Notes and miscellanea

Hanford radiation study

A note on: "Hanford radiation study III: a cohort study of the cancer risks from radiation to workers at Hanford (1944-77 deaths) by the method of regression models."

We shall refer to the above paper (p 156) as paper III, and to previous papers by these authors as I (ref 1 in the above paper), II (ref 5), and IV (ref 6).

Paper I reported that fatal malignancies were induced in radiosensitive tissues with much higher frequency per unit radiation dose than was generally accepted. It was also estimated that 6% of all cancers and 1% of all certified deaths were radiation-induced.

Criticism of this analysis focused on the authors' failure to take into account factors other than radiation that are known to influence the risk of death due to cancer such as age and calendar year. The authors were also criticised for basing their main conclusions on a proportional mortality analysis, and hence ignoring available information on those who survived to the end of the follow-up period. Paper II presented a reanalysis of the Hanford data, including additional deaths that occurred between 1972 and 1977. Revised numerical estimates of risk were given which were considerably lower than those reported in paper I, and it is estimated that approximately 5% of the deaths from cancer of Hanford workers were radiation-induced.

These conclusions were reached after controlling simultaneously for sex, age at death, date of death, internal radiation, and exposure period. However, the analysis in paper II still considered proportional mortality only.

The present paper (III) attempts to overcome the remaining criticisms of the authors' earlier work. The data have been divided into subgroups defined by different combinations of controlling factors specified in table 1. The hypothesis of no radiation effect is tested by comparing the total dose of those dying in any one year with the value expected if, in any subgroups, those dying form a random sample of those surviving to the beginning of that year. The evidence for various years of follow-up and the different subgroups is combined into a single t-statistic. The first three lines in table 4 give the values of this statistic considering deaths from all causes when all the controlling factors specified in table 1 are included, and also when some of them are omitted. A striking feature is the critical dependence on whether or not monitoring for internal radiation is included as a controlling factor.

The four levels of monitoring for internal radiation, described in the footnote to table 1, form a rough measure of internal radiation dose. It is therefore to be expected that there is a strong correlation between the level of monitoring for internal radiation and external radiation dose. This expectation is confirmed in table 2. Thus the inclusion of level of monitoring as a controlling factor is to some extent controlling for external radiation dose, since those at monitoring level 4 (which includes almost everyone in the highest external dose categories) are not compared with those at monitoring level 1 (which includes the majority of those in the lowest external dose category). The implications of such a procedure are unclear, as it potentially obscures a large part of the relevant information. Level of monitoring for internal radiation has been included as a controlling factor throughout the model fitting in the later part of the paper and upon which many of the conclusions are based.

Controlling for internal monitoring levels is the most important difference between this and other analyses of the Hanford data, and probably accounts for the remaining differences in conclusions. The justification for such a controlling factor, given in paper IV, is that it distinguishes between safe and dangerous occupations. We are unable to follow the logic of this argument, and although the highly significant negative t-values for all deaths, shown in the first two lines of table 4, certainly imply an inadequacy in the controlling factors, it does not follow logically that any factor which removes this negative effect is an appropriate one to include. The appearance of a significant negative t-value for group B cancers simultaneously with the significant positive result for group A cancers in the third line of table 4 suggests to us the opposite conclusion.

In the fourth line of table 4 level of monitoring is again included as a controlling factor, although workers are now allowed to progress through the monitoring levels. We feel that the implications of the procedure are no clearer here than in the previous case.

The introduction of a job hazard index based on job specification and made without any reference to film badge readings or bioassays seems to us to be a step in the right direction, and we should like to see the details of this index. The use of this index as a controlling factor, however, does not remove the large negative t-value for group B cancers, and this
indicates to us that there could still be inadequacies in the controlling factors used in the fifth line of table 4. The authors attempt to explain the negative finding for group B cancers by noting that it disappears when place of death is included as a controlling factor in a completely separate proportional mortality analysis which has been carried out after excluding all deaths ascribed to myocardial infarction and accidents. This explanation does not clarify our understanding of the issue.

Finally, we are glad to note that the present analysis makes no claim to find any evidence of radiation-related effects in the Hanford data when deaths from all causes are considered together, or when deaths from all types of cancer are taken as a single group. This conclusion is similar to that reached in other analyses of the Hanford data and now seems to represent a general consensus of opinion. There are, however, still important differences between the conclusions reached in this and other analyses of the Hanford data. These centre round the evidence for radiation-related effects for particular types of cancer. It is our view that the most likely explanation for these different conclusions is the use of level of monitoring for internal radiation as a controlling factor in the present analysis.

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References


Dr Stewart replies

The main objection to our paper and its conclusions appears to be that we have used as a controlling factor one that is difficult to interpret and that our results depend critically on it. According to Dr Reissland the four levels of this factor—called by us the level of monitoring for internal depositions—"form a rough measure of internal radiation dose." According to our reading of the relevant documentation, however, this is a complete misunderstanding.

Although 17,066 individuals, or over half the total workforce, were repeatedly subjected to urine analyses (see levels 2, 3, and 4 in table 2), only 225 workers (all men in level 4) ever had an estimable body burden of plutonium. A body burden was considered estimable if the maximum estimate consistent with the data exceeded one ten-thousandth of a micro-curie. For 9890 workers in levels 3 and 4 at least one of their urine analyses had detectable plutonium—that is, the disintegrations per minute for a 24-hour sample exceeded a detection limit that ranged from 0.066 before 1949 to 0.05 after 1953. Apart from these 225, however, further analysis never detected a body burden; therefore the plutonium must have come from accidental contamination of clothes or apparatus. The remaining distinction—that between levels 3 and 4—is that individuals in level 4 were considered to be at such risk of an internal deposition that they were subjected at least once to a whole body counter. But again, except for the 225, no estimate of plutonium body burden could be made.

Thus it is clear that, if the current ICRP risk estimates are wrong, it makes them less wrong to assume that the effect observed at Hanford is due to external radiation and not to any very minute internal depositions. Finally, since control for level of internal monitoring reduces to non-significance one of the paradoxical features observed in a first analysis, the true explanation of the paradox must be related to a variable that is highly correlated with level of internal monitoring. As stated in the paper, we have done some analyses, unpublished as yet, which indicate that the correct variable is related to deliberate selection of healthy workers to dangerous jobs.
Hanford radiation study.

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