International Commission on Radiological Protection Statement on Radon

Approved by the Commission in November 2009

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

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

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

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

exposures to below the national reference level. It is noted that the World Health Organisation now recommends a similar approach (WHO, 2009).

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

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

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Lung cancer risk from radon and progeny

DRAFT

Editor

……………………..

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CONTENTS

71   **ABSTRACT**

74   This report reviews recent epidemiological studies of lung cancer risk linked to
75   exposure to radon and its progeny. It concentrates on the results from pooled
76   case-control studies of residential exposures and cohorts of underground miners
77   exposed to low levels of radon and radon progeny. Consistent with the approach
78   used in ICRP Publication 65 (1993), recent miner data are used to recommend a
79   revised detriment-adjusted nominal risk coefficient of $5 \ 10^{-4}$ per WLM ($14 \ 10^{-5}$
80   per mJ h m^{-3}), replacing the ICRP Publication 65 value of $2.8 \ 10^{-4}$ per WLM (8
81   10^{-5} per mJ h m^{-3}). Furthermore, pooled analyses of epidemiological studies of
82   lung cancer risk from residential exposures demonstrate a statistically significant
83   increase per unit of exposure below average annual concentrations of about 200
84   Bq m^{-3}. The risk estimates derived from these pooled analysis are consistent with
85   those from underground miners and are sufficiently robust to enable protection
86   of the public to be now based on residential concentration levels. However, for
87   occupational protection purposes, dose estimates are required to demonstrate
88   compliance with limits and constraints. Dose estimates also allow comparisons
89   between various sources of public exposure. ICRP Publication 65 recommended
90   that doses from radon and its progeny should be calculated using a dose
91   conversion convention based on miner epidemiological studies. ICRP now
92   proposes to treat radon and radon progeny in the same way as other
93   radionuclides and will publish dose coefficients calculated using dosimetric
94   models for use within the ICRP system of protection.

95

96 **PREFACE**

97

98 A Task Group was appointed by ICRP Committee 1, with representation of
99 several members from Committee 2 and one member from Committee 4 to review
100 risks from alpha emitting radionuclides. The Commission subsequently asked the
101 Task Group to concentrate initially on radon. This report reviews epidemiological
102 studies of lung cancer risk associated with the inhalation of radon and radon progeny
103 in homes and in underground mines.
104
105 The members of the Task Group were:
106

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134

135

EXECUTIVE SUMMARY

136

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

152   (b) In 1998, the BEIR VI report presented a comprehensive analysis of available
153   miner cohorts (NRC, 1998). Recent studies of lung cancer in miners include
154   relatively low concentrations of radon and radon progeny, long duration of follow-
155   up and good quality of individual exposure data (UNSCEAR, 2009). A recent
156   pooled analysis of the French and Czech miner cohorts also provided risk estimates
157   associated with low levels and good quality of individual exposure (Tomášek et al.,
158   2008a). These results, consistent with previous analyses of combined miner studies,
159   demonstrate statistically significant associations between cumulative radon exposure
160   and lung cancer mortality at levels of exposure as low as 50 Working Level Months
161   (WLM), i.e. 180 mJ h m^{-3}. On the basis of calculations of lifetime excess absolute
162   risk (LEAR), using Publication 103 (ICRP, 2007) reference background rates and
163   risk models from pooled analyses (NRC, 1998; Tomasek et al., 2008a), a detriment-
164   adjusted nominal risk coefficient of 5 10^{-4} per WLM (14 10^{-5} per mJ h m^{-3}) is now
165   recommended for radiological protection purposes. This nominal risk coefficient
166   replaces the Publication 65 value of 2.8 10^{-4} per WLM (8.0 10^{-5} per mJ h m^{-3}).

167   (c) Three comprehensive publications have provided joint analyses of data from
168   domestic case-control studies for Europe (Darby et al., 2005), North America
169   (Krewski et al., 2005; 2006) and China (Lubin et al., 2004). Each joint analysis
170   demonstrated an increased risk of lung cancer with increasing domestic radon
171   concentration, considering exposures over a period of 30 years preceding the
172   diagnosis of cancer. The estimates of an increase of lung cancer per unit of
173   concentration in the three joint analyses are very close to each other and statistically
174   compatible: the values obtained were 1.08, 1.10 and 1.13 per 100 Bq m^{-3}. A
175   combined estimate calculated for Europe, North American and China was 1.09 per
176   100 Bq m^{-3} (UNSCEAR 2009). All of these results were obtained after adjustment
177   for smoking habits. It is noted also that the slope of the linear exposure-response
178   relationships increased to 1.11 per 100 Bq m^{-3} when the analyses focused on those
179   cases and controls with more complete estimation of cumulated individual exposure
180   (UNSCEAR 2009) .

181   (d) The joint analyses also made adjustments to take account of uncertainties
182   associated with variations in radon concentration. For example, in the European
183   pooled analysis (Darby et al., 2005), taking account of such uncertainties increased

184   the estimate of relative risk from 1.08 to 1.16 per 100 Bq m^{-3}. Limiting the European
185   analysis to those cases and controls with a relatively low annual exposure, there is
186   evidence of an increased risk below 200 Bq m^{-3}. It is concluded that the residential
187   studies provide a reasonable estimate of lung cancer risk and a basis for risk
188   management related to low protracted radon exposures in homes, considering
189   cumulated exposure during a period of at least 25 years.

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

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

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

204   (h) However, for the purposes of control of occupational exposures using dose
205   limits and constraints, estimates of dose per unit exposure are required. In ICRP
206   Publications 65 and 66 (ICRP, 1993; 1994), the effective dose per unit exposure to
207   radon and radon progeny was obtained using the so-called dose conversion
208   convention. This approach compared the detriment per unit exposure to radon and
209   its progeny with the total detriment associated with unit effective dose, estimated
210   largely on the basis of studies of Japanese survivors of the atomic bombings (ICRP,
211   1993). The values given were 5 mSv per WLM (1.4 mSv per mJ h m^{-3}) for workers
212   and 4 mSv per WLM (1.1 mSv per mJ h m^{-3}) for members of the public.

213   (i) Doses from radon and radon progeny can also be calculated using different
214   dosimetric models. A review of published data on the effective dose per unit
215   exposure to radon progeny obtained using dosimetric models is included as
216   Appendix B of this report. Values of effective dose range from about 6 to 20 mSv
217   per WLM (1.7 to 5.7 mSv per mJ h m^{-3}), with results using the ICRP (1994) Human
218   Respiratory Tract Model (HRTM) in the range from about 10 to 20 mSv per WLM
219   (3 to 6 mSv per mJ h m^{-3}) depending on the scenario of exposure.

220   (j) ICRP has concluded that doses from radon and radon progeny should be
221   calculated using ICRP biokinetic and dosimetric models, including the HRTM and
222   ICRP systemic models. That is, radon and its progeny should be treated in the same
223   way as other radionuclides within the system of protection. ICRP will provide dose
224   coefficients per unit exposure to radon and radon progeny for different reference
225   conditions of domestic and occupational exposure, with specified equilibrium
226   factors and aerosol characteristics. Until dose coefficients are published, the
227   previously recommended Publication 65 values should continue to be used. It should
228   be recognised, however, that the dose coefficients to be published will be larger by
229   about a factor of two or more.

230

231   **GLOSSARY**

232   Case-control study

233   Type of epidemiological study design in which a group of subjects with the
234   disease of interest (the cases with lung cancer) is compared to a group of
235   subjects that are free of this disease (the controls) but have similar
236   characteristics (sex, attained age…). This type of epidemiological design
237   was especially used in indoor radon studies. For each individual, past
238   exposures are estimated from measurements of radon concentration in
239   current and previously occupied dwellings.

240

241   Cohort study

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

247   Dose conversion convention

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

251   Equilibrium equivalent concentration (EEC)

252   The activity concentration of radon gas, in equilibrium with its short-lived
253   progeny which would have the same potential alpha energy concentration as
254   the existing non-equilibrium mixture.

255   Equilibrium factor, F

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

260   Existing exposure situations

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

264   Human Respiratory Tract Model (HRTM)

265   Model used in ICRP Publication 66 (1994) to evaluate the deposition and
266   clearance of inhaled particles in the respiratory airways as well as the
267   resulting dose to the lung tissues.

268   Planned exposure situations

269   Planned exposure situations are situations involving the deliberate
270   introduction and operation of sources. Planned exposure situations may give

271   rise both to exposures that are anticipated to occur (normal exposures) and to
272   exposures that are not anticipated to occur

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

278   Radon progeny
279   The decay products of radon-222, used in this report in the more limited
280   sense of the short-lived decay products from polonium-218 through
281   polonium-214. Radon progeny are sometimes referred to as "radon decay
282   products".

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

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

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

298   • Excess relative risk (ERR)
299   Relative risk – 1.

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

304   • Risk coefficient
305   Increase of risk per unit exposure or per unit dose. In general, expressed as
306   ERR per WLM, per $J\,h\,m^{-3}$, per 100 $Bq\,m^{-3}$ or per Sv.

307   • Risk model

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

312

313 • Lifetime risk

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

322 • Detriment

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

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

334 Thoron progeny

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

338 Unattached fraction

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

341

342 Upper reference levels

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

345 Working level (WL)

346 Any combination of the short-lived progeny of radon in one litre of air that
347 will result in the emission of 1.3×10^5 MeV of potential alpha energy. 1 WL
348 $= 2.08 \ 10^{-5}$ J m^{-3}.

349 Working Level Month (WLM)

350 The cumulative exposure from breathing an atmosphere at a concentration of
351 1 working level for a working month of 170 hours.

352 Units

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

354 Potential alpha energy concentration:

355 Radon progeny :

356 1 Bq m^{-3} of radon at equilibrium = $3.47\ 10^4$ MeV m^{-3} = $5.56\ 10^{-9}$ J m^{-3}

357 Thoron progeny :

358 1 Bq m^{-3} of thoron at equilibrium = $4.72\ 10^5$ MeV m^{-3} = $7.56\ 10^{-8}$ J m^{-3}

359 Working level:

360 1 WL = $1.3\ 10^8$ MeV m^{-3}

361 1 WL = $2.08\ 10^{-5}$ J m^{-3}

362 Working level month (WLM) :

363 1 WLM = $3.54\ 10^{-3}$ J h m^{-3}

364 1 WLM = $6.37\ 10^5$ Bq h m^{-3} EEC of radon

365 1 WLM = $6.37\ 10^5$/F   Bq h m^{-3} of radon[a]

366 1 Bq m^{-3} of radon during 1 year = $4.4\ 10^{-3}$ WLM at home[b]

367 1 Bq m^{-3} of radon during 1 year = $1.26\ 10^{-3}$ WLM at work[b]

368 1 WLM = $4.68\ 10^4$ Bq h m^{-3} EEC of thoron

369 (a) F = equilibrium factor

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

372

373

1. INTRODUCTION

375 (1) Radon-222 is a naturally occurring radioactive gas, with a half-life of 3.8
376 days. It is formed as the decay product of radium-226 (half-life 1600 years), which
377 is a member of the uranium-238 decay chain. Uranium and radium occur naturally in
378 soil and rocks and provide a continuous source of radon. Radon gas emanates from
379 the earth's crust and as a consequence is present in the air outdoors and in all
380 buildings, including workplaces. There is a large variation of indoor air
381 concentrations of this gas, depending mainly on the geology of the area and factors
382 that affect the pressure differential between the inside and outside of the building,
383 such as ventilation rates, heating within the building and meteorological conditions.
384 (2) Because radon is inert, nearly all of the gas that is inhaled is subsequently
385 exhaled. However, ^{222}Rn decays into a series of solid short-lived radioisotopes
386 which deposit within the respiratory tract. Because of their relatively short half-lives
387 (less than half an hour), the radon progeny decay mainly in the lung before clearance
388 can take place. Two of these short-lived progeny, polonium-218 and polonium-214,
389 emit alpha particles and it is the energy from these alpha particles that dominates
390 dose to the lung and the associated risk of lung cancer.
391 (3) The historical unit of exposure to radon progeny applied to the uranium
392 mining environment is the working level month (WLM) which is related to the
393 potential alpha energy concentration of its short-lived progeny. One WLM is defined
394 as the cumulative exposure from breathing an atmosphere at a concentration of 1
395 working level (WL) for a working month of 170 hours. A concentration of 1 WL is
396 any combination of the short-lived radon progeny in one litre of air that will result in
397 the emission of $1.3 \ 10^5$ MeV of alpha energy. One WLM is equivalent to $3.54 \ 10^{-3}$ J
398 h m^{-3} in SI units. Exposures can also be quantified in terms of the activity
399 concentration of the radon gas in Bq h m^{-3}. The two units are related via the
400 equilibrium factor, F, which is a measure of the degree of disequilibrium between
401 radon and its short-lived progeny (1 WLM = $6.37 \ 10^5$ / F Bq h m^{-3}; 1 J h m^{-3} = 1.8
402 10^8 / F Bq h m^{-3}). Thus, an annual domestic exposure of 227 Bq m^{-3} gives rise to
403 1 WLM, assuming occupancy of 7000 h y^{-1} and F value of 0.4.
404 (4) Radon has long been recognised as a cause of lung cancer and it was
405 identified as a human lung carcinogen in 1986 by the World Health Organisation
406 (WHO, 1986; IARC, 1988). The main source of information on risks of radon-
407 induced lung cancer has been epidemiological studies of underground miners (ICRP,
408 1993), and more recent studies have provided informative data on risks at lower
409 levels of exposure (e.g., Lubin et al., 1997b; NRC, 1998; EPA, 1999; 2003,
410 Tomášek et al., 2008a). In addition, recent combined analyses of data from case-
411 control studies of lung cancer and residential radon exposures have demonstrated
412 raised risks (Darby et al., 2005; 2006; Krewski et al., 2006; Lubin et al., 2004).
413 (5) A complication in the specification and control of doses and risks from
414 radon has been that doses can be calculated in two ways: the so-called
415 "epidemiological" approach and the "dosimetric" approach. Publication 65 (ICRP,
416 1993) recommended an epidemiological approach in which the risk of fatal lung
417 cancer per unit radon exposure (in J h m^{-3} or WLM) was compared with the total
418 risk, expressed as detriment, per unit effective dose (in Sv). Hence, values of mSv
419 (effective dose) per mJ h m^{-3} or WLM were obtained and referred to as the dose
420 conversion convention. Alternatively, various dosimetric models of the human
421 respiratory tract, including the ICRP (1994) model can be used to estimate

422 equivalent dose to the lungs and effective dose per unit radon and radon progeny
423 exposure. Given the uncertainties inherent in the estimation of risks from radiation
424 exposure, and in the calculation of doses using dosimetric models, it is not
425 surprising that the two approaches to calculating effective dose per unit radon
426 exposure have resulted in different values. In fact, the differences are remarkably
427 small. However, the use of different values by different organisations, notably by
428 ICRP (1993) and UNSCEAR (2000) suggests the need for clarification and the
429 formulation of a consistent approach. ICRP now intends to treat radon and its
430 progeny in the same way as other radionuclides and publish dose coefficients
431 calculated using models for use within the ICRP system of protection.

432   (6)   This report considers epidemiological data on radon risks published since
433 ICRP Publication 65 (ICRP, 1993) focusing on studies involving low levels of
434 protracted exposure. Results of pooled residential case-control studies are discussed
435 in Chapter 2 and results of recent miner epidemiological studies with low exposures
436 are discussed in Chapter 3. The miner data are used to recommend a revised estimate
437 of lung cancer lifetime risk per unit radon progeny exposure at low protracted levels
438 of exposure to radon and its progeny. Appendixes provide additional information on
439 epidemiological results obtained from miner studies (Appendix A) and review
440 published results of dose per unit exposure to radon progeny and thoron progeny,
441 calculated using dosimetric models of the human respiratory tract (Appendix B).

442

443 **2. EPIDEMIOLOGY OF LUNG CANCER RISK ASSOCIATED WITH**
444 **RESIDENTIAL EXPOSURES TO RADON AND RADON PROGENY**

445 **2.1. Introduction**

446 (7) In 1988, the International Agency for Research on Cancer (IARC, WHO)
447 classified radon as a human lung carcinogen, based on a review of evidence from
448 experimental data on animals and from epidemiological studies of underground
449 miners exposed to relatively high radon and radon progeny concentrations. This
450 report is focussed on those epidemiological studies able to provide information on
451 the dose-response relationship between lung cancer risk at relatively low annual
452 exposures to radon and radon progeny. Studies which include both individual
453 exposure assessment and individual assessment of potential confounding factors or
454 co-factors, such as tobacco use, are given special emphasis. Ecological studies of
455 cancer rates and average exposure per country or per region do not provide
456 individual exposure data and are not considered: they are unable to provide reliable
457 information on risk and are limited due to the unknown effect of confounding
458 factors, including smoking, and the unknown influence of population movement into
459 and out of the study areas (WHO, 1996; NRC 1998).
460 (8) The applicability of studies of underground miners to estimate
461 radon-induced lung cancer for residential concentrations of radon has been an
462 important uncertainty over the last twenty years. A variety of factors need to be
463 considered in this extrapolation from mines to homes, including the linearity of the
464 dose-response relationship, any differences between risks for adult males and the
465 general population which includes women and children, the difference in other
466 environmental exposures which may include arsenic, quartz and diesel exhaust
467 amongst others, different equilibrium factors between radon and its short-lived
468 progeny, and different breathing rates.
469 (9) Because of the desirability of having direct information on risks associated
470 with domestic radon concentrations, a large number of residential epidemiological
471 studies were launched in the late 1980s and early 1990s. There was also an
472 awareness that pooling of data may be required to provide the statistical power to
473 demonstrate a significant risk at residential exposure concentrations (Lubin et al.,
474 1997a). Reliable estimates of individual exposure conditions over long periods of
475 time were an important prerequisite of the epidemiological studies, with long-term
476 radon measurement in the current and previous homes of each individual.
477 Individuals' habits and ventilation conditions in dwellings had to be considered.

478 **2.2. Studies published since 1990**

479 (10) This section includes analytical epidemiological studies that have been
480 published since 1990 that have included at least 200 lung cancer cases, as well as
481 long-term domestic radon measurements. Selected studies are listed in Table 2.1;
482 more extensive details are available elsewhere (UNSCEAR, 2009).
483 (11) In most of the studies, year-long measurements of radon and its decay
484 products were made using standard methodologies in order to integrate any
485 variations in the specific conditions of the dwellings and any climatic and seasonal
486 changes. Most measurements are of concentrations in air using alpha track detectors.
487 In a few studies, glass-based retrospective detectors were also used.

488   (12) A number of European studies were designed with the intention of
489 conducting a pooled analysis (see Section 2.3). Considerable efforts were made to
490 have comparable protocols before starting studies in different countries. They were
491 all case-control studies, with face-to-face interviews whenever possible of both the
492 cases (lung cancer patients) and the controls (hospital controls or controls from the
493 general population). The same detailed questionnaire was used to analyse lung
494 cancer risk in relation to domestic radon exposure, adjusting for tobacco
495 consumption, occupational exposures, and indicators of socio-economic status.
496 These studies provide information on lung cancer risks from radon, for smokers and
497 non-smokers, and allow adjustment to be made relating to years as a smoker, the age
498 smoking began, years since stopping smoking, and the number of cigarettes per day.
499 Several large case-control studies were also conducted in Canada and in the USA, as
500 well as two studies in China, one in Shenyang and one in Gansu. Table 2.1
501 summarizes 20 studies published between 1990-2006.
502

503   Table 2.1. Residential radon cases-controls studies and one cohort study with more
504   than 200 lung cancer cases published between 1990 and 2006
505

Reference	Region	Population	No of Cases / Controls	Measurement. period	Relative risk per 100 Bq/m^3	95 % CI
Schoenberg et al., 1990	USA (New Jersey)	Women	480 cases, 442 controls	1 year	*1.49*	*0.89-1.89*
Blot et al., 1990	China-Shenyang	Women	308 cases, 356 controls	1 year	*0.95*	*undefined-1.08*
Pershagen et al., 1992	Sweden	Women	201 cases, 378 controls	1 year	*1.16*	*0.89-1.92*
Pershagen et al., 1994	Sweden	-	1281 cases, 2576 controls	3 months	*1.10*	*1.01-1.22*
Letourneau et al., 1994	Canada	-	738 cases, 738 controls	1 year	*0.98*	*0.87-1.27*
Alavanja et al., 1994	USA (Missouri)	Women, never-smokers	538 cases, 1183 controls	1 year	*1.08*	*0.95-1.24*
Auvinen et al., 1996	Finland	-	517 cases, 517 controls	1 year	*1.11*	*0.94-1.31*
Ruosteenoja et al., 1996	South Finland	Men	318 cases, 1500 controls	2 months	*1.80*	*0.90-3.50*
Darby et al., 1998	United Kingdom	-	982 cases, 3185 controls	6 months	*1.08*	*0.97-1.20*
Alavanja et al., 1999	USA (Missouri)	Women	477 cases, 516 controls	1 year	*1.27*	*0.88-1.53*
			387 cases, 473 controls		*1.3*	*1.07-2.93*
Field et al., 2000	USA (Iowa)	Women	413 cases, 614 controls	1 year	*1.24*	*0.95-1.92*
Kreienbrock et al., 2001	Germany (West)	-	1449 cases, 2297 controls	1 year	*0.97*	*0.82-1.14*
Lagarde et al., 2001	Sweden	Never-smokers	436 cases, 1649 controls	3 months	*1.10*	*0.96-1.38*
Wang et al., 2002	China-Gansu	-	768 cases, 1659 controls	1 year	*1.19*	*1.05-1.47*
Kreuzer et al., 2003	Germany (East)	-	1192 case, 1640 controls	1 year	*1.08*	*0.97-1.20*
Baysson et al., 2004	France	-	486 cases, 984 controls	6 months	*1.04*	*0.99-1.11*
Bochicchio et al., 2005	Italy	-	384 cases, 404 controls	6 + 6 months	*1.14*	*0.89-1.46*
Sandler et al., 2006	USA(Connecticut + (Utah-South Idaho)	-	1474 cases,1811 controls	1 year	*1,01*	*0.79-1.21*
Tomášek et al., 2001	Czech republic	-	173 cases in a cohort of 12 000 inhabitants	1 year	*1.10*	*1.04-.1.17*

506   CI: confidence interval;   - = men and women

507   (13) The studies listed in Table 2.1 evaluated the association between lung
508 cancer and domestic radon exposure. Results are presented in terms of the relative
509 risk per 100 Bq m^{-3} averaged for most studies over 20 to 30 years prior to lung
510 cancer diagnosis Two studies considered only never-smokers; most studies
511 considered males and females, smokers and non-smokers. Risks of radon exposure
512 are adjusted for smoking habits, and in several studies are also adjusted for
513 occupational exposures known to be potential lung carcinogens (e.g. exposure to
514 asbestos). Most of the twenty studies (17 out of 20 independent studies) reported a
515 positive trend in lung cancer risk with increasing exposure, but few of the trends
516 were statistically significant. A few studies were also consistent with the absence of
517 a positive trend. Each study considered alone had low statistical power and provided

518 an estimate of the risk per unit of exposure with a large confidence interval. Most of
519 the studies included only a small number of lung cancer cases that were never-
520 smokers and thus were limited in evaluating associations between radon decay
521 products and lung cancer in non-smoking populations.

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

2.3. Pooled studies

536 (15) Since 2000, several joint analyses were published, integrating the basic data
537 from individual case and control subjects and applying a standard methodology,
538 both in defining selection criteria and statistical analysis. It is noted that several
539 informative meta-analyses of radon studies have been conducted but did not have
540 the strengths of these pooled analyses which handle individual data in the same
541 manner (Lubin 1997a; NRC 1998; UNSCEAR 2009). Three joint analyses have
542 been conducted based on data from Europe (Darby et al., 2005), North America
543 (Krewski et al., 2005; 2006) and China (Lubin et al., 2004) (Table 2.2). Each joint
544 analysis showed evidence of lung cancer risk, increasing with cumulated domestic
545 exposure to radon. The exposure period considered was at least 30 years prior to
546 diagnosis for the North American and Chinese joint analysis and 35 years for the
547 European joint analysis. In each analysis the radon concentrations estimated for the
548 5 years prior to diagnosis were not considered since a minimum lag time of 5 years
549 was assumed from lung cancer induction to diagnosis based on data from studies of
550 underground miners (NRC 1998). In consequence the estimated risk per unit of
551 exposure is based on a time weighted average exposure for a window period 5 to 30
552 years prior to diagnosis (5 to 34 years for the European pooled analysis). The
553 estimates of the increase of lung cancer per unit exposure in the three joint analyses
554 are very close to each other and statistically compatible (Table 2.2): the values
555 obtained were 1.08, 1.10 and 1.13 per 100 Bq m^{-3} for Europe, America and China,
556 respectively. The combined estimate for Europe, North American and China was
557 1.09 per 100 Bq m^{-3} (UNSCEAR 2009).

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

563 (17) It is noteworthy that the slope of the linear exposure-response relationships
564 increased when analyses were restricted to those cases and controls with more
565 precise estimates of cumulated individual exposure, for example, if data were

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

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

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

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

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

Joint analysis	Number of studies included	Number of cases	Number of controls	Relative risk per 100 Bq m^{-3} (95% CI)
European *(Darby et al., 2006)*	13	7148	14208	1.08 (1.03-1.16)
North American	7	3662	4966	1.10 (0,99-1.26)

(Krewski et al., 2006)

Chinese *(Lubin et al., 2004)*	2	1050	1995	1.13	(1.01-1.36)

CI: confidence interval

(21) In conclusion, the joint analyses of lung cancer risk from residential radon exposures show an increase in risk of at least 8 % per 100 Bq m^{-3}, considering a period of exposure from 5 up to 30 - 35 years preceding the date of cancer diagnosis. Confining analysis to those with presumably more precise exposure measurements, the observed risk is increased in each of the joint analyses. The European pooling reported an excess relative risk increase of 16% per 100 Bq m^{-3} when uncertainties in the measured radon activity concentrations were considered. This value may be considered as a reasonable estimate of the risk associated with relatively low and prolonged radon exposures in homes, considering an exposure over a period of 25 - 30 years.

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

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

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

652
653

3. EPIDEMIOLOGY OF CANCER RISK ASSOCIATED TO RADON AND RADON PROGENY EXPOSURE IN UNDERGROUND MINES

654

3.1. Review of results since Publication 65

655 (25) ICRP Publication 65 (ICRP, 1993) estimated the risk of lung cancer death
656 from radon exposure on the basis of studies on seven cohorts of miners (Colorado
657 USA, Ontario Canada, New Mexico USA, Beaverlodge Canada, Western Bohemia
658 Czech Republic, CEA-COGEMA France and Malmberget Sweden) (Table A1 in
659 Appendix A). The total number of miners was 31,486. The weighted average of the
660 excess relative risk (ERR) per 100 WLM for these studies was 1.34 (95%CI = 0.82-
661 2.13). This ERR coefficient applied to a follow-up period of 20 years, taking into
662 account a lag-time (minimum latency) of 5 years, i.e., radon results for exposures
663 experienced 5 years prior to death from lung cancer (or comparable date for other
664 miners) are excluded from the analyses. A model was derived, taking account of the
665 modifying effects of age at exposure and time since exposure (ICRP, 1993).
666 (26) A comprehensive analysis of epidemiological results based on 11 cohorts of
667 radon-exposed miners was published in 1994 (Lubin et al., 1994). In comparison to
668 the ICRP Publication 65 report, results for some cohorts were updated (Colorado
669 USA, Ontario Canada, Beaverlodge Canada, Western Bohemia, Czech Republic and
670 Malmberget Sweden) and other cohorts were added (Yunnan China, Newfoundland
671 Canada, Port Radium Canada, and Radium Hill Australia). This analysis gave an
672 ERR per 100 WLM of 0.49 (95% CI = 0.2-1.0) (Lubin et al., 1994). After some
673 minor updates of the same 11 cohorts, a new joint analysis was published in the
674 BEIR VI report (NRC, 1998). This joint analysis relied on a total of 60,606 miners,
675 with a total of 2,674 lung cancer deaths (Table A2 in Appendix A). The estimated
676 combined ERR per 100 WLM was 0.59, assuming an exposure lag-time of 5 years.
677 Two models were derived, taking account of modifying effects of age at exposure
678 and time since exposure, as well as either duration of exposure or mean rate of
679 exposure. Analyses on restricted ranges of cumulative exposure of less than 100 or
680 50 WLM were also performed (NRC, 1998).
681 (27) Since the BEIR VI report (NRC, 1998), new results have been published
682 for the West-Bohemian cohort (uranium mines) and North Bohemian cohort (tin
683 mines) in the Czech Republic (Tomášek and Placek, 1999; Tomášek, 2002;
684 Tomášek et al., 2003; Tomášek and Zarska, 2004), the Newfoundland cohort
685 (fluorspar mines) (Villeneuve et al., 2007) and the Eldorado cohort (including
686 workers from Port Radium and Beaverlodge) (Howe, 2006) in Canada, the Colorado
687 Plateau cohort (Schubauer-Berigan et al., 2009) in the US, the Wismut uranium
688 mines in Germany (Kreuzer et al., 2002; Grosche et al., 2006; Kreuzer et al, 2008)
689 and the CEA-COGEMA mines in France (Rogel et al., 2002; Laurier et al., 2004;
690 Vacquier et al., 2008; Vacquier et al., 2009).
691 (28) The UNSCEAR 2006 (2009) report provided a comprehensive review of
692 available epidemiological results from nine studies (the New Mexico USA and
693 Australian studies were not included), including a total of more than 126 000 miners
694 (Table A3 in Appendix A). The weighted mean average ERR per 100 WLM was
695 0.59 (95%CI = 0.35-1.0) (UNSCEAR, 2009).
696 (29) Since the UNSCEAR 2006 (2009) report, the results of a joint analysis of
697 the Czech and French miner cohorts have been published. This analysis included
698 10,100 miners with a relatively long follow-up (mean, about 24 years) and relatively

699 low levels of cumulative exposure (mean, 46.8 WLM). The estimated ERR per 100
700 WLM was 1.6 (95%CI = 1.0-2.3) (Tirmarche et al., 2003; Tomášek et al., 2008a).
701 (30) Although other miner studies have been published, they are generally not
702 included here or in other comprehensive summaries since they provide little to no
703 quantitative information on the relationship between radon and cancer risk.

704 ## 3.2. Summary of estimates of Excess Relative Risk per WLM

705 The results from combined analyses summarised in Table 3.1 are presented as
706 simple linear estimates of the ERR per WLM. They apply across the whole
707 population of the cohorts under consideration but do not reflect variations of risk
708 between or within the cohorts. Some characteristics of the cohorts may explain
709 variations in the estimated ERR per WLM, including duration of follow-up, attained
710 age, duration of work, exposure levels and background rates of lung cancer. It is
711 important, therefore, to consider such factors in the assessment of the risk associated
712 with radon and radon progeny exposure. Nevertheless, the three large-scale analyses
713 that summarise most currently available information (Lubin et al., 1994; NRC, 1998;
714 UNSCEAR, 2009) provide estimates of the association between cumulated WLM
715 exposure and lung cancer risk that are highly concordant.
716
717 Table 3.1 Summary of Excess Relative Risk per WLM published from combined
718 analyses of miners studies
719

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

720 SE: standard error   CI: confidence interval
721
722
723 (31) All of the combined analyses and some of the individual studies
724 demonstrate a modifying effect of time since exposure (TSE) and, to a lesser extent,
725 of age at exposure (AE) (ICRP, 1993; Lubin et al., 1994; NRC, 1998; Howe, 2006;
726 Tomášek et al., 2008a). An inverse exposure-rate effect (or protraction enhancement
727 effect) has also been observed in most analyses (Lubin et al., 1994; NRC, 1998),
728 although such a modifying effect is not seen at low levels of cumulative WLM
729 exposure (Lubin et al., 1995; Tomášek et al., 2008a) or was no more evident using
730 improved individual dosimetric data (Howe, 2006; Vacquier et al., 2009). Models
731 have been developed to combine the modifying effects of TSE, age and exposure
732 rate. Two models were proposed in the BEIR VI report: the TSE-age-concentration
733 model and the TSE-age-duration model (NRC, 1998). These models provide risk
734 coefficients for different windows of cumulative exposure, with additional
735 modifying effect of age and concentration/duration based on categorical variables.
736 An alternative approach has been proposed in the joint analysis of the Czech and
737 French cohorts, modelling the risk associated with cumulative radon exposure and

738   integrating the modifying effects of TSE and AE as continuous variables (Tomášek
739   et al., 2008a).
740   (32) For current radiation protection purposes, the most relevant results from
741   miner studies are those derived for populations with low levels of cumulative
742   exposure, long duration of follow-up and good data quality. In general, the ERR per
743   100 WLM estimated from cohorts with low level of exposure (for example, the
744   Ontario, Beaverlodge and French cohorts) are higher than those estimated from
745   cohorts with high levels of cumulated exposures, although the confidence intervals
746   are broader (Table A3 in Appendix A). Some publications have provided estimates
747   based on analyses on restricted ranges of exposure (Lubin et al., 1997b). In the
748   BEIR VI report, such analyses resulted in estimated ERR per 100 WLM of 0.81 and
749   1.18, below 100 WLM and 50 WLM, respectively (NRC, 1998). In addition,
750   coefficients corresponding to low exposure rates can be obtained from models that
751   take account of modifying factors. In the BEIR VI report, an ERR per 100 WLM of
752   3.41 was obtained for low exposure rates < 0.5 WL (TSE-age-concentration model,
753   for an attained age of 55 - 64 years and at 15-24 years following exposure) (NRC,
754   1998). Recent analyses from the French and Czech cohorts have provided risk
755   estimates associated with low levels of exposure and reasonably good quality
756   exposure assessment ("measured exposures"), with values of ERR per 100 WLM
757   varying between 2.0 and 3.4 (Tomášek et al., 2008a; Vacquier et al., 2008). A
758   summary of these risk estimates is presented in Table 3.2, demonstrating significant
759   associations between cumulative radon exposure and lung cancer mortality at low
760   levels of cumulative exposure.
761
762   Table 3.2 Estimates of the ERR per WLM based on subgroups with low levels of
763   exposure and low exposure rate
764

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

765   CI: Confidence interval - WL: Working level - WLM Working Level Month
766   * for an attained age of 55 - 64 years at 15 – 24 years following exposure
767   ** restricted to miners with measured radon exposures

768 ### 3.3. Lung cancer risks from radon and smoking

769   (33) Although smoking is by far the strongest risk factor for lung cancer, most
770 studies of underground miners could not take account of smoking habits. Several
771 studies have partial smoking data, including the Chinese Yunnan cohort, the
772 Colorado Plateau cohort (United States), the Newfoundland cohort (Canada), the
773 Sweden cohort, the New Mexico cohort (United States) and the Radium Hill cohort
774 (South Australia). Case-controls studies among miners have also been conducted to
775 investigate the interaction between radon exposure and smoking on lung cancer risk
776 (Qiao et al., 1989; Lubin et al., 1990; L'Abbe et al., 1991; Thomas, 1994; Yao et al.,
777 1994; Brüske-Hohlfeld et al., 2006; Leuraud et al., 2007; Amabile et al., 2009).
778 More information on the lung cancer risk associated with both radon and cigarette
779 smoking should be available in the future as new datasets from cohort and case-
780 control studies are currently under development in Canada (Ontario cohort) and in
781 Europe (Czech, German and French cohorts) (Tirmarche et al., 2009).

782   (34) Considering currently available data (up to 2009), the results indicate that
783 the relationship between lung cancer mortality and radon exposure persists when
784 account is taken of smoking habits. The analyses conducted for the BEIRVI report
785 demonstrated a sub-multiplicative interaction between radon exposure and smoking
786 status (NRC, 1998). In a recent French nested case-control study, the ERR for lung
787 cancer related to cumulative radon exposure, adjusted for smoking, was 0.85 per 100
788 WLM (Leuraud et al., 2007), while the value obtained from the total French cohort,
789 when smoking information is ignored, was 0.82 per 100 WLM (Rogel et al., 2002;
790 Tirmarche et al., 2003). Tirmarche et al. (2003) concluded that presently available
791 models derived from cohort studies of underground miners that do not take account
792 of smoking status appear acceptable for the estimation of radon associated lung
793 cancer risks in a population including both smokers and non-smokers. When the
794 smoking status is known, the estimated ERR generally appears to be larger (even if
795 not significantly) among non-smokers than among smokers (Lubin et al., 1994;
796 Tomasek et al., 2002).
797

798

4. ASSESSMENT OF THE DETRIMENT FROM RADON AND RADON PROGENY EXPOSURE

4.1. Risks other than lung cancer

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

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

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

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

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

842   **4.2. Calculation of lung cancer lifetime risk estimates for underground miners**

843   (40) Most miner studies have demonstrated the existence of time modifying
844   factors of the relationship between cumulated radon exposure and lung cancer risk,
845   such as age at exposure or time since exposure. Due to variations in the
846   characteristics of the study populations (attained age, duration of follow-up), the
847   direct comparison of ERR estimates obtained from different cohorts may be
848   misleading. Account can be taken of such variations in the calculation of the lifetime
849   risk associated with a specific exposure scenario (Thomas et al., 1992). In outline,
850   calculation of lifetime risk requires:
851   1. Risk coefficients derived from an epidemiological study or studies, with or
852   without modifying factors such as attained age.
853   2. A projection model, enabling extrapolation of risk outside the range considered
854   by the epidemiological study (exposure range, sex, age) and transport to other
855   populations.
856   3. Background reference rates for all-cause and lung cancer mortality.
857   4. A scenario of exposure to radon concentrations.
858   (41) This approach was used in the ICRP Publication 65 to estimate the risk of
859   lung cancer associated with prolonged exposure to radon concentrations based upon
860   studies of underground miners (ICRP, 1993). Since that, several lifetime risk
861   estimates have been published (NRC, 1998; EPA, 2003; Tomášek et al., 2008a), but
862   cannot be easily compared due to differences in the nature of the estimates or in the
863   underlying assumptions. We focus here on estimates of the lifetime excess absolute
864   risk (LEAR) of lung cancer death from radon and radon progeny, as considered in
865   ICRP Publication 65, and we exclude those derived for background rates
866   corresponding to a specific country. We prioritized models derived from pooled
867   analyses rather than from single studies. The published estimates are summarized in
868   Table 4.1.
869
870   Table 4.1 Estimates of the lifetime excess absolute risk (LEAR) of lung cancer
871   associated with radon and radon progeny concentrations in underground mines (ICRP
872   Publication 65 scenario of a constant exposure to 2 WLM/y from age 18 to 64y)
873

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

874 ICRP 60 reference rates: averaged over males and females and over 5 countries
875 ICRP 103 reference rates: averaged over males and females and over asian and euro-american populations
876 * model relying on periods of work with the best quality of exposure assessment
877
878   (42) The scenario of exposure considered in estimating the LEAR shown in
879   Table 4.1 is as proposed in ICRP Publication 65 (ICRP, 1993): constant low level
880   exposure to 2 WLM per year during adulthood from 18 years to 64 years, with risk

881 estimated up to 90 or 94 years of age. Using the reference background rates of lung
882 cancer from Publication 60 (ICRP, 1991), Publication 65 (ICRP, 1993) adopted a
883 LEAR for lung cancer (also denoted as the nominal probability coefficient or fatality
884 probability) of 2.8 10^{-4} per WLM for radon exposure. Since detriment was entirely
885 due to lung cancer mortality, the Commission adopted a total detriment coefficient
886 equal to this fatality coefficient (ICRP, 1993).

887 (43) Applying the same risk coefficient as in Publication 65 (ICRP, 1993) to the
888 reference background rates found in Publication 103 (ICRP, 2007), Tomášek et al.
889 (2008b) calculated a LEAR of lung cancer of 2.7 10^{-4} per WLM. This comparison
890 shows that the modification of the reference population for background cancer rates
891 between Publication 60 and Publication 103 has only a small impact on the
892 estimated LEAR.

893 (44) Using the same scenario of exposure as in Publication 65 (ICRP, 1993) and
894 reference background rates from the Publication 103 (ICRP, 2007), Tomášek et al.
895 (2008b) also calculated the LEAR using the BEIR VI TSE-age-concentration model
896 (NRC, 1998). This model relies on the combined analysis of data from 11 cohorts of
897 miners, and takes into account modifying effects of attained age, TSE, and exposure
898 rate (note that the scenario corresponds only to the lowest category of exposure
899 rate). The LEAR estimate based on this model was 5.3 10^{-4} per WLM.

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

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

917 (47) Additional LEAR calculations were performed by the Task Group in order
918 to validate the published results, and to provide a sensitivity analysis of the different
919 underlying hypotheses using different models, scenarios and background rates.
920 Some calculations were performed independently by different experts to provide an
921 internal quality check; we especially thank Doug Chambers and Ladislav Tomášek.
922 Results confirmed the higher LEAR estimated using the BEIR VI model and the
923 Czech-French model. In addition to these models derived from pooled analyses,
924 other recent models obtained from single studies were also considered (French
925 CEA-AREVA cohort (Vacquier et al., 2008; Canadian Eldorado cohort (Howe,
926 2006), German Wismut cohort (Grosche et al., 2006)). It showed that the estimated
927 LEAR can vary from about 3 to 7 10^{-4} per WLM according to the model used. These
928 results illustrate the sensitivity of the estimate to the choice of the model, and
929 reinforced our preference for models derived from pooled analyses. Other
930 calculations also illustrated the sensitivity of LEAR estimates to background rates.
931 Using the rates for Euro-American males instead of the reference rates averaged

over males and females and over Euro-American and Asian populations (ICRP 2007), the estimated LEAR is about 7 10^{-4} per WLM. This difference is due to the higher background lung cancer rate among euro-american males. Conversely, using lower background lung cancer rates (such as females or non smokers) would lead to lower estimated LEAR per WLM.

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

4.3. Comparison of results from underground mine and domestic exposures

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

(50) Estimated primary risk coefficients are presented in Tables 2.1 and 2.2 for indoor studies and Tables 3.1 and 3.2 (and Appendix A) for miner studies. According to ICRP Publication 65, assuming an occupancy of 7000 h y^{-1} and an equilibrium factor (F) of 0.4, a concentration of 1 Bq m^{-3} radon gas leads to an exposure of $4.40.10^{-3}$ WLM indoor (ICRP, 1993). Most indoor case-control studies have estimated radon concentrations for periods of 30 or 35 years before diagnosis, with an exposure - lag time of five years. Therefore, considering a period of 30 years (ie. the last 35 years before diagnosis with a lag time of 5 years) and a time weighted averaged concentration of 100 Bq m^{-3}, the cumulated exposure of 2.1 10^{7} h Bq m^{-3} corresponds to a cumulated exposure of approximately 13 WLM assuming F-0.4. Using these values, an ERR per 100 Bq m^{-3} of 0.16 for indoor exposures (as obtained in the European pooling study with uncertainty correction; Darby et al., 2006) corresponds to an ERR of 1.2 per 100 WLM, which is similar to the value obtained in the BEIR VI analysis restricted to low levels of exposure below 50 WLM (NRC, 1998; see Table 3.2). This approach indicates a reasonably good agreement between the risk coefficients estimated for lung cancer mortality from indoor studies and miner studies at low levels of exposure. The same reasoning has been presented by several authors and led to the same conclusion (Zielinski et al., 2006; UNSCEAR, 2009; Tomášek et al., 2008a).

980   (51) The above approach does not consider the modifying effects of age and
981 time since exposure on the exposure risk relationship demonstrated by miner studies.
982 Lifetime estimates of lung cancer risk can account for these modifying factors and
983 provide another method for comparing underground miner study results with those
984 from indoor radon investigations. Nevertheless, due to differences in background
985 rates, duration of life considered and scenario of exposure, considerable caution is
986 needed in comparing published lifetime estimates obtained from miner studies
987 (ICRP, 1993; NRC, 1998; EPA, 2003; Tomášek et al., 2008a),and from indoor
988 studies (Darby et al., 2006).
989   (52) To allow comparison of estimated risks between miner studies and the
990 European indoor study, additional calculations were performed by the Task Group
991 using parameters chosen to respect as closely as possible the characteristics of the
992 available data. A specific scenario was elaborated in order to reflect the
993 characteristics of the subjects included in the European indoor study (attained age of
994 70 years corresponding to the average age at diagnosis, constant exposure to 100 Bq
995 m^{-3} over a time window of 5 to 30 years before diagnosis). To reflect the fact that
996 miner studies provide risk estimates for males, we used the ERR per 100 Bq m^{-3} of
997 0.25 obtained in the European pooling study for males only (Darby et al., 2006).
998 Using these parameters, the cumulated absolute risk up to 70 years of age estimated
999 for two pooled analyses of miner studies (BEIR VI and French-Czech) and for the
1000 European pooled analysis of indoor exposures were 3.5, 2.7 and 2.7 10^{-4} per WLM,
1001 respectively.
1002   (53) In conclusion, the currently available results show reasonably good
1003 consistency between lung cancer risk estimates obtained from miner and from
1004 indoor studies.
1005

5. CONCLUSIONS

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

- There is compelling evidence from cohort studies of underground miners and from case-control studies of residential radon exposures that radon and its progeny can cause lung cancer. For solid tumours other than lung cancer, and also for leukaemia, there is currently no convincing or consistent evidence of any excesses associated with radon and radon progeny exposures.
- The three pooled residential case-control studies (in Europe, North America and China) gave similar results and showed that the risk of lung cancer increases at least by 8% for an increase of 100 Bq m^{-3} in the radon concentration (Darby et al., 2005; Krewski et al., 2006; Lubin et al., 2004).
- After correcting for random uncertainties in the radon activity concentration measurements, the European pooled residential case control study gave an excess relative risk of 16% (5% to 32%) per 100 Bq m^{-3} increase (Darby et al., 2005). This value may be considered as a reasonable estimate for risk management purposes at relatively low and prolonged radon exposures in homes, considering that this risk is linked to an exposure period of at least 25 years.
- There is evidence from the European pooled residential case- control study that there is a risk of lung cancer even at levels of long-term average radon concentration below 200 Bq m^{-3} (Darby et al., 2005).
-
- The cumulative risk of lung cancer up to 75 year of age is estimated for lifelong non-smokers as 0.4%, 0.5% and 0.7% for radon activity concentrations of 0, 100 and 400 Bq m^{-3}, respectively. The lifetime cumulative risks of lung cancer by age 75 for lifelong smokers are close to 10%, 12% and 16% for radon activity concentrations of 0, 100 and 400 Bq m^{-3}, respectively (Darby et al., 2005; 2006). Cigarette smoking remains the most important cause of lung cancer.
- Appropriate comparisons of lung cancer risk estimates from miner studies and from indoor studies show good consistency.
- Based upon a review of epidemiological studies of underground miners, including studies with relatively low levels of exposure, a detriment adjusted nominal risk coefficient of 5 10^{-4} per WLM (0.14 per J h m^{-3}) is adopted for the lung detriment per unit radon exposure. This value of 5 10^{-4} WLM^{-1} (0.14 per J h m^{-3}) is derived from recent studies considering exposure during adulthood and is close to twice the value calculated in Publication 65 (ICRP, 1993).

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

(56) For occupational protection purposes, dose estimates are required to demonstrate compliance with limits and constraints. In addition to the review of

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

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1281
1282

1283   **APPENDIX A.**

1284   **Results from epidemiological studies of underground miners**

1285

1286   Table A1 Characteristics of the cohorts used in ICRP Publication 65 (ICRP, 1993)
1287

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

1288   ERR : excess relative risk   CI : confidence interval   * < 2000 WLM
1289
1290

1291
1292   Table A2 Characteristics of the cohorts considered in the BEIR VI report (NRC, 1998)
1293

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

1294   ERR : excess relative risk   se: multiplicative standard error   * among exposed
1295

1296   Table A3 Characteristics of the cohorts considered by UNSCEAR (2009)
1297

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

1298   ERR : excess relative risk   CI : confidence interval
1299

1300   **References for Appendix A**

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1309

1310   **APPENDIX B.   DOSIMETRY**

1311   **B.1.   Radon**

1312   (B 1)   The equivalent dose to the lung following the inhalation of radon and its
1313   short-lived progeny can be calculated using the ICRP Human Respiratory Tract
1314   Model (HRTM) (ICRP, 1994) and other models of the human respiratory tract.
1315   Nearly the entire lung dose arises from the inhalation of the radon progeny and not
1316   from the gas itself as almost all of the gas that is inhaled is subsequently exhaled.
1317   However, a large proportion of the inhaled radon progeny deposits in the respiratory
1318   airways of the lung.   Because of their short half-lives (less than half an hour), dose
1319   is delivered to the lung tissues before clearance can take place, either by absorption
1320   into blood or by particle transport to the alimentary tract.   Two of the short-lived
1321   radon progeny (^{218}Po, ^{214}Po) decay by alpha particle emission and it is the energy
1322   from these alpha particles that accounts for the relatively high dose to the lung. In
1323   comparison, doses to systemic organs and gastrointestinal tract regions are low and
1324   can be ignored in the calculation of effective dose.   The equivalent dose to the
1325   extrathoracic region is not small but its contribution to the effective dose is quite
1326   small.

1327

1328   (B 2)   The radon progeny aerosol in the atmosphere is created in two steps.
1329   After decay of the radon gas, the freshly formed radionuclides (^{218}Po, ^{214}Pb, ^{214}Bi)
1330   react rapidly (< 1 s) with trace gases and vapours and grow by cluster formation to
1331   form particles around 1 nm in size.   These are referred to as unattached particles.
1332   The unattached particles may also attach to existing aerosol particles in the
1333   atmosphere within 1 – 100 s forming the so-called attached particles.   The attached
1334   particles can have a trimodal activity size distribution which can be described by a
1335   sum of three lognormal distributions (Porstendörfer, 2001). These comprise the
1336   nucleation mode with an activity median aerodynamic diameter (AMAD) between
1337   10 nm and 100 nm, the accumulation mode with AMAD values 100 – 400 nm and a
1338   coarse mode with an AMAD > 1 μm. Generally, the greatest activity fraction is in
1339   the accumulation mode which has a geometric standard deviation of about 2.
1340   (B 3)   A dosimetric model for the respiratory tract needs to describe the
1341   morphometry, the deposition of the inhaled material, clearance from the respiratory
1342   tract and the location of target tissues and cells at risk.   For radon progeny, it is the
1343   dose to the target cells in the bronchial and bronchiolar regions of the lung that are
1344   of importance. In comparison, the dose to the alveolar region is significantly lower
1345   (UNSCEAR 1982, Marsh and Birchall, 2000).
1346   (B 4)   ICRP (1987) used values of dose per unit radon exposure based on an
1347   NEA (1983) review of available dosimetric models (Jacobi and Eisfed, 1980, 1982,
1348   James et al. 1982, Harley and Pasternack, 1982, Hofmann et al. 1980). UNSCEAR
1349   (1982; 1988; 1993) used similar estimates of dose from radon inhalation and the
1350   2000 report retained a value of effective dose of 5.7 mSv WLM^{-1} (1.6 mSv per
1351   mJ h m^{-3}, i.e. 9 nSv per Bq h m^{-3} of equilibrium equivalent concentration (ECC) of
1352   radon) for indoor and outdoor exposures (Table B.1). In the 2000 report, UNSCEAR
1353   recognised that more recent calculations with new dosimetric models resulted in
1354   higher values of this dose conversion factor. However, because of the lower values
1355   calculated using the dose conversion convention (ICRP, 1993), it was concluded that

1356 the previous value of 9 nSv per Bq h m^{-3} (ECC) should continue to be used in dose
1357 evaluations (UNSCEAR, 2000, 2009).
1358
1359
1360
1361
1362 Table B.1 Published values of effective dose to an adult male from the inhalation of
1363 radon and progeny calculated using dosimetric models
1364

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

1365 [a]A value of an absorbed dose of 6 mGy WLM^{-1} (1.7 mGy per mJ h m^{-3}) was
1366 calculated for the bronchial region. The effective dose per unit exposure was
1367 then calculated with a radiation weighting factor for alpha particles of 20 and a
1368 tissue weighting factor of 0.08 (= 2/3 ×0.12) for lung (ICRP, 1993).
1369 [b]Home without cigarette smoke.
1370 [c]No hygroscopic growth was assumed.
1371 [d]HRTM: Human Respiratory Tract Model (ICRP, 1994).
1372
1373
1374 (B 5) Table B.1 also shows values of effective dose per unit radon progeny
1375 exposure (mSv per WLM or mSv per mJ h m^{-3}) calculated using the ICRP
1376 Publication 66 Human Respiratory Tract Model (HRTM) (ICRP, 1994) and other
1377 models, including deterministic airway generation models (Winkler-Heil and
1378 Hofmann, 2002, Porstendörfer, 2001, Harley et al. 1996) and a stochastic airway
1379 generation model (Winkler-Heil et al. 2007). Results of selected recent calculations

1380 are given in Table B.1; more comprehensive tabulations of values published
1381 between 1956 and 1998 are given by UNSCEAR (2000).

1382 (B 6) The main sources of variability and uncertainty in the calculation of the
1383 equivalent dose to the lung per unit radon progeny exposure include:

1384 • the activity size distribution of the radon progeny aerosol,
1385 • breathing rates,
1386 • the model used to predict aerosol deposition in the respiratory tract,
1387 • the absorption of the radon progeny from lung to blood,
1388 • the identification of target cells and their location within bronchial and
1389   bronchiolar epithelium,
1390 • the relative sensitivity of different cell types to radiation,
1391 • the regional differences in the radiation sensitivity of the lung.

1392 Marsh and Birchall (2000) performed a sensitivity analysis to identify those HRTM
1393 parameters that significantly affect the equivalent dose to lung (H_{lung}) per unit
1394 exposure to radon progeny under conditions found in houses. Other sensitivity
1395 analyses have been reported (NCRP, 1984; NRC, 1991; Zock et al., 1996; Tokonami
1396 et al., 2003) and UNSCEAR (1988) noted that equivalent dose may vary by a factor
1397 of about 3 according to the target cells considered.

1398 (B 7) Winkler et al. (2007) compared the results of the effective dose for radon
1399 progeny inhalation obtained using the HRTM, a deterministic airway generation
1400 model and a stochastic airway generation model, with the same input parameter
1401 values. Similar results were obtained ranging from 8.3 to 11.8 mSv WLM^{-1} (2.3 to
1402 3.3 mSv per mJ h m^{-3}) (Table B.1). The authors noted that one of the important
1403 issues affecting the comparison is the averaging procedure for the doses calculated
1404 in airway generation models.

1405 (B 8) Porstendörfer (2001) calculated doses from radon progeny exposure for
1406 different exposure scenarios using an airway generation model developed by Zock et
1407 al. (1996). The effective dose calculated for 'normal' aerosol conditions in homes,
1408 workplaces and outdoors ranged from 8.0 to 11.5 mSv WLM^{-1} (2.3 to 3.3 mSv per
1409 mJ h m^{-3}) (Table B.1). However, in places with one dominating aerosol source
1410 producing a high number particle concentration (e.g. cigarette smoking or
1411 combustion aerosols by diesel engines) the effective dose was calculated to be
1412 lower, ranging from 4.2 to 7.1 mSv WLM^{-1} (1.2 to 2.0 mSv per mJ h m^{-3}). The
1413 activity size distributions and unattached fractions assumed for these calculations
1414 were based upon their measurements in indoor and outdoor air, and in the air at
1415 different workplaces in Germany.

1416 (B 9) James et al. (2004) calculated effective doses from radon progeny for
1417 mines and homes using the HRTM. The activity size distributions given in the
1418 BEIR VI report (NRC, 1998) were assumed. The authors calculated a range of
1419 values for mines (18 – 21 mSv WLM^{-1}; 5.1 – 5.9 mSv per mJ h m^{-3}) and homes
1420 (16 – 21 mSv WLM^{-1}; 4.5 – 5.9 mSv per mJ h m^{-3}) depending upon whether or not
1421 the attached particles double in size in the respiratory tract due to hygroscopic
1422 growth and depending upon the presence or absence of cigarette smoke in homes.
1423 These estimates are higher compared with other estimates (Table B.1), mainly
1424 because the activity size distribution assumed differed from those used by other
1425 investigators. Marsh et al. (2005), also using the HRTM and activity size
1426 distributions for mines and homes based upon measurements carried out in Europe,
1427 calculated values of about 13 mSv WLM^{-1} (3.7 mSv per mJ h m^{-3}) for mines and
1428 homes (Table B.1).

1429 (B 10) Calculations performed with the HRTM showed that the equivalent dose
1430 to lung per unit exposure is relatively insensitive to age (BEIR VI, 1999, Marsh and

1431 Birchall, 2000; Marsh et al., 2005, Kendall and Smith, 2005). For example the lung
1432 dose for an adult compared with that of children (> 1y) differs only by about 10%.
1433 The reason for this is that there are competing effects that tend to cancel out.
1434 Children have lower breathing rates which decreases the intakes and lung doses,
1435 while this is partly compensated by the smaller mass of target tissue which increases
1436 the doses. Also children have smaller airways which increase deposition by
1437 diffusion, but this is also compensated in part by smaller residence times that
1438 decrease deposition by diffusion.

1439 (B 11) The values of effective dose from radon progeny inhalation derived from
1440 the HRTM range from about 10 to 20 mSv WLM^{-1} (3 to 6 mSv per mJ h m^{-3})
1441 depending on the scenario of exposure. For typical aerosol conditions in home and
1442 mines the effective dose is about 13 mSv WLM^{-1} (3.7 mSv per mJ h m^{-3}) (Marsh et
1443 al., 2005). However, assuming the same aerosol conditions as for a home but with a
1444 breathing rate for a standard worker (1.2 m^{-3} h^{-1}) the effective dose increases from
1445 13 mSv WLM^{-1} (3.7 mSv per mJ h m^{-3}) to about 20 mSv WLM^{-1} (6 mSv per mJ h
1446 m^{-3}).

1447 (B 12) The Commission has concluded that doses from radon progeny should be
1448 calculated using ICRP biokinetic and dosimetric models, including the HRTM and
1449 the ICRP systemic models. In other words, radon progeny should be treated in the
1450 same way as any other radionuclide within the system of protection. One of the
1451 advantages of this approach is that doses to organs other than lung can also be
1452 calculated. ICRP will provide dose coefficients per unit exposure to radon progeny
1453 for different reference conditions of domestic and occupational exposure (i.e. of
1454 equilibrium factor and aerosol characteristics).

1455 ## B.2. Thoron

1456 (B 13) Thoron (^{220}Rn) gas is a decay product of radium-224 and is part of the
1457 thorium-232 decay series. Thoron (^{220}Rn) has a short half-live (56 s) and decays
1458 into a series of solid short-lived radioisotopes, including lead-212 which has a
1459 half-life of 10.6 h. Because of the short half-life of thoron, it is less able than radon
1460 (^{222}Rn) to escape from the point where it is formed. As a consequence, building
1461 materials are the most usual source of indoor thoron exposure.

1462 (B 14) As for radon, doses from the inhalation of thoron and progeny are
1463 dominated by alpha particle emissions from decay of the progeny (Jacobi and
1464 Eisfed, 1980, 1982). Because of its very short half-life, the gas activity
1465 concentration of thoron can vary substantially across a room and so it is not possible
1466 to use thoron gas concentration in dose evaluation. Therefore, for control purposes,
1467 the potential alpha energy concentration of the thoron progeny should be determined
1468 for the estimation of thoron exposure. However, it is usually sufficient to control
1469 the intake of ^{212}Pb for protection purposes because the potential alpha energy (PAE)
1470 per unit activity inhaled is about 10 times higher for ^{212}Pb than for other thoron
1471 progeny (ICRP, 1987).

1472 (B 15) UNSCEAR (2000) and the BEIR VI committee (NAS, 1999) presented
1473 data for the ratio of potential alpha energy concentration (PAEC) arising from
1474 thoron (^{220}Rn) progeny to that from radon (^{222}Rn) progeny. The values ranged from
1475 0.1 to 5. The highest values were for wood-frame and mud houses found in Japan
1476 and for some houses in Italy that used building materials of volcanic origin.
1477 UNSCEAR also noted that in the UK, a value as high as 30 was observed for a
1478 house with a high ventilation rate and an unusually low radon concentration

1479 (UNSCEAR, 2000, Cliff et al. 1992). The BEIR VI committee concluded that for
1480 dwellings with high radon (^{222}Rn) concentrations, it appears that the thoron (^{220}Rn)
1481 progeny will not be an important additional source of exposure and dose (NRC,
1482 1999).
1483 (B 16) A summary of dose coefficients for thoron progeny, calculated using
1484 dosimetric models, is given in Table B.2. Values range from 1.5 – 5.7 mSv WLM^{-1}
1485 i.e. 0.42 - 1.6 mSv per mJ h m^{-3} or 10 – 122 nSv per Bq h m^{-3} (EEC).
1486 (B 17) The dose coefficient given in ICRP Publication 50 (ICRP, 1987) is based
1487 on the work of an Expert Group of OEC/NEA (NEA, 1983), which reviewed the
1488 models of Jacobi and Eisfed (1980; 1982) and of James et al. (1980; 1982). Only
1489 doses to the bronchial epithelium and pulmonary tissue were considered.
1490 (B 18) In its 1982 report, UNSCEAR not only considered the doses to the lung
1491 based upon the work of Jacobi and Eisfed (1980) but also considered doses to other
1492 tissues by applying the dosimetric models given in ICRP Publication 30 (ICRP,
1493 1979). Values of 1.9 mSv WLM^{-1} (0.54 mSv per mJ h m^{-3}) and 2.5 mSv WLM^{-1}
1494 (0.71 mSv per mJ h m^{-3}) were recommended for indoor and outdoor exposures,
1495 respectively. The effective dose coefficients for thoron progeny given in the 1988
1496 UNSCEAR report were based upon the calculations of Jacobi and Eisfed (1982) and
1497 corresponded to an effective dose per unit PAE of 0.7 mSv mJ^{-1}. These values were
1498 retained in the 1993 report (UNSCEAR, 1993) and are given in Table B.2.
1499 UNSCEAR (2000, 2009) has since adopted a value 40 nSv per h Bq m^{-3} (ECC) (i.e.
1500 1.9 mSv per WLM or 0.54 mSv per mJ h m^{-3}) for indoors and outdoor exposures,
1501 which is similar to the value given in ICRP Publication 50 (ICRP, 1987).
1502
1503 Table B.2 A summary of the calculated dose conversion factors[a] for thoron progeny using
1504 direct dosimetry.
1505

Publication	Model Type	Exposure scenario	Effective dose[b] mSv WLM^{-1}	Effective dose, mSv per mJ h m^{-3}
ICRP 50, 1987	NEA (1983)	Indoors and outdoors	1.8	0.51
UNSCEAR, 1993	Jacobi and Eisfed, 1982	Indoors	1.5	0.42
		Outdoors	0.47	0.13
Marsh and Birchall, 1999	HRTM, (ICRP, 1994)	Dwellings	3.8	1.1
Porstendörfer, 2001	Zock et al. (1996)	Indoors	2.4	0.68
		Outdoors	2.0	0.56
Ishikawa et al., 2007	HRTM, (ICRP, 1994)	Indoors	5.4	1.5
Kendall and Phipps, 2007	HRTM, (ICRP, 1994)	Indoors	5.7	1.6

1506 (a) Calculated for an adult male
1507 (b) 1 WLM = 4.68 10^4 Bq h m^{-3} of EEC of ^{220}Rn.
1508
1509
1510 (B 19) The values of the dose coefficients obtained using the HRTM (Marsh and
1511 Birchall, 1999; Ishikawa et al., 2007, Kendall and Phipps, 2007) are higher than the
1512 values recommended by ICRP (1987) and UNSCEAR (1993). Kendall and Phipps
1513 (2007) calculated the effective dose conversion factor for thoron progeny with the
1514 HRTM and the most recent biokinetic models for lead (ICRP, 1993) and bismuth
1515 (ICRP 1979). The authors showed that typically the dose to the lung contributed

1516 more than 97% of the effective dose and that the intake from ^{212}Pb alone contributes
1517 to about 85% of the total dose. Calculations for different age groups (> 1 y) showed
1518 that the dose per unit exposure differed by 10% or less (Kendall and Phipps, 2007).
1519   (B 20) Following the decision to treat radon isotopes in the same way as other
1520 radionuclides for protection purposes, biokinetic and dosimetric models will be used
1521 to provide dose coefficients for radon-220 as well as radon-222.

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