Soz Präventivmed 1991; *36*: 255–265

0303-8408/91/050255-11 $1.50 + 0.20/0


# Lung cancer and alpha radiation

Werner Burkart

Institut für Strahlenhygiene/BfS, Neuherberg/München

Both from the point of view of contracted dose and radiosensitivity, the human lung is the most critical organ for late somatic health effects from exposure to ionizing radiation in our environment. Since the total radiation dose is dominated by the exposure of the lung to natural airborne decay products of the uranium and thorium decay chains in the indoor environment, the tracheobronchial region of the respiratory system generally receives radiation doses which are at least an order of magnitude above those of any other organ. The high-LET radiation of the predominant alpha-particles produces highly localized lung exposures which are qualitatively different from the beta- and gamma-irradiation dominating in the rest of the body.

## Radon, thoron and decay products

The noble gases radon ($^{222}$radon), thoron ($^{220}$radon) and their short-lived decay products are members of the natural $^{238}$uranium and $^{232}$thorium series. In the long decay chains from the actinides to stable lead, only gaseous radon is volatile and hence able to enter the indoor environment or the atmosphere in large quantities to become, through its decay products, a major contributor to the collective dose to mankind[1]. Figure 1 gives the half-lives and decay energies for the most important natural series starting with $^{238}$uranium and ending with the stable lead isotope $^{206}$Pb. Radon mainly acts as a carrier of potential alpha-decay energy from the ground to the biosphere. Upon its decay in the air, the short-lived radon daughters $^{218,214}$polonium, $^{214}$lead, and $^{214}$bismuth will become attached to aerosol particles. Inhalation and deposition of these particles lead to a highly localized irradiation of the linings of the respiratory tract. The direct source of radon and its decay products is the $^{226}$radium present ubiquitously in soil, water, building materials and natural gas. The half-life of 3.8 days allows the noble gas $^{222}$radon to diffuse or to be carried by air or water over considerable distances.

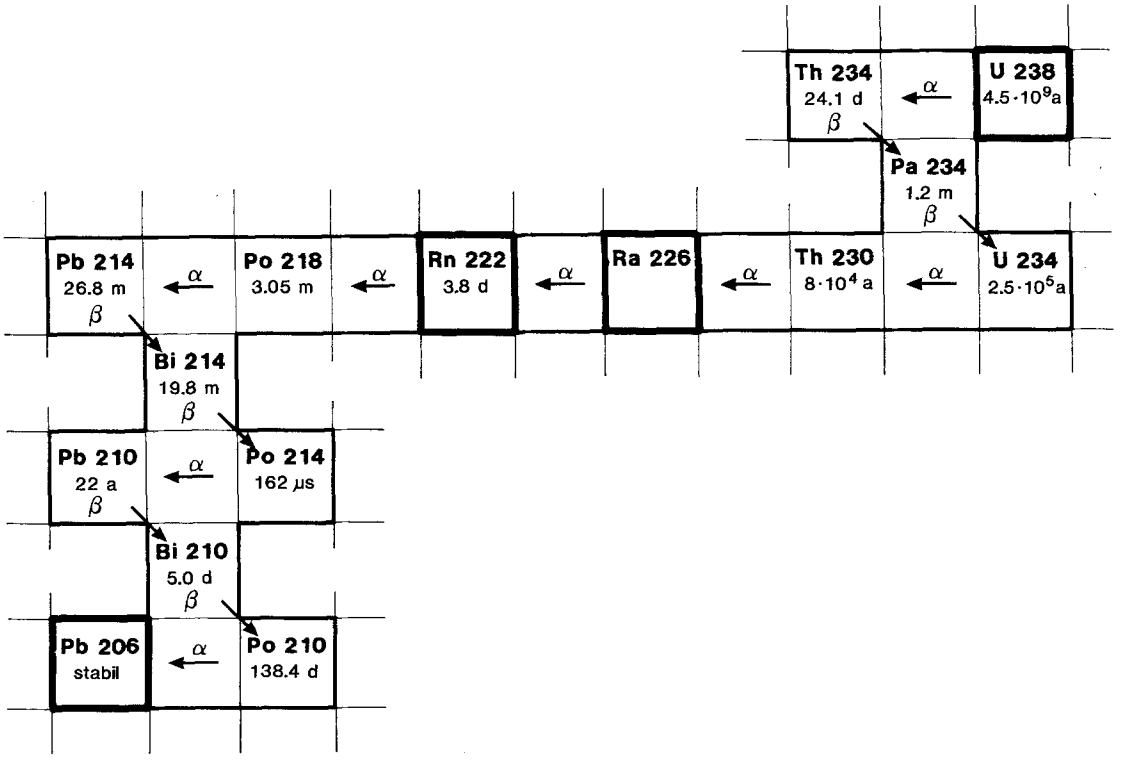


Fig. 1. Decay scheme from $^{238}$uranium to stable $^{206}$lead with half-lives, decay modes and energies (modified from Seelmann et al. (1981)).

$^{220}$radon and $^{219}$radon (actinon) and their decay products, formed in the $^{232}$thorium and $^{235}$uranium series, are of lesser importance because of their short half-lives of 55.6 and 3.9 seconds, respectively. The short average life prevents a large fraction of these radon atoms from leaving the matrix before further decay to nuclides of heavy metal occurs.
Radiation exposures from radon and its decay products provide the most important fraction of the exposure to the general public from ionizing radiation. In Switzerland the estimated average annual lung dose is about 16 mSv, leading to 2 mSv $H_e$ per year.

## Estimates of lung cancer risk from exposure to radon daughters

Studies of uranium miners, which have sufficient statistical power to allow an analysis of the shape of the dose/effect relationship between exposure to radon daughters and attributable excess lung cancer risk, point to or are compatible with a linear relationship, without threshold, in the dose range from zero to about 4 Sv $H_e$. At higher cumulative doses, the curve is often slightly downward concave, i.e. predicts a lower relative or absolute excess risk per unit dose[2,3]. Cumulative doses above 4 Sv $H_e$ were accumulated in uncontrolled exposures such as the ones prevailing in early uranium mining. However, an assessment of the radiogenic lung cancer risk in the controlled workplace or in dwellings is restricted to much lower doses for which a linear dose/effect relationship is the best estimate[4]. The assumption of a proportional increase of risk with dose still leaves several questions unanswered. Beside smoking and the two different risk concepts treated separately in this chapter, age at exposure, sex, and confounding factors such as diesel fumes in mines, have to be considered.
Epidemiological data for lung exposure to alpha-radiation is mainly based on male populations. Earlier findings of the Life Span Study in Hiroshima and Nagasaki on low-LET irradiation suggested a similar absolute excess risk of lung cancer for males and females[5]. Since the non-radiogenic lung cancer incidence in females is lower, this would translate into a much greater relative excess risk from low-LET radiation for females. In view of a new assessment of the data[6], yielding no significant differences in the risk factors for the two sexes, the same relative risk for lung cancer is suggested by ICRP[3].
For adults, the relative lung cancer risk per unit dose seems to be independent of the age at exposure. However, data on exposed children from the Life Span Study of the atomic bomb survivors suggest a relative risk which is at least twice as large as that for adults in the same study[7]. On the other end of the age scale, a recent paper by Hornung and Meinhardt[2] shows a statistically significant effect of age at initial exposure for U.S. uranium miners. This contradicts the notion, based in part on the findings from Hiroshima and Nagasaki, that the risk coefficients in a relative model tend to decrease with increasing age at exposure. Specifically, a miner with the same radon daughter exposure and smoking history, who was initially exposed 10 years later in age than the control miner, would have a 32 percent higher risk of lung cancer.
Confounding chemical and physical factors other than smoking seem to be of surprisingly small importance for the induction of lung cancer in miners. Ore dust serves as an example. Its concentration is more elevated near crushers above the ground than in the mine proper, but this was shown to have no influence on the lung cancer risk of the personnel[8].
Beside the inference of risk from the epidemiological investigation of exposed populations, lung cancer risks associated with the exposure to $^{222}$radon and its decay products can also be characterized via the radiation dose to the target cells. This dosimetric approach which is used by many investigators and committees (for an overview see Nuclear Energy Agency[9]; ICRP[3]), is based on the quantification of dose to the stem cells of the tracheo-bronchial tree. Unfortunately, the quantitative information needed to derive a risk coefficient solely from these dosimetric models is clearly not sufficient. Although aerosol characteristics, deposition probabilities, and morphometric data of the lung may be available for specific exposure situations, much of the additional information needed is based on assumptions which can not be directly verified. Type, number, location, and transformation probabilities of the target cells for cancer induction are important but inadequately characterized model parameters. Therefore, the U.S. National Research Council, in its BEIR IV report on the health risk of radon and other internally deposited alpha-emitters[10], chose a purely epidemiological approach for estimating lung cancer risks. Even so, it was accepted that dosimetric models play an important role in the scaling and extrapolating of data from adult, physically working male populations, which dominate the epidemiological studies on radon risks, to the general public exposed to $^{222}$radon and its decay products in the indoor environment.

## Relative risk models

A concise treatment of the relative risk projection model for radon-induced lung cancer can be found in ICRP Report 50[3]. In the scope of this treatise, dealing mainly with environmental exposures to

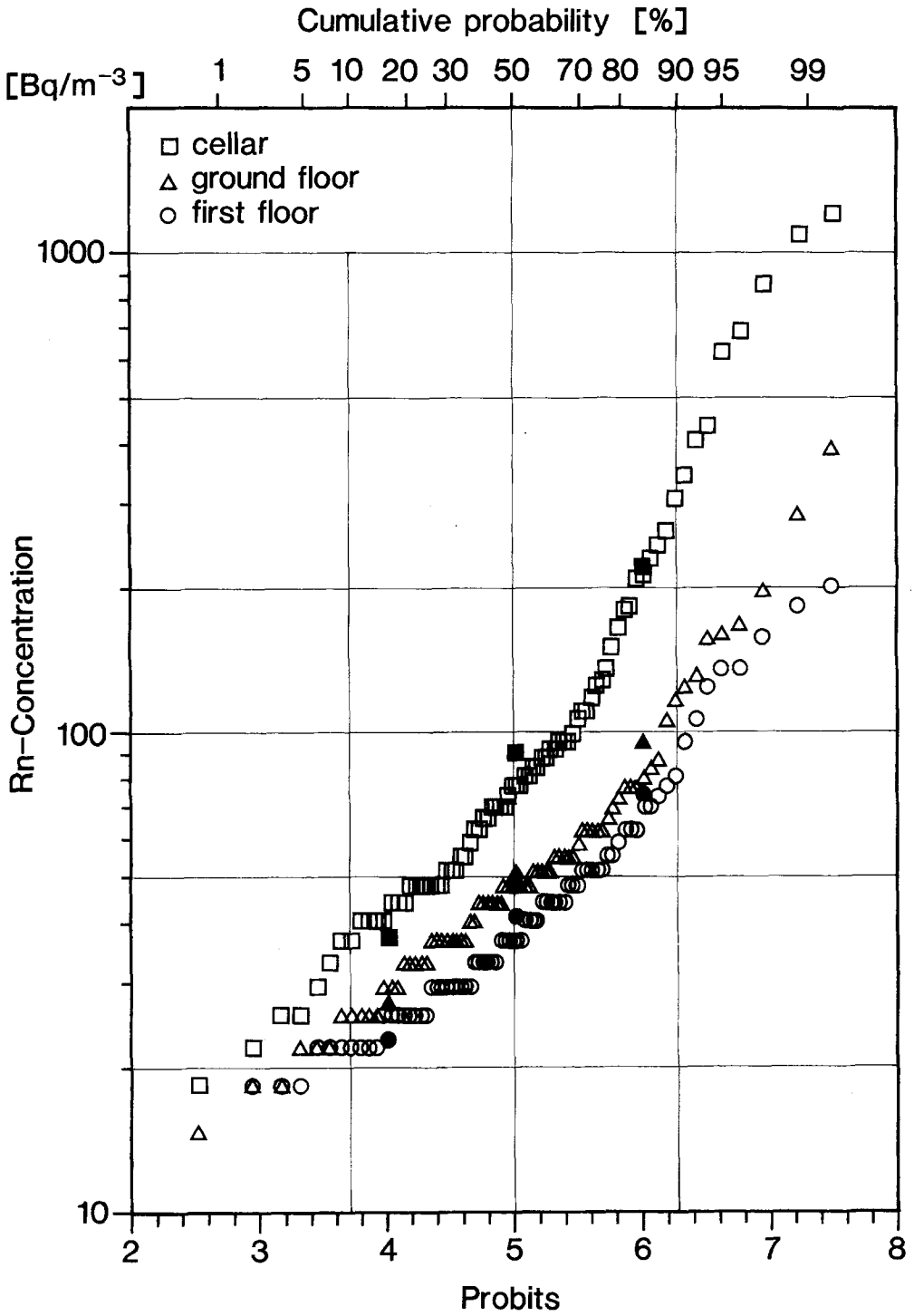


Fig. 2. Cumulative probability distribution of the time averaged indoor $^{222}$radon concentrations during the winters of 1981/82 and 1982/83 in a sample of 152 single family dwellings in the Molasse Basin (Mittelland) of Switzerland (Crameri et al., 1989).

radon daughter radioactivity, two simplifying assumptions can be made. If the exposure rate and the relative excess cancer risk coefficient are constants, i.e. not changing with time or age, the following equation for the total age-specific lung cancer rate emerges:

$$\delta(t) = \delta_0(t) \times [1 + r \times E \times (t - \gamma)]$$

$\delta(t)$: age-specific lung cancer mortality rate

$\delta_0(t)$: spontaneous lung cancer mortality rate in the unexposed individual

t: age

r: relative risk coefficient (assumed constant over age)

E: annual exposure to radon daughters

$\gamma$: latency period (time between exposure and onset of elevated risk) (assumed to be constant)

The proportional hazard model can also be applied to smokers. This would imply a multiplicative or synergistic influence of tobacco smoke on the lung cancer mortality rate. Such a relationship is often suggested by epidemiological studies with a short to moderate follow-up period. However, an update of the lung cancer risk assessment of the U.S. uranium miners suggests the presence of a considerable additive component for the extended follow-up of populations at risk [2]. Assuming the following equation for smokers not exposed to ionizing radiation:

$$\delta_{0,s}(t) = \delta_{0,ns}(t) \times [1 + S_s(t)]$$

$\delta_{0,s}(t)$, $\delta_{0,ns}(t)$: age-specific lung cancer mortality rate for smokers and non-smokers, respectively

$S_s(t)$: smoking factor characterizing the synergistic influence of smoking

the total age-specific lung cancer mortality rate for smokers in a relative risk model is given by:

$$\delta_s(t) = \delta_{0,ns}(t) \times (1 + S_s(t)) \times [1 + r \times E \times (t - \gamma)]$$

spontaneous smoking radon

The three terms "spontaneous", "smoking" and "radon" denote the corresponding contributions to the total, age-specific lung cancer mortality rate. For chronic smokers who start smoking at the age of 20 years and continue smoking at the same rate until the end of their life, the smoking factor $S_s$ amounts to about 0.7 per cigarette/day [11,12]. For non-smokers the second term is one or near to one if possible detrimental health effects from passive smoking are taken into account here.

In recent publications, relative risk models are clearly favoured over absolute models for radiation-induced carcinogenesis in man. Although they tend to be in better agreement with the epidemiological data, the large influence of age makes them more prone to large errors for time intervals not covered by the epidemiological follow-up. Under the assumption of a fixed age-independent relative risk coefficient for the life span remaining after the onset of expression of the cancer risk (age at exposure plus latency period), many fatalities will occur late in life in a period not yet covered in epidemiological studies. Figures 3 and 4 illustrate this point graphically for an acute and a chronic life-time exposure, respectively. In both situations, a constant relative risk coefficient and an infinite expression period is assumed. The initial steeper increase of the RR in the bottom graph of Figure 4 is a reflection of the three times higher risk coefficient for ages below 20 assumed by ICRP [3].

**Absolute risk models**

An absolute excess risk model assumes that the radiogenic and other pathways leading to the appearance of lung cancer act independently. Therefore no temporal correlation with the spontaneous or tobacco-dependent lung cancer rates, which are both highly age-dependent, increasing over a considerable part of the human life span with the $4.5^{th}$ and $5^{th}$ power of age, respectively, is sought. Although the prevalence of such models in the past was mainly a matter of the methods in use at that time, a biological basis could also be given. A cancer induction mechanism with only one rate-limiting step, such as a mutational event in a single oncogene, may well serve as the basic theory for the independence of radiogenic cancer induction in man from other chemical or endogenous pathways.

For a single, acute exposure to ionizing radiation, an absolute excess risk model will predict that, after a latency period of several years, a constant excess rate of lung cancer will be discernible in the population for the remaining lifetime or for a fixed expression period. For a linear dose/effect relationship, the model can be described by the following equation:

$$\delta_e(t_e, t) = a(t_e) \times E(t_e) \quad \text{for} \quad t > t_0 \quad \text{and} \quad t > t_e + \tau$$

$\delta_e$: annual radiogenic excess lung cancer rate

$t_e, t$: age at exposure, age at risk, respectively

a: absolute risk coefficient

E: Exposure at time $t_e$

$\tau$: latency period

$t_0$: 40 years (accounts for the fact that lung cancer below the age of 40 is very rare)

For a chronic exposure situation such as the exposure to radon daughters in the indoor environment, the annual excess lung cancer rate is given by integrating exposures multiplied with age-specific absolute risk coefficients. Assuming an age-averaged risk factor ($a_a$) and stable exposure conditions, the excess radiogenic lung cancer rate is given by:

$$\delta_e(t) = a_a \times E_a \times (t - \tau) \quad \text{for} \quad t > 40 \text{ years}$$

$a_a$: age-averaged absolute risk coefficient

$E_a$: annual exposure in chronic exposure situations

For $^{222}$radon in the indoor environment, ICRP gives an age-averaged risk coefficient ($a_a$) of $1.1 \times 10^{-11}\,\text{year}^{-1} \cdot (\text{Bq/m}^3 \cdot \text{h})^{-1}$ [3].

From the models adopted by major committees in the eighties, only the NCRP 78 model [13] is based on absolute risk, i.e. an additive rather than multiplicative relationship between excess and baseline risk. The radiation-induced excess risk is assumed to be independent of smoking and sex. The risk at a given age is only determined by exposure level and time since exposure. There is a latent period of 5 years with no excess risk; later the lung cancer risk from radon exponentially de-

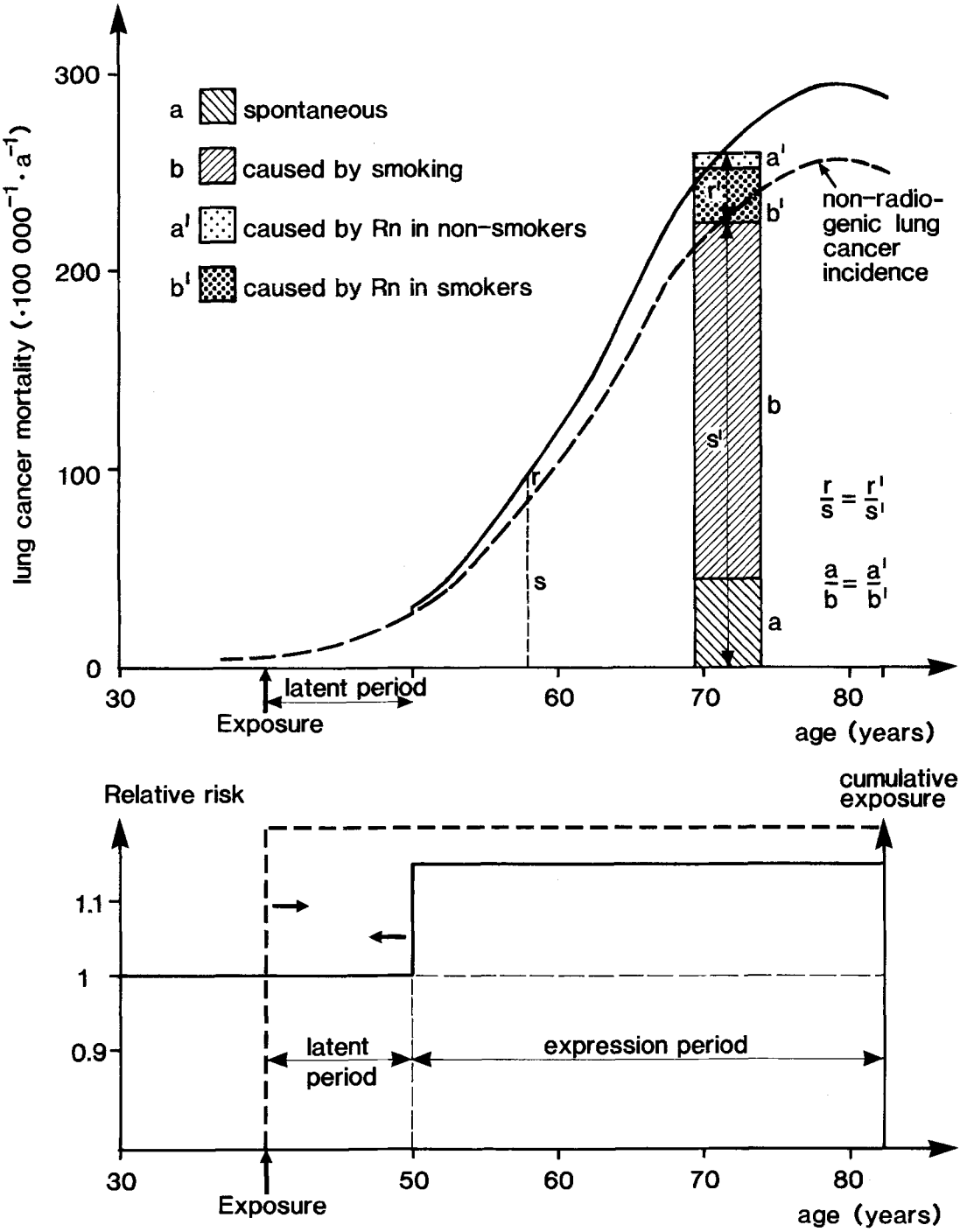


Fig. 3. Age dependency of excess lung cancer mortality in the relative risk model after a single acute exposure of the lung leading to a RR of 1.15. The curve for the "non-radiogenic" rate is based on the Swiss mortality for 1984 to 1985 (both sexes, smokers and non-smokers; Schüler, personal communication, 1987).

creases. In addition it is assumed that there is no excess lung cancer risk before the age of 40. The NCRP 78 model results in the following equation:

$$\delta_e(t_e, t, E, S) = \delta_0(t, S) + 10^{-5} \times a(t) f(t - t_e) \times E$$

$$a(t) = 1 \quad \text{if} \quad t > 40$$
$$= 0 \quad \text{if} \quad t < 40$$

and

$$f(t - t_e) = e^{-(t-t_e) \times \ln(2)/20} \quad \text{if} \quad t - t_e > 5$$
$$= 0 \quad \text{if} \quad t - t_e < 5$$

$\delta_e$: annual radiogenic incidence lung cancer rate

$\delta_0(t, S)$: baseline rate for persons of age t with smoking history S

$t_e, t$: age at exposure, age, respectively

a: absolute risk coefficient

E: Exposure at time $t_e$

The decrease in absolute risk coefficient with time since exposure is based on a biological model which assumes that stem cells in the bronchial

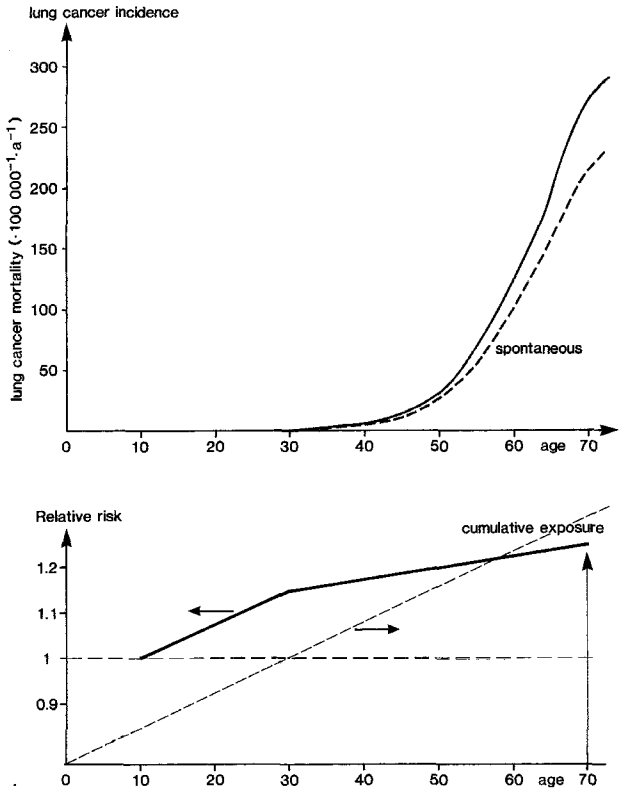


Fig. 4. Age dependency of excess lung cancer mortality in the relative risk model for a chronic exposure to $^{222}$radon and its decay products. The lower graph shows the accumulation of dose and the increase of the relative risk with time under the assumption of a ten year latency period. The steeper increase of the RR at the beginning is due to the higher excess risk coefficient below the age of 20.

epithelium, which are transformed by the radon daughter exposure, have themselves a half-life for survival or proliferation competence, i.e. are cleared from the lung with time. The maximal excess lung cancer risk occurs 5 years after cessation of exposure. From then on the radiogenic risk decreases with age, which is in marked contrast to the non-radiogenic risk, which increases dramatically with age until age 70. In the last few years, the poor fit of the model with epidemiological data from extended follow-up of miners and the absence of a risk modifier for smoking led to the demise of the NCRP approach.

## Fitting the data, the BEIR IV model

Both the relative and the absolute excess risk models for α-radiation-induced lung cancer can be fitted to available epidemiological data. However, prediction of risk for age groups poorly represented in the human populations studied for radiogenic lung cancer may vary considerably between the different models. Since recent developments indicate that the long-term excess risk is somewhere between the projections from relative and absolute models[2,5,10], a pure relative risk model with a life-long expression period may substantially overestimate the lung cancer mortality risk from ionizing radiation for age segments with high spontaneous lung cancer mortality, i.e. older age groups. The resulting misperception is further increased because of the decreasing individual and societal detriment with increasing age at diagnosis of the lung cancer. In many recent approaches, a cancer occurring in old age is weighted less than a fatal cancer early in life. This is done by normalizing the impact of the health detriment on an index of harm, e.g. on a scale of loss of life expectancy[14]. The first problem can be alleviated by introducing correcting elements into the models. To get a better fit of the relative excess model for the Hiroshima/Nagasaki data, the relative excess risk coefficient for exposure below the age of 20 is taken to be 3 times larger than for adults, thus correcting the very low risk prediction of this model for younger age groups[3]. This finding from low-LET radiation has a very low statistical power. To use it on exposures to α-irradiation early in life is prone to large uncertainties. A limitation of the expression period or a decrease of the relative excess risk coefficient after a certain time since exposure for radiogenic lung cancer mortality could also be envisaged in view of the latest findings[2,10]. In the absolute model, which tends to overestimate risk at lower ages and to underestimate at higher ages, the validity of the model may be restricted to people above the age of 40, and a longer latency period may be assumed[13].

The BEIR IV[10] report contains a careful and sophisticated approach to fit a model to epidemiological data from miner populations. In its analyses, the committee focused on the following potential risk factors:

- cumulative exposure
- duration of exposure
- age at which risk is being evaluated
- age at first exposure
- time since cessation of exposure
- time since different parts of exposure
- effects of smoking (only in the Colorado Plateau uranium miner cohort)

The BEIR IV committee used a relative risk model, i.e. the ratio of the excess risk to the background age-specific risk, to examine how the age-specific risk depends on the above variables. The report states that this was done by making a cross-classification of numbers of lung-cancer deaths and person-years at risk, by categories of these variables, and then fitting models to the rates given by the ratio of deaths to person-years in such a tabular cross-classification. The regression models were fitted with a Poisson probability model for the number of deaths in each cell of the table, where the expected value was taken as the product of the person-years at risk for the

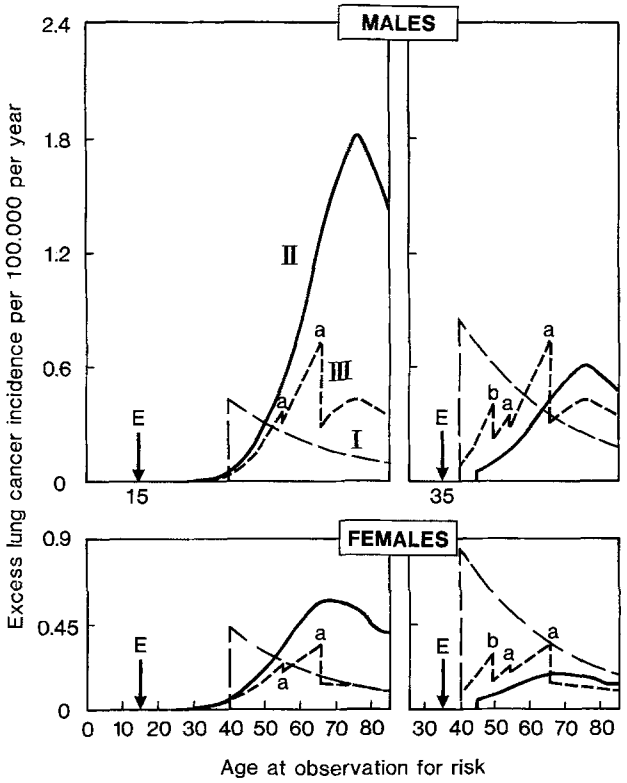


Fig. 5. Excess incidence rates for lung cancer induced by exposure to 1 WLM (about 7 mSv $H_e$) by sex and two exposure ages in non-smokers as predicted by the NCRP 78, ICRP 50, and BEIR IV models (Land, 1989, based on U.S. life tables and non-radiogenic incidence rates). **I**: NCRP 78; **II**: ICRP 50; **III**: BEIR IV; E: Exposure a, b: Changes in the BEIR IV risk estimate due to "age at risk"-dependent reduction steps (a) and due to "time since exposure"-dependent reduction steps (b).

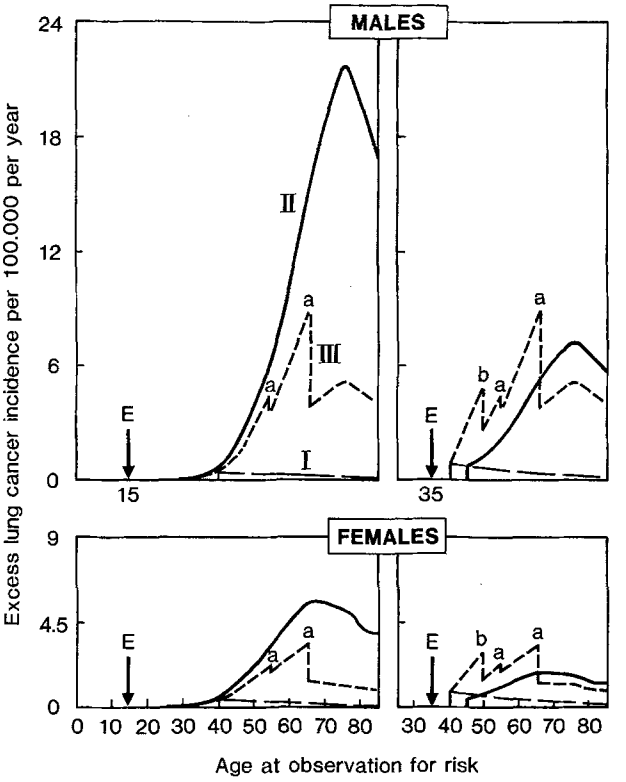


Fig. 6. As figure 5, but for smokers. **I**: NCRP 78; **II**: ICRP 50; **III**: BEIR IV.

cell and a cancer rate given by the parametric model. This work is described in depth in Annex 2A of the BEIR IV report [10]. The starting point of the analysis was the following general mathematical form of the relative risk:

$$\delta(a) = \delta_0(a)[1 + \{\beta \times \gamma(v) \times W\}]$$

$\delta(a)$: lung-cancer mortality rate for a given age and calender period

$\delta_0(a)$: background or baseline risk of the lung-cancer mortality in the population

W: cumulative exposure in WLM at 5 years before age a.
WLM (working level month): cumulative exposure, equivalent to the exposure to one working level (WL, 3700 Bq · $m^{-3}$ of $^{222}Rn$ in equilibrium with its short-lived daughters) for a working month (170 hours); 1 WLM corresponds to a dose of 8.5 mSv $H_e$ (occupational) and 5.5 mSv $H_e$ (non-occupational), respectively

$\beta$: basic slope of the dose-response relation

$\gamma(v)$: modifying effects of variables ($v$) other than cumulative exposure

This general form assumes that exposures have no substantial effect on risk of lung-cancer mortality for the first 5 years, and that risk increases linearly with cumulative dose. The analyses were made both by comparison with external population rates and by internal comparisons (e.g. low to high exposures). For external comparisons, population rates were standardized to make them consistent with the experience of the cohort(s) at zero (surface workers) and low exposures. Thus, external rates were used only to incorporate knowledge of lung-cancer mortality trends in age and calendar time. No significant effects on relative risk due to age at first exposure or duration of exposure (dose rate) were found when the model was applied to the four cohorts studied. However, risk per unit exposure decreased at very high cumulative doses above 2,000 WLM. Consistent and significant effects of age a, at which risk is evaluated (age at risk), and of time since cessation of exposure were seen. The excess relative risk per unit exposure decreased substantially with an increase in each of these two variables.

The BEIR IV committee felt that for prolonged exposures, early exposures may contribute less to the risk later in life, and tried to fit a time since exposure (TSE) model, which contained three time intervals prior to a specified age at risk. The 3 intervals were first the 5$^{th}$ to the 9$^{th}$ year (5–10), the 10$^{th}$ to the 14$^{th}$ year (10–15), and 15 and more years. Correspondingly, the equation was modi-

fied to contain weighting factors for the different exposure periods:

$$\delta(a) = \delta_0(a)[1 + \beta(W_1 + \theta_2 W_2 + \theta_3 W_3)]$$

$\beta$: estimated parameter set arbitrarily to give $\theta_1 = 1$

$W_1, W_2, W_3$: dose in WLM received in three time periods prior to a specified age at risk.

$\theta_2, \theta_3$: weighting factors; effect of exposures relative to those in the time-interval window 5–10 years ago.

Analyses of the separate cohorts with this model showed consistent effects of time since exposure. It was further determined by the committee that effects of age at risk on the relative risk coefficient had to be taken into account. In addition, more precise estimations of parameters were possible by pooling the data from the different cohorts. These last steps in the formulation of the equation yielded:

$$\delta(a) = \delta_0(a)[1 + \beta\gamma(a)(W_1 + \theta_2 W_2 + \theta_3 W_3)]$$

$\beta$: estimated parameter set arbitrarily to give $\theta_1 = 1$ for each cohort

$\gamma(a)$: effect of age at risk

The four analyses of different cohorts showed quite consistent results with a decline in excess relative risk with both age at risk evaluation and time since exposure. The differences between the cohorts, albeit quite large, were as close to one another as could be expected from statistical variation, even if all the exposures had been estimated perfectly or with the same bias. No consistent effects from age at first exposure or rate of exposure on the excess relative risk in the combined data from the four cohorts were found. Since the risk due to the exposures in the first two intervals (5–10 and 10–15 years) showed no significant differences, the two time periods were pooled. To account for the decrease of relative risk dependent of age at risk, three age categories of less than 55, 55–64, and 65 years of age or more were formed. The BEIR IV committee finally arrived at the following numerical values for the general equation:

$$\delta(a) = \delta_0(a)[1 + 0.025\gamma(a)(W_1 + 0.5 W_2)]$$

$\delta_0(a)$: age-specific background lung-cancer mortality rate

$\gamma(a)$: 1.2 for age a less than 55 years
1.0 for age a between 55–64 years
0.4 for age a being 65 years or more

$W_1$: dose in WLM incurred between 5 and 15 years before risk evaluation

$W_2$: dose in WLM incurred 15 years or more before risk evaluation.

The decreasing values of $\gamma(a)$ with age are a reflection of the increase of real risk with age, as seen in the epidemiological studies, which is clearly less than predicted from the relative risk model with constant risk coefficient. The risk for the miner cohorts studied has both an absolute and a relative component. Whereas in the BEIR III report [15], an absolute model was fitted to the data by introducing absolute risk coefficients increasing with age, the new approach reduces the relative risk coefficients with age. The abrupt changes of risk at the boundaries of the age intervals are artefacts of the simple step function. The actual pattern is thought to be gradual [10].
Applying the BEIR IV model to the US population living in single-family homes, Lubin and Boice [16] estimate that about 14% of lung cancer deaths among such residents (about 13300 deaths per year) may be due to indoor radon exposure.

**Risk in terms of loss of life expectancy**

For a risk which is highly skewed over the average life-span, the loss of life expectancy is a much better index of harm than the pooling of deaths occurring at different stages of life. The influence of acute or chronic exposure to ionizing radiation on survival probability can be calculated for both models based on exposures, risk coefficients and age-dependent lung cancer frequencies for unexposed populations [17] when using a relative risk model. If $L_0$ is the life expectancy at birth without any radiation exposure to the lung, the loss of life expectancy ($\Delta_{Lr}$) is given by:

$$\Delta_{Lr} = L_0 - L$$

L: predicted life expectancy for exposed individual

ICRP Report 50 uses mean relative excess lung cancer risk coefficients for $^{222}$radon (EEC) of $3 \times 10^{-8}$ and $1 \times 10^{-8}$ per $Bq \cdot h \cdot m^{-3}$ for exposures below and above the age of 20 years, respectively. These values have been used to derive the following relationship for a reference population exposed to $^{222}$radon and its progeny in the indoor environment:

$$\Delta_{Lr} \approx 1.4 \times 10^{-4} \times E_a \quad \text{(days)}$$

$E_a$: annual indoor exposure in $Bq \cdot h \cdot m^{-3}$ (activity as EEC)

The mean life expectancy in the absence of any exposure to radiation ($L_0$) in the reference population is taken as 72.5 years and the exposure is assumed to be constant over the whole life-span. For a typical average $^{222}$radon indoor concentration of 15 $Bq \cdot m^{-3}$ (EEC) over a lifetime, the resulting annual exposure of about $1.1 \times 10^5$ $Bq \cdot h \cdot m^{-3}$ would lead to a cumulative dose of 70 mSv $H_e$ and to an attributable loss of life

expectancy of about 16 days[3]. Taking the time-averaged indoor radon gas concentrations at the boundary of the upper 10th percentile and 1st percentile of the dwellings measured in Southeastern Switzerland of 650 and 1,560 Bq · $m^{-3}$, and assuming an equilibrium factor of 0.5[18], the theoretical attributable loss of life expectancy amounts to about 340 and 810 days, respectively. Even such a considerable risk will be difficult to detect, because few people will live their entire life in a dwelling in which these indoor radon levels prevail for decades.

## Comparison of the major risk projections

From 1984 to 1988, three committees published independent assessments of the risks from $^{222}$radon and its short-lived daughter products. The models put forward in these publications by the U.S. National Council on Radiation Protection and Measurements, NCRP, in the NCRP report 78[13], by ICRP in ICRP report 50[3], and by a committee of the U.S. National Research Council in the BEIR IV report[10] are quite different. Since they are based mainly on the same epidemiological base, and for the first two, on similar dosimetric and morphometric data of the lung, the different concepts of risks and numerical risk coefficients evolving are a vivid remainder of the many uncertainties involved in quantifying radiation hazards even at moderate to high doses. These uncertainties make the interpretation of essentially the same body of data highly dependent on the choices and philosophies of the expert groups involved. A comparison of the three models may help in understanding the general problems and expose the unsettled issues. The following discussion is based mainly on a comparative review of the models by Land[19].

The NCRP model uses an absolute risk concept. This means that the radiation-induced excess risk is independent of smoking. The dependence on age is restricted to the assumption that there is no excess risk before the age of 40. Excess risk starts 5 years after exposure and decreases subsequently as a negative exponential. The relative risk models of ICRP and BEIR IV both assume that the excess risk from radiation exposure is a multiple of the baseline rate and therefore depends strongly upon age, sex, and smoking history. In the ICRP model, the relative risk coefficient is dependent on age at exposure, i.e. 3 times higher below the age of 20. In the BEIR IV model, the coefficient is independent of age at exposure but dependent upon attained age. It decreases by 17 percent (from 1.2 to 1) at age 55 and another 60 percent (from 1 to 0.4) at age 65. Another difference is in the minimal latent period which is 5 years for BEIR IV (and NCRP 78) but 10 years for ICRP 50. In addition, the excess relative risk of BEIR IV remains constant for 15 years, and then drops to 50 percent for the time thereafter. Figures 5 and 6 adapted from Land[19] show the large differences in predicted risk per unit exposure between the three expert groups. Predictably, the range of model projections is relatively small for exposures at ages covered well in the cohorts studied, i.e. 20 to 50 years. For the prediction of risk from exposures during childhood and from smoking as a cofactor in $^{222}$radon induced lung cancer, the range of values becomes very large. The greatest range in Table 1 is found for smoking males exposed at age 15. The difference between the ICRP 50 estimate of 327 excess lung cancers lifetime risk in 100,000 smoking males exposed to 1 WLM at the age 15 versus only 7.2 lung cancer deaths according to the NCRP 78 model amounts to a factor of 45[19]. This disagreement for a scenario not covered by the available observations of radon exposed cohorts is a result of the higher relative risk coefficient for younger age groups (age at exposure less than 20 years) and of the synergistic (multiplicative) model for the influence of smoking used by ICRP. In the model of the NCRP 78 report, smoking has no effect on the absolute excess cancer risk from radon. The assumption that there is no excess risk before the age 40 even reduces the lifetime risk from early exposures (before age 35), because the early part of the expression period with the highest annual risk per unit dose no longer causes any lung cancer deaths with the NCRP assumption. The effect on lifetime risk is important since the excess risk function used in the NCRP model decreases exponentially with increasing time since exposure.

Tab. 1. Estimated lifetime excess lung cancer risk per 100000 persons exposed to 1 WLM (about 7 mSv $H_e$) in a single year, by model, smoking status (non-smokers vs. present smokers of 1 or more packs per day), exposure age, and sex (Land, 1989). Based on U.S. lifetables and smoking risks. Lifetables corrected for greater all-cause mortality among smokers.

| Exposure age | Sex | Model | | |
|---|---|---|---|---|
| | | NCRP 78 | ICRP 50 | BEIR IV |
| *Non-Smokers* | | | | |
| 15 | Female | 8.8 | 9.5 | 3.9 |
| | Male | 8.1 | 22.9 | 8.5 |
| 35 | F | 17.5 | 3.1 | 4.2 |
| | M | 16.2 | 7.5 | 9.0 |
| 55 | F | 11.9 | 1.9 | 3.4 |
| | M | 10.1 | 5.2 | 8.6 |
| *Smokers* | | | | |
| 15 | F | 8.3 | 164.8 | 69.9 |
| | M | 7.2 | 327.1 | 132.3 |
| 35 | F | 16.6 | 52.7 | 77.4 |
| | M | 14.4 | 106.6 | 143.5 |
| 55 | F | 10.4 | 30.5 | 60.1 |
| | M | 7.4 | 65.6 | 128.9 |

Tab. 2. Estimated absolute and relative risk coefficients for lung cancer mortality resulting from exposure to alpha-radiation from $^{222}$radon in the working environment (bold numbers denote original format).

| *Absolute risk model* | cases per $10^6$ person-years | | Lit. |
|---|---|---|---|
| | per WLM | per Sv $H_e$ [a] | |
| BEIR I | **6.5** | 760 | BEIR, 1972 |
| BEIR III | | | BEIR, 1980 |
| under 35 [b] | **0** | 0 | |
| 35–49 | **10** | 1,180 | |
| 50–65 | **20** | 2,350 | |
| 65+ | **50** | 5,880 | |
| ICRP 32 | **5–15** | 590 – 1,760 | ICRP, 1981 |
| ICRP 50 | **10** | 1,180 | ICRP, 1987 |
| NCRP 78 | **10** | 1,180 | NCRP, 1984 |

| *Relative risk model* | relative excess risk coefficient | | |
|---|---|---|---|
| | per WLM | per Sv $H_e$ | |
| ICRP 50 | 0.01 | 1.2 | ICRP, 1987 |
| BEIR IV | (0.0134) [c] | (1.6) [c] | BEIR, 1988 |

[a] Occupational conversion factor of 8.5 and 3 mSv per WLM exposure to $^{222}$radon and $^{220}$radon, respectively (Nuclear Energy Agency, 1983).
[b] Age at diagnosis of cancer (years).
[c] Average of all cohorts, modifying effects of variables other than cumulative exposure not taken into account, constant-relative-risk model.

Table 2 gives a comparison of risk coefficients for occupational exposures and adults.
Most disturbing are differences in risk projections from exposures typical of the experience of uranium miners, since data from miner cohorts form the base of all model validations. Land[19] finds a 10-fold difference between the BEIR IV and NCRP 78 model predictions for smoking males exposed at the age of 35 (Table 1). Such differences between the committees can only be resolved by adopting similar stands on the question of synergism with smoking, on the dependence of the risk on age at exposure, and on the decrease of risk with time since exposure. Additional follow-up may yield additional clues, but the decision of how much to rely on uranium miner data, and how much to rely on the information from the survivors of Hiroshima/Nagasaki, for the estimation of risk from low-level chronic exposure to $^{222}$radon in the indoor environment, is a matter of health physics policy. The adoption of only the conservative elements from both sets of data, i.e. synergism with smoking from the miner data, higher risk from exposure early in life, and a fixed non-decaying excess relative risk for the remaining life-span from the bomb survivors data, may overestimate the real risk. This approach is, however, in line with the philosophy of the ICRP and the health physics community in general.


## Summary

The considerable radiosensitivity of the human lung together with the highly localized α-doses in the bronchial and pulmonary regions from naturally occurring and man-enhanced radon decay products make the respiratory tract the most critical organ for cancer from exposure to ionizing radiation in our environment. From indoor radon, the tracheobronchial region of the lung generally receives radiation doses which are at least an order of magnitude above the total dose to any other organ. Excess lung cancer deaths found in epidemiological studies on heavily exposed populations of miners can be fitted reasonably well to a relative risk model, when declines in relative risk with both age at risk evaluation, and time since exposure, are incorporated. Smoking seems to act synergistically. A comparison of the major radon risk projections shows considerable discrepancies in the best estimates of risk, indicating that the uncertainties remain large.

## Résumé

### Cancer du poumon et rayonnement alpha

La radiosensibilité considérable du poumon combinée aux doses α très localisées dans les régions bronchiques et pulmonaires, provenant des dérivés du Radon engendrés de manière naturelle et par l'homme, rendent le système respiratoire l'organe le plus critique pour les cancers dûs au rayonnement ionisant de l'environement. Les doses de radiation provenant du Radon des bâtiments et reçues par la partie trachéobronchique du poumon, correspondent à un ordre de grandeur supérieur à la dose totale de tout autre organe. L'excès de cancers pulmonaires, ressortant d'études épidémiologiques sur des populations de mineurs fortement exposées, correspond relativement bien au modèle de risque, dans la mesure où celui-ci tient compte du risque relatif dû à l'âge et au temps écoulé depuis l'exposition. Fumer semble avoir un effet synergique. Une comparaison des principales projections de risque liés au Radon montre une divergence considérable entre les différents modèles étudiés. Cela démontre que l'incertitude dans l'interprétation des résultats reste grande.

## Zusammenfassung

### Lungenkrebs und Alpha-Strahlen

Die recht hohe Strahlenempfindlichkeit der Lunge zusammen mit den sehr lokalen α-Dosen im bronchialen und pulmonären Bereich durch natürlich und zivilisatorisch bedingte Radon-Zerfallsprodukte machen das Atemorgan zum kritischsten

Organ für Krebsinduktion durch ionisierende Umweltstrahlung. Die Strahlendosen im tracheobronchialen Bereich der Lunge, resultierend vom Radon in der Innenluft von Gebäuden, sind mindestens eine Grössenordnung höher als die Gesamtdosen anderer Organe. Die zusätzlichen Lungenkrebstoten, die in epidemiologischen Studien über stark exponierte Bergwerkarbeiter gefunden wurden, werden durch ein relatives Risikomodell recht gut vorausgesagt, sofern ein Abfall des relativen Risikos sowohl mit Alter bei der Risikoevaluation als auch mit der Zeitspanne seit Exposition angenommen wird. Rauchen scheint synergistisch zu wirken. Ein Vergleich der wichtigsten Radon-Risikoprojektionen ergibt bedeutende Diskrepanzen zwischen den verschiedenen Modellaussagen. Dies zeigt auf, dass die Projektionen weiterhin grosse Unsicherheiten aufweisen.

**References**


1 *Nazaroff WW, Nero AV* (eds.). Radon and its decay products in indoor air. Wiley and Sons, New York (1988).
2 *Hornung RW, Meinhardt TJ.* Quantitative risk assessment of lung cancer in U.S. uranium miners. Health Phys 1987; *52*: 417–430.
3 International Commission on Radiological Protection. Lung cancer risk from indoor exposures to radon daughters. Publication 50. Oxford: Pergamon Press, 1987.
4 *Burkart W.* Assessment of radiation dose and effects from radon and its progeny in energy-efficient homes. Nuclear Techn. 1983; *60*: 114–123.
5 *Preston DL, Kato H, Kopecky KJ, Fujita S.* Life span study report 10, Part 1. Cancer mortality among a-bomb survivors in Hiroshima and Nagasaki, 1950–82. Radiation Effects Research Foundation, Hiroshima, 1986.
6 *Kopecky KJ, Nakashima E, Yamamoto T, Kato H.* Lung cancer, radiation and smoking among a-bomb survivors of Hiroshima and Nagasaki. RERF Report TR 13.86, Hiroshima, 1986.
7 *Shimizu Y, Kato H, Schull WJ.* Studies of the mortality of a-bomb survivors. 9. Mortality, 1950–1985: Part 2. Cancer mortality based on the recently revised doses (DS86). Radiat. Res. 1990; *121*: 120–141.
8 *Radford EP, St Clair Renard KG.* Lung cancer in swedish miners exposed to low doses of radon daughters. New England J Med 1984; *310*: 1485–1994.
9 Nuclear Energy Agency of the OECD. Dosimetry aspects of exposure to radon and thoron daughter products. Paris NEA/OECD, 1983.
10 Committee on the biological effects of ionizing radiations. Health risks of radon and other internally deposited alpha-emitters, BEIR IV. Washington: National Academy Press, 1988.
11 *Wynder EL, Stellman SD.* Comparative epidemiology of tobacco-related cancer. Cancer Res. 1977; *37*: 4608–4622.
12 *Wynder EL, Goodman MT.* Smoking and lung cancer: some unresolved issues. Epidemiol Rev 1983; *5*: 177–207.
13 National council on radiation protection and measurements. Evaluation of occupational and environmental exposures to radon and radon daughters in the United States. Washington, NCRP-Report No. 78, 1984.
14 International commission on radiological protection, Principles of monitoring for the radiation protection of the population. Publication 42. Oxford: Pergamon Press, 1985.
15 Committee on the biological effects of ionizing radiations. The effects on populations of exposure to low levels of ionizing radiation, BEIR III. Washington, National Academy Press, 1980.
16 *Lubin JH and Boice JD.* Estimating Rn-induced lung cancer in the United States. Health Phys. 1989; *57*: 417–427.
17 *Whittemore AS and McMillan A.* Lung cancer mortality among U.S. uranium miners: a reappraisal. J. Nat. Cancer Inst. 1983; *71*: 489–499.
18 *Burkart W.* Radiation Biology of the Lung. The science of the total environment 1989; *89*: 1–230.
19 *Land CE.* The ICRP 50 Model. In: Radon, Proceedings of the 23rd annual meeting of the national council on radiation protection and measurements. Bethesda, 1989.



**Address for correspondence:**
Prof. Werner Burkart
Institut für Strahlenhygiene/BfS
Ingolstädter Landstrasse 1
D-8042 Neuherberg/Germany