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RADIATION PROTECTION

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Lung Cancer Risk from Indoor Exposures to Radon Daughters

A report of a Task Group of the
International Commission on Radiological Protection

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"We must bear in mind that all of us are continuously inhaling the radium and thorium emanations and their products, and ionized air. . . . Some have considered that possibly the presence of radioactive matter and ionized air in the atmosphere may play some part in physiological processes, ..."

Ernest Rutherford

In: Some cosmical aspects of radioactivity, Paper presented at a Meeting of the Astronomical Society of Canada, April 3, 1907 (*J. R. Astronom. Soc. Canada*, 1907, May–June, pp. 145–165)

CONTENTS

PREFACE

In 1981, the Commission established a Task Group to analyse the possible lung cancer risk associated with the natural radiation exposure of populations to inhaled radon daughters.

This report of the Task Group relates specifically to the risk associated with indoor exposure, particularly that resulting from inhaled ^{222}Rn-daughters. This type of exposure contributes the largest fraction of the natural radiation dose to populations living in the temperate regions of the world. A major part of this indoor exposure depends strongly on social factors and individual living habits. For this controllable fraction of natural radiation exposure, the principles for limiting exposure of the public to natural sources of radiation which have been recommended by the Commission (IC84) should be observed. In this context, the results presented in this report may provide guidance to the competent national authorities for the setting of action levels in existing houses and for the optimization procedure in the planning of future houses.

The Task Group members wish to record their appreciation to F. Schindel (GSF) for his substantial amount of work in the mathematical evaluations for the relative risk model, and to Irmgard Gerisch, Ingrid Goddeng and Ellen Reinhard for their technical assistance in the preparation of the manuscript. The Task Group members also wish to thank all their scientific colleagues for valuable suggestions relating to the exposure and risk analysis.

Members of the Task Group:

W. Jacobi (Chairman)
J. Lafuma
C. E. Land
H. G. Paretzke

1. INTRODUCTION

Some background information is presented here for those readers who are not familiar with the radon problem in houses and its biological significance.

1.1. The Physical Background

The radionuclides formed within the three natural decay series are principally radioisotopes of heavy metals, e.g. U, Th, Ra, Po, Bi, Pb. However, in each of these decay chains there is one link which is a radioisotope of the noble gas radon (Rn):

$^{238}U \rightarrow \ldots \rightarrow {}^{226}Ra \rightarrow {}^{222}Rn$ ("Radon") $\rightarrow \ldots$
$^{235}U \rightarrow \ldots \rightarrow {}^{223}Ra \rightarrow {}^{219}Rn$ ("Actinon") $\rightarrow \ldots$
$^{232}Th \rightarrow \ldots \rightarrow {}^{224}Ra \rightarrow {}^{220}Rn$ ("Thoron") $\rightarrow \ldots$

Furthermore, because the parent atoms of these decay chains can be found in all natural materials, from all surfaces of rocks, soils and building structures radon is released into the air. Of principal importance are the releases of ^{222}Rn (Radon, Rn), which has a radioactive half-life of 3.8 days, and of ^{220}Rn (Thoron, Tn), with a half-life of 55 s. Compared with these, the contribution of ^{219}Rn (Actinon, An) is negligible, due to its short half-life of 3.9 s and the low $^{235}U/^{238}U$ atomic ratio of 0.00725 in natural uranium. For this reason, the inhalation of actinon and its decay products is not discussed in this report.

Radon exhaled from the earth's surface into the free atmosphere is rapidly dispersed and diluted by vertical convection and turbulence. Considerably higher radon levels can occur if radon is released to confined air spaces, such as underground mines and houses. In such areas, the radon level increases with decreasing ventilation rate. Surveys in various countries have shown that the time-averaged concentration of ^{222}Rn in indoor air varies greatly from house to house, ranging from a few up to several thousand Bq/m^3.

Radon decays to a sequence of short-lived radionuclides, the so-called radon daughters, as shown in Fig. 1. Consequently, the release of radon into air leads also to a buildup of these short-lived daughters in air. As these nuclides are radioisotopes of Po, Bi and Pb, most of them become attached to dust particles, particularly to those in the sub-micron size range, forming a radioactive aerosol. Owing to surface deposition and ventilation, radioactive equilibrium between radon and its daughters is not reached in confined air spaces, such as mines or houses.

1.2. The Biological Background

Because of its lack of reactivity, inhaled radon is not chemically bound in body tissues. Furthermore, because its solubility in body tissues is rather low, the radiotoxicity of inhaled radon is relatively small compared with inhaled non-gaseous radionuclides, such as radon daughters (IC81).

In contrast, inhaled radon daughters are deposited in the respiratory tract. Because of their short radioactive half-lives, most of the deposited daughter atoms, except ^{212}Pb, the longest-lived daughter of ^{220}Rn, decay in the respiratory tract, giving rise mainly to a radiation dose to the bronchial epithelium. As some of these daughter atoms are alpha emitters, the relatively high biological effectiveness of alpha radiation has also to be taken into account. The

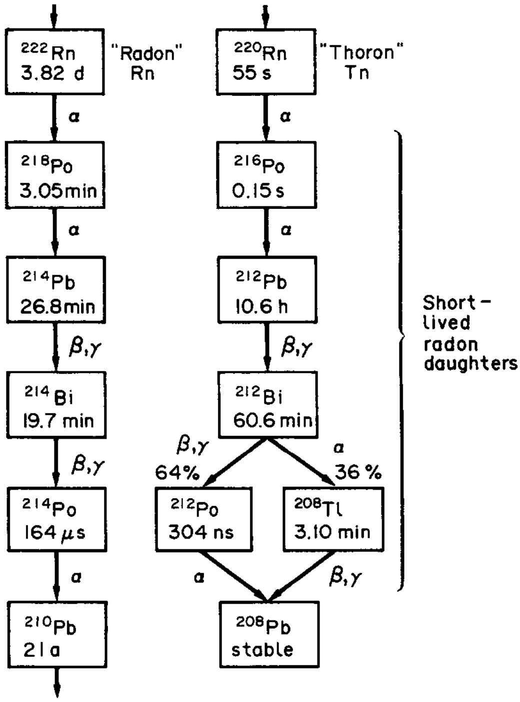

Fig. 1. The decay chains of ^{222}Rn and ^{220}Rn.

Commission has recommended a quality factor of 20 for alpha particles, compared with a mean quality factor of one for the beta, gamma-radiation of these daughter nuclides (IC77a;81).

Because of the enhanced level of radon daughters in indoor air, the estimated mean dose equivalent to the bronchial epithelium from indoor inhalation of radon daughters, averaged over the total population in the temperate latitudes, is about ten times higher than the mean total radiation dose equivalent to other body tissues from natural sources (UN82). Taking into account the great variation of radon daughter levels in indoor air, the bronchial dose for some individuals, living in houses with high radon levels, can reach considerably higher values.

So far, there are insufficient data on direct epidemiological studies on such population groups to allow a quantification of the possible risk of lung cancer, particularly bronchial cancer, which may be associated with the natural radiation exposure to inhaled radon daughters. In contrast, an increased frequency of bronchial cancer has been observed among various groups of underground miners who were exposed to radon during their underground work. In particular, the epidemiological studies among Rn-exposed uranium miners yield a clear correlation between their Rn-daughter exposure and excess lung cancer frequency, which cannot be explained by other influences. This finding is confirmed by experimental studies on Rn-exposed animals. The results of the epidemiological studies among Rn-exposed miners, taking into account appropriate corrections, can be used to estimate the possible lung cancer risk associated with the indoor exposure of the population to radon daughters.

1.3. Radiation Protection Principles

It should be emphasized that man, like any organism, has always been exposed to ionizing radiation from natural sources. One must be aware, however, that this natural exposure is not constant and that some components of it can be strongly influenced by human activities and living habits. This is particularly valid with respect to indoor exposure to radon and its short-lived daughters. This exposure is greatly dependent on the location, construction, type of building materials and ventilation of houses. Restricted ventilation leads to an increased radon level in indoor air. Thus, the indoor exposure to radon daughters is controllable to a large extent. This conclusion is valid both for existing and for future houses, although the degree of control and the types of control measures are different for the two situations.

The Commission has recently outlined the general principles for limiting exposure of the public to natural sources of radiation (IC84;Bo85). It has stated explicitly that the recommended annual dose limit of 1 mSv effective dose equivalent for chronic exposure of members of the public to artificial sources does not apply to natural sources, such as radon in houses. However, the controllable part of such natural exposures should be kept as low as reasonably achievable, economic and social factors being taken into account.

For existing houses with high Rn levels, the Commission proposes a system of remedial actions. In deciding whether to take action, it emphasizes that "the hazard or social costs involved in any remedial measure must be justified by the reduction of risk that will result". In addition, the decision on action levels for radon in houses is influenced by the degree to which the inhabitants are present voluntarily. The Commission has concluded that "for all these reasons, it would not be helpful to suggest a generally applicable value of an action level" for radon in houses. However, it states that "if the remedial action is fairly simple, an action level for the equilibrium equivalent concentration of ^{222}Rn in houses in the region of 200 Bq/m^3 might be considered. For severe and disrupting remedial action, a value several times larger might be more appropriate" (IC84). The definition of the quantity "equilibrium-equivalent concentration (EEC)" is given in the Appendix of this report.

With respect to the planning of future houses, the Commission recommends a limitation system similar to that for artificial sources. In this case, the exposure should be limited by the application of an upper bound for the acceptable radon level in houses. This upper bound should be lower than the action level for existing houses. The Commission believes "that a reasonable upper bound for the equilibrium-equivalent concentration of ^{222}Rn is of the order of 100 Bq/m^3 and that, in many countries, a value of this magnitude would prevent radon from becoming a dominating source of risk in dwellings" (IC84). It should be recognized that the setting of such an upper bound may influence building standards for the siting, construction and ventilation of future houses.

1.4. Objectives and Structure of the Report

The considerations set out above indicate that the perception and judgment of the radon problem in houses, and the decision-making for reasonable regulations or actions, are strongly linked with the assessment of the possible lung cancer risk associated with this natural radiation exposure. Such a risk analysis is the main objective of this report. This analysis relates mainly to the induction of bronchial cancer. In this report, the term "lung cancer" is used synonymously with "bronchial cancer", as specified in the International Classification of Diseases (ICD(8)—162).

In Sections 2 and 3, current knowledge on sources and levels of radon and its daughters in indoor air is summarized and the resulting doses to target tissues in the lung are estimated. In Section 4, the basic epidemiological and experimental findings on radiation-induced lung cancer are outlined. Main emphasis is given to the analysis of the results of the epidemiological studies on Rn-exposed uranium miners. On the basis of these findings, in Section 5, different concepts and models are described which can be used to estimate the individual risk and the population-related frequency of lung cancer from inhaled ^{222}Rn and ^{220}Rn daughters. In addition to the usually-applied absolute risk projection model, the Task Group opted to consider also a relative risk projection model. This latter model enables a simpler application of the data on miners to the different exposure conditions of populations, taking into account the chronic exposure rate, the age-dependency of the appearance rate of lung tumours and the possible synergistic influence of cigarette smoking. In Section 6, the results of the different risk models, as a function of the concentration of radon and its daughters in indoor air or the corresponding annual exposure, are given and compared, with the main emphasis being on the conclusions which can be drawn from the relative-risk projection model.

The reader's attention is drawn to the Appendix to this report. This defines the special quantities and units which are used to characterize concentrations of radon and its daughters, and exposures to these radionuclides.

2. SOURCES AND LEVELS

In this chapter, a short summary of sources and levels of $^{222}Rn(Rn)$, $^{220}Rn(Tn)$ and their short-lived daughters in indoor and outdoor air is presented, giving main emphasis to the activity balance in houses. For more detailed information, the reader is referred to several review reports (UN82; NC84a,b) and to recently published proceedings and journal issues on this subject (HP83; RP84; SE85).

The radon concentration in air is given in the unit Bq/m^3; the concentration of a radon daughter mixture is expressed in terms of the equilibrium-equivalent concentration (EEC) of ^{222}Rn or ^{220}Rn, as appropriate. The special quantities and units which are commonly in use to characterize the concentration of radon daughters are described in the Appendix to this report.

2.1. Radon Sources in Houses

Sources of radon in houses are:

—the radon exhalation from building materials;
—the radon influx from the underlying soil;
—the radon infiltration from outdoor air due to ventilation;
—the radon released from water supplies and from natural gas used for heating and kitchen appliances.

Usually, only the first three of the sources mentioned are of importance. Radon entry via water and natural gas need only be considered if their radon content is unusually high.

The radon entry rate from a given source is expressed in terms of the rate of supply of radon activity to the considered indoor area per unit time and per unit indoor air volume. The estimated ranges of the ^{222}Rn entry rates from different sources in typical houses are summarized in Table 1.

2.1.1. *Entry from building materials*

Only a small fraction of the total ^{222}Rn activity produced by decay of ^{226}Ra in building structures diffuses to wall surfaces and is released into indoor air. This fraction depends strongly on the emanating power and porosity of the building materials, as well as the bulk diffusion coefficient of radon in them. Due to the influence of these parameters, the rate of ^{222}Rn entry

Table 1. Volume-specific entry rate and concentration of ^{222}Rn in indoor air from different sources; predicted mean range and normal range of variation (excluding extreme values)

Source	Specific entry rate (Bq/m^3h) Estimated mean	Range of variation	Indoor concentration[a] (Bq/m^3) Estimated mean	Range of variation
Building materials				
brick or concrete houses	2–20	1 –50	3–30	0.7 –100
wooden houses	<1	0.05–1	≤1	0.03–2
Soil	1–40	0.5 –200	2–60	0.5 –500
Outdoor air	2–5	0.3 –15	3–7	1 –10
Other sources (water, natural gas)	≤0.1	0.01–(10)	≤0.1	0.01–(10)
All sources	6–60	2 –200	10–1000	2 –500

[a] Referring to a mean ventilation rate of 0.7 h^{-1} (normal range 0.3–1.5 h^{-1}).

from building materials cannot be predicted with sufficient accuracy from their ^{226}Ra content alone.

However, the rate of entry can be estimated from the exhalation rates which have been measured for different building materials. These data indicate, for houses built with ordinary building materials, a specific ^{222}Rn entry rate of 0.05–50 Bq/m^3h (UN77,82; Cl83; In83; Ne84,85a). For wooden houses, a value in the lower part of this range, less than 1 Bq/m^3h, is typical.

Exceptionally high entry rates are associated with houses built using concrete based on alum-shales with extremely high ^{226}Ra contents, as in Sweden, for which entry rates of up to 500–1000 Bq/m^3h have been observed (Sw84; UN82). Also, in houses built with tuff, phosphate slags or phosphogypsum, entry rates from building structures of up to several hundred Bq/m^3h can occur.

From measurements in the USA (Ne84,85b), the UK (Cl83) and the FRG (Bl86), the frequency distribution of ^{222}Rn entry rates from building structures can be roughly estimated. On the basis of these studies, a mean entry rate of 2–20 or less than 1 Bq/m^3h should be expected for concrete and brick houses or for wooden houses, respectively (In83; Na84; UN82).

In addition, the release of ^{220}Rn (Tn) from building materials has to be considered. The ^{232}Th content of normal building materials (concrete, bricks) is 10–100 Bq/kg which, on an activity basis, is comparable with their ^{226}Ra content. Because of its short half-life of 55 s, only ^{220}Rn atoms produced in the inner surface layer of building structures can diffuse into indoor air. In the past, only a few data on the release of ^{220}Rn from building materials were available (UN82). Recent measurements (Fo84) indicate, for normal concrete and brick houses, an average entry rate of 100–500 Bq/m^3h (range of variation 50–5000 Bq/m^3h). This means that, in such houses, the rate of ^{220}Rn-activity release from building materials into indoor air might be about a factor 10–100 higher than for ^{222}Rn(Rn). However, this ^{220}Rn release rate is strongly influenced by the wall covering (e.g. plaster, paints). In wooden houses, the ^{220}Rn entry rate is probably at the lower end of the range of variation given above.

2.1.2. *Entry from soil*

The mean ^{222}Rn concentration in the soil air is about 1000 times higher than the ^{222}Rn concentration in free atmospheric air near ground level. Thus, if substantial transport of radon from the underlying soil into indoor air can occur, it can lead to rather high indoor concentrations. Possible transport mechanisms are molecular diffusion and convective flow. Of most importance is the latter process, which is induced by pressure differences between soil and indoor air (Ea84; Ne83a,b,84,85a; Sw84). These pressure differences arise from the thermal stack effect, caused by the difference in temperature between indoors and outdoors, and from the wind laoding on the building.

The resulting entry rate from soil depends strongly on the permeability of the underlying soil and of the understructure of the house, taking into account cracks and openings. It can, therefore, vary markedly from house to house. Direct measurements of the radon entry rate from soil into indoor air are not available. Considering the uncertainties of the parameters involved, a theoretical prediction of this source term for specific houses is difficult. The possible contribution of Rn entry from soil can only be estimated from the observed total entry rate, subtracting the expected contribution from building materials.

Simultaneous measurements of the ^{222}Rn concentration in indoor air and of the ventilation rate of houses, which have been carried out in several countries, yield a broad distribution function for the total entry rate, with country-averaged mean values in the range from 6 to 60 Bq/m^{3}h (Ne85a); an exception might be Sweden, where a somewhat higher value was found. Subtracting the estimated mean contributions from other sources, particularly from building materials (see Table 1), the country-averaged mean values of the ^{222}Rn entry rate from soil cover a range of about 1 to 40 Bq/m^{3}h.

In general, in houses with a tightly-sealed substructure, the entry rate from soil will normally be less than 10 Bq/m^{3}h. Cracks and openings, however, can lead to much higher values. Particularly in areas with high natural soil activity, as in Sweden, entry rates of up to about 1000 Bq/m^{3}h have been observed, which can only be explained by the convective transport of ^{222}Rn from the underlying soil.

Taking into account its short radioactive half-life, the entry rate of ^{220}Rn from soil is probably lower than that of ^{222}Rn. However, in houses with no efficient barriers between soil and indoor air space, it cannot be excluded that the rate of ^{220}Rn entry from soil might be comparable with that of ^{222}Rn (Sc85a).

2.1.3. *Contribution from outdoor air*

The distribution of ^{222}Rn, ^{220}Rn and their short-lived daughters in atmospheric air is determined by radon and thoron exhalation from the soil surface and meteorological distribution processes. Therefore, their concentrations near ground level show strong local and temporal variations. The available measurements indicate, for areas with normal natural soil activity, long-term mean concentrations of 1–10 and 0.5–20 Bq/m^3 for ^{222}Rn and ^{220}Rn, respectively, in the air layer 1–10 m above ground level (UN82; Ge83; NC84a). With respect to the global situation, a mean value of 3–7 Bq/m^3 for both radon and thoron should be expected.

The specific entry rate i_o of radon from outdoor air into houses due to ventilation is given by the relationship:

$$i_o = \lambda_v a_o \tag{1}$$

where a_o is the radon concentration in outdoor air, and λ_v is the air exchange rate constant between outdoor and indoor air.

The ventilation rate of houses can be strongly influenced by personal living habits (opening of windows and doors). In naturally ventilated houses, it also depends on the meteorological conditions; it increases with the wind speed and the temperature gradient between indoor and outdoor air. A time-averaged ventilation rate constant λ_v of 0.3–1.5 per hour seems to be typical of most houses.

For a ^{222}Rn concentration of 1–10 Bq/m^3 in outdoor air, eqn (1) yields a typical variation range for the entry rate from outdoor air of about 0.3–15 Bq/m^3h. Assuming a mean ventilation rate of 0.7 h^{-1}, averaged over all houses, gives a mean entry rate of 2–5 Bq/m^3h. With respect to ^{220}Rn, the mean entry rate from outdoor air is probably somewhat lower, by a factor of about two.

2.2. Predicted Radon Balance in Indoor Air

In Table 1, the predicted normal variation range and the estimated mean value of the specific entry rate of ^{222}Rn from different sources are summarized, as they follow from the considerations in the previous section. As noted, extreme values, which might occur particularly in houses with a highly-permeable floor structure built on grounds with high ^{226}Ra-content and porosity, are excluded.

Summing the contributions from these source terms, a mean entry rate of 6–60 Bq/m^3h is estimated (Ne85a). For houses with ground floor accommodation, the soil is likely to be the strongest source. This is particularly so for wooden houses.

Assuming a homogeneous mixing in the indoor air volume, the activity concentration of radon in indoor air, a, at steady-state equilibrium, can be calculated from the relationship

$$a = \frac{i}{\lambda_r + \lambda_v} \simeq \begin{cases} i/\lambda_v & \text{for } ^{222}\text{Rn} \\ i/\lambda_r & \text{for } ^{220}\text{Rn} \end{cases} \tag{2}$$

where i is the total specific entry rate of radon from all sources, λ_v is the time-averaged ventilation rate constant, and λ_r is the radioactive decay constant.

Because the radioactive decay constant of ^{222}Rn is only 0.00755 h^{-1}, $\lambda_r \ll \lambda_v$ and the ^{222}Rn concentration is nearly inversely proportional to the ventilation rate, unless the entry rate from soil and building structures is influenced by the ventilation. In the case of ^{220}Rn ($\lambda_r = 45.4\ h^{-1}$), $\lambda_r \gg \lambda_v$ and the concentration should be rather independent of the ventilation rate.

In the second section of Table 1, the predicted contributions of different sources to the indoor ^{222}Rn concentration are given. These are based on the estimated entry rates, together with a mean ventilation rate of 0.7 h^{-1} (range 0.3–1.5 h^{-1}). On the basis of this simplified model, a mean total indoor concentration in the range of 10–100 Bq/m^3 is predicted. This is about 2–20 times higher than the mean outdoor concentration. The derived total variation range of 2–500 Bq/m^3, excluding extreme situations, covers more than two orders of magnitude. This considerable variation is mainly due to the influence of radon entry from building materials and soil. However, values exceeding 100 Bq/m^3 should normally only be expected in houses with a strongly enhanced rate of radon entry from soil. Exceptions are houses with highly radioactive additions in their structural materials, such as houses built of alum-shale concrete with a high ^{226}Ra content, as found in Sweden (Sw84).

In the case of ^{220}Rn (Tn), the major source is probably the release from building materials. Inserting the estimated ^{220}Rn entry rate for houses built of normal concrete or bricks (see Section 2.1.1), eqn 2 implies a ^{220}Rn concentration in indoor air of such houses in the range of 1–100 Bq/m^3, with a mean value of about 2–20 Bq/m^3. This is a factor of about 5 lower than the predicted values for ^{222}Rn in indoor air (see Table 1). The possibility cannot be excluded that, in the basement or ground floor of houses with a highly-permeable floor structure, higher values could occur. Further studies are required to confirm these preliminary estimates.

2.3. Buildup of Radon Daughters; Equilibrium Factor

The buildup of radon daughters in indoor air depends on the radon entry rate, the ventilation rate and ventilation pattern within the house, and on the deposition rate of radon daughters on surfaces (floors, walls, furniture etc.). The resulting concentration of short-lived radon daughters, expressed in terms of the equilibrium-equivalent radon concentration (EEC_{Rn}), is related to the activity concentration a_{Rn} of radon by the relationship

$$EEC_{Rn} = Fa_{Rn} \tag{3}$$

where F is the so-called equilibrium factor (see Appendix).

Due to ventilation and deposition, no radioactive equilibrium between radon and its daughters is reached in indoor air. The results of simultaneous measurements indicate a mean value of the equilibrium factor for ^{222}Rn daughters in indoor air in the range of 0.3–0.6 (Ca84; Ge80; Ke82,84; Le84; Mc80; Po78,84; St80; Sw83; Wi82; BI86). This factor increases with decreasing ventilation rate and increasing aerosol concentration in indoor air. The available measurements indicate that the variation of the equilibrium factor with the absolute activity concentration is rather small. In outdoor air, the mean equilibrium factor is somewhat higher than in indoor air (UN82).

In this report, representative mean values of the equilibrium factor for ^{222}Rn daughters in indoor and outdoor air are assumed to be 0.45 and 0.7, respectively. The value for indoor air was selected as the mid-point of the range of reported values. Taking into account the predicted ^{222}Rn concentration in indoor air (see Table 1), a mean ^{222}Rn-daughter concentration corresponding to an EEC_{Rn} of about 5–50 (range of variation 1–250) Bq/m^3 is estimated for houses associated with normal levels of natural radionuclides in soil and building materials.

With respect to ^{220}Rn (Tn) daughters, theoretical estimates indicate a mean $^{212}Pb/^{220}Rn$ activity ratio, or equilibrium factor, of 0.02–0.1 in indoor air (UN82; Po84). Inserting a mean ^{220}Rn level of 2–20 Bq/m^3, as has been derived from the estimated ^{220}Rn entry rates (see Section 2.2), an average ^{212}Pb concentration of about 0.04–2 Bq/m^3 is predicted for indoor air of normal houses. As shown below, this theoretical estimate is in accord with the few available direct measurements in indoor air.

2.4. General Results of Surveys in Houses

The above considerations demonstrate that theoretical models enable only a rough prediction of the concentration of radon and its daughters in indoor air. To obtain more reliable data, during the last 10 years several larger measurement programmes on radon (^{222}Rn) in houses have been started. The preliminary results of these surveys, until 1981, are summarized in the report of UNSCEAR (UN82). More recent data have been published in the proceedings of special conferences and other review issues of journals (HP83; RP84; SE85). Here, a brief description is given of the general findings which are relevant to the exposure and risk analysis undertaken in this report.

As typical examples, Fig. 2 shows the frequency distributions of the ^{222}Rn concentration in indoor air of dwellings as derived from larger surveys in Canada (Mc80; Le84), the Federal Republic of Germany (Sc85b; BI86), Italy (Sc85c), the Netherlands (Pu85), the Nordic Countries (Ca84,85; Sw84; St86a), the United Kingdom (Wr84,85; Gr85) and the United States of America (Ne85b). In Fig. 2, the complementary cumulative frequency is plotted on a Gaussian probabilistic scale versus the logarithm of the radon concentration. Thus, for a given radon level the ordinate gives the expected percentage of dwellings in which the level specified

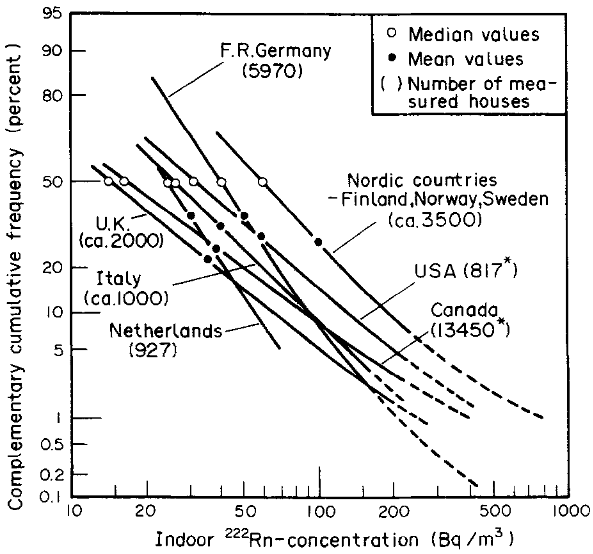

Fig. 2. Complementary cumulative frequency distribution of the ^{222}Rn-concentration in dwellings, estimated from the results of surveys in several countries (* single family houses only).

on the abscissa might be exceeded. In the figure, the resulting median and mean values are also marked.

In general, the measured frequency distribution of indoor Rn levels can be approximated by a log–normal distribution (corresponding to a straight line on the type of plot shown in Fig. 2. However, most of these surveys indicate a deviation towards higher frequencies at high radon levels (see Fig. 2). The investigations confirm that in most houses where high ^{222}Rn levels ($>$100–200 Bq/m^3) have been observed, the main cause is high rates of radon entry from the underlying soil (see Table 1); only in houses built with alum-shale concrete of high ^{226}Ra content do very high Rn levels results mainly from building materials.

For most countries where larger and randomly-distributed surveys have been made, the observed frequency distributions indicate a mean ^{222}Rn concentration in the indoor air of dwellings in the range of 20–60 Bq/m^3. Using a mean equilibrium factor $F = 0.45$, this yields a mean concentration of ^{222}Rn daughters in indoor air corresponding to an equilibrium-equivalent ^{222}Rn-concentration in the range of 9–30 Bq/m^3. Exceptions seem to be houses in the Nordic countries (Finland, Norway, Sweden), where higher mean values are estimated (see Fig. 2).

It can be assumed that the relative frequency distribution of ^{222}Rn-daughter levels in houses is similar to that of ^{222}Rn. Figure 3 shows the resulting complementary cumulative frequency distribution of the indoor concentration of ^{222}Rn daughters. The shadowed area indicates the expected range of variation in areas with normal background radioactivity. Averaged over the whole population in the temperate regions, the available data indicate, as a best estimate, the log–normal distribution function marked as "reference distribution" in Fig. 3. This distribution is based on a mean value of the equilibrium-equivalent concentration of ^{222}Rn in indoor air of 15 Bq/m^3, in accordance with the conclusion drawn in the report of UNSCEAR (UN82). In Table 2, the parameters of this log–normal reference distribution of ^{222}Rn daughters are listed. In addition, the expected percentages of dwellings exceeding certain levels are also given.

Compared with ^{222}Rn (Rn) and its daughters, very few measurements of ^{220}Rn (Tn) daughters in indoor air have been reported (Gu82; Ke82; St75; Wi79; UN82). They indicate an

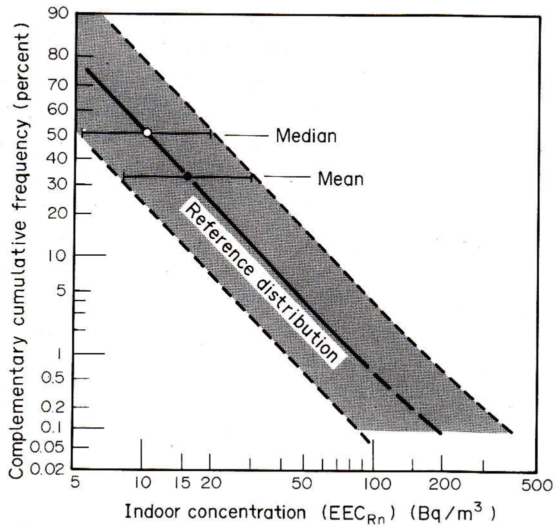

Fig. 3. Complementary cumulative frequency distribution of the equilibrium-equivalent ^{222}Rn-concentration in indoor air of dwellings; the shadowed area indicates the region which is covered by the distributions resulting from surveys in most countries of the temperate regions.

Table 2. Reference values of parameters for the frequency distribution of ^{222}Rn daughter concentrations in indoor air of private dwellings; concentrations are given in terms of the equilibrium-equivalent concentration of ^{222}Rn

Parameter		Reference value
Median concentration (Bq/m^3)		10
Mean concentration (Bq/m^3)		15
Equilibrium factor F		0.45
Rel. geom. standard deviation		2.5
Resulting percentage of dwellings with a level exceeding (in Bq/m^3):	50	5
	100	1
	200	ca. 0.1
	400	< 0.01

average equilibrium-equivalent concentration (EEC$_{Tn}$) in the range from 0.1–1 Bq/m^3, which is mainly attributable to ^{212}Pb. This result agrees with the expected range derived from the ^{220}Rn (Tn) exhalation of building materials (see Section 2.2). In this report, a mean EEC$_{Tn}$ of 0.5 Bq/m^3 for the population exposure to ^{220}Rn daughters in houses is assumed; this is about a factor of 2–3 higher than the mean level in outdoor air.

By comparison with the reference value for the mean indoor level of 15 Bq/m^3 for ^{222}Rn daughters, it follows that the ratio EEC$_{Tn}$/EEC$_{Rn}$ in indoor air has a mean value of 0.03. Using the conversion factors given in the Appendix, this corresponds to a ratio of potential alpha energy concentrations between ^{220}Rn (Tn) and ^{222}Rn (Rn) daughters of about 0.4. Simultaneous measurements of ^{220}Rn and ^{222}Rn daughters in the indoor air of houses with normal levels confirm this conclusion (Ke82; Wi79).

At the end of this chapter, some general remarks are necessary. It should be emphasized that most of the surveys for radon and radon daughters in houses are still not completed. Thus, only preliminary results are available. Furthermore, the methods for the selection of houses involved in these studies, and the type of measuring methods, were different. Thus, the preliminary results from these surveys and their comparability must be considered with some caution. Particularly, it has to be clarified whether the observed frequency distributions and their mean values are representative for the whole population in the considered region or country.

Furthermore, it should be recognized that the measured Rn levels in single houses refer to the conditions during the smapling period. For the estimation of the corresponding individual lung cancer risk, however, the cumulative lifetime exposure of the inhabitants of these houses must be known, taking into account their mobility and residence times. There are strong indications that new heating systems and energy-saving measures have led to reduced ventilation and consequently to an increase of the mean radon level in houses during recent decades. For some countries, an increase by a factor of about two to four has been suggested (Ha84; UN82; Sw86).

These surveys agree with the conclusion that, in most houses where strongly enhanced indoor ^{222}Rn levels have been measured, the main source is ^{222}Rn entry from soil.

3. EXPOSURE AND DOSE TO LUNG TISSUES

The lung cancer risk from inhaled radon daughters depends on the cumulated exposure to radon daughters and the associated dose to lung tissues as functions of time or age. In this chapter, methods for the evaluation of exposure and dose as functions of the radon daughter concentration in air are outlined, and reference conversion factors are given. Main emphasis is given to indoor exposure to ^{222}Rn daughters.

The quantity "radon daughter exposure" is generally defined as the time integral of the radon daughter concentration in the inhaled air. In this report, the radon daughter concentration is represented by the quantity equilibrium-equivalent concentration (EEC) of ^{222}Rn or ^{220}Rn, respectively. Also, in this report, the unit used for equilibrium-equivalent radon concentration integrated over time is Bq h/m^3. In the literature, the exposure is often expressed in terms of the time integral over the potential alpha energy concentration of the daughter mixture in the inhaled air. The SI-unit for this quantity is J h/m^3. Also used is the unit Working Level Month (WLM); where 1 WLM corresponds to 0.0035 J h/m^3. The relationships between these different exposure quantities and units are explained in the Appendix.

3.1. Evaluation of Annual Exposure

Three components of the radon daughter exposure have to be distinguished:

—indoor exposure at home (index ih);
—indoor exposure elsewhere (index ie);
—exposure during residence in outdoor areas (index o).

The exposure to radon daughters depends on the residence time in these areas. In the report of UNSCEAR (UN82), a mean residence probability p_i of 0.8 within houses, corresponding to about 7000 h per year, was assumed. This value refers to the total residence probability indoors, without specifying the contributions from residence at home and elsewhere.

In the United Kingdom, a survey on the mean residence probability of individuals in private houses, other buildings (mainly during occupational work), and out of doors as a function of time of day has been carried out (Br83). On the basis of these data, a residence model was chosen which is shown in the upper graph of Fig. 4. As a long-term average, this model yields mean

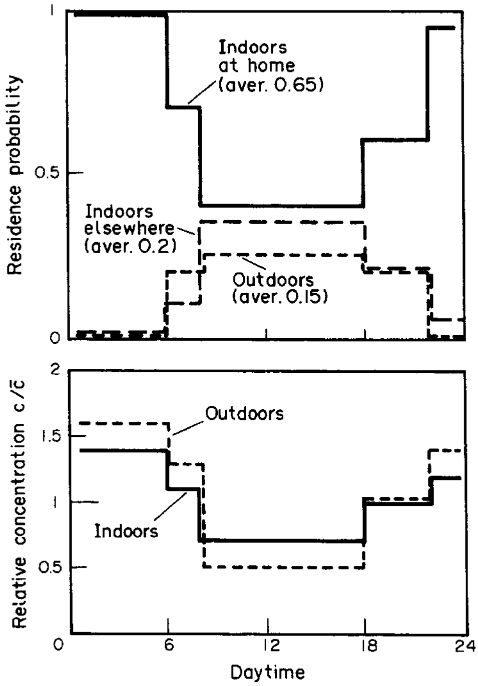

Fig. 4. Mean diurnal variation of residence probabilities (upper graph) and of the relative radon-daughter concentration (lower graph), indoors and outdoors, assumed in the specified exposure model.

residence probabilities of 0.65, 0.2 and 0.15 in private homes, other buildings and outdoor air, respectively. This would correspond to mean residence times of 5700, 1750 and 1300 h per year in these areas.

In most surveys, the long-term mean concentration of radon or radon daughters is measured. Neglecting the diurnal variation of the radon daughter concentration indoors and outdoors, this simplified residence model yields an annual equilibrium-equivalent exposure, E, in Bq h/m^3 (Rn- or Tn-eq):

$$E = 5700\ h \times \bar{c}_{ih} + 1750\ h \times \bar{c}_{ie} + 1300\ h \times \bar{c}_{o} \tag{4}$$

In this equation, $\bar{c}_{ih}$, $\bar{c}_{ie}$ and $\bar{c}_o$ are the time averaged mean values of the equilibrium-equivalent concentration (EEC), in Bq/m^3, in the air of private homes, other buildings, and in outdoor air, respectively.

The available measurements indicate a relatively large diurnal variation of the radon daughter concentration in indoor and outdoor air (Ge83; St80; UN82). As an annual average, the concentration outdoors during the night seems to be about a factor 2–5 higher than around noon; in indoor air this diurnal variation is probably less pronounced. To investigate the implications of this diurnal variation, a more specific exposure model was examined. It includes diurnal variation of the relative radon daughter concentration $c(t)/\bar{c}$, as shown in the lower graph of Fig. 4. Taking into account the corresponding residence probabilities, this model yields an annual radon daughter exposure:

$$E = 6400\ h \times \bar{c}_{ih} + 1400\ h \times \bar{c}_{ie} + 1000\ h \times \bar{c}_{o} \tag{5}$$

Compared with eqn (4), this model leads to a somewhat higher contribution from the indoor exposure at home, due to the higher concentration overnight; correspondingly, the other contributions are somewhat lower. However, with respect to indoor exposure, the differences of the annual exposure calculated from the two exposure models do not exceed 20–30%.

Taking into account the uncertainties involved in exposure estimates, the Task Group recommends the following reference relationship for the evaluation of the annual exposure to ^{222}Rn and ^{220}Rn daughters:

$$E = 6000\ h \times \bar{c}_{ih} + 1500\ h \times \bar{c}_{ie} + 1000\ h \times \bar{c}_{o} \tag{6}$$

with E in Bq h/m^3 and the equilibrium-equivalent concentrations, $\bar{c}$, in Bq/m^3. For a person not engaged in employment outside the house, the coefficient of the first term is about 20% higher, but the second term is correspondingly reduced.

In Table 3, the mean annual exposures from the different residence areas are listed for the proposed reference values of the mean concentrations of ^{222}Rn and ^{220}Rn daughters in indoor and outdoor air (see Section 2.4 and Table 2). These result in a total annual exposure of about

$$1.2 \times 10^5\ \text{Bq h/m}^3 \equiv 0.66\ \text{mJ h/m}^3 \equiv 0.19\ \text{WLM} \tag{7}$$

from ^{222}Rn daughters and about

$$4 \times 10^3\ \text{Bq h/m}^3 \equiv 0.30\ \text{mJ h/m}^3 \equiv 0.09\ \text{WLM} \tag{8}$$

from ^{220}Rn daughters. About 75% of this exposure is associated with indoor residence in private homes; only about 5% is due to outdoor exposure.

With respect to individuals living in private homes with strongly enhanced radon levels, it seems rather improbable that they are exposed to similarly high levels during their residence

Table 3. Estimated mean annual exposure to radon daughters for the given reference values of the mean concentration in indoor and outdoor air

Contribution from residence	Mean equil. equiv. concentration (Bq/m^3)	Annual exposure[a]: Equivalent equilibrium exposure (10^3Bq h/m^3)	Annual exposure[a]: Potential alpha energy exposure (mJ h/m^3)	Potential alpha energy exposure (WLM)
^{222}Rn daughters (^{218}PO–^{214}PO):				
Indoors				
at home	15	90	0.51	0.14
elsewhere	15	23	0.13	0.036
Outdoors	4	4	0.022	0.0064
Total, ^{222}Rn daughters		120	0.66	0.19
^{220}Rn daughters (^{212}Pb–^{212}Po):				
Indoors				
at home	0.5	3.0	0.23	0.065
elsewhere	0.5	0.75	0.057	0.016
Outdoors	0.2	0.2	0.015	0.0043
Total, ^{220}Rn daughters		4.0	0.30	0.09

[a] Exposure conversion factors:
1 Bq h/m^3(Rn-eq.) ≡ 0.00562 μJ h/m^3 ≡ 1.60×10^{-6}WLM.
1 Bq h/m^3(Tn-eq.) ≡ 0.0758 μJ h/m^3 ≡ 2.16×10^{-5}WLM.

time in other buildings, particularly at their working place. Usually, it can be assumed that the radon levels in these buildings will be comparable with the regional- or country-averaged mean indoor concentration. Under these conditions, the total annual ^{222}Rn-daughter exposure of such population groups can be roughly estimated from the relationship

$$E \approx 30\,000 \text{ Bq h/m}^3 + 6000\, h \times \bar{c}_{ih} \quad (9)$$

where $\bar{c}_{ih}$ is the long-term mean value of the equilibrium-equivalent concentration of ^{222}Rn in their private homes, in Bq/m^3. Figure 5 gives a graphical presentation of this relation. For such population groups, living in homes with enhanced ^{222}Rn levels, the additional contribution from ^{220}Rn daughters is small.

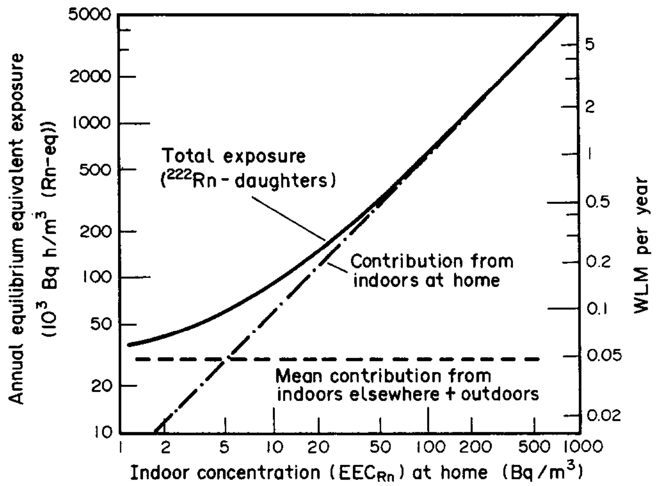

Fig. 5. Annual, equilibrium-equivalent ^{222}Rn-exposure as a function of the indoor concentration at home, assuming normal contributions from residence indoors elsewhere and outdoors.

3.2. Lung Dosimetry

With respect to lung carcinogenesis, the cells at risk are located in the basal and mucus cell layer of the tracheobronchial epithelium and in the alveolar tissue (IC80,81). Therefore, the lung dosimetry for inhaled radon daughters has to relate mainly to two target tissues. These are the tracheobronchial epithelium and the pulmonary region, with the latter including the alveoli and the non-ciliated terminal bronchioles.

In recent years, improved models for the lung dosimetry of inhaled radon daughters have been developed. A comprehensive review and comparison of these models and their results has been prepared by an expert group of the NEA/OECD (NE83). There, and in a NCRP report (NC84b), a sensitivity analysis was carried out to study the influence of the various physical, anatomical and physiological parameters on the dose distribution in the human respiratory tract; an updated summary has recently been published by James (Ja84c).

In general, all dosimetric models agree with the conclusion that the alpha dose to the bronchial target tissue from inhaled ^{222}Rn daughters is about a factor of 5–10 higher than the pulmonary alpha dose. This dosimetric result is in accord with the histological finding, among Rn-exposed miners, that the observed carcinomas originate in the bronchial epithelium (see

Section 4.2). With respect to the lung cancer risk to populations from natural exposure to radon daughters, the induction probability of bronchial carcinoma is of most interest.

For typical exposure conditions, the dose to the basal cell layer of different sizes of bronchial airways varies by a factor of less than five (Ho82; Ha82; Ja84c; NE83; NC84b). Taking into account the uncertainties involved in the estimation of the dose distribution in the bronchial tree and in the location of critical cells, it seems reasonable to consider the mean dose in the critical cell layer of the bronchial epithelium, averaged from the main bronchi down to the subsegmental bronchi. With respect to this mean bronchial dose, the results of the different dosimetric models for given exposure conditions agree to within a factor of two. The term "bronchial dose", used in this report, refers to this average dose and is identified by the subscript B. For a given radon daughter exposure, the bronchial dose to an individual depends mainly on the anatomic structure of the bronchial tree, the breathing pattern, the fraction f of unattached daughter atoms in the inhaled air, and on the activity median diameter (AMD) of the carrier aerosol of the attached daughter atoms.

In general, the bronchial dose equivalent to exposure ratio, H_B/E, increases nearly linearly with the unattached fraction, f, of the total inhaled potential alpha energy of the daughter mixture. This unattached fraction is relatively small, with a long-term average value in the range 0.01–0.05 (1–5%) in indoor and outdoor air (UN82; NE83; Ja84c; Po84). Also, the ratio H_B/E increases with decreasing AMD. Because both f and AMD are influenced by the aerosol concentration, it is normally found that an increase of the ventilation rate leads to an increase of the bronchial dose to exposure ratio. The influence of these parameters on the lung dosimetry of inhaled radon daughters has been summarized by James (Ja84c).

Based on this review, Fig. 6 shows, for reference adults, the estimated mean bronchial dose equivalent per unit of indoor exposure to ^{222}Rn daughters as a function of breathing rate, taking into account a mean quality factor of 20 for alpha radiation (IC77a). The mean values shown represent an average of best estimates derived from different dosimetric models (Ha82; NE83; NC84b; Ja84c). These best estimates refer to an AMD of 0.15 μm and an unattached fraction, f, of 0.03 of the total potential alpha energy of ^{222}Rn daughters in indoor air; these parameter values are believed to be representative of the long-term conditions in most houses. The error

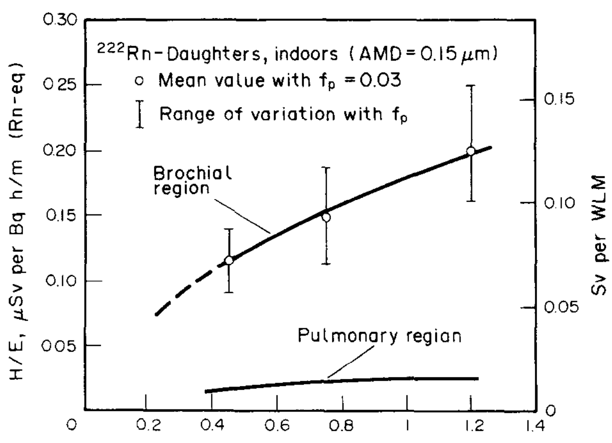

Fig. 6. Mean dose equivalent H to the bronchial and pulmonary region per unit of equilibrium-equivalent radon exposure, E, to ^{222}Rn daughters in indoor air as a function of breathing rate; best estimate for adults from different dosimetric models.

bars indicate the expected variation in these dose coefficients for a range of values of f from 0.01 to 0.05. For the pulmonary region, identified by the subscript P, the dosimetric models for ^{222}Rn daughters yield a dose to exposure ratio, H_P/E, which is a factor of 5–10 lower than the ratio H_B/E for the bronchial target tissue B. In this report, a mean dose ratio of 1/8 is assumed for H_P/H_B.

3.3. Reference Dose Coefficients

In the report of the ICRP Task Group on "Reference Man", reference values for the breathing rate were proposed (IC75). Taking into account the residence probabilities indoors and outdoors (see Fig. 4), mean daily breathing volumes of about 10, 5 and 4 m^3 result for the adult specified herein, during residence indoors at home, indoors elsewhere, and outdoors, respectively. Thus, for the total indoor residence, a breathing volume of about 15 m^3 per day and a mean breathing rate of 0.75 m^3/h can be assumed, in accordance with the value used in the report of UNSCEAR (UN82). For the outdoor residence period, a mean breathing rate of about 1 m^3/h is applicable.

Allowing for the described residence and breathing patterns and the best estimates of the bronchial dose as a function of breathing rate, given in Fig. 6, conversion coefficients between the long-term mean radon daughter exposure and the dose equivalent to target tissues in the lung can be derived. On the basis of this analysis, the Task Group suggests, for adults, the reference values of the dose to exposure ratio given in Table 4, which are in good agreement with the conclusions drawn in the report of UNSCEAR (UN82).

With respect to ^{220}Rn daughters, the dosimetric models yield, for an AMD of 0.2 μm, a bronchial dose equivalent in the range of $(20\text{–}80) \times 10^{-5}$ mSv per Bq h/m^3 exposure (NE83). The dose to the pulmonary tissue is a factor 3–5 lower. The proposed reference dose coefficients listed in Table 4 refer to the middle of this range.

Table 4. Mean dose conversion coefficients for indoor and outdoor exposure of adults to radon daughters (reference conditions)

	Dose equivalent per unit equilibrium-equivalent exposure[a] (mSv per Bq h/m^3)			Annual dose equivalent per unit equilibrium-equivalent concentration[b] (mSv per Bq/m^3)		
Residence area	H_B/E	H_P/E	H_E/E^c	H_B/c	H_P/c	H_E/c^c
^{222}Rn(Rn) daughters						
indoors at home				0.90	0.12	0.061
indoors elsewhere	15×10^{-5}	2.0×10^{-5}	1.0×10^{-5}	0.23	0.030	0.016
outdoors	20×10^{-5}	2.7×10^{-5}	1.4×10^{-5}	0.20	0.027	0.014
^{220}Rn(Tn) daughters						
indoors at home				3.0	0.90	0.23
indoors elsewhere	50×10^{-5}	15×10^{-5}	3.9×10^{-5}	0.75	0.23	0.06
outdoors				0.50	0.15	0.04

[a] To obtain H/E-values in mSv/WLM the listed values have to be multiplied by a factor of 6.3×10^5 (Rn daughters) or 4.63×10^4 (Tn daughters).

[b] Calculated using the exposure/concentration ratios in Table 3.

[c] Including only the dose contributions to the bronchial epithelium and the pulmonary tissue, using a weighting factor of 0.06 for each of these tissues.

In addition, Table 4 includes conversion coefficients for the effective dose equivalent, H_E, calculated as

$$H_E = 0.06\ H_B + 0.06\ H_P \tag{10}$$

in which weighting factors of 0.06 have been applied to the mean dose equivalents to the bronchial epithelium and the pulmonary tissue (IC81). The doses to other tissues from inhaled short-lived ^{222}Rn daughters are negligible. In the case of inhaled ^{220}Rn daughters, however, the transfer of ^{212}Pb leads also to significant doses to tissues outside the lung, particularly bone surfaces, bone marrow, kidneys and liver. Including these dose contributions results in a total effective dose equivalent from inhaled ^{220}Rn daughters of about 5×10^{-5} mSv per Bq h/m^3 exposure.

In the right part of Table 4, the corresponding conversion coefficients between the annual dose equivalent and the mean concentration of radon daughters in the air of the considered residence areas are listed. These are based on the relationship between concentration and annual exposure given in eqn 6. For inhaled ^{222}Rn daughters, the total annual dose equivalent H_B (in mSv) to the bronchial epithelium of adults is given by

$$H_B = \beta_1\ \bar{c}_{ih} + \beta_2\ \bar{c}_{ie} + \beta_3\ \bar{c}_o \tag{11a}$$

where the conversion coefficients β_1, β_2 and β_3 have values of 0.90, 0.23 and 0.20 mSv per Bq/m^3, respectively. The corresponding effective dose equivalent is given by

$$H_E = \varepsilon_1\ \bar{c}_{ih} + \varepsilon_2\ \bar{c}_{ie} + \varepsilon_3\ \bar{c}_o \tag{11b}$$

with conversion coefficients ε_1, ε_2 and ε_3 of 0.061, 0.016 and 0.014 mSv per Bq/m^3, respectively.

Using mean equilibrium-equivalent ^{222}Rn-concentrations of 15 Bq/m^3 in indoor air and 4 Bq/m^3 in outdoor air (see Section 2.4 and Table 2) the total bronchial dose equivalent is estimated to be 18 mSv per year and the total effective dose equivalent is estimated to be 1.2 mSv per year. Only about 5% of these dose values are attributed to the outdoor exposure.

As mentioned above, the conversion coefficients in Table 4 refer to adults. For children, the dosimetric models indicate a bronchial dose per unit exposure which is up to a factor of 2 higher than for adults (Ho82; NE83; Ja84c; NC84b). In the NEA-report (NE83), an average factor of 1.5 for the age group from 0–10 years is suggested.

4. RADIATION-INDUCED LUNG CANCER: GENERAL FINDINGS

The results of epidemiological studies on radiation-induced lung cancer, particularly among radon-exposed underground miners and the atomic bomb survivors in Hiroshima and Nagasaki, are described and critically reviewed in several reports (UN77,82; My81; NA80; NC84b; SC84; Th85). In this Section, a summary is given of the basic epidemiological and experimental findings on radiogenic lung cancer which are of importance in the context of this report. They concern the dose–risk relationship, the possible influence of smoking, the latency period and age distribution, and the estimation of the lifetime risk.

The cumulative radon daughter exposure of miners is usually expressed in terms of the potential alpha energy concentration of radon daughters in air at their workplace, integrated over their cumulative working time. In all epidemiological studies on these miners, this exposure is expressed in the unit "Working Level Months" (WLM). For this reason, this special unit is used also in this chapter of the report. For the definition of the special quantities and units used for quantifying radon daughter exposure and the corresponding conversion factors, the reader is referred to the Appendix to this report. With respect to ^{222}Rn daughters, 1 WLM corresponds to an equilibrium-equivalent ^{222}Rn-activity exposure of 6.3 10^5 Bq h/m^3.

4.1. Epidemiological Studies

4.1.1. *Radon-exposed underground miners*

Radiogenic lung cancer is the oldest type of radiation-induced malignancy known. It was recorded as early as in the 15th–16th century among miners in the Schneeberg–Jachymov region in the Erzgebirge. This so-called "Schneeberger Krankheit" was diagnosed as lung cancer in 1879. Its possible association with radon was suggested about 60 years ago, when the high radon levels in mines of this region were discovered. However, the real cause of this disease, the inhalation of short-lived ^{222}Rn daughters, was not recognized before the 1950s, when the first attempts at lung dosimetry were made.

Since that time, several epidemiological studies on Rn-exposed underground miners have been initiated. Of main importance for this report are the results from the three larger study groups of uranium miners in Colorado, USA (Lu71; NA80; Wa81; Wh83), Bohemia, CSSR (Se76; Ku79), and Ontario, Canada (Ch81b; Mu83,85). The basic data for these study groups are summarized in Table 5. The observed excess lung cancer frequency among these uranium miners indicates a strong correlation with their cumulated Rn daughter exposure. It cannot be explained by the inhalation of non-radioactive air pollutants occurring in the atmosphere of such mines.

This conclusion is confirmed by the findings among other small groups of Rn-exposed underground miners, e.g. the fluorspar miners in Newfoundland, Canada (Mo81,85), several smaller groups of metal ore miners, particularly iron ore miners, in Sweden (Sn73; Ax78; Da82; Ra84), China (Zh81) and the United Kingdom (Bo70; Fo81). Also, the recently published preliminary results of a survey among uranium miners in France yield a significant excess incidence of lung cancer (Ti85).

Overall, a total of about 25 000–30 000 Rn-exposed underground miners are included in

Table 5. Basic data for epidemiological studies on uranium miners

Quantity		Colorado[a] USA 1950–77	Bohemia[b] CSSR 1948–75	Ontario[c] Canada 1955–81	France[d] 1947–83
Initial number of miners		3 366	2 433	ca. 13 400	1 957
Average follow-up-period per miner (years)		19	26	15	25.9
Surviving fraction at end of follow-up (%)		72		ca. 80	81
Median age at start of uranium mining (years)		30	35–40	ca. 25	ca. 30
Average working period in uranium mines (years)		9	10	ca. 2	11.4
Number of person-years at risk (PYR)		62 556	ca. 60 000	202 795	50 784
Mean cumulated exposure [WLM]		820	310	60 ± 25	
Fraction of chronic cigarette smokers (%)		ca. 70	ca. 70	50–60	ca. 70
Number of lung cancer cases	observed	194	ca. 250	82	36
during follow-up	expected	40	ca. 50	57	18.8
	excess	154	ca. 200	25	17.2
Relative risk, observed/expected cases		4.8	ca. 5.0	1.45	1.9

[a] White miners only.
[b] Study group A only; the total group involved 4 364 miners (Sc82; Se71).
[c] Only uranium miners without prior gold mining.
[d] Exposure data not yet available.

these epidemiological study groups; of these, more than 80% are uranium miners in Canada, the CSSR, France and the USA.

4.1.2. *Atomic bomb survivors*

An excess lung cancer frequency has been observed among the atomic bomb survivors in Hiroshima and Nagasaki, who were exposed to external gamma radiation at a high dose rate. Among the 48 275 persons in the T65-kerma range (T65 = tentative 1965 dosimetry) of more than 0.01 Gy, 303 lung cancer deaths were observed in the period from October 1950 to December 1978, whereas 276 were expected (Ka82). Up to the end of 1982, the observed number of lung cancer deaths increased to 392, compared with an expected number of 348 (Pr84). Taking into account preliminary revised kerma values (Lo81; RE84), this group received a mean gamma dose to the lungs of 0.4–0.5 Gy.

4.1.3. *Ankylosing spondylitis patients*

An increased incidence of lung cancer has been also observed among a group of ankylosing spondylitis patients in the United Kingdom who were treated with x radiation during the period from 1935 to 1954. These data were recently re-examined and updated to 1970 (Sm82).

Among the final study group of 14 111 patients given a single course of treatment, 88 lung cancer deaths were observed during a mean follow-up period of 10 years, starting 9 years after exposure, compared with 59 expected deaths from lung cancer during this period. From the calculated dose distribution under these irradiation conditions, a mean fractionated bronchial dose of about 2.5 Gy can be estimated for these patients (Dr85; Ja86).

4.1.4. *Correlation studies on population groups exposed to enhanced natural radiation levels*

So far, only a few, small-scale epidemiological studies have been carried out, or initiated, relating to the possible influence of the natural radiation background on the etiology of lung cancer. In Sweden, after a first small-scale study (Ax79), two further case-control studies were started in local areas where strongly enhanced radon levels in houses have been measured (Ed83,84; Pe84). Due to the small number of lung cancer cases and the uncertainty of exposure estimates, the preliminary results of these pilot studies do not permit a significant correlation analysis. Also, in a recently published case-reference study based on 292 female lung cancer cases in the Stockholm area, a non-significantly higher proportion of cases than matched controls were residents of dwellings with enhanced ^{222}Rn levels (Sv85).

In Finland, a comparative regional survey on lung cancer frequency was carried out among a population of about 60 000 residents living in rural areas where high radon levels in houses have been measured (Ca85). From measurements in 754 houses, a current mean ^{222}Rn concentration of 370 Bq/m^3 in the houses of this study region was estimated, compared with an assumed mean value of about 90 Bq/m^3 for the entire population of Finland (i.e. including urban areas). A comparison of the reported lung cancer cases in the Finnish tumour registry during the period 1955–74 indicates no significant difference between the lung cancer frequency in this study area and that of the total Finnish population. The authors point out that "too far-reaching conclusions should not be drawn from this fact, because the radon exposures in the past were lower than now" (Ca85).

Recently, first results of a correlation analysis in Norway have been reported (St86b). In this study, two variables were taken into account, the Rn concentration in dwellings and the smoking habits, expressed as average number of cigarettes smoked per day. In a first step, the age-adjusted lung cancer incidence for men and women in the period 1966–85 was correlated against smoking habits, applying the data compiled in the Norwegian tumour registry. In the second step, the ratio between the actual incidence rate and the rate predicted by the correlation between lung cancer and smoking was correlated against Rn concentration, using the results of 1500 ^{222}Rn measurements in dwellings. The mean concentration varied from 80 to 180 Bq/m^3 for the population groups involved in this study.

The results of this analysis support a relative risk model (see Section 5.1) and indicate, for chronic exposure conditions, a relative excess risk coefficient for the lifetime risk from inhaled ^{222}Rn daughters in the range of 0.002–0.06 per Bq/m^3 at the 95% confidence level. On the basis of these preliminary results, Stranden (St86b) suggests that between 10 and 30% of lung cancers in the Norwegian population might be initiated by the radon daughter exposure in dwellings.

The correlation between lung cancer and natural radiation has also been investigated in a survey in the Guangdong province in China; in this study, cancer frequencies in an area with enhanced gamma background were compared with those in an area with normal background (Ho85; We85). The measured mean indoor concentration of ^{222}Rn daughters in the high background area is, however, relatively low, about 17 Bq/m^3, compared with a mean value of about 5 Bq/m^3 in the control area. The preliminary results indicate an astonishingly low overall lung cancer incidence (about one order of magnitude less than all other known values) and no statistically significant difference between the two areas studied. In consideration of the large number of persons included in this study, a validation of the death certificates with respect to lung cancer would be very valuable.

In the USA, a correlation study between cancer frequency and natural radioactivity in water has been carried out. A survey in Maine has shown a significant positive correlation between lung cancer mortality and the radon content in tap or well water (He83). It is doubtful, however, whether this finding can be related to the radon level in houses.

Summarizing, the preliminary results of the correlation studies available so far do not enable a reliable, quantitative estimate of the possible contribution of the natural radon daughter exposure to the observed lung cancer frequency in populations. The predominant role of smoking, the influence of factors other than natural radiation, such as occupational and environmental exposure to chemical air pollutants, and the variation of life expectancy, are probably the main reasons for the rather strong local variations in lung cancer incidence. Extended, carefully designed, epidemiological studies are necessary to verify any estimates for the contribution from indoor radon.

4.2 Histological Findings

The earliest publications on the histology of lung cancers in uranium miners led to the conclusion that irradiation specifically increased the incidence of anaplastic oat-cell cancers. Later, an increase of epidermoid cancers was also observed. Both oat-cell and epidermoid cancer originate from the bronchial epithelium. More recent investigations (Ho77,80; Sa81) have given further information on the induction by irradiation of various histological types of lung cancers in uranium miners.

The study on the CSSR-miners (Ho77,80) included an investigation of the response relationship for oat-cell, epidermoid and other lung cancers. It showed that the response for induction of the first two varieties was similar, even though there seemed to be a threshold for epidermoid cancers. Unfortunately, this study did not take tobacco consumption into account.

The study among the Colorado-miners (Sa81,84) was more exhaustive. It considered four histological types of lung cancer: oat-cell cancer, epidermoid carcinoma, adenocarcinoma and other lung cancers. Many factors were investigated: total dose, dose-rate, latency, age of the time of diagnosis, cigarette consumption and age at the beginning of work.

Summarizing, the histological findings indicate that the time factor and age at diagnosis are the main sources of the difference of responses between oat-cell cancers and epidermoid carcinomas. That is because the latency period, the age at the time of diagnosis and the total cigarette consumption all have an effect. The responses to these three, time-dependent factors are different for oat-cell cancers and epidermoid carcinomas. This explains why an excess of oat-cell cancers has been specially observed at the onset of the surveys. Epidermoid cancers are also more frequent in miners who were older when they started work. Adenocarcinomas have a different mode of response. They appear mainly in young heavy smokers, who started work early in life and who were exposed to high doses from radon daughters.

In conclusion, exposure to radon and its daughter products does increase the frequency of all types of lung cancers in uranium miners. The fundamental processes are certainly complicated and strongly related to cigarette consumption. The dose–effect–time relationship will, therefore, vary according to the histological type and many other factors beside irradiation.

4.3. Exposure–Risk Relationship

So far, only the epidemiological studies among the three larger groups of Rn exposed uranium miners enable an analysis of the shape of the relationship between exposure, or lung dose, and the associated lung cancer risk. Figure 7 shows, on a log–log scale, the relative increment of lung cancer frequency as a function of the cumulative exposure to ^{222}Rn daughters, as it follows from these studies (Ja85a; Mu85). For the uranium miners in Ontario, Canada, two data sets are given; they refer to different types of exposure estimates ("WLM-standard" (lower estimate) and "WLM-special" (upper estimate)) for the cumulative exposure of these miners (Mu85).

Taking into account the indicated 95% confidence limits and the uncertainty of the exposure data, the relationships between exposure and relative increment in lung cancer risk for these groups of uranium miners are in reasonable agreement, with the values for the Ontario miners lying between the regression lines for the Colorado miners and the Czechoslovakian uranium miners.

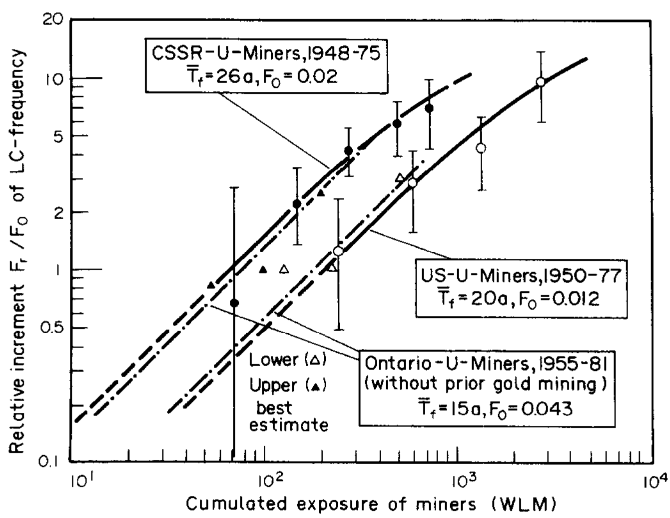

Fig. 7. Relative increment of normal lung cancer frequency among uranium miners in the CSSR, Canada and the USA as a function of their cumulative exposure to ^{222}Rn daughters (with 95% confidence limits). F_o is the normal lung cancer frequency and F_r is the radiation-exposure related frequency.

The data for the Colorado miners were tested for their consistency with various dose–response functions (St84); a linear response was found to yield the best fit in the exposure region below about 1000 WLM. Also, in the studies of the uranium miners in the CSSR and in Ontario, Canada, the presumption of linearity, without threshold, cannot be rejected at the 5% significance level (Th82,85; SC84). Thus, the criterion of maximum likelihood does not provide any reason to reject a proportional relationship, with respect to the absolute or relative excess of lung cancer incidence associated with the inhalation of radon daughters, up to exposure levels of several hundred WLM, corresponding to a mean bronchial dose equivalent of up to about 50 Sv. This conclusion is confirmed by the results of animal experiments; the observed increase of lung tumour incidence in Rn-exposed rats yields the same shape of exposure–response relationship as that shown in Fig. 7 for miners (Ch81a,82,85).

In this context, it should be emphasized that the data from the Czech and Ontario miners yield a statistically-significant excess risk above 50–100 WLM, corresponding to a mean bronchial dose equivalent above about 5–10 Sv. Recently, among a subgroup of Czech miners, a statistically-significant excess of lung cancer in the exposure cohort below 50 WLM was reported (Se85). The available measurements in houses indicate that lifetime exposures of small groups of the population, living in houses with high radon levels, exceed this statistical threshold. The mean indoor exposure of about 0.2 WLM per year, as is typical for most countries, yields a lifetime exposure of 10–20 WLM; this is about a factor 2–5 below the statistical level above which an excess lung cancer frequency among the uranium miners has been detected.

The epidemiological studies among Rn-exposed uranium miners, as well as the animal experiments, indicate a decreasing slope of the exposure–risk function at high exposure levels (see Fig. 7). It has been assumed that this reduction might be due to non-stochastic effects in the lung, resulting from cell killing at high doses. However, a reanalysis of the epidemiological data, on the basis of a relative risk model, shows that this downward curvature can be partly attributed to the reduced life expectancy of these miners at high exposure levels (Ja85a).

4.4. Lung Cancer Risk Coefficients for Rn-Exposed Miners

Assuming a linear exposure–risk relationship, risk coefficients for lung cancer from inhaled ^{222}Rn daughters can be derived from the epidemiological studies among ^{222}Rn-exposed miners. They are defined as the attributable excess risk per person year at risk (PYR) and per unit of exposure, averaged over the follow-up period, or as the relative increment of the expected lung cancer frequency per unit of exposure.

In Table 6 are listed the mean ranges of these risk coefficients as estimated for the three larger study groups of uranium miners, averaged over all age groups at start of mining and taking into account a minimum latency period of 5–10 years. These values are based on the summary of data given in recent review studies (NC84b; Ja85a; Th85), including the updated values from the Ontario miners (Mu85); with respect to relative excess risk coefficients see also Fig. 7. Applying a mean dose conversion coefficient of 0.12 Sv/WLM for these miners (NE83), the corresponding risk values per unit of mean dose equivalent to the bronchial epithelium can be calculated.

Absolute risk coefficients, derived for the respective follow-up periods, from mostly small-scale studies among Rn-exposed non-uranium miners, cover a range of $(2–20) \times 10^{-6}$ PYR^{-1} per WLM (UN77; NC84b; Th85), except for a small group of lead and zinc miners in Sweden, for whom an excess rate of about 30×10^{-6} PYR^{-1} per WLM has been estimated (Ax78). The relatively large range of absolute risk coefficients can be mainly attributed to differences with

Table 6. Mean absolute and relative risk coefficients for lung cancer resulting from studies among Rn-exposed uranium miners[a]

Study group of (follow-up)	Risk-exposure coefficient: Absolute risk coefficient (cases/10^6 person-years per WLM)	Relative excess risk coefficient (% per WLM)
Colorado/USA, whites (1950–77)	2–8	0.3–1.0
CSSR, group A 1948–75)	10–25	1.0–2.0
Ontario/Canada (1958–81)[b]	3–7	0.5–1.3
Average (probable range)	10 (5–15)	1.0 (0.5–1.5)

[a] Averaged over all age groups at start of mining and taking into account a minimum latency of 5–10 years; including risk contribution from external gamma irradiation and inhaled long-lived radionuclides in mines.
[b] From Ref. Mu85, miners with prior gold mining experience excluded.

respect to age distribution, follow-up period and smoking, and to the uncertainty of exposure estimates. Taking into account also the rather large statistical confidence limits of these studies, the results are in accord with the findings among the uranium miners.

At the bottom of Table 6 are listed the average values of the absolute and relative risk coefficients which follow from the various studies among the uranium miners. The Task Group believes that these values can be regarded as best estimates for the radiogenic lung cancer risk among miners averaged over all age groups at the start of mining. The probable ranges of uncertainty for these averages are given in parentheses. With respect to the relative excess risk coefficients, the range of error on the best estimate might be about $\pm 50\%$. Concerning the absolute risk coefficients, the influence of age, length of follow-up and of smoking have to be taken into account (see Section 4.5 and 4.6).

For the conditions in these mines, a mean bronchial alpha dose, averaged over the bronchial basal cell layer, of about 4–8 mGy per WLM exposure to ^{222}Rn daughters has been estimated (Ja85c). Applying a reference value of 6 mGy per WLM (NE83) and a quality factor of 20 for alpha radiation, the average risk coefficients given in Table 6 yield a mean excess lung cancer rate of about

$$0.08\ (0.04\text{–}0.12) \times 10^{-6}\ \mathrm{PYR}^{-1} \text{ per mSv mean bronchial dose equivalent} \qquad (12a)$$

and a relative excess risk coefficient of about

$$0.008\ (0.004\text{–}0.012)\% \text{ per mSv mean bronchial dose equivalent} \qquad (12b)$$

The estimated probable uncertainty range, given in parenthesis, does not include errors in the dosimetric evaluation.

It should be noted that the excess lung cancer frequency observed among these Rn-exposed miners includes contributions attributable to external gamma irradiation and to the inhalation of long-lived radionuclides (^{238}U, $^{210}Pb + ^{210}Po$) during their underground work. The available data indicate that the sum of these contributions might be small, about 10% of the total (see Section 5.2.2).

For the atomic bomb survivors, who were exposed mainly to low LET radiation at a high dose rate, a linear regression analysis yields a mean absolute risk coefficient for lung cancer of $(0.06\text{–}0.07) \times 10^{-6}$ PYR^{-1} per mGy (or mSv) mean lung dose (Ka82; NH85; Ja86). This value is only slightly lower than the mean value for the Rn-exposed miners given in eqn (12a).

4.5. Influence of Smoking

About 70% of the white uranium miners in Colorado were cigarette smokers, with an average smoking rate of somewhat less than 20 cigarettes per day (NC84b). Although 30% of these miners were non-smokers, only 9 of the 159 lung cancer cases, or 6%, were non-smokers (NA80). The updated follow-up of this group, involving 185 lung cancer cases, indicates the same strong influence of smoking on risk as is seen generally in non-irradiated persons (Wh83; Sa84). No individual smoking histories have been reported for the Czechoslovakian and Canadian miners, but about 70% of all the Czechoslovakian miners, and 50–60% of the Canadians, were smokers (Table 5). It is likely, therefore, that most of the lung cancer cases were among smokers.

The influence of smoking has also been investigated in Rn-exposed iron ore miners in Northern Sweden. Among a group of 1415 iron ore miners in Malmberget, Sweden, 51 lung cancer cases were observed. From a preliminary analysis of these data, Radford *et al.* (Ra84) concluded that the absolute lung cancer risk might be only about 50% higher for smokers than for non-smokers. However, in a larger case-control study among iron ore miners in northern Sweden, comprising about 600 lung cancer cases, a clear synergistic effect has been found, which can be approximated rather well by a multiplicative influence of radon daughter exposure and smoking (Da82).

Whittemore *et al.* have carried out an internal correlation analysis of the lung cancer incidence among the white uranium miners in Colorado (Wh83). From the risk functions examined, the best agreement with these data is obtained with a relative risk factor

$$\begin{aligned} R &\sim (1+0.3\times 10^{-2}E)(1+0.5\times 10^{-3}C) \\ &\sim 1+0.3\times 10^{-2}E+0.5\times 10^{-3}C+1.5\times 10^{-6}EC \end{aligned} \tag{13}$$

where E is the cumulative ^{222}Rn-daughter exposure in WLM, and C is the number of packs of cigarettes (1 pack = 20 cigarettes) smoked, cumulated up to 10 years before death. A recent reanalysis by Thomas *et al.* (Th85) also strongly rejects an additive, in favour of a multiplicative, model for these miners.

From this multiplicative function, one could conclude that at the same radon-daughter exposure, the relative radiation-induced lung cancer risk should be equal for smokers and non-smokers, but that the absolute excess risk is increased by smoking. The synergistic influence of smoking is represented by the multiplicative term in eqn (13). This synergistic factor depends both on the cigarette consumption and on radon-daughter exposure E. It should be emphasized, however, that the uncertainty range of this factor is rather large.

For uranium miners in the CSSR, smoking histories are only available for the lung cancer cases in a limited group; from the observed cases, 110 occurred among smokers and 5 among non-smokers (Ho77). With certain assumptions on the basis of these data, the number of expected cases in both groups can be roughly estimated. The comparison indicates a similar relative radiation risk for smokers and non-smokers, suggesting a multiplicative model (NA80; Th85).

In contrast to the multiplicative synergism between the alpha radiation from radon daughters and smoking suggested by the findings among Rn-exposed miners, the epidemiological data from the atomic bomb survivors yield no evidence for such a relationship (Ka82; Pr83). The preliminary results of a case-control study of lung cancer in Hiroshima and Nagasaki suggest more an additive than a multiplicative influence of smoking (Bl84).

To study the combined influence of radon daughter exposure and tobacco smoke, some animal studies have been carried out. Their results indicate that this interaction depends upon the amount of radon exposure and on the temporal sequence of administration of the two agents.

In dogs which inhaled both agents simultaneously, the observed lung cancer frequency was lower than in animals which were exposed only to radon daughters (Cr82,83). For the explanation of this antagonistic influence of tobacco smoke, it was suggested that the increased mucus production by smoking might lead to a lower dose to the bronchial epithelium. It should be noted that, in these experiments, very high exposure levels were used, and non-stochastic radiation effects probably also occurred.

Large-scale studies on the combined influence of radon daughters and tobacco smoke on lung cancer induction in rats were performed at different exposure levels from 100 to 4 000 WLM (Ch81a,82). In contrast to the study on dogs, the animals were chronically exposed to tobacco smoke after their exposure to radon daughters. The results indicate, particularly at low exposure levels, a synergistic effect related to exposure to both agents, and not an additive effect.

Summarizing, it can be concluded that the data on lung cancer in Rn-exposed miners, as well as results of animal experiments, suggest the existence of a synergistic interaction between inhaled radon daughters and tobacco smoke for chronic smokers. This agrees with the conclusions drawn in the report of UNSCEAR (Annex L in UN82). In the BEIR III-report (NA80), it was estimated that, because of this synergistic effect, the absolute excess lung cancer risk for non-smoking Rn-exposed miners might be a factor 2 to 6 lower than that for the smoking miners. However, the number of lung cancer cases among the non-smoking miners is too low to quantify the magnitude of the synergistic factor with sufficient accuracy.

The probability of such an interaction is supported by the fact that the inhalation of tobacco smoke influences the target region in the bronchial epithelium (basal and mucus cells) which receives the highest dose from inhaled radon daughters. The interaction of tobacco smoke and ionizing radiation can be interpreted either as influencing the initiation of bronchial carcinoma; or as a promoting action of tobacco smoke on the development and manifestation of such cancers, leading to a reduction of the latency period. Both modes of interaction can be considered as synergistic (UN82).

To describe and to quantify the influence of smoking, it seems reasonable to consider the effect of smoking on lung cancer induction by carcinogenic agents other than ionizing radiation. Of main importance are the quantitative conclusions which can be drawn from comparative studies on age-specific lung cancer rates in smokers, ex-smokers and non-smokers among the general population (Do64; US82; WH75; Wy77,83). These indicate that the influence of smoking decreases rather rapidly with time after cessation of smoking. This can be interpreted as suggesting that smoking probably acts mainly as a promoter, leading to a time-shift of the age-specific lung cancer appearance rate in comparison with non-smokers. It cannot be excluded that this promoting effect is associated with the non-specific structural and functional damage to the bronchial epithelium caused by chronic smoking.

On the basis of these studies, the promoting influence of tobacco smoke on the appearance rate of radiation-induced lung cancer can be estimated. This suggests the application of a proportional hazard model which combines the epidemiological findings on lung cancer among Rn-exposed miners with those concerning the promoting influence of smoking on lung cancer in populations; this model is described in Section 5.1.

4.6. Latency Period, Age and Sex Dependency

The available epidemiological data on radiogenic lung cancer refer to a restricted follow-up period. The assessment of the resulting lifetime risk depends strongly on the assumptions made and models used to represent the latency distribution as a function of age and time since exposure. In this section, the empirical findings concerning this time–response function are outlined, based on epidemiological data relating to the Rn-exposed miners and atomic bomb survivors. Previous studies (Ka82;Ja85a) have indicated a correlation with the normal incidence rate of lung cancer, which increases strongly with age. Therefore, main emphasis is given here to the analysis of the relative lung cancer risk (observed/expected cases) as a function of time since radiation exposure and its variation with age at time of exposure.

4.6.1. *Relative risk versus time*

Figure 8 shows the relative lung cancer incidence rate among the Colorado uranium miners as a function of time since starting uranium mining, derived from the data up to the end of 1977, as reported by Waxweiler *et al.* (Wa81). It must be pointed out that the average working or exposure period of these miners was rather short, about 9 years (see Table 5). Furthermore, most of these miners received most of their radon daughter exposure during the first few years after the start of mining, when the Rn concentration in these mines was still high. Therefore, the absolute and relative lung cancer risk of these miners refers, on the average, to an exposure period of only a few years. Taking into account the given 95% confidence range, the resulting relative risk is, after a time lag of 5–10 years, rather constant with time up to about 40 years after exposure. Similarly, the data from the CSSR uranium miners can be fitted with a time- or age-independent relative risk coefficient (Ja85a).

A similar time-invariance of the relative lung cancer risk follows from an analysis of the lung cancer data on the atomic bomb survivors for the period 1950–1982 (5–37 years since exposure), which were reported recently by Preston (Pr84,85). Figure 9 shows, as a function of time, the resulting relative risk for the T65-kerma groups above 1 Gy (upper graph) and above 0.1 Gy (lower graph), as compared with the low-exposed group (kerma $K < 0.1$ Gy) involved in the Life Span Study of the survivors in Hiroshima and Nagasaki.

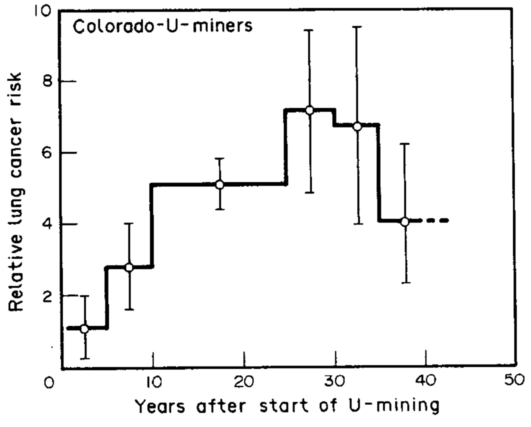

Fig. 8. Relative lung cancer risk of the Colorado uranium miners as a function of the period after the start of underground uranium mining (derived from data of Waxweiler *et al.*, 1981).

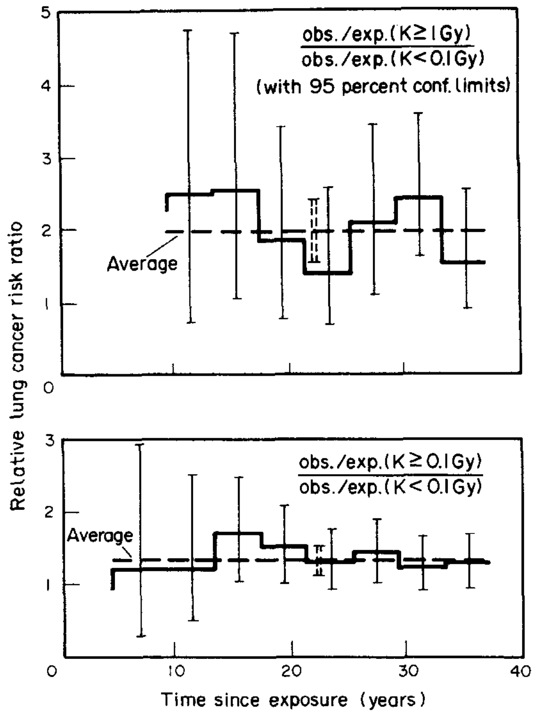

Fig. 9. Ratio of relative lung cancer risks in the atomic bomb survivors (1950–1982) as a function of time since exposure, compared with the 0–0.09 Gy kerma group (derived from data of the Life Span Study 1950–82, Preston *et al.*, 1984, 1985).

4.6.2. *Age dependence*

The observations among Rn-exposed uranium miners as well as among the atomic bomb survivors yield, for the given, restricted, follow-up period, a strong increase in the absolute excess lung cancer risk with age at time of the start of mining or with age at time of exposure, respectively (Ku79; Ka82; NC84b; La84; Ja85a). Furthermore, it has been shown that the epidemiological findings among the uranium miners in Canada, the CSSR and USA can be well fitted with a relative risk model, assuming, for adults, age-independent relative risk coefficients as given in Table 6 (Ja85a,86; Mu85).

This conclusion is confirmed by an analysis of the data from the Life Span Study among the atomic bomb survivors, which were reported for the period from 1950–1982 (Pr84,85). Figure 10 shows the normalized relative lung cancer risk as a function of age at exposure for two different T65-kerma groups. Taking into account the indicated 95% confidence range, these results are consistent with an age-independent relative risk for exposures at adult ages $\geqslant$20 years), in accord with the epidemiological finding for the Rn-exposed miners.

For the younger age groups (age at exposure less than 20 years), the study among the atomic bomb survivors indicates that the relative risk (R/R_0) is about a factor of two higher than in adults. This would correspond to a relative radiation-induced increment (R_r/R_0) of the normal lung cancer frequency (R_0) for children and juveniles, which might be up to a factor of about four higher than for adults. Owing to the restricted follow-up period, the total number of lung cancer cases observed so far in this young age group is rather low. Thus, the higher value for this age group may be modified by increased follow-up.

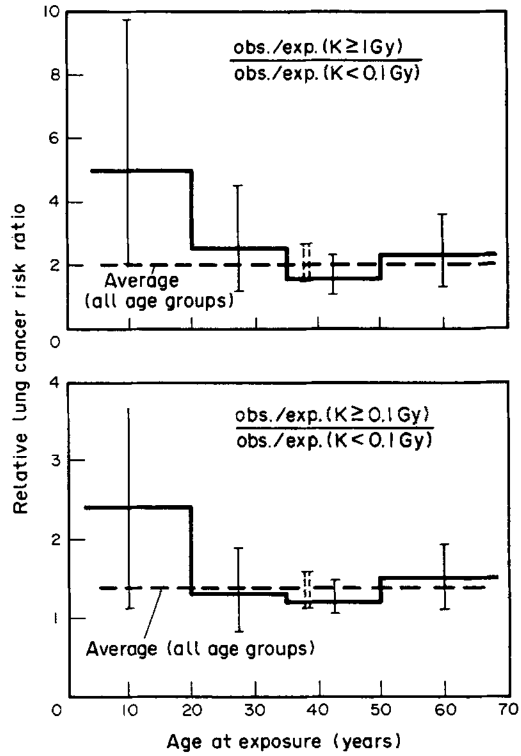

Fig. 10. Ratio of relative lung cancer risks in the atomic bomb survivors (1950–1978) as a function of age of exposure, compared with the <0.1 Gy kerma group (derived from data of the Life Span Study 1950–1978, Kato *et al.*, 1982).

4.6.3. *Sex dependence of lung cancer risk*

The lung cancer risk coefficients derived from data for Rn-exposed miners, which are given in Table 6, refer to males. Similar data for females exposed to significantly increased Rn-levels are not available. In general, it cannot be excluded that the sensitivity with respect to carcinogenic effects in the lung might be different for males and females. For example, the studies among non-smokers in the USA yield, on the average, an age-specific lung cancer rate which is about a factor of two higher for non-smoking males than for non-smoking females (Ga80,81; En80). This difference, however, can also be attributed to the influence of environmental factors.

With respect to radiation-induced lung cancer, the findings among the atomic bomb survivors can be quoted. A histological study among these survivors indicates that squamous cell carcinoma and adenocarcinoma of the lung seem to develop more rapidly in males than in females (Ha83). An analysis on the basis of the Nagasaki tumour registry yields, without correction for smoking, an absolute risk coefficient which is about a factor 2 to 3 higher for males than for females (Wa83).

More representative seem to be the results of the Life Span Study. In accordance with the previous findings (Ka82), the analysis of these data for the period 1950–82 indicated no significant difference of the average absolute excess risk of lung cancer between males and females (Pr85). As a consequence, the relative excess risk for women appears to be about 3–4 times higher than for males (Pr84,85), because the baseline rate of lung cancer among the females is about one third of that for males. In a study of lung cancer incidence over the period

1950–80, based on about 29 000 atomic bomb survivors in the same cohort for whom smoking data were available, it was revealed that, after adjusting for the effects of smoking, the baseline rate for females was about 80% of that for males. As this is not a significant difference, the relative excess risk as well as the absolute excess risk of lung cancer did not differ significantly between the sexes (Ko86).

On the basis of these results, the same relative risk coefficient for lung cancer induction is assumed for both males and females in assessment of the risks of indoor exposure to radon daughters (see Section 5.1).

4.7. Main Conclusions

Summarizing, the findings on radiogenic lung cancer lead to the following general conclusions.

(1) A linear exposure–risk relationship is a good fit to the available epidemiological and experimental data on lung cancer from inhaled radon daughters, if exposures above about 500 WLM are excluded.

(2) The appearance rate of radiation-induced lung cancer as a function of time is similar to the age-dependent distribution of the normal lung cancer rate in a comparable non-exposed population.

(3) Consequently, estimation of the attributable lifetime risk on the basis of a relative risk concept seems to be more appropriate than an absolute risk model that assumes no temporal correlation with the normal lung cancer rate.

(4) The relative lung cancer risk for adults is independent of the age at exposure and seems to be nearly equal for both sexes. For children and juveniles (age at exposure <20 years), the relative lung cancer risk is probably somewhat higher than for adults.

(5) With respect to bronchial cancer from inhaled radon daughters, the epidemiological and experimental findings suggest a more than additive influence of smoking. This influence can be approximated by a multiplicative model.

The observed time invariance of the relative risk can be interpreted on the basis of two-stage and multi-stage models for the development of lung cancer. If radiation is considered mainly as an initiator in the formation of intermediate or potentially malignant cells, then the expression rate is determined by other factors (e.g. age, smoking) which influence the tissue kinetics and the final tumour growth. Proceeding from the above mentioned, empirically founded, conclusions, the Task Group has developed a proportional hazard model for the estimation of the possible life-time risk of lung cancer among populations due to the inhalation of radon daughters in the environment.

5. CONCEPTS AND MODELS FOR THE EVALUATION OF THE LUNG CANCER RISK AMONG POPULATIONS

In this chapter, different concepts and approaches are outlined to estimate the probable lung cancer risk associated with environmental exposure to radon daughters. This risk can be expressed in terms of the absolute or relative lifetime risk to individuals, the radiation-induced lung cancer frequency among populations, or, finally, in terms of the corresponding loss of life expectancy.

Two different concepts for the evaluation of the radiogenic lung cancer risk are considered in this report:

(1) The relative risk projection, applying a proportional hazard model.

(2) The absolute or excess risk projection, which assumes no correlation between the radiation-induced excess rate and the normal, strongly age-dependent, appearance rate of lung cancer.

Main emphasis is given to the former concept which seems, with respect to lung cancer, to be more consistent with the epidemiological findings than are absolute risk models (Ja84b,85b; Mu85; NH85; Th85). Furthermore, it enables a simpler, and probably also a more realistic, transfer of the data from Rn-exposed miners to the exposure conditions of the general public.

5.1. The Relative Risk Projection Model

5.1.1. *Basic relationships*

The basic quantity from which this model proceeds is the age-specific lung cancer mortality rate $\lambda(t)$. This quantity defines the probability per unit time of dying from lung cancer at a given age t after having reached this age (conditional probability). Observed values of this quantity, as a function of age among the male and female populations of various countries, are compiled in a WHO report (WH83).

Irradiation of critical target cells in the lung can lead, after a certain time lag τ (minimum latency), to a radiation-induced increment $\lambda_r(t)$ of the baseline rate $\lambda_0(t)$ in the subsequent lifetime, giving a total rate:

$$\lambda(t)=\lambda_0(t)+\lambda_r(t) \tag{14}$$

The proportional hazard model used in this report is based on two assumptions, which are suggested by the epidemiological findings described in the previous chapter: a single exposure $E(t_e)$ to radon daughters at an age t_e leads, after a constant lag time τ, to an excess of the age-specific lung cancer rate which is proportional to this exposure and is also proportional to the normal rate λ_0 in the considered population group, without exposure to radon daughters.

For the case of a single, short-term exposure $E(t_e)$ at an age t_e (age at exposure), these two assumptions yield, for $t \geqslant [t_e+\tau]$, a radiogenic increment in the age-specific rate:

$$\lambda\lambda_r(t,t_e)=r(t_e)\; E(t_e)\; \lambda_0(t) \tag{15}$$

and a total rate:

$$\lambda(t,t_e)=\lambda_0(t)\;[1+r(t_e)\; E(t_e)] \tag{16}$$

In these equations, the relative excess risk coefficient $r(t_e)$ defines the relative increment of the age-specific lung cancer rate per unit of exposure at age t_e. In general, this coefficient depends on the age at exposure. It can be derived from the results of the epidemiological studies among radon-exposed miners (see Table 6), taking into account appropriate correction factors, which are discussed in Section 5.2.2.

For a chronic exposure to radon daughters at an age-dependent exposure rate $\dot{E}(t_e)$, this proportional hazard model yields a radiogenic increment:

$$\lambda_r(t)=\lambda_0(t)\int_0^{t-\tau} r(t_e)\; \dot{E}(t_e)\; \mathrm{d}t_e \tag{17}$$

and a total rate:

$$\lambda(t)=\lambda_0(t)\ [1+\int_0^{t-\tau} r(t_e)\ \dot{E}(t_e)\ dt_e] \tag{18}$$

In the special case of a constant exposure rate, or a constant annual exposure, respectively, the total age-specific lung cancer rate is given by

$$\lambda(t)=\lambda_0(t)\ [1+\bar{r}\ E(t-\tau)] \tag{19}$$

where $\bar{r}$ is the age-averaged, relative excess risk coefficient and $E(t-\tau)=\dot{E}\cdot[t-\tau]$ is the cumulative exposure to radon daughters up to age $t-\tau$.

This proportional hazard model implies a synergistic or promoting influence of smoking on the appearance rate of lung cancer induced by exposure to radon daughters. Without exposure to radon daughters, the age-specific lung cancer rate $\lambda_{o,s}$ for chronic smokers (index s) can be related to the corresponding rate $\lambda_{0,ns}$ for non-smokers (index ns) by the general formula:

$$\lambda_{0,s}(t)=\lambda_{0,ns}(t)\ [1+S_s(t)] \tag{20}$$

The function S_s characterizes the enhancement of this rate by smoking. To estimate the combined influence of smoking and radon daughter exposure, it is assumed, in the proportional hazard model, that the enhancement by smoking is the same for bronchial cancers initiated by the alpha radiation from inhaled radon daughters as it is for those initiated by other agents. With this assumption, a total age-specific lung cancer rate for chronic smokers of

$$\lambda_s(t)=\lambda_{0,ns}(t)\ [1+S_s(t)]\ [1+\bar{r}\ E(t-\tau)] \tag{21}$$

is expected for chronic radon-daughter exposure. In the numerical evaluation, the smoking factor S_s was introduced as a variable parameter (see Section 6).

The structure of this relationship corresponds to the risk function which has been derived by Whittemore *et al.* (Wh83) from a regression analysis of the data from the Colorado uranium miners (see eqn (13)). This equation also gives a good fit to the observed, excess lung cancer frequency among the Czechoslovak uranium miners (Ja85a). The relationship given by eqn (21) implies a multiplicative influence of smoking with respect to the age-specific lung cancer rate for radon-exposed individuals. The numerical evaluation shows, however, that this input function leads to a less than multiplicative influence of smoking on the integral lifetime risk of lung cancer from inhaled radon daughters (see Section 6).

5.1.2. *Evaluation of integral risk quantities*

The integral risk of lung cancer from inhaled radon daughters can be expressed in terms of three, interrelated, quantities:

—The individual lifetime risk of lung cancer (R_r)
—The radiation-induced lung cancer frequency among populations (F_r)
—The corresponding loss of life expectancy from radiation-induced lung cancer (ΔL_r)

These quantities can be derived from the radiogenic increment $\lambda_r(t)$ of the age-specific lung cancer rate as a function of radon daughter exposure rate ($\dot{E}$), using the proportional hazard model described in Section 5.1.1.

By definition the *individual lifetime risk* from inhaled radon daughters is

$$R_r=\int_0^{\infty} p(t)\ \lambda_r(t)dt \tag{22}$$

where $p(t)$ is the survival probability from birth until age t. Taking into account the reduction in life expectancy due to radiation-induced lung cancer, the survival probability $p(t)$ is given by the relationship

$$p(t)=p_0(t)\exp\left[-\int_0^t \lambda_r(t')\mathrm{d}t'\right] \tag{23}$$

where $p_0(t)$ is the value of $p(t)$ without radiation exposure.

The corresponding *loss of life expectancy* attributable to the lung cancer risk from inhaled radon daughters is given by the relationship

$$\begin{aligned}\Delta L_r=L_0-L= -\int_0^{\infty} t[\dot{p}_0(t)-\dot{p}(t)]\mathrm{d}t &= \int_0^{\infty}[p_0(t)-p(t)]\mathrm{d}t \\ &= \int_0^{\infty} p_0(t)\left\{1-\exp\left[-\int_0^t \lambda_r(t')\mathrm{d}t'\right]\right\}\mathrm{d}t \end{aligned} \tag{24}$$

where L_0 is the life expectancy at birth without any radiation exposure and $\dot{p}(t)$ is the differential quotient of the survival probability at age t.

In the relevant range of indoor exposures, the exponential function in eqn (24) can be represented by a first-order series expansion and the loss of life expectancy can be approximated by

$$\Delta L_r= -\int_0^{\infty}\left\{p_0(t)\int_0^t \lambda_r(t')\mathrm{d}t'\right\}\mathrm{d}t \tag{25}$$

For the assessment of the population-related detriment from radiation-induced lung cancer, the *attributable annual lung cancer frequency* among current populations is of interest. By integration over all age groups this annual frequency is:

$$F_r=\int_0^{\infty} v(t)\,\lambda_r(t)\mathrm{d}t \tag{26}$$

where $v(t)=n(t)/N$ is the relative age distribution of the considered population of N persons. Values of the currently observed total lung cancer frequency among the male and female populations of various countries are compiled in reports of the World Health Organization (WH83). They are usually expressed as number of observed lung cancer deaths per 10^5 persons per year.

On the basis of these data and for the purposes of this report, a reference male and female population was defined, for which a steady state was assumed (see Section 6.1). With this equilibrium condition, the survival probability $p(t)$ and the relative age distribution $v(t)$ are linked by the relationship

$$v_{eq}(t)=p(t)/L \tag{27}$$

where L is the life expectancy (at birth) of the considered population. Consequently, the corresponding annual frequency of radiation-induced lung cancer is given by:

$$F_r=R_r/L \tag{28}$$

and can be derived directly from the individual lifetime risk R_r given by eqn (22). The basic data for the reference population are listed in Section 6.1.

For the evaluation of the lung cancer risk from natural exposure to radon daughters, the relative increments of these integral risk quantities, related to the values without exposure, i.e.

R_r/R_0, F_r/F_0 and $\Delta L_r/L_0$, are of importance. For the same exposure conditions, these ratios are rather independent of the normal lung cancer risk and the life expectancy of populations, and are also nearly equal for non-smokers and smokers (see Section 6). This result is a direct consequence of the proportional hazard model used here.

5.2. Assessment of Risk Coefficients for Populations

5.2.1. *Transferability of Miners' Data*

The available epidemiological data on lung cancer from inhaled radon daughters refer to ^{222}Rn-exposed underground miners. These studies yield as best estimate (see Table 6) a relative risk increment

$$r_{\text{miners}} = \begin{cases} 0.010 \text{ per WLM} \\ 1.6 \times 10^{-8} \text{ per Bq h/m}^3 \end{cases} \tag{29a}$$

and an absolute risk, defined as the probability of death per unit time per unit exposure

$$a_{\text{miners}} = \begin{cases} 10 \times 10^{-6} \text{ year}^{-1} \text{ per WLM} \\ 1.6 \times 10^{-11} \text{ year}^{-1} \text{ per Bq h/m}^3 \end{cases} \tag{29b}$$

The mean values from different study groups of uranium miners differ by about a factor of two from these reference values.

It must be emphasized that these risk coefficients refer to male underground workers and include the radiation risks from external gamma radiation and from inhaled long-lived radioactive aerosols (uranium ore dust, $^{210}Pb + ^{210}Po$) in these mines. Furthermore, it can be argued that the observed enhanced lung cancer frequency among these miners might be attributed partly to the inhalation of other non-radioactive dusts and vapours which are present in mine atmospheres.

Also, the physical state of inhaled radon daughters (unattached fraction, particle size distribution of their carrier aerosol) in mine air is different from that found in indoor and outdoor conditions for populations. Taking into account the additional influence of breathing rate (see Fig.6), this leads to a bronchial dose/exposure ratio H_B/E for members of the public which differs from that for miners (dosimetric correction factor). In addition, the general question arises as to how these data from male miners can be utilized for populations including females and children, especially as the influence of smoking has to be taken into account.

In general, it must be emphasized that the risk coefficients derived from data on miners cannot be directly transferred to the general public for its different exposure conditions. The Task Group has attempted to estimate appropriate correction factors. However, these factors are tentative estimates based on present knowledge. Further studies will be necessary to validate these estimates and to reduce the uncertainties involved.

5.2.2. *Estimation of correction factors*

Correction for co-carcinogenic influences in mines. Recently, the results of a comprehensive study on the frequency of lung cancer among various groups of miners in Ontario, Canada, have been published (Mu83,85). Except for Rn-exposed uranium miners and a smaller group of gold miners, no significant excess of lung cancer among these miners was observed, as compared with the age-adjusted frequency among the general male population in the region. The reasons for the enhanced level in the small group of gold miners are still unclear; a possible influence of asbestos in these gold mines has been suggested (Mu83).

Chambers *et al.* (Ch85) examined the potential carcinogenic and co-carcinogenic effects of agents, other than ionizing radiation, which may currently be present in uranium mine atmospheres. They concluded that, at the current low levels of such toxic agents in Canadian uranium mines, a significant contribution to the workplace hazard seems to be unlikely. The same conclusion is probably valid for most other groups of uranium miners for which epidemiological data are available.

Recent studies among uranium miners in the USA have shown significant lung burdens of inhaled uranium ore dust and $^{210}Pb + ^{210}Po$ (Si85; Pa85). Dosimetric estimates indicate that, in mine areas with high uranium ore dust content, committed bronchial doses from these long-lived radionuclides can reach values which are comparable with those from short-lived ^{222}Rn daughters (Ha85). However, under normal conditions, the dose ratio is smaller, particularly in mines with high Rn content. Harley (Ha85) suggests that, on the average, the committed bronchial dose from inhaled long-lived dusts might be about one order of magnitude lower than that from short-lived ^{222}Rn daughters. Also, referring to those mines from which the epidemiological data have been obtained, the additional lung dose to the uranium miners from external gamma radiation is relatively small.

Summarizing, these findings support the conclusion that the observed excess lung cancer frequency among the study groups of uranium miners is mainly attributable to the inhalation of short-lived ^{222}Rn daughters. The risk contribution from inhaled long-lived radionuclides and gamma radiation might be, on the average, about 10–20% of the total. Also, the additional co-carcinogenic or synergistic influence of non-radioactive dusts and vapours in these mines seems to be relatively small. An overall risk contribution of 20% from all sources other than ^{222}Rn daughters is assumed in this report, corresponding to a multiplicative correction factor of 0.8 on the risk coefficients given in eqn (29).

Dosimetric corrections of the risk/exposure ratio. The mean breathing rate of members of the public is lower than that of miners during their underground work. This is particularly valid for the resting phase, during the night at home. Appropriate correction factors can be derived from Fig. 6. However, in indoor air the fraction of unattached daughter atoms is somewhat higher, and the activity median diameter (AMD) of the carrier aerosol of attached daughter atoms is probably somewhat smaller, than in mine atmospheres. This would lead to an increase in the bronchial dose to exposure ratio, compensating to a large extent for the reduction of this ratio resulting from lower breathing rates indoors.

The possible influence of these factors has been investigated in several dosimetric studies and has been reviewed in an NEA-report (NE83, see also Ja84c,85c). In that report, a mean bronchial alpha dose of 1.8 Gy per J h/m^3 ($\simeq 6$ mGy per WLM) ^{222}Rn-daughter exposure was suggested for underground miners. Taking into account a quality factor of 20 for alpha radiation (IC77a,81), this corresponds to a mean bronchial dose equivalent of 1.9×10^{-4} mSv per Bq h/m^3 equilibrium-equivalent ^{222}Rn exposure. A comparison of this number with the estimated mean values of the bronchial dose to exposure ratio H_B/E for adults in indoor and outdoor air, listed in Table 4, leads to a mean dosimetric correction factor for adult males and females of:

$$\frac{(H_B/E)_{\text{indoors}}}{(H_B/E)_{\text{mines}}} \approx 0.8$$

$$\frac{(H_B/E)_{\text{outdoors}}}{(H_B/E)_{\text{mines}}} \approx 1.0 \qquad (30)$$

For children (0–10 years), the dosimetric correction might be about a factor of 1.5 larger than for adults (NE83; NC84b).

Sex and age dependency. The risk coefficients for miners refer primarily to males and not to females. In general, however, the available epidemiological data on radiation-induced cancers indicate, for most types of tumours (except, e.g., for breast and thyroid cancer), no marked sex difference with respect to the relative sensitivity for radiation carcinogenesis. The observed sex-specific incidence ratios for radiation-induced tumours are similar to those observed for the same tumour types in non-irradiated populations. The sex ratio for lung cancer following irradiation can be estimated from the observations on the atomic bomb survivors, which were summarized in Section 4.6.3. In this report, on the basis of the most recent analysis (Ko86), the same relative risk coefficient r for the age-specific lung cancer rate is assumed for both sexes.

Observations on the Rn-exposed miners yield an increase of absolute lung cancer risk with increasing age at exposure, whereas the relative risk seems to be rather age-independent (see Section 4.6.2). This agrees with the findings among the adult (age > 20 years) atomic bomb survivors (see Fig. 10). At ages at exposure of less than 20 years, the atomic bomb survivor data indicate an increase of relative risk with decreasing age, particularly at higher doses (Ka82; Bl84; Pr84,85). The data given in the lower graph of Fig. 10 show a mean value of the relative risk R/R_0 in the range of 2–3 for the age group between 0–20 years, compared with a mean value of about 1.4 for adults. This yields a mean value for the relative radiation-induced increment R_r/R_0 of about 1–2 for the 0–20 years age group and of about 0.4 for adults. Of this difference, by a factor of 2.5–5, only a small fraction can be attributed to the higher lung dose to kerma ratio in children.

On the basis of this comparison, the Task Group has assumed, with respect to inhaled radon daughters, an average relative risk coefficient for the 0–20 years age group which is three times larger than that for adults. However, as shown in Fig. 10, the uncertainty of this risk factor for children is large, due to the limited number of lung cancer cases. The influence of this uncertainty on the estimated lifetime risk is discussed in Sections 6.2 and 6.4.

5.2.3. *Risk coefficients for the age-specific lung cancer rate*

In the upper part of Table 7 are summarized the values of the relative risk coefficients $r = \lambda_r/\lambda_0 E$ for indoor exposure to ^{222}Rn daughters of the different age groups (age at exposure) which were used in the numerical evaluation of the described proportional hazard model. They were derived from the average relative excess risk coefficients for ^{222}Rn-exposed miners, given in eqn (29a), taking into account the previously-described correction factors. The corresponding absolute risk coefficients for the age-specific lung cancer rate follow from the relationship

Table 7. Estimated mean values of the relative excess risk coefficient, referring to the age-specific lung cancer rate, as a function of age at time of exposure

	Relative excess risk coefficient $\lambda_r/\lambda_0 E$	
Age at exposure	$(\mathrm{Bq\ h/m^3})^{-1}$	WLM^{-1}
^{222}Rn(Rn) daughters		
0–20 a	3.0×10^{-8}	0.019
< 20 a	1.0×10^{-8}	0.0064
^{220}Rn(Tn) daughters		
0–20 a	10×10^{-8}	0.0045
< 20 a	3.3×10^{-8}	0.0015

$a = r\lambda_0 = \lambda_r/E$, where λ_0 is the normal rate without radon daughter exposure. Reference values of λ_0 as a function of age, which were used in the numerical evaluation, are given in Section 6.1.

Relative excess risk coefficients for ^{220}Rn daughters are given in the lower part of Table 7. They are estimated on the basis of a dosimetric comparison with ^{222}Rn daughters (see Table 4).

The risk coefficients given in Table 7 are best estimates. Taking into account the range of variation of the mean relative risk coefficients resulting from the epidemiological studies on uranium miners (see Table 6) and the probable range of correction factors for the exposure conditions of populations, the resulting overall uncertainty of the risk coefficients in Table 7 might cover a range from about 0.3 up to 2 times the values stated.

5.3. Absolute Risk Projection Models

In the past, mainly absolute or excess risk projection models (ARP models) have been considered for the evaluation of the lifetime lung cancer risk from ionizing radiation (UN77; NA80; NC84b). In the following section, the results of different approaches using such an ARP model are briefly summarized.

5.3.1. *Approach from data on miners*

Contrary to the proportional hazard model previously described, the ARP models presume no correlation between the appearance rate of radiogenic lung cancer and the normal, strongly age-dependent, lung cancer rate.

In the case of a single short-term exposure $E(t_e)$ to radon daughters at an age t_e (age at exposure), in these ARP models it is usually assumed that this exposure, after a certain time lag τ, leads to a potential excess rate of lung cancer which remains constant in the subsequent lifetime, but which can depend on the age at exposure. Using a linear exposure–response relationship, this model yields an excess age-specific lung cancer rate

$$\lambda_r(t_e, t) = a(t_e)\, E(t_e) \quad \text{for} \quad t > t_0 = t_e + \tau \tag{31}$$

The absolute risk coefficient a is expressed in terms of the number of excess cases per 10^6 person-years at risk per unit of exposure (see Table 6). To fit the epidemiological data, it is also often assumed that, for exposures at young ages, the excess rate starts at an age $t_0 = 40$ years. For exposures at higher ages, it is usual to take a time lag, τ, of 10 years (NC84b; NA80).

This ARP model yields, in the case of a chronic exposure to radon daughters ($\dot{E}$ = const.) an age-specific excess lung cancer rate:

$$\lambda_r(t) = \dot{E} \int_0^{t-\tau} a(t_e)\mathrm{d}t_e = \bar{a}\, \dot{E}\, [t - \tau] \quad \text{for} \quad t > 40 \text{ years} \tag{32}$$

When an ARP model is applied to the assessment of the lung cancer incidence in a population, the mean absolute risk coefficient, as incidence per person-year at risk, for Rn-exposed miners given in eqn (29b) can be used, together with the correction factors estimated above (see Section 5.2.2). On this basis, an age-averaged risk factor, $\bar{a}$, for the indoor exposure of populations to ^{222}Rn daughters is calculated as:

$$\begin{aligned} &\bar{a} = 1.1 \times 10^{-11} \text{ year}^{-1} \text{ per Bq h/m}^3 \\ \text{or} \quad &\bar{a} = 7 \times 10^{-6} \text{ year}^{-1} \text{ per WLM} \end{aligned} \tag{33}$$

The corresponding lifetime risk R_r for radiation-induced lung cancer can be evaluated from eqn (22) in the same way as for the proportional hazard model. For a population with a life expectancy without radon daughter exposure of $L_0 = 73$ years at birth, which is chronically exposed to ^{222}Rn daughters ($\dot{E}$=const.), this approach yields a lifetime risk per unit of annual indoor exposure rate to ^{222}Rn daughters of

$$\begin{aligned} &R_r/\dot{E} = 1.8 \times 10^{-8} \text{ per Bq h/m}^3\text{/year} \\ \text{or} \quad &R_r/\dot{E} = 0.011 \text{ per WLM/year} \end{aligned} \tag{34}$$

This corresponds to an age-averaged, excess lung cancer risk of

$$\begin{aligned} &R_r/E = 2.5 \times 10^{-10} \text{ per Bq h/m}^3 \\ \text{or} \quad &R_r/E = 1.5 \times 10^{-4} \text{ per WLM} \end{aligned} \tag{35}$$

It must be emphasized that this value refers primarily to males with smoking habits similar to those of the miners. Thus, no correction factors for females and smoking are taken into account. Results from different versions of this ARP model are presented in Section 5.3.3.

5.3.2. *Dosimetric approach*

In addition to the approach from data on miners, the ICRP has considered in its recommendations on exposure limits to radon daughters for workers (IC81) the so-called "dosimetric approach". This approach proceeds from the recommended age- and sex-averaged reference values $R_r/H_B = R_r/H_P = 1 \times 10^{-3}$ Sv^{-1} for the committed lifetime risk of lung cancer per unit of dose equivalent to the two target tissues in the lung (B = bronchial region, P = pulmonary region). Taking into account the estimated mean values of the dose to exposure ratio H_B/E and H_P/E given in Table 4, this dosimetric approach yields an age-averaged lifetime risk of lung cancer per unit of indoor exposure to ^{222}Rn daughters of

$$\begin{aligned} &R_r/E = 1.6 \times 10^{-10} \text{ per Bq h/m}^3 \\ \text{or} \quad &R_r/E = 1.0 \times 10^{-4} \text{ per WLM} \end{aligned} \tag{36}$$

In the case of a chronic, constant exposure rate ($\dot{E}$=const.) the cumulated lifetime risk per unit of annual exposure rate is calculated as

$$\begin{aligned} &R_r/\dot{E} = 1.1 \times 10^{-8} \text{ per Bq h/m}^3 \text{ in a year} \\ \text{or} \quad &R_r/\dot{E} = 7.0 \times 10^{-3} \text{ per WLM in a year} \end{aligned} \tag{37}$$

This estimate is about 0.6 of the mean value derived from the more direct epidemiological approach from the data on miners given in eqn (34).

5.3.3. *Comparison of different absolute risk approaches*

During recent years, several attempts have been made to estimate the possible lifetime risk due to lung cancer from inhaled radon daughters in the environment on the basis of absolute risk projection models. In Table 8, the estimated mean values resulting from these studies for chronic exposure conditions ($\dot{E}$=const.) are summarized. They refer to an annual exposure to ^{222}Rn daughters of 1.2×10^5 Bq h/m^3 ≡ 0.19 WLM, as might be typical for most populations at the current time (see Table 3).

At the top of this table is given the mean value derived from the ARP model described in this study (see eqn (34)). It agrees rather well with previous estimates cited in the NCRP report (NC84b), and by Jacobi (Ja84a), although different versions of the ARP model were used in these studies.

The NCRP model proceeds from an initial, age-averaged risk coefficient $a_0 = 1 \times 10^{-5}$ year^{-1} per WLM as derived from data on miners, without any corrections. In addition, this model

Table 8. Estimated lifetime risk of lung cancer from chronic exposure to ^{222}Rn daughters at a constant annual exposure rate of 1.2×10^5 Bq h/m^3 per year ($\equiv$0.19 WLM per year); comparison of mean values derived from different absolute risk approaches

Study, reference		Lifetime risk (%)
Approaches from miners' data referring primarily to males	This study (eqn 34)	0.22
	BEIR III (NA80)	1
	NCRP (NC84b)	0.18
	Jacobi (Ja84a)	
	Non-smokers	0.1
	Smokers (average)	0.3
Dosimetric approach (eqn 37)		0.13
Evans *et al.* (Ev81)		
upper bounded estimate for non-smokers		<0.15

assumes a decrease of this initial potential excess rate with time after exposure, according to an exponential function with a biological half-life of 20 years. This correction was made to give a better fit to the observed age dependency of the excess lung cancer frequency in Rn-exposed miners. It presupposes a removal half-life of 20 years for the radiation-induced transformations of stem cells in the bronchial epithelium. However, the epidemiological data already implicitly include the effects of such removal. As mentioned in Section 4, the observed age dependency can be explained simply on the basis of the relative risk concept, taking into account the restricted follow-up of these epidemiological studies (Ja85a).

As shown in Table 8, the results of the different absolute risk approaches are in reasonably good agreement. The only exception is the value resulting from the BEIR III-approach (NA80). This approach proceeds from risk factors calculated as a function of age at death, inserting overestimates of input parameters which are not consistent with the epidemiological findings (Co82; NC84b; NH85).

Excluding this approach, the results of the different ARP models listed in Table 8 yield, for the average population, a lifetime risk of 0.1–0.3% at the given reference exposure conditions. This corresponds to an age-averaged risk to exposure ratio

$$\begin{aligned} &R_r/E = (1-3)\times 10^{-10} \text{ per Bq h/m}^3 \\ \text{or}\quad &R_r/E = (0.6-1.8)\times 10^{-4} \text{ per WLM} \end{aligned} \tag{38}$$

for indoor exposure to ^{222}Rn daughters. It has to be pointed out that the approaches from data on miners refer primarily to smoking adult males and involve no correction for females. The value for non-smokers is probably at the lower end of the range given.

6. EXPECTED LUNG CANCER RISK FROM CHRONIC EXPOSURE TO RADON DAUGHTERS

The previously described concepts and models enable an estimation of the probable lung cancer risk which might be associated with the inhalation of radon daughters present in our environment. Taking into account the observed levels of ^{222}Rn in the air of houses and their variation, the risk contribution from indoor exposure to ^{222}Rn daughters is of prime importance. Main emphasis is given to the inferences from the proportional hazard model, which are described in Sections 6.2 to 6.5.

At the end of this section, (i.e. Section 6.6), the best estimates resulting from different risk projection models are compared. All of these approaches proceed from the assumption of a proportional relationship between the radon daughter exposure and the attributable increment of lung cancer rate.

The lung cancer risk values for inhaled radon daughters are expressed in terms of the attributable lifetime risk R_r to individuals (individual-related risk) or the attributable lung cancer frequency F_r among populations (population-related risk). In addition, the corresponding loss of life expectancy is estimated (see Section 6.5).

6.1. Assumed Exposure Conditions and Reference Populations

The following numerical results refer to a chronic radon daughter exposure, assuming a constant exposure rate throughout the whole lifetime ($\dot{E}(t)$=const.). In practice, actual exposure rates will vary because of the mobility of members of the public and the long-term variation of the radon levels in our houses. The possible influence of these factors is discussed below.

Risk estimates are given as functions of the annual radon daughter exposure, expressed in terms of the equilibrium-equivalent ^{222}Rn(Rn)- or ^{220}Rn(Tn)-exposure per year, in units of Bq h/m^3 per year. As outlined in Section 3, the total exposure to radon daughters is given by the sum of the exposure contributions during residence indoors at home (index ih), indoors elsewhere (index ie), and outdoors (index o). The exposure model proposed by the Task Group proceeds from the following mean residence probabilities: $p_{ih}=0.65$, $p_{ie}=0.20$, $p_o=0.15$.

The reference conversion coefficients between the radon daughter concentrations, $\bar{c}$, in indoor or outdoor air and the resulting annual exposures from residence in these areas, listed in Table 3 (see also eqn (6)), take into account a diurnal variation of the indoor and outdoor levels. If this variation is already taken into account in measured or calculated air concentrations, the annual exposure can be evaluated from eqn (4). The reference conversion coefficients can be applied to derive relationships between the radon daughter concentrations in indoor and outdoor air and lung cancer risk values.

The proportional hazard model used herein implies that the age dependence of the rate of appearance of radiogenic lung cancer should be similar to that observed for lung cancer from other causes. Data for the currently observed age-specific lung cancer rates among males and females in various countries are compiled in a WHO report (WH83).

In countries with a mean life expectancy (at birth) of 67–73 years for males and 72–80 years for females, the observed country-averaged mean values of the integral lung cancer frequency cover a range of 300–1 100 and 70–300 cases/10^6 residents per year for males and females, respectively. However, values at the upper end of these ranges refer to only a few countries, such as Belgium, the Netherlands and the United Kingdom for males; and Denmark, the United Kingdom and the USA for females. In most countries with populations of high life expectancy, the currently-observed lung cancer frequency lies in the range of 400–800 or 80–200 cases/10^6 residents per year for males and females, respectively (WH83).

On the basis of these data, a reference population of males and females was defined for the numerical evaluations using the proportional hazard model. The baseline values of the survival probability $p_o(t)$ from birth until age t, and of the age-specific lung cancer rate without radon daughter exposure, $\lambda_o(t)$, are listed for this reference population in Table 9. They correspond to mean life expectancies L_o (from birth) of about 70 years for males and 75 years for females, and yield integral lung cancer frequencies without radon daughter exposure, F_o, of 600 and 120 cases/10^6 residents per year among males and females, respectively.

Table 9. Survival probability p_o and age-specific lung cancer (LC) mortality rate λ_o of the reference population without exposure to radon daughters (L_o = life expectancy at birth)

Age, t (Years)	$p_o(t)$ Males $L_o = 70a$	$p_o(t)$ Females $L_o = 75a$	$\lambda_o(t)$ LC-cases/10^5 persons-a Males	$\lambda_o(t)$ LC-cases/10^5 persons-a Females
30	0.96	0.97	<0.1	<0.1
35	0.95	0.96	1.2	0.5
40	0.94	0.95	4	1.5
45	0.93	0.94	12	3.0
50	0.90	0.93	30	7.0
55	0.86	0.90	60	13
60	0.80	0.87	120	20
65	0.72	0.81	200	30
70	0.60	0.73	280	40
75	0.46	0.62	360	50
80	0.29	0.47	400	60
85	0.14	0.30	350	64
90	0.040	0.14	320	67
95	0.005	0.030	300	70
All ages			60	12

These reference values are in accord with the average values derived from the country-averaged lung cancer data, subtracting a fraction of about 10% for the attributable lung cancer rate from radon daughters (see Section 6.4). It should be recognized that this reference population represents a mixture of non-smokers, smokers and exsmokers. Due to the reduced life expectancy of chronic smokers, the fraction of non-smokers among the population increases strongly at higher ages.

In addition to this mixed reference population, the attributable lung cancer risk from inhaled radon daughters has been estimated for non-smokers and smokers. The epidemiological studies on lung cancer among non–smokers in the USA (Ga80,81; En80) and the United Kingdom (Do64,76,78; To78) yield a strong increase of the age-specific lung cancer rate with age. In the relevant age range, above 50 years, it can be approximated by a function $\lambda(t) \approx kt^6$ with $k = (1–5) \times 10^{-15}\ a^{-7}$. Taking into account a mean life expectancy of 75–77 years for non-smokers, this yields a lifetime lung cancer risk in the range of about 0.3–1.5%. The US study indicates, for non-smoking males, a lung cancer rate which is about a factor of two higher than that for females. It is not known whether this difference, which might be caused by occupational factors, is valid for other populations.

On the basis of these epidemiological studies, a baseline reference function

$$\lambda_{o,ns}(t) = kt^6 \quad \text{with } k = 2.5 \times 10^{-15}\ a^{-7} \tag{39}$$

for the age-specific lung cancer rate among non-smokers, averaged over both sexes, is assumed in this report. With a mean life expectancy (at birth) $L_{o,ns} = 76$ years for non-smokers, this corresponds to a lung cancer frequency $F_{o,ns} \approx 80$ cases/10^6 non-smokers per year and a lifetime lung cancer risk $R_{o,ns} \approx 0.6\%$.

Now, the radiogenic lung cancer risk for chronic smokers is estimated on the basis of the proportional hazard model. Taking the baseline rate for non-smokers from eqn (39), the baseline lung cancer rate for smokers is evaluated from eqn (20), with the smoking factor S_s introduced as a variable parameter (see Section 6.3).

In general, the considered populations are assumed to be in a steady state. Under this condition the following relationships are valid:

$$\begin{aligned} R_o &= F_o\, L_o \\ R &= R_o + R_r = FL = (F_o + F_r)(L_o - \Delta L_r) \\ R_r &= R - R_o = F_r L - F_o\, \Delta L_r \end{aligned} \tag{40}$$

where F_r and R_r are the attributable excess frequency and lifetime risk, and $\Delta L_r = L_o - L$ is the corresponding loss of life expectancy due to lung cancer from inhaled radon daughters, as they follow from the equations given in Section 5.1.2.

At low radon daughter exposures, $\Delta L_r \ll L_o$. With this assumption the attributable excess lifetime risk is given by

$$R_r \approx F_r L_o \tag{41a}$$

and the associated relative risk increment is given by

$$\frac{R_r}{R_o} \approx \frac{F_r}{F_o} \tag{41b}$$

6.2. Lifetime Risk versus Exposure: Results of the Proportional Hazard Model

Proceeding from the relative risk coefficients for the age-specific lung cancer rate given in Table 7, the integral values of the attributable lifetime risk and frequency of lung cancer from inhaled radon daughters can be calculated on the basis of the proportional hazard model for different exposure conditions and populations.

6.2.1. *Relative risk coefficients*

In Fig. 11, the relative attributable lifetime risk R_r/R_o per unit of the equilibrium-equivalent exposure rate $\dot{E}$ (in units of 10^5 Bq h/m^3 per year, equivalent to 0.16 WLM per year) as a function of this exposure rate is plotted. These results relate to the specified reference populations, for the case of chronic indoor exposure to ^{222}Rn daughters ($\dot{E}(t)$ = const.). As a consequence of the proportional hazard model used, the relative risk coefficients $R_r/R_o\dot{E}$ are similar for males and females, as well as for non-smokers only. The small deviations are caused by differences in life expectancy. The decrease at high exposure rates is due to the reduced survival probability caused by the increasing radiogenic lung cancer rate. This decrease is more pronounced for the mixed reference population (non-smokers + smokers), than for non-smokers alone.

At indoor exposure rates below about 2×10^6 Bq h/m^3 per year, or about 3 WLM per year, which is the case for most houses (see Section 3.1), the relative risk coefficient is constant, corresponding to a linear exposure–risk relationship. Under these exposure conditions, the relative risk coefficients listed in Table 10 can be regarded as central best estimates for populations with a life expectancy of 70–80 years. The value listed for ^{220}Rn daughters is derived from Table 7 and is based on a comparison of the bronchial dose/exposure ratio with that for ^{222}Rn daughters (see Table 4). The dosimetric comparison indicates that the attributable risk per unit outdoor exposure to ^{222}Rn daughters might be about a factor 1.3 higher than the value for indoor exposure given in Table 9.

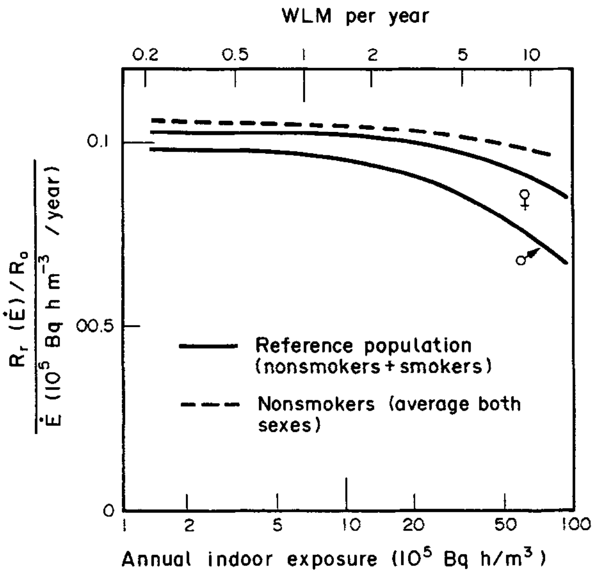

Fig. 11. Relative attributable lifetime risk R_r/R_o per unit of the annual indoor exposure to ^{222}Rn daughters as a function of this exposure rate.

Table 10. Relative risk coefficient $R_r/R_o\dot{E}$ for the attributable lifetime risk of lung cancer from chronic indoor exposure to radon daughters ($\dot{E}$=const.); best estimates resulting from the proportional hazard model[a]

Inhaled radionuclides	Relative excess risk per unit annual indoor exposure[b] (Bq h/m^3)$^{-1}$	WLM^{-1}
^{222}Rn(Rn) daughters	1.0×10^{-6}	0.64
^{220}Rn(Tn) daughters	3.3×10^{-6}	0.15

[a] In steady-state populations the same value is valid for the attributable relative excess of lung cancer frequency per unit annual exposure ($F_r/F_o\dot{E}$).

[b] The selected primary value is underlined and rounded to one significant figure; other values are derived from it.

With the proposed residence model, described in Section 3.1, the exposure rate can be related to the corresponding radon daughter concentration in air. Taking into account the conversion coefficients listed in Table 3 and the relative risk coefficients (see Table 10), results in the following relationship between the total relative lifetime risk of lung cancer from inhaled ^{222}Rn daughters and their concentrations in air:

$$R_r/R_o = F_r/F_o$$

$$\approx 0.001\ (\mathrm{Bq/m^3})^{-1}\ [6\ \bar{c}_{ih} + 1.5\ \bar{c}_{ie} + 1.3\ \bar{c}_o] \qquad (42)$$

In this equation $\bar{c}_{ih}$, $\bar{c}_{ie}$ and $\bar{c}_o$ are the annual mean values of the ^{222}Rn daughter concentration in indoor air at home, in indoor air elsewhere (e.g. at the working place), and in outdoor air, respectively, expressed in terms of the equilibrium-equivalent ^{222}Rn concentration, in Bq/m^3.

Of most importance is the risk contribution from residence indoors at home, for which a mean residence probability $p_{ih} = 0.65$ is assumed in this report. Normally, individuals living in dwellings with high ^{222}Rn levels would be exposed to considerably lower levels during their indoor residence elsewhere ($p_{ie} = 0.20$). Assuming normal levels of $\bar{c}_{ie} \approx 15$ Bq/m^3 and of $\bar{c}_o \approx 4$ Bq/m^3 for the equilibrium-equivalent ^{222}Rn concentration, (see Table 3), eqn (42) can be used to derive a relative attributable lifetime risk from inhaled ^{222}Rn daughters:

$$R_r/R_o = F_r/F_o$$
$$\approx 0.028 + 0.006\ (\mathrm{Bq/m^3})^{-1}\ \bar{c}_{ih} \tag{43}$$

Under these conditions, a doubling of the normal lung cancer risk R_o, without ^{222}Rn daughter exposure, should be expected for groups of the population who are chronically exposed throughout their whole lifetime to a mean equilibrium-equivalent ^{222}Rn concentration of about 150–200 Bq/m^3 in the residence areas of their homes.

These approximate relationships are valid up to indoor concentrations of about 300 Bq/m^3. At higher indoor levels, the attributable lifetime risk per unit of exposure becomes lower due to the decrease in survival as a result of the induction of radiogenic lung cancer. Appropriate correction factors can be derived from Fig. 11.

6.2.2. *Absolute risk coefficients*

As outlined, the proportional hazard model leads to the conclusion that, for the same exposure conditions, the attributable relative risk of lung cancer from inhaled radon daughters is nearly equal for populations with the same survival function or life expectancy. Because of the assumed multiplicative influence of smoking, the resulting absolute excess risk will be higher for populations with an enhanced baseline lung cancer frequency from smoking than for the non-smoking fraction of populations.

For chronic exposure conditions ($\dot{E}$ = const.), the values of the attributable excess lifetime risk R_r, or frequency F_r, of lung cancer per unit of annual indoor exposure can be derived for specified populations, taking into account their baseline lung cancer risk (R_o, F_o). Figure 12 shows the attributable excess lifetime risk R_r as a function of the annual indoor exposure to ^{222}Rn daughters for the mixed reference population (non-smokers + smokers) and for the assumed reference group of non-smokers (averaged over both sexes). The deviation from linearity at high exposure rates, particularly for the male reference population, is caused by the increasing loss of life expectancy due to the induction of radiogenic lung cancer (see also Fig. 11).

Taking into account the mean relative risk coefficient given in Table 10 and the baseline values for the lung cancer incidence (see Section 6.1), the absolute risk coefficients $R_r/\dot{E}$ and $F_r/\dot{E}$ listed in Table 11 are calculated for these reference populations. They refer to chronic indoor exposure to ^{222}Rn daughters ($\dot{E}$ = const.). For the total reference population, averaged over non-smokers and smokers and over both sexes, the proportional hazard model yields, as a best estimate, an absolute risk coefficient of

$$R_r/\dot{E} = 2.6 \times 10^{-8} \text{ per Bq h/m}^3 \text{ in each year}$$
or
$$R_r/\dot{E} = 0.016 \text{ per WLM in each year} \tag{44a}$$

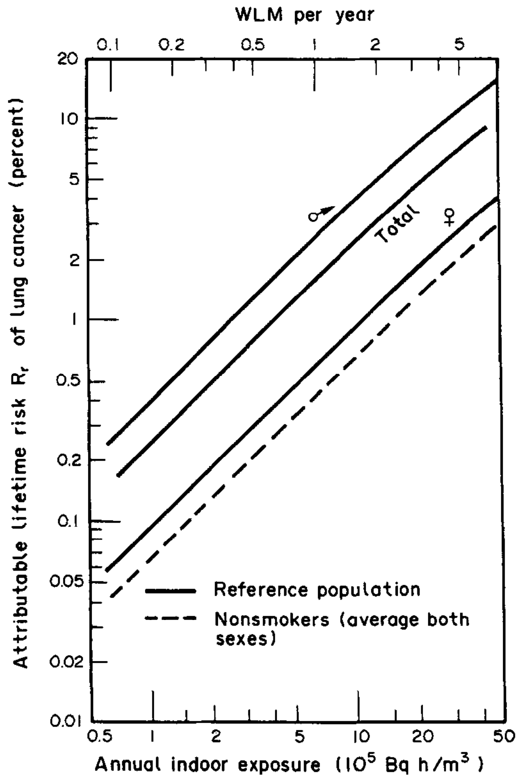

Fig. 12. Attributable absolute lifetime risk R_r of lung cancer from chronic exposure to ^{222}Rn daughters as a function of the annual exposure; the line "total" refers to the total reference population (males plus females).

Table 11. Attributable excess lifetime risk R_r and excess frequency F_r of lung cancer, and corresponding loss of life expectancy ΔL_r, from chronic indoor exposure to ^{222}Rn daughters at a constant level of 10^5 Bq h/m^3 or 0.16 WLM per year; best estimate derived from the proportional hazard model

Attributable absolute risk	Reference population[a] (non-smokers + smokers) Males $L_0 = 70a$	Females $L_0 = 75a$	Total $L_0 = 72.5a$	Non-smokers[b] $L_0 = 76a$
Lifetime risk R_r (%)	0.42	0.09	0.26	0.06
Frequency F_r, cases/10^6 persons per year	60	12	36	8
Loss of life expectancy ΔL_r, days	20	8	14	5.5

[a] Baseline annual lung cancer frequency: F_0(males) = 600 cases/10^6 persons per year; F_0(females) = 120 cases/10^6 persons per year

[b] Reference non-smokers, baseline annual lung cancer frequency: $F_0 = 80$ cases/10^6 persons per year, averaged over both sexes.

with respect to the individual lifetime risk, and an excess lung cancer frequency:

$$\begin{aligned} &F_r/\dot{E} = 3.6\times10^{-4} \text{ cases}/10^6 \text{ persons per year per Bq h/m}^3\text{/year} \\ \text{or}\quad &F_r/\dot{E} = 230 \text{ cases}/10^6 \text{ persons per year per WLM/year} \end{aligned} \tag{44b}$$

For the non-smoking fraction of this population, the absolute risk coefficients are a factor 4 to 5 lower.

According to Fig. 12, at high exposure rates a reduction factor has to be taken into account. Corresponding absolute risk coefficients can be derived for ^{220}Rn daughters, applying the relative risk coefficients listed in Table 10.

6.3. Attributable Radiation Risk for Smokers versus Non-smokers

In the numerical evaluation of the lung cancer risk from inhaled radon daughters for smokers, the smoking factor S_s, characterizing the promoting or synergistic influence of smoking, was inserted as a variable parameter (see eqn (21)). Figure 13 shows, as a function of this parameter, the ratio of the attributable lifetime risks R_r for chronic smokers and non-smokers at different values of the annual ^{222}Rn-daughter exposure.

This risk ratio increases non-linearly with the smoking factor S_s not only at high, but also at low exposure rates. This is because chronic cigarette smoking alone leads to a reduction of the survival probability with age and a corresponding reduction in life expectancy due to bronchial cancer and other smoking-related fatal diseases, particularly ischaemic heart disease. At ^{222}Rn daughter exposures in the range of 10^5–10^6 Bq h/m^3 per year, chronic strong smokers ($S_s > 10$)

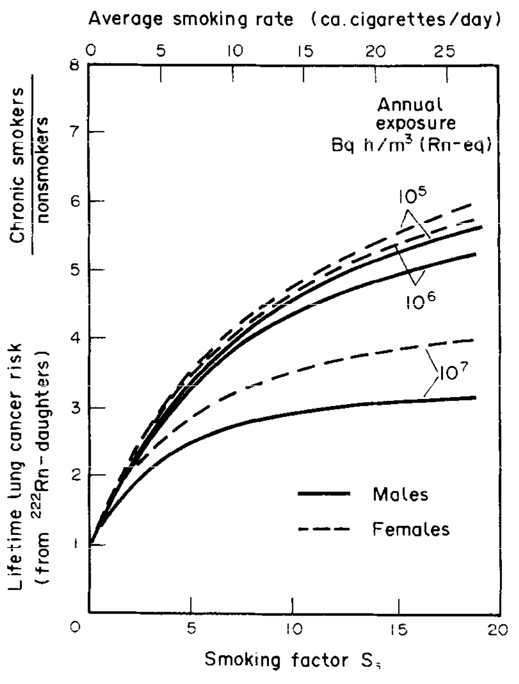

Fig. 13. Ratio of the attributable lifetime risk of lung cancer of chronic smokers versus non-smokers from chronic exposure to ^{222}Rn daughters, plotted as a function of the promoting or synergistic factor S_s.

have a 4–6 times higher radiation risk than non-smokers ($S_s = 0$). At higher exposure rates, this ratio decreases due to the additional loss of life expectancy from radiation-induced lung cancer.

It should be noted that the risk ratios shown in Fig. 13 are estimates for chronic cigarette smokers who start smoking at an early age (about 20 years) and continue smoking at the same rate until the end of their lives. Taking into account the observed recovery after cessation of smoking, for smokers with a shorter smoking period the risk ratio is lower.

The comparative studies between chronic cigarette smokers and non-smokers yield a strong correlation between the age-adjusted lung cancer mortality ratio and the smoking rate (Do64,71; To78; US82; WH75; Wy77,83). On the basis of the results of four of the larger studies which were compiled in a WHO report (WH75), as a first approximation, a linear relationship between the mean smoking rate $\dot{c}_s$ (expressed, e.g., in cigarettes per day) and the relative lung cancer risk of chronic smokers, or the attributed smoking factor S_s, can be assumed. These studies yield a mean ratio, $S_s/\dot{c}_s$, of 0.5–1.0 per cigarette/day. The corresponding mean smoking rate is plotted on the upper ordinate of Fig. 13, assuming a ratio, $S_s/\dot{c}_s$, of 0.7 per cigarette/day. It should be noted that these studies refer to males. The available data for females smokers indicate a somewhat lower value of this ratio (Wy77,83).

6.4. Attributable Mean Population Risk

Based on measurements in various countries, reference values of the current mean concentration of radon daughters in indoor and outdoor air are as given in Section 2.4. Taking into account the mean residence probabilities, corresponding mean values of current annual radon daughter exposures were derived and these are summarized in Table 3. These reference values are considered to be representative for the total population in the temperate regions. With respect to ^{222}Rn daughters, a mean equilibrium-equivalent ^{222}Rn concentration of 15 Bq/m^3 in indoor air is estimated, in accord with the conclusions drawn in the report of UNSCEAR (UN82).

Table 12 gives a summary of mean values of the attributable lung cancer risk estimated for chronic exposure at the given reference levels. These are based on the proportional hazard model, utilizing the risk coefficients given in Tables 10 and 11. In addition to the relative excess risk R_r/R_o, related to the normal lung cancer risk R_o without radon daughter exposure, for each source term k the attributable relative fraction

$$\frac{R_{r,k}}{R_{\text{total}}} = \frac{R_{r,k}}{R_o + \sum_k R_{r,k}} = \frac{R_{r,k}/R_o}{1 + \sum_k (R_{r,k}/R_o)} \tag{45}$$

of the total lung cancer risk is given. In the right columns of Table 12, the resulting absolute values of the attributable excess lung cancer frequency, $F_r = (R_r/R_o)F_o$, for the reference population (non-smokers + smokers) and for non-smokers (average over both sexes) are given.

On the basis of this proportional hazard model, it is estimated that about 10% of the total lifetime risk or frequency of lung cancer in a population might be associated with the indoor exposure to ^{222}Rn daughters. As mentioned earlier, this model yields nearly the same relative risk value for males and females. The additional contribution from ^{220}Rn(Tn) daughters is relatively small, about one tenth of the values for ^{222}Rn (Rn) daughters for the assumed exposure conditions.

With respect to the absolute risk, an attributable lung cancer frequency of about 40 cases/10^6 persons per year from exposure to ^{222}Rn daughters is estimated for the total reference

Table 12. Attributable relative lifetime risk and absolute excess frequency of lung cancer (LC) from chronic exposure to radon daughters at the given mean levels of the equilibrium-equivalent concentration and annual exposure indoors and outdoors

Source residence area	EEC (Bq/m^3)	Annual exposure[a] ($Bq\ h/m^3$)	Attributable relative risk (%)		Excess frequency F_r^b LC cases/10^6 persons per year			
					Reference population			Non-smokers (average both sexes)
			R_r/R_o	R_r/R_{total}	Males	Females	Total	
$^{222}Rn(Rn)$ daughters								
indoors at home	15	0.90	9.0	8.0	54	11	32	7.2
indoors elsewhere	15	0.23	2.3	2.0	14	2.7	8.1	1.8
outdoors	4	0.040	0.52	0.46	3.1	0.62	1.9	0.42
Total, ^{222}Rn daughters		1.2	11.8	10.4	71	14	42	9.4
$^{220}Rn(Tn)$ daughters								
indoors at home	0.5	0.030	1.0	0.88	6.0	1.2	3.6	0.80
indoors elsewhere	0.5	0.0075	0.25	0.22	1.5	0.30	0.90	0.20
outdoors	0.2	0.0020	0.66	0.058	0.40	0.079	0.24	0.053
Total, ^{220}Rn daughters		0.040	1.3	1.2	7.9	1.6	4.7	1.05
Total, $^{222}Rn + ^{220}Rn$ daughters			13	11.6	79	16	47	10.5

[a] Calculated with eqn (6).
[b] For steady-state populations.

population (males + females), compared with a total frequency $F = F_0 + F_r \approx 360 + 40 = 400$ cases/10^6 persons per year. For the reference group of non-smokers, the attributable frequency is about 9 cases/10^6 non-smokers per year, averaged over both sexes, compared with a total value $F \approx 80 + 9 \approx 90$ cases/10^6 non-smokers per year. As noted, these reference values of the absolute risk refer to populations with a life expectancy at birth of 70–80 years.

Furthermore, it should be recognized that the risk estimates given above assume a chronic, constant exposure to radon daughters at the given levels throughout the whole lifetime. Attributable mean risk values for other specified populations can be estimated from the relative risk coefficients given in Table 10, taking into account the specific exposure conditions and age-specific lung cancer rates of these populations.

6.5. Estimated Loss of Life Expectancy

The health detriment by lung cancer from inhaled radon daughters can also be expressed in terms of the attributable loss of life expectancy as defined by eqn (24). In Fig. 14, the calculated loss of life expectancy (at birth) from inhaled ^{222}Rn daughters as a function of the annual indoor exposure is plotted, as derived from the proportional hazard model for the defined male and female reference populations. This figure applies for the case of chronic exposure at a constant annual rate. It should be noted that the absolute risk model, normalized to the same number of cancers as the proportional hazard model, would predict a larger loss of life expectancy.

On the basis of the relative excess risk coefficients given in Table 7, the attributable loss of life expectancy, averaged over both sexes, is

$$\Delta L_r \approx 1.4 \times 10^{-4} \dot{E} \quad \text{(days)} \tag{46}$$

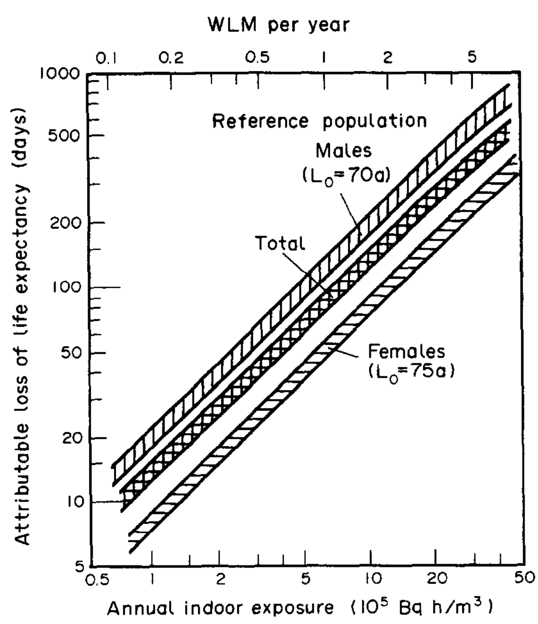

Fig. 14. Estimated loss of life expectancy at birth attributable to lung cancer from inhaled ^{222}Rn daughters as a function of the annual indoor exposure at the given rate.

where $\dot{E}$ is the annual indoor exposure in Bq h/m^3(Rn-eq). The corresponding specified risk coefficients are listed in Table 11.

At the present time, a mean indoor concentration in the range of 7–30 Bq/m^3, corresponding to an annual exposure of (0.5–2.3) 10^5 Bq h/m^3(Rn-eq) seems to be typical for most countries. Chronic exposure at such levels would lead to an attributable loss of life expectancy averaged over both sexes, of about 7–31 days. At the assumed reference indoor level of 15 Bq/m^3 a value of about 16 days is estimated. Compared with a mean life expectancy, L_0, of 72.5 years for this reference population, this corresponds to a mean relative loss of life expectancy $\Delta L_r/L_0 \approx 0.0006$ (or 0.06%) for chronic exposure at this reference level.

The mean loss of lifetime per lung tumour induced by inhaled radon daughters is defined by the ratio $\Delta L_r/R_r$. Inserting the absolute excess lifetime risk coefficient per unit of exposure listed in Table 11 and using eqn (46), a mean loss of lifetime per radiation-induced lung tumour averaged over both sexes, for the reference population can be calculated:

$$\Delta T_r(\text{per tumour}) = \Delta L_r/R_r$$

$$= \frac{1.4 \times 10^{-4}}{2.6 \times 10^{-8}}\ \text{days} \approx 5\,400\ \text{days} \approx 15\ \text{years} \tag{47}$$

This value is in rather good agreement with the estimate given in *ICRP Publication 27* (IC77b), where a quite different approach was used.

6.6. Summary and Conclusions

6.6.1. *Comparison and reliability of different risk approaches*

The assumptions and simplifications of absolute risk projection models are outlined in Section 5.3 of this report. In Table 13, best estimates from these models of the attributable lifetime risk of lung cancer for chronic indoor exposure to ^{222}Rn daughters are compared with the sex-averaged risk value resulting from the proportional hazard model. The latter value, referring to the defined mixed reference population (non-smokers + smokers), is about a factor of 1.5 higher than the results of absolute risk projection approaches based on data from Rn-exposed miners. With respect to the dosimetric approach, the difference is about a factor 2.5.

These differences between the various risk approaches are comparable with, or perhaps smaller than, the confidence ranges of the respective best estimates. Two error sources have to

Table 13. Comparison of estimates from different risk projection models for the attributable lifetime risk R_r of lung cancer from chronic indoor exposure to ^{222}Rn daughters ($\dot{E}$ = const.)

Type of approach	Lifetime risk R_r for a chronic exposure of 10^5 Bq h/m^3 in each year	Lifetime risk R_r for a chronic exposure of 1 WLM in each year
Relative risk projection		
reference population[a]	0.0026	0.016
non-smokers only[a]	0.0006	0.004
Absolute risk projection		
approach from miner's data, this study	0.0018	0.011
approach from miner's data, NCRP model	0.0015	0.0095
dosimetric approach[a]	0.0010	0.0070

[a]Averaged over both sexes.

be considered, the simplifying assumptions involved in the risk projection models and uncertainties in the values of their input parameters.

The Task Group believes that the empirically-founded proportional hazard model, which was developed for the purpose of this report, enables more reliable risk projection than absolute risk projection models, which assume no correlation between the time or age distributions of the radiogenic and the normal lung cancer rates. Furthermore, it enables an easier and more appropriate transfer of the lung cancer data from Rn-exposed miners to the exposure conditions of populations.

With respect to the uncertainty of input parameters, the error range is rather similar for all types of approaches which proceed from data on Rn-exposed miners. The probable uncertainty range of the primary absolute or relative risk coefficients has been estimated to about $\pm 50\%$ (see Table 6). Compared with this, the range of variation of the dosimetric correction factor and of the correction factor to allow for the risk to miners from gamma radiation and inhaled long-lived radioactive dusts in mine air, seem to be relatively small (see Section 5.2.2).

Another problem concerns the synergistic, or co-carcinogenic, influence of non-radioactive dusts and vapours in mine air on the observed enhanced lung cancer frequency among Rn-exposed miners. In this report, as in previous studies, this has not been taken into account. There is no epidemiological evidence for a strong influence of this factor, but its existence cannot be entirely excluded.

In the absolute risk projection models, no synergistic factor for smoking was introduced. In the proportional hazard model a multiplicative model was assumed with respect to the age-specific lung cancer rate. As pointed out, this assumption leads to a lifetime risk ratio between smokers and non-smokers which is somewhat less than multiplicative, owing to the considerable reduction in life expectancy of chronic smokers (see Fig. 13). This result is in rather good agreement with the available findings on Rn-exposed miners and enables an allowance to be made for the synergistic influence of smoking. The resulting absolute risk coefficient for non-smokers, averaged over both sexes, is about a factor three lower than the value for the mixed reference population (see Table 13).

The lung cancer data for Rn-exposed miners refer to adult males. Therefore, to evaluate risks to females and children, the observations on lung cancer incidence among the atomic bomb survivors have been taken into account in the proportional hazard model. On the basis of these data, a three times larger relative risk coefficient for the radiation-induced increment of the age-specific lung cancer rate was assumed for individuals exposed at ages of less than 20 years as compared with exposures at higher ages. Because of the limited number of lung cancer cases in this young age group, the uncertainty of this age-correction factor is rather large. If, for example, no age dependency of the relative risk coefficient were included, the lifetime risk resulting from the proportional hazard model would be about 0.7 of the value given in Table 13 and would then be comparable with the listed estimates based on absolute risk projection models.

Summarizing, the Task Group believes that the total uncertainty range of the best estimate values from the proportional hazard model, listed in Table 13, covers a range given by a factor of from 0.3 up to about 2. This includes some uncertainty as to the shape of the exposure–risk relationship in the region relevant to indoor exposure to ^{222}Rn daughters.

6.6.2. *Assessment of reference risk coefficients*

On the basis of the comparison given in Table 13, the Task Group recommends a rounded reference value of the attributable absolute lifetime risk of lung cancer from chronic indoor exposure to ^{222}Rn daughters of

$$R_r/\dot{E}=2\times10^{-8} \text{ per Bq h/m}^3 \text{ in each year}$$
or
$$R_r/\dot{E}=0.013 \text{ per WLM in each year} \tag{48}$$

averaged over both sexes. This absolute risk value seems to be appropriate for populations with a life expectancy, L, of 70–80 years, whose baseline lung cancer risk is comparable with that of the defined reference population (see Table 9). In steady-state populations, the corresponding risk coefficient for the attributable lung cancer frequency (per year), F_r, can be derived from the relationship $F_r/\dot{E}=R_r/\dot{E}L$ (L = life expectancy).

As noted, a relative risk projection model seems to have more validity than do absolute risk projection models. The Task Group believes, therefore, that it is most appropriate to select a primary reference value expressed in terms of the relative risk coefficient, $R_r/R_o\dot{E}$. Taking into account the baseline lung cancer risk, R_o, of the reference population, the rounded absolute risk value given in eqn (48) corresponds to a coefficient for the attributable relative risk of

$$R_r/R_o\dot{E}=F_r/F_o\dot{E}$$
$$=0.8\times10^{-6} \text{ per Bq h/m}^3 \text{ in each year}$$
or
$$R_r/R_o\dot{E}=0.5 \text{ per WLM in each year} \tag{49}$$

This value is 0.8 of the original value (see Table 10), which was derived from the proportional hazard model, taking into account a 3 times higher potential risk for exposures at young ages (0–20 years). Having in mind the uncertainty of the age dependence, this difference is small. The proportional hazard model indicates that this relative risk coefficient is generally applicable to all populations or groups of the population. It is recommended for males and females, as well as for non-smokers and smokers. Taking into account their baseline lung cancer frequency, the absolute attributable lung cancer risk of these specified populations can be estimated.

The reference risk coefficients recommended are based on the assumption of a linear dose–risk relationship for lung cancer induced by alpha radiation. On the basis of the available epidemiological and radiobiological data, it is reasonable to adopt such a linear relationship.

It has to be emphasized that the recommended risk coefficients refer to a chronic exposure at a constant annual rate throughout the whole lifetime. This simplification seems to be appropriate in developing protection guidelines, for decision making with respect to action levels and technical countermeasures in existing houses, and in the planning of future houses.

For evaluation of the attributable lung cancer risk in current populations the situation can be different. There is strong evidence that, on the average, the ^{222}Rn levels in houses have been increasing during recent decades. Therefore, the mean cumulated radon daughter exposure of the present population may be lower than that calculated from currently measured radon levels in houses. This long-term variation and secular changes in smoking habits have to be taken into account in the planning and evaluation of epidemiological lung cancer studies among populations.

7. CONCLUDING REMARKS

The risk analysis described in this report should be regarded as an attempt to quantify the possible lung cancer risk associated with the natural exposure to radon daughters. The results indicate that, although it is considered that cigarette smoking remains as the major cause of lung cancer in many countries, a significant fraction of the observed lung cancer frequency in populations may be attributed to the indoor exposure to ^{222}Rn daughters.

Further investigations are necessary to confirm, or to improve, this risk assessment. In several countries, epidemiological pilot studies on lung cancer among population groups exposed to enhanced ^{222}Rn levels in houses have been started or are planned. The main problems of such studies concern the retrospective estimation of the exposure and the competing influence of other occupational and environmental pollutants, and particularly of smoking.

REFERENCES

Ax78 Axelson, O. and Sundell, L. (1978). Mining, lung cancer and smoking. *Scand. J. Environ. Health* **4**, 46–52.

Ax79 Axelson, O., Edling, C. and Kling, H. (1979). Lung cancer and residency—A case-referent study on the possible impact of exposure to radon and its daughters in dwellings. *Scand. J. Work. Environ. Health* **5**, 10–15.

BI86 Bundesministerium des Innern (1986). Radon in Wohnungen und im Freien: Erhebungsmessungen in der Bundesrepublik Deutschland. Report of the Federal Ministry of the Interior, F.R. Germany, Bonn.

Bl84 Blot, W. J., Akiba, S. and Kato, H. (1984). Ionizing radiation and lung cancer: A review including preliminary results from a case-control study among A-bomb survivors. In: *Atomic Bomb Survivor Data: Utilization and Analysis*, pp. 235–248, (eds. R. L. Prentice, D. J. Thompson), SIAM-SIMS Conference Series 10. SIAM, Philadelphia.

Bo70 Boyd, J. T., Doll, R. and Faulds, J. S. (1970). Cancer of the lung in iron ore (haematite) miners. *Br. J. Ind. Med.* **27**, 97–105.

Bo85 Bouville, A., Snihs, J. O. and O'Riordan, M. C. (1985). Principles of protection against natural radiation. *Science of the Total Environment* **45**, 565–577.

Br83 Brown, L. (1983). National radiation survey in the U.K.: Indoor occupancy factors. *Radiat. Prot. Dosimetry* **5**, 203–208.

Ca84 Castren, O., Winquist, K., Mäkeleinen, I. and Voutilainen, A. (1984). Radon measurements in Finnish houses. *Radiat. Prot. Dosimetry* **7**, 333–336.

Ca85 Castren, O., Voutilainen, A., Winqvist, K. and Mäkelainen, I. (1985). Studies of high indoor radon areas in Finland. *Science of the Total Environment* **45**, 311–318.

Ch81a Chameaud, J., Perraud, R., Masse, R. and Lafuma, J. (1981). Contribution of animal experimentation in the interpretation of human epidemiological data. In: *Proc. Int. Conf. Radiation Hazards in Mining*, pp. 222–227, (ed. M. Gomez), Society of Mining Engineers, New York.

Ch81b Chovil, A. (1981). The epidemiology of primary lung cancer in uranium miners in Ontario. *J. Occup. Med.* **23**, 417–421.

Ch82 Chmelevsky, D., Kellerer, A. M., Lafuma, J. and Chameaud, J. (1982). Maximum likelihood estimation of the prevalence of nonlethal neoplasms—An application to radon daughter inhalation studies. *Radiat. Res.* **91**, 589–614.

Ch85 Chameaud, J., Masse, R., Morin, M. and Lafuma, J. (1985). Lung cancer induction by radon daughters in rats; present state of the data in low-dose exposures. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining, pp.* 350–353, *(ed. H. Stocker), Canadian Nuclear Association, Toronto.*

Ch85a Chambers, D. B. and Marchant, R. E. (1985). Potential co-carcinogens in the uranium mine environment. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 615–622, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Cl83 Cliff, K. D., Wrixon, A. D., Green, B. M. R. and Miles, J. C. H. (1983). Radon daughter exposures in the U.K. *Health Phys.* **45**, 323–330.

Co82 Cohen, B. J. (1982). Failures and critique of the BEIR III lung cancer risk estimates. *Health Phys.* **42**, 267–284.

Cr82 Cross, F. T., Palmer, R. F., Filipy, R. E. *et al.* (1982). Carcinogenic effects of radon daughters, uranium ore dust and cigarette smoke in beagle dogs. *Health Phys.* **42**, 33–52.

Cr83 Cross, F. T., Palmer, R. F., Bush, R. H., Dagle, G. E., Filipy, R. E. and Regan, R. H. (1983). An overivew of the BNL radon experiments with reference to epidemiological data. *Proc. 22nd Hanford Life Sciences Symposium, Richland, Sept. 1983.*

Da82 Damber, L. and Larsson, L. G. (1982). Combined effects of mining and smoking in the causation of lung carcinoma. *Acta Radiologica Oncology* **21**, 305–313.

Do64 Doll, R. and Hill, A. B. (1964). Mortality in relation to smoking: Ten years obsevations of British doctors. *Br. Med. J.* **1**, 1399–1410.

Do71 Doll, R. (1971). The age distribution of cancer. Implications for models of carcinogenesis. *J. Roy. Soc., Ser. A* **134**, 133–166.

Do76 Doll, R. and Peto, R. (1976). Mortality in relation to smoking: 20 years observations on male British doctors. *Br. Med. J.* **2**, 1525–1536.

Do78 Doll, R. and Peto, R. (1978). Cigarette smoking and bronchial carcinoma: Dose and time relationships among regular smokers and life-long non-smokers. *J. Epidemiol. Community Health* **32**, 303–313.

Dr85 Drexler, G. and Williams, G. (1985). Körperdosen in der Strahlentherapie, ein Beitrage zur Quantifizierung des Strahlenrisikos. In: *Strahlenschutz in Forschung und Praxis Bd. XXV*, pp. 208–220, G. Thieme Verlag, Stuttgart.

Ea84 Eaton, R. S. and Scott, A. (1984). Understanding radon transport into housing. *Radiat. Prot. Dosimetry* **7**, 251–254.

Ed83 Edling, C. (1983). Lung cancer and radon daughter exposure in mines and dwellings. *Linköping University Medical Dissertations No. 157*, Linköping, Sweden.

Ed84 Edling, C., Wingren, G. and Axelson, O. (1984). Radon daughter exposure in dwellings and lung cancer. In: *Proc. Int. Conference Indoor Air Quality, Stockholm, August 1984*, pp. 29–34, Liber Tryck AB, Stockholm.

En80 Enstrom, J. E. and Godley, F. H. (1980). Cancer mortality among a representative sample of non-smokers in the United States during 1966–68. *J. Nat. Cancer Inst.* **65**, 1175–83.

Ev81 Evans, R. D., Harley, J. H., Jacobi, W., McLean, A. S., Miles, W. A. and Stewart, C. G. (1981). Estimate of risk from environmental exposure to radon-222 and its decay products. *Nature* **290**, 98–100.

Fo81 Fox, A. J. *et al.* (1981). A study of the mortality of Cornish tin miners. *Br. J. Ind. Med.* **38**, 378.

Fo84 Folkerts, K. H., Keller, G. and Muth, H. (1984). Experimental investigations on diffusion and exhalation of ^{222}Rn and ^{220}Rn from building materials. *Radiat. Prot. Dosimetry* **7**, 41–44.

Ga80 Garfinkel, L. (1980). Cancer mortality in non-smokers: Prospective study by the American Cancer Society. *J. Nat. Cancer Inst.* **65**, 1169.

Ga81 Garfinkel, L. (1981). Time trends in lung cancer mortality among nonsmokers and a note on passive smoking. *J. Nat. Cancer Inst.* **66**, 1061.

Ge80 George, A. C. and Breslin, A. J. (1980). The distribution of ambient radon and radon daughters in residential buildings in the New Jersey–New York area. In: *Natural Radiation Environment III. CONF-780 422*, pp. 1272–1292.

Ge83 Gesell, T. F. (1983). Background atmospheric radon-222 concentrations outdoors and indoors: A review. *Health Phys.* **45**, 284–302.

Gr85 Green, B. M. R., Brown, L., Cliff, K. D., Driscoll, C. M. H., Miles, J. C. H. and Wrixon, A. D. (1985). Surveys of natural radiation exposure in UK dwellings with active and passive measurement techniques. *Science of the Total Environment* **45**, 459–466.

Gu82 Gunning, C. and Scott, A. G. (1982). Radon and thoron daughters in housing. *Health Phys.* **42**, 527–528.

Ha82 Harley, N. H. and Pasternack, B. S. (1982). Environmental radon daughters alpha dose factors in a five-lobed human lung. *Health Phys.* **42**, 789–799.

Ha83 Hayabuchi, N., Russell, W. J. and Murakami, J. (1983). Slow-growing lung cancer in a fixed population sample: Radiological assessments. *Cancer* **52**, 1098–1104.

Ha84 Harley, J. H. (1984). Indoor living, long life and radiation risk. *Radiat. Prot. Dosimetry* **7**, 19–22.

Ha85 Harley, N. H. and Fisenne, I. M. (1985). Alpha dose from long-lived alpha emitters in underground uranium mines. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 518–522, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

He83 Hess, C. T., Weiffenbach, C. V. and Norton, S. A. (1983). Environmental radon and cancer correlations in Maine. *Health Phys.* **45**, 339–348.

Ho77 Horacek, J., Placek, V. and Sevc, J. (1977). Histological types of bronchogenic cancer in relation to different conditions of radiation exposure. *Cancer* **40**, 832–835.

Ho80 Horacek, J. and Sevc, J. (1980). Histological types of pulmonary carcinoma after long-term exposure to radiation. *Studia Pneumol. Phtiseol. Cechoslov.* **40**, 324–328.

Ho82 Hofmann, W. (1982). Dose calculations for the respiratory tract from inhaled natural radioactive nuclides as a function of age. II. Basal cell dose distribution and associated lung cancer risk. *Health Phys.* **43**, 31–44.

Ho85 Hofmann, W., Katz, R. and Zhang Chunxiang (1985). Lung cancer incidence in a Chinese high background area—Epidemiological results and theoretical interpretation. *Science of the Total Environment* **45**, 527–534.

HP83 Indoor radon (A. V. Nero, W. H. Lowater, editors), 1983. *Health Physics* **45**, 273–574.

IC75 ICRP (1975). Report of the Task Group on Reference Man. *ICRP Publication 23*, Pergamon Press, Oxford.

IC77a ICRP (1977). Recommendations of the International Commission on Radiological Protection. *ICRP Publication 26*, Pergamon Press, Oxford.

IC77b ICRP (1977). Problems involved in developing an index of harm. *ICRP Publication 27*, Pergamon Press, Oxford.

IC80 ICRP (1980). Biological effects of inhaled particles. *ICRP Publication 31*, Pergamon Press, Oxford.

IC81 ICRP (1981). Limits for inhalation of radon daughters by workers. *ICRP Publication 32*, Pergamon Press, Oxford.

IC84 ICRP (1984). Principles for limiting exposure of the public to natural sources of radiation. *ICRP Publication 39*, Pergamon Press, Oxford.

In83 Ingersoll, J. G. (1983). A survey of radionuclide contents and radon emanation rates in building materials used in the U.S. *Health Phys.* **45**, 363–368.

Ja84a Jacobi, W. (1984). Possible lung cancer risk from indoor exposure to radon daughters. *Radiat. Prot. Dosimetry* **7**, 395–402.

Ja84b Jacobi, W. (1984). Expected lung cancer risk from radon daughter exposure in dwellings. In: *Proc. Int. Conf. Indoor Air Quality, Stockholm, August 1984*, Vol. 1, pp. 31–42, Swedish Council for building research, Stockholm.

Ja84c James, A. C. (1984). Dosimetric approaches to risk assessment for indoor exposure to radon daughters. *Radiat. Prot. Dosimetry* 7, 353–366.

Ja85a Jacobi, W., Paretzke, H. and Schindel, F. (1985). Lung cancer risk assessment for radon-exposed miners on the basis of a proportional hazard model. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 17–24, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Ja85b Jacobi, W. and Paretzke, H. G. (1985). Risk assessment for indoor exposure to radon daughters. *Science of the Total Environment* **45**, 531–562.

Ja85c James, A. C. (1985). Dosimetric assessment of risk from exposure to radioactivity in mine air. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 415–426, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Ja86 Jacobi, W. (1986). Carcinogenic effects of radiation on the human respiratory tract. In: *Radiation Carcinogenesis* (eds. A. C. Upton, R. E. Albert, F. J. Burns and R. E. Shore) Elsevier Science, New York.

Ka82 Kato, H. and Schull, W. J. (1982). Studies of the mortality of A-bomb survivors. 7. Mortality 1950–78: Part I. Cancer mortality. *Radiat. Res.* **90**, 395–432.

Ke82 Keller, G., Folkerts, K. H. and Muth, H. (1982). Activity concentrations of radon-222, radon-220 and their decay products in German dwellings; dose calculations and estimate of risk. *Radiat. Environ. Biophys.* **20**, 263–274.

Ke84 Keller, G., Folkerts, K. H. and Muth, H. (1984). Special aspects of the ^{222}Rn and daughter product concentrations in dwellings and in the open air. *Radiat. Prot. Dosimetry* **7**, 151–154.

Ko86 Kopecky, K. J., Nakashima, E., Yamamoto, T. and Kato, H. (1986). Lung cancer, radiation and smoking among A-bomb survivors of Hiroshima and Nagasaki. RERF Report TR 13.86, September 1986.

Ku79 Kunz, E., Sevc, J., Placek, V. and Horacek, J. (1979). Lung cancer in man in relation to different time distribution of radiation exposure. *Health Phys.* **36**, 699–706.

La84 Land, C. E. and Tokunaga, M. (1984). Induction period. In: *Radiation Carcinogenesis: Epidemiology and Biological Significance*, pp. 421–436, (eds. J. D. Boice and J. F. Fraumeni). Raven Press, New York.

Le84 Letourneau, E. G., McGregor, R. G. and Walker, W. B. (1984). Design and interpretation of large surveys for indoor exposures to radon daughters. *Radiat. Prot. Dosimetry* **7**, 303–308.

Lo81 Loewe, W. E. and Mendelsohn, E. (1981). Revised dose estimates at Hiroshima and Nagasaki. *Health Phys.* **41**, 663–666.

Lu71 Lundin, F. E., Wagoner, J. K. and Archer, V. E. (1971). Radon daughter exposure and respiratory cancer; quantitative and temporal aspects. National Institute for Occupational Safety and Health, National Institute of Environmental Health Sciences, Joint Monograph No. 1., U.S. Department of Health, Education and Welfare, Washington, D.C.

Mc80 McGregor, R. G., Vasudev, P., Letourneau, E. G. *et al.* (1980). Background concentrations of radon and radon daughters in Canadian houses. *Health Phys.* **39**, 285–289.

Mo81 Morrison, H. I., Wigle, D. T., Stocker, H. and de Villiers, A. J. (1981). Lung cancer mortality and radiation exposure among the Newfoundland fluorspar miners. In: *Proc. Int. Conf. Radiation Hazards in Mining*, pp. 372–376, Society of Mining Engineers, New York.

Mo85 Morrison, H. I., Semenciw, R. M., Mao, Y., Corkill, D. A., Dory, A. B., de Villiers, A. J., Stocker, H. and Wigle, D. T. (1985). Lung cancer mortality and radiation exposure among the Newfoundland fluorspar miners. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 365–368, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Mu83 Muller, J., Wheeler, W. C., Gentleman, J. F., Suranyi, G. and Kusiak, R. A. (1983). Study of mortality of Ontario miners, 1955–77, Part I. Report Ontario Ministry of Labour, Ontario Worker's Compensation Board, Atomic Energy Control Board of Canada.

Mu85 Muller, J., Wheeler, W. C., Gentleman, J. F., Suranyi, G. and Kusiak, R. A. (1985). Study of mortality of Ontario miners. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 335–343, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

My81 Myers, D. K., Stewart, C. G. and Johnson, J. R. (1981). Review of epidemiological studies on hazards of radon daughters. In: *Proc. Int. Conf. Radiation Hazards in Mining*, pp. 513–524, (ed. M. Gomez), Society of Mining Engineers, New York.

NA80 National Academy of Sciences/National Research Council (1980). The effects on populations of exposure to low levels of ionizing radiation. BEIR III Report, Washington, D.C.

Na84 Nazaroff, W. W. and Nero, A. V. (1984). Transport of radon from soil into residences. In: *Proc. Int. Conf. Indoor Air Quality, Stockholm, August 1984*, Vol. 2, pp. 15–20, Swedish Council for Building Research, Stockholm.

NC84a National Council on Radiation Protection and Measurements (1984). Exposures from the uranium series with emphasis on radon and its daughters. NCRP Report No. 77, Bethesda.

NC84b National Council on Radiation Protection and Measurements (1984). Evaluation of occupational and environmental exposures to radon and radon daughters in the United States. NCRP Report No. 78, Bethesda.

NE83 Nuclear Energy Agency (1983). Dosimetry aspects of exposure to radon and thoron daughter products. Report NEA/OECD, Paris.

Ne83a Nero, A. V. (1983). Airborne radionuclides and radiation in buildings; A review. *Health Phys.* **45**, 303–322.

Ne83b Nero, A. V. and Lowder, W. M. (1983). Indoor Radon. *Health Phys.* **45**, 273–574.

Ne84 Nero, A. V. and Nazaroff, W. (1984). Characterizing the source of radon indoors. *Radiat. Prot. Dosimetry* **7**, 23–40.

Ne85a Nero, A. V., Sextro, R. G., Doyk, S. M., Moed, B. A., Nazaroff, W. W., Revzan, K. L. and Schwehr, M. B. (1985). Characterizing the sources, range and environmental influences of radon-222 and its decay products. *Science of the Total Enivronment* **45**, 233–244.

Ne85b Nero, A. V., Schwehr, M. B., Nazaroff, W. W. and Revzan, K. L. (1985). Distribution of airborne ^{222}Radon concentrations in U.S. homes. Lawrence Berkeley Laboratory/University of California Report LBL 18274, July 1985.

NH85 National Institute of Health (1985). Report of the ad hoc working group to develop radioepidemiological tables. National Institute of Health Publication No. 85-2748, Washington, D.C., January 1985.

Pa85 Paschoa, A. S., Wrenn, M. E., Singh, N. P., Miller, S. C., Jones, K. W., Cholewa, M., Hanson, A. L., Saccomano, G. and Bruenger, F. W. (1985). Uranium bearing particles in miners' and millers' lungs. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 511–517, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Pe84 Pershagen, G., Damber, L. and Falk, R. (1984). Exposure to radon in dwellings and lung cancer: A pilot study. In: *Proc. Int. Conf. Indoor Air Quality, Stockholm, August 1984*, pp. 73–78, Swedish Council for Building Research, Stockholm.

Po78 Porstendörfer, J., Wicke, A. and Schraub, A. (1978). The influence of exhalation, ventilation and deposition processes upon the concentration of radon, thoron and their decay products in room air. *Health Phys.* **34**, 465–473.

Po84 Porstendörfer, J. (1984). Behaviour of radon daughter products in indoor air. *Radiat Prot. Dosimetry* **7**, 107–114.

Pr83 Prentice, R. L., Yoshimoto, Y. and Mason, M. W. (1983). Relationship of cigarette smoking and radiation exposure to cancer mortality in Hiroshima and Nagasaki. *J. Nat. Cancer Inst.* **70**, 611–622.

Pr84 Preston, D. L. (1984). Cancer mortality and incidence in the life span study: Statistical methods used in reports five through ten. In: *Atomic Bomb Survivor Data: Utilization and Analysis*, pp. 35–50, (eds. R. L. Prentice and D. J. Thompson). SIAM-SIMS Conference Series 10, SIAM, Philadelphia.

Pr86 Preston, D. L., Kato, H., Kopecky, K. J. and Fujita, S. (1986). Life Span Study Report 10, Part 1. Cancer mortality among A-bomb survivors in Hiroshima and Nagasaki, 1950–82. In preparation.

Pu85 Put, L. W. and de Meijer, R. J. (1985). Survey of radon concentrations in Dutch dwellings. *Science of the Total Environment* **45**, 389–396.

Ra84 Radford, E. P. and St. Clair Renard, K. G. (1984). Lung cancer in Swedish iron miners exposed to low doses of radon daughters. *New England J. Med.* **310**, 1485–1494.

RE84 Radiation Effects Research Foundation (1984). Reassessment of atomic bomb radiation dosimetry in Hiroshima and Nagasaki. *Proc. Workshop at Hiroshima, November 83.* Radiation Effect Research Foundation, Hiroshima.

RP84 Indoor exposure to natural radiation and associated risk assessment (1984). Proc. Int. Seminar, Capri, Oct. 1983; CONF. 83/049. *Radiat Prot. Dosimetry* **7**, (1–4).

Sa81 Saccomano, G., Archer, V. E., Auerbach, O., Kushner, M., Egger, E., Wood, S. and Mick, R. (1981). Age factor in histological type of lung cancer among uranium miners; a preliminary report. In: *Proc. Int. Conf. Radiation Hazards in Mining*, pp. 675–679, (ed. M. Gomez), Society of Mining Engineers, New York.

Sa84 Saccomano, G., Yale, C., Dixon, W. and Auerbach, O. (1984). Incidence and development of lung cancer due to cigarette smoking and radiation exposure in uranium miners. *Int. Conf. Occupational Radiation Safety in Mining, Toronto/Canada, October 1984.*

Sc82 Schüttmann, W. (1982). Ionisierende Strahlung und Bronchialkarzinom. *ZS. Erkrank. Atm. Organe* **159**, 3–15.

SC84 SENES Consultants (1984). Assessment of the scientific basis for existing federal limitations on radiation exposure to underground uranium miners. Report for the American Mining Congress. SENES Consultants Ltd., Toronto.

Sc85a Schery, S. D. and Zarcone, M. J. (1985). Thoron and thoron daughters in the indoor environment. In: *"Environmental Radiation '85", Proc. 18th Midyear Topical Symposium, Health Physics Society, 1985.*

Sc85b Schmier, H. and Wicke, A. (1985). Results from a survey of indoor radon exposure in the Federal Republic of Germany. *Science of the Total Environment* **45**, 307–310.

Sc85c Sciocchetti, G., Scacco, F. Baldassini, P. G., Battella, C., Bovi, M. and Monte, L. (1985). The Italian national survey of indoor radon exposure. *Science of the Total Environment* **45**, 327–333.

Se71 Sevc, J., Placek, V. and Jerabek, J. (1971). Lung cancer risk in relation to long-term radiation exposure in uranium mines. In: *Proc. 4th Conference in Radiation Hygiene, CSSR*, pp. 315–326.

Se76 Sevc, J., Kunz, E. and Placek, V. (1976). Lung cancer in uranium miners and long-term exposure to radon daughter products. *Health Phys.* **30**, 433–437.

Se85 Sevc, J., Placek, V., Smid, A. and Stromp, L. (1985). Lung cancer risk at low level of radiation exposure. Paper presented at the 19th Annual Meeting of the European Society for Radiation Biology, Prague, August 26–30, 1985.

SE85 Exposure to enhanced natural radiation and its regulatory implications (1985). Proc. Seminar held in Maastricht/Netherlands, March 25–27, 1985. *Science of the Total Environment* **45**, 1–699.

Si85 Singh, N. P., Bennett, D., Saccomano, G. and Wrenn, M. E. (1985). Concentrations of ^{210}Pb in uranium miners' lungs and its states of equilibria with ^{238}U, ^{234}U and ^{230}Th. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 503–506, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

Sm82 Smith, P. G. and Doll, R. (1982). Mortality among patients with ankylosing spondylitis after a single treatment course with X-rays. *Br. Med. J.* **284**, 449–460.

Sn73 Snihs, J. O. (1973). The approach to radon problems in non-uranium mines in Sweden. In: *Proc. 3rd Int. Congress of IRPA, US-AEC Report Conf. 730907*, pp. 900–911.

St75 Steinhäusler, F. (1975). Long-term measurements of ^{222}Rn, ^{220}Rn, ^{214}Pb and ^{212}Pb concentrations in the air of private and public buildings and their dependency on meteorological parameters. *Health Phys.* **29**, 705–714.

St84 Stewart, C. G. (1984). Comments upon basis of intake limits for radon and thoron daughters, proposed by the Atomic Energy Control Board of Canada in Consultative Document C-78, entitled Limitation of Exposure to Ionizing Radiation, issued for Comment, 14 November, 1983, SENES Consultants Ltd., Toronto.

St80 Stranden, E. and Berteig, L. (1980). Radon in dwellings and influencing factors. *Health Phys.* **39**, 275–284.

St86a Stranden, E. (1986). Radon in Norwegian dwellings and the feasibility of epidemiological studies. *Radiat. Environ. Biophys.* **25**, 37–42.

St86b Stranden, B. (1986). Radon-222 in Norwegian Dwellings. Paper Presented at the American Chemical Society Radon Symposium, New York, April 14–16, 1986.

Sv85 Svensson, C., Eklund, G. and Pershagen, G. (1985). Indoor exposure to radon from the ground and bronchial cancer among women. Paper presented at the IV World Conference on Lung Cancer, Toronto, August 1985 (submitted for publication in Archives of Environmental Health).

Sw83 Swedjemark, G. A. (1983). The equilibrium factor F. *Health Phys.* **45**, 453–462.

Sw84 Swedjemark, G. A. and Mjönes, L. (1984). Radon and radon daughter concentrations in Swedish houses. *Radiat Prot. Dosimetry* **7**, 341–346.

Sw86 Swedjemark, G. A., Buren, A. and Mjönes, L. (1986). A comparison of radon levels in Swedish homes in the 1980s and thirty years ago. Paper presented at the American Chemical Society Radon Symposium, New York, April 14–16, 1986.

Th82 Thomas, D. C. and McNeill, K. G. (1982). Risk estimates for the health effects of alpha radiation. *Research Report INFO-0081*, Atomic Energy Control Board, Ottawa.

Th85 Thomas, D. C., McNeill, K. G. and Dougherty, C. (1985). Estimates of lifetime lung cancer risks resulting from Rn progeny exposure. *Health Phys.* **49** (5), 825–846.

Ti85 Tirmanche, M., Brenot, J., Piechowski, J., Chameaud, J. and Pradel, J (1985). The present state of an epidemiological study of uranium miners in France. In: *Proc. Int. Conf. Occupational Radiation Safety in Mining*, pp. 344–349, (ed. H. Stocker), Canadian Nuclear Association, Toronto.

To78 Townsend, J. (1978). Smoking and lung cancer: A cohort data study of men and women in England and Wales 1935–1970. *J. Roy. Statist. Soc., Ser. A* **141** (Part 1), 95–107.

UN77 UNSCEAR (1977). Sources and effects of ionizing radiation. United Nations, Publication No. E.77.IX.1, New York.

UN82 UNSCEAR (1982). Ionizing radiations: Sources and Biological Effects. United Nations, Publication No. E.82.IX.8., New York (with annex D: Exposures to radon and thoron and their decay products).

US82 U.S. Department of Health and Human Services (1982). The health consequences of smoking—Cancer: A report of the Surgeon General. DHHS Publ. No. (PHS) 82-50179.

Wa81 Waxweiler, R. J., Roscoe, R. J., Archer, V. E., Thun, M. J., Wagoner, K. J. and Lundin, F. E. (1981). Mortality follow-up through 1977 of white underground miners cohort examined by the US-Public Health Service. In: *Proc. Int. Conf. Radiation Hazards in Mining*, pp. 823–830, (ed. M. Gomez), Society of Mining Engineers, New York.

Wa83 Wakabayashi, T., Kato, H., Ikeda, T. and Schull, W. J. (1983). Studies of the mortality of A-bomb survivors, Report 7. Part III. Incidence of cancer in 1959–1978, based on the tumor registry, Nagasaki. *Radiat. Res.* **93**, 112–146.

We85 Weihui He *et al.* (1985). A summary of third stage investigations on cancer mortality in high background radiation area (1970–83). *Chinese J. Radiol. Med. Prot.* **5** (2), 109–113.

WH75 World Health Organization (1975). Smoking and its effects on health. WHO Tech. Report Series No. 568, Geneva.

WH83 World Health Organization (1983). World health statistics; annual vital statistics and causes of death. WHO Report, Geneva.

Wh83 Whittemore, A. S. and McMillan, A. (1983). Lung cancer mortality among U.S. uranium miners: A reappraisal. *J. Nat. Cancer Inst.* **71**, 489–499.

Wi79 Wicke, A. (1979). Untersuchungen zur Frage der natürlichen Radioaktivität der Luft in Wohn- und Aufenthaltsräumen. Doctor Thesis, University of Giessen, F.R.G.

Wi82 Wicke, A. and Porstendörfer, J. (1982). Radon daughter equilibrium in dwellings. In: *Natural Radiation Environment*, pp. 481–488, (eds. K. Vohra *et al.*), Wiley Eastern Ltd., New Delhi.

Wr84 Wrixon, A. D., Brown, L., Cliff, K. D. *et al.* (1984). Indoor radiation surveys in the U.K. *Radiat. Prot. Dosimetry* **7**, 321–325.

Wr85 Wrixon, A. D. and O'Riordan, M. C. (1985). The control of indoor radiation exposure. *Science of the Total Environment* **45**, 657–676.

Wy77 Wynder, E. L. and Stellmann, S. D. (1977). Comparative epidemiology of tobacco-related cancers. *Cancer Res.* **37**, 4608–22.

Wy83 Wynder, E. L. and Goodman, M. T. (1983). Smoking and lung cancer: Some unresolved issues. *Epidemiol. Rev.* **5**, 177–207.

Zh81 Zhang, J. Y. *et al.* (1981). Investigation of the radioactive aetiological factors of lung cancer among miners. *Acta Academiae Medicinae Sinicae* (No. 3)., 127–131.

APPENDIX: SPECIAL QUANTITIES AND UNITS

A.1. Potential Alpha Energy of Radon Daughters

The potential alpha energy ε_p of a radon daughter atom in the decay chain of ^{222}Rn (radon) or ^{220}Rn (thoron) is the total alpha energy emitted during the decay of this atom to ^{210}Pb or ^{208}Pb, respectively. Consequently, the potential alpha energy per unit of activity (Bq) of a daughter nuclide is $\varepsilon_p/\lambda_r = \varepsilon_p T_r/\ln 2$ where λ_r is the decay constant and T_r the radioactive half-life of the considered radionuclide. Values of ε_p and $\varepsilon_p/\lambda r$ are listed in Table A1.

Table A.1. Potential alpha energy per atom and per Bq activity of radon daughters

Radio nuclide	T_r	Potential alpha energy per atom (MeV)	per atom (10^{12}J)	per unit of activity (MeV/Bq)	per unit of activity (10^{-10}J/Bq)
^{222}Rn(Rn) daughters:					
^{218}Po	3.05 min	13.7	2.19	3 620	5.79
^{214}Pb	26.8 min	7.69	1.23	17 800	28.6
^{214}Bi	19.7 min	7.69	1.23	13 100	21.0
^{214}Po	164 μs	7.69	1.23	2×10^{-3}	3×10^{-6}
^{220}Rn(Tn) daughters:					
^{216}Po	0.15 s	14.6	2.34	3.32	5.3×10^{-3}
^{212}Pb	10.64 h	7.8	1.25	431 000	691
^{212}Bi	60.6 min	7.8	1.25	40 900	65.6
^{212}Po	304 ns	8.78	1.41	3.8×10^{-6}	6.2×10^{-9}

A.2. Potential Alpha Energy Concentration in Air

The potential alpha energy concentration of any mixture of short-lived ^{222}Rn(Rn) or ^{220}Rn(Tn) daughters in air is the sum of the potential alpha energy of all short-lived daughter atoms present per unit volume of air. Thus, if $c_{act,i}$ is the activity concentration of a daughter nuclide i, the potential alpha energy concentration c_p of the daughter mixture is given by:

$$c_p = \underset{i}{\mathrm{j}}\, c^{p,i} = \underset{i}{\mathrm{j}}\, c_{act,i} \times \varepsilon_{p,i}/\lambda_{r,i} \qquad \text{(A1)}$$

summing over all short-lived daughter nuclides of ^{222}Rn or ^{220}Rn, respectively. This quantity is expressed in the SI unit J/m^3, where 1 J/m^3 = 6.24×10^{12} MeV/m^3.

A.3. Equilibrium Equivalent Concentration in Air

The potential alpha energy concentration of any daughter mixture in air can also be expressed in terms of the so-called equilibrium-equivalent concentration (EEC) of their mother nuclide, ^{222}Rn(Rn) or ^{220}Rn(Tn), respectively. The EEC of a non-equilibrium mixture of short-lived radon daughters in air is that activity concentration of ^{222}Rn(Rn), or ^{200}Rn(Tn), in radioactive equilibrium with its short-lived daughters, which has the same potential alpha energy concentration, c_p, as the actual non-equilibrium mixture.

This definition leads to the following relationships:

$$\mathrm{EEC_{Rn}} = 1.81 \times 10^8\ c_p \text{ for } ^{222}\mathrm{Rn(Rn)} \text{ daughters} \tag{A2}$$

$$\mathrm{EEC_{Tn}} = 1.32 \times 10^7\ c_p \text{ for } ^{220}\mathrm{Rn(Tn)} \text{ daughters} \tag{A3}$$

where $\mathrm{EEC_{Rn}}$ and $\mathrm{EEC_{Tn}}$ are the equilibrium-equivalent concentrations of radon and thoron, respectively, in units of Bq/m^3; and c_p is the potential alpha energy concentration of radon or thoron daughters, in units of J/m^3.

A.4. Equilibrium Factor in Air

The "equilibrium factor" F is defined as the ratio of the EEC to the actual activity concentration c_{act} of the mother nuclide in air:

$$F_{\text{Rn daughters}} = \frac{\mathrm{EEC_{Rn}}}{c_{\mathrm{act,Rn}}}$$

$$F_{\text{Tn daughters}} = \frac{\mathrm{EEC_{Tn}}}{c_{\mathrm{act,Tn}}} \tag{A4}$$

Thus, this factor characterizes the disequilibrium between the radon daughter mixture and their mother nuclide in terms of potential alpha energy.

A.5. Radon Daughter Exposure

The quantity "radon daughter exposure", E, of an individual, as used in this report, is defined as the time integral over the potential alpha energy concentration c_p of the daughter mixture in air, or the corresponding equilibrium-equivalent concentration (EEC] of radon, to which the individual is exposed over a given time period T, for example one year (annual exposure):

$$E_p(T) = \int_0^T c_p(t)\mathrm{d}t$$

$$= \text{potential alpha energy exposure}$$

$$E_{act}(T) = \int_0^T \mathrm{EEC}(t)\mathrm{d}t$$

$$= \text{equilibrium-equivalent activity exposure.} \tag{A5}$$

The SI unit of the exposure E_p is J h/m^3. The radon daughter exposure of miners is often expressed in the unit WLM (Working Level Months), where one Working Level Month corresponds to exposure at one Working Level for 160 h, a period appropriate to conditions of occupational exposure. Thus;

$$1\ \mathrm{WLM} = 160\ \mathrm{WL\ h} = 3.5 \times 10^{-3}\ \mathrm{J\ h/m^3} = 3.5\ \mathrm{mJ\ h/m^3} \tag{A6}$$

(or 1 J h/m^3 = 285 WLM).

The corresponding unit of the activity exposure E_{act} is Bq h/m^3 (Rn- or Tn-eq). From eqns

(A2) and (A3), the following conversion factors between the units of the two exposure quantities result:

^{222}Rn(Rn) daughters

$1 \text{ Bq h/m}^3 = 5.52 \times 10^{-9} \text{ J h/m}^3 = 1.60 \times 10^{-6} \text{ WLM}$

or $1 \text{ J h/m}^3 = 1.81 \times 10^{8} \text{ Bq h/m}^3$

$1 \text{ WLM} = 6.3 \times 10^{5} \text{ Bq h/m}^3$ (A7)

^{220}Rn(Tn) daughters

$1 \text{ Bq h/m}^3 = 7.58 \times 10^{-8} \text{ J h/m}^3 = 2.16 \times 10^{-5} \text{ WLM}$

or $1 \text{ J h/m}^3 = 1.32 \times 10^{7} \text{ Bq}(\text{EEC}_{\text{Tn}})\text{h/m}^3$

$1 \text{ WLM} = 4.63 \times 10^{4} \text{ Bq h/m}^3$ (A8)

In this report, the radon daughter exposure of individuals during their residence time indoors and outdoors is expressed in terms of the equilibrium-equivalent activity exposure to ^{222}Rn(Rn) or ^{220}Rn(Tn), respectively, using the unit Bq h/m^3.

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