Carcinogenic risk from hot particle exposures - has ICRP got it right?

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The argument has become very familiar—that radionuclides introduced into the environment from nuclear installations, fall-out from weapons testing, or whatever source, are responsible for substantial increases in cancer rates, and, because current risk estimates do not support this conclusion, they must be very wrong. It is argued that there must be some way in which low levels of artificial radionuclides, levels that result in tissue doses lower than from naturally-occurring radionuclides, pose a risk that is yet to be appreciated. One obvious problem with current risk estimates, it is suggested, is the simplistic averaging of doses from hot particles—see, for example, the home page of the Low Level Radiation Campaign website (www.llrc.org).

It is true that the most widely used estimates of dose and carcinogenic risk from incorporated radionuclides, provided by the International Commission on Radiological Protection (ICRP), are based on calculations of average dose. Dose estimates do consider, however, the distribution of target cells within tissues. Doses to the respiratory tract from inhaled radionuclides take account of the position of target cells for the induction of different lung cancer types in the bronchial and bronchiolar airways (ICRP 1994). Similarly, the distribution of target cells in skeletal tissues is taken into account in estimates of risk of bone cancer and leukaemia (ICRP 1979, Gössner et al 2000). In other cases, the liver for example, the assumption is made that target cells are distributed throughout the whole organ. Thus, the target tissue volume can vary from a single layer of cells to a whole organ. But, having considered the available animal and human data on hot particle effects, ICRP concluded that risk should be estimated on the basis of average dose within the target tissue (ICRP 1980, 1991). Claims that dose from hot particles can be orders of magnitude more carcinogenic than dose delivered uniformly were judged to be poorly founded.

Charles and Mill (pages 5–28 of this issue) have provided a review of in vitro and in vivo experimental studies and epidemiological evidence relevant to the hot particle question. Most information comes from animal studies but with support from recent in vitro studies. Charles and Mill conclude that ICRP dose averaging is likely to provide a reasonable estimate of carcinogenic risk, within a factor of ±3. The evidence includes a quite surprising concordance of risk estimates for radiation-induced lung cancer from three sources: Russian Mayak workers exposed to plutonium-239, mainly by inhalation, underground miners exposed to radon-222 and its progeny, and Japanese atomic bomb survivors exposed to external radiation. Taking account of differences in relative biological effectiveness of alpha particles and low LET radiations, the risk estimates derived were similar despite differences including the time-course of dose delivery and the heterogeneity of energy deposition from plutonium-239 oxide particles (see also Grogan et al 2001, Harrison and Muirhead 2003). While it is important to recognise the uncertainties associated with these risk estimates, including lifetime risk projection and application to different populations for the Japanese data and estimates of lung target tissue doses from plutonium-239 oxide particles in Mayak workers, the values obtained suggest that hot particle irradiation is not unexpectedly effective. This conclusion is also supported by comparisons of risk estimates for radiation-induced liver cancer and leukaemia obtained from
the Japanese data and from studies of patients given injections of Thorotrast, an alpha-emitting thorium-232 oxide particulate, used as a vascular imaging agent in the 1930s and 1940s. Additional evidence is provided by studies of cancer rates and radiation exposures, including plutonium-239 oxide exposures, in workers at the British Nuclear Fuels plant at Sellafield, UK Atomic Energy Authority, UK Atomic Weapons Establishment, and Los Alamos and Rocky Flats in the USA. These studies show no evidence of radiation-induced lung or liver cancer at the relatively low levels of exposure of these working populations.

In considering carcinogenic risks from hot particle irradiation, it is worth bearing in mind the extent to which they may ordinarily be incorporated into body tissues unless introduced directly into blood (e.g. Thorotrast). While particles are deposited in the respiratory tract and can pass through the alimentary tract, they are effectively prevented from reaching the blood stream and hence reaching other body tissues. An exception is the slow clearance of particles from lung tissue to the associated tracheobronchial lymph nodes (TBLN), a process that can lead to concentrations of, for example, plutonium-239, that exceed concentrations in all other tissues (see e.g. Popplewell et al 1985). The ICRP respiratory tract model includes particle transfer to lymph nodes. However, it appears that TBLNs are insensitive to radiation-induced malignancy—no lymphoid tumours have been observed in uranium and thorium oxide workers or in animal studies (see e.g. The Royal Society 2001).

It can be concluded that, on current evidence, hot particle effects do not provide a mechanism for doses from environmental levels of artificial radionuclides to be more effective in causing cancer than larger doses from naturally-occurring radionuclides. The ICRP approach of averaging dose to target cells and tissues appears to give reasonably reliable estimates of risk.

A real problem with hot particles is, of course, the possibility of exceeding threshold doses for the development of deterministic effects—acute damage to tissues due to cell killing. Charles and his colleagues have been leading contributors to the development of knowledge and standards of protection, particularly with regard to effects of local irradiation of skin (see e.g. Charles et al 2000). Studies in progress include detailed assessments of possible doses and effects following skin contact with or ingestion of hot particles released from the Dounreay fast reactor site (Darley et al 2003). These studies, involving collaboration between the University of Birmingham and NRPB, will include estimates of cancer risk and consideration of the very low probability of encountering such particles.

The whole question of doses and risks from internal emitters is currently being considered by the Committee Examining Radiation Risks from Internal Emitters (CERRIE), set up in 2001 under the auspices of the Government’s advisory Committee on Medical Aspects of Radiation in the Environment (COMARE). The remit of CERRIE, formed after discussions between the Department of the Environment, Food and Rural Affairs and the Department of Health, is ‘to consider the present risk models for radiation and health that apply to exposure to radiation from internal radionuclides in the light of recent studies and identify any further research that might be needed’. The paper by Charles and Mill (pages 5–28) has been presented as evidence to CERRIE. The Committee will continue its work throughout 2003 and should report in 2004 (see www.cerrie.org). The report should include objective evaluation of scientific data and their application, and identification of important uncertainties with recommendations for further research.

References

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