

PREFACE

At its 1990 meeting, the Commission established a Task Group of Committee 4 to prepare a report on protection against radon in buildings. In 1991, it set up a Working Party to prepare a report on limits for the radiation exposure of workers in mines.

The members of the Commission Working Party were W. Jacobi (Chairman) and H. J. Dunster. Members of the Committee 4 Task Group were R. V. Osborne (Chairman), J. H. Harley✱, A. C. James, M. C. O'Riordan, A. G. Scott, G. A. Swedjemark and P. Zettwoog.

Both reports were submitted to the Commission for discussion at its meeting in November 1992. It was then decided to combine the two studies into a single report on protection against radon in dwellings and workplaces, including mines. This combined report was prepared by a Commission Task Group with the following membership:

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A draft of the report was issued by the Commission for consultation in April 1993. A revised text was approved for publication in September 1993.

1. INTRODUCTION

(1) The naturally radioactive noble gas radon (^{222}Rn) is present in the air outdoors and in all buildings, including workplaces. It is thus an inescapable source of radiation exposure both at home and at work. High radon levels in air can occur in buildings, including workplaces, in some geographical locations. This applies particularly in workplaces such as underground mines, natural caves, tunnels, medical treatment areas in spas, and water supply facilities where ground water with a high radon concentration is treated or stored.

(2) This report summarises the extent of current knowledge about the health effects of inhaled radon and its progeny and makes recommendations for the control of this exposure in both dwellings and workplaces. It aims to give guidance to national advisory and regulatory agencies and to practitioners of radiological protection concerned with radon in dwellings and workplaces.

1.1. The Structure of the Report

(3) In its 1990 recommendations, *ICRP Publication 60* (ICRP, 1991), the Commission deals separately with practices and intervention and with occupational and public exposure. Exposures to radon have implications in all these situations. Radon is present in all buildings. In existing dwellings, the exposures can be reduced only by some form of intervention. In workplaces, it is necessary to consider both the need for intervention (as in dwellings) and the continued control of radon exposures as part of the practice carried out in the workplace. The future exposure in new buildings also has to be considered. This report, which is intended to deal coherently with all these issues, has the following structure.

(4) The rest of Section 1 provides introductory material about radon and the quantities and units used in specifying radon concentrations and exposures, followed by a summary of the main principles of protection applicable to radon. Throughout the report, the term "radon" is used most often to include its short-lived progeny, not necessarily in equilibrium. The term "radon concentration," however, relates to the concentration of the parent nuclide alone.

(5) Section 2 deals with the current information about the health effects of exposure to radon. It provides estimates of both the fatality and detriment coefficients for lifetime exposure to radon progeny of workers and the general public. These coefficients are then used to give a direct conversion, based on equal detriment, between radon exposure and effective dose.

(6) Section 3 deals briefly with radon in buildings, indicating the practical approaches to reducing the concentrations of radon and its progeny. Section 4 deals with the policy for limiting radon in dwellings, leading up to recommendations for action levels of radon in dwellings, above which remedial measures (intervention) should be taken. The requirements for new buildings are also discussed.

(7) Section 5 deals with radon in workplaces. Radon occurs in all workplaces and action may be needed to reduce existing concentrations. Guidance is given on the concentration of radon above which remedial measures (intervention) to reduce radon

concentrations should be taken. The Commission recommended in *ICRP Publication 60* that exposure to radon at work should be excluded from its system of protection for practices unless the relevant regulatory agency has ruled otherwise. Section 5 gives guidance on the level of exposure to radon that should be used in making that ruling.

(8) Once the decision to apply the Commission's system of protection is made, it becomes necessary to apply an exposure limit. This has been derived to correspond to the same level of detriment as that resulting from an effective dose equal to the Commission's recommended dose limit. Some additional guidance is given on practical control measures in workplaces.

1.2. The History of Radon

(9) The existence of a high mortality rate among miners in central Europe was recognised before 1600, and the main cause of death was identified as lung cancer in the late nineteenth century (Haerting and Hesse, 1879). It was suggested that the cancers could be attributed to radon exposure in 1924 (Ludewig and Lorensen, 1924).

(10) Early environmental measurements were largely confined to outdoor air for the study of diverse phenomena such as atmospheric electricity, atmospheric transport and exhalation of gases from soil. The first indoor measurements were made in the 1950s (Hultqvist, 1956), but attracted little attention. In recent years, there has been an upsurge in the interest in radon in dwellings and workplaces.

(11) A more comprehensive review of the history of radon is given as a separate publication in this issue of the *Annals of the ICRP*.

1.3. Radon and its Progeny

(12) The two significant isotopes of radon are radon-222, the immediate decay product of radium-226, deriving from the uranium series of natural radionuclides, and radon-220, the immediate decay product of radium-224, deriving from the thorium series. Because of their origins, the two isotopes are commonly known as radon and thoron. The element is a noble gas and both isotopes decay to isotopes of solid elements, the atoms of which attach themselves to the condensation nuclei and dust particles present in air. The problems posed by radon-220 (thoron) are much less widespread, and generally more tractable, than those posed by radon-222. For protection against thoron, it is usually sufficient to control the intake of the decay product, lead-212, which has a half-life of 10.6 hours. This report is concerned with protection against radon-222. The main decay properties of the short lived progeny are shown in Table 1. Radon-222 decays by alpha emission to polonium-218 with a half-life of 3.82 days. Polonium-214 decays to lead-210 which has a half-life of 23.3 years and which eventually decays to stable lead-206.

(13) The biological processes linking the inhalation of radon and its progeny to the generation of an increased risk of lung cancer are complex. The special quantities that have been developed for use with radon have proved useful in practice in the provision of simple relationships between exposure and risk. However, their quantitative significance for this purpose may be modified by physical factors not included in the definition of the quantities themselves, such as the unattached fraction (see Annex C).

Table 1. Decay properties of radon-222 and short lived progeny

Radionuclide	Half-life	Main radiation energies and yields (y)					
		Alpha		Beta		Gamma	
		Energy (MeV)	y (%)	Energy (max) (MeV)	y (%)	Energy (MeV)	y (%)
²²² Rn	3.824 days	5.49	100	—	—	—	—
²¹⁸ Po	3.05 min	6.00	100	—	—	—	—
²¹⁴ Pb	26.8 min	—	—	1.02	6	0.35	37
				0.70	42	0.30	19
				0.65	48	0.24	8
²¹⁴ Bi	19.9 min	—	—	3.27	18	0.61	46
				1.54	18	1.77	16
				1.51	18	1.12	15
²¹⁴ Po	164 μ s	7.69	100	—	—	—	—

Sources: Browne and Firestone (1986) and ICRP (1983).

1.4. Special Quantities and Units

(14) This section sets out the special quantities and units that are used to characterise the concentration of the short-lived progeny of radon in air, and the resulting inhalation exposure.

Potential alpha energy

(15) The potential alpha energy, ϵ_p , of an atom in the decay chain of radon is the total alpha energy emitted during the decay of this atom to stable ²¹⁰Pb. The potential alpha energy per unit of activity (Bq) of the considered radionuclide is $\epsilon_p/\lambda_r = (\epsilon_p t_r / \ln 2)$ where λ_r is the decay constant and t_r the radioactive half-life of this nuclide. Values of ϵ_p and ϵ_p/λ_r are listed in Table 2.

Table 2. Potential alpha energy per atom and per unit activity

Radionuclide	Half-life	Potential alpha energy			
		Per atom		Per unit of activity	
		(MeV)	(10 ⁻¹² J)	(MeV Bq ⁻¹)	(10 ⁻¹⁰ J Bq ⁻¹)
Radon (²²² Rn) progeny:					
²¹⁸ Po	3.05 min	13.69	2.19	3615	5.79
²¹⁴ Pb	26.8 min	7.69	1.23	17 840	28.6
²¹⁴ Bi	19.9 min	7.69	1.23	13 250	21.2
²¹⁴ Po	164 μs	7.69	1.23	2 × 10 ⁻³	3 × 10 ⁻⁶
Total (at equilibrium), per Bq of radon				34 710	55.6

Concentration in air

(16) The potential alpha energy concentration, c_p , of any mixture of short-lived radon progeny in air is the sum of the potential alpha energy of these atoms present per

unit volume of air. Thus, if c_i is the activity concentration of a decay product nuclide i , the potential alpha energy concentration of the progeny mixture is

$$c_p = \sum_i c_i (\epsilon_{p,i} / \lambda_{r,i})$$

This quantity is expressed in the SI unit J m^{-3} ($1 \text{ J m}^{-3} = 6.242 \times 10^{12} \text{ MeV m}^{-3}$).

(17) The potential alpha energy concentration of any mixture of radon progeny in air can be also expressed in terms of the so-called equilibrium equivalent concentration, c_{eq} , of their parent nuclide, radon. The equilibrium equivalent concentration, corresponding to a non-equilibrium mixture of radon progeny in air, is the activity concentration of radon in radioactive equilibrium with its short-lived progeny that has the same potential alpha energy concentration, c_p , as the actual non-equilibrium mixture. The SI unit of the equilibrium equivalent concentration is Bq m^{-3} .

(18) The equilibrium factor, F , is defined as the ratio of the equilibrium equivalent concentration to the activity concentration of the parent nuclide, radon, in air. This factor characterises the disequilibrium between the mixture of the short-lived progeny and their parent nuclide in air in terms of potential alpha energy.

Inhalation exposure of individuals

(19) The quantity "exposure," P , of an individual to radon progeny is defined as the time integral of the potential alpha energy concentration in air, c_p , or the corresponding equilibrium equivalent concentration, c_{eq} , of radon to which the individual is exposed over a given period T , e.g. one year.

$$\begin{aligned} \text{Potential } \alpha \text{ energy exposure} \quad P_p(T) &= \int_0^T c_p(t) dt \\ \text{Equilibrium equivalent exposure} \quad P_{eq}(T) &= \int_0^T c_{eq}(t) dt \end{aligned}$$

(20) The unit of the exposure quantity P_p is J h m^{-3} ; for the exposure quantity P_{eq} the unit is Bq h m^{-3} . The potential alpha energy exposure, P_p , of workers is often expressed in the historical unit Working Level Month (WLM). 1 WL was originally defined as the concentration of potential alpha energy associated with the radon progeny in equilibrium with 100 pCi l^{-1} (3700 Bq l^{-1}). This concentration was about $1.3 \times 10^5 \text{ MeV l}^{-1}$, but the precise value depended on the estimates of alpha energy per disintegration. The Working Level is now defined as a concentration of potential alpha energy of $1.300 \times 10^8 \text{ MeV m}^{-3}$. Since the quantity was introduced for specifying occupational exposure, 1 month was taken to be 170 hours. Since $1 \text{ MeV} = 1.602 \times 10^{-13} \text{ J}$, the relationship between the historical and the SI units is as follows:

$$\begin{aligned} 1 \text{ WLM} &= 3.54 \text{ mJ h m}^{-3} \\ 1 \text{ mJ h m}^{-3} &= 0.282 \text{ WLM} \end{aligned}$$

(21) Here, and elsewhere in this report, values that will be used in later calculations may be given to more significant figures than are usually needed and sometimes to more than the precision of the data justifies. Whenever rounded values are given for quantities that will be used in subsequent calculations, the unrounded values are retained for use in these calculations. Most values are given in SI units. However, the

historical units are still widely used and converted values are also given where it is likely that this will be helpful.

(22) The conversion coefficients between the concentration quantities, potential alpha energy, C_p , and equilibrium equivalent concentration, C_{eq} ; and between the exposure quantities, potential alpha energy exposure, P_p , and equilibrium equivalent exposure, P_{eq} , are given in Table 3.

Table 3. Conversion coefficients for the different concentration quantities and for the corresponding exposure quantities for radon-222

Quotient	Conversion coefficients
C_p/C_{eq}	5.56×10^{-9} (J m ⁻³) per (Bq m ⁻³)
C_{eq}/C_p	1.80×10^8 (Bq m ⁻³) per (J m ⁻³)
P_p/P_{eq}	5.56×10^{-9} (J h m ⁻³) per (Bq h m ⁻³) 1.57×10^{-6} WLM per (Bq h m ⁻³)
P_{eq}/P_p	1.80×10^8 (Bq h m ⁻³) per (J h m ⁻³) 6.37×10^5 (Bq h m ⁻³) per WLM

Quantities: C_p —concentration of potential alpha energy, C_{eq} —equilibrium equivalent concentration of radon, P_p —time-integrated exposure to potential alpha energy concentration, P_{eq} —time-integrated exposure to equilibrium concentration of radon.

(23) The relationship between the annual exposure and the radon concentration at home or at work can be obtained from Table 3. For most purposes, it is adequate to use an equilibrium factor of 0.4 and an occupancy of 2000 hours per year at work or 7000 hours indoors (UNSCEAR, 1988). On this basis, a continued exposure to a radon concentration of 1 Bq m⁻³ results in an annual exposure at home of 1.56×10^{-2} mJ h m⁻³ (4.40×10^{-3} WLM). The corresponding figure at work is 4.45×10^{-3} mJ h m⁻³ (1.26×10^{-3} WLM).

1.5. The Principles of Protection

(24) In *ICRP Publication 60*, attention is drawn to the need for protection against natural sources of radiation both in dwellings and workplaces. Key extracts from the recommendations as they relate to radon are presented here.

(25) The Commission distinguishes between two circumstances of exposure to radiation, one where human activities introduce new sources or modes of exposure and thus increase the overall exposure and the other where they decrease the exposure to existing sources. The first it calls practices and the second intervention. It also identifies the circumstances under which exposure to radon at work may need to be subject to the Commission's system of protection for practices and where the need for action against exposure to radon in homes should be considered. Radon occurs in all buildings and the concentrations vary widely from building to building. In the workplace, there is sometimes a difficulty in making a sharp distinction between radon concentrations that should be treated as being due to a practice or as due to an existing situation for which

intervention may be needed. One of the aims of this report is to give guidance on that distinction.

(26) The system of radiological protection recommended by the Commission for proposed and continuing practices is based on the following general principles. Here, and throughout the document, direct quotations and paragraph references from *ICRP Publication 60* are italicised. This extract is from *Paragraph 112*.

- “(a) No practice involving exposures to radiation should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes. (The justification of a practice.)”*
- “(b) In relation to any particular source within a practice the magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received should all be kept as low as reasonably achievable, economic and social factors being taken into account. This procedure should be constrained by restrictions on the doses to individuals (dose constraints), or the risks to individuals in the case of potential exposures (risk constraints), so as to limit the inequity likely to result from the inherent economic and social judgements. (The optimisation of protection.)”*
- “(c) The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits, or to some control of risk in the case of potential exposures. These are aimed at ensuring that no individual is exposed to radiation risks that are judged to be unacceptable from these practices in any normal circumstances. Not all sources are susceptible of control by action at the source and it is necessary to specify the sources to be included as relevant before selecting a dose limit. (Individual dose and risk limits.)”*

(27) For intervention, the Commission recommends that two general principles be followed. These general principles are set out in *Paragraph 113* of which the relevant part reads:

- “(a) The proposed intervention should do more good than harm, i.e. the reduction in detriment resulting from the reduction in dose should be sufficient to justify the harm and the costs, including social costs, of the intervention.”*
- “(b) The form, scale, and duration of the intervention should be chosen so that the net benefit of the reduction of dose, i.e. the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximised.”*

(28) The Commission qualifies this advice in *Paragraph 131*, of which the relevant part reads:

“The dose limits recommended by the Commission are intended for use in the control of practices. The use of these dose limits, or of any other pre-determined dose limits, as the basis for deciding on intervention might involve measures that would be out of all proportion to the benefit obtained and would then conflict with the principle of justification. The Commission therefore recommends against the application of dose limits for deciding on the need for, or scope of, intervention. Nevertheless, at some level of dose, approaching that which would cause serious deterministic effects, some kind of intervention will become almost mandatory.”

More detailed accounts of the Commission's policy are given in Sections 4 and 5.

2. THE HEALTH EFFECTS OF INHALED RADON AND ITS PROGENY

(29) Estimates of the consequences to health of exposures to ionising radiation are best based on epidemiological studies of human populations. In the context of radiation, epidemiology is concerned with the establishment of statistical associations between exposures and health effects. These studies have established beyond any reasonable doubt that radiation is a causative agent of cancer in many organs and tissues of the body, including the lung. The establishment of quantitative association is more difficult.

(30) It is a vital tenet of statistics that events that are correlated in time or space are not necessarily correlated in cause. Indeed, chance associations have a definite likelihood of occurrence. To establish a quantitative causal relationship it is necessary to supplement the epidemiological data by the use of models based on biological evidence. When a range of such models is proposed, it is legitimate to use epidemiology to indicate statistical preferences between them. Epidemiology may also suggest improvements to the proposed models or further possible models. However, it is not legitimate to create or modify models solely to improve the statistical fit of the data in a single epidemiological study. There must also be confirmatory findings in other studies and plausible biological support.

(31) Epidemiological studies have shown a correlation between exposure to radiation and excess lung cancer. These include the Life Span Study of the survivors of the atomic bombs at Hiroshima and Nagasaki, patients treated for ankylosing spondylitis, cancer of the cervix, Hodgkins disease and breast cancer, and miners exposed to radon at work. The two main sources of quantitative information about the risks resulting from the exposure of the lungs to radiation are the Life Span Study and the studies of miners. The Life Span Study provides estimates of the cancer fatality coefficient for exposure, principally to gamma radiation, that is fairly uniform over the whole lung. The studies on miners provide information on the relationship between the incidence of fatal lung cancer and the concentration of radon progeny in the mining environment.

(32) In the last ten years or so, there have also been many studies aimed at detecting a correlation between the incidence of lung cancer and exposure to radon in dwellings. Some of these have shown positive correlations, but many have not. Reviews of these studies has been made by Samet (1989) and Stidley and Samet (1993). Most of these studies have been geographical correlation studies. These involve selecting two or more areas, some of high and others of low, average concentration of radon in dwellings. The current lung cancer incidences are examined and a statistical comparison made.

(33) Unfortunately, geographical correlation studies are difficult to interpret, even qualitatively, because of the presence of several serious confounding factors. One possible confounding factor is a correlation of radon concentrations with other environmental features. Areas of high radon are often in rocky and hilly regions rather than in the river valleys and alluvial plains where populations and industrial developments are likely to be concentrated. There may thus be an inverse correlation of radon concentration and industrialisation. If, as is likely, there is a direct correlation between lung cancer and industrialisation, probably associated with smoking, this may mask, or appear to reverse, any link between lung cancer and radon.

(34) Even if an allowance can be made for confounding factors, it remains difficult to draw quantitative conclusions, because many of those who die in an area have not consistently lived in that area. The concentrations observed are then not typical of the

exposures of individuals. These difficulties can largely be avoided by the use of cohort and case-control studies. Several of these are currently (1993) in hand.

(35) When the problem of confounding factors is recognised, case-control studies of radon (e.g. Schoenberg *et al.*, 1990) in dwellings are not inconsistent with the mining studies, but, as yet, most of them provide no quantitative data. However, some quantitative data, albeit statistically weak, are provided by two case-control studies from Sweden (Pershagen *et al.*, 1992, 1993). For the present, the Commission continues to rely mainly on the data from epidemiological studies on miners, because of the lack of statistical power in the studies on dwellings.

(36) There are several sources of uncertainty in the radon epidemiology. These include the statistical limitations imposed by the size of the exposed populations, the need to select a projection model to estimate lifetime risks and the need to postulate an exposure-response relationship to provide estimates of risk at levels of exposure below those for which there are directly observable excess risks. In addition to the statistical uncertainties, there are several sources of non-random uncertainty in these studies:

- (a) the uncertainty of individual exposure estimates;
- (b) the difficulty of selecting an appropriate control group;
- (c) the different working atmospheres in the mines, including the influence of other non-radioactive ore dusts;
- (d) the different smoking habits; and
- (e) the differences (by a factor of about two) in the mean follow-up periods.

Furthermore, the quantity inhaled potential alpha exposure may not be the most appropriate quantity, because of variation in physical parameters such as the particle size distribution of the inhaled aerosols. However, this is the quantity in which all the epidemiological data for miners are expressed.

(37) The Commission has adopted an improved dosimetric model of the respiratory tract for use in a very wide range of circumstances (ICRP, 1994). The practical applications of this model are still being developed. The use of this model for assessing the fatality and detriment coefficients for inhaled radioactive materials is complicated by uncertainties in several important areas. The deposition and retention aspects of the model lead on to dosimetric stages involving the geometrical relationship between the deposited material and the cells at risk. It is then necessary to assess the relative importance of the dose to cells in different parts of the tract. The present estimates of the probability that these doses will result in cancer depend on the estimation of risk coefficients for lung cancer caused by uniform, high dose-rate exposure to low LET radiation obtained from the Life Span Study. Statistical limitations prevent the direct observation of the excess relative risk at low doses. The use of these data for estimating the risk from radon exposures in homes and workplaces therefore depends on the choice of the dose and dose rate effectiveness factor for the induction of lung cancer by low LET radiation and of the radiation weighting factor for alpha radiation.

(38) Although there are uncertainties in both above approaches, they do not lead to widely different results. The Commission has concluded that the use of the epidemiology of radon in mines is more direct, and therefore involves less uncertainty and is more appropriate for the purposes of this report than the indirect use of the epidemiology of low LET radiation from the Japanese data. The Commission therefore recommends that the dosimetric model should not be used for the assessment and control of radon exposures. The fatality coefficients in this report are therefore based

on the epidemiological studies on miners exposed to radon. Since these results relate essentially to adult males, it is necessary to make further judgements to predict the risks to females and children from the observed risks to males. See Sections 2.2.1 and 2.2.3.

2.1. Lung Cancer in Radon-Exposed Miners

(39) There have been several epidemiology studies of lung cancer in miners exposed to radon. These are continuing and the results are combined and reviewed from time to time, both by individuals and groups (e.g. U.S. National Research Council, NRC). Several studies and reviews are in preparation or in press at the date of preparation of this report. As an indication of the methodology of such studies, and to give a general indication of typical results, the Commission has conducted a limited review, summarised in Annex A of this report. In this developing situation, the Commission has not made its own definitive analysis.

2.1.1. Epidemiological studies

(40) The epidemiological evidence for the induction of lung cancer following inhalation of radon comes from several cohort and case-control studies of underground miners, particularly uranium miners. These findings have been summarised and reviewed in other reports (UNSCEAR, 1986, 1988; NRC, 1988; IARC, 1988; ICRP, 1991). For the quantitative risk analysis, the following studies of uranium miners cohorts are of special importance: Bohemia (Sevc *et al.*, 1988, 1993), Colorado, USA (Whittemore and McMillan, 1983; Hornung and Meinhardt, 1987), New Mexico, USA (Samet *et al.*, 1989, 1991), Ontario, Canada (Muller *et al.*, 1985, 1989), Saskatchewan (Beaverlodge), Canada (Howe *et al.*, 1986; SENES, 1991; Chambers *et al.*, 1992), France (Tirmarche *et al.*, 1992a) and Port Radium, Canada (Howe *et al.*, 1987). An excess rate of lung cancer has also been observed in iron miners in Malmberget, Sweden (Radford and Renard, 1984), fluorspar miners in Newfoundland, Canada (Morrison *et al.*, 1988), workers in a tin mine in Yunnan, China (Lubin *et al.*, 1990, Xiang-Zhen *et al.*, 1993) and gold miners in Ontario (Kusiak *et al.*, 1991).

(41) Many of these studies are consistent with a proportional (linear, non-threshold) relationship between excess risk and cumulative exposure. Some, however, show evidence of a higher excess relative risk per unit exposure at low exposures compared with the mean value for the whole exposed group (Darby and Doll, 1992). Studies on rats, reviewed by the US Department of Energy (DOE, 1988), support a non-threshold, linear, exposure-risk relationship at low levels of exposure. There are several possible explanations of this discrepancy. The expression of the exposure in terms of potential alpha energy concentration may conceal the effect of other factors, such as particle size distribution, ventilation rate, and the unattached fraction. The exposure-risk relationship might also be distorted by the presence of other carcinogens, such as arsenic.

(42) When the results of several studies have been amalgamated, it has usually been on the basis of the estimated excess relative risk per unit exposure. This implies the use of a causative relative risk model in which the excess risk results from a multiplication of the age-specific baseline risk (including any enhancement from smoking). However, any enhancement of the baseline risk caused by earlier parts of the occupational exposure is ignored. If it were included, the model would show the excess risk rising

more rapidly than linearly at higher levels of exposure. Such a rise has not apparently been observed. If there is a true causative relative risk model, it seems to be more complex than is usually assumed.

2.2. Lung Cancer Risk Estimates for Chronic Exposure

(43) The epidemiological findings have to be extended to provide information for long periods of exposure, for lifetime risks, and for other populations than those studied. For estimating lifetime risk from data covering shorter periods, the Commission has used a multiplicative projection model rather than an additive one (ICRP, 1991). It warned, however, that there was no adequate basis for choosing between a relative and an absolute risk model for transferring estimates from one population to another. In this report, the estimates of absolute excess risks are assumed to apply to a wide range of populations. There is, as yet, no *a priori* basis for selecting a model for transferring the risk estimate for males to the risk estimate for females. The choice is complicated by the interaction of the effects on the lung of radiation and of smoking. This issue is discussed in Section 2.2.1.

(44) The general policy of the Commission towards protection makes use of the attributable lifetime risk and detriment from stochastic radiation effects. It is therefore necessary to estimate the lifetime absolute probability of attributable death starting from the data over the more limited follow-up periods provided by the epidemiology studies.

2.2.1. Risk projection models for lung cancer

(45) Different types of risk projection models, some with modifications for factors such as time since exposure, have been proposed to estimate the possible lifetime risk of lung cancer from inhaled radon progeny from the results of the epidemiological studies with limited follow-up periods (Harley *et al.*, 1981; NCRP, 1984a,b; ICRP, 1987; NRC, 1988; Jacobi, 1992).

(46) At present, multiplicative projection models, which assume a correlation with the age dependency of the normal baseline rate of lung cancer, are considered to be more representative of the time distribution of the excess risk. Assuming a proportional exposure-risk relationship, these relative risk models proceed from the age specific mortality rate for lung cancer to the age specific excess rate resulting from chronic exposure starting at 18 years of age. The integration considers a time lag (minimum latency) between exposure and the expression of lung cancer from inhaled radon progeny.

(47) In *ICRP Publication 60*, the Commission used a projection model with a constant multiplier (relative risk factor) for most cancers and low-LET radiation. However, the epidemiological findings from radon-exposed miners now yield convincing evidence that the excess relative risk factor for lung cancer varies strongly with time since exposure and with attained age. This follows from the analysis of the data from the uranium miners in the USA and Canada in the BEIR IV study (NRC, 1988) and from the data of the uranium miners in Bohemia (Sevc *et al.*, 1988, 1993). On the basis of these findings, modified multiplicative risk projection models have been developed (NRC, 1988; Jacobi *et al.*, 1992). They are compared in Annex A.

(48) With respect to smoking, some studies on lung cancer in radon-exposed miners suggest qualitatively a synergistic or multiplicative effect, whereas some do not. Some

quantitative information comes from a large case-control study among the Colorado uranium miners. This study yields a somewhat less than multiplicative effect of smoking and rejects an additive model (Whittemore and McMillan, 1983; Hornung and Meinhardt, 1987). It should be noted, however, that these miners were exposed to very high radon levels. Furthermore, it has been reported that, with increasing follow-up, this relationship is moving towards an additive model (Jacobi, 1991). A similar result is reported in the New Mexico studies (Samet *et al.*, 1989). The latter tendency might be related to the different latency distributions of small-cell and squamous cell carcinoma, which are two most common types of carcinoma included in the generic term lung cancer. In short, the epidemiological evidence from miners does not yet provide a firm quantitative conclusion on the influence of smoking (IARC, 1988).

(49) In this context, it should be mentioned that the Life Span Study of the atomic bomb survivors yields for females an excess relative risk of lung cancer per unit equivalent dose to the lung that is 3 to 4 times higher than that for males (Shimizu *et al.*, 1988). The absolute excess risk per unit dose was much the same in males and females. It has been demonstrated that this sex difference in relative risk diminished after adjustment had been made assuming additivity of the effect of smoking and external radiation exposure (Kopecky *et al.*, 1986).

(50) It is biologically plausible that the absolute risk coefficient should be about the same for men and women of similar habits, including smoking. In the absence of a clear indication to the contrary, the Commission has now decided to use, for protection purposes, the same absolute lifetime risk per unit exposure to radon progeny for both males and females. In so doing it is recognised that the risk factor may be over-cautious for females. The use of the same relative risk would have predicted a lower absolute fatality coefficient for females, probably related to a lower level of smoking.

2.2.2. *Lifetime risk from chronic occupational exposure*

(51) As in *ICRP Publication 60*, the Commission has adopted nominal probability coefficients for chronic exposure of workers (ages 18 to 65 years). Since the epidemiology is all related to the exposure to concentrations of potential alpha activity, rather than to intake, the coefficients relate to exposure. They can be converted to nominal coefficients for intake using a standard breathing rate of $1.2 \text{ m}^3 \text{ h}^{-1}$. The published estimates of risk are similar to those in the BEIR IV report (NRC, 1988). This report gave a lifetime fatality coefficient of 3.5×10^{-4} per WLM for a U.S. population, 9.99×10^{-5} per (mJ h m^{-3}) . The Commission's reference population has baseline values of survival probability, and of the age-specific lung cancer mortality rate, corresponding to the reference data for the "average population" and defined as the unweighted average of the values listed by Land and Sinclair (1991) for the populations of Japan, the United States, Puerto Rico, the United Kingdom and China. This population has a somewhat lower baseline cancer mortality. On this basis, the Commission has adopted a nominal probability coefficient (fatality) for males and females of 8.0×10^{-5} per (mJ h m^{-3}) . The corresponding value in historical units is 2.83×10^{-4} per WLM, which has been rounded to 3×10^{-4} per WLM.

2.2.3. *Lifetime risk from chronic exposure of the public*

(52) The fatality probability coefficient for the general public might be somewhat larger than that for miners because of the inclusion of children. However, the effect of any high relative risk in the period soon after exposure of children would be offset by

the decreasing excess relative risk with time. For the mortality coefficient for cancer in general, the Commission has used fatality coefficients of 5×10^{-2} per Sv for the public and 4×10^{-2} per Sv for workers—a factor of 1.25 (ICRP, 1991). However, for exposure to radon, the Commission knows of no reason to adopt a lifetime risk coefficient for children different from that for adults. Many other factors may influence the difference in coefficient for occupational and public exposure to radon progeny. They include dust loading, particle size, the degree of attachment of radon progeny to condensation nuclei and dust particles, and the properties of the respiratory tract as a function of age. To adjust the risk coefficient, it is necessary to consider all these factors.

(53) On balance, variations in the values of the physical and biological parameters suggest a lower dose (and therefore risk) per unit exposure in buildings than in mines. Several authorities have made adjustments for this difference, either implicitly or explicitly. The results are expressed as the factor by which the risk coefficient for exposure in mines should be changed to give the coefficient for exposure in buildings. Calculations by NEA (1983) yield a factor of 0.65, by NCRP (1984b) a value of 1.4 in the case of adult males, and by Harley (1984) a range from 0.8 to 1.2. ICRP (1987) adopted a value of 0.8, NRC (1988) adopted a default value of unity, but later calculated a range from 0.6 to 0.9 (NRC, 1991).

(54) Despite the importance of the unattached fraction, these adjustments are all close to unity. Taking this into account, and accepting the implicit degree of approximation, the Commission has concluded that, for protection purposes, there is insufficient justification for adopting a nominal probability coefficient (fatality) for the public different from that for workers, i.e. 8×10^{-5} per (mJ h m⁻³).

2.2.4. *Detriment coefficients*

(55) In order to establish a consistent policy for exposure to radon and to other radiation sources, it is necessary to take account of the factors that convert mortality into detriment. In *ICRP Publication 60* (ICRP, 1991), the Commission took account of non-fatal cancer, hereditary effects and the length of life lost or impaired. The principal detriment due to the inhalation of radon and its progeny is that associated with the fatal lung cancer. There is a slight addition due to curable lung cancer and a slight reduction due to a smaller length of life lost than for the average of all cancers. From the values in Table B-20 of *ICRP Publication 60*, the detriment coefficient for lung cancer is 0.95 times the fatality coefficient. There will also be some detriment resulting from the exposure of tissues outside the lung as the result of radon transferred to these tissues by the blood, and of radon progeny inhaled. The information in Annex B shows that these will result in an increase in detriment of about 2%. In view of these various factors, the Commission has concluded that the selection of a detriment coefficient different from the fatality coefficient for radon exposure is not justified.

2.2.5. *The conversion from exposure to effective dose*

(56) Because most workers exposed to radon will also be exposed to other sources of radiation, it is helpful to provide a conversion from radon exposure to effective dose. Since the Commission has not used a dosimetric approach for radon, this conversion has been obtained by a direct comparison of the detriment associated with a unit effective dose and a unit radon exposure. The detriment per unit effective dose is 5.6×10^{-5} per mSv for workers and 7.3×10^{-5} per mSv for the general public (ICRP, 1991). The detriment per unit exposure to radon progeny is 8.0×10^{-5} per (mJ h m⁻³)

for workers and the same for members of the public. In terms of detriment, an exposure to radon progeny of 1 mJ h m^{-3} is equivalent to an effective dose of 1.43 mSv for workers or 1.10 mSv for members of the public. The corresponding figures for 1 WLM are 5.06 mSv for workers and 3.88 mSv for members of the public. This difference is entirely due to the different detriment coefficients for effective dose in *ICRP Publication 60*. The conversions obtained in this way are called conversion conventions. They are based on an equality of detriment, not on dosimetry. Rounded values are given in Table 7.

3. RADON IN BUILDINGS

(57) A building above ground, especially if it is made of traditional earthen materials quarried locally and has a basement, may be considered as a transition between the lithosphere and the atmosphere. If all the doors and windows are open, indoor air will not be very different from the outdoor air: if the openings are all tightly closed, the indoor radon concentration will be appreciably higher than that outdoors.

(58) Underground workings, such as those involved in tunnelling, are not strictly buildings and the options for reducing the concentration of radon and its progeny are somewhat different from those available in buildings. A description of the mining environment is given in *ICRP Publication 47* (ICRP, 1986) and is relevant to other underground workings.

3.1. Radon Concentrations in Buildings

(59) For both dwellings and workplaces, the distributions of radon concentrations are approximately lognormal, with some tendency for high concentrations to lie above those predicted by the lognormal distribution. The geometric mean (GM) and geometric standard deviation (GSD) describe the distribution. The arithmetic mean (AM) is used to estimate the average probability of detrimental health effects. Comprehensive data on indoor radon concentrations are compiled by the United Nations Scientific Committee on the Effects of Atomic Radiation. The Committee concluded (UNSCEAR, 1988) that the worldwide, population-weighted, values of these parameters for dwellings are $\text{AM} = 40 \text{ Bq m}^{-3}$, $\text{GM} = 25 \text{ Bq m}^{-3}$ and $\text{GSD} = 2.5$. It also adopted a typical value of 0.4 for the equilibrium factor.

(60) Radon concentrations in dwellings differ between countries because of differences in geology and climate, in construction materials and techniques, and in domestic customs. National values mask marked regional variations in radon concentrations. Elevated regional values ranging up to several times the central UNSCEAR values occur fairly widely and values of several thousands of Bq m^{-3} have been found in thousands of houses in Finland (Castrén, 1987) and Sweden (Socialstyrelsen, 1988). Systematic investigations of above-ground workplaces are still rare, with the principal exception of public buildings such as schools and nurseries.

3.2. Building Occupancy

(61) To calculate the radon exposure from measured concentrations, a value of the occupancy factor is needed. UNSCEAR (1988) uses 0.80 indoors and 0.20 outdoors for worldwide calculations. In northern countries, the indoor occupancy factor seems

to be higher according to studies in the UK (Brown, 1983) and Sweden (Mjönes, 1986; Westrell, 1984). On average, more than 90% of time in the UK is spent indoors, 75% being spent in dwellings; differences between summer and winter are small. The Swedish studies show that about 85% to 90% of time is spent indoors, 65% being spent in dwellings: this changes to 60% when the holiday period is included. Some 5% to 10% of time is spent outdoors, and the same percentage is used for travelling. The occupancy factor for women who remain at home in France is said to be 90% (Roy and Courta, 1991). A rounded occupancy factor of 0.8 is adopted here, corresponding to 7000 hours per year. For workplaces, it is customary to assume an occupancy of 2000 hours per year. These values are reasonable reference values but do not necessarily reflect the conditions in any particular building. This uncertainty reinforces the need to use rounded, but fairly representative, occupancy values.

3.3. The Value of Identifying Radon-Prone Areas

(62) In the Commission's view, there is merit in defining radon-prone areas in which the concentration of radon in buildings is likely to be higher than is typical of the country as a whole. This allows attention to be focused on radon where it is most exigent and action to be concentrated where it is most likely to be effective. Any definition of radon-prone areas will have to be in fairly general terms, so it must be remembered that some locations with high radon concentrations may occur outside radon-prone areas. One way of selecting areas to be treated as "radon-prone" is to use the results of surveys in dwellings and to define a radon-prone area in terms of a selected proportion of dwellings with concentrations above some selected value. The choice of these figures is discussed in Section 4.2.2.

(63) Whereas knowledge of the geology and the type of soil is important in identifying likely radon-prone areas, especially in the first phase of a radon programme, the most reliable way to delineate radon-prone areas is by measuring the radon concentrations in a representative sample of existing dwellings. The radiological information, which also reflects the nature and use of the dwellings, may then be used to improve the use of the geological information in identifying other radon-prone areas. Correlations with superficial and bed-rock geology, soil radon and permeability may be used to adjust or explain the boundaries of the areas (Miles *et al.*, 1992). In some regions, the correlations may be strong enough for geological criteria to be applied directly (Åkerblom *et al.*, 1990; Clavensjö and Åkerblom, 1992), but what succeeds in some cases may not succeed in all.

3.4. Remedial and Preventive Measures

(64) The principal methods for reducing high radon concentrations indoors are as follows:

- (a) To reduce the radon supply by reversing the pressure differential between the building and the soil, often called soil depressurisation. This is most easily achieved by using a small fan to withdraw the radon from the region under the floor, either in a porous area under (or close to) the dwelling or in the space under a suspended floor.

- (b) To reduce the radon supply by raising the resistance of the foundations to soil gas entry or by treating building materials to reduce radon escape. This process of sealing is difficult to make effective in existing buildings because there are many routes of entry for radon from the ground.
- (c) To remove the radon source, which is likely to be feasible only for the water supply and in, extreme cases, solid materials such as the underlying soil.
- (d) To dilute the radon and its progeny by increasing the ventilation rate. The effectiveness of this process is limited because the ventilation rate in most buildings is already as high as the occupants want, and further ventilation will increase heating or cooling costs. Some forms of ventilation will decrease the pressure in the building, thus increasing the radon input.
- (e) To reduce the concentration of radon progeny, e.g. by filtration or by increased movement of indoor air to enhance the deposition of radon progeny.

Some of these remedial measures, e.g. (a) and (d), depend on a continued expenditure if they are to be effective. Local circumstances and the material giving rise to the radon will influence the choice of methods.

(65) Table 4 shows a qualitative summary of the costs and effectiveness of the various options for radon remedial work. The cost and effectiveness of the methods are likely to vary locally and national authorities are best placed to adapt their policies to their particular circumstances.

Table 4. Guide to the cost and effectiveness of various remedial measures for buildings

Method	Cost	Effectiveness ^a
Soil depressurisation	Moderate	High
Floor sealing	Moderate	Moderate
Water treatment	Moderate	High
Subsoil removal	High	High
Increased ventilation	Moderate	Low
Increased air movement	Low	Low

^a The effectiveness is judged in terms of the effect on the part of the concentration of radon progeny to which the remedial measure applies.

4. THE APPROACH TO PROTECTION IN DWELLINGS

4.1. Policy Issues

(66) Radon in dwellings is singled out for special attention by the Commission in *ICRP Publication 60* because of the magnitude of individual and collective doses (*Paragraphs 216–218*). The Commission has dealt with radon in dwellings only in the context of intervention. It has not treated the occupancy of dwellings as a practice. It envisages that intervention would involve “*modifications to the dwellings or to the behaviour of the occupants*”. Behaviour is taken here to mean the manner in which occupants use a dwelling.

(67) The Commission goes on to recommend the use of action levels for initiating intervention:

"... to help in deciding when to require or advise remedial action in existing dwellings. The choice of an action level is complex, depending not only on the level of exposure, but also on the likely scale of action, which has economic implications for the community and for individuals". Whereas owner-occupiers may be left to decide whether to take action, firm national action levels may be required."

In a crucial passage, the Commission recommends that

"the best choice of an action level may well be that level which defines a significant, but not unmanageable, number of houses in need of remedial work. It is then not to be expected that the same action level will be appropriate in all countries."

(68) By dealing with radon in dwellings in this way, the Commission has emphasised intervention to protect the more highly exposed individual members of the population. In this report, it has not dealt with the wider public health implications of the exposures to the whole population. Any action affecting the whole housing stock of a country would be extremely costly, although it might still be cost-effective in terms of the reduction in the national collective dose. It is for national authorities to decide whether the necessary funds would be available and best spent on general radon reduction or other aspects of housing improvement.

(69) Following the policy set out in Section 1.5, consideration is given in this section to the circumstances under which the principles of protection against natural sources of radiation might be applied to radon in buildings and to the practical procedures for doing so. The section deals primarily with dwellings, but many of the issues are equally relevant to buildings used as workplaces. The special problems of workplaces are dealt with in Section 5.

(70) It is clear that elevated levels of radon do occur in some dwellings, that it is possible to identify the conditions under which they arise, that remedial and preventive measures are usually simple and of moderate cost, and that there are appreciable risks attendant on elevated exposures. Intervention is therefore feasible. The main matter is the determination of the action level at which intervention should be undertaken.

4.2. Practical Protection in Dwellings

4.2.1. Action level for intervention in dwellings

(71) It is now appropriate to examine the basis for adopting an action level for intervention in dwellings. Here, and throughout the report, action levels relate to the annual mean concentration of radon in a building. It is important that the action taken should be intended to produce substantial reduction in radon exposures. It is not sufficient to adopt marginal improvements aimed only at reducing the radon concentrations to a value just below the action level. Once intervention is decided, the degree of the intervention should be optimised.

(72) It seems clear that some remedial measures against radon in dwellings are almost always justified above a continued annual effective dose of 10 mSv. For simple remedial measures, a somewhat lower figure could be considered, but a reduction by a factor of five or ten would reduce the action level to a value below the dose from natural background sources. The choice of action level for annual effective dose is thus

limited to the range of about 3–10 mSv. The Commission recommends that the action level should be set within this range by the appropriate authorities.

(73) The corresponding rounded value of radon concentration is about 200–600 Bq m⁻³, with an annual occupancy of 7000 hours and an equilibrium factor of 0.4. Continuous domestic exposures at average concentrations of 200 Bq m⁻³ and 600 Bq m⁻³ would imply annual exposures as in Table 5.

Table 5. Annual exposures for action levels of 200 (Bq m⁻³) and 600 (Bq m⁻³) in dwellings

Action level (effective dose)	3 (mSv y ⁻¹)	10 (mSv y ⁻¹)
Action level (radon concentration)	200 (Bq m ⁻³)	600 (Bq m ⁻³)
Annual exposure to radon gas	1.4 (MBq h m ⁻³)	4.2 (MBq h m ⁻³)
Annual exposure to progeny	3.11 (mJ h m ⁻³)	9.33 (mJ h m ⁻³)
	0.88 WLM	2.63 WLM

4.2.2. Implementation of action levels

(74) It is for national authorities to decide whether to make action levels mandatory or advisory. Much will depend on the view taken of the social and legal circumstances. If there is a high proportion of rented dwellings together with a legal system based on statutes, there may be a willingness to oblige landlords to comply with the action level. In common-law jurisdictions with a preponderance of owner-occupiers, compulsion may be deemed undesirable. In either case, it is of considerable importance to ensure that occupiers, both tenants and owners, are fully aware of the risks of radon and the remedial options. Because of the uncertainty inherent in any measurement of indoor radon level, it is also important to allow some flexibility in cases marginally above or below the action level. It must also be remembered that the risk estimates relate to a mixed population of smokers and non-smokers. Unless the effect of smoking is purely additive, the action level will be over-cautious in relation to the risks to non-smokers. The conventional conversion from radon exposure to effective dose will also over-estimate the risks to non-smokers. In a few situations, the readily available counter-measures may not be sufficient to bring the radon concentrations in a dwelling down to the action level. It must then be remembered that the action level recommended by the Commission relates only to the simple measures discussed in this section. More severe measures, such as relocation, would not be appropriate unless the irreducible concentrations were an order of magnitude or more higher than the action levels adopted.

(75) Although exposure to radon is unlikely to be an acute threat to health, it will be wise not to delay remedial action unduly once an elevated level has been found. National authorities recognise the importance of this point and have developed various protocols. In Sweden, for example, householders are advised to take simple precautions temporarily, such as increasing the ventilation, until a permanent remedy can be effected (Socialstyrelsen, 1990). There has been a tendency to relate rapidity of action to the level of radon. National authorities should be aware that such schemes may result in procrastination, or even inaction, on the part of some occupiers of dwellings where the radon concentrations are not markedly above the action level.

(76) Attention was drawn in Section 3.3 to the concept of radon-prone areas. A radon-prone area might be defined as one in which about 1% of dwellings had a radon concentration of more than ten times the national average value. In any particular case,

both the country-wide distribution and the choice of action level will influence the definition. General quantitative advice can be no more than indicative. The spatial clustering of buildings requiring action because of geological circumstances is advantageous: it facilitates the establishment of programmes of measurement and intervention. Furthermore, it helps to order priorities in a national scheme. In setting priorities, it is prudent to take action more urgently in areas of high radon concentration while not necessarily basing the urgency of action on the concentrations found in each individual house.

4.2.3. *Application to new dwellings*

(77) The emphasis in the preceding sections has been on existing dwellings, but the approach adopted is also relevant to future dwellings. Differences in the action levels set by some authorities for the two circumstances are not large and not all authorities have regarded the differentiation between old and new dwellings as helpful, partly because such refinement may prove difficult to explain and partly because the figure for new dwellings cannot be applied rigorously until the dwelling is completed and occupied.

(78) The aims in imposing restrictions on the construction of new dwellings in radon-prone areas are to keep the radon concentrations in the finished buildings as low as can reasonably be achieved and to provide for the easy introduction of further remedial measures if the initial construction fails to achieve concentrations below the action level for existing buildings. These aims are best achieved by issuing guidance on construction practices. Particularly careful consideration should be given to developments on made-up ground if there are indications that radium-bearing wastes have been dumped there. A thorough quantitative assessment will be needed in this circumstance, possibly supported by measurements in a temporary structure on the proposed site.

(79) When new buildings are to be erected in a radon-prone area, it will be advisable to modify the design of the foundations so as to prevent elevated radon levels. There are two types of modified foundation, those that readily permit later remedial measures and those that are resistant to radon, or, more correctly, to soil-gas. In some circumstances, elevated radon concentrations could be caused by the use of ground fill or building materials with elevated radium-226 content. As such materials can be readily detected by the gamma-ray emission, consideration should be given to identifying them and preventing or limiting their use.

(80) The radon-resistant approach requires bigger changes in foundation design and construction to prevent soil gas from entering the building by passive means. It then has no further costs. The simpler solution is the ready-remedy approach, in which a low-resistance fill layer with a low radium content is provided under the floor slab so that the radon may be extracted. Space may also be left for an interior exhaust duct for the extracted air.

(81) Either approach will reduce radon exposure. The approach favoured by national authorities will depend on local building styles, the extent and severity of radon-proneness, and the regulatory regime. In the initial phase of a national radon programme, the authorities will need to monitor closely the outcome of preventive and remedial procedures to ensure that they are reliable and durable. The most effective option may prove to be a combination of the two approaches.

5. THE APPROACH TO PROTECTION IN WORKPLACES

(82) Radon is present in all workplaces. In some, such as uranium mines, it is a recognised source of exposure and is already subject to control. In others, such as buildings and non-uranium mines, it is widely ignored. As indicated in Section 1.5, there is some difficulty in distinguishing between radon concentrations that should be treated as being due to a practice and those that should be regarded as being due to an existing situation. The Commission now recommends the use of action levels to clarify the basis for this choice.

(83) In the first place, an action level is needed to define workplaces, including mines, in which intervention should be undertaken to reduce radon exposures. Secondly, it is necessary to define the workplaces in which the Commission's system of protection for practices should be applied to radon exposures, with other workplace not being subject to this system. This definition can also be expressed as an action level.

5.1. The Selection of Relevant Workplaces

(84) It is likely that elevated levels of radon will occur in buildings used as workplaces in radon-prone areas defined for dwellings. However, such areas may have been defined only for residential areas. When defining radon-prone areas, national authorities ought also to take into account non-residential areas.

(85) It would be advisable for regulatory or supervisory agencies to ensure that a systematic survey is conducted in places of work in radon-prone areas. It would also be prudent to make additional measurements in a representative sample of workplaces throughout the country to ensure that no geographical area of importance is being overlooked. If reliance is being placed on measurements in dwellings to define the areas of concern for workplaces, care must be taken to ensure that any systematic differences in the two types of building are taken into account. There is, however, a strong argument in favour of the same boundaries of radon-prone areas for dwellings and workplaces. The confusion likely to be caused by different boundaries would then be avoided. Underground workplaces, and other workplaces such as spas, should be considered separately.

5.1.1. *Workplaces in which intervention is needed*

(86) Workers who are not regarded as being occupationally exposed to radiation are usually treated in the same way as members of the public. It is then logical to adopt an action level for intervention in workplaces at the same level of effective dose as the action level for dwellings. The action levels for intervention in workplaces can be most easily derived from the range of action levels for dwellings by multiplying by 7000/2000 (the ratio of the occupancy) and by 3.88/5.06 (the ratio of the dose conversion coefficients). The resulting range (rounded) is 500–1500 Bq per m⁻³. When selecting action levels for dwellings and workplaces, authorities should choose values that are similarly located within the two ranges. In some mines, the equilibrium factor may be significantly different from 0.4. National authorities may then wish to use a different action level in terms of radon concentration in such mines.

5.1.2. *Workplaces in which the system of protection for practices should be applied*

(87) For workplaces, the Commission recognises in *ICRP Publication 60* the ubiquity of radiation and the need to avoid the conclusion that all workers should be subject to a regime of radiological protection. To avoid unrealistic and unnecessary protective measures, the Commission has concluded that its system of protection for practices should be applied at work only when the exposures incurred at work are a result of *situations that can reasonably be regarded as being the responsibility of the operating management.*

(88) To some extent, radon in workplaces can be so regarded. Nevertheless, the Commission recognises (*Paragraph 135*) that

“... there is some exposure to radon in all workplaces, and it is important not to require the use of a formal system of separate decisions to exempt each individual workplace where controls are not needed. They should be excluded from the control of occupational exposure by some general system. Considerable knowledge and judgement is needed to define such a system.”

The Commission goes on to recommend that exposure to radon should be excluded from its system of protection and treated separately, *unless the relevant regulatory agency has ruled otherwise, either in a defined geographical area or for defined practices.* Guidance is offered in this section on the basis for such a ruling.

(89) There are clearly advantages in adopting the same action level for requiring the application of the system of protection and for instituting remedial measures. The Commission therefore recommends the adoption of an action level within the range of 500–1500 Bq m⁻³ for both purposes. The corresponding range of annual effective dose is 3–10 mSv. When simple countermeasures do not reduce the radon concentrations below the action level, the Commission's system of protection should be applied to the practice.

(90) The control of radon may also need to be considered in workplaces where there is already a need for controls on the exposures directly associated with the work, that is to say, from artificial sources. For such circumstances, the Commission recommends that *“it will be sufficient to take account of the exposures to natural sources if, and only if, they would be controlled in their own right. . . . Elsewhere, they would not need to be included in radiation monitoring results, or in statistical reports of occupational exposures”* (*Paragraph 137*).

5.1.3. *Workplaces used by members of the public*

(91) Some workplaces are also used by members of the public. If the public occupancy is low, e.g. in offices, libraries and theatres, these workplaces need no special treatment. If the occupancy is high, e.g. in hospitals, residential institutions and schools, the premises should be treated as dwellings for the purpose of setting an action level for remedial measures. Workers should be subject to the Commission's system of protection for practices on the same basis as in any other workplace.

5.2. **Practical Protection in Workplaces**

(92) Having adopted an action level, the regulatory agency or the employer will need to determine what is to be done with a workplace where the radon concentration

exceeds that level. It would seem most sensible to start by taking whatever remedial measures are necessary to reduce the radon concentration to a value well below the action level. In many buildings, there will be little difficulty in taking such measures, but this may not be so in large complex structures. Preventive measures should be incorporated in new buildings in radon-prone areas.

(93) Should it prove unreasonably difficult, either in all or some parts of a building or an underground workplace, to reduce the radon below the action level, the system of radiological protection should be the same as when workers are exposed to artificial airborne activity at work. If radon concentrations vary widely in different parts of the workplace, the action level may be based on the annual time-weighted average concentration in the different parts of the workplace.

5.2.1. *The choice and application of exposure limits*

(94) The dose limit recommended by the Commission for effective dose is 20 mSv per year averaged over a period of 5 years with the proviso that the effective dose should not exceed 50 mSv in any single year (ICRP, 1991). For workers on short term contracts, the regulatory agency might consider an averaging period not exceeding the period of the contract of employment. The selection of the corresponding figure for exposure to radon progeny can best be done with the help of the convention, based on equal detriment, for the equivalence of radon exposure and effective dose. As indicated in Section 2, the Commission has decided to base its risk estimates primarily on the results of the radon epidemiology. The dosimetric estimate of the effective dose per unit exposure to radon progeny has therefore not been used in selecting the exposure limit.

(95) From the conversion coefficient of 1.43 mSv per (mJ h m^{-3}), 20 mSv corresponds to 14.0 mJ h m^{-3} (4.0 WLM) and 50 mSv corresponds to 35.0 mJ h m^{-3} (10.0 WLM). The corresponding figures for radon are thus:

14 mJ h m^{-3} per year (4 WLM per year), averaged over 5 years and
35 mJ h m^{-3} in a single year (10 WLM in a single year).

(96) Even if all the 1-year exposure is incurred in a short period, the absorbed doses to lung tissues will not be sufficiently high to cause deterministic effects. The derived air concentration for radon (occupancy of 2000 hours per year, equilibrium factor of 0.4) would be about 3000 Bq m^{-3} (average over 1 year). The exposure limits and the derived air concentrations are not the primary basis of control. The whole of the Commission's system of protection for practices should be applied, with emphasis on the optimisation of protection, which includes the use of any constraints on the choice of options.

5.2.2. *The application of the system of protection*

(97) The system of protection for practices recommended by the Commission applies to radon in workplaces where the radon concentration exceeds, or may exceed, the action level in the same way as it does in any workplace where radioactive substances are handled in unsealed forms. The issues relating specifically to mines were set out by the Commission in *ICRP Publication 47* (ICRP, 1986). They are still valid. More general guidance is given in the following paragraphs and stems directly from the recommendations of *ICRP Publication 60*. This guidance relates only to workplaces in which it has been decided to apply the Commission's system of protection.

(98) *Designation of areas.* Areas of workplaces where the radon is not directly associated with the operations in the workplace will need to be treated as supervised areas in which periodic measurements may be needed to confirm that concentrations have not increased with time. Exceptionally, the concentrations may be high enough to require special operating procedures and therefore to require the use of controlled areas. If the radon concentration is largely due to the operations, it is more likely that controlled areas will be needed with special working procedures adopted to control the exposure to radon.

(99) *Monitoring of individual exposures.* Employers will need to ensure that exposure of their workers in controlled areas is monitored in a systematic fashion (see Section 7.5 of *ICRP Publication 60*). It will sometimes be sufficient to use workplace, rather than individual, monitoring. Devices such as track-etch detectors may be used for either purpose, provided that workplace monitoring is related to working periods. Gross exposures rather than net values above the action level should be determined. The action level is merely the device for deciding to apply the system of protection to the radon exposures, all of which are then regarded as being the responsibility of the operating management.

(100) *Additivity of exposures.* It is possible that workers may be exposed both to radon above the action level, and to other sources, such as an x-ray machine, to which the system of protection for practices applies. In mines, there will often be exposures to radioactive ore dusts and gamma radiation. In such circumstances, it will be necessary to aggregate the doses for comparison with the dose limit. To do so, the dose conversion convention should be employed to translate the radon exposure into effective dose, which should then be added to the other effective doses for overall assessment. More generally, where workers are exposed to radon above the action level and to other sources, either internal or external, the conventional procedure of summing the quotients of the separate annual exposures and limits for comparison with unity should be followed to check for compliance with the recommended dose limits. The Commission recognises that no allowance has been made for exposures other than to radon in interpreting the epidemiological data. The requirement to add the dose from these sources for control purposes therefore errs somewhat on the side of caution.

6. SUMMARY

(101) The Commission has used an epidemiological basis for the assessment and control of radon exposure in this report. Since all the available epidemiological studies use the quantity inhaled potential alpha energy, this has been used as the primary quantity in this report. The Commission does not recommend the use of the dosimetric human respiratory model (ICRP, 1994) for the assessment and control of radon exposures.

(102) The Commission sees practical advantages in the delineation of radon-prone areas where more buildings than usual have elevated radon levels. For dwellings, it is suggested that areas with more than 1% of buildings with radon concentrations exceeding ten times the national average concentration might be designated as radon-prone, but the choice will depend on local conditions. A similar approach might be adopted in non-residential areas. Action against radon should be focused on such radon-prone areas.

(103) The imperatives of intervention against adventitious exposure to radon in buildings are clear. Above appropriate action levels, intervention is practicable and usually more cost-effective than other investments in radiological protection.

(104) Two types of building need to be considered, dwellings and workplaces. In both cases, radon concentrations are most likely to be elevated by the ingress of soil gas from the subjacent ground. Preventive and remedial measures to avoid this circumstance are recommended. The action levels adopted should fall within the recommended range of values given in Table 7.

(105) Proven measures against radon are readily available. For remedial work, the technical procedure that is most likely to maintain the radon level to a value well below the action level should be adopted from the outset. Intervention should take place soon after the discovery of elevated levels, especially if the concentrations are substantially above the action levels adopted by the competent authority. For preventive work, construction codes and building guides should be devised that will consistently achieve low concentrations of radon in the completed buildings.

(106) In workplaces, both in buildings and underground, where the radon concentrations remain above the recommended action level after any appropriate remedial measures have been taken, the Commission's system of protection should be applied and radon should be treated in the same way as any other radioactive material at work.

(107) The relevant data on conversion coefficients are given in Table 6 and the main quantitative recommendations are summarised in Table 7. Corresponding values in historical units are given in Table 8.

Table 6. Summary of conversion coefficients

Quantity	Unit	Value	Section
Exposure and radon gas conversions (equilibrium factor 0.4)	(mJ h m ⁻³) per WLM	3.54	1.4
	(mJ h m ⁻³) per (Bq h m ⁻³)	2.22×10^{-6}	
	WLM per (Bq h m ⁻³)	6.28×10^{-7}	
Annual exposure per unit radon concentration ^a			
	(mJ h m ⁻³) per (Bq m ⁻³)	1.56×10^{-2}	1.4
	(mJ h m ⁻³) per (Bq m ⁻³)	4.45×10^{-3}	
	WLM per (Bq m ⁻³)	4.40×10^{-3}	
	WLM per (Bq m ⁻³)	1.26×10^{-3}	

^a Assuming 7000 hours per year indoors or 2000 hours per year at work and an equilibrium factor of 0.4.

Table 7. Summary of values recommended in this report

Quantity	Unit	Recommended value	Section
Nominal fatality and detriment coefficient at home and at work	$(\text{mJ h m}^{-3})^{-1}$	8×10^{-5}	2.2.2. 2.2.3. 2.2.4.
Dose conversion convention, effective dose per unit exposure:			
at home	mSv per (mJ h m^{-3})	1.1	2.2.5.
at work	mSv per (mJ h m^{-3})	1.4	
Action level (dwellings)			
Radon concentration	(Bq m^{-3})	200–600 ^a	4.2.1.
Annual effective dose	mSv	3–10	
Action level (workplaces)			
Radon concentration	(Bq m^{-3})	500–1500 ^a	5.1.
Annual effective dose	mSv	3–10	
Occupational annual limit on exposure	(mJ h m^{-3}) per year, averaged over 5 years (mJ h m^{-3}) in a single year	14 35	5.2.1.

^a Assuming 7000 hours per year indoors or 2000 hours per year at work and an equilibrium factor of 0.4.

Table 8. Summary of quantities in historical units corresponding to those in Table 7

Quantity	Unit	Recommended value	Section
Nominal fatality and detriment coefficient at home and at work	$(\text{WLM})^{-1}$	3×10^{-4}	2.2.2. 2.2.3. 2.2.4.
Dose conversion convention, effective dose per unit exposure:			
at home	mSv per WLM	4	2.2.5.
at work	mSv per WLM	5	
Action level (dwellings)			
Radon concentration	(Bq m^{-3})	200–600 ^a	4.2.1.
Annual effective dose	mSv	3–10	
Action level (workplaces)			
Radon concentration	(Bq m^{-3})	500–1500 ^a	5.1.
Annual effective dose	mSv	3–10	
Occupational annual limit on exposure	WLM per year, averaged over 5 years WLM in a single year	4 10	5.2.1.

^a Assuming 7000 hours per year indoors or 2000 hours per year at work and an equilibrium factor of 0.4.

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ANNEX A. AN ILLUSTRATIVE EXAMPLE OF EPIDEMIOLOGY OF MINERS EXPOSED TO RADON AND ITS PROGENY

NOTE: This annex is a summary of a study done in 1992. It is used here only to illustrate the methodology of combining studies and of interpreting the combined finding in terms of the lifetime risk of chronic exposure.

(A1) This annex summarises the epidemiological findings of several studies that are of importance for the assessment of exposure limits.

(A2) One general uncertainty in all these studies stems from the estimates of the miners' exposure to radon and its progeny. This concerns particularly those miners who received their exposure at earlier times when no radon measurements were carried out in these mines. Among the more reliable exposure estimates for this earlier mining period are those available for the Bohemian uranium miners. This study cohort comprises 4042 miners who started underground work between 1948 and 1957 at a mean age of 32 years (mean duration of employment 8.2 years) and were followed-up from 1953–1985. The mean annual number of radon measurements per mine-shaft in this mining district increased from about 100 in 1948 to about 700 in 1970 (Sevc *et al.*, 1993).

A.1. The Principal Study Groups

(A3) Characteristics of the seven more quantitative cohort studies of underground miners are summarised in Table A.1. For the Beaverlodge (Eldorado) miners, a mean cumulative exposure of 22 WLM was previously estimated (Howe *et al.*, 1986). A re-analysis of the exposure conditions and the working history of these miners is still in hand, but early results indicate that their real exposure was probably, on average, about a factor of two higher (SENES, 1991; Chambers *et al.*, 1992).

(A4) The cohort studies listed in Table A.1 comprise in total about 31,500 underground miners with a mean age at the start of exposure of about 30 years. The mean employment period of the uranium miners was about 8 years, averaged over all cohort studies. The weighted mean value of their cumulative exposure during their underground work, weighted by the number of miners in each group, was about 120 WLM. The mean follow-up period of these epidemiological studies varies from 14 to 32 years; the mean period, weighted by the person years at risk in each study, is about 20 years.

A.2. Exposure–Risk Relationship

(A5) In general, the cohort studies of miners listed in Table A.1 yield a monotonic increase of the excess relative lung cancer risk with the cumulative exposure to radon progeny. Taking into account the statistical confidence range of the excess relative risk, the data can be fitted by a proportional exposure–risk relationship up to cumulative exposures of a few hundred WLM (NRC, 1988).

Table A.1. Characteristic data of seven study cohorts of underground miners

Cohort study follow-up period	Number of miners	Person years at risk	Mean cumulative exposure (WLM)	Number of lung cancer deaths	
				Obs	Exp
Uranium miners					
Colorado, USA, 1951–1982, cumulative exposure <2000 WLM ^a	2975	66 237	510	157	48.7
New Mexico, USA, 1957–1985	3469	66 500	111	68	17
Ontario, Canada, 1955–1981	11 076	217 810	37	87	57.9
Beaverlodge, Saskatchewan, Canada, 1950–1980	6847	114 170	44 (22) ^b	65	28.7
Bohemia, 1953–1985	4042	97 913	227	574	122
France, 1946–1985	1785	44 005	70	45	21.1
Iron miners					
Malmberget, Sweden, 1951–1976	1292	27 397	98	51	14.9
Total, all studies	31 486	635 022	120 ^c	1047	310

^a The higher exposures were eliminated because of the likelihood of the effect of cell killing on the incidence of lung cancer.

^b Original value (Howe *et al.*, 1986). See Paragraph (A7).

^c Weighted by number of miners in each study.

(A6) Furthermore, the analysis of the excess risk data as a function of time since exposure indicates some correlation with the baseline risk, R_0 , of lung cancer without radiation exposure. This supports the use of the multiplicative risk projection model.

(A7) Table A.2 shows the mean values of the excess relative risk of lung cancer per unit exposure that follow from the cohort studies listed in Table A.2. These relative risk

Table A.2. Mean excess relative risk of lung cancer per unit cumulative exposure to radon progeny resulting from the different cohort studies of underground miners; the values are averaged over the follow-up period and all exposure cohorts^a

Study group, follow-up period	Excess relative risk per J h m ⁻³ (282 WLM) ^b	
	Mean value	95% confidence interval
Uranium miners		
Colorado, USA, 1951–1982, exposure <2000 WLM	1.7	0.85–4.0
New Mexico, USA, 1957–1985	5.1	2.0–15.4
Ontario, Canada, 1955–1981	4.0	1.7–9.4
Beaverlodge, Saskatchewan, Canada, 1950–1980	3.7 ^c	1.7–8.5
Bohemia, 1953–1985	4.8	3.4–6.8
France, 1946–1985	1.7	0–4.6
Iron miners		
Malmberget, Sweden, 1951–1976	4.0	0.85–27
Weighted mean, all studies^d	3.79	2.3–6.0

^a Referring to the studies listed in Table A.1. For references see text.

^b To obtain the risk coefficient per 100 WLM, the values have to be divided by a factor 2.82.

^c Revised value taking into account new exposure estimates for these miners (SENES, 1991).

^d Weighted for person years at risk.

coefficients are averaged over the follow-up period and all exposure cohorts of each study; in the case of the Colorado miners the cohort with exposures above 7 J h m^{-3} (2000 WLM) was excluded. The data for the Colorado, Ontario, and Malmberget miners are based on the BEIR IV study (NRC, 1988). The values for the uranium miners in Bohemia, in New Mexico, and in France were taken from new or updated publications (Sevc *et al.*, 1993; Samet *et al.*, 1991; Tirmarche *et al.*, 1992a,b). In the case of the Beaverlodge miners, in addition to the previous value (Howe *et al.*, 1986), a revised value is given, based on the new exposure estimates for these miners (SENES, 1991).

(A8) The excess relative risk coefficients that follow from the different cohort studies in Table A.2 do not differ significantly. Summing over all these studies results in a weighted mean value of the excess relative risk coefficient, averaged over the follow-up period, of

3.79 (2.3 to 6.0) per (J h m^{-3})

or 1.34 (0.8 to 2.1) per 100 WLM

(95% confidence interval in brackets).

(A9) The studies of non-uranium miners that are referred to in this section but not listed in Table A.1, provide only qualitative or weak quantitative information about the link between radon exposure and the excess relative risk of cancer. The quantitative results are consistent with those in Table A.2.

(A10) In addition, the statistical analysis of the Bohemian uranium miners leads to the suggestion that the excess relative risk coefficient might be somewhat higher at exposures below 100 WLM compared with the mean value listed in Table A.2 (Sevc *et al.*, 1993). However, owing to the absence of an internal control group, the possibility that this tendency may be due to other confounding factors such as smoking cannot be excluded. The same is true for the observed tendency of a somewhat higher effectiveness per unit exposure at protracted exposures. A similar exposure-rate effect has been derived from the data of the Colorado miners (Hornung and Meinhardt, 1987). However, the analysis of the data sets from four cohort studies of uranium miners, carried out by the BEIR IV Committee, yields no consistent pattern on this issue. Another factor that could account for higher risk coefficients at low exposures might be the presence of other carcinogens, the exposures to which were not correlated with the radon exposures. The lack of information about exposures in dwellings may also play a part.

(A11) Dosimetric models lead to the conclusion that the bronchial dose per unit of exposure increases as the fraction of the radon progeny attached to condensation nuclei decreases. The bronchial dose per unit exposure then increases with increasing ventilation of working areas in mines or with decreasing dust concentration (NRC, 1991). Following these dosimetric arguments, a higher excess lung cancer risk per unit exposure in well ventilated mines would be expected compared with that in poorly ventilated ones. This tendency may go some way towards explaining the finding that the excess risk per unit exposure is larger at lower exposures. The results of the Swedish case-control study in dwellings (Pershagen, 1993) suggest that the effect does not influence the risk estimates of Table A.2, but the statistical limitations prevent a categorical conclusion.

(A12) Finally, it should be recognised that the primary data on the excess risk of lung cancer in radon-exposed miners include the risk contribution from external radiation and from inhaled, long-lived, emitters in mines. Under the exposure conditions of the considered cohorts of uranium miners, the relative contribution from these other

occupational radiation sources was probably small. In *ICRP Publication 50* (ICRP, 1987), a relative risk correction of about 10–20% was assumed. For reasons set out in Annex B, the Commission no longer makes any correction for these factors. There may also have been some exposure to other carcinogens. Quantitative estimates are available only in a few studies, so no account has been taken of their contribution to the observed mortality, all of which has been assigned to the radon exposures.

(A13) In summary, the epidemiological findings from these studies provide no firm conclusion on the real shape of the exposure–risk relationship, particularly at low cumulative exposures. They are broadly consistent with a proportional relationship. They are also consistent with other dose–response relationships, including both threshold relationships and those with enhanced risk coefficients at low exposures. The proportional relationship leads to a central estimate of the excess relative risk of fatal lung cancer per unit exposure at work of

3.79 per (J h m^{-3})
or 1.34 per 100 WLM.

This central estimate of the excess relative risk coefficient refers to a follow-up period of 20 years, taking into account a time lag (minimum latency) of 5 years.

(A14) To obtain estimates of the lifetime risk of chronic exposure it is necessary to postulate a projection model. The Commission uses a multiplicative projection model for this purpose. Three different versions of this model are used in this annex:

- (a) The multiplicative model used in *ICRP Publication 50* (ICRP, 1987) which uses a persistent (excess) relative risk (PRR model).
- (b) The modified projection model proposed in the BEIR IV study (NRC, 1988) which takes into account the variation of the excess relative risk with time since exposure (TSE) and with attained age (BEIR IV model).
- (c) The modified projection model which has been developed at the GSF (Jacobi *et al.*, 1992) which considers the age-specific excess rate of lung cancer as function of age at exposure and of time since exposure (GSF model).

These models and their original input parameters are summarised at the end of this annex. The GSF model was developed primarily for the evaluation of the probability of causation for lung cancer among the uranium miners in Saxonia and Thuringia in eastern Germany.

(A15) The original models used an excess relative risk coefficient of 1.6×10^{-2} per WLM, reduced in the case of the ICRP model to 1×10^{-2} per WLM because of the expected overestimate due to the use of a constant excess relative risk coefficient. In this report, the excess relative risk coefficient is lower by a factor of 1.33/1.6, or 0.83.

(A16) Although the three models show some differences in the age specific excess risks, the estimate of the attributable lifetime risk of lung cancer is much less sensitive to the choice of model.

(A17) The input parameters of these risk projection models are based on the epidemiological data from male miners. In *ICRP Publication 50* (ICRP, 1987) and in the BEIR IV study (NRC, 1988), it was assumed that, at the same exposure to radon progeny, the relative excess of the age-specific lung cancer rate in females would be equal to that in males. The validity of this assumption, which implies a purely multiplicative influence of smoking, is questionable.

(A18) The lifetime values of the excess relative risk and of the excess absolute risk of fatal lung cancer from occupational exposure to radon progeny that follow from these projection models are listed in Table A.3. These central estimates refer to a chronic exposure at constant annual levels of 3.5, 7 and 14 mJ h m⁻³ per year (1, 2 and 4 WLM per year).

Table A.3. Excess lifetime relative risk and lifetime probability of fatal lung cancer for the 'Male Reference Worker', attributable to chronic occupational exposure to radon (²²²Rn) progeny from age 18 to 64 (baseline risk $R_0 = 0.042$)

Risk quantity	Annual exposure mJ h m ⁻³ (WLM)	Projection model ^a		
		PRR model ICRP 50 (1987)	TSE model BEIR IV (1988)	TSE model GSF (1992)
Excess relative risk	3.5 (1.0)	0.35	0.29	0.31
	7.0 (2.0)	0.68	0.56	0.62
	14.0 (4.0)	1.33	1.12	1.19
Excess absolute risk	3.5 (1.0)	0.015	0.012	0.013
	7.0 (2.0)	0.029	0.024	0.026
	14.0 (4.0)	0.056	0.047	0.050

^a The excess relative risk coefficients underlying these models have been modified by a factor of 0.83 from the original values used in the models (see text).

(A19) The nominal fatality probability coefficient for occupational exposure to radon progeny can now be derived giving the results shown in Table A.3. Of the three models available, the original ICRP model is unsatisfactory because the constant excess risk factor is not appropriate. The BEIR IV model and the GSF model give similar results, but the smooth variation of the excess relative risk factor in the GSF model is more plausible biologically. The basis adopted in this annex is the attributable probability of fatal cancer for an exposure of 7 mJ h m⁻³ per year of working life using the GSF projection model. This leads to a probability coefficient (fatality) of

$$0.026 / (7.0 \times 47) = 7.90 \times 10^{-5} \text{ per (mJ h m}^{-3}\text{)} \\ (2.80 \times 10^{-4} \text{ per WLM}).$$

These values have been derived for a male workforce with a working lifetime from age 18 to 64 years, inclusive (47 years). They are assumed also to apply to a similar female workforce.

A.3. Risk Projection Models

(A20) This section summarises the form of the risk projection models used in this annex. All the models are multiplicative risk projection models, assuming a correlation between the excess age-specific lung cancer rate λ_r , caused by a preceding exposure, and the normal baseline rate of lung cancer, λ_0 at the attained age a (age at risk). This implies a multiplicative influence of smoking. Furthermore, all these models proceed from a proportional relationship between the exposure P_p , received at an age t_e (age at exposure) and the attributed excess rate of lung cancer in the subsequent years, taking into account a time lag τ (minimum latency) between exposure and expression of lung

cancer. As originally used, all the models used the same excess relative rate, λ_r/λ_o , for males and females. All these projection models proceed from the general equation:

$$\lambda_r(a) = \lambda_o(a)f(t_e, a)P_p(t_e) \text{ for } a \geq (t_e + \tau).$$

Besides the inserted age-specific baseline rate, the models apply different functions $f(t_e, a)$ which express the variation of the excess relative rate with time since exposure, $T = a - t_e$, and with the attained age a .

A.3.1. *The model used in ICRP Publication 50 (ICRP, 1987)*

(A21) The relative model used in *ICRP Publication 50* proceeds from a mean, time-averaged, relative risk coefficient and a persisting excess relative risk factor until the end of life (PRR model):

$$\lambda_r(a)/\lambda_o(a) = fP_p(t_e) \text{ for } a > t_e + \tau,$$

with $\tau = 5$ years.

A.3.2. *The BEIR IV model*

(A22) The model proposed by the BEIR IV Committee (NRC, 1988) is based essentially on an analysis of the lung cancer data from uranium miners in Canada and the U.S.A. It is a modified multiplicative projection model that takes into account the time since exposure as well as the attained age. The finally recommended model is given by the relationship:

$$\lambda_r(a)/\lambda_o(a) = s\gamma(a)[P_1 + 0.5 P_2]$$

with $\gamma(a) = 1.2$ for $a < 55$ years
 $\gamma(a) = 1.0$ for $a = 55-64$ years
 and $\gamma(a) = 0.4$ for $a = 65$ years

s is a constant of proportionality with exposure, taken by the original BEIR IV model to be 0.025. P_1 is the potential α energy exposure, in WLM, incurred between 5 and 15 years before the age a , and P_2 , in WLM, is the exposure incurred 15 years or more before this age.

Thus, this model leads to a stepwise reduction of the age-specific excess rate.

A.3.3. *The GSF model*

(A23) The primary objective of this recently developed model (Jacobi *et al.*, 1992) was the evaluation of the probability of causation of lung cancer among the previous uranium miners in eastern Germany.

(A24) Like the BEIR IV model, this is a modified multiplicative projection model. Its main variable is the time since exposure, $T = a - t_e$. The basic equation of this model, referring to a single potential energy exposure, P_p , is:

$$\lambda_r(a)/\lambda_o(a) = s(t_e)P_p(t_e)\phi(T) \text{ for } a > t_e + \tau$$

where $\tau = 4$ years.

The function $\phi(T)$, with T in years, characterises the relative latency distribution,

which is normalised to one at its maximum. Beyond this maximum a decrease of the excess relative rate with a half-life of 10 years is assumed.

$$\begin{aligned} \phi(T) &= 0 && \text{for } T \leq 4 \text{ years} \\ \phi(T) &= 0.25(T - 4) && \text{for } 4 \text{ years} < T < 8 \text{ years} \\ \phi(T) &= 1 && \text{for } 8 \text{ years} \leq T \leq 12 \text{ years} \\ \text{and } \phi(T) &= \exp[-(\ln 2/10)(T - 12)] && \text{for } T > 12 \text{ years.} \end{aligned}$$

The function $s(t_e)$ is a function of proportionality with exposure taking into account the decreasing carcinogenic susceptibility of the lung with increasing age t_e at time of exposure. In the original model it decreased from 0.036 WLM^{-1} for an age at exposure of 20 years, to 0.017 WLM^{-1} for age at exposure of 60 years.

Compared with the BEIR IV-step model the GSF model provides a monotonic variation of the excess relative lung cancer with age or time since exposure.

A.3.4. Excess lifetime risk

(A25) Assuming a proportional exposure–risk relationship, all these relative risk models proceed from the following general equation for the age specific excess rate of lung cancer, λ_r , at an attained age a . The baseline rate at this age is $\lambda_0(a)$;

$$\lambda_r(a, P) = \lambda_0(a) \int_{18y}^{a-\tau} f_r(a, t_e) \dot{P}(t_e) dt_e$$

This relationship refers to a chronic exposure starting at an age t_e (age at time of exposure) of 18 years. $\dot{P}(t_e)$ is the exposure rate at age t_e . $f_r(a, t_e)$ is the attributed excess relative risk at the attained age a per unit of exposure at age t_e . The integration considers a time lag τ (minimum latency) between exposure and the expression of lung cancer from inhaled radon progeny.

(A26) Taking into account a survival probability $p(a)$ from start of exposure until the considered age a , the excess lifetime risk up to an age of 90 years becomes

$$R_r = \int_{18y}^{90y} p(a) \lambda_r(a, P) da$$

ANNEX B. THE MAGNITUDE OF POSSIBLE CORRECTIONS TO THE EXPOSURE LIMITS

(B1) Three possible corrections to the exposure limits are considered. The first relates to the estimation of the total detriment associated with a fatal lung cancer caused by exposure to radon and its progeny. The other two are concerned with the possible contribution to the incidence of the observed lung cancers from exposures other than that due to radon progeny.

B.1. Detriment Other than from Lung Cancer

(B2) In assessing the total detriment associated with a fatal lung cancer attributable to radon progeny, it is necessary to assess the contributions to the effective dose due to radon dissolved in tissues other than the lung and due to the direct inhalation of radon

progeny. The effective dose equivalent rate from continued exposure to a radon concentration of 1 Bq m^{-3} with no contribution from radon progeny except that from the decay of radon after inhalation has been estimated (Peterman and Perkins, 1988). Table B.1 is based on their data, with some increase in the contributions from organs with a high fat content. The tissue weighting factors of *ICRP Publication 60* have been used.

Table B.1. The effective dose from exposure of tissues other than lung due to the inhalation of unit concentration of radon free of progeny

Organ or tissue	Equivalent dose per unit exposure $10^{-10} \text{ Sv per (Bq h m}^{-3})$	Tissue weighting factor	Contribution to effective dose 10^{-10} Sv
Gonads	0.38	0.2	0.076
Bone marrow ^a	2.00	0.12	0.24
Colon	0.66	0.12	0.079
Stomach	0.66	0.12	0.079
Bladder	0.33	0.05	0.165
Breast ^b	1.50	0.05	0.075
Liver ^a	1.30	0.05	0.065
Oesophagus ^c	0.66	0.05	0.033
Thyroid	0.66	0.05	0.033
Bone	0.15	0.01	0.0015
Skin ^c	0.66	0.01	0.0066
Remainder ^c	0.66	0.05	0.033
Total (rounded)			0.74

^a Approximately twice the value from Peterman and Perkins (1988).

^b Approximately five times the value from Peterman and Perkins (1988).

^c Taken as "vessel rich" from Peterman and Perkins (1988).

(B3) For the exposure of workers to radon and its progeny with an equilibrium factor of 0.4 and an occupancy of 2000 hours, a concentration of radon of 500 Bq m^{-3} is equivalent to an annual effective dose of about 3 mSv, i.e. 1 Bq h m^{-3} is equivalent to $3 \times 10^{-6} \text{ mSv}$. The effective dose from the inhalation of 1 Bq h m^{-3} of radon alone (Table B.1) is $0.74 \times 10^{-7} \text{ mSv}$, or about 2% of the total effective dose.

(B4) It is difficult to estimate the effective dose from tissues other than the lung due to directly inhaled radon progeny because the estimate depends critically on the rates of transfer from the point of deposition to other organs and tissues. Preliminary estimates suggest that the contribution will be a few percent of the total effective dose.

(B5) For lung cancer alone, the detriment coefficient is 0.95 times the fatality coefficient. On the basis of the two additional contributions to the effective dose, and thus to the total detriment, the Commission has adopted a total detriment coefficient equal to the fatality coefficient.

B.2. Lung Cancer due to External Radiation and Ore Dust in the Study Groups

(B6) A typical level of exposure to radon progeny in the mines for which the epidemiological data have been obtained is in the region of 10 WLM per year. This corresponds to an effective dose of about 50 mSv per year, giving a fatality rate of about 3×10^{-3} per year. Estimates of annual gamma doses in uranium mines were given

by the Commission in *ICRP Publications 32 and 47* (ICRP, 1981, 1986). These estimates were in the region of 5–10 mSv per year. The fatality coefficient for lung cancer from Table B.20 in *ICRP Publication 60* is 8.5×10^{-3} per Sv, giving a lung cancer fatality rate from external radiation in the region of 110^4 per year. This is not sufficient to justify inclusion in the interpretation of the epidemiology. Any such correction would tend to decrease the estimated risk coefficient associated with the radon progeny.

(B7) The possible contribution from the inhalation of ore dust is very uncertain. Concentrations of respirable dust of the order of 10 mg m^{-3} are not uncommon in uranium mines. The specific alpha activity of the particles is low and any carcinogenic effects may be influenced more by the physical and chemical forms of the dust than by the radioactive content. There seems to be no adequate basis for making an allowance for possible lung cancer due to the dust. Any such correction would tend to decrease the estimated risk coefficient associated with the radon progeny.

ANNEX C. GLOSSARY

Action level: The concentration of radon at which intervention is recommended to reduce the exposure in a dwelling or workplace.

Bronchial tree: The branching airways of the respiratory tract from the trachea to the entry to the gas exchange or pulmonary region of the lung.

Condensation nuclei: Any small particles or ions capable of serving as a site for the condensation of vapour.

Dose conversion convention: The method used to relate exposure to radon progeny expressed in WLM, to effective dose expressed in mSv on the basis of equal detriment.

Equilibrium equivalent concentration, c_{eq} : The activity concentration of radon, in equilibrium with its short-lived progeny which would have the same potential alpha energy concentration as the existing non-equilibrium mixture.

Equilibrium factor, F : The ratio of the equilibrium equivalent concentration and the radon gas concentration.

Potential alpha energy concentration, c_p : The concentration of short-lived radon progeny in air in terms of the alpha energy released during complete decay through polonium-214.

Potential alpha energy exposure, $P_p(T)$: The time integral of the potential alpha energy concentration in air, c_p to which an individual is exposed over a given time period T , e.g. one year.

Radon progeny: The decay products of radon-222, used herein in the more limited sense of the short-lived decay products from polonium-218 through polonium-214.

Radon prone: An area in which the characteristics of the ground cause more buildings than usual to have elevated radon levels.

Reference level: Used to establish values of measured quantities such as recording level or investigation level, above which some specified action or decision should be taken.

Risk: Terms relating to risk are grouped together here.

Risk.

In this report, the probability that a fatal lung cancer will occur.

Relative risk.	The ratio of the risk in an exposed population to that in a similar unexposed population.
Excess relative risk.	Relative risk - 1.
Absolute risk.	The probability that a fatal lung cancer will occur.
Excess or attributable risk.	The absolute excess or attributable risk due to an exposure.
Risk coefficient.	The risk per unit exposure.
Risk projection model.	A model describing the variation of risk as a function of the time since exposure. It may be related by a factor to the age specific baseline risk (multiplicative) or added to the baseline risk (additive).

Soil gas: A gas in the free space within a volume of soil.

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

Working level: Any combination of the short-lived progeny of radon in one litre of air that will result in the emission of 1.3×10^5 MeV of potential alpha energy.

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

THE HISTORY OF THE RADON PROBLEM IN MINES AND HOMES

W. Jacobi

The radon saga is a scientific thriller with tragic features and political confounders. The historical roots of this saga reach back to the 15th century. It is a field full of dilemmas, controversies and frustrations, some of which still persist. One should learn from this history which has been described in part by Schüttmann (1988) and Stannard (1988).

1. THE 'SCHNEEBERGER LUNG DISEASE'

About 1470, extensive mining of silver commenced in the region of Schneeberg, a small city in Saxony/Germany at the northern slope of the "Erzgebirge" (Ore Mountains). Silver was also mined in the region of Joachimsthal (now Jachymov) at the southern Bohemian side of the Ore Mountains at about the same time. The mining techniques which were applied in both of these regions at the beginning of the 16th century have been described and illustrated by Agricola (1494–1555), referred to as the father of mineralogy, who worked from 1527–1533 as physician in Joachimsthal (Agricola, 1556). His most famous book *De Re Metallica* was translated from Latin to English by the American mining engineer Herbert C. Hoover (who later became the president of the United States) and his wife Lou. Agricola indicated that in Jachymov, the silver ore was mined at or near the surface, whereas in Schneeberg already the ore was mined at greater depths. Some shafts reached a depth of about 400 m.

An unusually high mortality from lung disease, occurring in younger workers, was observed among the miners in the Schneeberg region in the early 16th century. The first report stems from Paracelsus (1493–1541) in his book *Über die Bergsucht und andere Bergkrankheiten* (About the 'Bergsucht' and other Miner's Diseases). The word *Bergsucht* is a summary term for the lung diseases observed in miners. Paracelsus had written this book in the year 1537, but it was printed only after his death (Paracelsus, 1567, new edition 1925).

The frequency of this lung disease, which was later called 'Schneeberger Lungenkrankheit' ('Schneeberger Lung Disease'), increased in the 17th and 18th centuries, when the mining of silver, cobalt and copper was intensified (see Rosen, 1943). The disease was eventually identified as lung cancer by Haerting and Hesse (1879). Originally, it was assumed to be a lymphatic sarcoma, originating from the bronchial lymph nodes and it was somewhat later classified as bronchial cancer. Haerting and Hesse mention that at about this time 75% of the miners in the Schneeberg region died from lung cancer. The available reports indicate that the percentage was probably lower among the miners of Jachymov.

2. THE SEARCH FOR THE CAUSES

Paracelsus (1567) labelled the 'Schneeberger Lung Disease' as "Mala Metallorum". It was assumed that the lung cancer was caused by inhaled ore dust containing different

metals. Contributory carcinogenic factors were thought to include tubercular disease and the presence of arsenic in the dust.

In 1898, Marie and Pierre Curie had extracted radium (^{226}Ra) and polonium (^{210}Po) from Jachymov ores (Curie, 1898). The so-called radium emanation, later called radon (^{222}Rn) was identified as a radioactive noble gas produced by the decay of radium. Starting with the first radon measurements by Elster and Geitel (1901), a high radon concentration in the air of mines at Schneeberg and Jachymov was subsequently demonstrated. The first cases of cancer, particularly skin cancer, induced by the x-rays of radium radiation, were reported at the beginning of the 20th century (Friebe, 1902; Hesse, 1911).

It was on the basis of these findings, that a relation between lung cancer and the high radon content in these mines was assumed. Schüttmann (1988) considered that H. E. Müller, a mining director in Zwickau, Saxony, was the first person to recognize the causal link. Müller concluded that the Schneeberger lung cancer was a specific occupational disease, caused by the radium content of the ore and the high radon content of the air in these mines which, when inhaled, initiated a carcinogenic process in the airways of the lung.

This hypothesis was supported by more precise radon measurements carried out in the 1920s in the Schneeberg (Lorenser and Ludewig, 1924) and Jachymov mines (Pirchan and Sikl, 1932). However, the role of radon as a causative factor for the Schneeberger lung cancer was not generally accepted. In a summary report of a group of pathologists from Dresden which was published in 1926, the opinion was expressed that this cancer type might be initiated by the inhalation of toxic ore dusts (Rostosky *et al.*, 1926). Lorenz (1944) later claimed that arsenic and other mine contaminants, as well as the poor health of the miners, were the primary causes.

A research programme in Germany which was initiated in 1936 by B. Rajewsky from the Kaiser-Wilhelm-Institute (later Max-Planck-Institute) for Biophysics in Frankfurt, Main, provided further clarification of the relation between radon concentration and lung cancer. This comprehensive study involved radon measurements in the mines near Schneeberg; and measurements of the alpha activity in tissue samples and histopathological analysis of lung tissues from miners who had died from lung cancer (Hueck, 1939; Rajewsky, 1940). At that time, the average radon concentration in most mines at Schneeberg was within the range of 70–120 kBq m⁻³. In one mine, however, a mean value of about 500 Bq m⁻³ was observed. It was known that most workers in this mine died from lung cancer; it was called “death mine”. On the basis of these observations and supporting biological studies, it was concluded that in the Schneeberg mines, the inhalation of radon must be regarded as a possible cause for the high lung cancer frequency among the miners in Schneeberg region (Rajewsky, 1940).

This summarises the extent of knowledge in 1945. The available data from Schneeberg and Jachymov did not enable any quantitative estimate of the relationship between radon exposure and lung cancer. Furthermore, the possible role of the inhaled short-lived decay products of radon was not yet realised.

3. URANIUM MINING AND THE ROLE OF THE RADON DAUGHTERS

The extensive mining and processing of uranium for military purposes started in the 1940s. The main sources at this time were the uranium deposits in the Belgian Congo

(now Zaire), Canada and in Colorado, USA. In 1946, the intensive mining of uranium commenced under a directive of the USSR government in the historical mining region of Aue, Schneeberg in East Germany. Proceeding from the radium production in Jachymov, the mining of uranium in Bohemia began in 1948. At the same time, uranium mining started in France.

During this early phase of uranium mining, little attention was paid to the radiological protection of workers. It was believed that the radon levels in these new mines were considerably lower than in the older mines in the Ore Mountains and only a few radon measurements were reported in this period. In the Colorado mines, radon samples were not taken before 1950; and in the uranium mines in East Germany, radon data are only available after 1955 (Jacobi, 1992).

During this period, dosimetric studies and radiobiological research on the possible effects of the inhaled radon were continuing. However, all attempts failed to explain the induction of lung cancer by inhalation of radon gas alone, until William F. Bale (Bale, 1951) in Rochester introduced the idea that the decay products of radon might be the causative agent. John Harley confirmed the presence of high concentrations of these radon decay products by measurements in air (Harley, 1953). Bale stated in his memorandum: "In these and other past evaluations of the hazard associated with radon, the vital fact seems to have been almost entirely neglected that the radiation dosage due to the disintegration products of radon, *present in the air* under most conditions where radon itself is present, conceivably and likely will far exceed the radiation dose due to radon itself and to disintegration products formed while the radon is in the bronchi".

In the following years, experimental studies on the deposition and retention of inhaled radon and thoron decay products in the lung were carried out at the University of Rochester (Bale and Shapiro, 1956; Shapiro, 1956); and at the Max-Planck-Institute for Biophysics in Frankfurt, Main (Schraub *et al.*, 1955; Aurand *et al.*, 1955; Jacobi *et al.*, 1956). The results of these studies enabled a quantitative estimate to be made of the mean alpha dose to the bronchial epithelium from inhaled decay products of radon. Dosimetric lung models for the evaluation of the activity and dose distribution along the bronchial airways were also developed (Altschuler *et al.*, 1964; Jacobi, 1964). It was concluded from these various studies that the maximum alpha dose should be expected in the target cells of the segmental bronchi of the airways. Lung dosimetry of inhaled short-lived radon decay products, now called radon progeny, is still an important and controversial area of research.

As a consequence of these studies, there followed the development of more reliable methods for the monitoring of radon progeny in mines. In the United States, the concept of the potential alpha energy concentration of radon progeny in air, the so-called 'Working Level Concept' was created (Holaday, 1957). Practical experience has confirmed the appropriateness of this simplifying concept for the purposes of monitoring, and for the evaluation of the exposure of miners to radon progeny.

About the same time, a study on the health status and the relation to radiation exposure of the uranium miners in Colorado was carried out by the Division of Occupational Health of the US Public Health Service. The preliminary results indicated a significant excess of lung cancer among these miners (Wagoner *et al.*, 1964). Following the hearings before the Joint Committee on Atomic Energy of the Congress of the United States in 1967, revised guidelines for the control of radiation hazards in uranium mining were established by the US Federal Research Council (FRC, 1967).

The first quantitative analysis of the cohort study among the US uranium miners, covering the period from 1950 through 1967, was published by Lundin *et al.* (1971). One year later, the results of a similar study among uranium miners in Czechoslovakia was reported (Sevc *et al.*, 1972; see also Sevc *et al.*, 1976). Both studies concluded that the lung cancer risk increased monotonically with the cumulative exposure to radon progeny. But the resulting slope of this dose-response relationship was considerably higher for the Czech miners. Several other cohorts of uranium miners have since been followed up. The available updated results of all these studies, which constitute a follow-up on about 30,000 uranium miners, are summarised in this issue, *ICRP Publication 65*.

Averaging these studies provides an excess relative risk of lung cancer of about three. It is noteworthy that this excess is appreciably higher than the excess relative risk from all types of cancer in the life-span study of the atomic bomb survivors in Hiroshima and Nagasaki. It should be emphasized that the epidemiological cohort studies involve only a small fraction of all uranium miners. For example, the available information indicates that in the uranium mines in East Germany, about 250,000 persons worked underground during the critical years from 1946–1955. Their average exposure to radon progeny is estimated to be about 100–200 WLM per year (Jacobi, 1992). So far, no epidemiological data are available from these studies. Thus, it can be assumed that from 1945 to the present, a total of about 500,000 persons worldwide have worked in uranium mines.

High radon levels have also been observed in non-uranium ore mines. Epidemiological surveys of some of these mining cohorts indicate an excess risk of lung cancer, for example, amongst fluorspar miners in Newfoundland, Canada (Morrison *et al.*, 1988), Chinese tin miners (Lubin *et al.*, 1990, 1993) and iron ore miners in Sweden (Radford *et al.*, 1984).

4. PERCEPTION OF THE RADON PROBLEM IN HOMES

Compared with the situation in mines, the possible influence of radon on lung cancer risk to the general public was discovered much more recently. One year after the discovery of radon, the measurements of Elster and Geitel (1901) revealed that radon (at that time called 'radium emanation') was a ubiquitous constituent of atmospheric air. In a paper entitled "Some Cosmical Aspects of Radioactivity" presented at a meeting in Canada in April 1907, Ernest Rutherford said: "We must bear in mind that all of us are continuously inhaling radium and thorium emanations and their products, and ionising air. Some have considered that possibly the presence of radioactive matter and ionised air may play some part in physiological processes" (Rutherford, 1907). It is noteworthy that the balneological application of radon was started in the following years. The first 'Radium Inhalatory' (more appropriately called Radon Inhalatory) was opened in 1912 in Bad Kreuznach, Germany.

Early environmental measurements of radon were largely confined to outdoor air. The first set of indoor radon measurements which involved 225 houses in Sweden, were published by Hultqvist (1956). This study, which had been initiated by Rolf Sievert, indicated rather high radon levels in a few houses built of alum-shale concrete with a high radium content. Little attention was paid internationally to this finding because it was believed that this was a local Swedish problem.

About 20 years later, larger surveys on indoor radon were made in several countries. Their results are summarised in the reports of UNSCEAR (1977, 1982, 1988, 1993).

These studies reveal the extremely large variation in the radon level in houses, covering a range from a few Bq m^{-3} up to $100,000 \text{ Bq m}^{-3}$. This means that some members of the population are being exposed to indoor radon levels comparable to those of underground uranium miners in the early phase of uranium mining. It was recognised that in most houses with high radon levels, the main source was not the building material but the convective radon influx from the soil. This finding has proved to be of great importance for the planning of efficient intervention techniques to reduce the radon level.

The mean values of the indoor level of radon progeny from these national studies cover a range of the equilibrium equivalent concentration from about $5\text{--}50 \text{ Bq m}^{-3}$. UNSCEAR (1988) assumes a global mean value of about 15 Bq m^{-3} and a mean value of the attributable equivalent dose to the bronchial epithelium of about 15 mSv per year which is about a factor of ten higher than the mean dose of extra-pulmonary tissues from all other natural radiation sources. Consequently, on the basis of a world average, about half the total effective dose from natural radiation sources is due to the inhalation of radon progeny in wellings (ICRP, 1987; UNSCEAR, 1988, 1993).

Estimates of the possible lung cancer risk from indoor exposure to radon progeny are presently based upon the epidemiological data of radon-exposed underground miners (ICRP, 1987; NRC, 1988). As has been pointed out by Stidley and Samet (1993), direct geographical or ecological correlation studies seem to be of low value, due to the strong influence of other confounding factors. One possible exception might be the recently published findings on the lung cancer frequency in Umhausen, a small community in Tyrol, Austria (Ennemoser *et al.*, 1993). Of more promise are case-control studies on indoor radon in several countries (Neuberger, 1992). Preliminary results of some smaller studies of this type have been published (Schoenberg *et al.*, 1990; Pershagen *et al.*, 1992, 1993). Although these preliminary findings indicate a positive correlation between indoor radon levels and lung cancer, in agreement with the range from miner's data, the statistical error range is large.

In summary, perception of the radon problem in houses has three components: (1) a large variation in the range of indoor levels; (2) the relatively high equivalent dose to the sensitive bronchial epithelium; and (3) the convincing epidemiological evidence of an excess risk of lung cancer in radon-exposed miners. A major uncertainty remaining is the evaluation of the carcinogenic effects from indoor exposure to radon and its progeny, including the synergistic influence of smoking. A reliable quantitative answer to this vital question is still awaited.

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