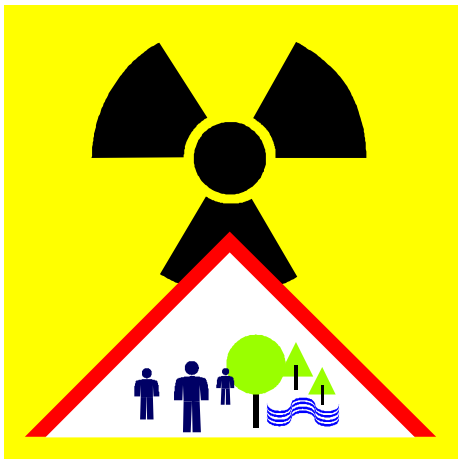
Radiation protection 98



Scientific Seminar on Radiation Protection in relation to Radon

European Commission

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1998

Directorate-General
Environment, Nuclear Safety
and Civil Protection

CONTENTS

FOREWORD

Title VII of Council directive 96/29/Euratom of 13 May 1996 laying down basic safety standards for the protection of the health of workers and the general public against the dangers arising from ionizing radiation includes Title VII, devoted to significant increase in exposure due to natural radiation sources. In many circumstances, exposure to radon and radon daughters is the major contributor to the dose from natural radiation sources incurred by workers and members of the public. Exposure to radon in dwellings is the subject of Commission Recommendation of 21 February 1990 on the protection of the public against indoor exposure to radon (90/143/Euratom).

Although it has been demonstrated that exposure to radon and radon daughters induces lung cancers in humans, especially in miners, a quantification of the excess cancer risk due to exposure to radon is still subject to uncertainties. Knowledge in the field is progressing and studies on dosimetry and modelization of the respiratory tract as well as epidemiological studies are carried out, especially under the fourth Euratom framework programme on research and training.

The European Commission, Directorate General XI, Environment, Nuclear Safety and Civil Protection, and Directorate General XII, Science, Research and Development jointly organised a scientific seminar on radiation protection in relation to radon, with a view to present in a compact form the latest developments in the knowledge of the effects of exposure to radon likely to have an impact on the regulatory texts ensuring protection of the workers and the population against the dangers of this ubiquitous source of ionizing radiation.

AN OVERVIEW OF RADON INDOORS

Gun Astri SWEDJEMARK

Older history (1470-1945)

About 1470 extensive mining of silver started in the region of Schneeberg in southern Germany. An unusually high mortality from lung disease, occurring in younger workers was observed, described by Paracelsus (Pa1567). In the 1900th century about 75% of the miners died from lung cancer (Ha79). After the identification of radon by Marie and Pierre Curie, the first measurements in the mines were made in 1901 (El01). After more precise measurements in the 1920s a correlation between radon exposure and lung cancer was suggested (Lu24). This hypothesis was confirmed in 1940 (Ra44).

One year after the discovery of radon, measurements (El01) revealed that radon (at that time called radium emanation) was a ubiquitous constituent of atmospheric air. Ernest Rutherford (Ru07) said in 1907: ”We must bear in mind that all of us are continuously inhaling radium and thorium emanations and their products, and ionising air. Some have considered that possibly the presence of radioactive matter and ionised air may play some part in physiological processes.”

It was seen as healthy to be exposed to radon and health spas were built in many countries. Some of them are still working. Many measurements of concentrations of radium and radon in ground water sources were carried out in these days.

In the following I will present measures taken in European and other countries and recommendations from international organisations in a historical review. However, a rather big portion will be focused on judgements and actions taken in Sweden. The reasons are that Sweden was the first country to introduce limits and recommendations for the general public and that I was involved in this work between 1971 and 1994.

1946-1969

Intensive mining of uranium began in the 1940s in Africa, Canada and USA. Little attention was paid to radiation protection of the workers. The first measurements in these mines were made in 1950. Bale (1951) introduced the idea that it was not only radon but the decay products which might be the causative agent. More reliable measurement methods were developed and revised guidelines for uranium mines were established by the US Federal Research Council (FRC67). The first quantitative analysis of uranium miners and lung cancer was published in 1971 (Lu71). High radon levels were also found in non-uranium mines.

Early environmental measurements of radon were largely confined to outdoor air. The first set of indoor radon measurements were published by Hultqvist (1956). This study, initiated by Rolf Sievert, resulted in high radon levels in some of the alum-shale concrete houses but also in a few dwellings built from other materials with very poor ventilation rates and in some basements.

Table 1 - The radon concentrations in air in four towns in central Sweden in the early 1950s, in houses built before 1946 (Hu56). Both single-family houses and apartments in multi-family houses are included.

Radon concentration[a], Bq m^{-3}				
Building materials	Unaired houses[b]	Aired houses[c]	Weighted average	Maximum
All dwellings	77	36	62	592 (1740[d])
Wood	15	15	15	63
Brick	47	26	40	303
Alum shale concrete	133	67	116	592 (1740[d])

a) 1 Bq m^{-3} = 0.027 pCi/liter
b) No thorough airing was carried out since the day before the sampling of air for measurement.
c) Dwellings were aired the morning before the sampling of air for measurement.
d) This value was not included in the original sample of dwellings. The ventilation rate was extremely poor.

The results showed that the radon content in the air caused higher radiation doses than the gamma radiation, which was also measured. Limits were discussed but not introduced because good air exchange rates were judged to be necessary for other hygienic reasons and that the ventilation rate was important for the concentration of radon. Little attention was paid internationally to these findings because it was believed to be a local Swedish problem.

During the 1950s a few studies on natural radiation indoors were carried out also in other countries. The gamma levels in Scottish houses were measured and the study showed the difference between houses built from granite and from sandstone (Sp57).

At the end of the 1960s, the Swedish Radiation Protection Institute, SSI, issued information about the natural radiation in buildings (Li68). During the 1960s radon indoors was studied in some countries but the extent of the research was limited, illustrated in the UNSCEAR reports of this time.

Drinking water was studied with respect to the natural radionuclides including radon in several countries e.g. Finland and Sweden. High values were found in some ground water wells. The dominating part of the radiation dose from radon in water is caused by inhalation of the radon gas that evaporates from the water.

1970s

At the beginning of the 1970s there was an increasing interest in the ionising radiation indoors. Restrictions on building materials were suggested in the Soviet Union, Great Britain etc. (Kr71, NRPB72). In USA requirements were established for both the gamma and the radon progeny exposures in houses built on or from waste materials from the uranium industry (US71).

In 1973 a task group was established by OECD/Nuclear Energy Agency (NEA) on exposure to radiation from natural radioactivity in building materials. This work resulted in a report which described the problems (NEA79). Measurements of radon and radon progeny concentrations indoors were carried out, in particular in houses built on or from waste materials but also in dwellings in general, e.g. in Hungarian buildings (Ge72). Bo Lindell said at that time: ”If you measure houses in your country you will find some houses with high radon levels. Even in a tent you will find levels higher than outdoors.”

In Sweden we tried to inform the other involved national boards about the health risks with radon exposure indoors, but with little success. We saw the radon concentration increase when residents saved energy by decreasing the ventilation rates. At that time the Swedish radiation protection act could not be used to specify recommendations or limits for natural radiation and no international organisation had recommended restrictions on natural radiation. The Swedish Radiation Protection Institute published a booklet for information of the public, but nobody cared. One journalist which we contacted said that it was not ethical to inform the Swedish people about such a problem just before Christmas. SSI informed the company that produced the alum shale concrete, and subsequently, in 1974, they stopped the production. The material had then been produced since 1929.

In 1977 the International Commission on Radiological Protection, ICRP, published new recommendations where exposure to natural radiation was not excluded as in the earlier recommendations. Two years later ICRP started a committee for natural radiation. The radiation protection boards in the Nordic countries set up a task group for natural radiation.

After several thousands of Bq m^{-3} had been found in Swedish houses and mass media had the news on the first pages, the government granted Bo Lindell’s request for a government commission to work with a limitation system for radiation exposure in dwellings. The Commission received a fund for targeted research. As one result, a random sampling was carried out in 1980-82 in the 1976 housing stock (Sw84), Table 2, mitigation methods were investigated, a calibration room was built etc. This research also implied that the knowledge about radon, earlier concentrated at SSI, was undergoing a rapid growth in other organisations. Much effort was put into informing health inspectors, builders, residents etc. The Swedish Radon Commission also issued recommendations on the distribution of responsibility between the different Swedish authorities.

Table 2 - Radon concentrations in single-family houses and apartments in multi-family houses in the 1976 Swedish housing stock (Sw84).

The measurements were made in 1980-82.

Type of dwelling	Arithmetic mean, Bq m^{-3}	Geometric mean, Bq m^{-3}	>800 Bq m^{-3}	Maximum[a)], Bq m^{-3}
Single-family	122	69	2 %	3310
Multi-family	85	53	1 %	920

[a)]Average of measurements in two rooms.

1980s

The recommendations of the Swedish Radon Commission were used by the responsible national boards to set requirements on radon levels lower than 400 Bq m^{-3} of EER (see App.) for existing dwellings and 70 Bq m^{-3} EER for newly built dwellings. It was also recommended that the level in existing dwellings should be decreased in the range 70 - 400 Bq m^{-3} EER when this could be done with simple methods. The risk estimates were based on the results of the epidemiological studies of miners, both uranium and non-uranium. The existence of an excess risk from radon exposure was also based on the results from animal research and from the atom bomb survivors. Swedish recommendations for miners were published in 1972. We meant, while waiting for better risk estimates, that the population should not be exposed to more radon than the miners. Consideration was taken to the different conditions for the population in dwellings and miners by a very simple model. The limits were decreased in 1990 when we had better measurement techniques, know more about mitigation and the health risks had been more thoroughly studied.

From the end of the 1970s larger surveys on radon indoors were performed in several countries. Their results are summarised in the reports of UNSCEAR. These studies revealed the extremely large variation in the radon levels in houses, not only in Sweden, covering a range from a few Bq m^{-3} up to 100 000 Bq m^{-3}. This means that some members of the public are being exposed to indoor radon levels comparable to those of underground uranium miners in the early phase of uranium mining. It was recognised that in most houses with high radon levels, the main source was not the building material but the convective radon influx from the soil.

The country averages of the indoor level of radon from these studies cover a range from about 10-100 Bq m^{-3}. UNSCEAR (1988, 1993) assumes a global mean of about 40 Bq m^{-3}, earlier 15 Bq m^{-3}. On the basis of a world average, about half of the total effective dose from natural radiation sources is due to the inhalation of radon in dwellings (ICRP87, UN88 and 93).

An intensive research on radon was going on in many countries shown in e.g. the International Seminar on Indoor Exposure to Natural Radiation and Associated Risk Estimates held in 1984 at Anacapri (In84). The principles of a possible limitation system were discussed.

The ICRP-recommendations about natural radiation came in 1984 (ICRP84) and those from the Nordic radiation protection boards in 1986 (No86). The World Health Organisation's Regional Office for Europe published their guidelines in 1987 (WHO87).

The interest for radon in water increased. A representative sampling of consumption water in Sweden was measured and recommendations for limiting the radon concentration in consumption water were given, principally limiting the inhalation exposure. The exposure from intake of the water was considered lower and would be limited at the same time.

During the 1980s the first epidemiological studies of radon in dwellings and lung cancer were performed. They were, however, small or designed in such a way that they could not be expected to give any quantitative correlation between radon exposure indoors and lung cancer occurrence. Calculation of the absorbed and effective doses had been started

during the 1970s (NEA83). At the 1980s the magnitude of the risk estimated on the basis of epidemiological studies of miners and that from dose calculations were similar.

1990s

Many well designed case-control epidemiological studies in dwellings are going on in the 1990s. The results from the published studies support the risk estimates based on the studies of miners. However, the lung dose model for radon exposure was not included in ICRP's lung dose calculations (ICRP94). Therefore, ICRP based their recommendations of 1993 (ICRP93) on risk estimates based on epidemiological studies of miners. For comparison with other radiation sources ICRP recommended the concept of "dose conversion convention" (see App.).

The radon exhalation from the ground varies from place to place and by time. Over the oceans the radon concentration is very low. Therefore, the radon concentration in coastal regions vary more than over continents depending on the wind direction. Temperature inversions can give very high concentrations in regions with high radon concentrations in the earth's crust. This can be a problem in the application of low limits for radon levels indoors. UNSCEAR has estimated the world-wide average outdoor concentration to be 10 Bq/m^3. The concentration indoors also depends on the building technique, the climate, and the occupancy habits. The building technique changes over time and therefore radon averages in homes might be quite different for various areas despite the same ground conditions.

Table 3 - Residential radon concentrations in some European countries

Countries	Average, Bq/m^3	> 200 Bq/m^3	> 400 Bq/m^3
Sweden	108	14 %	4.8 %
Finland	123	12.3	3.6
Norway	60	5	1.6
Italy	77	5	1
Ireland	60	3.8	1.6
Germany	50	2	1
UK	20	0.5	0.2

Many surveys of indoor radon concentrations have been carried out during the last decades. Country averages from a few countries are summarised in Table 3. The distributions are approximately log normal. The table illustrates the wide variety of country averages and portion of levels above 200 Bq m^{-3} between the countries.

A European atlas of radon indoors and gamma radiation outdoors and indoors has been published with support from the EU (CEC91). It illustrates the differences in averages between countries and regions.

Several countries have decreased their action and design levels for radon indoors during the 1990s. Recommended action levels for some countries are given in Table 4.

Table 4 - Recommended action levels for the annual average radon concentration in air in dwellings.

	Action level (Bq m^{-3})	
	Existing dwellings	Future dwellings
EU	400	200
ICRP	200-600	-
WHO [a)]	800	200
Canada	800	800
Czech Republic	400	200
Finland	400	200
Germany	250	250
Ireland	200	200
Norway	400	200
Sweden	400	200
Switzerland	1000	400
UK	200	200
USA	150	150

a) For F=0.5. The recommendations are under revision (Le97).

During the 1990s the interest for the exposure to radon in water increased, especially in Sweden. A limitation system was issued in 1997, stating that water with a radon concentration exceeding 1000 Bq/l is unfit for consumption. More than 100 Bq/l is a compulsory action level for public water plants. In 1992 Finland limited the effective dose to 0.5 mSv/year from exposure to natural nuclides in water from public water plants (An93). That corresponds to activity index $1 = C_\alpha + C_\beta + C_{Rn}/300$, which should be less or equal to one. C_α , C_β and C_{Rn} are the concentrations of total alpha, total beta and radon in the water in Bq/l. The requirement means that the radon concentration has to be less than 300 Bq/l with a good margin because the decay products from radon are always present in the water when radon is found. The Czech Republic has three levels when the radon concentrations should be observed (Ha93). More than 20 Bq/l is an indication that the concentration might be high. Public water works should not have higher concentrations than 50 Bq/l and the concentration in water from all types of dwellings should not exceed 1000 Bq/l.

Thoron is the name of ^{220}Rn, an isotope to ^{222}Rn described above. The ultimate parent nuclide ^{232}Th, is found in the earth's crust in similar concentrations as ^{238}U, the ultimate parent nuclide of ^{222}Rn. The physical half-life of thoron is only 55 seconds. Therefore, only a small part of the gas exhales from surfaces. Thoron indoors can be measured, reported for example in the 1950s (Hu56). It has been shown to be no problem in most countries with a few exceptions e.g. in some Japanese houses (Do94). The contribution

from thoron exposure relative to the radon exposure has been found to be of the same magnitude when the radon exposure indoors is low.

EU Research Program

An intensive research program for radon studies has been supported by the EU Radiation Protection Research Program and also by some other EU programs. The radiation protection program was earlier principally directed towards studies of the radon concentrations indoors; representative distributions of levels, time variations indoors, dynamics, mitigation etc. The program has principally been changed to studies with the objective to assess the human risk. To reach this aim a combination of several topics is required involving a multidisciplinary approach addressing five main topics: radioactive aerosol studies, lung modelling, human studies, animal studies and retrospective assessment of radon exposure studies. Another part of the radiation protection program includes studies of mitigation methods for reduction of radon and other natural radionuclides in consumption water.

Conclusions

The Swedish experience is that it takes at least a ten year period to disseminate the knowledge to health inspectors, builders, inhabitants etc. Most times when something has happened, for example when Hultqvist's study was published in 1956, and when very high radon concentrations were found in 1978, the Swedish mass media have been very interested. Most people in Sweden know something about radon. However, after 16 years with a radon limitation system, only 10-20 % of the expected number of dwellings with levels higher than the limit have been mitigated. The experiences from several of the other countries which have a limitation system for radon indoors are similar to the Swedish experiences.

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Appendix

1. Concentrations

2. Radiation dose concepts

APPENDIX I

Concentration and exposure quantities

based on publication 65 of the International Commission on Radiological Protection ICRP

	Bequerel	Activity concentration
Activity of radon	1 decay per second	For air Bq m^{-3}, For liquids Bq litre $^{-1}$

The potential alpha energy concentration, c_p, of any mixture of short-lived radon progeny in air is the sum of the potential alpha energy of these atoms present per unit volume of air, expressed in the SI-unit J m^{-3}.

The same concept can also be expressed in terms of **equilibrium equivalent concentration**, c_{eq}, of their parent nuclide, radon, also called EEC or EER. EEC is the activity concentration of radon in radioactive equilibrium with its short-lived progeny that has the same potential alpha energy concentration, c_p, as the actual non-equilibrium mixture. The SI unit of the EEC is Bq m^{-3}.

The unit of the **exposure quantity** P, of an individual to radon progeny is defined as the time integral of the potential alpha energy concentration in air c_p, *or* the corresponding equilibrium equivalent concentration, c_{eq}, of radon to which the individual is exposed over a given period T, e.g. one year. The unit of the exposure quantity P_p is J h m^{-3}. For the exposure quantity P_{eq} the unit is Bq h m^{-3}.

The potential alpha energy exposure, P_p, of workers is often expressed in the historical unit **Working Level Month** (WLM). 1 WL is now defined as a concentration of potential alpha energy of 1300 x 10^8 MeV m^{-3}. 1 month was taken to be 170 hours.

The conversion coefficients between the mentioned quantities and units are given in Table 1.

Table 1 Conversion coefficients for the different concentration quantities and for the corresponding exposure quantities for radon-222 (ICRP-publication 65).

Quotient	Conversion coefficients
c_p / c_{eq}	5.56 x 10^{-9} (J m^{-3}) per (Bq m^{-3})
c_{eq} / c_p	1.80 x 10^8 (Bq m^{-3}) per (J m^{-3})
P_p / P_{eq}	5.56 x 10^{-9} (J h m^{-3}) per (Bq h m^{-3})
	1.57 x 10^{-6} WLM per (Bq h m^{-3})
P_{eq} / P_p	1.80 x 10^8 (Bq h m^{-3}) per (J h m^{-3})
	6.37 x 10^5 (Bq h m^{-3}) per WLM

Quantities:
c_p - concentration of potential alpha energy,
c_{eq} - equilibrium equivalent concentration of radon,
P_p - time-integrated exposure to potential alpha energy concentration,
P_{eq} - time-integrated exposure to equilibrium equivalent concentration of radon.

APPENDIX II

RADIATION DOSE CONCEPTS

based on publication 60 of the International Commission on Radiological Protection (ICRP)

Absorbed dose, D

The energy absorbed per unit mass.
Unit: joule per kilogram
Special name: gray (Gy)

Radiation weighting factors, w_R

The absorbed dose is weighted by a factor related to the quality of the radiation to receive

Equivalent dose, H_T

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

where $D_{T,R}$ is the absorbed dose averaged over the tissue or organ T, due to radiation R.
Unit: joule per kilogram
Special name: sievert (Sv)

Tissue weighting factors, w_T

The equivalent dose is weighted by a factor related to the relative contribution of that organ or tissue T to the total detriment resulting from uniform irradiation of the whole body to receive

Effective dose, E[a]

with consideration to the different radiation sensitivity of organs

$E = \sum_T w_T \cdot H_T$

where H_T is the equivalent dose in tissue or organ T and w_T is the weighting factor for tissue T.
Unit: joule per kilogram
Special name: sievert (Sv)

Detriment coefficient

is a risk factor combined of morbidity, mortality and expected loss of life·
Unit: $(\text{sievert})^{-1}$

Risk estimate

= The effective dose times the detriment coefficient

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[a] For radon exposure ICRP has recommended the concept **dose conversion convention** (ICRP-65) by which the ”effective dose” is obtained as the epidemiological risk estimate for miners and residentials divided by the detriment coefficient described above.

Mitigation

The mitigation method should be chosen according to the radon source. Mitigation methods are described in detail in a few books (Clavensjö and Åkerblom 1994, EPA) and in booklets given in several countries for example in UK (BRE), Finland, Norway and Sweden. Mitigation methods in many countries have been reported. The following principles for radon reduction indoors in existing buildings are applicable

When the **soil** below the building is the major radon source:

1. Reversing the pressure differential between the building and the soil, often called soil depressurisation. This is most easily achieved by using a small fan to withdraw the radon from the region under the floor or, when the ground is very porous, near the house (radon well). Moderate cost and high effectiveness.
2. Raising the resistance of the foundations to soil gas entry. Very effective and low cost when the radon entry can be easily found. Otherwise sealing is difficult to make effective in existing buildings because there are mostly many routes of entry for radon from the ground, then moderate cost and moderate effectiveness.
3. Removing the radon source. This is likely to be feasible only in extreme cases such as removal of the underlying soil. High cost and high effectiveness.
4. Diluting the radon by increasing the ventilation rate. Some forms of ventilation will decrease the pressure in the building, thus increasing the radon input. Moderate cost and low effectiveness.
5. Diluting the air with indoor or outdoor air under the house, e.g. in a crawl space. If correctly applied, moderate cost and high effectiveness.

When the **building material** is the major radon source:

1. Diluting the radon by increasing the ventilation rate. Moderate cost and moderate to high effectiveness.
2. Sealing of the walls etc. Moderate cost and moderate effectiveness.
3. Removing the radon source. High cost and moderate to high effectiveness.

When the **water** is the major radon source:

1. Air the water before it comes into the building. Several companies sell such equipment. A problem might be growth of bacteria. Moderate cost and high effectiveness.
2. Filter the water through a charcoal filter. Problems might be radium in the filter and growth of bacteria. Moderate cost and moderate effectiveness.
3. Changing water supply. This is usually not possible for private wells. Mostly high cost and high effectiveness.

Some of these remedial measures depend on a continued expenditure if they are to be effective. Local circumstances will influence the choice of methods. The costs vary between the methods but also for the same method between countries.

It is important to check the radon concentration after the measure, but also in the future at regular intervals, e.g. 10 years. It has been shown that most measures will last many years (Clavensjö 1997, Naismith 1997). However, the radon concentration has increased in a portion of buildings with low radon concentrations after mitigation. This was the case for all kinds of mitigation methods and reasons of various kinds. In many cases erroneous mitigation method had been chosen, e.g. increased ventilation with increased inflow of radon. One reason which could be prevented was that the function of the fans decreased or stopped.

New buildings planned to be built on radon risk soil should of practical and often also cost-effective reasons be built radon protective.

Literature

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MEASUREMENTS OF RADON IN DWELLINGS: STATE OF THE ART AND RESEARCH PROSPECTS

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ABSTRACT

Methods for measuring radon 222 and radon 220 and their decay products in dwellings are presented and discussed. Special attention is devoted to quality control and quality assurance of measurements of both nuclides. Research prospects in each field are suggested. Utility of gamma spectrometry *in situ* in some situations is also shortly analysed. Conclusions are drawn on what type of experimental efforts should be planned in the near future in international projects.

INTRODUCTION

It is well known that interest in radon concentration in indoor air as a pollutant emerged during the energy crisis of the Seventies which led to reduced ventilation in dwellings. Since then, much experimental work has been done and the knowledge of this gas and its decay products, their behaviour and related relevant health risks have been the aims of research for many scientists around the world. Attention has essentially focused on Rn-222 isotope (of the U-238 chain) and its progeny. In fact, the 220 isotope (the so-called *thoron* of the Th-232 chain) and its progeny - due to the short decay time of the parent ($T_{1/2}$ = 55.6 s) - generally do not show high concentration indoors. However, considerable research in recent years has been carried out on Rn-220 and its progeny (M. Doi et al. 1994, M. Zhukovsky et al. 1997, M. Buzinny et al. 1997, F. Bochicchio et al. 1996b, L.Mijones et al. 1996, F. Steinhäusler 1996a), showing that in some zones or regions they can not only reach significant concentrations, but relevant effective doses comparable to those due to Rn-222 and its decay products. For this reason after discussing the current situation of research on radon 222 in dwellings, a short final paragraph will also cover radon 220. At the end a short paragraph is devoted to a particular technique - γ spectrometry *in situ* – which is a powerful method in those dwellings where building materials are rich of natural radioactivity.

MEASUREMENTS OF RADON 222 IN DWELLINGS

At present, a large number of methods, both active and passive, are available for measuring concentration of radon 222 and its decay products in an indoor environment (see e.g. the review in A.C. George 1996). The choice of one method over the others is strongly guided by both the aim of the measurement and the related degree of accuracy required. That is, one must have the final goal clear before measurement, whether it is to be a screening aimed at identifying indoor environments with high radon concentration, an assessment of the distribution of radon concentration indoors, the identification of the source of radon in order to take remedial action, the re-test of the dwelling after remediation, the estimate of the cumulative exposure of people over many years, the organisation of an intercomparison exercise, etc. Indeed, an ideal method for one objective might not be suitable, and usually isn't, for a different objective. A detailed review of all methods and their ideal applications cannot be done here due to space

limitations. In the discussion which follows, attention will therefore be devoted only to some particular items or open points of discussion emerging from the scientific literature and the most recent seminars and congresses on these themes.

In order to evaluate the exposure - that is, the mean annual radon concentration distribution and resulting risk - of the population to Rn-222 and its decay products in a single country's dwellings, *statistically representative national surveys* should be organised. This type of survey is often called a *second generation* survey, in order to distinguish it from the previous ones, with which many research groups since the Seventies have carried out screenings, often in limited zones, or tests of sampling procedures and experimental methods.

Second generation surveys are usually conducted over extended periods by means of passive integrating detectors. The most tested and widely used method is a passive detector containing a track etch detector, even if alternative methods, such as small ionisation chambers containing electrets, are preferred by some research groups. In most cases until now, the physical quantity to be measured in dwellings has been considered radon concentration, even if the true cause of respiratory system irradiation is the alpha particles of its decay products. In order to measure radon concentration in most types of passive detectors (so-called *closed detectors*) the track detector is in a closed container, which allows diffusion of external Rn-222 but not that of radon decay products. This way, inside alpha particle tracks can be correlated to the radon concentration in external air. A great number of different closed detectors have been designed and tested both in laboratory and in field conditions and shown to be useful and generally reliable. However, their reliability is highly dependent on many parameters including the skill and diligence of the operator, making repeated intercomparison exercises essential (see below).

A different approach is used by some laboratories (see e.g. I. Burian et al. 1997, I. Los' et al. 1996) which utilise bare nuclear track detectors (so-called *open detectors*). In this case, the Rn-222 decay products are recorded and the results are obtained by assuming a value for the equilibrium factor.

Because of the high variability of radon concentration in the course of a single year, mainly due to wind, temperature, atmospheric pressure, differential pressure between inside and outside air, and so on, measurements are made for periods from several months (see e.g. J.Pinel et al. 1995) to a year (see e.g. Wrixon et al. 1988, M.K. Langroo et al. 1991, O. Castrén 1994, F. Bochicchio et al. 1996a). In the former case a correction factor should be used to predict the mean annual value based on the measurement. Up to now a *seasonal correction factor* has generally been estimated for the country, comparing large numbers of measurements carried out in different seasons, but recently a good correlation seems to have been obtained between radon concentration indoors and outdoor temperature (L.Hubbard et al. 1996, J.Miles 1997, L.Minach 1996), so that a new type of correction factor might soon be used in routine estimates.

At present, these integrated measurements of Rn-222 concentration are the basis of the assessment of the radiological risk related to the inhalation of this gas, but additional information about the degree of equilibrium between Rn-222 and its decay products is essential for the estimate. There is a general agreement at present on the mean value of this factor to be assumed for domestic environments, 0.4 (see e.g. UNSCEAR 1993, ICRP 65). However, this factor was rarely assessed for extended periods, and the assumed mean value was obtained and confirmed by studies conducted mainly in

Northern European and non-European countries, primarily in single or semi-detached houses. Consequently, little data is available for temperate countries (F. Bochicchio et al. 1995) - and even less for hot countries - and for apartments in large buildings, where upper storeys presumably have greater natural ventilation rates.

A comment must be made about the use of charcoal canisters for this type of estimate. Due to its ease of use, it remains a popular system for measuring radon concentration indoors. However, its experimental limitations can lead to large errors. In 1984 George et al. (A.C. George 1984, A.C. George et al. 1990) showed that open-faced charcoal canisters do not act as real integrators because, due to the capacity of activated charcoal to both adsorb and desorb radon gas, they follow the radon concentration variations. Moreover, Scarpitta (S.C. Scarpitta et al. 1990) pointed to the fact that charcoal also absorbs environmental water vapour and reaches a *break point* beyond which its active sites can no longer adsorb radon. These are the reasons why the EPA decided to adopt diffusion charcoal canisters (see e.g. D.J. Gray et al. 1992), where a barrier (e.g. of foam) slows the diffusion of radon concentration (both adsorption and desorption) in the charcoal canister - thus improving the integrating capability of charcoal - but slows the diffusion of water vapour to a greater degree. With this method the possible integrating period is extended from 1-2 days to about seven days, without reaching the break point. A special protocol was set up by the EPA (EPA 1989) for conducting measurements. Radon concentration indoors should, for example, be measured in the basement or lower floor of the dwelling, during the winter, after having closed all windows and doors in the room for 12 hours so as to maximise the concentration. Thus the measured value serves as a screening result, e.g. to identify areas with high radon risk, but it cannot be considered an accurate estimate of radon concentration in that dwelling. Unfortunately, several laboratories around the world continue to use open charcoal canisters or diffusion canisters while neglecting to follow the EPA protocol and publishing their results as mean values, sometimes even predicting annual radon exposure for inhabitants.

A different type of approach to integrated radon concentration measurements comes from epidemiological studies, where knowledge of the life-time exposures for both *cases* and *controls* is essential. Instead of measuring present radon concentration in the dwellings inhabited by the person and multiplying the exposure by the relevant years, cumulative exposure to radon concentration could be assessed studying the tracks of alpha particles of Po-210 implanted in glass surfaces. This method first proposed by Samuelsson (C. Samuelsson 1988) appeared powerful and was adopted by several research groups. However, its results are strongly affected by aerosol particles present and the uncertainty for radon exposure easily exceeds a factor of two (S. Oberstedt 1997a). For this reason, above all, estimates obtained with this method have not been used as the basis of exposure in epidemiological studies, but as a support of other estimates. That is, in case-control epidemiological studies these estimates can help to eliminate cases and controls found to have lived substantial period of time in dwellings where radon levels in the past differ from those at present by an amount greater than could be due to uncertainties in the retrospective assessment techniques (J.P. Mc Laughlin, 1997). Moreover, the combination of this method with the analysis of the Po-210 activity deposited in volume traps (such as sponge materials) has recently been proposed and discussed (S. Oberstedt 1997b).

Finally, the quality assurance and quality control of radon indoor measurements is worth examining. It is of primary importance that reliable and reproducible results are obtained,

that traceability to national standards is ensured and that national standards are made consistent. In this field a large experimental effort has taken place over the last few years.

First of all, several laboratories have developed a Rn-222 reference standard. Repeated international intercomparison trials were conducted in 1990, with the participation of several national standards laboratories, in 1992, in the framework of a EUROMET project, and again in 1994 as a EUROMET project. The aim of the 1994 exercise was to improve the consistency of measurements among European countries as some discrepancies between participating laboratories had become evident during the first two intercomparison exercises (P. De Felice 1996). As a matter of fact, in 1992 the observed spread of results of the 12 laboratories involved in the exercise was of the order of ± 20%, whereas in 1994 it reduced to about ± 5% for the laboratories which had already participated in the previous one. This improvement was due partially to the adoption of a common source of nuclear decay data and to the use of traceable calibration standards (J. C. J. Dean et al., 1996).

The next step of this work, now underway in several laboratories, is to create standard atmospheres traceable to Rn-222 reference standards.

Moreover, since the early Eighties, seven international intercomparisons of passive radon detectors - the latest still ongoing - have been organised by the National Radiological Protection Board, with the financial support of the Commission of the European Union (EC 1996). In the same period analogous initiatives were periodically organised by IAEA, EML, etc. The NRPB exercises concentrated mainly on EU Member States, but participation by other countries increased over the years, as did the different types of dosemeters involved in the exercises. Exposures were generally arranged in the NRPB radon chamber, and sometimes in a building as well. Over the years a clear improvement was observed in the measurement quality of detectors and regularly participating laboratories, and some general conclusions could be drawn about dosemeter performances.

The last intercomparison concluded, that of 1995 (EC 1996), also included for the first time three exposures in three homes in northern, central and southern Europe. These exposures were organised by the University of Gent (Belgium) while the facilities and monitoring of conditions were provided by the Swedish Radiation Protection Institute (SSI), by the Centre Universitaire in Luxembourg and the Italian National Institute of Health (ISS). In order to provide a comparison of exposure in the laboratory and in the domestic environments, continuous radon and radon decay product monitors were exposed in the laboratory and then used to monitor the homes. A final paper on this exercise has not yet been published; however, preliminary results demonstrate the utility of this experience and the need to prepare and test detailed protocols for this type of exposure (EC 1996, F. Bochicchio et al. 1998, A. Poffijn et al. 1997). In the author's opinion it is extremely important that this type of exercise be repeated, in the framework of laboratory intercomparisons, because the overall quality of measurements should be verified under actual field conditions.

MEASUREMENTS OF RADON 220 IN DWELLINGS

In recent years increased attention has been devoted by researchers to Rn 220 concentration in dwellings. In the countries where high concentrations of this gas or its progeny are found, their origin is generally building materials and sometimes soil (Y Li et al. 1992). For example in Italy it seems that high concentrations indoors are mainly

related to the use of natural building materials of volcanic origin, such as tuff and pozzolana (see e.g. F. Bochicchio et al. 1994, G. Sciocchetti et al. 1992), whose thorium and uranium content is typically in the hundreds of Bq/kg. On the contrary, in some other countries, high concentrations of Rn-220 were detected where certain kinds of building materials of artificial origin had been used. The most common one is phosphogypsum (see e.g. A. Poffjin 1985, D. Georgescu 1997) a by-product of the fertiliser industry.

To measure the concentration of Rn-220 and its decay products, a different approach is needed from that used for Rn-222 and its decay products. First of all, due to the very short half life of Rn-220, its concentration in an environment is highly dependent on distance from the source. Therefore, defining an equilibrium factor for this isotope has scarce meaning, and in order to make measurements of this gas comparable, the distance of the measurements from the source should be agreed upon in international protocols. In contrast with what happens with Rn-222 concentration, due to the impossibility of defining a mean equilibrium factor Rn-220 concentration does not have a direct dosimetric meaning, which exists only for Rn-220 decay product concentration (C. Nuccetelli et al. 1997). However, at present, only scattered and short-term measurements of concentration of both radon 220 and its decay products in dwellings have been published. Neither systematic nor statistically representative data is available for any country; this means that, as far as Rn-220 is concerned, knowledge is still at the stage of first generation surveys (C. Nuccetelli et al. 1997).

Measurement methods of Rn-220 and its decay products, compared with those of Rn-222, are also much less developed at present. Some methods were set up in recent years for active and short-term measurements, but passive integrating methods are essential for representative surveys. Only one passive track etch detector for measuring Rn 220 concentration is described in the literature: it was set up by Japanese researchers some years ago and used for local surveys (M. Doi et al. 1992). It consists of two small hemispheric chambers each containing one track etch detector; the two chambers are connected to each other and to external air by means of appropriate filters. Taking advantage of the different diffusion coefficients of the two isotopes in the two chambers, one chamber can record the alpha tracks due to both Rn-222 and Rn-220 concentration, while the other records only those for Rn-222. Another passive and integrating system for Rn-220 concentration was set up by Kotrappa (P. Kotrappa 1988, P. Kotrappa 1996) modifying the *type S electret-passive environmental radon monitors* (E-Perm). With this system, the Rn-220 concentration is obtained by subtracting the Rn-222 concentration from the sum of the two. In any case, the hypotheses assumed in the calculations are not particularly convincing.

As far as integrated measurements of Rn-220 decay products are concerned, the author knows of no passive measuring system presently available, and this represents a future challenge for researchers in this field.

Another less developed aspect in Rn-220 measurements is quality assurance and quality control. At present, although some countries may have a reference standard for Rn-220, they have certainly never been intercompared at the international level. Moreover, some countries (such as Canada, Sweden, and the United States) are now able to calibrate equipment in a Rn-220 chamber, but no international intercomparison exercise has yet been organised on the measurements of this gas and its decay products. Therefore no guarantee can currently be given about the traceability of these measurements.

From this summary and short discussion it should be clear that a great deal of experimental and research work remains to be done and that greater attention to Rn-220 would be fruitful.

BUILDING MATERIALS AS A SOURCE OF RADON CONCENTRATION AND GAMMA DOSE RATE INDOORS

In some countries (see e.g. E. Stranden et al. 1986, G. Carrera et al. 1997) building materials contribute significantly to the radiation exposure of the inhabitants in some zones. In these dwellings, not only radon concentration but also gamma exposure can contribute seriously to the total effective dose (G.Campos Venuti et al. 1988). In these cases a new tool has became available. This tool, gamma spectrometry *in situ*, was developed more than twenty years ago by H.l.Beck (H.L.Beck et al. 1972) to calculate the radioactive content of soil by analysing the full absorption peaks of the spectrum. It was subsequently adapted to an indoor geometry to evaluate the relative contribution of radioactive families in building materials to the indoor gamma dose (M.Miller et al. 1982). This powerful and promising method can be useful in diagnosis of the origin of high Rn-222 concentrations (or Rn-220 concentration) and of possible high levels of indoor gamma exposure. The advantages are that it can be run in a short time interval (one or several hours depending on the efficiency of the gamma spectrometer and the activity content of building materials), without the need to sample the material and measure it in a laboratory. An example of the application of this method in a local survey in Italy showed (F. Bochicchio et al. 1994) that the fraction of gamma dose rate due to the Th-232 family can in some situations be quite higher than that of U-238.

CONCLUSIONS

Over the last twenty five years a great deal of experimental work has been done in order to assess the concentration of radon and radon decay products indoors and to increase knowledge about this gas and its behaviour. Evidence comes from the numerous seminars and congresses on this issue held in various countries and the impressively large number of papers devoted to it in many scientific journals. However, some aspects require further analysis and experimental effort.

First of all, until now the calculation of the effective dose for the population due to radon concentration indoors has been made based on measurements of radon concentration in homes using several mean factors or assumed parameters (e.g. equilibrium factor, occupancy factor, breathing rate, attached fraction, etc.) whose estimate is affected by a high degree of uncertainty. This fact may introduce a large uncertainty in the final estimate (see e.g. A. Birchall et al. 1994, F. Steinhäusler 1996b) and emphasises the need to develop more precise evaluations in the future (see e.g. C.Baixeras et al. 1997).

Moreover, quality assurance and quality control of measurements of both Rn-222 and Rn-220 and their decay products is essential. This means that metrological institutes should continue to improve their efforts to obtain consistent standards for the different quantities and national institutes (such as NRPB, EML, etc.) should repeat intercomparison exercises in both laboratory and field conditions. Particular attention should be devoted to expanding these practices to countries which - for completely different reasons - are only now beginning to discuss their scientific work in international seminars and congresses.

Lastly, as stated above, it is essential that appropriate passive detectors for measuring the concentration of Rn-220 decay products be developed in order to obtain an estimate of their distribution and an assessment of its overall health significance.

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PULMONARY DOSIMETRY; STATE OF THE ART

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Abstract

The majority of the concern for health risks from radon exposure is due the inhalation of Rn-222 and its progeny which results in the irradiation of the different tissues of the respiratory tract. The dose to the different regions of the lung arising from inhalation of radon progeny cannot be measured directly. It must be calculated using models which take into account the various processes involved : inhalation, deposition, clearance and decay of radon progeny in the airways of the lung. This paper reviews the main historical dosimetric models and the different morphometric models used for dosimetric purpose. The new ICRP lung model and the influence of the localisation and distribution of target cells and of deposition and clearance parameters on dose calculations are also described. A comparison between the different dosimetric models and the influence of modelling variables on the uncertainties of dose calculations are also addressed.

Introduction

Radon is a noble gas that occurs in several isotopic forms. These are Rn-222, Rn-220 and Rn-219 which are formed in the decay series of U-238, Th-232 and U-235, respectively. The majority of the concern for health risks from radon exposure is due to the presence in the environment of the isotope Rn-222, which has the longest half-live, 3.824 days, of the three isotopes. Thus, this paper will be restricted to the lung dosimetry of inhaled Rn-222 progeny. Rn-222 is ubiquitous since it is a highly mobile gaseous radionuclide and its precursor radionuclides U-238 and Ra-226 are present everywhere in the earth' crust. The decay products of Rn-222 are radioisotopes of heavy metals (polonium, lead, bismuth). They have the physical and chemical properties of the corresponding solids and behave accordingly in the atmosphere. Radon decay products occur as unattached ions or atoms or attached to aerosol particles already present in the atmosphere. The probability of attachment is high and depends strongly on the concentration of particles in the air. The size of aerosol particles that carry radon decay products is a mixture of pre-existing particles to which radon decay products have become attached and of newly formed radon decay products which have not yet become attached to carrier particles.

The dose to the different regions of the lung arising from inhalation of radon progeny cannot be measured directly. It must be calculated using models which take into account the various processes involved: inhalation, deposition, clearance and decay of the radon progeny in the airways of the lung. The dose of alpha-radiation delivered to lung tissues is a function of:

(i) the concentration of radon decay products in air,

(ii) the volume of air inhaled by the subject (worker or member of the general public) over the monitoring period,

(iii) the fraction of inhaled radon decay products which remain deposited in the various regions of the respiratory tract.

In turn, the fraction of radon progeny deposited in the respiratory tract is a function of the geometry of the airways themselves, the breathing rate, the breathing pattern (through the mouth or the nose), and the size of inhaled particles. Among these parameters, the only quantity which is measured routinely is the concentration of radon progeny in air. The other parameters, which are depending of environmental or personal factors, are average morphometric values (dimension of human airways), the size distribution of the inhaled activity and breathing rate and pattern. Therefore, the dose due to the inhalation of radon progeny is always affected by errors and uncertainties of generally unknown magnitude. Despite these uncertainties, efforts have been made to quantify the dose received by lung tissues after inhalation of radon progeny.

The first modern lung models describing the inhalation, deposition and retention of radon progeny appeared in 1964 (Altschuler et al., 1964; Jacobi, 1964). In 1966, a revised lung model was published (TGLD, 1966). The dosimetric model used for the respiratory system (*ICRP Publication 30*, 1979) was a slightly modified version of the model developed and published in 1966. In both these models, the calculated dose was averaged for the total mass of the blood-filled lungs to give a mean lung dose (Mean Lung Dose concept). In the *ICRP Publication 30*, the contents of the thoracic lymph nodes were included with those of the lungs. In *ICRP Publication 32* (1981), the ICRP recommended a dosimetic approach, the Regional Lung Dose concept, in which separate doses were calculated for the bronchial basal tissue layer and for the pulmonary region; half the lung weighting factor for stochastic risks was applied to each region. In 1994, the ICRP published its "Human Respiratory Tract Model for Radiation Protection" (*ICRP Publication 66*, 1994 b), which is a major revision of the respiratory tract dosimetric model used in *ICRP Publication 30* (1979). Recently, the NCRP published its "Deposition, Retention and Dosimetry of Inhaled Radioactive Substances" report (NCRP Report n° 125, 1997).

Morphometry

Lung Morphologyical Models

The first step in evaluating absorbed doses is to calculate the fraction of inhaled radon progeny deposited on airway surfaces within the lung. To do this, it is necessary to use a simplified geometrical model of airway size and branching. Three principal lung morphological models were used for lung dosimetry purpose :

The Weibel A model (Weibel, 1963).

This model is based on histological sections and casts of human lungs. The lung is represented as a single lobe structure in which an airway divides dichotomously. The Jacobi-Eisfeld lung dosimetry model uses the Weibel model of the lung.

The Yeh and Schum model (Yeh and Schum, 1980).

This model is based on human lung replica casts. In this, the lung is treated as a five lobe system in which each lobe has its own characteristic set of branching patterns. In this representation, the airway dimensions of the model are slightly larger than those of the

Weibel A model. Both the Harley-Pasternack (1982) and the James-Birchall (1980) lung dosimetry models use the Yeh and Schum model of the lung or an adapted form of it.

The University of California at Irvine (UCI) model (Phalen et al., 1985).

This model is based on a large number of human replica casts from persons of various ages. The airway dimensions are generally smaller than those given by the Weibel A or Yeh and Schum models.

It is not clear which of the above models is the most appropriate for lung dosimetry purpose. In its new respiratory tract model, the ICRP (ICRP 66, 1994 b) has adopted values for airway dimensions in the bronchial and bronchiolar regions which are adjusted average values from the above three referenced models.

The New ICRP Morphometric Model (ICRP 1994 b)

The model considers the respiratory tract as divided in four anatomical regions :

(1) The extrathoracic region (ET), which comprises the anterior nose (ET_1) and the posterior nasal passages, larynx, pharynx, and mouth (ET_2),

(2) The bronchial region (BB), consisting of the trachea and bronchi from which deposited material is cleared by ciliary action,

(3) the bronchiolar region (bb), which consists of the bronchioles and terminal bronchioles, and,

(4) the alveolar-interstitial region (AI), consisting of the respiratory bronchioles, the alveolar ducts and sacs with their alveoli, and the interstitial connective tissue. All these four regions contain lymphatic tissue (LT) or components of it. Fluid accumulated in the interstitial connective tissue is collected in lymph capillaries, lymph vessels and passes through one to several lymph nodes: LN_{ET} drain the extrathoracic region (ET), and LN_{TH}, which are located in the bronchial region (BB), drain this region as well as the bronchiolar (bb) and alveolar-interstitial (AI) regions.

For dosimetric calculations, a typical airway is represented by a cylindrical tube of appropriate internal calibre and wall thickness. Various scaling factors must be used to scale the airways for age, gender and ethnicity.

Respiratory physiology

Radiation doses to tissues and cells of the respiratory tract are determined to a large extent by breathing characteristics and by certain respiratory parameters. These are largely a function of body size, level of physical activity (sleep, rest, light or heavy exercise). For dosimetric calculations, the *ICRP Publication 66* recommends a set of reference values for respiratory parameters for the general Caucasian population. Guidance is provided for adjusting these values for other ethnic groups.

Radiation biology

Knowledge of the types and locations of cancers that occur in the respiratory tract, is helpful in identifying the most likely cells and tissues at risk.

Respiratory tract cancers

Extrathoracic airway

A large variability exists in wordwilde risks of upper airway cancers according to ethnic origin and way of life. The incidence of cancers of the mouth, pharynx, and larynx is strongly influenced by smoking and alcohol comsumption. Radiation-induced cancers of the extrathoracic airways have not been found in excess in the epidemiological studies of the atomic bomb survivors or patients treated for ankylosing spondylitis (Darby et al., 1985). However, paranasal sinus cancers were observed amongst radium dial painters (Rowland, 1994).

Tracheobronchiolar airways and the lung

Most respiratory cancers are thoracic and the largest proportion of lung tumours is arising from the bronchi. Half of the lung tumours arise centrally from the main bronchi, to the point of division of the segmental bronchi (generation 3), and half arise distally to the segmental bronchi. In general terms, the range of frequencies of the four major classes of lung cancers among human populations can be summarized as follows (*ICRP Publication 66*, 1994 b) :

- Squamous cell (epidermoid) carcinoma : one-third to one-half,
- Small cell carcinoma : one-sixth to one-third,
- Adenocarcinoma : one tenth to one-third,
- Large cell carcinoma : less than one-tenth to one-fifth.

The frequencies of these four major classes differ between smokers and non smokers. For non-smokers about 30% of lung cancers are squamous cell carcinomas, 20% are small cell carcinomas, 45% are adenocarcinomas and 5% are large cell carcinomas. For smokers, the proportion of squamous cell carcinoma may increase up to 50%, while the proportion of adenocarcinomas may be about 25%. Small cell carcinoma and squamous cell carcinoma are mainly central in the lobe. Squamous tumours of peripheral origin might be underestimated, as suggested by the study in tin miners in China exposed to high concentrations of radon and progeny which showed that 44% of all tumours were peripheral and 59 % of those were epidermoid. In contrast adenocarcinoma are mainly peripheral.

It is known that small cell carcinoma and squamous cell carcinoma were closely related to cigarette smoking. However, during the last twenty years, the prevalence of adenocarcinoma strongly increased in relation with the consumption of less irritating low tar and low nicotine cigarettes. In parallel, in uranium miners with heavy exposures (Saccomanno et al., 1964; 1971; Horacek et al., 1977) exceptionnally high rates of small cell carcinoma were reported. A 66% incidence of this tumour type was observed in a cohort of Colorado Plateau uranium miners (Yessner, 1981). However, the most recently diagnosed cases among the Colorado cohorts do not confirm a high ratio for small cell carcinoma (Saccommano et al., 1996). Squamous cell carcinoma became predominant and small cell carcinoma decreased progressively to 22%. Adenocarcinoma increased up to about 15% in those who started mining when they were age 20-29 years. In both types

of exposure, the decreased incidence of small cell carcinoma might be related with lower doses, lower doses of irradiation for miners, and lower doses of tar and nicotine for cigarette smokers.

In humans, tumours originating from lung parenchyma, either from alveolar pneumocyte type II cells and from Clara cells are extremely rare. They are frequently induced in experimental animals, mice, rats and dogs. Distal airway tumours might be the main consequence of long-term retention of insoluble radionuclides in the alveolar region of the human lung, as well as in experimental animals. However peripheral tumours are also found in animals treated with chemicals such as arsenic, beryllium, nickel sulphide, vinyl chloride, instead of the more central location of tumours usually found in exposed people. It is likely that peripheral tumours in animals reflect a specific animal sensitivity of the distal airways.

Two different mechanisms might be involved leading to the development of peripheral carcinoma. As mentionned by Kuschner (1995), scar tumours, mainly adenocarcinomas, occur when scarring is a common process, as in workers exposed to asbestos or in cured tuberculosis patients, as well as in experimental animals. The other process is the expanding adenomatous to carcinomatous lesion found in experimental animals.

Cells at risk

It is presumed that the cells of the lung that are sensitive to radon-induced cancers are those that are not terminally differentiated but have still the capability for division and differentiation. Among the numerous types of cells in the bronchial and bronchiolar epithelium, only basal and secretory cells appear to have the potential for proliferation. These two cell types are presently considered the most likely progenitor cells for lung cancers by both the US NRC (1990) and the ICRP (*ICRP Publication 66*, 1994 b). Other bronchial cells such as goblet (mucous) cells and serous cells are fully differentiated cells and therefore unlikely to be involved in tumour development.

For dosimetric modelling purpose, the ICRP assumes that the basal and secretory target cells are distributed as follows : (i) for a typical bronchus in region BB, the nuclei of columnar secretory and short basal cells are considered to be the sensitive targets and are assumed to occur uniformly throughout a 30-µm-thick layer of tissue at 10-µm depth, and in a 15-µm layer at 35-µm depth respectively; (ii) for a typical bronchiole in the region bb, the nuclei of secretory (Clara) cells are considered the sensitive targets and are assumed to occur uniformly throughout the 8-µm-thick layer of tissue at 4-µm depth. The target cells in the alveolar-interstitial region (AI) are the secretory (Clara) cells of the respiratory bronchioles and type II epithelial (pneumocyte) cells. Type II epithelial cells cover only about 7% of the alveolar surface, but there are twice as many as the much larger type I epithelial cells.

Arguments, however, have been presented against the basal cells being the primary target cells based on the fact that the peripheral epithelium has a very low density of basal cells. In some lung dosimetry models, such as the James-Birchall model and the new NCRP model, doses are calculated over the whole thickness of the bronchial epithelium which is effectively equivalent to assuming that sensitive cells may found throughout this tissue layer. Experimental data suggest that the secretory cells may be the major progenitor cells for the bronchial epithelium and that basal cells have a limited differentiation capacity (Johnson and Hubbs, 1990).

Deposition

In the ICRP 66 Respiratory Tract model, each region of the respiratory tract is represented by an equivalent particle filter, in which particles are deposited during inhalation as well as exhalation.

In the new ICRP deposition model, a semiempirical approach is used to describe values of regional deposition derived from experiments and/or theory using fluid mechanics approaches.

The dose delivered to respiratory tissues depends mainly on the initial repartition of particles in the airways during inhalation. The total deposition probabilities for a given aerosol size distribution may be obtained by combining the probabilities of the various deposition mechanisms. The deposition efficiency of each respiratory tract filter is represented by two components, η_{ae}, which arises from the "aerodynamic" deposition processes of impaction and gravitational settling, and η_{th}, which arises from the "thermodynamic" process of particle diffusion by Brownian motion.

The separate deposition mechanisms are combined in such a manner that the regional deposition efficiencies η_i may be expressed by an equation which has the same form for thermodynamic or aerodynamic deposition processes :

$$\eta_{ae} \quad \text{or} \quad \eta_{th} = 1 - \exp\left(-aR^p\right)$$

Each formula defining a reference deposition efficiency is expressed in terms of three parameters : *a*, *R* and *p* for each deposition regime. These parameters are different for each of the five regions, ET_1, ET_2, BB, bb and AI, and for nose breathing and for mouth breathing.

Parameters *a* and *p* are numerical while the values of parameter *R* depend explicitly on the particle sizes and on the volumetric airflow rate or the transit time of the air through a region. The resulting components of particle deposition are combined to give the overall deposition efficiency, η_i, in each region as a function of particle size and the relevant respiratory parameters.

The initial filter η_0, represent the potential loss of particles at either of the two intake points, i.e., before entering mouth or nose. The deposition efficiency of this imaginary prefilter η_0, is the complement of the inhalability or intake efficiency η_i , ($\eta_0 = 1- \eta_i$). Inhalability is the ratio of particles with a particular aerodynamic diameter inspired into the respiratory tract, through nose or mouth, to the number concentration of particles with the same aerodynamic diameter present in the inspired volume of ambient air. In the conditions of the indoor environment, most of the radon progeny are generally unattached or attached to submicrometer sized particles, and thus, their inhalability is equal to unity and $\eta_0 = 0$.

In the model, each filter has two characteristic parameters : its volume v_j and its overall efficiency η_j for removing airborne particles. Taking these parameters into account, the fractional deposition in the individual filters is obtained by means of a recursive expression. By this way, it is possible to model the deposition of inhaled activity within the respiratory tract. For lung dosimetry of radon progeny in indoor air, it appears that diffusional deposition in regions BB and bb is the most important deposition mechanism.

On the other hand, it should be mentionned that inhaled radon progeny may be hygroscopic on entering the saturated conditions of the respiratory tract airways and that it may be expected to grow in size to about twice their original size. In recent deposition and dosimetry calculations, the general effect of particle hygroscopic growth during inhalation appears to be to reduce the velocity of particles and therefore to reduce deposition and doses in the bronchial region.

Clearance

In the bronchial tree, radon progeny are assumed to be deposited on the surface of the mucus layer. They can be removed toward the throat by the normal process of mucociliary clearance, with the additional possibility of absorption through the epithelium and elimination by the bloodstream. In the alveolar-interstitial (AI) region, radon progeny are deposited onto a thin layer of surfactant fluid in close contact with blood capillaries.

Direct measurements of the elimination of Rn-220 (thoron) progeny from human and animal lungs have been made. In contrast, elimination of the Rn-222 progeny cannot be measured directly since it is difficult to assess their initial deposition and radioactive transformations.

For dose calculations, most of the models assume that radon progeny are relatively insoluble and are cleared with mucus. In all the models, the absorption half-time is long compared with the radioactive half-lives of radon progeny : 3 minutes for Po-218, 27 minutes for Pb-214 and 20 minutes for Bi-214. However, Jacobi and Eisfeld (1980) have modelled the effect on lung dose of absorption into the blood. In this model, which applies to the conditions of mining exposure, the unattached fraction is absorbed rapidly. However, the unattached fraction for mine conditions is only 1 to 5%, and this rapid absorption does not affect too much the dose.

Dosimetry

Calculation of the dose to the alveolar-interstitial (AI) region is given by the following expression (James, 1988) :

$$D_{AI} = nE_{\alpha} / m_{AI}$$

where n is the number of alpha decays, E_{α} is the alpha-particle energy, and m_{AI} is the mass of the A-I region. This simple expression is valid, because in the alveolar-interstitial (AI) region, the interalveolar septa and the walls of blood and lymphatic capillaries are sufficiently thin to ensure that sensitive target cells are uniformly distributed throughout the tissue mass. The average dose received by the target cells may therefore be assumed to be the same as that received by the whole tissue mass.

Calculation of absorbed dose in the bronchial (BB) and bronchiolar (bb) region is more complicated. Dose calculations have been made using various levels of sophistication ranging from those using a very simple approach in which the total energy deposited in some arbitrary mass of lung tissue to detailed calculations where doses to specific cells at define tissue depths in each airway are calculated, taking into account clearance mechanisms.

For dosimetric modelling, the identification and spatial distribution of target cells is of major importance. One of the most important reason is that the dose to the lung due to Rn-222 progeny is due entirely to the short-lived progeny because they decay shortly after being deposited in the respiratory tract, whereas radionuclides with longer half-lives have more time to be cleared and do not release all, or any, of their potential radiation energy in the lung tissues. Since the energy of alpha particles is deposited along the short path of the particle in human tissues and fluids (47 μm and 71 μm for Po-218 and Po-214 alphas, respectively), the dose to lung tissues come entirely from the emissions ultimately released by Po-218, Pb-214, Bi-214, and Po-214.

In order to determine the dose received by the target cells, whether basal or secretory cells, from the sources, the decaying radon progeny, a cylindrical model is used for bronchi (BB region) and bronchioles (bb region) in which the positions of the target and the sources are represented.

Three main lung dosimetry models have been used to calculate doses from inhaled radon progeny, which are : (i) the Jacobi-Eisfeld model (Jacobi and Eisfeld, 1980), (ii) the James-Birchall model (James et al. (1980) and, (iii) the Harley-Pasternak model (Harley and Pasternak, 1982), complemented by the Harley model (Harley et al., 1996).

In all these three models, the principal target cells are considered to be the basal cells of the bronchial epithelium. The main differences between the models are in the chosen lung morphometry models, the depth of the chosen target cells and the assumptions regarding the deposition and clearance of short-lived radon progeny.

In the Harley-Pasternak model, clearance is by mucus only and uptake to the blood is ignored. In contrast, uptake to the blood is included in both the Jacobi-Eisfeld and James-Birchall models. In the Harley-Pasternak model, doses are calculated at a fixed depth of 22 μm below the surface in the first 10 bronchial generations and at 10 μm beyond the 10th generation. In their recent model, Harley et al. (1996) calculated the dose to the basal cells from the bronchial region. Measurements on surgical specimens of bronchi (airway generation 3 to 6) from over 100 persons resulted in a mean basal depth of 27 μm. In these measurements, the basal depth was from the midpoint of the nucleus to the free epithelial surface. In contrast, the variable depth of target cells is taken into account in both the Jacobi-Eisfeld (1980) and James-Birchall (1980) models.

The effective dose per unit exposure from radon progeny

The doses per unit exposure to radon progeny potential alpha energy P_p are expressed in terms of absorbed dose (mGy / WLM). In order to calculate effective doses E from absorbed doses to the lung, it is necessary to use appropriate weighting factors which are essentially :

(i) a radiation weighting factor W_R (the former quality factor Q). For alpha particles, the recommended value given by *ICRP Publication 60* (1990) is : $W_R = 20$.

(ii) a tissue weighting factor W_T. For the lung, the value of W_T recommended by *ICRP Publication 60* (1991) is 0.12. In its new respiratory tract model, ICRP recommends that separate weighting factors (apportionment factors) are to be assigned for the partition of detriment among the various respiratory tract tissues, as indicated in Table 1.

Table 1 - Weighting factors assigned for the partition of radiation detriment among respiratory tract tissues

Tissue	Assigned fractions
Extrathoracic region **($W_T = 0.025$ from remainders)**	
- ET1 (anterior nose)	0.001
- ET2 (posterior nasal passages, larynx, pharynx, and mouth)	1
- LN_{ET} (lymphatics)	0.001
Thoracic region **($W_T = 0.12$)**	
- BB (bronchial)	0.333
- bb (bronchiolar)	0.333
- AI (Alveolar-interstitial)	0.333
- LN_{TH} (lymphatics)	0.001

When the new ICRP respiratory tract model is applied to calculate the effective dose per unit exposure in mines, the resulting effective dose per unit exposure is between 2 to 3 times that derived from epidemiological studies. Dosimetry estimates are around 15 mSv per WLM, while epidemiological estimates are around 5 mSv per WLM (Birchall and James, 1994). For typical underground uranium mines conditions, the value of the dose conversion coefficient, E / Pp, has been found to be 13.4 mSv per WLM.

The ICRP has resolved the incompatibility between the two approaches using an alternative method of deriving E / Pp. First, the estimated number of excess lung cancers in miners per unit exposure, projected directly from uranium miner epidemiology, is divided by the predicted excess lung cancer per unit dose. This latter quantity is projected

from lung cancer incidence per unit of whole body gamma dose obtained from the Japanese survivors of the atomic bombs.

This method leads to a so-called dose-conversion convention of about 5 mSv per WLM for workers, assuming 2000 hours per year at work and an equilibrium factor of 0.4 (ICRP, 1994 a). For members of the public, the corresponding conversion convention is 3.88 mSv, assuming 7000 hours per year indoors and an equilibrium factor of 0.4. These conversion conventions are based on equality of detriment and not on dosimetry.

An uncertainty analysis of the effective dose per unit exposure from radon progeny has been performed by Birchall and James (1994) to determine whether the uncertainty in parameter values in the dosimetry approach can account for the discrepancy with the epidemiological approach. Their conclusion was that if values of 20 and 0.12 are used for the dosimetric risk weighting factors, W_R and W_T, respectively, it is unlikely that the effective dose per unit exposure to radon progeny, E / Pp, in an underground mine could be as low as the value determined from the uranium miner epidemiological data, i.e., 5 mSv per WLM. The uncertainty analysis suggests that the three-fold discrepancy between the dosimetric and epidemiological risk estimates is unlikely to be explained by uncertainties in the dose calculation alone, i.e, in aerosol conditions, lung model parameters, target cell assumptions. The implication of the analysis is that there may be systematic errors in at least one of the ICRP recommended values for the risk "weighting factors" W_T, W_R or DDREF. Furthermore, if this implication is accepted for radon progeny exposure, then it must be considered for any inhaled alpha emitting radionuclides, such as plutonium isotopes.

Comparison between the different dosimetry models and the influence of modelling variables

Over the last 10-15 years, all of the radon dosimetry models have tended to predict a larger dose per unit exposure. The reasons for this are that, despite the complexity of lung dosimetry modelling, the resulting dose seems to be fairly robust and insensitive to many of the assumptions. The dose estimates obtained using the various models for different particle sizes, unattached fractions and breathing volumes usually agree within a factor of approximately 2 within the normal range of these factors.

However, the use of the ICRP 66 respiratory tract model increases the weighted equivalent lung dose per WLM (H_W / P_p) by a factor of approximately 2 compared with that of both the Jacobi-Eisfeld and James-Birchall models (Marsh and Birchall, 1998).

The ICRP 66 respiratory tract model considers secretory cells as well as basal cells in the bronchial region. The introduction of the secretory cells in the BB region increases the dose to the BB region by a factor of 1.6 and increases the equivalent lung dose H_W / P_p by a factor of 1.2 (Marsh and Birchall, 1998).

Concerning the depth of target cells, defined as the distance to the cells from the bottom of the mucus gel layer, differences exist between the different models. In the BB region, the depth of basal cells is considered to be 41 μm in the ICRP 66 model, 20 to 38 μm in the Jacobi-Eisfeld model and 25 to 85 μm in the James-Birchall model. In the bb region, the depth of secretory cells is considered to be 8 μm in the ICRP 66 model, the depth of basal cells 15 to 25 μm in the Jacobi-Eisfeld model, whereas the stem cells are considered to be distributed throughout the depth of the epithelium in the James-Birchall model. If the depth of secretory cells in the bb region was 18 μm instead of 8 μm of the

ICRP 66 model, then the absorbed dose to the bb region decreases by 0.76 and the equivalent lung dose H_W / P_p decreases by 0.87 (Marsh and Birchall, 1998).

The ICRP Publication 66 assumes an assigned fraction (A) of tissue weighting factor of 0.333 for each of the three regions of the lung : BB, bb and A-I. Thus, this is equivalent of an A values of 0.666 for the TB region and 0.333 for the pulmonary region, whereas the ICRP 32 model assumes 0.5 for the TB region and 0.5 for the pulmonary region. Therefore, the use of 0.666 instead of 0.5 for the TB region is to increase the dose by a factor of 1.3 (Marsh and Birchall, 1998).

A number of studies have been performed on the effect on dose calculations of changes in parameters of unattached fraction and aerosol particle size distribution. Reinecking et al. (1992) made measurements of aerosol size characteristics, unattached fractions, and equilibrium factors of short-lived radon and thoron progeny under realistic conditions in houses and outdoors which were used for dose calculations. The main conclusion of this study was that dose calculations, based on James (1988) and Jacobi-Eisfeld (1980) models, were less influenced by aerosol characteristics compared to model assumptions concerning the type and location of target cells, uncertainties in extrathoracic filtration (nasal and oral filtration efficiencies), and model estimates of regional deposition probabilities.

Conclusion

The relationship between exposure to airborne short-lived radon progeny and doses to the cells considered to be at risk for the induction of cancers in the human respiratory tract is extremely complex. There are no major fundamental conflicts between the current models available including the new ICRP respiratory tract model. For most given exposure conditions, all the models produce dose values which agree within a factor of two. The difference that exist between the models arises from the identification of target cells, the choice of morphological model and physiological parameters.

Further research is needed to reduce the uncertainties in lung dosimetry modelling. The main areas of uncertainty that need to be addressed are the activity weighted size distributions of radon progeny in workplaces and homes, the hygroscopic growth of aerosols in the respiratory tract (aerosol parameters), the intersubject variability in lung morphology and physiology, oral and nasal breathing patterns in miners and in public, deposition patterns in the respiratory tract (subject related parameters), the identification of cells at risk and their importance in lung carcinogenesis (target cell parameters), the physical behaviour of radon progeny deposited in mucus and the absorption rates of radon daughters (solubility and clearance rates) and the values of the "Risk Weighting Factors" W_T , W_R and DDREF that are appropriate to alpha irradiation of the lung by radon progeny in indoor or occupational situations.

Given the extremely wide range of aerosol size distributions found in the natural environment, in dwellings and at the workplace, the wide variety of physical and chemical forms of aerosol particles that carry radon progeny - which affect their hygroscopic growth -, and the equally wide range of respiratory patterns, it is difficult to imagine that individual exposures and doses will be known with a better accuracy in the future. However, a better characterization of the parameters that govern aerosol penetration in the airways, their deposition and their clearance rate would allow to

establish a realistic overall range of uncertainty in the exposure-to-dose conversion factors, both for workers and the general population.

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RADON EPIDEMIOLOGY: STATE-OF-THE-ART, WITH EMPHASIS ON DOMESTIC EXPOSURE

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Abstract

There is a substantial amount of information on the risk of lung cancer associated with exposure to radon in mines. In contrast, up until recently there has been little direct evidence of the risks from exposure to radon to homes. However, a large number of studies of lung cancer and indoor radon have now been undertaken or are nearing completion. Combined analyses of these data will allow comparison with risk estimates based on the miner studies.

Studies of miners

The main epidemiological evidence for the role of radon exposure in the induction of lung cancer comes from a multitude of studies of miners of uranium, tin and iron ores, conducted mostly in Europe and North America[1,2]. The total number of men included in these studies is over 60,000, with cumulative exposures varying over a very wide range. The observed number of lung cancers is around 2,600 in total, far in excess of the value expected from national or regional rates of around 750; indeed, each study shows a statistically significant excess[2]. A combined analysis of eleven miner studies[1,3] indicates that the lung cancer risk increases linearly with increasing cumulative exposure. The exposures at which some of the studies yield a significantly raised risk are only about a factor of two to five above the mean lifetime indoor exposure received in countries such as the UK and the USA, and are less than the lifetime exposure from a radon concentration of 200 Bq m^{-3} (ie. equal or close to the action level in several European countries)[2].

While other carcinogens were sometimes present in mines, the associations between lung cancer and radon have been seen in a wide range of mines, including those where respiratory carcinogens such as arsenic were absent. Further evidence comes from animal studies in the US and France, which show similar patterns of increased risks of lung cancer in rats and dogs exposed chronically to radon[4]. Based on such information, the International Agency for Research on Cancer[5] has concluded that there is sufficient evidence to classify radon as a carcinogen in humans.

The International Commission on Radiological Protection (ICRP)[6] and other organisations such as the fourth US Committee on the Biological Effects of Ionising Radiation (BEIR IV)[4] have used the miner studies to estimate the risk over a lifetime for a general population exposed to radon in an indoor environment. Based on models derived from the miner data that utilise a measure of cumulative exposure called the Working Level Month (WLM)*, ICRP has recommended a risk factor for fatal lung cancer in the UK of 3 per 10,000 per WLM, based on a reference world population of all ages and both sexes[6]. This risk factor, which is similar to the BEIR IV value for a US

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* Technical terms are defined in the Glossary.

population[4], implies that lifetime exposure in a dwelling at 200 Bq m^{-3} would increase the risk of fatal lung cancer by around 50% relative to the baseline risk.

ICRP has also adopted a dosimetric model of the respiratory tract[7], the practical applications of which are still being developed. It has been shown that, by combining lung doses calculated using this model with risk factors for low-LET radiation from the Japanese atomic bomb survivors, the estimated risk of radon-induced fatal lung cancer is greater than that based on the miner data by a factor of about two to three[8]. However, the former "dosimetric" calculation involves several sources of uncertainty that do not enter the latter "epidemiological" approach based on the miner data; for example, in the tissue weighting factor for the lung for low-LET irradiation and in the radiation weighting factor for alpha radiation[6,8]. ICRP has therefore concluded that the "epidemiological" approach using the miner data is more direct and is to be preferred for the purposes of estimating radon risks[6].

There are, however, some uncertainties in using the miner data to calculate risks from exposure in dwellings. These include: the conversion of doses from a mining to an indoor environment; the effect of smoking on radon risks; the lack of information on risks to females and from exposure in early life; and the effect of protracting exposure over many years. Some of these issues are amenable to analysis using the miner data. For example, the joint effect of radon and smoking on lung cancer risks appears to be greater than the sum of the individual components (ie. risks from radon are greater among smokers than non-smokers), although there is some uncertainty about whether the risk is a multiple of the radon and smoking components or somewhat less than this[1]. Furthermore, for a given total exposure, the combined analysis of miner studies suggests that the risk is higher if the exposure is protracted over decades rather than received over a short time[9]. However, this effect appears to diminish and possibly disappear at low exposures[9].

Research to examine these topics using miner data is continuing. For example, the European Commission is currently funding work to obtain a better understanding of lung cancer risks from protracted exposures based on data for uranium miners in France, the Czech Republic, and the large group of Wismut miners in Germany. In addition, a new Committee on the Biological Effects of Ionising Radiation (BEIR VI) that was convened by the US National Research Council will shortly issue a report on radon risks, largely based upon analyses of miner data. However, in addition to these data, direct information on the effects of indoor radon is now being obtained from epidemiological studies of such exposures.

Epidemiological studies of lung cancer and indoor radon exposure

Methodological Issues

Various case-control studies have been conducted or are in progress throughout Europe and North America. These studies involve assessments of radon exposures in dwellings for persons with and without lung cancer, plus the collection of data on potential confounding factors, the most important of which is smoking. In contrast to case-control studies, in which data on radon and smoking are obtained for specific individuals, some geographical studies have been conducted in which attempts were made to correlate lung cancer rates averaged over geographical areas with average radon levels for the same areas[10,11]. However, such geographical correlation studies are vulnerable to bias[12,13], ie. to give results that are systematically in error. The potential for bias is particularly important here because smoking is a very strong determinant of lung cancer risk. The absence of

individual smoking information in geographical studies means that, even when data on average smoking rates in areas are available, it is not possible to make adequate adjustment for smoking effects. Furthermore, the data in geographical correlation studies do not take account of, for example, how long someone has lived in a house and what exposures may have been received in previous dwellings. For methodological reasons such as these, it has been concluded that geographical correlation studies are uninformative about lung cancer risks in relation to indoor radon[12].

Although methodologically more sound than geographical correlation studies, case-control studies of indoor radon and lung cancer are not always easy to interpret. This reflects largely the low statistical power of many of the studies, given that the radon exposures and predicted risks are generally lower than in the miner studies. Also of importance are uncertainties in the assessment of domestic exposures. These can arise, for example, from the use of contemporary measurements to characterise past radon levels, imprecision in the percentage of time spent in a dwelling or particular rooms thereof, and possibly a lack of information on levels in past homes occupied within the previous two or three decades (eg. if access to the dwelling is denied or if it no longer exists). It has been shown that uncertainties in assessed exposures can reduce statistical power[14] and lead to biased estimates of risk[15]. While the latter problem can be addressed through appropriate statistical analyses[15], the effect on statistical power may mean that individual studies - unless they are very large - lack the precision required to make meaningful comparisons with the results of the miner studies. As a consequence, there is particular interest in synthesising the results from different studies.

Results from studies to date

A meta-analysis of eight case-control studies of lung cancer and indoor radon has recently been conducted[16]. Details of these studies are shown in Table 1. It can be seen that these studies varied in the numbers of subjects, the levels of radon and, to some extent, in the level of risk, although the associated confidence intervals are often wide. In the meta-analysis, published findings from these studies were combined in order to arrive at an overall estimate of risk. Based on a total of over 4,000 lung cancer cases and 6,000 controls, there was a statistically significant increase in risk with increasing radon levels[16] (p-value of 0.03), as shown in Figure 1. In particular, the combined estimate of the relative increase in risk from residence in a dwelling with a concentration of 200 Bq m^{-3} can be estimated as 19% (95% confidence interval (CI) from 1% to 42%). No single study dominated the combined results. It can also be seen from Figure 1 that the results from this analysis of the indoor radon case-control studies are consistent with the level of risk predicted from the miner studies (assuming an average of 25 years exposure in the homes considered in the indoor studies). In contrast, the findings from geographical correlation studies differ[11] considerably from those of the case-control and miner studies, so underlining the methodological deficiencies of geographical studies.

Ongoing studies and combined analyses

A range of other studies of indoor radon and lung cancer are now being completed in Europe and North America[17]. Among the largest studies in Europe are:

(i) a study initially of around 1,500 cases and 2,300 controls in western Germany (described in more detail by Professor Wichmann during the seminar) which will

subsequently increase to cover about 2,500 cases and 2,500 controls, plus data on over 1,500 cases and a similar number of controls in Eastern Germany;

(ii) a multi-centre study of about 1,200 cases and twice as many controls performed in the Ardennes-Eifel region covering Belgium, France, Germany and Luxembourg[18];

(iii) a study of around 1,000 cases and 3,000 controls conducted in the south-west of England;

(iv) a study in several regions of France, covering approximately 600 controls and twice as many controls.

Each of the above studies has focused on areas with known high radon levels, so as to maximise statistical power. In order to arrive at more precise conclusions, the European Commission is currently supporting initiatives to combine studies in Europe. In particular, a European Collaborative Group on Residential Radon and Lung Cancer met in Oxford in early 1997, and it is planned to assemble a combined dataset with around 9,000 lung cancer cases, scheduled for analysis in 1999. A similar pooling exercise is also in progress in North America[17], again based on data for several thousand cases. These analyses differ from the meta-analysis described above in that they will make use of the data for individuals in the studies (their ages, radon exposures, smoking habits, etc), rather than summaries of findings quoted in publications. Consequently, the combined analyses will allow the comparability of the studies to be examined in more detail and will make it possible for data to be analysed in a uniform manner. The participation of the original investigators in such a task is highly important.

Conclusions

The substantial amount of data on lung cancer among underground miners is likely to continue to form the basis of radon risk estimates for the foreseeable future. However, combined analyses of case-control studies of indoor radon will allow the validity of these estimates to be examined. To date, the domestic and miner findings are in broad agreement.

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Table 1

Summary of Eight Completed Case-control Studies of
Lung Cancer and Indoor Radon (after Lubin and Boice[16])

Study	Cases	Controls	Mean radon concentration (Bq m^{-3})	Relative risk (95% CI) at 200 Bq m^{-3}
Finland-I	238 (males)	434	220	1.42 (1.12-1.79)
Finland-II	517 (males and females)	517	103 (cases) 96 (controls)	1.01 (0.92-1.12)
New Jersey (USA)	433 (females)	402	26	2.24 (1.20-4.14)
Shenyang (China)	308 (females)	362	118	0.79 (0.72-0.88)
Winnipeg (Canada)	738 (males and females)	738	120	0.95 (0.82-1.11)
Stockholm (Sweden)	210 (females)	400	128	2.24 (1.48-3.39)
Sweden (nationwide)	1281 (males and females)	2576	107	1.28 (1.18-1.38)
Missouri (USA)	538 (females)	1183	67	1.16 (0.89-1.51)
Combined	4263	6612		1.19 (1.01-1.42)

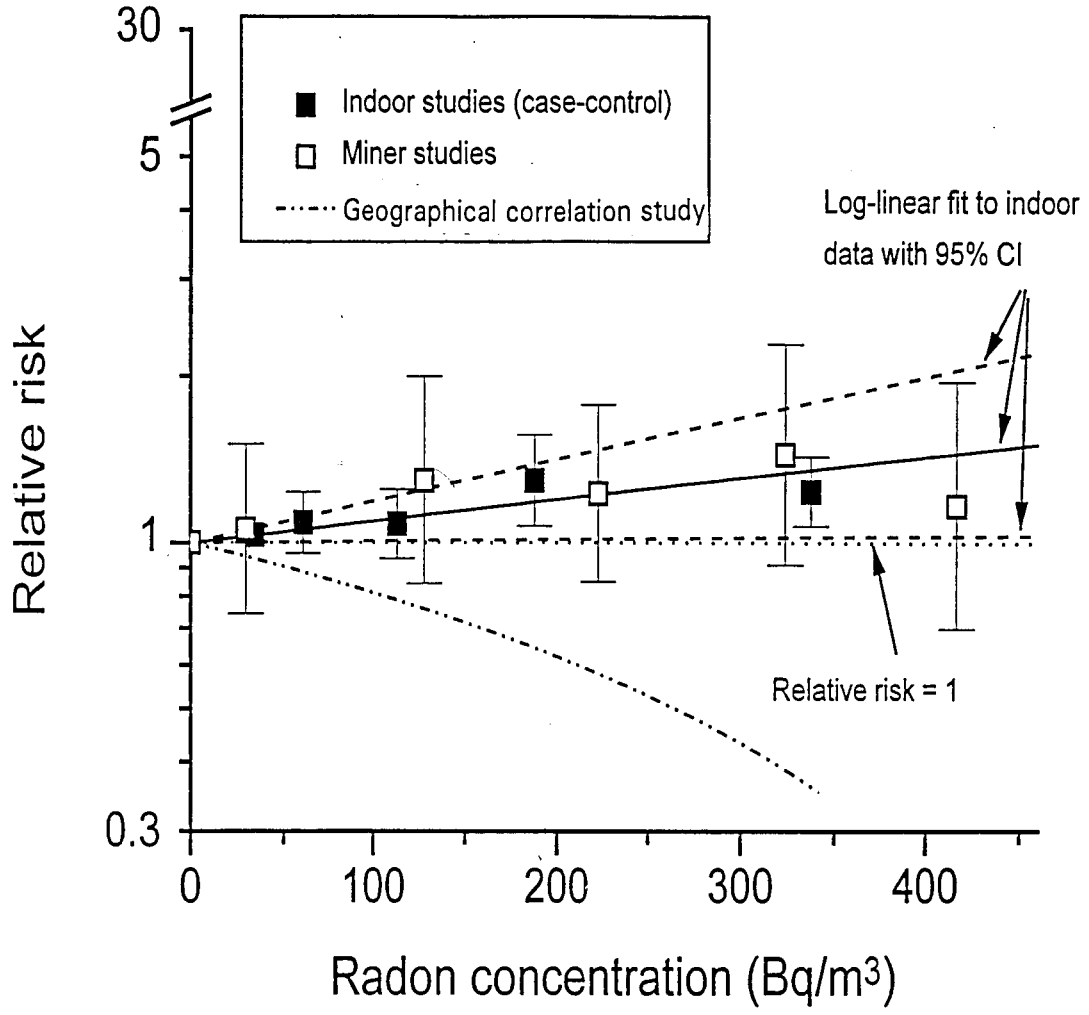

Figure 1 Risks of lung cancer in relation to radon, based on: a combined analysis of miner studies (restricted to exposures under 50 WLM)[3]; a meta-analysis of eight indoor radon case-control studies[16]; and the geographical correlation study of Cohen[11].

GLOSSARY

Case-control study	An investigation into the extent to which a group of persons with a specific disease (the cases) and comparable persons without the disease (the controls) differ with respect to exposure to putative risk factors.
Confidence interval	An interval calculated from data when making inferences about an unknown parameter. In hypothetical repetitions of the study, the interval will include the parameter on a specified percentage of occasions (eg. 95% for a 95% confidence interval).
Confounder	A factor that is correlated with the exposure of interest and, independently, is related to the disease under investigation.
Geographical correlation study	An attempt to correlate disease rates averaged over geographical areas with levels of exposure averaged over the same areas.
Meta-analysis	A synthesis of results from different studies, usually based on summary findings reported in publications.
p-value	The probability that a result as extreme as that observed would have occurred in the absence of a true effect. A result is often said to be statistically significant if the p-value is less than 0.05.
Relative risk	The ratio of disease rates in exposed and unexposed groups, usually adjusted for factors such as age and sex.
Statistical power	The probability that, with a given degree of confidence, an underlying effect of a certain magnitude will be detected in a study.
WLM	Working Level Month: a measure of cumulative exposure to radon, used in studies of miners. One WLM equates to exposure to $2.1\ 10^{-5}$ J m^{-3} for a working month of 170 hours, or to a year's exposure in a house with a radon concentration[19] of 144 Bq m^{-3}.

RESULTS OF THE WESTERN GERMAN CASE-CONTROL STUDY ON INDOOR RADON AND LUNG CANCER IN THE CONTEXT OF OTHER EPIDEMIOLOGICAL STUDIES

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Abstract

In a case-control study in Western Germany the lung cancer risk due to exposure to radon was investigated. From 1990 to 1996 incident lung cancer patients (histologically/cytologically confirmed) (cases) as well as a random sample recruited from the population (controls) were interviewed in participating lung clinics according to their residential history, smoking, occupational exposure and other risk factors. Two charcoal canisters and two alpha track detectors (exposure time 3 days, 1 year respectively) were placed in the present and former dwellings of the participants. On the one hand the evaluation included 1449 cases and 2297 controls recruited from the entire study area (parts of North Rhine Westphalia, Rhineland Palatinate, the Saar and Eastern Bavaria), on the other hand 365 cases and 595 controls recruited from radon-prone matching areas (Eifel, Hunsrück/Westerwald, Upper Palatinate/Lower Bavaria).

Odds ratios were estimated according to a logistic regression model simultaneously adjusted for smoking and exposure to asbestos, furthermore the linear trend was calculated. In the entire study area no odds ratios were different from one, in the radon-prone matching areas a clear influence of exposure to radon on the lung cancer risk was observed. The adjusted odds ratios [95 %-CI] referring to the long time measurement in the last dwelling were 1.59 [1.08, 2.27], 1.93 [1.19, 3.13] and 1.93 [0.99, 3.77] for 50 - 80, 80 - 140 and over 140 Bq/m^3 compared to 0 - 50 Bq/m^3. The odds ratio for the linear trend was 1.13 [0.88, 1.46] for an increase in exposure of 100 Bq/m^3. Other quantifications of exposure show similiar results.

The results in the radon-prone matching areas are compatible with the so far largest Swedish study, with the result of a meta-analysis including eight important published studies, and with risk models from the analysis of cohorts of miners. The absence of an exposure-effect-relation in the entire study area may be explained by misclassification since there is a high percentage of dwellings with low exposure.

Introduction

Studies on miners show an increased lung cancer risk after exposure to radon and radon daugthers. They suggest that exposure to indoor radon also may be relevant and may contribute to the incidence of lung cancer in the general population. Some direct epidemiological investigations from other countries indeed show such a relationship. However, these results are not consistent and the statistical power of many studies is

limited. For Western Germany it has been estimated earlier that 4 - 12 % of all lung cancers could be due to indoor radon (see BMU 1992). This figure was confirmed by more recent estimates (7 %, Steindorf et al. 1995).

From 1990 to 1996 a case control study has been performed in Western Germany which has the aim to find direct evidence for a radon related risk in the German population. This study was funded by the Federal Ministry of the Environment (Bundesministerium für Umwelt, Naturschutz und Reaktorsicherheit). The German report (Wichmann et al. 1997b) desribes the study design, the field work, the results, the risk estimates and the discussion. A second study for Eastern Germany is on the way. Thus, this report is only the first step for the evaluation of the role of radon in Germany.

Material and Methods

The study took place in radon-prone areas as well as in areas with lower radon concentrations in Western Germany. It is a case-control study with incident lung cancer cases (interviewed within 3 month after diagnosis) and population-based controls under 75 years. Both groups participated in face to face interviews by trained interviewers. An extended part dealt with residential history, housing conditions, reconstruction of the house and ventilation habits. Furthermore, questions on active and passive smoking, occupational exposure, nutrition, leisure activities and the personal and familial medical history were asked. Radon was measured in the present home and in past homes.

Cases and controls were frequency matched by sex, age and area. The cases were interviewed in the participating lung clinics, the controls were interviewed at home. The latter were recruited either via the official mandatory registries or by random digit dialing.

Radon measurements were performed in the present home and in the past homes (identified via telephone and mandatory registries) up to 35 years back. In the living room and the bedroom charcoal canisters were exposed for three days and alpha track detectors for one year. In the following only homes are considered for which complete alpha track measurements are available.

The following areas belong to the entire study area: Southern Nordrhine-Westfalia, Rhineland-Palatinate, Saar and Eastern Bavaria. Since about half of the study area has only low radon concentrations, in addition radon-prone matching areas are considered separately. These are Eifel, Westerwald/Hunsrück and Upper Palatinate/Lower Bavaria. A matching area is classified as radon-prone if most of it belongs to a zone with high geological radon potential, due to the classification of Kemski et al. (1996).

Radon exposure of the participants was quantified in two ways. In the first approach a weighted average of the radon concentrations in the living room and the bedroom of the present home was calculated, taking into account the time spent in both rooms. The second approach considers the cumulative exposure 5 - 15 years before interview, which is the most relevant time interval with respect to the lung cancer risk due to radon. Here in addition to the measurements in the present homes the concentrations in the past homes were used, corrected by changes due to reconstruction of the house or different ventilation habits of the participants and the present inhabitants.

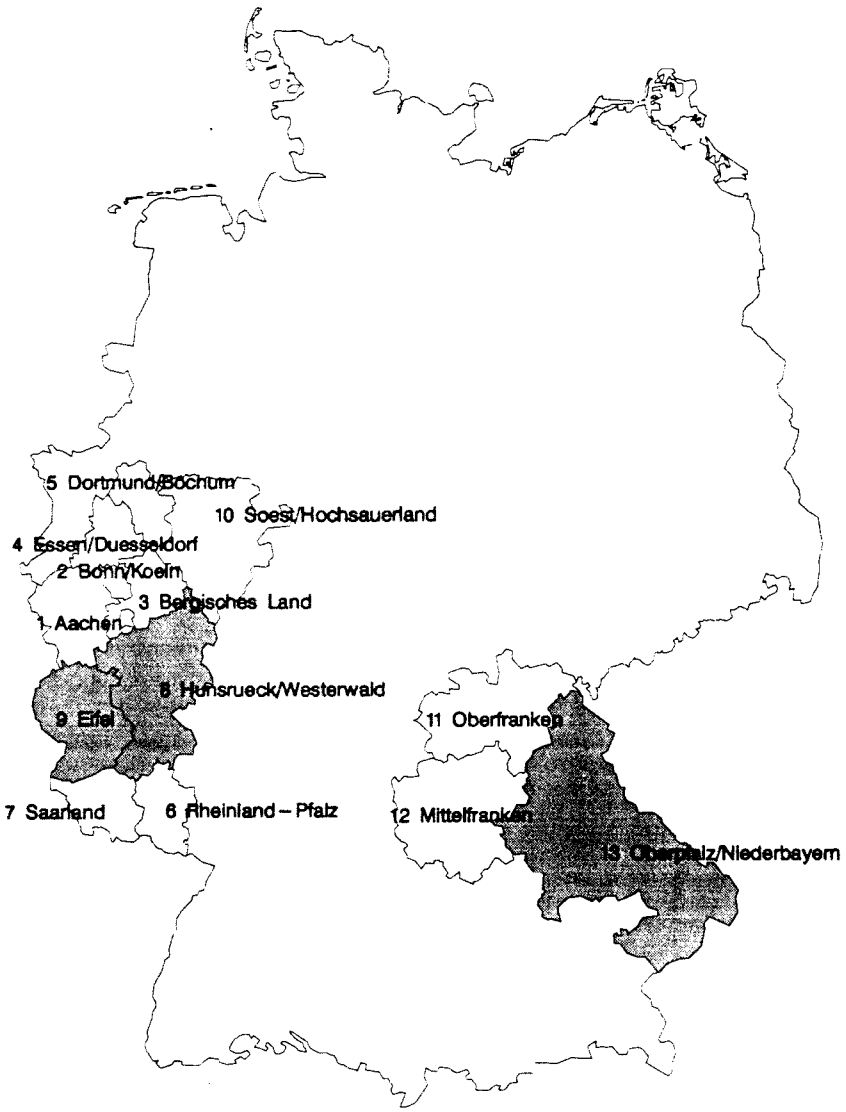

Figure 1: Entire study area and radon-prone matching areas (grey shaded)

Smoking was quantified based on detailed information from the questionnaire. Lifelong smoking history was asked in phases of constant smoking habits, for cigarette smoking and other types of smoking material. Active smokers and exsmokers are summarized as smokers and compared to never smokers. In the analysis years of smoking, age at the beginning of smoking, average cigarettes per day, packyears and time since cessation are considered.

For all participants a complete occupational history has been asked. For each new job at the same or a different employer a new „occupational phase“ started. Occupational exposures were evaluated on the basis of additional job-specific questionnaires and the lifelong history of all jobs and industries was coded. For the present analysis, only exposure to asbestos (yes/no) is considered, more detailed occupational analyses are described separately (Wichmann et al., 1997a).

All cases have a histopathologically or cytologically verified tumor of the lung. The following subtypes are considered: small cell (oat cell) carcinoma, squamous cell carcinoma, adenocarcinoma and other bronchial cancers.

In the statistical analysis odds ratios were calculated using a logistic model where matching is considered by strata for age, sex and area. The cutpoints for radon have been chosen as in the study of Pershagen et al. (1994) at 50, 80 and 140 Bq/m^3. The highest cutpoint of that study at 400 Bq/m^3 has been omitted due to too few highly exposed homes. In addition the linear trend has been calculated.

Results

In the entire study area 2527 cases and 3439 controls have been interviewed, with response rates of 79 % and 38 % respectively. For the present analysis only participants with complete questionnaire information and complete long-term radon measurements are considered, which reduces the numbers to 1449 and 2297. In the radon-prone matching areas the numbers are roughly one third to one quarter of the entire study area.

The radon concentrations (weighted average of the one year measurements in living room and bedroom) show a lognormal distribution. In the entire study area the mean concentrations are 49 Bq/m^3 for cases and 50 Bq/m^3 for controls, and in the radon-prone matching areas they are 67 and 60 Bq/m^3 respectively (Table 1). The maximum concentration is 922 Bq/m^3.

Table 1: Average radon concentrations in Bq/m^3 in entire study area and radon-prone matching areas

	Entire study area		radon-prone matching areas	
	cases	controls	cases	controls
n	1449	2297	365	595
Min	8	8	10	11
Max	760	922	455	922
Med	40	40	52	46
Mean	49	50	67	60
Std Dev	41	47	50	66
CV %	85	95	75	110
geom Mean	40	41	55	48
geom Stddev	2	2	2	2

For the entire study area no increased lung cancer risk is found. This holds true for the logistic regression with and without adjustment for smoking (log (pack years +1), years since quitting in categories) and asbestos (exposed vs. not exposed), as well as for the linear trend test (Table 2). In contrast, for the radon-prone matching areas an exposure response relationship is found (Figure 2, Table 2). The OR is increased in all categories, compared to the reference category of 0 - 50 Bq/m^3, and the increase is slightly stronger after adjustment. The linear trend test shows an elevated OR of 1.13 for an increase of the radon concentration by 100 Bq/m^3.

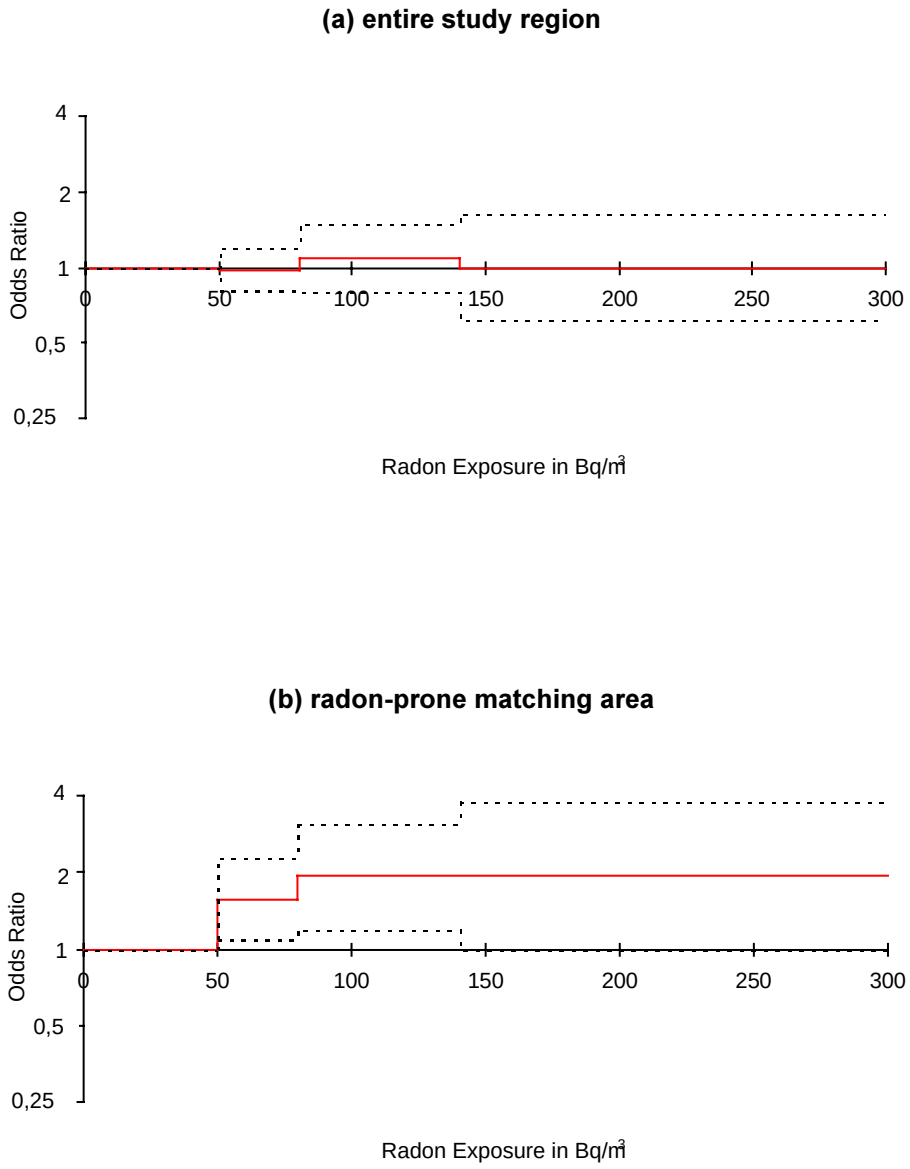

Figure 2: Lung cancer risk due to indoor radon in Western Germany - Odds Ratios by average radon concentration in last dwelling

If one considers the cumulative radon exposure during the last 5 - 15 years, for the entire study area also no elevated OR is found, although the ORs are slightly higher than based on the present home (Table 2). For the radon-prone matching areas again a positive exposure response relationsship is observed, which also is slightly higher than based on the present home (Table 2).

Table 2: Lung cancer risk due to radon in dwellings in Western Germany

	cases		controls			
	n	%	n	%	OR[2,3)]	95 % CI
Radon in Bq/m^3 [1)] from over … to …	Radon concentration in present dwelling **Entire study area**					
0-50	1022	70.5	1542	67.1	1.	
50-80	284	19.6	512	22.3	0.98	(0.81, 1.20)
80-140	108	7.5	178	7.7	1.09	(0.80, 1.48)
>140	35	2.4	65	2.8	0.99	(0.61, 1.63)
Total	1449	100.0	2297	100.0		
Trend [4)]					0.98	(0.82, 1.17)
Radon in Bq/m^3 [1)] from over … to …	Radon concentration in present dwelling **radon-prone matching areas**					
0-50	178	48.8	345	58.0	1.	
50-80	104	28.5	156	26.2	1.57	(1.08, 2.27)
80-140	58	15.9	65	10.9	1.93	(1.19, 3.13)
>140	25	6.8	29	4.9	1.93	(0.99, 3.77)
Total	365	100.0	595	100.0		
Trend [4)]					1.13	(0.88, 1.46)
Expos. in Bq/m^3×a [1)] from over … to …	Cumulative radon exposure during the last 5 - 15 years **Entire study area**					
0-20	459	44.9	710	43.7	1.	
20-40	410	40.1	621	38.2	1.16	(0.94, 1.43)
40-60	87	8.5	184	11.3	0.82	(0.58, 1.16)
>60	67	6.5	111	6.8	1.21	(0.81, 1.81)
Total	1023	100.0	1626	100.0		
Trend [4)]					0.97	(0.82, 1.14)
Expos. in Bq/m^3×a [1)] from over … to …	Cumulative radon exposure during the last 5 - 15 years **radon-prone matching areas**					
0-20	56	22.8	122	30.2	1.	
20-40	109	44.3	170	42.1	1.67	(1.04, 2.69)
40-60	40	16.3	66	16.3	1.55	(0.83, 2.90)
>60	41	16.7	46	11.4	2.60	(1.38, 4.93)
Total	246	100.0	404	100.0		
Trend [4)]					1.09	(0.86, 1.38)

[1)] Livingroom measurement substitutes bedroom measurement, should the occasion arise, and vice versa; measurement adjusted dwelling conditions, time-weighted means of living- and bedrooom measurement
[2)] matched by age, sex and region
[3)] adjusted for smoking (log(packyears + 1), years since quitting), asbestos (exposed vs. non exposed)
[4)] Trend test: linear model for continuous variable, unit 100 Bq/m^3

The histopathological subtypes show similar results. For no type an effect is seen for the entire study area and for all subtypes there is a tendency for elevated ORs in the radon-prone matching areas, which mostly is not significant due to small numbers. The strongest effect is observed for small cell carcinoma (Table 3).

Table 3: Lung cancer risk for small cell carcinoma due to radon in dwellings in Western Germany

Radon[1)] in Bq/m^3 from over … to …	cases n	%	controls n	%	Crude Odds Ratios [2)] 95 % C			Adjusted Odds Ratios[2,3)] 95 % CI		
Entire study area										
0-50	215	69.6	1340	67.2	1.			1.		
50-80	59	19.1	440	22.1	0.84	(0.61,	1.15)	0.99	(0.69,	1.42)
80-140	24	7.8	156	7.8	0.90	(0.56,	1.46)	1.01	(0.59,	1.74)
>140	11	3.6	58	2.9	1.12	(0.56,	2.23)	1.48	(0.69,	3.18)
Total	309	100.0	1994	100.0						
Trend [4)]					0.94	(0.72,	1.23)	0.99	(0.77,	1.27)
radon-prone matching areas										
0-50	52	51.5	300	57.9	1.			1.		
50-80	25	24.8	133	25.7	1.20	(0.71,	2.04)	1.35	(0.72,	2.53)
80-140	14	13.9	60	11.6	1.49	(0.75,	2.97)	1.55	(0.70,	3.46)
>140	10	9.9	25	4.8	2.42	(1.07,	5.45)	3.41	(1.33,	8.73)
Total	101	100.0	518	100.0						
Trend [4)]					1.16	(0.89,	1.51)	1.11	(0.83,	1.47)

1) Livingroom measurement substitutes bedroom measurement, should the occasion arise, and vice versa; measurement adjusted dwelling conditions, time-weighted means of living- and bedrooom measurement
2) matched by age, sex and region
3) adjusted for smoking (log(packyears + 1), years since quitting), asbestos (exposed vs. non exposed)
4) Trend test: linear model for continuous variable, unit 100 Bq/m^3

For cigarette smoking, an elevated OR of 16.1 (95 %-CI 10.5 - 24.8) is observed which is nearly unchanged after adjustment for asbestos. For other tobacco products the OR is 2.6 (95 %-CI 1.2 - 5.9). Exposure to asbestos leads to an increased risk after adjustment for smoking of 1.7 (95 %-CI 1.4 - 2.0) for males, for females only 6 cases and no controls were exposed.

To understand the different results of the entire study area and the radon-prone matching areas, these are compared in more detail. The latter have a somewhat higher response rate of the controls (46 % vs. 38 %), but not for the cases (77 % vs. 79 %). Other differences are not pronounced. A different approach to understand this difference is to look at known „risk factors“ of high radon concentrations in homes. The following seven categories can be considered as unfavourable dwelling conditions:

(1) houses in villages below 5000 inhabitants,
(2) houses built before 1900,
(3) half-timbered houses ,
(4) houses with no or partial basement,
(5) houses with basement floor of loam or natural stone,
(6) houses with poor insulation of basement,
(7) infrequent opening of the windows

The cumulative frequency distributions for these subgroups show a shift to higher exposure for cases already for the entire study area. This effect is more pronounced if the radon-prone matching areas are considered and it is also more pronounced if more than one of these radon-relevant criteria is used to define the subgroup.The effect is demonstrated in Figure 3.

Discussion

The analysis is based on 1449 cases and 2297 controls which is the largest size of a single radon study published until now. Nevertheless, already in the planing phase the relatively small fraction of homes with higher radon concentrations in Western Germany was seen as a limiting factor. Therefore it was very helpful that German reunification allowed us to perform a second study in Eastern Germany where more areas with high radon concentrations are located. This second investigation is still ongoing and will have a comparable study size. After it is finished both German studies will be pooled. Until then, further radon measurements in present and past homes will also be finished, and the Western German data set also will be more complete.

In most big case-control studies on lung cancer risk due to radon cases were recruited via cancer registries. This has the advantage of a complete coverage of the study area, but it has the disadvantage that due to the poor prognosis of lung cancer most cases are already dead at the time of the interview and the radon measurements. Thus, one depends on information of next of kin as well as on measurements in homes of other persons.

One limitation of this study is the low response rate of controls. To understand the reasons, for a random sample of nonresponders a short telephone interview was performed (Kreienbrock 1994). It showed that a large percentage did not participate because they did not want to have radon detectors in their homes. Furthermore, it was analysed how socioeconomic status influenced the results. Although better educated people also in this study tended to participate more frequently a bias could not be identified, since the housing characteristics of the responders and the nonresponders did not differ.

Much effort was invested to quantify individual exposure in an appropriate way.Therefore a detailed analysis of the radon concentrations depending on the characteristics of the home and the ventilation habits was performed. Thus it was possible to adjust for differences of the ventilation habits between participants and present inhabitants of the past homes as well as for reconstructions like the change of windows or of the heating system. Furthermore times spent in the different rooms and outside the home were taken into account.

In the study the risk is not elevated for the entire study area. Therefore, it is surprising that a significantly increased risk is seen if radon-prone areas or radon-relevant constallations are considered. This is surprising, because theoretically one would not expect such a discrepancy if the chosen quantification of radon exposure would completely correspond to the real exposure: The results should not change if participants living in homes with low exposure were added or not.

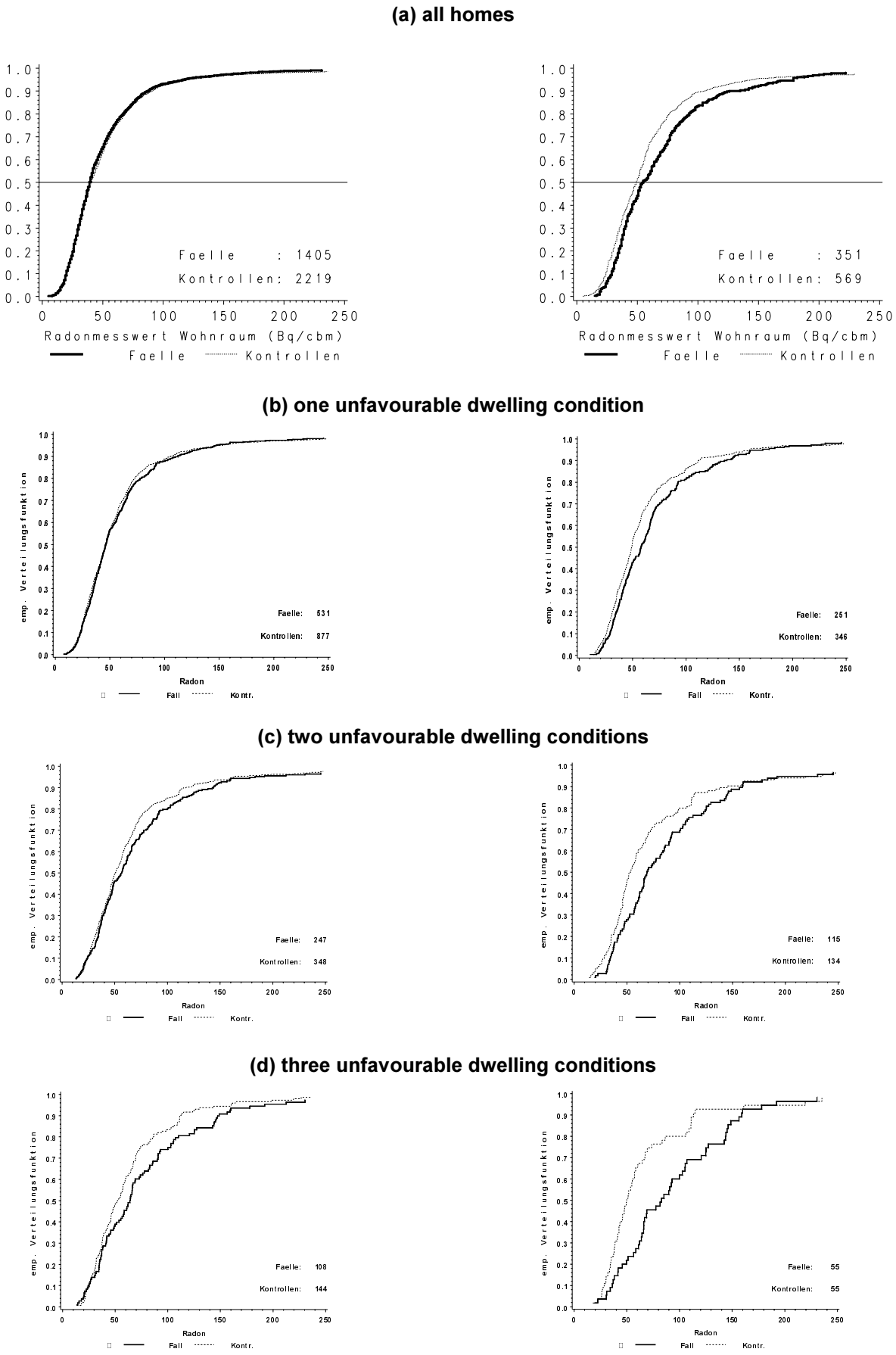

Figure 3: Cumulative distribution of radon concentration by cases and controls in groups with unfavourable dwelling conditions; left column: entire study region, right column: radon-prone matching areas

However, the perspective changes if the quantification of radon exposure - despite of the effort - would still differ strongly from the real exposure in the sense of random fluctuations. In this situation it is indeed possible that, adding a large number of persons with low exposure, the noise is increased in such a way that the existing effects cannot be identified. Exactly this is found if one goes from the radon-prone matching areas to the entire study area. The former have a small number of highly exposed participants with a clear exposure-response relationship. Adding a large number of participants with low exposure might dilute the data and the exposure-response relationship cannot be identified any longer. The same argument holds true for the subgroups with radon-relavant housing characteristics.

Such a phenomenon is well known in epidemiology. It is called misclassification of exposure by measurement error. Therefore this study due to its size and the large amount of homes with low exposure is able to illustrate this methodological problem especially clear. Theoretical papers addressing this topic (e.g. Thomas et al. 1993, Reeves et al. 1996) show that under normal conditions a „regression towards the null“ is observed, i.e. that the observable effect is partly or completely lost by random misclassification. This may be one of the reasons why in most of the published radon studies no exposure-response relationship is found. The reanalysis of the study of Pershagen et al. (1994) by Lagarde et al. (1997), which leads to a further increase of the published risk estimates, also emphazises the importance of random misclassification.

A good basis to compare the results of this study with those of other studies is the metaanalysis of Lubin & Boice (1997). In that paper, the most important eight case-control studies from the United States (Schoenberg et al. 1990, Alavanja et al. 1994), Canada (Létourneau et al. 1994), Sweden (Pershagen et al. 1992, 1994), Finland (Rousteenoja 1991, Auvinen et al. 1996) and China (Blot et al. 1990) are comprehended. In total it considers 4263 cases and 6612 controls. The original studies are brought into a comparable form, using a loglinear model of the higher categories, compared to the reference categories of the mutual papers. A significantly increased risk is found for the Finish study of Rousteenoja (1991), the two Swedish studies and the American study of Schoenberg et al. (1990).

In the metaanalysis the studies are weighted essentially by their inverse random error which depends on the number of cases and controls and on the distribution of exposure. The metaanalysis shows a statistically significant trend for a risk that increases with exposure. The OR is 1.14 (95 %-CI 1.01 - 1.30) for an increase of 150 Bq/m^3.

Compared to this our study shows no trend with an OR of 0.97 for an increase of 150 Bq/m^3 of the linear trend analysis in the entire study area. In the radon-prone matching areas the corresponding OR is 1.20 (95 %-CI 0.83 - 1.76). The linear trend from the original paper from Pershagen et al. (1994) leads to an OR of 1.15 (95 %-CI 1.01 - 1.33). This results are comparable with our results for the radon-prone matching areas

Finally, if one evaluates the pooled analysis of the 11 miners studies of Lubin et al. (1994) in a similar way for the relevant group of those with low exposure (total exposure below 50 WLM), after plausible recalculations a relative risk of 1.13 (95 %-CI 1.0 - 1.2) is seen at 150 Bq/m^3. This is practically identical with the metaanalysis of the indoor studies of Lubin & Boice (1997).

In total one can summarize that the available case-control studies on indoor radon and the corresponding extrapolations from data from miners suggest that exposure to radon indoors contributes in a relevant manner to the lung cancer risk in the general population. The results of this study, for the radon-prone matching areas, also support this finding.

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TABLE 1

Using the new ICRP respiratory tract model and the radiation weighting and tissue factors recommended by ICRP Publication 60, the effective dose per unit exposure in mines is 2-3 times that derived from epidemiological studies. This descrepancy between the dosimetric and epidemiological risk estimates is unlikely to be explained by the uncertainties in the dose calculation alone. There may be systematic errors in at least one of the ICRP recommended values for the risk weighting factors.
Case-control studies of indoor radon and lung cancer often have low statistical power. However, the result of a meta-analysis of eight case-control studies is consistent with the level of risk predicted from miner studies. The findings of a very large case-control study carried out in Germany from 1990 to 1996, suggest that the risk is not elevated for the entire study area: however, a significantly increased risk is seen if radon-prone areas or radon-relevant constellations are considered. The results from this German study are compatible with the so far largest Swedish study, with the meta-analysis mentioned above and with risk models from the analysis of cohort of miners. All these findings suggest, that exposure to radon indoors contributes in a relevant manner to the lung cancer risk in the general population.
Many parameters (e.g. equilibrium factor, attached fraction, aerosol parameters, target cell parameters, solubility and clearance rates, etc.) whose estimates are affected by a high degree of uncertainty may introduce a large uncertainty in the estimate of the effective dose. It is difficult to imagine that individual exposure and doses will be better known by a better accuracy in the future.
Geographical correlation studies are uninformative about lung cancer risk in relation to indoor radon.
A wide range of recommended action levels for existing dwellings have been fixed by the different countries. It takes a long time to disseminate knowledge about radon; in areas with increased radon levels the public is very interested and well informed but mitigation is much less than expected.

SUMMARY OF THE DISCUSSION

Mr. Feider, Chairperson of the Seminar, opened the discussion presenting his understanding of the main conclusions by the five speakers as laid down in Table 1.

He asked the participants whether they concurred on such conclusions. The participants did not challenge the conclusions however, clarifications were asked on some points of the presentation, and the speakers gave the following additional information.

Mr. Wichmann gave a possible explanation to the fact that the German study indicates an increased risk in radon prone areas, while in the entire study area the risk is not increased. He said that in the entire study area the noise is higher than in the radon prone areas, and this leads to a flatter exposure-effect relationship.

Mr. Wichmann also said that epidemiological studies in North America (in contrast to Europe) do not show clear indications of increased risk, a possible reason for that could be the greater mobility of people who in Europe change home much less than in North America. This leads to a less precise estimation of the exposure and therefore to an increased noise. He reported on a recent seminar held in Stockholm, where the results of the published Finnish study of Auvinen et al. were corrected, which now confirm the existence of an increased risk correlated with exposure to radon in dwellings (meanwhile published as Erratum Journal of the National Cancer Institute 90, 4 01-2, 1998).

Mr. Muirhead said that while data on epidemiological studies in dwellings point to an increasing lung cancer risk with increasing exposure, they are too sparse to tell whether, for a given total exposure, the magnitude of the risk is affected by exposure rate. However, there is some evidence from the miner data and from biological considerations to suggest that the effect seen at high exposures in the miner studies (ie, that for a given total exposure, the risk decreases with increasing exposure rate) disappears at low exposures.

He also said that it seems unlikely that epidemiological studies would show any increased risk for levels of radon up to about 50 Bq/m^3.

Mr. Muirhead and Mr. Wichmann emphasized that the main elements to be determined in a well-designed epidemiological study are the individual exposure and the individual smoking habits. The studies they considered, include both smokers and non-smokers however, the results from the smokers are the dominant effect.

Ms. Trimarche reported of a US study on non-smoking women of Missouri exposed to a radon concentration up to 200 Bq/m^2, which did not show any increased risk.

Mr. Monchaux supported the view that smoking increases considerably the risk.

ABSTRACT

Radon is a radioactive noble gas that occurs naturally in the earth crust from the decay of primordial radionuclides. It can migrate to the atmosphere, where it is inhaled together with the other components of the atmosphere, contributing substantially to the exposure of man to ionizing radiation.

The European Union, on the basis of the Euratom Treaty, has responsibilities in the field of protection against the dangers of ionizing radiation. Knowledge in the field is permanently progressing stimulated, amongst other things, by the European Research Programmes.

The presentations made at the Luxembourg Seminar summarise the state of the art on some aspects of radiation protection in relation to radon, in particular measurements, dosimetry and epidemiology.

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