (in Bq m^{-3}) can be converted to the PAEC by the relationships [U6]:

$$1 \text{ Bq m}^{-3} = 5.56 \times 10^{-9} \text{ J m}^{-3} = 2.67 \times 10^{-4} \text{ WL } (^{222}\text{Rn})$$

$$1 \text{ Bq m}^{-3} = 7.56 \times 10^{-8} \text{ J m}^{-3} = 3.64 \times 10^{-3} \text{ WL } (^{220}\text{Rn})$$

A4. The equilibrium factor, F, is defined as the ratio of the EEC to the radon gas concentration (C_{Rn}). In other words, it is the ratio of the PAEC for the actual mixture of radon progeny to that which would apply at radioactive equilibrium:

$$\text{EEC} = F \times C_{Rn}$$

A5. Exposure is the time integral of the concentration. The potential alpha energy (PAE) exposure is the time integral of the PAEC in air and has the historical unit of the working level month (WLM).

The WLM is defined as the cumulative exposure from breathing an atmosphere at a concentration of 1 WL for a working month of 170 hours. The relationship between the historical and SI units is:

$$1 \text{ WLM} = 3.54 \text{ mJ h m}^{-3}$$

$$1 \text{ mJ h m}^{-3} = 0.282 \text{ WLM}$$

A6. The exposure in terms of EEC (in h Bq m^{-3}) can be converted to the PAE exposure by the relationships:

$$1 \text{ h Bq m}^{-3} = 5.56 \times 10^{-6} \text{ mJ h m}^{-3} = 1.57 \times 10^{-6} \text{ WLM } (^{222}\text{Rn})$$

$$1 \text{ h Bq m}^{-3} = 7.56 \times 10^{-5} \text{ mJ h m}^{-3} = 2.14 \times 10^{-5} \text{ WLM } (^{220}\text{Rn})$$

For ^{222}Rn, if the exposure is expressed in terms of the radon gas concentration then the two units are related via the equilibrium factor: 1 WLM = $(6.38 \times 10^5/F)$ h Bq m^{-3}.

II. DOSIMETRIC MODELS

A7. Calculations of the absorbed dose to the target tissues of the lung per unit exposure to radon progeny can be carried out with dosimetric models of the human respiratory tract. A dosimetric model for the respiratory tract needs to describe the morphometry, the respiratory physiology, the deposition of the inhaled material, clearance from the respiratory tract and the dosimetry including the location of target tissues and cells at risk.

A8. The human respiratory tract is composed of three functional regions: *(a)* the extrathoracic region, consisting of the anterior nose, posterior nasal passages, larynx, pharynx and mouth, acting primarily as a filter to protect the lungs; *(b)* the tracheobronchial region, consisting of bronchial and bronchiolar airways, whose primary task is to conduct air to the gas-exchange region; and *(c)* the alveolar interstitial (pulmonary) region, where the gas exchange between lung and blood takes place via the alveoli. Clearance of deposited material is a competitive process between absorption into blood and particle transport to the alimentary tract and lymph nodes. The tracheobronchial and alveolar regions differ in their particle transport mechanisms, with relatively fast mucociliary clearance in the bronchial and bronchiolar airways and relatively slow macrophage-mediated transport in the alveolar region.

A9. There are currently three different type of modelling approaches used to calculate doses to the lung from inhaled radon progeny:

- *Deterministic airway generation models (DAGM)*. These are anatomical or biological models in which the absorbed dose to the sensitive target cells for each airway generation of the tracheobronchial tree is calculated. Such models include those developed by Winkler-Heil et al. [W12, W13]; Harley et al. [H1] and Zock et al. [Z5]. To calculate an average lung dose, a weighting procedure for combining the target cell doses needs to be applied.

- *Stochastic airway generation models (SAGM)*. These are also anatomical or biological models but here the inherent asymmetry and variability of the human airways system is described in terms of probability density functions of airway parameters [H9, H10]. Monte Carlo methods are applied to obtain a distribution of absorbed doses to the sensitive target cells for each airway generation reflecting inter- and intra-subject variability in the morphology of the lung.

– *Semi-empirical compartment models*. Such as the ICRP human respiratory tract model (HRTM) [I7]. In comparison with the airway generation models, these models simplify the anatomical representation of the respiratory tract by combining airway generations into compartments.

A10. In comparison with airway generation models, the overall structure of the HRTM has been simplified for the purposes of dosimetry. For example, the lung is divided into three regions: the bronchial region (BB), the bronchiolar region (bb) and the alveolar interstitial (AI) region. Target cell layers within the BB and bb regions are identified. For the BB region, two layers are defined; one that contains basal cells and the other that contains secretory cells, which lie at different depths. The HRTM defines the absorbed dose to the BB region as the average dose of two layers, giving equal weighting to the basal and secretory layers: $D_{BB}=0.5D_{bas}+0.5D_{sec}$. The absorbed dose to the bb region is given by the average dose to the target cell layer in that region, which are Clara cells (a type of secretory cell). It is the dose to the target cells in the BB and bb regions of the lung that are of importance for radon progeny. In comparison, the dose to the alveolar region is significantly lower [M1, U1].

A11. In the DAGM, the tracheobronchial tree consists of about 15 airway generations with all airways in a given generation being identical in terms of length, diameter and branching angle. The SAGM consists of a variable number (12 to 20) of asymmetric airway generations with airways in a given generation having variable dimensions. Basal and secretory cells are considered to be the target cells for bronchial carcinomas located at different depths in the epithelium. Cellular doses in each airway are calculated and the depth distribution of the targets cells is assumed to be a function of airway diameter, whereas in the HRTM a constant depth for the target cell layer is assumed for each anatomical region [W12, W13]. Hofmann and Winkler-Heil weigh the doses to basal and secretory cells in the bronchial region by their volumetric densities in the epithelium [H10]. This results in a higher weight to the basal cells compared with the secretory cells in the bronchi (e.g. 0.83:0.17 in generations 0–4) [W12]. Mucociliary clearance flow rates are also assumed to be proportional to airway diameter [H9, H10].

A12. The equivalent dose to an organ and the effective dose are ICRP quantities used for radiation protection purposes. The HRTM calculates the equivalent dose to the lung as follows: $H_{lung}=w_R\times(A_{BB}\,D_{BB}+A_{bb}\,D_{bb}+A_{AI}\,D_{AI})$, where D_{BB}, D_{bb} and D_{AI} are the absorbed doses to the bronchial (BB), bronchiolar (bb) and alveolar interstitial (AI) regions. The apportionment factors, A_i represent the regional's estimate sensitivity relative to that of the lung and the assumed values are: $A_{BB}=⅓$, $A_{bb}=⅓$, $A_{AI}=⅓$ [I11]. The ICRP radiation-weighting factor, w_R, for alpha particles is 20 [I9]. As H_{lung} contributes most of the effective dose following inhalation of radon progeny, the effective dose can be approximated by multiplying H_{lung} by the lung tissue weighting factor, w_{lung}, of 0.12.

III. DOSIMETRIC PARAMETERS

A13. Equivalent doses to the lung depend mainly on the potential alpha exposure (i.e. radon progeny concentration in air and the duration of exposure), the breathing rate and the aerosols' properties, including the activity size distribution of the radon progeny aerosol and the unattached fraction. If the exposure is characterized by radon gas measurements, a value for the equilibrium factor, F, is required to estimate the concentration of radon progeny in air, or more precisely the EEC. When using dosimetric models to calculate doses to the lung, additional information on various parameters is required, such as the fractional deposition in the airways, mucous clearance rate, absorption into blood, and the location of the target cells in the airways. The ICRU Report 88 [I14] and ICRP Publication 137 [I13] give a detail discussion of model parameter values. A discussion of aerosols parameter values for homes, indoor

workplaces and mines is given in this appendix. Target cell depth in bronchial epithelium, airway dimensions of the bronchial region and the effects of cigarette smoke on the lung mucociliary clearance are also discussed.

A. Aerosol parameters

A14. Radon (^{222}Rn) gas decays into a series of short-lived radionuclides creating an aerosol of solid particles suspended in air. As described in the ICRU Report 88 [I14], the aerosol is created in two steps: after decay of the radon gas, the freshly formed radionuclides react rapidly (<1 s) with trace gases and vapours and grow by cluster formation to form particles around 0.5 to 5 nm in size. Within 100 s after formation, this unattached progeny may also attach to existing aerosol particles in the atmosphere. The attached progeny may have a trimodal activity size distribution, which can be approximated by a combination of three log-normal distributions [P6]. These consist of the nucleation mode with activity median diameters (AMD) between 10 and 100 nm, the accumulation mode with AMD values of 100–450 nm and a coarse mode with an AMD > 1 μm. Generally, the greatest fraction of PAEC is in the accumulation mode.

A15. Measurements carried out by several researchers of the size distribution of the unattached progeny were found to be unimodal with AMD in the range 0.5 to 1.7 nm and geometric standard deviations (σ_g) between 1.2 and 1.4 [I13, M6]. However, Porstendörfer measured a trimodal distribution with AMDs of 0.6, 0.85, and 1.3 nm with σ_g of approximately 1.2 [P6]. The largest mode (1.3 nm) was not measured in places with high radon concentrations. For the purposes of dose calculation and for simplicity, a unimodal distribution is assumed for the unattached progeny with AMD values between 0.8 to 1.2 nm and a σ_g of 1.3 for all exposure scenarios (tables 1–3 of the annex).

B. Homes and indoor workplaces

A16. Typically, nucleation and coarse modes are introduced when there is a specific source such as small aerosols released in cooking or large aerosols from dispersion activities. However, inter-comparison measurements performed in a house in Germany, without additional aerosols (i.e. for an aged aerosol), showed nucleation and accumulation modes with the fraction of the attached PAEC in the nucleation mode, f_{pn}, being about 0.2 [R7]. The AMD of the nucleation mode was about 50 nm. Measurements of the activity size distribution of the attached progeny in a dwelling in Japan also showed a nucleation mode with an activity fraction of 0.14 and an AMD between 15 and 40 nm [K4]. This is in contrast to measurements carried out in France, where the attached size distribution consisted only of the accumulation mode for an aged aerosol [H16].

A17. Porstendörfer [P6] summarized the measurement results of the activity size distribution of the attached radon progeny for dwellings and reported values of f_{pn}=0.3 (0–0.4) and AMD=20–40 nm for the nucleation mode and AMD=210 nm (120–350) for the accumulation mode. It was noted that in low ventilated rooms without additional aerosol sources, the coarse mode is insignificant because of the greater plate-out rate of large aerosol particles on room surfaces. The AMD for the accumulation mode is consistent with results from other studies, which range from 100 to 350 nm for dwellings [I13, I14].

A18. As described in the ICRU Report 88 [I14], only a few activity size measurements have been carried out in indoor workplaces. Reichelt et al. [R6] carried out activity size measurements of radon (^{222}Rn) progeny in offices, workshops, factories, kitchens, agricultural facilities and public buildings, such as schools, hospitals and art galleries. Porstendörfer [P6] summarized their results and suggested dividing workplaces into three categories regarding activity size distribution and particle concentration:

- Workplaces in rooms without coarse particles;
- Workplaces with coarse particles generated by human activities and dispersion processes;
- Workplaces with one dominant aerosol source such as combustion aerosols, resulting in a uni-modal distribution for the attached progeny.

A19. For the first two categories, the nucleation mode represents about 0.3 of the attached PAEC, with a range of f_{pn}=0.2 to 0.5. AMD values for the nucleation mode range from 15 to 40 nm [P6]. Coarse particles produced by re-suspension may occur when ventilation conditions vary considerably and when the ventilation rate is above 0.5 h^{-1} [R6].

A20. The magnitude of the unattached fraction, f_p, depends mainly on ambient particle concentration, which depends on local conditions. Measurements of f_p in homes and indoor workplaces have shown a wide range of values typically between 0.03 and 0.15 with some values greater than 0.2. In working places with additional aerosol sources due to technical processes, combustion and human activities, the particle concentration can be high resulting in low f_p values of about 0.01 or less. However, f_p is greater than 0.10 for poorly ventilated rooms (ventilation rate <0.5 h^{-1}) without additional aerosol sources, rooms with an operating air cleaner, and poorly ventilated underground caves [I14, P6].

A21. Because radon progeny in the air can be removed by plate-out (i.e. by deposition on surfaces), the activity concentrations of the short-lived radon progeny in the air are less than that of the radon gas. This is quantified by the equilibrium factor (*F)*, which is a measure of the degree of disequilibrium between the radon gas and its progeny. The equilibrium factor (*F*) is decreased further (i.e. there is greater disequilibrium) as a result of ventilation because as the ventilation rate increases, there is less time for the radon progeny to grow-in. If the exposure is characterized by radon gas measurements, then a value for the equilibrium factor (*F*) is required to estimate the radon progeny concentration in air for the calculation of doses to the lung.

A22. The UNSCEAR 1988 Report adopted a value of 0.4 as the average *F* value for dwellings and this value was retained in the UNSCEAR 2006 Report [U2, U6]. The Committee noted that while indoor measurements carried out in the United States and in India show a range from 0.1 to 0.9, most were within 30% of the typical value of 0.4 [H12, R4]. The ICRP recommended an *F* value of 0.4 for indoor workplaces and homes [I13]. A recent review of more than 14,500 measurements in 20 countries showed that measured *F* values vary widely, from as low as 0.1 to as high as 0.8, with a mean of 0.41 and a standard deviation of 0.11 [C12]. The *F* values are significantly higher in poorly ventilated houses and in homes, where smokers abide. The wide range of *F* values suggests that location specific values are more appropriate than a worldwide average value when calculating lung bronchial doses based on radon gas measurements.

A23. For indoor air, where the ventilation rate is not too high, *F* is negatively correlated with f_p [I13, I14, U2]. Consequently, it has been shown that for indoor air, the radon gas concentration is a better index of dose than the PAEC or EEC under a range of aerosol conditions normally encountered [V5, V6]. Based on their 430 measurements of f_p and *F*, Vargas et al. derived a log-power equation between f_p and *F* [V6]. Using this equation with the HRTM it can be shown that as *F* varies between 0.2 and 0.7, the effective dose rate per radon gas concentration only varies from 7–13 nSv per h Bq m^{-3} (i.e. within 30% of the

mean) [M1]. However, if the correlation is not considered then the effective dose rate would vary from 5–19 nSv per h Bq m^{-3} (i.e. within 60% of the mean). Thus, if location specific values of F are used when calculating doses from radon gas measurements, an appropriate value of f_p should also be used.

A24. Some of the ambient aerosols, to which radon progeny are attached, are hygroscopic and are assumed to grow rapidly on inhalation. Typically, this is modelled by assuming the AMD increases instantaneously by the hygroscopic growth factor (*hgf*) as the particle enters the nose or the mouth. It is generally assumed that *hgf* have values between 1 and 2 for attached progeny [I13, I14]. The size of the unattached progeny is assumed to remain constant in the lung.

A25. Sinclair et al. [S11] carried out growth measurements of atmospheric particles, originating from an industrial area close to the sea, and found that the diameters increased by a factor of two when the relative humidity (RH) increased from zero to 98%. The authors expected the ambient aerosol to consist of a mixture of NaCl and $(NH_4)_2SO_4$ salts with a mixture of acids (HNO_3, H_2SO_4 and HCl). The data also showed that the particle diameters would increase by factors of 1.5 and 1.8 as the humidity increases from the relative humidity of indoor air (~30–50%) to 99% relative humidity in the respiratory tract. Measurements of growth factors of a background continental aerosol had two modes with values of 1.5 for the low hygroscopic mode and 2.9 for the more hygroscopic mode [P1]. Li and Hopke measured *hgf* of indoor combustion aerosols including cigarette smoke, incense smoke, candle flame, natural gas flame and propane fuel flame [L8]. The average *hgf* values ranged from 1.5 to 1.9, which represents maximum growth as these measurements were made starting with a dried aerosol and increasing the humidity to 99% relative humidity. In contrast, radon progeny attached to aerosols produced from cooking oil are hydrophobic [D9]. Based on these data, it could be assumed an *hgf* value of 1.5–2.0 for attached progeny following indoor exposure (tables 1–3 of the annex).

C. Underground mines

A26. The activity size distribution of the radon progeny in underground mines depends on the different types of mining conditions and activities such as use of diesel- or electric-powered equipment, different ventilation rates, and use of propane heaters, blasting, drilling and ore carrying equipment. Results from activity size measurements carried out in mines have been summarized by the ICRU [I14] and by the ICRP [I13]. For example, in mines, where the aerosol is dominated by diesel particles, the size distribution of the attached progeny is unimodal with an AMD of about 250 nm [I13].

A27. Tu et al. [T20] carried out measurements in the winter of 1995 to characterize the aerosol in a wet underground uranium mine (Eagle Point mine) in northern Saskatchewan, Canada. The mine uses diesel-powered equipment, propane heaters, and is ventilated at a high rate (~one air change per 3 minutes) [C1]. Measurements carried out at a stope and a drilling area, where miners were working, showed predominately a two-modal distribution for the attached progeny. The fractions of the attached PAEC associated with the "nucleation" and accumulation modes were approximately 65 and 35%, with mean AMDs of ~60 and ~330 nm, respectively. For the unattached progeny, the AMD was about 0.6 nm and f_p was 0.01. Measurements were also carried out at a bolt-storage bay next to a major mine exhaust, which gives an estimate of the "average" mine atmosphere. Most of these measurements showed that the attached progeny consisted of the nucleation mode containing approximately 97% of the attached PAEC, on average, with AMD values between 55 and 75 nm. The coarse mode accounted for the remaining 3% of attached PAEC with AMAD between 2 and 8 μm. For the unattached progeny, the measured AMD at the bolt-storage bay was mainly about 1 nm (0.6–3 nm) with f_p less than 0.02. The authors concluded that a combination of diesel particles and combustion particles from burning

propane gas caused a modal diameter shift to a smaller size range, 50–85 nm compared with the previous reported values (100–200 nm). Calculations with the HRTM for a mine atmosphere show that a shift from an AMD of 200 to 65 nm would increase the effective dose coefficient by a factor of two [I13]. The authors also noted that there was no nuclei mode in the range of 2–30 nm.

A28. Aerosol measurements were also carried out during the summer time of 1996 at the same Canadian mine (Eagle Point mine) in northern Saskatchewan [C1, C2, C3]. During the summer months propane heaters are not used. Throughout the mine, the measured AMD values ranged from 50 to 120 nm with a mean value of 85 nm and σ_g of approximately 2.0. The average value of f_p was approximately 6%, whereas the expected value based on particle concentration was 0.3%. This unexpectedly high f_p value was theoretically shown to occur under conditions of high ventilation, where the radon progeny is far from equilibrium [C1]. The average value of the equilibrium factor F was 0.08.

A29. In 2013, activity size distribution measurements of radon progeny were carried out at the Olympic Dam underground uranium mine, Roxby Downs, South Australia [S14]. Measurements were made with a multiscreen continuous diffusion battery at six locations representing a range of working atmospheres. Two were at underground workshops, characterized by high ventilation rates, low PAEC and low F values. The activity size distribution at these sites were mainly bimodal, one with unattached and accumulation modes and the other with nucleation and accumulation modes (table 3 of the annex). The other four sites were in locations within the operational areas of the mine with higher levels of PAEC. Three of these sites had bimodal distributions consisting of nucleation modes with mean AMDs of ~30 to 40 nm and accumulation modes with mean AMDs of ~180 nm. The fraction of the nucleation mode varied from 0.1 to 0.3 across these three sites. The fourth site near an ore crusher showed a single accumulation mode with a mean AMD of 114 nm. These measurements are important as they show the presence of the nucleation mode and because there is very little data on aerosol characteristics in modern mines (table 3 of the annex).

D. Target cell depth in bronchial and bronchiolar epithelium

A30. Measurements of the basal and secretory cell depths in human bronchial epithelium have been made mainly for radon progeny bronchial dose models [B3, B4, G2, M8, R10]. Robbins et al. [R10] obtained human bronchial specimens removed for various pathologies from 115 persons and dissected airway generations 3 to 6 from non-involved areas of the lungs. The cell depth was measured from the midpoint of the nucleus to the free epithelial surface. The arithmetic mean basal and secretory cell depths averaged over airway generations 3 to 6 were 27 and 19 μm, respectively [I14]. The average cell depths were approximately the same for male and female smokers, non-smokers and ex-smokers. Generation 3 also had a small percentage of samples having basal cell depths ranging from 40 to 70 μm.

A31. Baldwin et al. [B3] obtained tissue from 29 patients undergoing lobectomy for lung carcinoma and non-specific inflammatory changes. Measurements of the basal cell depth (epithelial surface to basal cell nucleus) gave similar results as reported by Robbins et al. [R10]. However, the airways in the Baldwin study [B3] were characterized by diameter, which does not permit exact identification of airway generation as a range of diameters exist for each generation [I14].

A32. Mercer et al. [M8] obtained three lung lobes from surgical resections that were used for study of the airway epithelium of the human lung. The lobes were from non-smokers who had normal pulmonary function tests and chest radiographs. The lobes were used to measure the distribution of nuclear cell depth for different cell types in the bronchi and bronchioles. In large and small bronchi, the basal cell depths

averaged for the three lungs ranged mainly from 36 to 48 μm with most cells at 48 μm. For the secretory cells, the nuclei depths ranged mainly from 12 to 36 μm in the large bronchi and from 24 to 48 μm in the small bronchi. In the bronchioles, basal cells were absent, and the secretory cell depths ranged mainly from 4 to 8 μm. The nuclei depths were measured from the epithelial surface to the cell nucleus (excluding cilia).

A33. The histological measurements of Mercer et al. [M8] are the basis of the model of the target cell depth distribution (basal and secretory cells) of the HRTM [I7], the DAGM and SAGM models of Zock et al. [Z5], Winkler-Heil et al. [W12, W13] and Hofmann et al. [H9, H10]. In contrast, based on the data of Robbins et al. [R10], the DAGM of the bronchial region developed by Harley et al. [H1] assumes smaller target cell depths, particularly for the basal cell nuclei. Thus, the target cell depth distribution is a source of uncertainty. Harley [H3] estimated that the bronchial dose can change by ±20% due to the different target cell assumptions selected by various models. With the HRTM, Marsh and Birchall [M1] estimated that the dose conversion factor increases from 15 to 21 mSv WLM^{-1}, if the target cell depth and thickness is decreased by a factor of 2 (table 4 of the annex).

E. Airway dimensions

A34. Available morphometric models of the human tracheobronchial tree are based on measurements of a small number of individual measurements from a few laboratories. To determine the degree of variability of the bronchial tree, Nikiforov and Schlesinger performed an analysis of airway lengths, diameters and branching angles using solid casts of the upper bronchial tree from eight human right lungs [N7]. The casts were made from the level of the main bronchus (generation 1) through the eighth branching level beyond the trachea (generation 8). The results showed that there is considerable variability in the lengths, diameters and branching angles within the same generation. The intersubject differences in dimensions generally increased with increasing generation number. For example, the coefficient of variation for diameters by generation for the eight casts varied linearly from 13% for generation 1 to 40% for generation 8. The intersubject variation of the airway dimension affect deposition of inhaled radon progeny and consequently the bronchial doses [H9].

A35. The morphometric models published by Weibel [W4] and by Yeh and Schum [Y1] were compared with the airway dimension data of Nikiforov and Schlesinger [N7]. The length and diameter mean of the Weibel model were in good agreement with the corresponding means of the measured data of Nikiforov and Schlesinger (within 20%). The length and the angle mean of the Yeh and Schum model were also in similar agreement with the means of the measured data but the mean diameters of the Yeh and Schum model were considerably larger. However, comparisons should take account of relative inflation of the fixed lungs of the cast. Yeh and Schum [Y1] acknowledged their preparation of the casts resulted in enlargement of the airways, probably to the total lung capacity. Thus, they recommended that the airway dimensions should be scaled down to functional residual capacity. Before evaluating the effect of different morphometric models on lung dose, Yu et al. [Y3] scaled the morphometric models to the functional residual capacity of ICRP Reference Man [I8].

F. Lung clearance of smokers and non-smokers

A36. Studies of the effects of cigarette smoke on the lung mucociliary clearance have shown conflicting results [I7, I14]. Based on studies with inhaled radioactive labelled particles, Albert et al. [A3] showed that the 90% clearance time (i.e. the time after which 90% of the inhaled particles were cleared from the lungs) was the same for both smokers and non-smokers. In comparison, Aldas et al. [A4] observed an overall faster clearance in smokers probably because of more central deposition. However, the authors stated that the clearance rate in the large proximal airways was slower than in non-smokers ($T_{1/2}$=2.3 hours in smokers; $T_{1/2}$=42 minutes in non-smokers), but faster in the more peripheral airways. Generally, slower clearance in the trachea or the large proximal airways is observed for chronic smokers compared with non-smokers [A1, C21, T17]. Vastag et al. [V7, V8] found that the lung mucociliary clearance (overall and inner zone) was less for smokers compared with non-smokers and decreased with cigarette consumption. The decrease was more pronounced in the central than the peripheral airways.

IV. PARTITION OF DETRIMENT AMONG THORACIC REGIONS

A37. Considering differences in radiation sensitivity between different regions of the lung apportionment factors, A_i are applied in the calculation of equivalent dose to the lung as follows: $H_{lung}=A_{BB}\,H_{BB}+A_{bb}\,H_{bb}+A_{AI}\,H_{AI}$, where H_{BB}, H_{bb} and H_{AI} are the equivalent dose to the bronchial (BB), bronchiolar (bb) and alveolar interstitial (AI) regions. The apportionment factors, A_i represent the regional's estimated sensitivity relative to that of the lung. Thus, A_i represents the fraction of the total lung detriment assigned to region *i* of the lung following uniform exposure of the lung. In deriving values for the apportionment factors, ICRP considered applying the relative risk concept, in which it is assumed that the induction of cancer by radiation exposure is proportional to the background cancer rates in tissue [I7].

A38. In the development of the HRTM, values of the apportionment factors, A_i were initially derived on the basis of regional distribution of background lung cancer rates in the general population [J1]. Initial estimates were: 0.8 for the BB region, 0.15 for the bb region and 0.05 for the AI region. These estimates were reviewed and values of 0.6 for the BB region, 0.3 for bb region and 0.1 for the AI region were obtained for a population of non-smokers and smokers [I7]. ICRP reviewed data from studies of underground miners exposed to radon, Japanese atomic bombing survivors and from experiments with laboratory animals [I7, L2, M11, Z6]. Following this review, ICRP noted that uniform irradiation of the thorax is more likely to lead to the induction of cancer in the BB region than in the AI region and probably also in the bb region. However, it was concluded that there is no basis for deriving factors, with any acceptable degree of confidence, to represent regional differences in radiation sensitivity among the three regions of the lung: BB, bb and AI. Consequently, equal weighting was assigned to each of the regions (A_{BB}=0.333, A_{bb}=0.333, A_{AI}=0.333).

A39. Saccomanno et al. [S1] measured the distribution of tumours in the bronchial tree for a cohort of 467 miners and 311 non-miners. Ninety-five per cent of the miners were cigarette smokers. The position and type of tumour in the miners were compared with those of non-miners of the same age, all with positive smoking histories, who developed lung cancer in the same calendar year as the miner. All the subjects were male. Three anatomical zones were considered: central (0–8 airway generations), middle (9–18 airway generations) and peripheral. These regions approximately correspond to the bronchial (BB), bronchiolar (bb) and alveolar interstitial (AI) regions of the HRTM. The results gave the following distributions of tumours in the BB:bb:AI regions as (0.68:0.15:0.16) for miners and (0.59:0.18:0.23) for non-miners. The majority of the tumours originated in the BB, mainly within the first few airway generations (1 to 3). The number of small cell carcinomas among the miners was a factor of 4 higher

compared with the non-miners, and these mainly occurred in the BB region (~90%). The number of squamous cell carcinomas originating in the BB region was only 20% higher for the miners compared with non-miners. The adenocarcinomas in the bb and AI regions were twice as high for the miners compared with non-miners. To conclude, the data indicates that for smokers, the inhalation of radon progeny preferentially induces small cell carcinomas in the BB region, although an increase was also observed in the bb region. Similar data for non-smokers would be useful information to determine the region's radiation sensitivity relatively to that of the whole lung.

A40. Winkler-Heil et al. [W14] tested the current assumption of equal apportionment factors by comparing different radon and thoron exposures, which produce different regional dose distributions, with pathological observed regional cancer distributions. Exposure conditions considered were *(a)* radon progeny inhalation; *(b)* thoron progeny inhalation; *(c)* thoron and thoron progeny exhalation (thorotrast patients); and *(d)* radon progeny inhalation in rats. Regional lung deposition of cigarette smoke particles was also calculated. Following inhalation of radon progeny or thoron progeny, the calculated regional dose factors were similar for the BB and bb regions, whereas the AI dose was only a few per cent of the average lung dose. The similarity of the BB and bb doses is inconsistent with pathological data that showed more tumours in the bronchial region compared with the bronchiolar region [S1]. This indicates higher radiation sensitivity for the BB region compared with the bb region. In addition, a lower sensitivity for the AI region compared with central airways was inferred from the thorotrast patient data. This was concluded as there were no excess cancers observed among the thorotrast patients despite the relatively high dose to the AI region compared with the bronchial and bronchiolar regions at levels, where lung cancer induction was expected. Taking account of dose enhancement at bronchial bifurcations during inhalation, which increases BB doses relative to bb doses, the authors concluded that apportionment factors of ~(A_{BB}=0.65, A_{bb}=0.30, A_{AI}=0.05) may represent a realistic estimate.

A41. Calculations with the HRTM [M1] and calculations performed by Winkler-Heil et al. using the SAGM gives similar doses to BB and bb regions of the lung [W14]. In contrast, other publications by the same group gave higher doses to BB compared with bb by 60:40 or more using their SAGM [H9, H10, H11]. Hofmann and Winkler-Heil noted that the average bronchial doses are higher than the average bronchiolar doses by a factor of ~2–3 [H11]. The reason for this discrepancy is due to different averaging procedures of doses among different airway generations. Due to the stochastic asymmetry of the airways, all airway generations between ~12–20 can contain both bronchioles and respiratory bronchioles with varying degree of probability. The respiratory bronchioles (bronchioles with some alveoli apposed) are defined as part of the AI region. Higher doses in BB region relative to bb region are obtained if the average bronchiolar dose is calculated by weighting the doses in a given airway generation by the relative frequency of bronchiolar airways in that generation. If the dose to BB region is higher than bb region this may partly explain the pathological data that showed more tumours in the BB region compared with the bb region [S1]. Similar doses to BB and bb regions were obtained when the average bronchiolar dose was calculated without considering the fraction of the bronchiolar and alveolated airways in a given generation [W15]. The revised HRTM [I12] gives similar doses to BB and bb regions of the lung for indoor workplace exposure (table 2 of the annex) whereas for mine exposure, the dose to BB is lower than to bb (table 3 of the annex) assuming ICRP reference aerosol parameter values [I13].

A42. The equivalent dose to the lung is sensitive to the assumed values of the apportionment factors; higher doses are obtained if more weight is given to the BB and bb regions compared with the AI region [H11, I14, M1]. Assuming apportionment factors (A_{BB}:A_{bb}:A_{AI}) of ~(0.65:0.30:0.05) [W14], (0.6:0.3:0.1) [I7], (0.6:0.2:0.2) [S1] and (0.333:0.333:0.333) [I7] gives effective doses calculated with the HRTM for home exposures of 21, 20, 17, 15 mSv WLM^{-1}, respectively [M1].

APPENDIX B. THORON EPIDEMIOLOGY

B1. In recent decades, it has become increasingly clear that exposure to thoron and its progeny cannot be ignored as it contributes to the risks otherwise assigned solely to exposure to radon and its progeny [U6]. Although only a limited number of measurements of thoron are available, levels of thoron in workplaces and residential dwellings of various types were reviewed and summarized in previous UNSCEAR reports [U3, U6].

B2. Up to now, there is no epidemiological study specifically designed to assess lung cancer risk due to thoron exposure in addition to exposure to radon. However, some radon epidemiological studies (both occupational and residential) evaluated the impact or the effect of thoron exposure on the assessment of radon risk [U6].

I. OCCUPATIONAL STUDIES

B3. For studies in workplaces, two studies mentioned thoron [U6]. In the studies of Ontario uranium miners [K11], the Canadian investigators recognized that estimates of radon decay product concentrations involved some uncertainty, especially for the early years of mining, and that thoron progeny contribute an appreciable radiological exposure in the Elliot Lake mines. However, the reconstruction of early underground uranium mining environments undertaken in 1985 confirmed that the exposure values used for the early years did not overestimate the working exposure at that time [U6].

B4. In the studies of French uranium miners [Z3], workplace area monitors were replaced by personal alpha and gamma dosimeters after 1983. Those personal dosimeters record energy depositions of short-lived progeny of radon and thoron, long-lived alpha emitters contained in ore dust as well as gamma radiation. However, there was no mention of either thoron levels recorded or associated effects [U6].

B5. In a subgroup of 592 males who worked for at least one year in selected jobs that had the highest exposure to thorium dust (mean alpha-particle emission per minute per cubic metre of air was 426) and thoron (mean concentration of 32.6 kBq m^{-3}) that was followed up more intensively, the standardized mortality ratio (SMR) for lung cancer was 1.68 (95% CL: 0.81, 3.09), while that for respiratory diseases was 1.20 (95% CL: 0.52, 2.37). The SMRs were assessed as a combined effect from exposure to thorium dust and thoron gas. Information on smoking habits in a sample of survivors suggested that smoking could have explained at least part of the excess mortality from lung cancer and from diseases of the respiratory system.

B6. Solli et al. [S13] investigated the incidence of cancer among 318 male employees of a niobium mining company which was operated between 1951 and 1965. Many of the workers, especially underground miners, were exposed to the daughters of radon and thoron and also to thorium dust. The mean radon daughter concentration was 1.0 WL (ranging from 0.5 to 2.2 WL) and 0.2 WL (ranging from 0.1 to 0.2 WL) for thoron daughters. However, the measurement technique was considered rather crude. The dust concentration in the air was very high at the time of the measurements. The accumulated doses to the workers from short-lived radon and thoron daughters in the mine atmosphere were assessed to be

relatively low; up to 300 WLM. For the radon and thoron daughter exposure, about 50 excess cases per million person-years at risk per WLM were observed. The authors considered thoron daughter exposure to the workers in the mine to be relatively low, and that thoron seemed to have contributed relatively little to the radiation hazard among the workers.

B7. The Bayan Obo Rare-earth Iron Mine is an open pit mine, and has been operated since 1958. Its ore contains not only iron, rare-earth elements and silica dioxide but also thorium (ThO_2) at a concentration of 0.04%; uranium (U_3O_8) at a concentration of 0.0001%; SiO_2 is found at a concentration of 10%. Chen and his colleagues have conducted follow-up studies on thorium lung burdens of thorium dust exposed miners for more than two decades [C13, C14, C15, C16, C17, C18, C19]. Air concentrations of dust containing thorium in the crushing workshop were extremely high; the average thorium concentration was 189 mg m^{-3} (ranging from 9–875 mg m^{-3}) and the average thoron progeny concentration was 1.08×10^4 MeV L^{-1} (ranging from 4.20×10^2 to 6.77×10^4 MeV L^{-1}), which was a factor of 10 higher than radon progeny concentration in the same area [C13]. The average value of the thorium lung burden from 1,270 measurements among 751 dust exposed miners was 1.6 Bq. About 23% of the 1,270 measurements reached the investigation level of 2.2 Bq for thorium lung burden [C19].

B8. For the total person-years of observation of the 62,712 dust exposed miners and the 34,672 unexposed miners during the 22 years follow-up period, the study showed that the lung cancer mortality of the dust exposed miners was significantly ($p<0.005$) higher than that of the non-exposed group [C16]. Significant increase of incidences of respiratory symptoms, lung function disorders and pneumoconiosis stage 0+ and stage 1 were reported too. They estimated aetiological attribution of risk factors, among the 27 cases of lung cancer deaths in the dust exposed miners, 1 case was induced by inhaled thorium, 2 cases by the inhaled short-lived thoron progeny, 12 cases with cigarette smoking, and the remaining 12 cases to SiO_2. In their latest report [C18], it was concluded that long-term exposure to thorium-containing dusts (carcinogens are ThO_2 and SiO_2) and to thoron progeny induced an excess of lung cancers. However, the high rate of smoking among the Bayan Obo miners (80%) and the high gamma radiation exposure in the mining workshops might have enhanced the carcinogenic effects of the inhaled thorium-containing dust and thoron progeny in inducing the excess lung cancers.

B9. In summary, nine publications were identified in the literature review that assess the exposure to thorium dust and/or thoron progeny in workplaces. In addition to thorium dust, exposure to thoron decay product also contributes to increased risk of developing lung cancer among exposed workers. However, no detailed assessment of thoron induced lung cancer is available to date.

II. RESIDENTIAL STUDIES

B10. In the UNSCEAR 2006 Report [U6], it was recognized that the results of radon epidemiological studies conducted in the Loess Plateau region of China were affected by the presence of thoron and its progeny, and reassessment of risks due to radon exposure may be needed to take the presence of thoron into account. However, for this evaluation, there were a few studies on health effects of residents who lived in cave dwellings in northwestern rural areas of China, where radon and thoron levels are relatively high, no industrial pollution exists and where there has been very little migration before the 1980s [G4].

B11. In order to explore the feasibility of an epidemiological study on lung cancer in areas with elevated indoor radon and thoron exposures, 202 residences including loess caves, brick caves, stone caves, and ordinary houses in 20 villages were selected from Yan'an and Luliang areas in the Chinese Loess Plateau [S20, S21]. Indoor levels of thoron and its progeny as well as radon were determined with passive radon-

thoron discriminative detectors and thoron progeny deposition rate devices. The exposure period covered from August 2001 through August 2002. The indoor radon concentration in loess caves ranged from 17 to 179 Bq m^{-3}, with geometric means of 73 and 71 Bq m^{-3} in Luliang and Yan'an, respectively. Thoron levels varied substantially depending on the distance between the device and the wall, ranged from 10 to 760 Bq m^{-3}, with geometric means of 145 and 185 Bq m^{-3} in Luliang and Yan'an, respectively. Geometric mean of EEC of ^{220}Rn (thoron) were estimated to be 1.6 Bq m^{-3} in Luliang and 2.2 Bq m^{-3} in Yan'an. The study revealed that the indoor air pollution, indexed by cooking fuel source, in Yan'an was low compared with that in Luliang. Migration levels before the 1980s were very low, 86% of the subjects had no history of migration in Yan'an. Ninety per cent of the cave-dwellings, where the subjects once lived, were available for measurements when the survey was conducted. The results showed the investigated cave-dwelling area with low levels of industrial pollution was suitable for conducting epidemiological study on residential thoron and radon exposure and lung cancer. Later on, a pilot case-control study on radon and thoron induced lung cancer was conducted there. However, the results are not yet available at the time of this evaluation.

B12. In summary, like in workplaces, evidence has shown that thoron exposure in residential homes contributes to the total radiation dose to the lung and increases the risk of developing lung cancer. However, no quantitative result of epidemiological studies on thoron induced lung cancer is yet available at the present time.

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