Letters

To the Editor

Delayed effects of A–bomb radiation

SIR—Recently, Stewart1 presented a reinterpretation of published data on the atom bomb survivors which suggests that previous estimates of radiation health effects derived from these data such as those published in the BEIR III Report2 and the 1977 UNSCEAR Report3 are low by a factor of 10. Since the conventional estimates are widely used in performing risk assessments related to regulatory activities, this finding was of considerable interest to us. On close examination we think that Stewart’s argument supporting her suggestion is questionable on a number of counts.

The contention that previous health effect estimates are low by an order of magnitude is based on a hypothesis that, though the A-bomb survivors show standardised mortality ratios (SMRs) of about 100 for many non-cancer diseases—that is, 100% of the deaths expected on the basis of national age and sex specific death rates or the same mortality experience as the general population—this apparent lack of effect is the product of two strong opposing “silent forces.” The first of these is a “healthy survivor” effect—that is, survivors were subject to considerable mortality immediately after the blasts, and those who were left alive to be incorporated into subsequent health studies were exceptionally healthy. This selection is, in Stewart’s theory, balanced by residual radiation damage to the bone marrow and other organ systems. Thus the survivors are a group of exceptionally healthy individuals that is made only average in mortality experience by residual radiation damage.

It is from a single disease category, cerebrovascular accidents, and a single city, Hiroshima, that Stewart derives her estimate of a residual radiation health effect. Stewart’s basic argument is that since cerebrovascular accidents show a 25% deficit in Hiroshima all other diseases should show SMRs of about 75. Therefore, an SMR of 100 represents a 30% increase over expectation. If one then assumes that the SMR for all deaths observed in the atom bomb survivors is approximately 100 and thus attributes 30% of mortality for all causes in this group to radiation, one obtains a ten-fold increase in radiogenic deaths as compared with conventional estimates that consider only those diseases showing radiation dose-related excess mortality—that is, malignancies.

It is arguably implausible that two such strong “silent forces” should balance almost exactly across a variety of diseases, and the selection of cerebrovascular accidents is at best arbitrary (and at worst circular; the SMRs for cerebrovascular accidents are low because this disease category is assumed to be unaffected by radiation and it is apparently unaffected by radiation because the SMRs are low.) More importantly, both the logic and the arithmetic of the analysis are faulty.

Firstly, it is assumed that at very high doses there is greater selection producing very healthy people which is opposed by correspondingly large residual health damage from radiation exposure. Similarly, at low doses, both the selection and the residual health damage are small. A major problem with this argument is that the SMRs for cerebrovascular accidents, which are supposedly not affected by radiation but which are affected by healthy survivor selection, do not vary appreciably across dose categories. (The zero dose group shows an SMR of 76, the highest dose shows an SMR of 80, while the others range from 72 to 79.) If Stewart’s hypothesis were accepted one would expect an SMR of about 100 in the zero dose (and therefore presumably least selected) group and progressively lower SMRs for higher dose categories because cerebrovascular accidents are supposed to be relatively immune to radiation damage and to reflect primarily the selective component of the atomic blasts. Thus the reference disease does not behave as it should, unless one assumes a constant healthy survivor effect. This, however, would imply that since radiation damage is necessarily dose related, all other diseases should show a trend of increase in the SMR from low to high dose. Such trends are not apparent in most disease categories given by Stewart in her table 2, and blood diseases, the one disease category that does show such a trend, accounts for less than 1% of all deaths. Thus her model does not fit the great majority of the data.

From a computational standpoint, Stewart’s calculation of an order magnitude underestimate of health effects is based on applying a correction of 30% to all deaths regardless of dose category. This is a problem for two reasons. Firstly, the SMR for diseases other than cerebrovascular accidents calculated from Stewart’s table 2 is 87 not 100 for the city of Hiroshima, which implies, if one accepts Stewart’s argument, an excess of 16%, not 30%, for deaths from all causes. One can raise the SMR from 87 to 92 by pooling the Hiroshima and Nagasaki data, but this ignores the fact that the SMR for cerebrovascular accidents for Nagasaki is 95 not 75. If these last two figures are pooled the result is an SMR of 79, which compared with the previous pooled SMR of 92 still suggests only a 16% excess for mortality from all causes.

It may be noted that Stewart deals briefly with the city differences in mortality level and says that they can be explained in terms of the raised August to
December 1945 death rate for first-day survivors in Nagasaki compared with Hiroshima and the higher radiation dose experienced by the Nagasaki survivors. This is taken by Stewart to in some sense support her hypothesis. Nevertheless, the SMR for cerebrovascular accidents is higher in Nagasaki than in Hiroshima so one must assume that higher mortality rates imply less selection. Alternatively, one can assume that cerebrovascular accidents are affected by radiation but show no dose response with increasing exposure in either city. Again, Stewart's argument lacks internal consistency.

Stewart's error in overestimating her correction factor—that is 30% v 16%—is magnified by being applied to all deaths, regardless of dose category. This ignores the fact that most survivors had relatively low doses. Indeed, about a third of Stewart's additional "radiogenic" deaths are attributed to people with zero dose! Stewart's estimate is therefore by her own model much too high. Aside from this, one is led to the unlikely conclusion that very small doses of radiation (less than 10 rad) can cause a 30% differential in mortality from diseases such as tuberculosis or digestive disease.

Stewart's most serious error, however, is one of omission. There was an additional group of 27 000 individuals included in the atom bomb survivor studies who were not in the cities when the bombs fell (the "not in city" cohort). These individuals were thus neither exposed to radiation nor subject to selection. An SMR of 75 was calculated for cerebrovascular accidents in the not in city cohort of Hiroshima. This is almost identical to the overall SMR (76) for cerebrovascular accidents shown by the actual survivors. It seems more credible to attribute the low SMR in Hiroshima to some unidentified set of variables than to embrace a "silent forces hypothesis" which is itself logically inconsistent.

To recapitulate, there are four main objections to Dr Stewart's model. Firstly, it is predicated on somewhat arbitrary and implausible assumptions. Secondly, it is internally inconsistent in that the data she presents do not behave as they should under her model. Thirdly, the model is misapplied to yield larger estimates of excess radiation-induced deaths than would be the case if it were valid, including many radiogenic deaths among those receiving little or no radiation. Finally, it is based on a biased data set, omitting the vital information from the not in city cohort. For these reasons we believe that Dr Stewart has not presented credible evidence that present estimates of radiation-induced health effects are seriously low.

Note: The views expressed here are those of the authors and do not reflect an official position of the Nuclear Regulatory Commission.

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References


Dr Stewart replies:

Ginevan and Puskin are right to insist that "it is arguably implausible that two such strong silent forces should balance almost exactly across a variety of diseases." In the definitive report of 1950–74 deaths, however, there is ample evidence of opposing forces that are not exactly matched.1 In this report all diseases other than neoplasms are compressed into six diagnostic groups and for a residual group (which is larger than any of the disease specific ones) there has been no attempt to distinguish between the two cities, the two sexes, or five exposure age groups. Even so, many dose response curves are more supportive of the "silent forces" than the "cancer only" theory (table 1).

If there had been no radiation effects apart from cancer there would have been no difficulty in fitting the dose-response curve for each group of non-cancer deaths to a smooth horizontal line. Two opposing forces of roughly equal strength would often have evoked this type of dose response, but there would have been other alternatives, such as a curve depicting a rising or falling trend with dose or a curve that failed to pass a homogeneity test because there was a significant peak or trough in the middle of the dose scale. Therefore, given several groups of non-cancer deaths, it would be reasonable to expect more than one type of curve. In fact, all five alternatives can be found in the 1977 report. For
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J Epidemiol Community Health 1983 37: 85-88
doi: 10.1136/jech.37.1.85

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