HEALTH HAZARDS FROM RADIOCAESIUM FOLLOWING THE CHERNOBYL ACCIDENT

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The WHO Regional Office for Europe has organized a series of meetings to assess the health impact of the Chernobyl nuclear accident. The most recent meeting in the Federal Republic of Germany reviewed the principal long-lived radionuclides emitted from the accident and concluded that ^{134}Cs and ^{137}Cs had the greatest potential for contributing to the human dose because they are still present, the dose will be delivered over a long term, and because of the accumulation in some edible plants and animal products. The observed contribution of radionuclides to the collective effective dose-equivalent in the first year is about 60-80% from ingestion, 30-40% from external irradiation, and 2-20% from inhalation.

ENVIRONMENTAL PATHWAYS

Ingestion of contaminated food and water can occur by direct and rapid, or indirect and long-term pathways. The most immediate are the drinking of contaminated milk and the ingestion of fresh, leafy vegetables and fruits contaminated by direct deposition. Other pathways involve the movement of radionuclides from soil to plants and on to animals.

The amount of direct contamination on plants depends on how the deposition occurs, the density and type of crop, and the weathering that occurs before the crop is harvested. Initial retention is typically 25%, but may vary between 5 and 40% depending on vegetation density and rainfall rate. Approximately half of the deposit may be lost in 2 weeks due to weathering and growth [1]. Uptake of radiocaesium by plants from soil depends on the physicochemical conditions of the soil and the type of plants. For example, soils with high clay content retain radiocaesium much better than certain acid, sandy and organic rich soils, and plant uptake from clays will be very low. The radiocaesium uptake from acid soils may be significantly higher, and contamination of plants grown in these soils will continue for long periods of time [2].

Transfer from plants to food products of animal origin, such as milk and meat, depends on how each radionuclide is metabolized by individual species. Radiocaesium is easily transferred from fodder to milk and meat. For humans, approximately 20-30% of the daily intake is secreted in human milk [3]. Of particular interest is the lichen-reindeer (caribou)-human pathway. The effective half-life of caesium in lichen is 8-10 years, and approximately 25% of the caesium contained in lichen is absorbed by reindeer [3]. The biological half-life (T_b) of radiocaesium in animals depends on the body mass, and on whether the animal is ruminant or monogastric, domestic or wild.

Ingestion of radionuclides through the aquatic food chain also contributes to the dose, although the impact is often more limited

than through the terrestrial pathways. Radiocaesium is soluble in water. The uptake in aquatic life is influenced by factors, such as dilution, feeding habits and water salinity. Marine fish show steady-state concentration factors of the order of 100 [3]. Freshwater fish may have concentrations which are greater than those of marine fish by one order of magnitude or more, although large differences occur between species [4,5].

EXTERNAL AND INTERNAL EXPOSURES

The external radiation dose received by individuals in urban and rural areas will vary markedly for a given amount of deposition, as the external dose from deposited radionuclides is influenced by the type and amount of deposited material, and the extent and time of exposure. In rural areas, compared to urban areas, a larger fraction of the airborne particles near the surface will be deposited as a consequence of differences in surface characteristics, but the dose rate will decline progressively due to the gradual penetration of caesium into the soil. In urban areas, deposited material will be washed away and weathered more rapidly; also, the buildings will provide a significant shielding. Moreover, the population in urban areas spends more time indoors than one in rural areas. Overall, therefore, the urban external dose rate will be considerably less than the rural one.

Seasonal variations strongly influence the level of radioactivity in different foods, such as cereal crops, fodder, and pasture grass. The state of grass growth and availability of pasture at the time of the accident exerted a strong influence on the contamination observed in some animal products.

Once radiocaesium has entered the food chain, dietary habits of different populations will exert a great influence on exposure of individuals to internal radiation because of food quantities eaten and selection of dietary items. Food processing may alter the degree of radioactive contamination. For example, it decreases during the preparation of fruit juices or bread, or increases due to drying or concentration of milk.

DOSE FACTORS

Whatever the entry route into the body may be, uptake of caesium to blood is generally quick and almost complete. Once absorbed, caesium distribution is diffuse and relatively homogeneous, and its concentration is approximately the same in all the internal organs and soft tissues. Consequently, the dose factors are virtually independent of the organ or tissue considered. When considering age, two parameters count: mass of organs and tissues, and retention. In the case of radiocaesium, the effect on the dose factors of their variation with age are opposite, and compensate each other. Indeed, going from the infant to the adult, dose factors should decrease because the mass of organs and tissues increases. However, this trend is counteracted as, at the same time, the long-term retention increases with age. The retention fraction and biological half-life (T_b) of the long-term

component of retention vary with age. An infant will retain a fraction of 0.4 (T_b = 20 d), whereas an adult will retain a fraction of 0.9 (T_b = 110 d). Consequently, dose factors are virtually independent of age, although the use of an age-independent dose factor may result in a small overestimate of the effect on children.

HEALTH RISKS

The possible health effects from radiocaesium after the accident may be expressed in terms of stochastic consequences. These are fatal cancers, serious genetically related ill health and possible teratogenic effects. They are added to the incidence of these ill effects which is normally present in the population. The calculations, based on a linear no-threshold model, will usually represent an upper or conservative limit of risk expectations. The radiation doses to individuals and population for the first year after the accident, as well as the committed doses projected for 50 years, have been considered. For fatal cancers induced by radiation, a risk factor of $2 \times 10^{-2} Sv^{-1}$ is used. For serious genetic health effects, the appropriate risk factor is $4 \times 10^{-3} Sv^{-1}$ for the first two generations. Teratogenic effects may be seen following high radiation doses, and present knowledge does not exclude the possibility of a threshold, especially for low doses, in the range of 1 mSv. At higher doses the risk of severe mental retardation is 0.4 Sv^{-1} for a fetus exposed in the period between the 8th and 15th weeks of gestation, and 0.1 Sv^{-1} for a fetus exposed between the 16th and 25th weeks.

The range of individual 50-year dose increments from the accident is estimated at 0-6 mSv. The dose estimates are based on the measurements and assessments made within individual countries from methods they have considered most suitable. No attempt has been made to "normalize" such estimates. Standardization of dietary habits or other patterns of behaviour that will vary widely even within countries, is not appropriate. However, the overall uncertainty will be of the order of no more than a factor of 2.

Concerning the health effects from all radionuclides contributing to population dose, the 50-year collective dose is about 0.33×10^6 person-Sv. Thus, the upper estimate of associated fatal cancer cases in Europe (excluding the USSR) is about $(0.02)\ (0.33 \times 10^6) = 7000$, which would be added to a normal expectation of about 110 million fatal cancers (assuming 20% is the fraction of overall spontaneous mortality due to cancer which is applied to the cohort of 550 million Europeans, excluding the USSR). This is equivalent to an additional incidence of up to about 0.006%, which is an added risk to the population of about 0.001% $(7000/(550 \times 10^6))$. The first-year dose is considered for both genetic and teratogenic health effects. Assuming 28×10^6 cases as a nominal incidence of serious genetic effects, the additional radiation-induced cases in the first generation are estimated at up to 700 cases (an extra 0.003%). Teratogenic effects, such as severe mental retardation, have been seen in children who were irradiated during their 8th to 25th weeks of fetal development. The expected number of live births in the year following the accident in the

550 million people in Europe is 12 000 per million, or a total of 6.6 million. The average individual dose in the first year is 0.3 mSv, and this is combined with risk factors of 0.4 for 8 weeks plus 0.1 for 10 weeks. Up to an additional 200 cases might therefore be added to the 50 000 cases which would be expected spontaneously - (extra 0.4%) or an additional risk of 0.003% in the newborn population. The up to some 7000 anticipated radiation-induced cancer fatalities will not be evenly distributed throughout Europe (excluding the USSR). The risk of such fatalities will be higher in areas where deposition was higher or where received doses were greater. Because of differences in population density in these areas, radiation-induced cancer fatalities may be a higher proportion of the normal incidence than is given for Europe as a whole. Clearly, the reverse also is true. However, the estimated range of radiation-induced increments of possible stochastic health effects risk is very low and probably could not be detected, even by the most careful study.

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REFERENCES

1. Aarkrog, A. Translocation of radionuclides in cereal crops. In: Ecological aspects of radionuclide release. Oxford, Blackwell Scientific Publication, 1983, pp 81-90.

2. Menzel, R.G. Soil-plant relationships of radioactive elements. Health physics, 11: 1325-1332 (1965).

3. Aarkrog, A. Environmental studies on radioecological sensitivity and variability with special emphasis on the fallout nuclides ^{90}Sr and ^{137}Cs. Thesis, University of Riso, 1979 (Report Number 437).

4. Carlsson, S. Caesium-137 in a dysoligotrophic lake, a radioecological field study. Thesis, University of Lund, 1976.

5. Camplin, W.C. et al. Radioactivity in surface and coastal waters of the British Isles. Monitoring of fallout from the Chernobyl reactor accident. Lowestoft, Ministry of Agriculture, Fisheries and Food, 1986 (Aquatic Environment Monitoring Report 15).