

Dr F. J. C. ROE (Independent Consultant): Darby and Reissland's paper might be seen as a response by "the establishment" to a challenge from an outsider to the effect that more people are at risk of developing cancer as a result of long-term low-dose exposure to ionizing radiation than has hitherto been calculated by the International Commission on Radiological Protection (ICRP). Risk from low-dose radiation has necessarily to be calculated by extrapolation from data on the effects of long-term exposure to higher doses or of acute exposure to the same total dose. The crux of the matter is whether such extrapolation is valid? Is the shape of the dose response curve in the observable range predictive of that in the sub-observable range? Are the cumulative effects of numerous small doses less than, equal to, or more than those of acute exposure to the same total dose? On the basis of data from the large nuclear facility at Hanford in the USA, Mancuso *et al.* (1977) claimed that the cancer risk from long-term low-dose radiation exposure is substantially higher than predicted by ICRP. Darby and Reissland are reporting the results of their analyses of the same data. They point to deficiencies in the data, particularly the lack of information on cause of death for 49 out of 2089 workers who died before 1974. They associate themselves with earlier criticism of Mancuso *et al.*'s analytical methods (which were confined to men who had died, made inadequate allowance for confounding variables, and underestimated cumulative radiation dose for men who died with cancer, etc.). They conclude that the claims made by Mancuso *et al.* (1977) are not substantial and that with the possible exception of multiple myeloma (3 deaths observed *versus* 0.28 expected) the Hanford data are compatible with ICRP predictions. On the other hand they add, cautiously, that the data are also compatible with a wide range of values for cancer-induction rates and that there is a need for the collection and analysis of more data.

I am not a statistician or an epidemiologist and so I am free to comment that I deplore the practice which seems to be particularly rife in the USA of rushing incautiously into print with analyses based on incomplete data. It should have been possible for someone to have found the missing 49 death certificates. I instinctively distrust proportionate mortality ratios and am much happier with analyses based on comparisons of age-standardized risk of various cancers in initially defined exposure groups than with limited case-control comparisons. Thus I am much happier with Darby and Reissland's analyses than with those of Mancuso *et al.*

It is plausible that low dose radiation is a risk factor in the aetiology of multiple myeloma since other important risk factors have not been identified. Moreover, other studies have suggested a similar association. In the absence of detailed information about smoking habits I believe it would be nonsense to draw any conclusion about the effect of radiation on risk of pancreatic cancer from the presently available Hanford data. The difference between one observed death from renal cancer in the 5 rem exposure group, as compared with 0.6 expected, does not provide grounds for suspecting any causal link. Despite the data for multiple myeloma, it would be my overview that the available data from Hanford provide welcome reassurance that ICRP's calculations of cancer risk from radiation are reasonably conservative. In particular, I am impressed by the absence of any apparent effect of exposure on longevity, even after allowance is made for the healthy worker phenomenon.

The no-threshold concept of carcinogenesis as applied to chemicals gains credence because of the consensus view that no dose of radiation can be regarded as safe. Those who quote this as a consensus view should realize that it is based more on an understanding of the nature of radiation damage at the molecular biological level than on hard data from epidemiological or animal studies. Inaccuracies of death certification and the impossibility of controlling or measuring human exposure to other relevant risk factors are likely always to blunt the epidemiological tool rather severely.

I regard Darby and Reissland's paper as soundly based on appropriate statistical and epidemiological methodology. My only question is, would it ever have been necessary if Mancuso *et al.* had refrained from rushing into print a paper based on short-cut methods and, seemingly, a lesser understanding of the pitfalls?

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