

ANNEX 3

INDOOR RADON – a background document to the report 'A Radiological Protection Policy Under Discussion'

Ulla Swarén¹, Lars Ehrenberg², Gun Astri Swedjemark³, and Fredrik Granath⁴

¹Bohusgatan 53, SE-116 67 Stockholm, ulla.swaren@privat.utfors.se; ²Dept of Environmental Chemistry, Stockholm University, SE-106 91 Stockholm; ³Båtsmansvägen 11, SE-192 48 Sollentuna, ga.swedjemark@swipnet.se; ⁴Clinical Epidemiology Unit, Karolinska Hospital, SE-171 76 Stockholm, fredrik.granath@mep.ki.se

Introduction

The radioactive noble gas radon has attracted increased interest as a specific risk factor over the last few decades. Originating from the decay of the heavy elements uranium and thorium contained in the crust of the earth, the gaseous element radon, Rn, and its immediate decay products, known as progeny (radioactive isotopes of certain heavy elements), are generally present in the air and are therefore one of the unavoidable sources of background ionising radiation. Outdoor exposures will vary depending on the geological properties of the ground, the cultural habits of populations, etc., and indoor air exposures will also vary depending on where and how a dwelling is built, including its ventilation and the building materials used. However, with exception of a few known areas in the world, outdoor levels of radon¹ are generally very low in comparison with what can be found in dwellings at particular locations. Radon may also be present in sources of water supply, especially ground water.

In Sweden, indoor radon is—besides radiation used in medical practice—a major source of the general population's exposure to ionising radiation. The present mean in Swedish dwellings is around 100 Bq m⁻³ (measured as radon gas) with a maximum measured value of 84,000 Bq m⁻³; the mean outdoor concentration is calculated at 10 Bq m⁻³ (1).

In contrast to most radiation exposures, it is possible (in assessing lung cancer risks from radon) to obtain meaningful information from epidemiological studies at actually occurring environmental levels. Initially such studies concerned the working environment (miners) but more studies of the indoor environment and the general population are gradually emerging.

2. Inhalation of radon: notes on biology and dose concepts

The decay of gaseous radon generates a series of radioactive intermediates, eventually leading to stable isotopes of lead. The main radon progeny is very short-lived with half-lives counted in minutes or less. Rn-222, the predominant isotope involved in inhalation risk, has a half-life of about four days. The first of the long-lived decay products of Rn-222 has a half-life of about 22 years, but it is the short-lived progeny that give the highest radiation doses to the respiratory passage and the lungs. Some of the radon gas is transported via the lymphatic system and the blood to other organs in the body, increasing the calculated detriment by about 2% (2), with lung cancer thus being the major cancer risk in exposure through inhalation.

Above all, the decay of radon gas and its progeny is characterised by alpha-radiation (helium ions). The range of alpha-particles in the body is limited, one to two cell diameters. Alpha-

¹ In the following, unless otherwise specified, the term radon denotes both the element radon and its progeny.

radiation implies very dense ionisation, which means that its high biological effectiveness can lead to complex damage of chromosomes and DNA. In the decay of radon gas and its progeny, some beta- and gamma-radiation is emitted as well; however, if we focus on health risks engendered by inhalation, the predominant contributing factor is alpha-radiation. For more information on the mechanistic basis of radon-induced lung cancer, see, e.g., what is known as the BEIR VI report, from the US National Research Council (3).

In epidemiological studies of radon in ambient air, it is a natural approach to express exposure dose as radioactivity 'concentration' in Bq m^{-3} , for example, since this can be measured or calculated over time from actual measurements. In many other cases, and especially when radiation doses and/or risks are to be compared, the 'effective dose' according to the International Commission on Radiological Protection, the ICRP, is the reference point in radiological protection (4). The detriment in radon-epidemiological studies is likely to be either the number of fatal lung cancers (according to some specific definition of the disease) or the incidence of lung cancer. Although generally speaking, the latter should be a more informative measure of risk, it makes little difference to lung cancer risk estimates, since this disease has still a very high mortality rate.

For comparisons with effective dose from other sources of ionising radiation in radiological protection, the ICRP has introduced a special means of conversion from radon air exposure (average concentration) to effective dose, which is obtained by a direct comparison with the detriment (2). In many contexts nowadays, the ICRP uses a multi-component detriment concept (4), one component of which is fatal cancer. In its consideration regarding the inhalation of radon, the ICRP has concluded that 'the selection of a detriment coefficient different from the fatality coefficient for radon exposure is not justified'. The value of the detriment is associated with a unit effective dose, in mSv, and a unit radon exposure as defined by the ICRP. Conversions obtained in this way are called 'conversion conventions' and this new 'dose' concept is thus 'based on an equality of detriment, not on dosimetry'. It should also be noted that any change in the assessed radon risk will yield a different figure for one and the same exposure dose.

With regard to exposure assessments *per se* in epidemiological studies that build on exposures in the past, there is first of all the general difficulty of calculating the doses in retrospective—and different scientific approaches are being used, sometimes in combination. This kind of uncertainty and its consequences for the risk estimation will be briefly illustrated in the following section. With regard to exposures to radon in particular, there are also specific issues to be considered if meaningful dose measurements are to be obtained:

A reasonably inexpensive and therefore common method of measuring radon 'concentration' is one in which the radon gas, but not its progeny, is 'trapped'. This may well give operative dose values in a study that can be compared with those obtained in other studies using similar methods. However, the radon progeny occurs in air to some extent as free ions, which easily bind to air-borne particles. Such particles, including components in tobacco smoke, interfere with the rate of uptake into the body of other agents, implying that actual exposures from indoor radon sources often will be higher in 'dusty' environments. Considerable variations have been shown, see e.g. (5). Depending on the methods used, two identical exposure measurement values may not in fact be identical with regard to the inhaled radon dose. From the point of view of biological effect, the ideal dose concept would then take into account - besides the distribution pattern of the individual progeny - the fact that particle size and the amounts and characteristics of the 'unattached' fractions of the radon progeny will influence the uptake in the body, the general picture being that more unattached fractions increase the uptake and thus offset the influence of air pollution by agents other than radon. This is discussed in depth in the BEIR VI report (3)

3. Radon in ambient air; certain risk assessments

In 1993 the ICRP made an assessment of indoor radon lung cancer risks based on studies of miners (2). However, a nation-wide Swedish case-control study of radon in dwellings that had at that time just been presented (6) was considered as supporting the conclusions by the ICRP about a radon-entailed risk to the general public. A few years later, in 1999, the BEIR VI report was published, which so far constitutes the most extensive review of available scientific knowledge. In this report (3), the lung cancer risk estimates were based on a number of 'pooled' cohort studies of miners, the BEIR committee being of the opinion that the lower exposures of people in the then-available case-control studies in dwellings 'and methodologic problems make it very difficult to identify the relationship between residential radon exposure and lung-cancer mortality in an individual study'. However, the committee did use a meta-analysis of eight residential epidemiological case-control studies, published in 1997 (7), in support of a linear extrapolation of results from the studies of miners to radon concentrations normally found in dwellings.

If the ICRP's risk coefficient is applied to Swedish exposure conditions, nearly 40% of all lung cancer deaths may be associated with radon. The corresponding figures based on the BEIR VI report (the Committee used two different calculation models) would be 23% and 33%, respectively. (8)

In Sweden, the Radiation Protection Authority, the SSI, revised in 2001 an earlier assessment of the lung cancer risk to the general public from residential radon exposure. While the previous assessment (9) in 1994 had been explicitly based on studies from both the occupational environments in mines and from dwellings, this time the SSI stated that they judged the case-control studies of indoor radon taken together to be a sufficient basis for risk assessment, preferring this to the studies of miners, in which exposure conditions and the exposed populations differed in certain respects (8). The next two paragraphs summarise the further considerations and conclusions presented by the SSI in that document.

The 1993 Swedish national case-control study mentioned above (6) had found about 16% of lung cancers to be associated with indoor radon exposure, with an uncertainty factor of about two, implying 200–900 cases at the current (2001) incidence rate. (This should be seen in relation to a population of just under 9 million.) Taking into consideration that the estimate is still uncertain and that the studies of miners have given higher risk figures than the studies of radon in dwellings, the SSI chose a figure of 500 expected cases annually with an upper limit of 1,000. The SSI also noted that an overwhelming majority of people, or nearly 90%, with radon-related cancer are also smokers.

In this context, the SSI also discussed the risk to non-smokers, referring in particular to a new Swedish case-control study on 'never-smokers', to be published shortly afterwards (10). The study shows that exposure to radon increases the lung cancer risk to never-smokers as well, with a relative risk increase of 0.10 per 100 Bq m⁻³. This figure is about the same as that from the Swedish nation-wide study of 1993 comprising both smokers and non-smokers (6). The whole of the increased risk seemed to occur among those never-smokers who were also exposed to environmental tobacco smoke, ETS, while the group not exposed to ETS did not show an increased risk with rising radon concentration. However, the SSI concurs with the authors of the study that the evidence of an interaction between ETS and residential radon exposure among never-smokers needs confirmation. It should also be recognised that, although the radon-related excess relative risk among never-smokers did not appear different from that in smokers, it is obviously much smaller in terms of excess absolute risk. Our own

further comment to this is to underline that the important issue of the theoretically possible lung cancer risk from radon alone, to never-smokers (not exposed to tobacco smoke at home), is still not known. For statistical reasons it is also possible that this issue will never be clarified by epidemiological means.

The issue of uncertainties in these and similar studies deserves a few more comments. One is the influence of age at exposure, which is also discussed in the BEIR VI report (3). The other is the retrospective calculation of the radon dose. There are different ways of approaching this, obviously also depending on the method(s) used for the exposure assessment. The following illustrates the importance of taking into consideration potentially significant errors in retrospective exposure analysis:

A study in 1991 (11) aimed at identifying where the largest uncertainties could be found and where efforts might most effectively be made in trying to determine long-term residential radon exposures. Using Swedish data on a number of relevant parameters, the lack of knowledge of which was numerically studied by means of a simple Monte Carlo technique. Later a new study was made, doing essentially the same thing but using a different mathematical approach (12). In this paper, the data from the previous study modelling Swedish conditions was used and applied to the nation-wide Swedish 1993 study. Here it may be added that the latter study (6) included a large number of lung cancer cases diagnosed from 1980 through 1984, as well as control groups. Radon measurements had been taken in about 3 out of 4 dwellings occupied by the study subjects over at least 2 years since 1947. The authors of the study (12) evaluated the possible bias in risk estimates related to random error in the retrospective assessment of time-weighted average radon concentration (TWA). Without adjustment for random error in the TWA estimates, the linear excess relative risk coefficient was 0.10 per 100 Bq m⁻³ (as mentioned above) but a value of 0.15 to 0.20 per 100 Bq m⁻³ was suggested following adjustment. Although this does not say anything about the size of the absolute risk, such an increase (50-100%) of the relative risk may be noteworthy.

4. Radon in drinking water; certain risk assessments

Risks from radon in drinking water have lately (1999) been extensively analysed by the US National Research Council, NRC, through another special committee (13). Its work is based on a comprehensive data inventory and some new modelling, especially of the distribution of ingested radon in the body. Radon in drinking water presents special problems *inter alia* with regard to the exposure analysis.

Most of the gaseous radon is transferred to the ambient air where it adds to the radon/radon progeny levels. If the committee's transfer coefficient is applied to water containing, e.g., 1,000 Bq L⁻¹ of radon activity, the calculated mean increase of radon in the indoor air would be about 100 Bq m⁻³ (under US conditions). Proceeding from data on radon activity in water, one may then assess the additional lung cancer risk on the basis of preferred epidemiological investigations. The NRC committee mentioned has used the BEIR VI data. It may be added that the example above (1,000 Bq L⁻¹) in Sweden also represents a limit above which the water is considered unfit for use, according to official recommendations.

For radon ingested via drinking water, there are no epidemiological studies. In the study (13), the ingestion risk—mainly of stomach cancer from radon gas—was calculated to be very small, some 5–50 cases per year in the whole of the US, based on a 'dose to risk' assessment using the ICRP coefficients for the relevant organs. As a result of the exposure analysis, the committee also concluded that radon progeny in drinking water is not likely to pose a health risk.

With respect to Swedish conditions, where the mean radon concentration in drinking water is about twice the figure given for the US, the SSI has stated that radon transferred from consumer water stands for a few percent of the total exposure to indoor radon and may have

caused a few tens of the deaths in lung cancer recorded each year. Using the NRC models, the SSI estimates the mean radiation dose to the Swedish population caused by ingested radon to be less than 0.01 mSv per year, implying a few cancer deaths annually. (8).

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