Health Physics Vol. 55, No. 6 (December), pp. 845–853, 1988
Printed in the U.S.A.

0017–9078/88 $3.00 + .00

● *Special Submission*

ENVIRONMENTAL RADIOACTIVITY AND MAN— THE 1988 SIEVERT LECTURE*

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INTRODUCTION

Rolf M.Sievert
1896-1966

ROLF SIEVERT was born in 1896, the year in which Henri Becquerel discovered the phenomenon of radioactivity. This can be regarded as one motive to devote this Sievert Lecture to some new and actual aspects of environmental radioactivity and its impact on man. Rolf Sievert himself was strongly interested in this field. Under his leadership, important studies on the sources and levels of natural radiation and radioactivity in our indoor and outdoor environment have been carried out in the Department of Radiophysics of the famous Karolinska Institute in Stockholm. As an example, I want to mention the doctoral thesis of his co-worker Bengt Hultqvist (1956). This study revealed for the first time the wide variation range of ^{222}Rn levels in the indoor air of Swedish houses. At this time Sievert and Hultqvist believed that the high Rn levels observed in some houses would be a specific Swedish problem, restricted to houses built of concrete containing alum-shale with a high Ra content. These pioneer studies have been successfully continued under the leadership of Bo Lindell and now of Gunnar Bengtsson at the National Institute of Radiation Protection in Stockholm which had its origin in Rolf Sievert's department.

Today we know that Rn in houses is a world-wide problem. Taking into account the improved models on lung dosimetry, it has become evident that inhalation of the short-lived decay products of ^{222}Rn in indoor air yields by far the highest contribution to the total population exposure from natural and man-made radiation sources

* Presented at the opening ceremony of the Seventh International Congress of the International Radiation Protection Association; Sydney, Australia; 10–17 April 1988.

(UNSCEAR 1982; NCRP 1987). The control and mitigation of Rn in existing houses and the implications on building codes for new houses represent one of the most important challenges to the radiation protection community and the responsible authorities. The solution to this Rn problem in houses is strongly linked with that of the risk of lung cancer which might be associated with this indoor exposure to Rn and its decay products. The discussion of this question will be the subject of one part of this Sievert Lecture.

The other part of this lecture is concerned with a problem in the outdoor environment—the transfer of radionuclides through food chains to man. The actuality of this field is well known; it is related with the reactor accident at Chernobyl which happened two years ago. In this part of my lecture I want to describe some results of our work at the GSF-Institute for Radiation Protection on the development of dynamic radioecological models and their validation by measurements after the Chernobyl accident.

ACTIVITY TRANSFER THROUGH FOOD CHAINS

Radioecological modelling

The transfer of radionuclides through food chains to man is, besides the dose from external irradiation, the most relevant exposure pathway in the case of a contamination of the outdoor environment. For chronic operational releases of activity from nuclear facilities, rather simple transfer models are used which consider only the reached steady-state equilibrium conditions. Many of these simplifying models are designed to lead to an overestimation of the actual exposure.

In the case of high accidental releases, more realistic radioecological models are required to enable appropriate decisions on eventually necessary countermeasures. In particular, such models should provide with sufficient accuracy a prediction of the expected activity concentration in relevant foodstuffs as function of time and of the cumulative potential radiation exposure via these pathways. Annual mean values of the overall-transfer coefficients of ^{90}Sr, ^{131}I and ^{137}Cs in major groups of foodstuffs have been estimated by a regression analysis of data from nuclear weapon tests. These results are summarized in the reports of UNSCEAR (1977, 1982). These values refer, however, to a long-term and a rather uniform spatial deposition of these radionuclides. They cannot be applied to the predictive evaluation of the dietary activity intake and the corresponding ingestion dose to the local and regional population in the case of short-term, accidental releases. This has been confirmed by the observations after the Chernobyl accident.

Already, in 1978, our laboratory started developing realistic dynamic models for predictive evaluation of spatial and temporal distribution of external and internal radiation exposure which might occur as a consequence of accidents at nuclear facilities. Main emphasis was laid to the simulation of the transfer kinetics through food chains, taking into account typical agricultural conditions and their seasonal variation in the Federal Republic of Germany. First results of this so-called ECOSYS model have been published in a comprehensive report in 1982 (Matthies et al. 1982). An improved version of this model was presented three years later (Müller et al. 1985).

Results of the ECOSYS model

In the following, some selected results of this dynamic model are outlined and discussed. For some important radionuclides Fig. 1 shows the effective dose equivalent commitment from the intake with the total diet, integrated over an intake period of 50 y, as a function of the calendar month in which the deposition occurs. The data in this figure refer to the first, simplified version of the ECOSYS model and take into account the average food consumption rates of adults in the Federal Republic of Germany (Matthies et al. 1982). They are normalized to a deposition density of 1 kBq m^{-2} and an interception factor of one on plant surfaces.

The strong seasonal variation of the dose commitment per unit deposition density is due to the direct deposition on plant surfaces and the subsequent uptake through the cuticula of plants during the vegetation period. This variation is, therefore, more pronounced for those

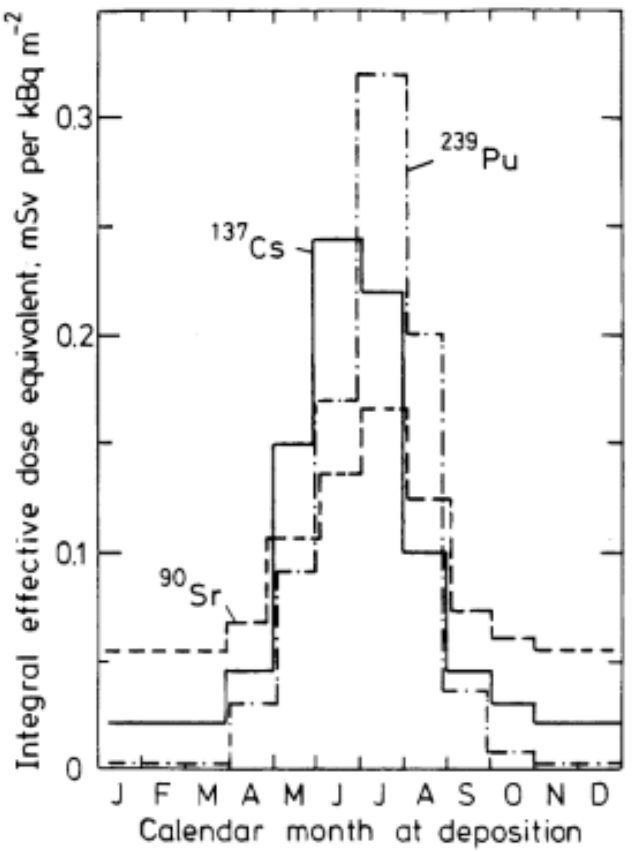

Fig. 1. Calculated ingestion dose commitment as a function of the calendar month at deposition, normalized to a deposition density of 1 kBq m^{-2} and an interception factor of one (ECOSYS-model, 1982).

radionuclides, like ^{137}Cs, for which the root uptake from normal soils is rather low. The dose contribution by uptake from soil is, under the vegetational conditions in Germany, the dominant source term for depositions during the winter months (see Fig. 1).

Another sensitive parameter is the interception factor of plants, which depends on the type and growth status of plants and on the type of deposition (wet or dry deposition). Figure 2 shows the cumulative ingestion dose as function of time after deposition for different values of the interception factor, as it results from an improved version of the ECOSYS model for a deposition of 1 kBq m^{-2} of ^{137}Cs in the first days of May (Müller et al. 1985, in press). For wet deposition by rainfall, the model proceeds from an interception factor of about 0.1–0.2. Higher values in the range of 0.5–1 may be appropriate in the case of dry deposition.

The increase of the ingestion dose with time after deposition (Fig. 2) reflects again the strong influence of direct foliar uptake. For depositions during the vegetation period, the contribution from this source determines the ingestion dose received during the first two years. The contamination of foodstuffs due to the uptake from soil is a long-term process, the kinetics of which are mainly determined by the migration and sorption processes of the deposited radionuclides in the soil. The dose contribution from this pathway is indicated by the dotted line.

Due to the direct deposition and foliar uptake by plants, the specific activity of milk, meat, leafy vegetables and cereal products varies strongly with time after deposition. For example, Fig. 3 shows the variation of the mean specific activity in major groups of foodstuffs, as it follows from the improved version of the ECOSYS model for the deposition of ^{137}Cs in early May, normalized to a deposition density of 1 kBq m^{-2} and a mean interception factor of 0.2 in the area where the foodstuffs are produced.

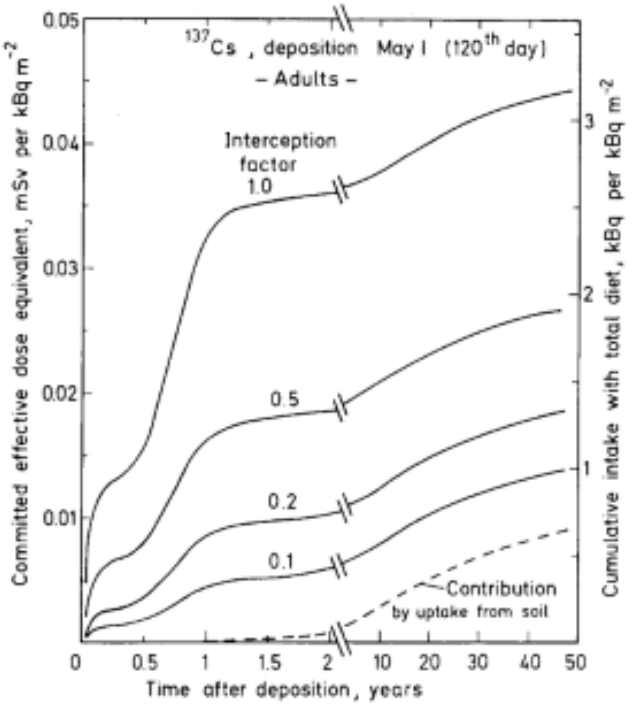

Fig. 2. Cumulative dietary intake of ^{137}Cs and dose to adults as a function of time, predicted with the ECOSYS-model for a uniform deposition of 1 kBq m^{-2} on 1 May.

Under these conditions, the model yields a peak value of the specific activity in milk and beef at about 5 or 30 d after deposition, respectively. For the rapid decrease afterwards, the growth of biomass is an important factor. At the beginning of the winter period, about six months after deposition, the specific activity in beef and milk increases again due to the feeding of contaminated hay and silage which were harvested during the summer period. After this period of dry feeding the contamination decreases and, after about two years, approaches the contamination level, which is caused by the Cs uptake from the soil.

Comparison with measurements after Chernobyl

An improved version of the ECOSYS model, which also involved a probabilistic dose assessment, was published a few months before the Chernobyl accident (Müller et al. 1985). This accident caused a rather high activity deposition in southern Bavaria in the days between 30 April and 2 May 1986; the main deposition occurred during a heavy rainfall with thunderstorm in a period of 1 h in the afternoon of 30 April. At our laboratory in the northern area of Munich, we measured a total deposition density of about 400 kBq m^{-2} from which about 100 and 20 kBq m^{-2} were allotted to ^{131}I and ^{137}Cs, respectively (ISS 1986; Hötzl et al. 1987). It is interesting to note that the latter value was comparable with the Cs deposition reported for the region of Kiew. This experience shows that, under adverse weather conditions, the activity release from major reactor accidents may lead to rather high depositions far away from the emergency site.

Since the Chernobyl accident, we have made more than ten thousand measurements in different environmental media and foodstuffs, as well as whole-body counting measurements. These data offered the possibility to validate the predictions of the ECOSYS model and to improve the model. In addition, in cooperation with other institutes, we have carried out specific feeding experiments with cows and pigs to study the transfer into milk and meat, and have started laboratory experiments to investigate the kinetics and mechanisms of the foliar uptake of radiocesium into grass and leaves.

In general, with a few exceptions, the observed mean values of the specific activity in relevant foodstuffs as a function of time after deposition agreed rather well with the predictions of the ECOSYS model (Müller et al. in press). This was particularly valid for milk. As an example, Fig. 4 shows a comparison between the measured daily mean values of ^{137}Cs activity in milk from a dairy farm near Munich during the period from May 1986 to August 1987. The closed line in this figure indicates the variation with time which was calculated with the ECOSYS model, inserting a deposition density of 20 kBq m^{-2} and an in-

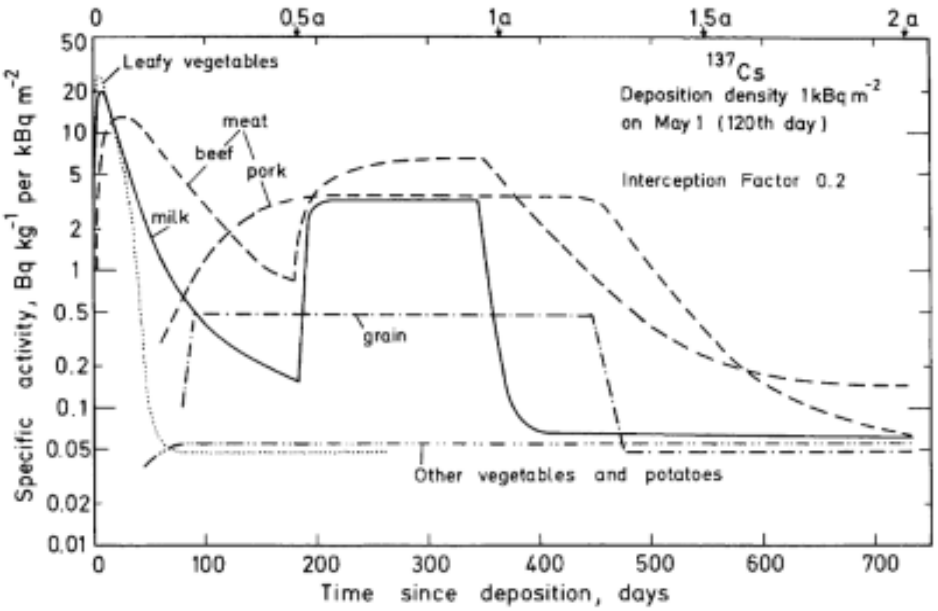

Fig. 3. Mean specific ^{137}Cs activity in foodstuffs as a function of time after deposition, expected from the ECOSYS-model for a uniform deposition of 1 kBq m^{-2} on 1 May (interception factor = 0.2).

terception factor of 0.2 for grass. As mentioned before, the second broad peak during the winter period (200th–400th day) is due to the feeding of contaminated hay, which was cut mainly in June 1986. Also for other major foodstuffs the measured and calculated mean values of the time integral over the specific activity, integrated over the first year after deposition, agree in most cases within a factor of two.

The final objective of such models should be the assessment of the cumulative activity intake with the total diet and the corresponding dose. Since the Chernobyl accident, many whole-body-counter measurements of persons living in the region of Munich have been carried out. The cumulative frequency distribution of the total intake of radiocesium (^{137}Cs + ^{134}Cs) during the first year, derived from these measurements, is shown in Fig. 5. The most reliable distribution function is represented by the curve for 21 adults who were measured at least once each month. In the same figure, the distribution functions are plotted from the probabilistic version of the ECOSYS model for an interception factor of 0.1 and 0.2, respectively (Müller et al. in press).

This comparison indicates that in the given situation, the ECOSYS model for radiocesium leads to an overes-

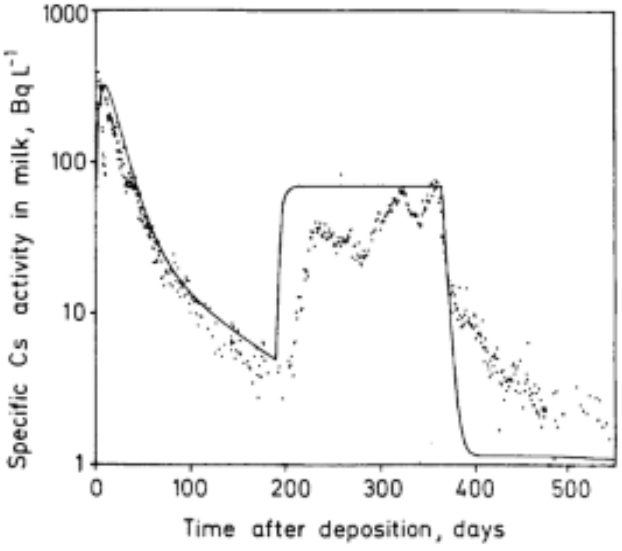

Fig. 4. Measured daily mean values of the ^{137}Cs in milk from a dairy farm near Munich after the Chernobyl accident (points); comparison with the time dependency predicted with the revised ECOSYS-model (closed line).

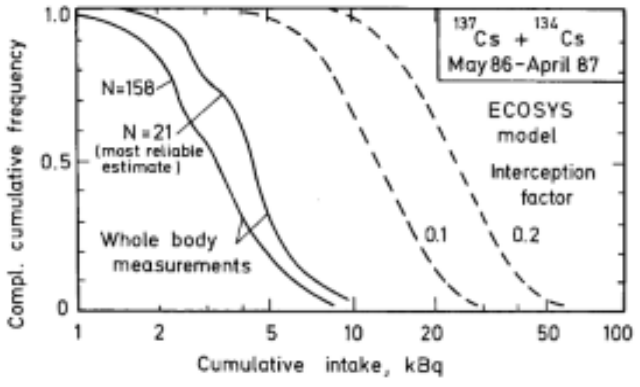

Fig. 5. Complementary cumulative frequency distribution of the radiocesium intake by adults during the first year after the Chernobyl accident; comparison of results from whole body measurements of persons in the region of Munich (closed lines) with the predictions of the probabilistic ECOSYS-model (plotted lines).

timation of the cumulative intake or dose by a factor of about three or five, respectively, depending on the inserted interception factor. This deviation can be explained by several reasons:

(1) the model assumes that all consumed foodstuffs are also produced in the same region of high contamination;

(2) normal food consumption rates have been assumed, whereas the real consumption rates of milk, fresh vegetables and partly also of meat were somewhat lower, at least until early summer 1986; and

(3) normal feeding practices have been assumed.

Probably, the main part of the observed deviation may be attributed to the first cause, because a considerable fraction of the foodstuffs consumed in the rather highly contaminated area of southern Bavaria will not be produced in this region. This influence of the spatial distribution of major foodstuffs is confirmed by the whole-body measurements of persons in other parts of Germany, where the deposition density after the Chernobyl accident was considerably lower, in average by a factor of about five.

In conclusion, dynamic radioecological models can be a very important and useful tool for emergency management at early and later times after nuclear accidents, in particular for an appropriate setting of intervention levels for major groups of foodstuffs (Jacobi et al. 1987). Our future work on this field is directed to the improvement and extension of the ECOSYS model, taking into account all experience obtained after the Chernobyl accident. This also involves the dose assessment from external radiation in urban and rural environments. Based on the ECOSYS model, we intend to develop, in cooperation with the Federal Health Office, a real time expert system. It will be part of the central monitoring and warning system in the Federal Republic of Germany which is provided by the German government for the future management of emergency situations.

LUNG CANCER RISK FROM INDOOR Rn

The second part of this Sievert Lecture is devoted to an entirely different field of environmental radioactivity. It deals with the population exposure to ^{222}Rn and its short-lived decay products, giving main emphasis to the assessment of the attributable risk of lung cancer.

General aspects and problems

Since the first studies of Rolf Sievert and his coworkers, comprehensive surveys on Rn in houses have been carried out or started in several countries. One of the most striking results is the observed large variation range of the Rn level from house to house. It covers a range from a few up to several thousand Bq m^{-3}, and in a few extreme cases values up to about 100,000 Bq m^{-3} have been measured. In most houses with strongly enhanced levels the main source term is the Rn supply from the underlying soil. This Rn influx from the soil is a very complex process. Under these circumstances a realistic quantitative prediction of the Rn level in existing houses seems rather impossible. Consequently, measurements are finally the only reliable method for the detection of houses with high Rn levels. A flood of measured values have been reported in the last years. But these data alone do not solve the Rn problem. Important is the fact that during the last five years efficient and low-cost techniques for the reduction of the Rn influx from soil, like sub-pressurization, have been developed and tested.

Under these aspects, the question arises: What is a reasonable lower boundary below which no actions should be taken? Or, with other words: Where is the borderline between normal natural radioactivity and artificial modifications of this natural source of exposure? One should also have in mind that this is not a new source. Our ancestors were exposed to Rn levels which were comparable with the values measured now. But they did not worry about it, because in Germany the saying goes: "Was ich nicht weiß, macht mich nicht heiß," which corresponds to the English saying, "Ignorance is bliss." But today we are aware of the problem, and the guiding principle of precaution to which we are obliged in radiation protection makes it necessary to follow the ALARA principle, or the principle of optimization, respectively.

In Publication 39, the ICRP has made a first attempt to outline the basic principles for the limitation of exposures to natural sources, but there is also stated that for several reasons "it would not be helpful to suggest a generally applicable value of an action level" for Rn in houses (ICRP 1984). It is obvious that a simple cost-benefit analysis alone cannot give the final answer to the setting of such action levels. Guidelines of national authorities must be in accordance with the demands of public health, having in mind the collective detriment to health from the controllable fraction of this exposure. On the other hand, they should warrant a margin for the individual freedom and voluntariness of decision-making. This underlines the necessity to inform and advise the general public in a clear and objective way.

It seems reasonable to apply a step-wise system of action levels for the concentration of Rn or its decay products in indoor air. The higher the observed concentration, the shorter should be the time period for remedial actions, if they are possible. The deciding quantity is the cumulative exposure. Thus action levels c_{AL} of the concentration should be derived from a primary action level E_{AL} of the cumulative exposure, applying the relationship

$$c_{AL} \times T_A = E_{AL},$$

where T_A is the time period in which the action should be taken. The possible lung cancer risk associated with this indoor exposure is an important criterion for the final judgment of the Rn problem in houses and the appropriateness of mitigation measures. The epidemiological studies of various groups of Rn-exposed miners, as well as the findings from the externally irradiated atomic bomb

survivors, indicate that the human lung, and particularly the bronchial epithelium, is a rather sensitive tissue for carcinogenic effects of ionizing radiation.

On the other hand, up to now no direct epidemiological studies on lung cancer from indoor exposure to Rn and its decay products are available which enable a quantitative risk assessment. Some smaller pilot studies in Sweden and Canada indicate a positive correlation. But their statistical and systematic error ranges are quite large, taking into account the overwhelming influence of smoking and the uncertainties of long-term exposure estimates. Case-control studies among non-smoking females seem to be more favourable, but one should have in mind that lung cancer in real non-smokers is a rather rare event.

Comparison of risk estimates

Under these circumstances, risk assessments based on data from Rn-exposed miners seem to be more reasonable, taking into account appropriate correction factors for the different conditions in mines and houses. During the last years, reports of three expert groups on this subject have been published in which different approaches have been considered (NCRP 1984; ICRP 1987; NAS-BEIR IV 1988). The central estimates of the age-averaged mortality risk of lung cancer per unit of indoor exposure to ^{222}Rn-decay products resulting from these studies are summarized in Table 1. The exposure is expressed in terms of the equilibrium-equivalent exposure to ^{222}Rn (unit: 1 Bq y m^{-3} = 8760 Bq h m^{-3}); risk values per unit of the potential α energy exposure to ^{222}Rn-decay products are given in brackets (unit: 1 WLM = 0.0035 J h m^{-3} $\triangleq$ 72 Bq y m^{-3}).

In all these studies, a proportional exposure-risk relationship has been assumed which yields the best fit to the miner's data in the relevant exposure range. In a subgroup of U miners in Czechoslovakia (CSSR), a statistically significant excess frequency of lung cancer has been already observed at an exposure level of about 0.14 J h m^{-3} (40 WLM). This "statistical threshold" lies only a factor two to five higher than the mean lifetime exposure of the populations in most countries to be expected from the presently measured mean concentrations in indoor air.

More controversial are the conceptions for the risk projection as a function of time after exposure. In the NCRP study an absolute risk projection model has been assumed. The data on lung cancer in Rn-exposed miners as well as in the A-bomb survivors indicate, however, that the appearance rate of radiogenic lung cancer is similar to the age-dependent distribution of the normal cancer rate in a comparable non-exposed population. On the basis of this finding, in the two more recent studies (ICRP 1987; NAS 1988) preference is given to a relative risk projection model. In the ICRP report, a constant relative excess risk was inserted, whereas in the NAS (BEIR IV) report, a slight decrease with time after exposure was inserted. The ICRP Task Group also made a comparison with the results of absolute risk projection models (Table 1). This includes, as well, the so-called "dosimetric approach" which proceeds from the age-averaged reference risk coefficients of 1×10^{-3} Sv^{-1}, recommended by the ICRP (1981) for each of the two target tissues in the lung: the tracheobronchial epithelium and the pulmonary tissue.

The estimates from different types of approaches differ only by a factor of three to four. This range is probably smaller than the total uncertainty of the input data involved in these models. The relative risk projection models primarily yield values for the relative excess lifetime risk. Due to this proportional hazard model, at equal exposure

Table 1. Age-average risk of lung cancer per unit of indoor exposure to ^{222}Rn decay products; comparison of central estimates from different studies and types of approaches.

Time projection model	Study, Population		Attributable cases per 10^6 persons per Bq y m^{-3} (per WLM) [a]	
Relative risk projection	ICRP (1987) Refer.population	males	5.3 (380)	average 3.2 (230)
		females	1.1 (80)	
	NAS/BEIR IV (1988) US-population	males	7.0 (500)	average 4.9 (350)
		females	2.7 (200)	
Absolute risk projection	NCRP (1984)	average, both sexes		1.8 (130)
	ICRP (1987)			2.1 (150)
	"Dosimetric approach" (ICRP 1987)			1.4 (100)

a) The unit Bq y m^{-3} refers to the equilibrium-equivalent exposure to ^{222}Rn; 1 WLM $\triangleq$ 72 Bq y m^{-3}.

conditions the relative risk is nearly equal for males and females. It is interesting to note that the relative risk coefficients resulting from the ICRP and the BEIR IV approaches are nearly equal; the difference is less than 20 percent. Consequently, the absolute number of excess cases increases proportionally with the normal lung cancer frequency in populations and will be higher for males than for females (Table 1).

The main reason for the different absolute numbers averaged over both sexes which result from the ICRP and NAS studies (Table 1). The ICRP estimate refers to a reference population with a life expectancy at birth of 70 y (males) and 75 y (females), and a normal lung cancer frequency of 600 and 120 cases (average both sexes: 360 cases) per 10^6 persons per year for the male and female population, respectively. These values correspond roughly with the worldwide average frequency, which results from the reported values in populations with high life expectancy. The NAS estimate refers to the United States' population only, for which, in recent years, an annual lung cancer frequency of about 450 cases per 10^6 persons averaged over both sexes has been reported.

The important question arises: Which fraction of the observed lung cancer frequency might be associated with the indoor exposure to Rn decay products? In the UNSCEAR report (1982) a country-averaged mean value of about 15 Bq m^{-3} for the equilibrium-equivalent ^{222}Rn concentration in indoor air and an occupancy factor of 0.8 is assumed. This would yield a mean indoor exposure of 12 Bq y m^{-3} per year. Applying the risk coefficients for the reference populations resulting from the ICRP relative risk approach (Table 1), a chronic exposure at this level would yield an attributable annual frequency of about 64 and 13 cases (average 38 cases) per 10^6 males and females, respectively. This means that nearly 10% of the totally observed lung cancer frequency among males and females might be associated with this indoor exposure. The attributed relative loss of life expectancy would be about 0.06%, averaged over both sexes, corresponding to an absolute value of about 20 d (ICRP 1987). On the basis of absolute risk projection models, these values are only about a factor of two lower.

This risk assessment indicates that if smoking is excluded, the indoor exposure to Rn decay products is probably the most important exogenic cause of lung cancer in most countries. It also underlines the necessity of mitigation measures in those houses where much higher concentrations, due to the Rn supply from soil, have been measured, taking into account that this source term is to a large extent controllable and can be efficiently reduced by rather low-cost techniques.

Risk models: problems and perspectives

The previous considerations may lead to the impression that a relative risk projection model enables a realistic assessment of the lifetime risk of lung cancer by ionizing radiation. This conclusion is premature. Some epidemiological data on lung cancer in American miners suggest a decrease of the relative risk with time after exposure

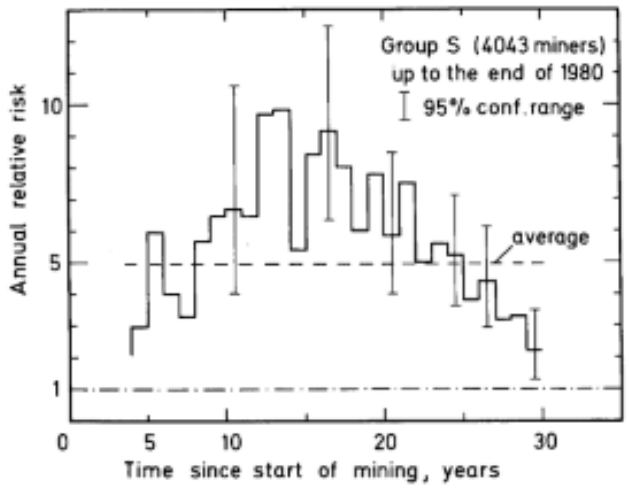

Fig. 6. Relative risk of lung cancer among Rn-exposed U miners (group S) in Czechoslovakia as function of time after start of mining (from data of Sevc et al. 1988).

(NAS 1988). A similar tendency can be deduced from the incidence of lung cancer in the A-bomb survivors (Yamamoto et al. 1987; Preston et al. 1987) if the data for all age groups at time of exposure are combined. However, in both cases, due to the large statistical error range, this decrease cannot be quantified. Recently the results of the extended follow-up of U miners in the CSSR have been published (Sevc et al. 1988). Figure 6 shows the time dependency of the relative lung cancer risk which can be evaluated from these data. A maximum value in the range of 5–10 is reached 10–20 y after start of mining. Afterwards the relative risk decreases strongly, and at the end of the follow-up period, 30 y after start of exposure, a value of only about 2 has been found. The decrease is highly significant as can be seen from the confidence limits in this figure.

These epidemiological findings strengthen the doubts in the appropriateness of relative risk models which assume a constant relative risk with time after exposure. Relative risk models also do not lead to a conclusive description of the interaction between inhaled Rn daughters and cigarette smoke for the induction of lung cancer (ICRP 1987; NAS 1988). Thus, neither absolute nor relative risk projection models may be appropriate for radiation risk assessments in every instance. This conclusion is also valid for other types of radiation-induced cancers (NAS 1980; Upton 1984).

The future improvement of risk models should take into account that carcinogenesis is a multifactorial multistage process. There is strong evidence that ionizing radiation, particularly at low doses, acts primarily on earlier stages of this process. On the other hand, the effects of aging and the influence of other promoting factors affect the later stages. Thus the latency distribution of radiogenic lung cancer is mainly determined by aging and other factors, like smoking, which stimulate the tumor growth. Consequently, a risk model considering the multiplicative

interaction of initiating and promoting effects seems to be more reasonable.

In a simplified way, a multiplicative two-stage model leads to the following general relationship for the age-specific incidence rate $\lambda(t)$ of lung cancer as function of age t:

$$\lambda(t) = P(t) \times \mu(t).$$

In this equation, $P(t)$ characterizes the latent hazard potential of potentially malignant cells which is built up by all initiating agents acting in the early phases of the carcinogenic process. The function $\mu(t)$ defines the probability for the growth and manifestation of tumors per unit time. This function increases strongly with age and will be amplified or shifted to lower ages by smoking.

If ionizing radiation is mainly regarded as an initiating agent, it leads to an increase of the latent hazard potential $P(t)$, but causes no significant change of the promotion probability $\mu(t)$. This is shown schematically in Fig. 7 for the example of a radiation exposure or dose D received at an age $t_e = 30$ y; for simplicity, no decrease of the radiation-induced increment $P_r(t, D)$ due to the removal or inactivation of radiation-induced potentially malignant cells is assumed. The total hazard function follows to

$$\lambda(t, D) = [P_0(t) + P_r(t, D)]\mu(t),$$

for $t > t_e + \tau$ (τ = time lag between irradiation and manifestation) which leads to a relative risk function:

$$\frac{\lambda(t, D)}{\lambda_0(t)} = \frac{P_0(t) + P_r(t, D)}{P_0(t)} = 1 + \frac{P_r(t, D)}{P_0(t)}.$$

As the baseline potential P_0 caused by initiating agents other than radiation increases with age, the radiation-induced relative risk decreases with time after irradiation, as shown in the lower graph of Fig. 7. Furthermore, in accordance with epidemiological findings, the initial relative risk decreases with increasing age at exposure.

Summarizing, a multiplicative, two-stage model suggests a decrease of the relative lung cancer risk with time after irradiation. Consequently the assumption of a constant relative risk might lead to an overestimation of the real lifetime risk.

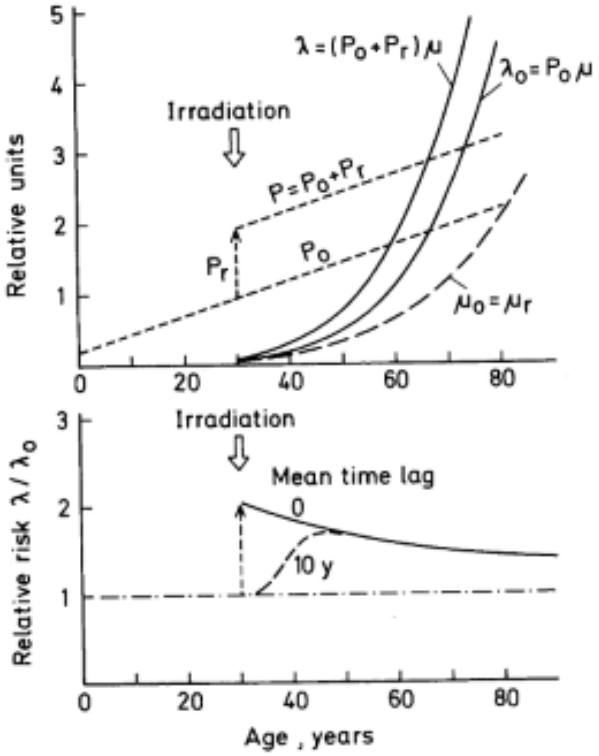

Fig. 7. Two-stage model for the age-specific lung cancer rate (schematically, explanation see text).

FINAL REMARKS

These considerations shall indicate some perspectives for the future improvement of risk models. Similar views might also be valid for other types of radiation-induced cancer. It is necessary to build a bridge between epidemiology and molecular tumor genetics. In my opinion, only the combination of the findings and experience from both these fields can lead to more realistic estimates of the cancer risk at low doses which are required for a well-founded system of dose limitation in radiation protection.

Such thoughts are in accordance with Rolf Sievert's ideas. His pioneer work has demonstrated the success of interdisciplinary cooperation and the strong linkage between research and practical radiation protection. The problems of environmental radioactivity outlined in this lecture confirm the necessity of this view. Like in the past, the concepts and experience of radiation protection can be and should be a guiding model for the protection against toxic non-radioactive chemicals in our environment.

Acknowledgments—I am greatly indebted to all scientific and technical co-workers in my institute who have contributed in a very efficient way to the work outlined in this lecture.

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