Strahlenschutzkommission

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Radon dose coefficients

Recommendation by the
German Commission on Radiological Protection

Adopted at the 290th meeting of the German Commission on Radiological Protection on 5 and 6 December 2017

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Radon-Dosiskoeffizienten

Empfehlung der Strahlenschutzkommission

Preface

For several years now, protection against the damaging effects of radon has drawn increased attention within radiation protection circles. The new German Radiation Protection Act (StrlSchG) establishes a comprehensive set of rules to protect against radon in workplaces and dwellings.

The conversion of radon exposure quantities into dose quantities is a problem that has not been solved sufficiently to date. The International Commission on Radiological Protection (ICRP) has put forwarded a number of suggestions and recommendations, but so far it has not been possible to reach a definitive agreement on the subject.

In light of this situation, the Federal Ministry for the Environment, Nature Conservation, Building and Nuclear Safety (BMUB) asked the German Commission on Radiological Protection (SSK) to prepare a recommendation as to which dose conversion should be used for public and occupational exposure to radon in Germany over the coming years under the given circumstances.

In order to prepare a draft of the present recommendation, a 'radon dose coefficient' working group of the Committee on Radiation Risk was created. The working group consisted of the following members:

- Prof. Dr. Joachim Breckow (THM University of Applied Sciences, Giessen)
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- Prof. Dr. Claudia Fournier (GSI Helmholtz Centre for Heavy Ion Research, Darmstadt)
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This recommendation provides extensive information about various exposure quantities and units, as well as how to convert them. The reference quantity is generally the cumulative activity concentration, expressed using the SI unit $Bq \cdot h \cdot m^{-3}$. Conversions to other exposure quantities and units are provided in round brackets. There are a few exceptions to this convention where a different reference quantity was used at the time or when the quoted literature was written.

The cut-off date for taking pertinent recommendations and literature into account was 13 November 2017, meaning that any recommendations or literature published after that date have not been taken into account in this recommendation.

Bonn, February 2018

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1 Introduction

For several years now, protection against the damaging effects of radon has drawn increased attention within radiation protection circles. Particular importance has been attached to protection against radon, most notably in Directive 2013/59/Euratom (Euratom 2014) and in the German Radiation Protection Act (StrlSchG) passed in 2017.

The designed legislation generally refers to the expression of airborne radon activity concentration or to a radon exposure level (activity concentration multiplied by time). By way of example, Directive 2013/59/Euratom and the German Radiation Protection Act (StrlSchG) stipulate an activity concentration of 300 $Bq \cdot m^{-3}$ as a reference level for Radon-222 (hereafter referred to as 'radon'[1]) in dwellings and in workplaces. Other than is the case in other areas of radiation protection, this stipulation does not refer to a dose quantity (e. g. to the effective dose). For the time being, corresponding radiation protection measures based on activity concentrations do not require explicit dose values. The reference to radon exposure is grounded in scientific findings, largely from radiation epidemiology, which indicate a link between radon exposure and the induced radiation risk.

Without any reference to dose quantities it is difficult to compare risk assessments and radiation protection provisions on radon exposure to those from other areas of radiation protection. In fact, it is imperative to be able to refer to reference dose quantities for a number of situations and issues. Practices involving NORM residues, for example, require regular effective dose estimates, while radon exposure doses are also essential to the German Federal Government's annual 'Environmental radioactivity and radiation exposure' reports.

Reference to the effective dose is especially required for occupational radon exposure protection rules, hence it is also provided for in Section 130 (2) and Section 130 (3) of the German Radiation Protection Act (StrlSchG). In these cases, the German Radiation Protection Act (StrlSchG) describes how exposures to radon at these workplaces are to be dealt with as planned exposure situations. As a result, the radon activity concentration is to be calculated for certain indoor workplaces for areas to be designated pursuant to Section 121 (1) (1) of the German Radiation Protection Act (StrlSchG), along with an estimate of workers'. In order to transpose Directive 2013/59/Euratom into national law, the German Radiation Protection Act (StrlSchG) prescribes the implementation of occupational protection measures if the effective dose may exceed 6 mSv per calendar year.

In principal, so-called dose coefficients (also known as dose conversion coefficients) can be used to convert an exposure quantity to a dose quantity. Section 95 (13) of the current German Radiation Protection Ordinance (StrlSchV) provides such dose coefficients for occupational radiation protection. The International Commission on Radiological Protection (ICRP) has previously issued a series of radon dose conversion recommendations aimed at effecting a change to the dose coefficients that have been in use to date. However, as there is currently no definitive recommendation or international agreement on the subject, it is unclear as to how things should proceed. In December 2015, a Workshop on Radon Dose Coefficients was held in Bonn involving representatives from the ICRP, the Federal Ministry for the Environment, Nature Conservation, Building and Nuclear Safety (BMUB), the Federal Office for Radiation

[1] An additional radon isotope, Rn-220, may also occur in the air together with its decay products if thorium is present in the environment. This may influence Rn-222 measurements if not corrected to account for the presence of Rn-220. Rn-220 and its decay products can also contribute to the risk of lung cancer, but this recommendation will focus exclusively on Rn-222.

Protection (BfS) and the German Commission on Radiological Protection (SSK) with the aim of investigating the problem (Müller et al. 2016).

Suggestions to stipulate dose coefficients, which in Germany will be enshrined by way of an ordinance, are to be put forward in draft legislation in the near future. When transposing Directive 2013/59/Euratom into national law, the Member States of the EU have the option of using 'approved updates' to the current dose coefficients. In light of this situation, the Federal Ministry for the Environment, Nature Conservation, Building and Nuclear Safety (BMUB) asked the German Commission on Radiological Protection (SSK) to prepare a recommendation as to which dose conversion should be used for public and occupational exposure to radon in Germany over the coming years under the given circumstances. The advisory mandate issued by the Federal Ministry for the Environment, Nature Conservation, Building and Nuclear Safety (BMUB) does not include anticipating the results of the ICRP deliberations. Instead it calls for practicable solutions that can be implemented in the time until a corresponding ICRP recommendation has been issued or an international agreement has been reached.

2 Background

Dose coefficients enable an exposure quantity to be converted into a dose quantity. A radon exposure can be expressed in working level months (WLM), as a cumulative alpha energy concentration in $mJ \cdot h \cdot m^{-3}$, or as an cumulative Rn-222 activity concentration in $MBq \cdot h \cdot m^{-3}$ (further information on exposure quantities are provided in the glossary). The organ equivalent dose of the lung or the effective dose, expressed in mSv, serves as the reference dose quantity. Converting exposure to dose, which is known as dose conversion, relies on a series of assumptions and models, along with a number of parameters and factors such as radiation and tissue weighting factors.

The dose coefficients provided in Section 95(3) of the current version of the German Radiation Protection Ordinance (StrlSchV 2001) are largely based on ICRP 65 (ICRP 1993). There, conversion to an effective dose is based on the dose coefficients 5 mSv/WLM ($F \cdot 7.8$ mSv/(MBq·h·m^{-3})) for workplaces and 4 mSv/WLM ($F \cdot 6.3$ mSv/(MBq·h·m^{-3})) for dwellings[2]. These values include assumptions relating to the exposure time and equilibrium factor F (or radon activity equilibrium equivalent), and are based on the so-called epidemiologic approach which uses a 'dose conversion convention' largely representing the quotient of the lifetime risk of fatal lung cancer per radon exposure and total detriment (total of all detriment-adjusted organ risk coefficients (see Section 3.1). Hereafter, the lifetime risk shall also be referred to as the Lifetime Excess Absolute Risk (LEAR).

By way of example, the above dose coefficient of 5 mSv/WLM ($F \cdot 7.8$ mSv/(MBq·h·m^{-3})) comes from the value for the LEAR of fatal lung cancer per radon exposure among miners of

[2] The historical unit WLM is not an SI unit and should therefore not be used. However, reference will be made to the WLM in this recommendation in order to be able to compare values as WLM is used in the quoted studies and recommendations as the unit for exposure. WLM is still commonly used as a unit in international literature. Conversions are provided in the glossary.

$2.8 \cdot 10^{-4}$/WLM ($F \cdot 4.4 \cdot 10^{-4}$/(MBq·h·m^{-3})) (ICRP 1993) and from the total detriment for the adult population of $5.6 \cdot 10^{-2}$/Sv from ICRP 60 (ICRP 1991) [3]:

$$\frac{2.8 \cdot 10^{-4} / \mathrm{WLM}}{5.6 \cdot 10^{-2} / \mathrm{Sv}} = 5\ \mathrm{mSv} / \mathrm{WLM} = 3.1\ \mathrm{mSv} / (\mathrm{MBq} \cdot \mathrm{h} \cdot \mathrm{m}^{-3}) \qquad (1)$$

These values were updated in ICRP 115 (ICRP 2010) and ICRP 126 (ICRP 2014). The nominal risk coefficient or fatal lung cancer risk[4] is stated as $F \cdot 7.8 \cdot 10^{-4}$/(MBq·h·m^{-3})·($5.0 \cdot 10^{-4}$/WLM) which, when applying the dose conversion convention with the latest risk coefficients from ICRP 103 (ICRP 2007), leads to a dose coefficient of $F \cdot 14.1$ mSv/(MBq·h·m^{-3}) (9 mSv/WLM) for dwellings and $F \cdot 18.8$ mSv/(MBq·h·m^{-3}) (12 mSv/WLM) for workplaces (Müller et al. 2016). This is roughly double the dose coefficient values compared to the values from ICRP 65 (ICRP 1993). An overview of the various dose coefficients is provided in Table 2.1.

Table 2.1:Dose coefficients based on the various ICRP models

Risk coefficient (1/WLM)	Category	Detriment (10^{-2}/Sv)		Dose coefficient F·(mSv/(MBq·h·m^{-3}))	Dose coefficient (mSv/WLM)	Factor of increase
$2.83 \cdot 10^{-4}$ (ICRP 65)	Public	7.3	ICRP 60	6.3	4	–
	Occupational	5.6		7.8	5	–
$5.0 \cdot 10^{-4}$ (ICRP 115)	Public	7.3	ICRP 60	10.9	7	1.7
	Occupational	5.6		14.1	9	1.7
$5.0 \cdot 10^{-4}$ (ICRP 115)	Public	5.7	ICRP 103	14.1	9	2.3
	Occupational	4.2		18.8	12	2.4

The ‘dose conversion convention’ refers to two different situations: The ICRP detriment with data pertaining to cancer incidence for various organs as a result of acute exposure to low LET radiation (data from the Life Span Study (LSS) for atomic bomb survivors with external, largely homogenous exposure), and fatal lung cancer risk as a result of a chronic exposure (with internal, non-homogeneous exposure) to high LET radiation (radon). As a result, it is important to emphasise that the risk due to radon exposure using the LNT model (Dose and Dose Rate Effectiveness Factor (DDREF)= 1) only includes fatal lung cancer, while the detriment from ICRP 60 (ICRP 1991) and ICRP 103 (ICRP 2007) using a DDREF of 2 also includes the induction of non-fatal cancers, the loss of duration and quality of life, and the induction of germline damage. Consequently, this approach somewhat negates the ICRP’s own argumentation in which it stated poor comparability of risks due to radon exposure and the atomic bomb explosion (LSS) as justification for its original preference for the epidemiologic approach over dosimetric models in ICRP 65 (ICRP 1993). In addition, not only a change to the estimated fatal lung cancer risk following radon exposure but also a change to the detriment for all cancer types and hereditary effects among the Japanese population would lead to a

[3] This corresponds with the radon dose conversion coefficient provided in Section 95 (13) of the German Radiation Protection Ordinance (StrlSchV) using $1\ \mathrm{WLM} = \frac{0.64}{F} \cdot \frac{MBq \cdot h}{m^3}$, with an equilibrium factor of F=0.4.

[4] ICRP 115 designates this quantity as ‘nominal risk coefficient’, ‘nominal probability coefficient’ or lifetime excess absolute risk (LEAR). This is the excess absolute risk of dying from lung cancer per exposure. In accordance with standard ICRP practice, the risk coefficients apply to a reference population. This also means that smokers and non-smokers were not evaluated separately.

change in the calculated radon dose coefficient (cf. equation (1)), as was the case following a reassessment in ICRP 115 (ICRP 2010) (ICRP 2014, Müller et al. 2016). However, such a dependency would be difficult to justify from a conceptual perspective if the effective dose were mistakenly understood as being a kind of dose (i. e. a physical and measurable quantity) rather than a quantity designed to represent the risk for radiation protection purposes.

As well as the epidemiologic approach, ICRP 115 (ICRP 2010) mainly pursues the so-called 'dosimetric approach' (cf. Section 3.2.2). Here, various biokinetic models are used with a particular focus on the Human Respiratory Tract Model (HRTM) developed in ICRP 66 (ICRP 1994) and modified in ICRP 130 (ICRP 2015) (see Section 3.2.3). The ICRP also uses such a dosimetric approach as part of a common conceptual framework for all other radionuclides.

The model describes the distribution of radon and its decay products within the human body as a function of time and includes the physical and chemical properties of all involved radionuclides. This enables the calculation of time-dependent activity concentrations for the inhalation pathway for every organ in the human body. When used in conjunction with radiation transport simulations based on Monte Carlo methods, and detailed computer models of the human body, the corresponding absorbed dose can be calculated for every organ. Radiation and tissue weighting factors can then be used to calculate the effective dose associated with the inhalation of radon and its decay products. The result is a radon dose coefficient as an expression of the effective dose for a given radon activity concentration or exposure. In contrast to the epidemiologic approach, a dose coefficient calculated in this manner is not dependent upon the lung cancer risk caused by radon exposure. A link to the radiation risk is thus created, as with any other kind of exposure, solely by way of the damage-weighted nominal risk coefficient, i. e. via the total detriment from ICRP 103 (ICRP 2007).

The dosimetric approach is used in ICRP 115 (ICRP 2010) to calculate dose coefficients ranging from $F \cdot 18.8\ \mathrm{mSv/(MBq \cdot h \cdot m^{-3})}$ (12 mSv/WLM) to $F \cdot 32.8\ \mathrm{mSv/(MBq \cdot h \cdot m^{-3})}$ (21 mSv/WLM) for workplaces (depending on the activity) and $F \cdot 21.9\ \mathrm{mSv/(MBq \cdot h \cdot m^{-3})}$ (14 mSv/WLM) for dwellings. Although the dosimetric approach is generally preferred (ICRP 2010), in a later publication 126 (ICRP 2014) the ICRP refers to the positive match between the epidemiologic and dosimetric approaches ("A dose coefficient of 11 mSv per WLM has been obtained for exposures in mines using the dosimetric approach, essentially the same as obtained by the dose conversion"). The ICRP also emphasises – albeit without any reference to the dose – that risk estimates from miner studies match indoor radon exposure studies in homes (e. g. Darby et al. 2005). The dose conversion convention should not be used in future ICRP recommendations on radon exposure, with preference instead given to biokinetic models like the ones already used for other radionuclides (ICRP 2014). Based on such biokinetic models, Part 3 of the ICRP's Occupational Intakes of Radionuclides (OIR 3) publication series (ICRP 2018) recommends occupationally exposed worker dose coefficients of $F \cdot 16.8\ \mathrm{mSv/(MBq \cdot h \cdot m^{-3})}$ (rounded to 10 mSv/WLM) for miners or indoor workers predominantly with a sedentary activity, and $F \cdot 32.5\ \mathrm{mSv/(MBq \cdot h \cdot m^{-3})}$ (rounded to 20 mSv/WLM) for indoor workers with a predominantly physical activity and in caves accessible to tourists (see Table 2.2).

Table 2.2: Dose coefficients for occupationally exposed workers recommended in OIR 3 (ICRP 2018)

Workplace	Dose coefficient F·(mSv/(MBq·h·m^{-3}))	Dose coefficient (mSv/WLM)	Factor of increase compared to ICRP 65
Mine, indoors (predominantly sedentary activity)	16.8	10	2.0
Indoors (predominantly physical activity), caves accessible to tourists	32.5	20	4.0

Calculation of the dose coefficients in ICRP 115 (ICRP 2010) and ICRP 126 (2014) is not without contradictions and conceptional gaps (Müller et al. 2016). Even more recent data from WISMUT studies (e. g. Kreuzer et al. 2015, 2017) or modified calculations (Müller et al. 2016, Beck 2016, 2017) cannot be aligned with the ICRP estimates without the need for further action as described below.

The present recommendation will investigate the following questions in connection with the state of the art of science regarding a radon dose coefficient estimate:

- Is the assumption of an approximate doubling of the lifetime risk of fatal lung cancer or a nominal risk coefficient for radon-induced lung cancer justified by new literature?
- What is the state of the art of science in terms of equating the risk coefficients due to occupational exposure and due to exposure in dwellings?
- How reliable are the radon dose coefficients calculated using on the epidemiologic and dosimetric approaches? How large are the uncertainties and how good are the matches?

3 Present state of scientific discussion

3.1 Epidemiologic studies[5]

To date, radon exposure only has proven links with the induction of lung cancer as there is no definitive data available linking radon exposure with the induction of other types of cancer. In publication 65 (ICRP 1993), the ICRP argued that epidemiologic studies on humans are best suited to estimating the health effects of radiation exposure. A dosimetric model of the respiratory tract was in fact available at the time (ICRP 1994), but it was deemed to be in need of further development. The ICRP also states an additional problem with a dosimetric approach in that an estimate of doses associated with exposure would have to be backed up by an estimate of risks based on the assumptions in the Life Span Study (LSS). This, in turn, means that all of the uncertainties (SSK 2016) regarding diversity of exposure conditions (short-term exposure involving predominantly gamma radiation vs. long-term exposure involving alpha emitters) would influence the estimate. As a result, the ICRP concluded that the dosimetric model should not be used to evaluate and regulate radon as the use of epidemiologic studies involving radon-exposed persons is more direct and hence better suited than the indirect use of epidemiology data for low LET radiation. Only a few studies with minimal statistical relevance as to the consequences of indoor radon exposure were available at the time, which is why the ICRP

[5] The exposure figures were taken from the original works using WLM as the unit and then converted to SI for the purpose of this recommendation (see glossary).

largely based its estimate on epidemiologic studies involving uranium miners. The available studies provided a nominal risk coefficient for fatal lung cancer per radon exposure of $F \cdot 4.4 \cdot 10^{-4}$/(MBq·h·m^{-3}) or $7.8 \cdot 10^{-5}$/(mJ·h·m^{-3}) ($2.8 \cdot 10^{-4}$/WLM) for occupational exposure. The ICRP saw no indications of a risk coefficient for the general population that deviated beyond the general uncertainty associated with estimates. For the sake of compliance with the provisions set out in ICRP 60 (ICRP 1991), the nominal risk coefficient for fatal lung cancer was converted into a detriment in which both quantities were equated.

As most workers were exposed to radon as well as to other sources of ionising radiation, the ICRP recognised the need to convert exposure to effective dose. This conversion was achieved by directly comparing the detriment values associated with radon exposure and the effective dose. The detriment applicable at the time in ICRP 60 (ICRP 1991) was $5.6 \cdot 10^{-2}$/Sv for workers and $7.3 \cdot 10^{-2}$/Sv for the public. As described above, the detriment per radon exposure was calculated as $F \cdot 4.4 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($2.8 \cdot 10^{-4}$/WLM) for both workers and the public. This means that 1 MBq·h·m^{-3} is equivalent to an effective dose of $F \cdot 7.8$ mSv for workers and $F \cdot 6.3$ mSv for the public. 1 WLM corresponds to approximately 5 mSv for workers and approximately 4 mSv for the public. These conversions are also known as dose conversion conventions.

In ICRP 115 (ICRP 2010), the nominal risk coefficient for fatal lung cancer per exposure of $F \cdot 4.4 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($2.8 \cdot 10^{-4}$/WLM) for occupational exposure was scaled up to $F \cdot 7.8 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($5 \cdot 10^{-4}$/WLM) on account of more recent epidemiologic studies. Together with the detriment values reduced by approximately 25% in ICRP 103 (ICRP 2007), this change leads to an effective dose estimate of $F \cdot 18.8$ mSv/(MBq·h·m^{-3}) (12 mSv/WLM) for workers and $F \cdot 14.1$ mSv/(MBq·h·m^{-3}) (9 mSv/LM) for the public when using the dose conversion convention. These effective dose figures are 2.3 to 2.4 times higher than those provided in ICRP 65 for the same exposure.

For the present recommendation the question of the state of the art of science regarding an estimate for the risk of fatal lung cancer following radon exposure has to be answered and, particularly, clarification is required to determine whether the assumption of an approximate doubling of the nominal risk coefficient for radon-induced lung cancer is justified by literature.

3.1.1 Estimate of a nominal risk coefficient for fatal lung cancer following miners' exposure to radon and its decay products

Seven cohorts with a total of around 31,000 workers were available for the uranium miner risk assessment for ICRP 65 (ICRP 1993). The weighted average Excess Relative Risk (ERR) per exposure was 0.0134 (0.0082-0.0213 95% confidence interval (CI)) per WLM. The exposure rate was not included as a so-called effect modifier[6] in the epidemiologic model used. In the years that followed, several comprehensive analyses were published using updated data for these cohorts and some new cohorts (Lubin et al. 1994, NRC 1999, UNSCEAR 2009). These three publications reported similar ERRs per exposure to one another across all of the cohorts. Surprisingly, however, these reported ERRs per exposure were less than half the ERRs per exposure calculated in ICRP 65 (ICRP 1993). Despite this, the ERR values cannot be directly compared due to the differing underlying characteristics of the cohorts and resulting potential effect modifiers (age reached, follow-up duration, etc.). Instead, lifetime risks have to be estimated using projection models and various assumptions of the exposure situation and background rates of fatal cancer, all of which are associated with high uncertainties.

[6] Effect modifiers are factors which modify the influence of exposure on the effect to be investigated in various subgroups of the population, e. g. age at time of exposure, time passed since exposure, exposure rate, etc.

Here, the ICRP has primarily taken studies into account involving populations with low cumulative exposures, a long follow-up, and good data quality. In general, the estimated ERRs per exposure for cohorts with a low exposure rate are higher than those for cohorts with a high exposure rate (inverse exposure rate effect), as confirmed in an embedded case-control study involving Czech, French and German miners (Hunter et al. 2015). For this reason, in ICRP Publication 115 (ICRP 2010) the ICRP referred to subgroups of the stated cohorts with low exposure for estimates of the LEAR per exposure for fatal lung cancer. Assuming the same exposure scenarios for LEAR estimates as those provided in ICRP 65, values of $F \cdot 8.3 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($5.3 \cdot 10^{-4}$/WLM) were calculated based on the BEIR VI model, and $F \cdot 6.9 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($4.4 \cdot 10^{-4}$/WLM) using the model for the joint French-Czech cohort (Tomášek et al. 2008). The ICRP propounds that the rise in LEAR estimates is partly due to only involving chronic exposures with a low dose rate, and partly because of the increased estimated ERRs per exposure in more recent studies (that were, however, not presented in the document). Other studies and analyses showed that LEAR estimates are sensitive to variations in the selected models and the assumed background rates. As a result, values of between $F \cdot 4.7 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($3 \cdot 10^{-4}$/WLM) and $F \cdot 10.9 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($7 \cdot 10^{-4}$/WLM) were reported (ICRP 2010). Based on these considerations, the ICRP issued a recommendation in ICRP Publication 115 (ICRP 2010) to use a nominal risk coefficient of $F \cdot 7.8 \cdot 10^{-4}$/(MBq·h·m^{-3}) ($5 \cdot 10^{-4}$/WLM) for fatal lung cancer, which is approximately double the value stated in ICRP Publication 65 (ICRP 1993). In view of the major uncertainties, the SSK does not consider it necessary based on the available data to use the above ICRP assumptions of an increased nominal risk coefficient. Consistency is a valuable commodity when it comes to radiological protection, meaning that changes should only be introduced if there is very strong evidence available to back up new findings.

The total detriment in ICRP 103 (ICRP 2007) and the LEAR for fatal lung cancer in ICRP 115 (ICRP 2010) are each calculated in several steps and depend on a range of assumptions and parameters that influence the result and hence the total uncertainty. These assumptions and parameters include: Selection of the risk model and modifying factors (age, sex, time since exposure), size and validity of the selected study population, observed disease (cancer and hereditary diseases vs. lung cancer; incidence vs. fatality), selection of the underlying reference population, assumptions regarding lifetime and exposure scenario, selection of the weighting factors for damage-weighted detriment (lethality factor, factor for reduced quality of life, factor for the relative loss of life expectancy), DDREF and assumptions regarding the interaction of radon and smoking for risk transfer (multiplicative or additive).

It should be noted that the evaluations presented in ICRP 115 (ICRP 2010) did not include the world's largest available cohort of uranium miners, the WISMUT cohort. Kreuzer et al. (2015, 2017) investigated the German WISMUT uranium miner cohort by performing comprehensive analyses into the radon-related excess relative lung cancer risk at low exposures and low exposure rates that take account factors that influence this risk (time since exposure, age at time of exposure, smoker/non-smoker). No LEARs have been published for the WISMUT cohort to date. Provisional results indicate an approximate 50% reduction in LEAR values compared to those calculated in ICRP 115 (ICPR 2010). However, the anticipated confidence intervals are large, meaning that some overlapping is expected (Müller et al. 2016, Kreuzer et al. 2017). Given these circumstances, the SSK suggests awaiting an evaluation of the WISMUT study that is comparable with other miner studies before stipulating nominal risk coefficients.

3.1.2 Estimate of a nominal risk coefficient for fatal lung cancer following exposure of the resident population to radon and its decay products

There are several reasons why it is difficult to compare risk coefficients between uranium miners and the resident population (different epidemiologic study types, exposure for a fixed period of time vs. ongoing exposure, etc.). ICRP 65 (ICRP 1993) discusses several factors, such as age distribution, particle sizes and the binding of radon progeny to dust particles, which may influence the risk coefficient for miners and the resident population at the same activity concentration. Due to the uncertainties associated with this, however, the ICRP stated in ICRP 65 (ICRP 1993) that there is no justification for stipulating a separate risk coefficient for the resident population. ICRP 115 (ICRP 2010) reviewed a series of epidemiologic studies released since the late 1980ies to estimate the risk coefficients for the radon-exposed resident population, despite the fact that the studies are of limited statistical value on their own. Pooled studies were also conducted using data from Europe (Darby et al. 2005), North America (Krewski et al. 2005, 2006), and China (Lubin et al. 2004). In contrast to occupational exposures, exposure among the resident population spans an entire lifetime, meaning that it can vary significantly over time. In the aforementioned studies, weighted average activity concentrations were calculated for the participants in every dwelling that was resided in over a period of 5 to 34 years or 5 to 30 years, respectively, prior to diagnosis. It should be noted that the age reached and the total period of residential radon exposure in these studies did not have any major influence on the calculated risk per activity concentration. The calculated risk estimates were therefore expressed per activity concentration rather than per exposure (i. e. without any reference to the duration of exposure).

The stated pooled studies showed very similar and statistically compatible relative risk estimates (ERR per activity concentration) of 0.08 (0.03-0.16 95% CI), 0.11 (0.00-0.28 95% CI) and 0.13 (0.01-0.36 95% CI)/(100 Bq·m^{-3}) for Europe, North America, and China, respectively. The joint estimate value was 0.09/(100 Bq·m^{-3}) (UNSCEAR 2009). Taking account of the uncertainties of the exposure estimate led to a rise in the risk estimate values. In the European study this led to an ERR per activity concentration of 0.16/(100 Bq·m^{-3}) when taking account of uncertainties in the radon activity measurements (Darby et al. 2005).

In ICRP 115 (ICRP 2010) the IRCP states that the risk estimates for fatal lung cancer among miners and persons exposed in dwellings are comparable and offers a discussion on a common dose coefficient for occupational and residential exposure (Müller et al. 2016). However, it should be noted that the comparisons are based on a series of assumptions largely pertaining to exposure. In the miner studies, the exposure durations are generally short (e. g. an average of 5.7 years for the 11 miners cohort (NRC 1999)), while residential exposure is far more prolonged with a typical age of > 50 at the time of lung cancer diagnosis (Darby et al. 2006). Exposures that took place more than 35 years in the past were not collected during the indoor studies and were classed as being insignificant. This is based on the results of miner studies which consistently demonstrated a significant decrease in the risk of lung cancer due to radon over an increasing period of time since exposure, and this includes low doses (UNSCEAR 2009). Here, it is unclear whether the assumed irrelevance of exposures further in the past apply to residential exposure in much the same way as they apply to miner exposure. The ICRP investigated this in ICRP 115 (ICRP 2010) by estimating an ERR of 1.2/(100 WLM) for a 30-year residential exposure of 100 Bq·m^{-3} and an excess relative risk of 0.16/(100 Bq·m^{-3}), which is within the ERR range seen in various studies involving miners with comparatively low exposure (0.8-2.7/(100 WLM)). It is evident that other assumptions regarding the duration of relevant exposure would lead to other results.

Chen et al. (2017) also compared lifetime risks, calculated using the risk coefficients from ICRP 115 (ICRP 2010), with various models from epidemiologic pooling analyses on

residential radon exposure, and concluded that the values from ICRP 115 (ICRP 2010) match the epidemiologic studies better than the previous values from ICRP 65 (ICRP 1993). However, the assumption of a relevant exposure here is also only implied for the period of 5 to 30 years and 5 to 34 years, respectively, prior to diagnosis.

The SSK notes that the uncertainties in the epidemiologic studies and the many assumptions required to compare the risks between miners and the resident population do not currently enable a definitive statement to be made regarding an equality or a difference in risk coefficients with the same activity concentration and duration of exposure. For this reason, the risk coefficients for miners and the general public, including workers who have light physical duties, could indeed differ in radon-prone areas.

3.2 Biokinetic and dosimetric studies

Radon gas and its decay products/progenies, which are also present in the air and in water, are incorporated via the epithelia (lung, gastrointestinal tract, skin). The radon progenies adhere (adsorb) to the epithelia, which diffuse the radon around the body. Radon is also cleared from the body by way of diffusion and then exhalation, while radon progenies leave the body by way of mechanical transport (lung) or excretion.

The stated organs as well as all of the organs that come into contact with radon and its progenies upon incorporation or clearance are potentially at risk of tumour induction. In order to estimate this risk, the effective dose is required, in turn requiring knowledge of the dose for the entire organism and individual organs. As direct measurements on human test subjects are difficult and partially impossible for ethical and logistical reasons, radiological protection uses models and individual measurements of the required parameters to provide an estimate of the effective dose.

3.2.1 Dose conversion: Dosimetric approach

The dosimetric approach used to calculate dose coefficients follows the generic method for calculating the dose in internal dosimetry: Biokinetic models describe how radionuclides behave in the body over time following their incorporation, diffusion around the body, and excretion. These models can then be used to calculate the number of nuclear transformations in individual parts of the body. Dosimetric models are used to calculate the mean absorbed dose in various body tissues due to the energy released during nuclear transformations in a certain part of the body.

Multiplying the respective absorbed dose with the radiation weighting factors enables the organ dose values to be calculated. The effective dose is calculated by totalling the sex-averaged organ dose values which are then multiplied by the respective tissue weighting factor. The tissue weighting factor of 0.12 for the lung is unchanged (since its introduction in ICRP 26 (ICRP 1977)).

The ICRP up to now applied the dosimetric approach to all radionuclides, excluding radon isotopes.

A deviation for radon is where the effective dose is not only expressed per incorporated activity (Sv/Bq) (separately for radon gas and short-lived decay products), but also per exposure (mSv/WLM or mSv/(Bq·h·m^{-3})) for radon including its decay products.

Part 3 of the ICRP publication series ‘Occupational Intakes of Radionuclides’ (ICRP 2018) states that the effective dose upon inhalation of radon is more than 95% determined by the lung dose and more than 95% by the short-lived decay products. This reduces the relevance of the biokinetic model for radon gas in the lung.

For this reason, the latest ICRP respiratory tract model, particularly for particle inhalation, is described in more detail below.

3.2.2 Human Respiratory Tract Model (ICRP)

The respiratory tract models developed by the ICRP include the following:

- Deposition of particles in various respiratory tract regions
- Mechanical transport to the upper respiratory tract with subsequent transfer to the digestive tract as a result of swallowing
- Mechanical transport to the regional lymph nodes, and
- Absorption into the bloodstream

These mechanisms were already provided in the lung model in ICRP 30 (ICRP 1979) (and, albeit in a very rudimentary way, in Publication 2 (ICRP 1959)) and revised significantly in the Human Respiratory Tract Model (HRTM) in Publication 66 (ICRP 1994). This model was amended in the first volume of the ICRP OIR series, namely in Publication 130 (ICRP 2015). Below is a description of the current HRTM from ICRP 130 (ICRP 2015).

3.2.2.1 Respiratory tract regions

The extrathoracic and thoracic regions of the respiratory tract are investigated here. The extrathoracic region is divided into the anterior nasal passage, ET_1, and the remaining extrathoracic region (posterior nasal passage, pharynx and larynx), ET_2. The thoracic region is divided into the bronchial region (trachea and bronchi) up to airway generation 8, BB, the bronchiolar region, generations 9 to 15, bb, and the alveolar-interstitial region, AI, which is the part of the lung in which air is exchanged. The extrathoracic region of the respiratory tract (ET_1 and ET_2) correspond with the nasopharynx (N-P) in the ICRP 30 (ICRP 1979) lung model, while the bronchial and bronchiolar regions (BB and bb) correspond with the tracheo-bronchial system (T-B), and the alveolar-interstitial region (AI) aligns with the pulmonary system (P).

3.2.2.2 Deposition

Deposition of particles in parts of the respiratory tract depends upon:

- The physical properties of the particles (average size and size distribution, density and shape of the particles), and
- The person's breathing patterns and age.

In terms of physical particle properties, deposition is particularly influenced by particle size. Larger particle sizes (average ≥ 0.3 µm) are expressed by way of their activity median aerodynamic diameter (AMAD), while smaller particles (< 0.3 µm) are expressed using their activity median thermodynamic diameter (AMTD).

Two additional parameters are of interest for radon decay products: the equilibrium factor *F*, a measure of the relationship between radon gas activity and its decay products, and the so-called unattached fraction, i. e. the fraction of radon progeny that does not attach itself to particles in the surrounding air. OIR 3 (ICRP 2018) assumed an equilibrium factor of 0.4 for indoors and caves accessible to tourists, and an equilibrium factor of 0.2 for ventilated mines. The value for indoors corresponds with the assumptions set out in ICRP 65 (ICRP 1993) and provided by UNSCEAR (UN 2000). The fraction of unattached radon progeny is assumed to be 0.08 for indoors (measurements involved a large range of between 0.03 and 0.2, or also more than 0.4), 0.01 for mines, and 0.15 for caves accessible to tourists. AMTDs of between 0.5 nm and 1.7 nm were measured for this fraction (ICRP 2018). An AMTD of 1 nm is assumed in OIR 3. The

particles to which the radon decay products are attached may exhibit a trimodal size distribution (Porstendörfer 2001), i. e. particles in nucleation mode (ultrafine particles nucleated by way of condensation processes and chemical reactions), in accumulation mode (fine particles accumulated by way of various processes), and in coarse mode (coarse particles that may arise in particular by way of mechanical processes). In OIR 3, only the accumulation mode with an AMTD of 250 nm is assumed for mines (although measurement results depend on the mine and exhibit large variability). The same applies to caves accessible to tourists where only the accumulation mode with an AMTD of 200 nm is considered. For indoors, OIR 3 provides an AMTD of 30 nm (15 nm to 40 nm was measured (Porstendörfer 2001)) for the nucleation mode (20%), and 250 nm (100 nm to 400 nm was measured (ICRP 2018)) for the accumulation mode (80%). However, according to the assumptions in OIR 3, inhalation leads to hygroscopic growth, meaning that the indoor values immediately increase by a factor of 2. No coarse particles (coarse mode) were assumed to be present in general. OIR 3 also contains studies into particle properties at waterworks and spas, but no reference levels were stipulated and no dose calculations were performed.

Respiration measurements involving partially radioactively marked particles were performed on human test subjects and in animal experiments to investigate how deposition is dependent upon particle size. Corresponding measurements in human test subjects are now likely to have been discontinued for ethical reasons. The present works showed a discontinuous curve progression, i. e. almost full deposition was observed for particles with a diameter of > 5 µm and minimal deposition for particles between 0.1 µm and 0.8 µm in size (summarised in Figs. 3 and 4 in Stuart 1984).

It is important in the deposition of radon decay products that deposition of particles less than 0.1 µm in size increases again, as demonstrated in other studies, primarily among miners at the Colorado Plateau (George and Breslin 1969, Holleman et al. 1969, Harley and Pasternack 1972, Stuart et al. 1970). For technical reasons, it is difficult to perform experiments to determine deposition in the pulmonary and alveolar-interstitial regions of the respiratory tract (Stuart 1984), and there is only very little experimental data available for humans in terms of deposition of particles the size of radon decay products (Wilson and La Mer 1948, Van Wijk and Patterson 1940, Lippmann and Albert 1969, Brown et al. 1950, Gessner et al. 1949), and this data only involves particles without any chemical bonding and does not include radioactive isotopes. These results showed that particles < 0.1 µm are primarily deposited in the pulmonary system (Beeckmans 1965), which negates the discontinuous dependency of deposition on particle size as described above.

In terms of the dependence of deposition of radon decay products on other parameters, such as attachment to carrier substances, studies involving human test subjects (Harley and Pasternack 1972, Jacobi 1964, Parker 1969) showed that under certain conditions, a high dose deposition is to be expected at the branches of the larger tracheo-bronchial airways (Nelson et al. 1971). These results are consistent with observations involving uranium miners who exhibited an increased incidence of bronchogenic carcinomas of the lung (Archer et al. 1973, Saccomanno et al. 1996). More recent epidemiologic studies into tumour type discuss an increased incidence of small cell lung cancer (SCLC) compared to bronchogenic carcinomas (Kreuzer et al. 2000, Taeger et al. 2006). Uncertainties due to heterogeneity within a tumour must also be taken into consideration when assigning tumours to a certain type of tumour. Dependency of deposition on respiratory parameters (aerodynamic diameter, respiratory volume, respiratory frequency) was also investigated using human test subjects, but without using radioactive isotopes (Lippmann and Albert 1969, Giacomelli-Maltoni et al. 1972, Foord et al. 1978, Chan and Lippmann 1980, Heyder et al. 1971, George and Breslin 1969, Holleman et al. 1969, Heyder et al. 1975, Heyder et al. 1980).

The deposition models developed on the basis of these works, and the stipulated reference particle properties for the short-lived radon decay products were used to calculate the following deposition values (see Table 3.1):

Table 3.1: Deposition values from OIR 3 (ICRP 2018) for radon decay products (ET_1 is the anterior nasal passage, ET_2 the posterior nasal passage, pharynx and larynx, BB the bronchial region, bb the bronchiolar region, and AI the alveolar-interstitial region)

Properties of the radon decay products	Deposition in the respiratory tract regions (%)					
	ET_1	ET_2	BB	bb	AI	Total
Unattached	51.91	27.95	7.93	10.05	0.59	98.43
Attached to particles in nucleation mode (indoors)	3.85	2.07	0.93	6.53	27.90	41.28
Attached to particles in accumulation mode (indoors)	10.68	5.75	0.60	1.43	9.05	27.51
Attached to particles in accumulation mode (mines)	3.16	1.70	0.41	2.16	9.94	17.37
Attached to particles in accumulation mode (caves)	3.42	1.84	0.47	2.61	11.94	20.28

When it comes to inhalation of gases, deposition depends upon chemical rather than physical properties, i. e. the extent to which the gases dissolve or react with the airway tissues. Neither is the case with noble gases (e. g. radon), and deposition also does not occur. In this case it is assumed that the activity concentration in the air found in the airways matches that of the air in the surroundings. Inhaled radon gas is partially absorbed into the arterial blood and transported to other body tissues, from where it is then transported back to the respiratory tract by way of venous blood and partially exhaled and partially reabsorbed into the arterial blood.

3.2.2.3 Mechanical transport

According to the model in ICRP 130 (ICRP 2015) (see Fig. 3.1 below), mechanical processes remove the deposited substance from the anterior nasal passage with a biological half-life of eight hours; around 2/3 is transferred to the following region of the extrathoracic respiratory tract (ET_2), with around 1/3 being removed by way of respiratory air.

Mucociliary clearance transports the deposited substance from all of the thoracic respiratory tract regions to the extrathoracic region (ET_2 compartment), from where it is transported to the gastrointestinal tract with a biological half-life of 10 minutes. This mucociliary clearance is slower in the lower lung region than in the higher lung region: The biological half-lives are 230 days from AI to bb, 3.5 days from bb to BB, and 1.7 hours from BB to ET_2.

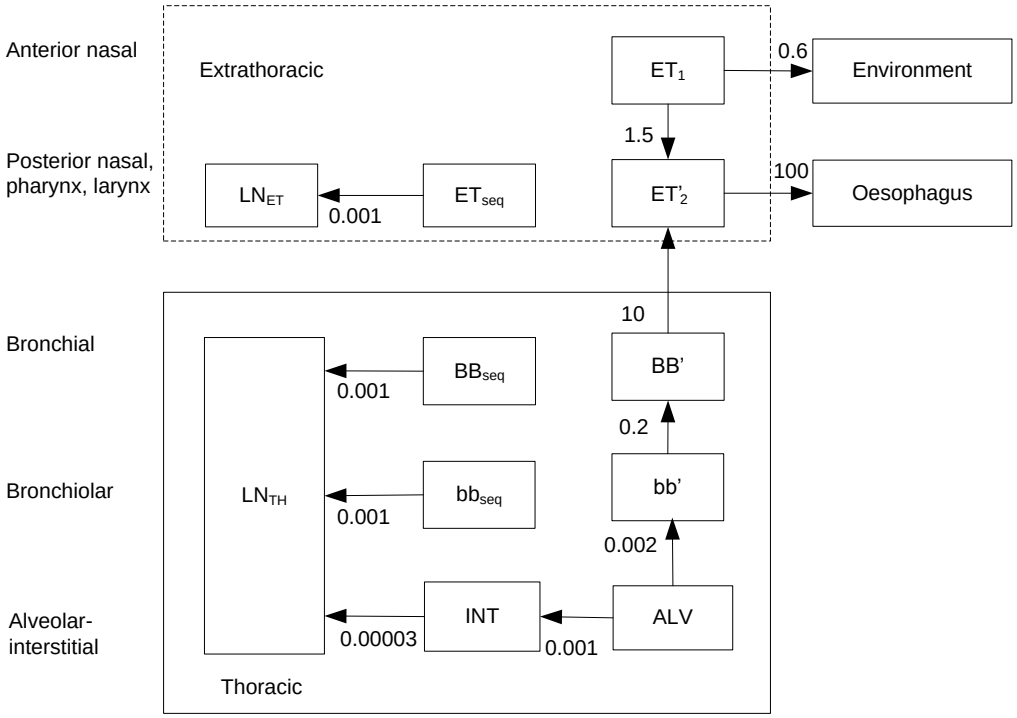

Fig. 3.1: Mechanical transport in the respiratory tract as described in ICRP 130 (ICRP 2015) with transport rates in d^{-1}. Deposition in the anterior nasal passage and alveolar-interstitial region takes place in the compartments ET_1 and ALV; deposition in the posterior nasal passage (including the pharynx and larynx), the bronchial region and the bronchiolar region takes place in the compartments ET'_2, BB' and bb' as well as in the compartments ET_{seq}, BB_{seq} and bb_{seq} (0.2% each) within the walls of the airways. INT is used to designate the interstitium in the alveolar-interstitial region, while LN_{ET} and LN_{TH} are used for the extrathoracic and thoracic lymph nodes.

Mechanical transport to the regional lymph nodes also occurs from every region except the anterior nasal passage. Other than the alveolar-interstitial region, it is assumed that 0.2% of the deposited substance is transported to the lymph nodes with a biological half-life of 1.9 years. With the alveolar-interstitial region, it is assumed that 1/3 of the deposited substance is transported to the lymph nodes with a biological half-life in excess of 600 years.

For short-lived radon decay products, only mucociliar clearance from the upper airways (bronchial region) and swallowing from the extrathoracic region are of importance. The other processes are so slow that it can only be roughly assumed that the deposited radon decay products decay at the place of deposition and that the quantitative uncertainties of the corresponding transfer parameters are of no importance to the short-lived radon decay products.

Multiple works have investigated the physiological bases of mucociliary clearance (summarised in Stuart 1984). Studies on human test subjects into mucociliar clearance following inhalation of radioactive particles (Lippmann 1977, Albert et al. 1973, Camner et al. 1971, Lourenço et al. 1972, Yeates et al. 1975, Clarke and Pavia 1980, Goodman et al. 1978, Wood et al. 1975) have shown a period of 3 to 24 hours for excretion which depends upon particle size, respiration frequency and airway anatomy. Mucociliary clearance was reported to subside with increasing age (Wong et al. 1977) and during sleep (Clarke et al. 1980), but this does not depend upon sex (Wong et al. 1977) or stature (Bateman et al. 1978).

3.2.2.4 Absorption

In the HRTM, absorption into the blood from every respiratory tract region except the anterior nasal passage takes place at the same transfer rates, generally with a fast and slow component. Absorption rates depend on the substance's solubility, in turn making them dependent upon the element and compound. Here, delayed absorption with temporary retention (known as a bound state) is also possible in the walls of the respiratory tract. The ICRP has defined default rates F, M and S (for fast, moderate and slow absorption) without taking the bound state into account, and also provides compound-specific parameters as and where the requisite knowledge is available.

With short-lived radon decay products, only a fast component with a biological half-life of 5.5 hours is assumed for polonium isotopes and 17 hours for bismuth isotopes without any consideration of the bound state. Fast absorption with a biological half-life of 10 minutes is assumed for 10% of the Pb-214, while the remainder is absorbed with a biological half-life of 9.8 hours. For 50% of the bound state, retention in the walls of the airways is assumed to have a biological half-life of 9.8 hours.

The fast fraction of Pb-214, which is deposited on the walls of the respiratory tract in a bound state, is of particular importance to the radon dose, with the alpha radiation emitted from the extremely short-lived Po-214 from the decay chain causing a significant dose in particular. The parameters used were derived by evaluating studies involving (a few) people and the Rn-220 decay product, Pb-212, where activity in the blood, excretions and, in part, lung retention was measured. Here, OIR 3 stated a much lower fraction that achieves the bound state than that observed in the evaluated studies, although the data used (only a few test subjects) imposes a great deal of uncertainty regarding the results.

3.2.2.5 Dosimetry

The average dose in radiosensitive cells is calculated for the various regions of the respiratory tract. Radiosensitive cells are distributed across the entire alveolar-interstitial region and regional lymph nodes, while in other areas they are assumed to be in a tissue layer at a certain depth within the airways: In the extrathoracic region these cell layers are assumed to be the basal cells at a depth of 40 µm to 50 µm, in the bronchial region they are assumed to be the secretory cells (10 µm to 40 µm) and the basal cells (35 µm to 50 µm), and in the bronchiolar region they are assumed to be the secretory cells (4 µm to 12 µm). The dose in the bronchial region is calculated as an arithmetic mean of the dose values for the secretory and basal cells.

The dose for the extrathoracic region is calculated as a weighted mean from the dose values for ET_1 (0.001) and ET_2 (0.999), while the lung dose is calculated as an arithmetic mean from the dose values for the bronchial region, the bronchiolar region and the alveolar region. The dose for the thoracic and extrathoracic lymph nodes are included in the dose calculation for the lymphatic tissue (0.08 in each case).

The effective dose upon inhalation of radon is determined in particular by the lung dose received from alpha radiation. This means that the radiation and tissue weighting factors are applied directly. The tissue weighting factor of 0.12 for the lung is unchanged from the very beginning (the effective dose concept was first introduced in ICRP 26 (ICRP 1077)). The radiation weighting factor for alpha radiation also remains unchanged at 20 and is generally used in radiation protection to assess the stochastic effects of radiation.

The definition of radiosensitive cells, for which dose values are calculated, and their depth within the tissue are of key importance to the dose received from alpha radiation. The definition and depth of radiosensitive cells have been defined in ICRP 66 (ICRP 1994) and are generally applied by the ICRP.

As the dose is not distributed homogeneously within the respiratory tract (the dose to the bronchial region and bronchioles is greater than that of the alveolar region), the method used to calculate the lung dose from the regional dose values for the bronchial region, bronchioles and alveolar region has a major impact. Examination of the (mass-weighted) total average dose, for example, would show that the lung dose for radon is lower than the dose from the OIR report, which is the arithmetic mean of the single partial dose values.

3.2.3 Effective dose for radon according to the ICRP dosimetric concept

OIR 3 (ICRP 2018) provides dose coefficients of $F \cdot 19$ mSv/(MBq·h·m^{-3}) (12 mSv/WLM) for mines, $F \cdot 33$ mSv/(MBq·h·m^{-3}) (20 mSv/WLM) for indoor workplaces and $F \cdot 37$ mSv/(MBq·h·m^{-3}) (24 mSv/WLM) for caves accessible to the public, all of which were calculated using the dosimetric concept. The values for miners are lower, in particular due to the ventilation measures providing for a lower equilibrium factor; the value is highest for workers in caves accessible to tourists because a larger unattached fraction is assumed. Indoor workers with sedentary activity are quoted as having a value of $F \cdot 22$ mSv/(MBq·h·m^{-3}) (14 mSv/WLM) (Harrison and Marsh 2012), while a value of $F \cdot 16.8$ mSv/(MBq·h·m^{-3}) (rounded to 10 mSv/WLM) is recommended for mines and indoor workplaces (with a predominantly sedentary activity). A value of $F \cdot 32.5$ mSv/(MBq·h·m^{-3}) (rounded to 20 mSv/WLM) is recommended for indoor workplaces involving physical activity and workers in caves accessible to tourists (cf. Table 2.2).

The ICRP will derive and publish dose coefficients for individual members of the public in the near future in its Environmental Intakes of Radionuclides (EIR) publication series. Deposition in the lung in particular will differ for individual members of the public due to their different respiratory behaviour and possibly different ages when compared to deposition in the lung for occupationally exposed persons. There are currently no models or calculations available for radon dose coefficients in the EIR publications. A publication by Marsh and Bailey (Marsh and Bailey 2013) is quoted in part 3 of the OIR series (ICRP 2018) as it provides an effective dose of $F \cdot 20$ mSv/(MBq·h·m^{-3}) (13 mSv/WLM) for individual adult members of the public in dwellings.

In general, the ICRP provides dose coefficients for reference persons without taking individual characteristics and behaviour patterns into account. There are no separate dose coefficients for smokers and non-smokers, for example. However, the respiratory tract model in ICRP 66 (ICRP 1994) does provide modifying factors that are recommended for special situations (influence due to smoking, air pollution, respiratory diseases). Such situations slow down clearance from the alveolar region to the bronchiolar region and from the bronchial region to the extrathoracic region of the respiratory tract by up to a factor of 2. In the alveolar region, this slowdown in mucociliary clearance due to smoking has no effect on the short-lived radon decay products due to the extremely long biological half-life when compared to the physical half-lives. The effective half-life for the individual decay products only increases by up to 12% for mucociliar clearance from the bronchial region to the extrathoracic region. The result of a study where total deposition of particles is 1 µm to 5 µm higher among smokers (Lippmann 1977) is not taken into account in the modifying factors in ICRP 66.

The ICRP dose coefficients for radon calculated using the dosimetric concept are values for reference persons with standardised environmental conditions. As well as individual variability and variability of environmental conditions, the reference levels include a number of uncertainties. Potential sources of these uncertainties include:

- Deposition values of the radon decay products in the lung regions directly related to the dose

- Mechanical transport rates of deposited radon decay products for mucociliar clearance from the bronchial region to the extrathoracic region, and clearance from the extrathoracic region to the gastrointestinal tract
- Absorption rates from the respiratory tract to the blood, particularly the Pb-214 fraction retained in the walls of the airways in a bound state, and
- Definition and location of radiosensitive cells of particular relevance in terms of alpha radiation.

The dose estimate is largely influenced by the deposition values, which have been verified to a relatively large extent by way of human studies, as well as the Pb-214 fraction attached to the walls of the airways in a bound state and the location of the radiosensitive cells. The data available for the bound state parameters in particular is quite poor, meaning that these parameters may have a larger uncertainty. Ultimately, additional radiation protection parameters other than physical dosimetry play a crucial part in calculating the effective dose, particularly the radiation weighting factor for alpha radiation, the tissue weighting factor for the lung, and the method used to calculate a lung dose based on partial lung dose values.

Potential uncertainties in the kinetics of radon gas and dose values away from the lung (e. g. in the lymph nodes) and their potential influence on the uncertainty of the effective dose coefficient for radon were not evaluated here.

3.3 The ICRP concept

Even though the ICRP intends to cease using the dose conversion convention in its epidemiologic approach in the future, it uses the good agreement with the dosimetric approach as an argument in favour of consistency of the dose coefficients developed therefrom (ICRP 2010, 2014). Due to the major uncertainties, and as a result of more recent epidemiologic studies, doubts have been raised as to the good agreement between the two approaches (e. g. Müller et al. 2016). But even if this were not the case, the dose conversion convention has a number of significant conceptual short comings, meaning that it can only be used as a supporting argument to a very limited extent. One of the more curious and implausible consequences of using this approach stems from the following simple consideration: The dose coefficient for radon (densely ionising radiation) is linked to the risk from radon exposure (cf. equation (1)) across the total detriment for all organs (calculated for sparsely ionising radiation). Therefore, if risk estimates for *other* types of cancer (other than lung cancer) and for *other* types of radiation (e. g. for sparely ionising radiation) were to change at some point in the future, the radon dose would, in turn, also change. It is hard to see why the effective dose due to radon exposure should change if, for example, the risk estimate for colon cancer due to x-rays were modified. However, if the effective dose is to be changed in any way other than by radon reasoning and if it were to be merely seen as a measure of the detriment due to external (low LET) radiation, the radon risk would have to be ‘updated’ and adapted as well.

Even when focussing solely on the lung, this implausibility remains the case. The detriment for the lung is based on the lung dose, making it independent of the type of radiation involved. In view of this, if the detriment or risk coefficient for the lung changes due to a change in the risk estimates for sparsely ionising radiation, the estimator for the radon risk also changes, expressed by way of the effective dose, even if the radon dose coefficient remains unchanged. This characteristic applies to both the epidemiologic and the dosimetric approaches (Müller et al. 2016).

The effective dose concept is based on the quantitative comparability of the risk contributions of various organs from various exposure conditions. This concept should not make an exception

for radon exposure. A real radiation protection situation is characterised by a certain individual exposure condition resulting from the properties of the radiation field and its influence on the exposed body, and is expressed by way of the values assigned to the organ doses. The tissue weighting factors are averaged over sex, age, and a number of other demographics (e. g. ethnicity, social and economic background, lifestyle, smoker or non-smoker, genetic predisposition). The effective dose is therefore a reference dose that is in fact assigned to a single person, but does not constitute any individual risk. It merely serves radiation protection purposes and is unsuitable for performing risk estimates for individuals or certain groups of people (SSK 2005).

As a result, there are limits as to how the effective dose can be applied. Although the concept was predominantly developed for partial-body exposures, it gives rise to certain restrictions if only individual organs are affected, as is the case with radon exposure. As the sum of the various tissue weighting factors is 1, it can be assumed that incorrect assumptions for the individual weighting factors would increasingly cancel each other out the more individual organs are affected by the exposure (and the closer to 1 arrived at by summation). On the other hand, internal compensation is not possible if only one individual organ is affected. In this case, the risk due to the effective dose is only insufficiently represented (ICRP 2007), meaning that the reference to the respective organ dose is decisive. This is one of the reasons why the ICRP now prefers the dosimetric approach over the epidemiologic approach.

In 2014, the SSK issued a recommendation where it called for an amendment and possibly also the discontinuation of the dose and dose rate effectiveness factor (DDREF)[7] (SSK 2014). Such a discontinuation would affect all organ risk coefficients and all organ detriments, including those of the lung. As the detriment is independent of the type of radiation and the exposure condition, its modification would mean a change to the detriment and to the risk per lung dose even due to radon exposure. Therefore, if both the DDREF and radon dose coefficient were to change, it has to be made sure that this is made in line with epidemiologic data.

3.4 Current developments

UNSCEAR is currently performing a detailed evaluation of a number of new scientific publications covering the risk following radon exposure in order to better understand combined effects, e. g. of smoking, and to derive the uncertainties in the epidemiologic and dosimetric models along with potential amendments to the radon dose coefficients. It will be a few years before a final report is published.

The ICRP is currently immersed in the biokinetic and dosimetric models for radon, its decay products and more recent epidemiologic studies. This recommendation takes account of the draft ICRP 137 publication (ICRP 2018).

In order to gain an overview of how other European countries are implementing Directive 2013/59/Euratom, particularly the question of new radon dose coefficients on the basis of ICRP 103 (ICRP 2007) and ICRP 115 (ICRP 2010), the SSK surveyed experts from 15 countries, who are involved in implementing the Directive in their respective country, about the current situation and progress of discussions into the introduction of new dose coefficients in their respective country.

[7] Within the scope of this amendment, the SSK also recommends updating all other parameters used for the detriment, i. e. specification of radiation damage, to bring them in line with the state of the art in science (SSK 2014).

The responses failed to provide a clear picture of current progress made and the next steps to be taken in the individual countries. Discussions as to whether and how the results of ICRP 115 (ICRP 2010) and ICRP 103 (ICRP 2007) should be taken into consideration are largely incomplete or have just started. Most of the experts surveyed assume that the existing models will be amended to reflect the new ICRP recommendations. However, only very few countries have actually proceeded to do this, as is the case in Switzerland where the revised radiation protection ordinance (federal level) introduced a reference level of 300 $Bq \cdot m^{-3}$ without providing a radon dose coefficient to calculate the dose to the population. To date, Switzerland has only provisionally introduced a radon dose coefficient of $1.87 \cdot 10^{-5}$ $mSv/(Bq \cdot h \cdot m^{-3})$ (or 12 mSv/WLM) for an equilibrium factor F of 1 in the subordinate dosimetry ordinance (departmental level) for use in calculating the workplace dose. This value depends on the situation and is either determined by measuring the weighting factor, or by applying ICRP recommendations. As a result, this value can be easily amended at a later time to accommodate a specific ICRP recommendation.

In most other countries, the radon dose coefficient stipulations have still been retained, with the introduction of new coefficients contingent upon an explicit ICRP recommendation. The dose coefficients in this recommendation should be subsequently introduced in corresponding ordinances and regulations. The stipulation of a certain value should generally be enshrined in subordinate rather than superordinate ordinances/directives, as is the case in Switzerland, so as to make it easier to amend the dose coefficients at a later date.

Some of the experts surveyed suggested lobbying the Group of Experts appointed by the European Commission as per Article 31 of the Euratom Treaty with the aim of persuading them to impose standard radon dose coefficients upon every Euratom member state.

4 Evaluation of discussions

In its most recent publications, the ICRP generally recommends the use of the dosimetric approach for radon and radon decay products in the same way as for all other radionuclides of radiological importance. In this context, it mentions the good agreement between estimates based on both the epidemiologic approach and the dosimetric approach as an argument in favour of consistency of the calculated values. Based on all of the uncertainties associated with both approaches, the ICRP propounds a value for occupationally exposed persons of $F \cdot 16.8$ $mSv/(MBq \cdot h \cdot m^{-3})$ (rounded to 10 mSv/WLM) for indoor workplaces with predominantly physical activity and $F \cdot 32.5$ $mSv/(MBq \cdot h \cdot m^{-3})$ (rounded to 20 mSv/WLM) (ICRP 2018) in caves accessible to tourists. It is assumed that the ICRP will also recommend a value of $F \cdot 16.8$ $mSv/(MBq \cdot h \cdot m^{-3})$ (rounded to 10 mSv/WLM) for individual members of the public. As a result, the use of new dose coefficients will lead to an increase in the effective dose associated with exposure to radon and radon decay products.

For existing exposure situations, the system of radiological protection provided in ICRP 103 (ICRP 2007) provides a band of dose constraints and reference levelsof 1 mSv to 20 mSv per year so as to be able to determine optimisation strategies. Given this, it is suggested to stipulate reference levels for the radon activity concentration in dwellings. If, for example, an effective dose of 10 mSv per year is used as a basis, this results in a reference level of 600 $Bq \cdot m^{-3}$ when applying the radon dose coefficients from ICRP 65 (ICRP 1993). The new values in ICRP 115 (ICRP 2010) and ICRP 126 (ICRP 2014) formed the basis for the reference level of 300 $Bq \cdot m^{-3}$ in Directive 2013/59/Euratom and in the German Radiation Protection Act (StrlSchG). It is important to note, however, that a conversion using the new dose coefficients is not absolutely

necessary in order to justify the stipulated reference level of 300 $Bq \cdot m^{-3}$ due to the intentionally broad band of dose constraints and reference levels provided by the ICRP.

The following example shows how this works for radon exposure in dwellings: The mean radon concentration in dwellings in Germany is approximately 50 $Bq \cdot m^{-3}$. Based on a typical period of time spent indoors throughout an entire year (7,000 hours), this equates to an exposure of $3.5 \cdot 10^5$ $Bq \cdot h \cdot m^{-3}$ or 0.22 WLM (with a radon equilibrium factor of F=0.4 as is typical indoors). Based on the ($F \cdot 6.3$ mSv/($MBq \cdot h \cdot m^{-3}$) or (4 mSv/WLM) dose coefficient for the population provided in ICRP 65 (ICRP 1993), this results in an annual effective dose of 0.9 mSv for exposure to radon in dwellings in Germany. This means that a dose constraint of 10 mSv leads to a value of 560 $Bq \cdot m^{-3}$, or 600 $Bq \cdot m^{-3}$ when rounded up, for the radon activity concentration.

However, if the value $F \cdot 14$ mSv/($MBq \cdot h \cdot m^{-3}$) (9 mSv/WLM) as suggested in ICRP 115 (ICRP 2010) is used together with F=0.4, this results in an annual effective dose of 2 mSv for exposure to radon in dwellings in Germany. If F=0.4, an assumed general value of $F \cdot 19$ mSv/($MBq \cdot h \cdot m^{-3}$) (12 mSv/WLM) would lead to an annual effective dose of 2.6 mSv for exposure to radon in Germany.

In view of the uncertainties present in both approaches, the good agreement between the epidemiologic and dosimetric approaches observed by the ICRP are of little import. The new evaluations of the WISMUT studies (Kreuzer et al. 2015) indicate risks per exposure at around half the level of those assumed in ICRP 115 (ICRP 2010) and ICRP 126 (ICRP 2014). When viewing uncertainties as a whole, both the old and new dose coefficients calculated using the epidemiologic approach appear to be reconcilable with the epidemiologic data. The uncertainty range in the dosimetric approach also covers a range that includes these values.

As a result, the radon exposure conversions (expressed in either $MBq \cdot h \cdot m^{-3}$ or in WLM) to effective dose values (expressed in mSv) are currently unclear, irrespective of whether they are based on, the so-called dosimetric approach or the epidemiologic approach. The radon dose coefficients resulting from both of these approaches exhibit significant uncertainties within which it is difficult to determine a specific value. The new radon dose coefficients put forward by the ICRP, particularly in recommendations ICRP 115 (ICRP 2010) and ICRP 126 (ICRP 2014), are in fact based on dosimetric and epidemiologic findings from recent years, but the derivation of these values still presents a number of conceptual flaws. On top of that, new studies, particularly the more recent WISMUT miner studies, were not taken into account sufficiently in these publications. The SSK therefore considers this to be an ongoing issue that may well require quantitative amendments in the foreseeable future along with, probably to a lesser extent, conceptual adjustments.

5 Recommendation

The SSK recommends keeping the radon dose coefficients in Germany unchanged until the ICRP provides definitive recommendations on the issue and, furthermore, until international regulatory agreement has been reached on the basis of in-depth scientific discussions. Until that is the case, the radon dose coefficients provided in Section 95(13) of the current German Radiation Protection Ordinance (StrlSchV) should remain valid in current draft legislation as they are within an uncertainty and error range provided by both the epidemiologic and the dosimetric approaches. Any prior national change not agreed upon on an international level would require much greater justification than is currently available.

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7 Glossary

Becquerel (Bq)

SI unit for the activity of a radioactive substance (number of decays per second), 1 Bq = 1 s^{-1}.

Dose

Absorbed dose *D*

Energy d*E* imparted to matter d*m* by ionising radiation:

$$D = \frac{\mathrm{d}E}{\mathrm{d}m} = \frac{1}{\rho} \cdot \frac{\mathrm{d}E}{\mathrm{d}V}$$

Unit: $J \cdot kg^{-1} = Gy$

where ρ: Density of matter d*m*

Organ dose H_T

Product of the organ absorbed dose $D_{T,R}$ averaged over the tissue, organ or body part T resulting from external or internal exposure to radiation R, multiplied by the corresponding radiation weighting factors w_R

$$H_T = \sum_R w_R \cdot D_{T,R}$$

Unit: Sv

The radiation weighting factor values depend on the type and quality of radiation (photons, electrons, neutrons, protons, alpha particles).

Effective dose *E*

Product of the sex-averaged organ doses H_T in relevant organs and tissues multiplied by the corresponding tissue weighting factors w_T

$$E = \sum_T w_T \cdot H_T = \sum_T w_T \cdot \sum_R w_R \cdot D_{T,R}$$

Einheit: Sv

Dose and Dose Rate Effectiveness Factor (DDREF)

Weighting factor used to account for a low biological effectiveness (per dose) of radiation exposure at low doses and low dose rates for some radiation effects. Factor by which a dose-response relationship to be described deviates from the LNT model. Unit: 1

Energy units

Joule (J): 1 J = $6.242 \cdot 10^{12}$ MeV

Equilibrium Equivalent Concentration (EEC) C_{eq}

Radon activity concentration C_{Rn}, in equilibrium with the decay products that have the same potential alpha energy concentration (PAEC) as the actually present compound of radon and its short-lived decay products that are not in equilibrium. The following equation applies in general:

$$C_{eq} = 0.105 \cdot C_{Po\text{-}218} + 0.516 \cdot C_{Pb\text{-}214} + 0.379 \cdot C_{Bi\text{-}214}$$

Unit: $Bq \cdot m^{-3}$

1 Bq·m^{-3} EEC = 2.5 Bq·m^{-3} Ra-222 applies indoors because Rn-222 and its decay products are not in equilibrium. An equilibrium factor of F = 0.4 is assumed. Typical equilibrium factors for dwellings range from 0.2 to 0.6.

Equilibrium factor *F*

The ratio of the equilibrium equivalent concentration C_{eq} to the radon activity concentration C_{Rn}:

$$F = \frac{C_{eq}}{C_{Rn}}$$

Unit: 1

F therefore describes the ratio of potential alpha energy concentration (PAEC) in a given compound of Rn-222 and its short-lived decay products to the PAEC, as would be the case in radioactive equilibrium.

Life Span Study (LSS)

Long-term cohort study of the health effects of ionising radiation on survivors of the Hiroshima and Nagasaki atomic bombs.

Lifetime Excess Absolute Risk (LEAR)

Excess absolute probability of the occurrence of a stochastic effect throughout an entire lifetime. It is approximated by the number of cases (e. g. of lung cancer) per number of people in a reference group of the population.

Unit: 1

Linear Energy Transfer (LET)

Quotient of d*E* and d*s* where, d*E* is the mean energy released to a medium by a charged particle of energy *E* due to collisions when traversing a path length d*s*, with no limit on the secondary particle's kinetic energy released by ionisation, given that d*s* is the corresponding path length:

$$\mathrm{LET} = \frac{\mathrm{d}E}{\mathrm{d}s}$$

Unit: keV·µm^{-1}

Linear No-Threshold (LNT) model

Model of the dose-response relationship based on the assumption that the risk of additional cancer cases and/or hereditary diseases observed at doses ranging from 0.1 Gy to around 2 Gy can be scaled down in a linear fashion to a dose of zero without assuming a threshold dose.

Potential Alpha Energy (PAE)

Sum of the alpha energy of Rn-222 and its short-lived decay products in radioactive equilibrium.

Unit: J

Potential Alpha Energy Concentration (PAEC)

Alpha energy emitted due to radon activity concentration C_{Rn} when Rn-222 decays through to Pb-210 in air volume V as a result of a random compound of short-lived Rn-222 decay products.

Unit: $J \cdot m^{-3}$

1 $Bq \cdot m^{-3}$ radon (Rn-222) in radioactive equilibrium with its short-lived decay products equates to a PAEC of $3.47 \cdot 10^4$ $MeV \cdot m^{-3} = 5.56 \cdot 10^{-9}$ $J \cdot m^{-3}$.

Cumulative potential alpha energy concentration (P_{PAEC})

Time integral of the potential alpha energy concentration (PAEC) over exposure time T:

$$P_{PAEC}(T) = \int_T PAEC(t)\, dt$$

Unit: $J \cdot h \cdot m^{-3}$

Radon activity (A_{Rn})

Quantity dN of radioactive decays of the radionuclide Rn-222 per time interval dt:

$$A_{Rn} = \frac{dN}{dt}$$

Unit: Bq

Radon activity concentration (C_{Rn})

Radon activity A_{Rn} per gas/air volume V :

$$C_{Rn} = \frac{A_{Rn}}{V}$$

Unit: $Bq \cdot m^{-3}$

Cumulative radon activity concentration (P_A)

Time integral of the radon activity concentration C_{Rn} over exposure time T:

$$P_A(T) = \int_T C_{Rn}(t)\, dt$$

Unit: $Bq \cdot h \cdot m^{-3}$

Radon exposure P

General term for cumulative radon activity concentration P_A and cumulative potential alpha energy concentration P_{PAEC}

Risk

A radiation risk is the probability of a certain group of the population for becoming ill or for dying due to the consequences of having been additionally exposed to ionising or other high-energy radiation.

Risk = Risk coefficient R × organ dose H. Unit: 1

Risk coefficient R

Probability of the occurrence of a stochastic effect per dose and per time or age interval. It is approximated by the number of cases (e. g. of cancer) per number of people in a reference group of the population and per dose. If the risk refers to the entire lifetime, the risk coefficient provides the lifetime risk coefficient as the lifetime excess absolute risk (LEAR) per dose.

Unit: 1/Sv

In reference to the risk of lung cancer, ICRP 115 (ICRP 2010) also uses 'nominal risk coefficient' and 'nominal probability coefficient' to describe this quantity. This is the lifetime excess absolute risk of dying from lung cancer per exposure.

Unit: $1/(Bq \cdot h \cdot m^{-3})$

In accordance with standard ICRP practice, the risk coefficients apply to a reference population. This also means that smokers and non-smokers were not evaluated separately.

Detriment

Also known as 'detriment-adjusted risk'. The detriment is the product of risk coefficient R and detriment quality d. The detriment depends on a number influencing parameters. It largely consists of stochastic effects: Probability of attributable fatal cancer, weighted probability of attributable non-fatal cancer, weighted probability of severe hereditary effects, and length of life lost if the harm occurs.

Unit: 1/Sv

Detriment quality d

Weighting factor for the radiation risk in order to designate the 'severity' of a stochastic effect and render such effects comparable with one another. The type of weighting is stipulated in ICRP 103 (ICRP 2007). Detriment quality includes the lethality of the disease, the loss of quality of life, and the relative loss of life expectancy due to the disease. Detriment quality is independent of exposure.

Unattached fraction

The fraction of potential alpha energy concentration of short-lived radon decay products not attached to particles.

Working Level (WL)

Working level (WL) is the unit used for every combination of Rn-222 and its short-lived decay products in a litre of air which emits a potential alpha energy of $1.3 \cdot 10^5$ MeV = $2.08 \cdot 10^{-8}$ J. 1 WL was originally defined as

100 pCi/L EEC = 3,700 $Bq \cdot m^{-3}$ EEC.

The following values apply to Rn-222: 1 WL = $1.3 \cdot 10^8$ $MeV \cdot m^{-3}$ = $2.08 \cdot 10^{-5}$ $J \cdot m^{-3}$. The unit WL is not an SI unit and should therefore no longer be used.

Working Level Month (WLM)

WLM is a unit for the radon exposure a worker receives during a month (170 working hours) at 1 WL.

The unit WLM is not an SI unit and should therefore no longer be used.

The following applies to radon with the equilibrium factor F:

$$1\ \mathrm{WLM} = 3.54\ \mathrm{mJ \cdot h \cdot m^{-3}} = \frac{0.64}{F}\ \mathrm{MBq \cdot h \cdot m^{-3}}$$

The following values apply with an equilibrium factor of $F = 0.4$:

$$1\ \mathrm{WLM} = 3.54\ \mathrm{mJ \cdot h \cdot m^{-3}} = 1.59\ \mathrm{MBq \cdot h \cdot m^{-3}}$$

1 WLM corresponds to a radon equilibrium equivalent concentration of $0.64\ \mathrm{MBq \cdot h \cdot m^{-3}}$.

$1\ \mathrm{Bq \cdot m^{-3}}$ of radon in the workplace for one year leads to an exposure of $1.26 \cdot 10^{-3}\ \mathrm{WLM} = 4.46 \cdot 10^{-6}\ \mathrm{J\ h\ m^{-3}}$ or $2.00 \cdot 10^{3}\ \mathrm{Bq \cdot h \cdot m^{-3}}$ based on the assumption of an indoor working time of 2,000 hrs per year and an equilibrium factor of $F = 0.4$ (ICRP 1993).

$1\ \mathrm{Bq \cdot m^{-3}}$ of radon in dwellings for one year leads to an exposure of $4.4 \cdot 10^{-3}\ \mathrm{WLM} = 1.56 \cdot 10^{-5}\ \mathrm{J\ h\ m^{-3}}$ or $7.00 \cdot 10^{3}\ \mathrm{Bq \cdot h \cdot m^{-3}}$ based on the assumption of spending 7,000 hrs indoors per year and an equilibrium factor of $F = 0.4$ (ICRP 1993).