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Limits for Inhalation of Radon Daughters by Workers

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Limits for Inhalation of Radon Daughters by Workers

A report of the International Commission
on Radiological Protection

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PREFACE

In November 1977 the Commission asked two of its members, D. J. Beninson and W. Jacobi, to prepare a report outlining a possible basis for setting an appropriate limit for occupational exposure to radon and its daughter products.

At its meeting in 1980 the Commission issued a Statement concerning occupational exposure to radon-222 and its daughters. This was published in *Annals of the ICRP*, **4**, (3/4), 1980. In the statement a reference was made to a Commission report being prepared on the topic of occupational exposure to radon and its daughter products; this is that report.

CONTENTS

INTRODUCTION

(1) One of the occupational risks of mining ore results from the exposure of miners to airborne radon gases (^{222}Rn, ^{220}Rn) and their short-lived decay products (radon daughters).* The inhalation of these radionuclides constitutes the most important occupational exposure in mines, especially in uranium mines. A previous report of the ICRP outlined the principles of monitoring and limitation of radiation exposure in uranium and other mines (IC 77b). It should be noted that radon and its daughters are also present in atmospheric and room air. The inhalation of these radionuclides constitutes a substantial fraction of the radiation exposure of the population from natural radiation sources.

(2) The exposure to these radionuclides is mainly due to the inhalation of the short-lived radon daughters, which leads to an inhomogeneous dose distribution within the human respiratory tract. Compared with this lung dose from inhaled daughters the dose contribution from the inhaled radon gas itself is small under normal exposure conditions (see Annex C). In the Commission's previous recommendations on internal exposure (*ICRP Publication 2*, 1960) a maximum permissible concentration for short-lived radon daughters in air was derived from a maximum permissible dose equivalent of 0.15 Sv (15 rem) to the epithelium of the large bronchi, which was regarded as the critical tissue. For this evaluation a simplified lung model was used which paid attention principally to the dose from inhaled daughter atoms not attached to airborne particulates. Since then, improved models for the estimation of the dose distribution in the respiratory tract from inhaled radon daughters have been developed.

(3) In its current basic recommendations the Commission introduced a system of dose limitation (*ICRP Publication 26*, 1977). This limits the total individual risk or health detriment from stochastic radiation effects (cancer, genetic effects), taking into account the dose in all risk-relevant tissues of the human body. This concept is based on the principle that at a given level of safety the stochastic risk should be equal whether the whole body is irradiated uniformly or whether there is a non-uniform or partial irradiation of the body. To meet this condition the quantity "effective dose equivalent" (H_E) has been introduced which is defined by the equation

$$H_E = \sum_T w_T H_T \qquad (\text{with} \sum_T w_T = 1) \tag{1}$$

summarized over all exposed, relevant tissues T. In this equation H_T is the mean dose equivalent in tissue T, and w_T is a weighting factor which represents the ratio of the stochastic risk resulting from tissue T to the total risk when the whole body is irradiated uniformly.† For the lung, as a

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* The main radioactive decay properties of these radionuclides are tabulated in Annex A. The special exposure quantities and units (and their conversion factors) which are used for the radionuclides in radiation protection are compiled in Annex B.

† The recommended values of the weighting factors w_T are:

Gonads	0.25	Thyroid	0.03
Breast	0.15	Bone surfaces	0.03
Red bone marrow	0.12	Remainder (total)	0.30
Lung	0.12		

Regarding the remainder, a value $w_T = 0.06$ is applied to each of those five tissues receiving the highest dose equivalent. The gastro-intestinal tract is treated as four separate tissues (stomach, small intestine, upper large intestine and lower large intestine). The skin, the lens of the eyes, hands, forearms, feet and ankles are not included in the remainder.

It is recognized that the risk associated with a given exposure will vary with the age and sex of the individual exposed. However, the values of w_T are recommended as appropriate for the protection of any worker, regardless of these sources of variability.

single organ comprising several tissues, a weighting factor $w_T = 0.12$ is recommended. This concept of the effective dose equivalent, which replaces the previous concept of the critical tissue, is used in the evaluation of intake limits for radionuclides (*ICRP Publication 30*, 1979) and can also be used for radon and radon daughters.

(4) The Commission has recommended a value of 0.05 Sv as the annual occupational limit of the effective dose equivalent. The assessment of this limit was determined by the requirement that the average occupational risk of radiation workers should not exceed the average occupational risk experienced in industries with a high standard of safety. It is the opinion of the Commission that this limit of effective dose equivalent or the corresponding level of radiation risk should be applied also to miners exposed to radon and its daughters.

(5) Occupational limits for intake or exposure of radon daughters can be derived in two different ways. The first procedure is based on the substantial epidemiological information, which has been obtained in the last 15 years, on the excess lung cancer risk among several groups of underground miners, especially uranium miners, exposed to relatively high ^{222}Rn-concentrations. From these data the relationship between radon daughter exposure and excess lung cancer risk can be estimated. Taking into account the primary risk limit for stochastic radiation effects, recommended by the Commission for radiation workers (*ICRP Publication 26*, 1977), this epidemiological approach allows a more direct assessment of exposure limits for short-lived ^{222}Rn-daughters. The second procedure is based on dosimetric models which provide a relationship between intake or exposure and the dose to the lung or the effective dose equivalent. Such a dosimetric approach proceeds from the recommended annual limit of 0.05 Sv for the effective dose equivalent. Both approaches, the epidemiological as well as the dosimetric, involve uncertainties. Because of this the Commission considered that its final decision should be based on a comparison of both approaches.

THE EPIDEMIOLOGICAL APPROACH

(6) An excess lung cancer risk has been observed among several groups of underground miners exposed to ^{222}Rn and its daughters: uranium miners in Colorado/USA, Czechoslovakia and Ontario/Canada, fluorspar miners in Newfoundland, and several smaller groups of non-uranium miners working in hard rock mines in Sweden and England. The results of these epidemiological studies are discussed in detail in the report of UNSCEAR (UN 77), where the literature is also listed. The Commission is not aware of any substantial new data that have been published since 1977, except for one paper which gives a more detailed analysis of the findings among the uranium miners in Czechoslovakia (KU 79), and another paper which completes the data for the uranium miners in USA up to 1974 (AR 78, see also BE 80).

(7) Of special importance and weight are the epidemiological studies for the two larger groups of uranium miners in the USA and Czechoslovakia, which have now been followed up for a period of nearly 30 years and which have received a mean exposure of about 700 and 300 WLM, respectively.* These studies indicate that a proportional relationship, without threshold, between the excess lung cancer incidence and potential α-energy exposure to ^{222}Rn-daughters up to levels of several hundred WLM cannot be excluded. Figure 1 shows the excess lung cancer risk among the Czech uranium miners (KU 79), who started mining between 1948 and 1952 and were followed up until the end of 1975 (mean follow-up period 24 years).

The slope of this curve corresponds to a lung cancer risk of about (2–3) 10^{-4} per WLM exposure. In the low-exposure group (< 100 WLM) which received a mean annual exposure of less than 10–20 WLM, the excess is not significant at the 5% level. The slopes of the corresponding curves are, however, different for different subgroups of these miners. The observations indicate a decrease of the risk coefficient with decreasing age at the start of mining and—at high-exposure levels—with increasing exposure rate (KU 79).

(8) The values of the mean excess lung cancer risk rate per unit of potential α-energy exposure to short-lived ^{222}Rn-daughters which can be derived from the various epidemiological studies among ^{222}Rn-exposed miners cover a range from about 2–20 cases per 10^6 person-years per WLM (UN 77, AR 78, CO 80, BE 80). The breadth of this range is partly due to the observed age dependency of the risk coefficient. Averaged over all age periods during occupational work, 5–15 cases per 10^6 person-years per WLM can be regarded as the most probable range. Assuming a mean manifestation period of 30 years this corresponds to a total life time risk for lung cancer of

$$1.5\text{–}4.5\ 10^{-4} \text{ per WLM} = 0.043\text{–}0.13 \text{ per J h m}^{-3} \qquad (2)$$

in accordance with the conclusions of the 1977 UNSCEAR Report (UN 77). The Commission believes that a mean breathing rate of 1.2 $m^3\ h^{-1}$ during the occupational working period of 2 000 h per year, assumed for other radiation workers, seems to be also an appropriate mean value for miners. On this assumption an excess lung cancer risk of

$$0.036\text{–}0.11 \text{ per Joule} \qquad (3)$$

inhaled potential α energy of short-lived ^{222}Rn-daughters follows from eqn 2. For higher

* The definition of the units "Working Level Month (WLM)" and "Working Level (WL)" and their conversion factors to SI-units is given in Annex B of this report.

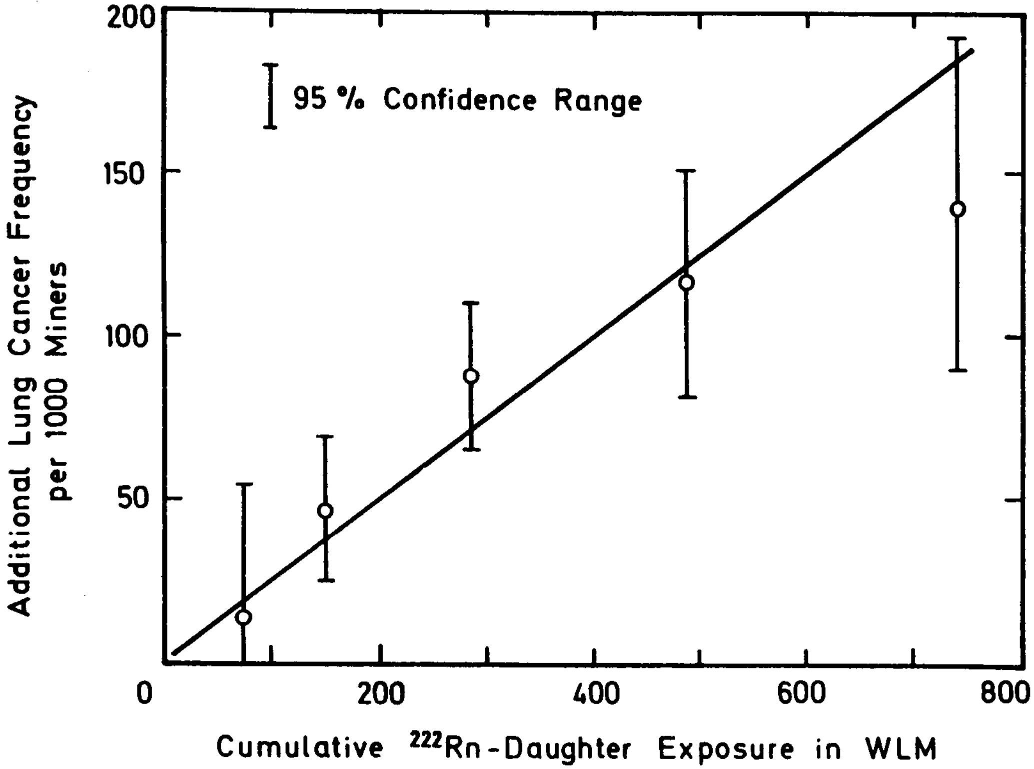

Fig. 1. Observed excess lung cancer frequency among uranium miners in Czechoslovakia (1948 1975) as a function of their potential α-energy exposure to short-lived ^{222}Rn-daughters (from Kunz *et al.*, 1979).

breathing rates this value will be lower. It should be noted that the risk values for the Colorado-uranium miners lie in the lower part, and the values for the Czech uranium miners in the upper half of this range.

(9) The annual limit of 0.05 Sv of the effective dose equivalent for workers was recommended by the Commission on the basis of a risk concept (see paragraph 4), assuming a proportional dose-risk relationship. On this basis the annual exposure of individual miners to ^{222}Rn-daughters should be limited to the same risk as that corresponding to this effective dose limit of 0.05 Sv, applying a risk conversion factor of 1.65 10^{-2} Sv^{-1} (IC 77). Taking into account the range of the lung cancer risk coefficient for miners, given in eqn (3), this epidemiological approach yields an annual occupational limit of intake (ALI) in the range of

$$0.0075\text{–}0.023 \text{ Joule} \tag{4}$$

for the total inhaled potential α energy of a mixture of short-lived ^{222}Rn-daughters. With a mean breathing rate of 1.2 m^3 h^{-1} this would correspond to an annual limit of potential α-energy exposure (ALE) of

$$0.0063\text{–}0.020 \text{ J h m}^{-3} = 1.8\text{–}5.5 \text{ WLM} \tag{5}$$

(10) The Commission wishes to point out that the risk coefficients given in paragraph 8 include the excess lung cancer risk from external γ radiation in mines and from other carcinogenic chemical agents to which these miners were exposed. They also take into account

possible cocarcinogenic or synergistic influences. Thus the real risk from the inhalation of ^{222}Rn-daughters alone might be lower. Consequently this would lead to somewhat higher intake or exposure limits for workers who are only exposed to radon daughters. However the main uncertainty of the epidemiological approach results from the low reliability of the exposure data for those miners who died from lung cancer. Systematic errors in the calculation of their individual exposure on the basis of area monitoring measurements in mines cannot be excluded (DO 79, ZE 81).

THE DOSIMETRIC APPROACH

(11) Retention studies indicate a biological half-life of a few hours up to about 1 day for inhaled ^{222}Rn- and ^{220}Rn-daughters in the human lung. This means that the main fraction of the potential α energy of ^{222}Rn-daughters deposited in the lung will be absorbed in this organ; the dose to other tissues delivers a negligible contribution to the effective dose. The critical cells with respect to the induction of lung cancer or other stochastic radiation effects are assumed to be located in the basal cell layer of the bronchial epithelium and in the pulmonary epithelium (IC 80). These two tissues can be regarded as target tissues in the lung for the dosimetry of inhaled radon daughters. Only in the case of the more long-lived ^{220}Rn-daughter ^{212}Pb (ThB) will a considerable fraction of the deposited activity in the lungs be transferred to other tissues, particularly the blood (red blood cells), the kidneys and the bone surfaces (IC 79, JAC 80).

(12) The risk concept for stochastic radiation effects like lung cancer, as recommended by the Commission for purposes of radiation protection, assumes a proportional relationship without threshold between the dose to risk-relevant target tissues and the associated excess probability for the induction of cancer in the range of low doses. The excess lung cancer incidence among radon-exposed miners (see paragraph 7) does not exclude the possibility of such a proportional relationship up to potential α-energy exposures of about 2 J h m^{-3} or several hundred WLM. On the basis of this concept the risk-relevant dosimetric quantities for radon daughters in the lung are the *mean* dose or dose equivalent to the two target tissues mentioned above, the basal cell layer in the tracheo-bronchial (TB) region and the mean dose to the epithelium in the pulmonary (P) region.

(13) In the years following the first recommendations of ICRP Committee 2 (IC 60) better estimates of the dose distribution in the bronchial tree from inhaled radon daughters have been published (e.g. AL 64, JA 64, HA 67, WA 70, HA 72). These studies led to the conclusion that the maximum dose should be expected in the basal cells of the segmental–subsegmental bronchi. From these studies, and assuming a quality factor of 20 for α radiation, one can derive a dose equivalent to the target cells in these bronchial airways per unit of potential α-energy exposure to ^{222}Rn-daughters; this leads to values in the range of about 20–400 Sv per J h m^{-3} (0.07–1.4 Sv per WLM).

The broad range of this dose factor is mainly due to the different biological and physical parameters that were used in these dosimetric models. However, new, more realistic analyses indicate that under normal conditions the dose to the basal cells in the segmental–subsegmental bronchi will be in the lower part of the range given above (HA 80, JAC 80, JAM 80). The following dosimetric approach is based on the results of two extensive studies which have been published recently (JAC 80, 81; JAM 80, 81).* In particular these two models take into account the observed large variation in the depth of stem cells throughout the bronchial tree (GA 72) which is one of the most sensitive parameters. Both models agree in the conclusion that under normal conditions in mines the dose distribution over the bronchial generations from inhaled radon daughters is more uniform than previously assumed, with a broad maximum in the region from the lobar bronchi down to the upper bronchioles.

(14) As was pointed out in paragraph 12 the risk-relevant dose quantities for the lung are taken to be the mean dose to the bronchial basal cell layer and the mean dose to the pulmonary epithelium. This basic concept is supported by the dosimetric findings mentioned above.

* A detailed comparison of these two models and their results is being prepared by a OECD/NEA Expert Group.

Table 1. Estimated conversion functions for the mean dose equivalent to the target tissues in the lung and to the total lung from inhaled ^{222}Rn-daughters (occupational exposure)[1,2]

Target tissue, dosimetric model	Mean dose equivalent (Sv) per Joule inhaled potential α energy	Mean dose equivalent (Sv) per WLM potential α-energy exposure
Bronchial basal cell layer		
JACOBI–EISFELD model	$18+170f_p$	$0.076+0.72f_p$
JAMES–BIRCHALL model	$14+560f_p$	$0.060+2.4\ f_p$
ICRP lung model[3]	$15+100f_p$	$0.064+0.42f_p$
Pulmonary region		
JACOBI–EISFELD model[2]	$5.2\ (1-f_p)$	$0.022\ (1-f_p)$
JAMES–BIRCHALL model[2]	*ca.* 2	*ca.* 0.01
ICRP lung model	$8\ (1-f_p)$	$0.033\ (1-f_p)$
Total lung ($m=1$ kg)		
JACOBI–EISFELD model	$7+3f_p$	$0.030+0.013f_p$
JAMES–BIRCHALL model	not evaluated	
ICRP lung model	$10+4f_p$	$0.042+0.016f_p$

[1] Referring to a mean breathing rate of 1.2 $m^3\ h^{-1}$ and an AMAD = 0.2–0.3 μm for the carrier aerosol of attached daughters.

[2] The values from the Jacobi–Eisfeld study refer to the anatomical lung model of Weibel (A), whereas the values from the James–Birchall study refer to the Yeh–Shum lung model.

[3] Mean dose to the total TB region, assuming a mass of 0.04–0.05 kg for this region (IC 75).

Furthermore it follows from these dosimetric studies that the dependency of the mean dose to these two target tissues from the parameters characterizing the deposition, translocation and retention of radon daughters in the lung is relatively small. In Table 1 the dose-conversion functions for the mean dose equivalent to target tissues in the lungs and to the total lung are listed which result from these two dosimetric studies as best estimates for the occupational exposure to short-lived ^{222}Rn-daughters using a quality factor of 20 for α radiation.

Dose values are given as a function of the unattached fraction f_p of the total potential α energy of the daughter mixture. Included in Table 1 are the conversion functions which can be obtained if the ICRP lung model for clearance class D (IC 79) is applied to the inhalation of ^{222}Rn-daughters (JA 72, UN 77); using this model the dose is averaged over the total mass of the considered lung region. It should be noted that this ICRP lung model overestimates the deposition probabilities of radon daughters in the TB region and P region of the lung.

(15) The available experience indicates that for most occupational exposure conditions the unattached fraction of the potential α energy of daughter mixtures, averaged over the total annual working period, is in the range $f_p=0$–0.05. For this range the dose factors given in Table 2 result from the different dosimetric models (see Table 1).

(16) In conclusion, a mean dose equivalent to the bronchial basal cell layer in the range of 15–40 Sv per Joule inhaled potential α energy of short-lived ^{222}Rn-daughters can be derived for occupational exposure conditions from the different dosimetric models. This corresponds to about 0.064–0.17 Sv per WLM. For mine areas with high dust concentration a dose factor in the lower part of this range should be expected, whereas for high-ventilated areas with low dust generation a dose factor in the upper half of this range seems to be more appropriate. The mean dose to the bronchial basal cell layer is about a factor 4–10 higher than the mean pulmonary

Table 2. Estimated mean dose conversion factors for occupational exposure to ^{222}Rn-daughters, assuming an unattached fraction $f_p = 0$–0.05 of the total potential α energy of the daughter mixture (AMAD = 0.2–0.3 μm)

Target tissue, dosimetric model	Mean dose equivalent (Sv) per Joule inhaled potential α energy	Mean dose equivalent (Sv) per WLM potential α-energy exposure
Bronchial basal cell layer		
JACOBI–EISFELD model	18–27	0.076–0.11
JAMES–BIRCHALL model	14–42	0.060–0.18
ICRP lung model	15–20	0.064–0.085
Pulmonary region		
JACOBI–EISFELD model	5.2–4.9	0.022–0.021
JAMES–BIRCHALL model	*ca.* 2	*ca.* 0.01
ICRP lung model	8.0–7.0	0.033–0.031
Total lung ($m = 1$ kg)		
JACOBI–EISFELD model	7.1	0.030
JAMES–BIRCHALL model	not evaluated	not evaluated
ICRP lung model	10	0.042

dose. When the dose is averaged over the total lung, conversion factors in the range of 7–10 Sv per Joule, or 0.03–0.04 Sv per WLM, respectively, are obtained.

(17) In its basic recommendations the Commission has proposed that the total lung (NP + TB + P + L region) should be considered as a composite organ. This means that the recommended risk-weighting factor $w_T = 0.12$ for the lung refers to the mean dose to the total lung. This "Mean Lung Dose (MLD)"-concept is reasonable, when the dose to the pulmonary region of the lung is the dominant factor. In the special case of inhaled short-lived radon daughters however, the dose to the bronchial epithelium is considerably higher than the mean dose in the pulmonary region or in the total lung. Taking into account a similar radio sensitivity of the critical cells in the bronchial epithelium and the pulmonary tissue, it might be appropriate to split up the weighting factor $w_T = 0.12$ for the total lung, as recommended for the GI-tract. As an alternative to the MLD-concept, the effective dose equivalent from inhaled radon daughters is derived by applying separate weighting factors ($w_T = 0.06$) for the mean dose to the basal cell layer of the tracheo-bronchial region and to the pulmonary region. This alternative is called the "Regional Lung Dose (RLD)"-concept.

(18) The effective dose equivalent for ^{222}Rn-daughters, resulting from both weighting concepts, are shown in Figure 2 as a function of the unattached fraction of the total potential α energy of ^{222}Rn-daughters in the inhaled air. The curves were derived from the dose factors listed in Table 1. The weighted dose from tissues other than the lung is small and can be neglected for ^{222}Rn-daughters. The experience in mines shows that the time-averaged fraction f_p of the unattached α energy of the radon daughter mixture in mine air is less than a few percent ($f_p \leq 0.05$). Figure 2 shows that under these conditions the total potential α-energy intake or exposure, respectively, might be an adequate quantity, for purposes of radiation protection, to characterize the effective dose equivalent from any mixture of ^{222}Rn-daughters.

(19) For occupational exposure to ^{222}Rn-daughters the following range for the effective dose

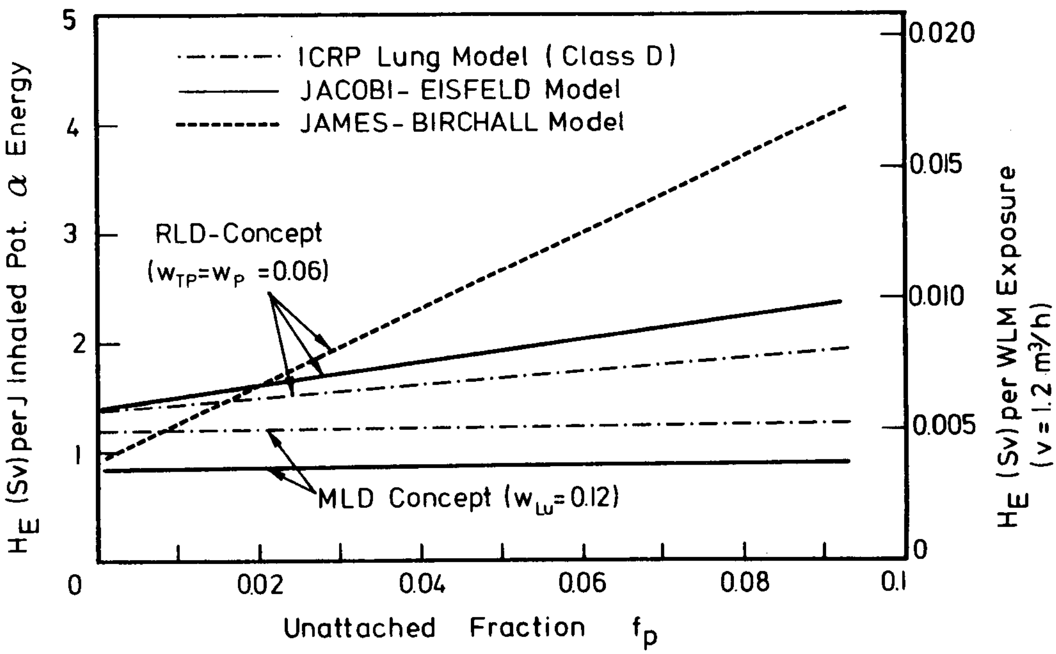

Fig. 2. Effective dose equivalent from inhaled ^{222}Rn-daughters as a function of the unattached fraction of the potential α energy of the daughter mixture; comparison of different dosimetric models and weighting concepts.

equivalent per unit of potential α-energy intake (I_p) or exposure (E_p) results, for $f_p \leq 0.05$, from the different weighting concepts (see Figure 2):

Mean lung dose–concept ($w_{LV} = 0.12$)

$H_E/I_p = 0.85$–1.2 Sv per J

$H_E/E_p = 0.0036$–0.0050 Sv per WLM (6a)

Regional lung dose–concept ($w_{TB} = w_p = 0.06$)

$H_E/I_p = 1.0$–2.5 Sv per J

$H_E/E_p = 0.0042$–0.011 Sv per WLM (6b)

It follows from eqn (6b) that a ^{222}Rn-daughter mixture in air which corresponds to an equilibrium equivalent ^{222}Rn-activity exposure of 1 Bq h m^{-3} leads to an effective dose equivalent of about (0.7–1.7) 10^{-8} Sv from inhaled ^{222}Rn-daughters. This value is about a factor of 40–100 higher than the effective dose equivalent per Bq h m^{-3} exposure to ^{222}Rn alone (H_E from $^{222}\text{Rn} \approx 1.8\ 10^{-10}$ Sv per Bq h m^{-3}; see Annex C). Thus the contribution from ^{222}Rn to the effective dose is only of importance if the equilibrium factor of the daughter mixture in air is below $F = 0.1$.

(20) The dose to target tissues in the lung from inhaled ^{220}Rn-daughters has been estimated using the same models as for ^{222}Rn-daughters (HA 73, JAC 80, JAM 80). As the radioactive half-life of ^{212}Pb (ThB) is comparable to its biological half-life in the lung, the ratio of the bronchial to pulmonary dose is lower than for ^{222}Rn-daughters and a considerable fraction of the deposited ^{212}Pb-activity is transferred to blood and other tissues. The dose contribution from these tissues can be evaluated on the basis of the metabolic and dosimetric models described in *ICRP Publication 30* (IC 79) and its supplements.

Table 3. Mean effective dose equivalent from inhaled ^{220}Rn and its daughters (from JAC 80)

Inhaled radionuclide	Effective dose equivalent (Sv)		
	per 10^{10} Bq inhaled activity	per J inhaled potential α energy	per WLM potential α-energy exposure
^{220}Rn(Tn)	0.9 } 1	—	—
^{216}Po(ThA)[1]	0.1 }	—	—
^{212}Pb(ThB)	400	0.6	0.0025
^{212}Bi(ThC)	60	0.9	0.0038

[1] Assumed to be inhaled in form of unattached atoms.

(21) Estimated mean values of the effective dose equivalent to workers per unit of inhaled activity and potential α energy of ^{220}Rn-daughters are summarized in Table 3. For comparison the value for inhaled ^{220}Rn-gas (see Annex C) is listed in the table. The data for ^{212}Pb (ThB) and ^{212}Bi (ThC) refer to an AMAD of 0.2–0.3 μm for their carrier aerosol, which can be considered as an appropriate value for most mine atmospheres.

Taking into account the radioactive equilibrium between ^{220}Rn and ^{216}Po(ThA) in air, one can calculate a total effective dose equivalent from both nuclides of about 1 10^{-10} Sv per Bq inhaled ^{220}Rn. This value is small compared with the effective dose from ^{212}Pb(ThB) + ^{212}Bi(ThC) provided the ^{212}Pb(ThB)/^{220}Rn(Tn)-activity ratio in air is higher than about 0.02.

(22) It follows from Table 3 that the effective dose equivalent per unit potential α-energy intake or exposure is not very different for ^{212}Pb(ThB) and ^{212}Bi(ThC). Thus the total potential α-energy intake or exposure of these two nuclides can be used as an appropriate monitoring quantity. For these purposes an effective dose equivalent of

$$H_E/I_p = 0.8 \text{ Sv per J} \tag{7a}$$

inhaled potential α energy, or of

$$H_E/E_p = 0.0034 \text{ Sv per WLM} \tag{7b}$$

potential α-energy exposure is recommended for any mixtures of ^{212}Pb(ThB) and ^{212}Bi(ThC).

(23) The effective dose factors for ^{222}Rn- and ^{220}Rn-daughters given in the previous paragraphs refer to the occupational exposure of an adult Reference Man. For indoor and outdoor exposure of members of the public, correction factors have to be applied. For children and infants especially, the age-dependency of breathing rate, deposition, retention and tissue masses has to be taken into account (HO 79).

THE DEVELOPMENT OF RECOMMENDED LIMITS

(24) The Commission recommends that the annual limit for the effective dose equivalent of 0.05 Sv for workers should be applied also to miners exposed to radon and its daughters. From this basic limit secondary limits for the annual intake or exposure for ^{222}Rn, ^{220}Rn and their short-lived daughter mixtures can be derived, taking into account the effective dose factors for these radionuclides given in the previous section.

(25) The possible range for the setting of occupational intake and exposure limits for ^{222}Rn-daughter mixtures in air, resulting from the different dosimetric and weighting concepts (see Figure 2) is graphically presented in Figure 3 as a function of the unattached fraction f_p of the total potential α energy of the daughter mixture in the inhaled air. The limit resulting from the mean lung dose (MLD) weighting-concept is approximately 1.5–3 times higher than the limit derived from the regional lung dose (RLD) weighting-concept.

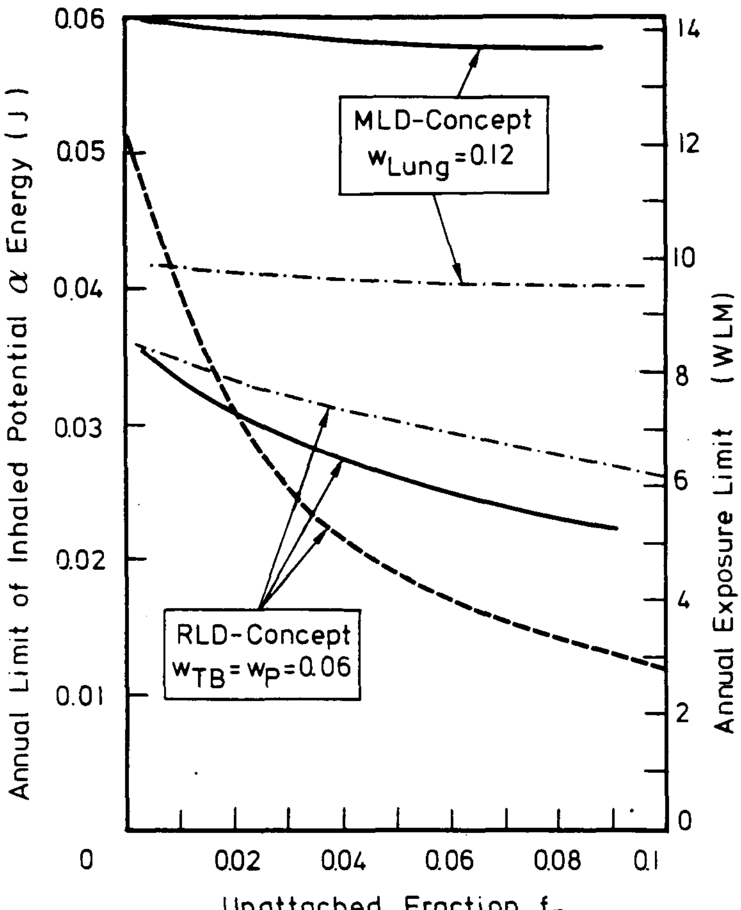

Fig. 3. Range of intake and exposure limits for short-lived ^{222}Rn-daughters resulting from different dosimetric models and weighting concepts. ——— JACOBI–EISFELD model (JAC 80, 81); — — — JAMES–BIRCHALL model (JAM 80, 81); —·—·— ICRP lung model, class D.

(26) In most radon atmospheres, particularly in mines, the long-term mean value of the unattached fraction of the potential α energy of the ^{222}Rn-daughter mixture is below 0.05. Under these conditions the range of limits summarized in Table 4 is obtained from the dosimetric approaches, compared with the limit derived from the epidemiological approach (see paragraph 9). The recommendation of a final limit requires a careful judgment of the uncertainties involved in both types of approach.

(27) The main uncertainty of the dosimetric approach is due to the fact that little is known about the specific radiosensitivity of the cells at risk in the bronchial and alveolar epithelium with respect to the induction of lung cancer. While the tumours observed among radon-exposed miners are mainly of the bronchial type, some animal experiments indicate that the dose to the whole lung has an important influence on the initiation of cancer (CH 81).

It should be noted that the weighting factor $w_T = 0.12$ for the total lung recommended by the Commission (IC 77) has been derived from cases with external exposure, where the total lung is irradiated nearly uniformly. The Commission believes that in the special case of inhaled radon daughters, which leads to a considerably higher dose to the bronchial than to the pulmonary epithelium, it seems more realistic to attempt to take account of the average doses in these two target tissues of the lung.

(28) Assessments based on epidemiological studies seem to be more straightforward. However, as mentioned, they are also qualified by substantial uncertainties. The radiation risk estimates for lung cancer among miners exposed to radon are biased towards higher values because other carcinogenic agents, which are involved in mine atmospheres, were given no weight in the risk assessment. Secondly these risk estimates are not corrected for the risk contribution of external γ radiation, whose level is at present in the order of 0.01 Sv per year in some uranium mines. A third, substantial cause of uncertainty is the variable quality of the exposure estimates in these epidemiological studies. There is some evidence, especially in the earlier phases of uranium mining, that the individual exposure of miners was higher than that indicated by the results of area monitoring which were used for the estimation of their radon exposure; a factor of about two cannot be excluded (DO 79, ZE 81). All such uncertainties would bias the risk factor assessments to higher values. Consequently, if the limits were derived from the epidemiological approach this could be regarded as a conservative estimate, especially for high-ventilated mines.

(29) On the basis of these considerations the Commission recommends for workers an annual limit of intake (ALI_p) for the potential α energy of any mixture of short-lived ^{222}Rn-daughters:

$$ALI_p = 0.02 \text{ J} \tag{8a}$$

Table 4. Comparison of limits for ^{222}Rn-daughters in air, derived from the dosimetric and epidemiological approach.

Type of approach	Annual limit (potential α energy) Intake limit in J	Exposure limit in WLM[2]
Dosimetric approach[1]		
Mean lung dose-concept	0.042 – 0.059	10–14
Reg. lung dose-concept	0.020 – 0.050	4.8–12
Epidemiological approach	0.0075– 0.023	1.8–5.5

[1] For $f_p = 0$–0.05.
[2] Assuming a mean breathing rate of 1.2 $m^3 h^{-1}$.

Taking into account a mean breathing rate of $v = 1.2\ m^3\ h^{-1}$ this limit corresponds to an annual limit of potential α-energy exposure:

$$ALE_p = 0.017\ J\ h\ m^{-3} = 4.8\ WLM \tag{8b}$$

From these limits a "Derived Air Concentration (DAC)"

$$DAC_p = 8.3\ 10^{-6}\ J\ m^{-3} = 0.40\ WL \tag{9a}$$

can be derived, if an annual working period of 2 000 hours is applied. Expressed in terms of the equilibrium-equivalent ^{222}Rn-concentration $EC_{Rn\text{-}222}$ (see Annex B) a derived air concentration

$$DAC\ (EC_{Rn\text{-}222}) = 1\ 500\ Bq\ m^{-3} \tag{9b}$$

can be obtained. It should be noted that the potential α-energy intake is the primary quantity for which the Commission recommends the limit.

(30) For the exposure to ^{222}Rn-gas alone, without daughters, the Commission recommends an annual limit of activity exposure

$$ALE\ (^{222}Rn) = 3\ 10^8\ Bq \cdot h\ m^{-3} \tag{10}$$

taking into account the effective dose factor for ^{222}Rn derived in Annex C. From the limit it follows that the DAC for ^{222}Rn without daughters would be:

$$DAC\ (^{222}Rn) = 1.5\ 10^5\ Bq\ m^{-3} \tag{11}$$

This value is a factor of 100 higher than the equilibrium-equivalent DAC $(EC_{Rn\text{-}222})$ for ^{222}Rn-daughters given in eqn (9b). Thus under normal conditions the contribution from ^{222}Rn-gas itself can be neglected.

(31) For ^{220}Rn and its daughters in air the Commission recommends the following annual limits and DAC-values for workers, derived on the basis of the dosimetric approach (see paragraphs 21, 22):

$\underline{^{220}Rn(+^{216}Po)}$

$ALE = 5\ 10^8\ Bq \cdot h\ m^{-3}$ (Primary Limit)

$$\begin{aligned} ALI &= 6\ 10^8\ Bq \\ DAC &= 2.5\ 10^5\ Bq\ m^{-1} \end{aligned} \tag{12}$$

$\underline{^{212}Pb(ThB) + ^{212}Bi(ThC)}$

$$\begin{aligned} ALI_p &= 0.06\ J \quad \text{(Primary Limit)} \\ ALE_p &= 0.050\ J\ h\ m^{-3} = 14\ WLM \\ DAC_p &= 2.5\ 10^{-5}\ J\ m^{-3} = 1.2\ WL \end{aligned} \tag{13}$$

The latter value corresponds to a DAC, expressed in terms of the equilibrium-equivalent ^{220}Rn-concentration, of

$$DAC(EC_{Rn\text{-}220}) = 330\ Bq\ m^{-3} \tag{14}$$

This DAC for ^{220}Rn-daughters is about 1/500 of the DAC for $^{220}Rn(+^{216}Po)$. Thus under most conditions the exposure to ^{220}Rn can be neglected.