Radon in the Human Environment

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Introduction

Extensive screening of radon concentrations during recent years has revealed that these concentrations are markedly higher inside of buildings than outside (e.g. ICRP 50, 1987). The exposure to radon and to radon daughters results in an, on the average, tenfold higher radiation dose to the bronchial epithelium (i.e. about 10 mSv) than to most other tissues of the body due to background exposure. For children the models of calculation even predict double the dose estimated for adults (ICRP 50, 1987, page 17). This comparatively high burden for the population requires thorough studies of a possible risk exerted by radon and its progeny.

Physical Background

Radon-222 originates from uranium-238 via radium-226. The gaseous radon disintegrates by emission of alpha-rays to solid, but radioactive polonium-, lead- and bismuth-nuclides. In particular the polonium nuclides (polonium-218 and -214) make the major contribution to the radiation dose to the respiratory tract. This is mainly due to the fact that these solid alpha-emitters are bound to the aerosolic fraction of the air and are deposited in the respiratory ducts after inhalation, whereas radon itself (physical half-life = 3.82 days) is predominantly exhaled shortly after entering the respiratory tract.

Uranium and consequently also radium are distributed almost ubiquitously, though in varying concentrations, in soil and rock. Thus, human beings are exposed to radon everywhere, however, to grossly different doses depending on the environmental conditions.

Radon Sources and Concentrations

As outlined above, the outdoor concentration of radon is caused by the radium-226 content of rock and soil. The concentration is about 9 Bq/m³ worldwide (Gesell, 1983). There are considerable differences when the concentrations are compared either with regard to time of the day (2 to 5 times higher values during night than around noon) and to time of the year (2 to 4 times higher values during late summer and autumn than during spring time). Also concentration diminishes with increasing distance from the ground, so that children are exposed to slightly higher concentrations than adults.

Most of the indoor radon derives from the radium located in the
soil below the buildings; variable amounts are contributed by the building material, air from outside, tap water, and natural gas. This sums up to a mean value of approximately 50 Bq/m³ in dwellings (for reviews see UNSCEAR, 1986; ICRP 50, 1987; BEIR IV, 1988). Variability, however, is pronounced: the indoor range is between a few Bq/m³ to several thousand Bq/m³. With the exception of a specific building material used in Sweden, it is almost exclusively the soil underneath the buildings that is responsible for unusually high radon concentrations indoors.

When estimating radiation risk, one has to take into account that indoors no equilibrium between radon and its daughters is reached. Therefore, one has to correct for this fact by multiplying the radon concentration with an equilibrium factor (usually in the range between 0.3 and 0.5); radon concentration is then expressed as "equilibrium-equivalent radon concentration" (or abbreviated: Rn-eq).

**Health Risks that May be Expected**

In general, four different types of risk can be expected after exposure to ionizing radiation: lethality, teratogenicity, mutagenicity, and carcinogenicity (detailed information by Streffer, 1989). For various reasons (Müller and Streffer, 1990), only the carcinogenic risk must be considered in the context of radon exposure.

The major target organ is, of course, the respiratory tract. As our current knowledge suggests that there is no threshold dose with regard to radiation-induced tumour risk, one should expect a certain contribution to the lung tumour frequency by radon and radon daughters. In the remaining part of the paper, the procedure will be described, how to achieve an estimate of this contribution.

**Epidemiologic Studies and Risk Coefficient**

Already in the 16th century radiation-induced lung tumours caused a high frequency of deaths in miners ("Schneeberger Krankheit"), though nobody, of course, was aware of the nature of the disease and the involvement of ionizing radiation in its genesis at that time. Only at the end of the 19th century, the disease was identified as a lung tumour and only some decades ago the causation by radon was realized.

Recent epidemiologic studies of miners (in particular uranium miners) clearly point to an increase in the frequency of lung tumours in dependence of the amount of radon exposure (for reviews see ICRP 50, 1987 and BEIR IV, 1988). The relative risk coefficient derived from these studies amounts to about 1.6% per 1 million Bq h/m³ Rn-eq. That is: the radon concentration (in Bq/m³) at the place of work is multiplied with the duration of stay (in hours); this time integrated concentration gives the exposure of the individual (in Bq h/m³) and is, after consideration of the equilibrium factor, the basis for risk estimation.