Letters

Radiation Estimates

The article by Eliot Marshall on the estimates of radiation dose received by the survivors in Hiroshima and Nagasaki (News and Comment, 22 May, p. 900) is essentially accurate concerning what various people have said about the situation, but it gives an erroneous view of the implications. It is unfortunate that, although the revisions proposed by William Loewe and Edgar Mendelsohn at the Lawrence Livermore National Laboratory of the T65 dosimetry have been widely circulated in summary form, they have not been published and therefore have not yet been subjected to the scrutiny of the scientific community. The Livermore revisions are being publicized instead by individuals whose statements concerning them are unfortunately, sometimes in error. Thus, Edward Radford, in a recent statement to the Environmental Protection Agency concerning the proposed Federal Radiation Protection Guidance for Occupational Exposures, stated that ". . . there is indication from the new evaluation that the gamma ray doses in both cities have been overestimated in the 1965 data, and for this reason the risk estimates previously derived even on the linear dose-response hypothesis are too low" Radford is wrong on both counts: Loewe and Mendelsohn assert that the gamma dose in Hiroshima was grossly *under*estimated in the T65 dosimetry, while the gamma doses in Nagasaki and the neutron doses in both cities were overestimated. The net result of all this is that the risks for cancer, per rad, estimated on the linear hypothesis, change very little.

Radiobiologists and students of radiation carcinogenesis have been intensely interested in the contrast between the effects of high LET and low LET (linear energy transfer) radiation because of the light that might be cast on the nature of the intracellular event that results in a cancerous cell. This discussion will have to begin again from square one if the Livermore dosimetry revision, or anything like it, is deemed to be the most likely after probing scrutiny by the scientific community. But the usefulness of the Hiroshima-Nagasaki data for the purposes of radiation protection is not challenged by this dispute concerning the dosimetry, nor are the actual linear risk estimates affected appreciably.

As Marshall makes clear in his article, the workers at Livermore reexamined the Hiroshima-Nagasaki dosimetry at this late date under the impetus of Harald Rossi's contention that the risks from neutrons are larger than had previously been supposed. Since the weapons laboratories have a real stake in the question of the magnitude of risks from neutrons, and hence the appropriate exposure limits, they cannot be regarded as disinterested parties. As Marshall says, the record of this controversy is a compelling argument for bringing the data into the public arena through the traditional modes of open publication and peer review and discussion. It is unacceptable to base discussion of important public policy decisions, such as occupational exposure limits, on rumor, hearsay, and privately circulated, privileged documents.

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We wish to comment on Marshall's article dealing with our new estimates of the Hiroshima and Nagasaki radiation doses (really free-in-air kermas). Of concern to us is an inference drawn by Marshall and by some of the persons whom Marshall interviewed and quoted; this inference in turn sets the tone for the entire article. We take exception to statements that our results show gamma radiation is much more hazardous than previously assumed.

Marshall states that our much lower neutron dose estimates for Hiroshima imply "that the gamma rays were more toxic than had been thought." He fails to mention that the reduction in neutron dose was accompanied by a substantial increase in gamma dose, which counters that argument. The following example illustrates that point:

Hiroshima doses at 2 kilometers.

Estimates	Tissue rads	
	Neutrons	Gamma rays
T65D*	0.54	1.9
LLNL†	0.064	7.7

*Tentative dose estimates compiled in 1965. †Lawrence Livermore National Laboratory estimates.

For Nagasaki, the T65D estimates have shown a predominant gamma dose. Therefore the radiobiological effects observed in Nagasaki have previously been attributed solely to gamma radiation. Although different, to first order our gamma dose for Nagasaki is similar to that of T65D. Thus any conclusions drawn in the past concerning the radiobiological effects of gamma radiation in Nagasaki could not change significantly on the basis of our new dose estimates alone. In addition, leukemia data for Hiroshima now tracks the Nagasaki experience when plotted against our dose values (*1*). We therefore fail to see how our work can be a basis for assigning a greater hazard to gamma radiation than was the case for T65D. (However, see below for additional discussion.)

We believe that definitive conclusions concerning the implications of our new doses with respect to radiobiological effects cannot be drawn until further work has been completed. The data base containing information on the individual Japanese survivors must be revised to reflect our new estimates. In addition, a recalculation of gamma building transmission factors [they might be reduced by a factor of roughly 1.6 (*2*)] and body transmission factors appear to be important. Such new calculations would increase the gamma radiation risk, in proportion to any decrease of the transmission factors.

We also wish to correct the following statement in Marshall's article: "This stalemate existed for several years until the summer of 1980 when Loewe decided to rework the calculations." A correct statement would be: "Unaware of ongoing work at Oak Ridge National Laboratory, in 1978 Loewe and Mendelsohn carried out what they believed to be the first calculations of Hiroshima and Nagasaki doses to have an air-ground interface explicitly included. In August

1980, after identifying significant errors in previous calculations elsewhere and establishing agreement of their own calculations with in situ data at Hiroshima, they presented their results in a preliminary but detailed report which received widespread distribution."

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2. J. Marcum, "House attenuation factors for radiation at Hiroshima and Nagasaki" (Memorandum, R & D Associates, Marina del Rey, Calif., 15 May 1981).

A number of conclusions in Marshall's article do not follow from the new dose estimates for Hiroshima and Nagasaki calculated by Loewe and Mendelsohn.

We have recently reported (*1*) an analysis of the biological implications of the new doses. The Loewe-Mendelsohn estimates (*2*) were combined with published biomedical data from the Japanese A-bomb survivors (*3*) and dose-response relations were analyzed for several major effects, including leukemia, breast cancer, and total malignancies. What the combined data show can be summarized as follows.

- The mortality data for leukemia and total malignancies show dose-response curves for low LET (linear energy transfer) that are clearly sigmoidal in shape; the data cannot be fitted adequately by linear regressions. The carcinogenic efficiency (effect per unit dose) of gamma rays is less at low doses than it is at high doses. In the case of breast cancer incidence, although the situation is not so clear, the data are again well fitted by a sigmoidal (linear-quadratic) curve.
- Because of statistical uncertainties in the A-bomb–survivor data, the dose-response curves are still unable definitively to demonstrate either the presence or the absence of low LET "threshold" doses for human malignancies.
- Risk coefficients for leukemia and breast cancer are consistent with generally accepted values (*4*) at low doses. The low LET coefficient for total malignancies, however, appears lower than the earlier estimates (*4*). Only at high doses, above those relevant to radiation protection standards, do the risk coefficients (for certain malignancies, for example, leukemia) become significantly higher.
- Clear evidence for high neutron RBE (relative biological effectiveness, compared to low LET radiation) is lacking from the new dose-response curves for leukemia and breast cancer (in both cases curves for the two cities are not significantly different from each other). In the case of total malignancies, however, significant differences between the cities are seen. There were more cancer deaths in Hiroshima than in Nagasaki at any given dose. If these differences are a result of the larger neutron component in Hiroshima (the new dose estimates still show more neutrons in Hiroshima than in Nagasaki), the data suggest that the RBE of neutrons increases with decreasing dose (because of the decreasing effectiveness of gamma rays) and may reach quite high values; interestingly, a significant fraction of the A-bomb–related cancer deaths would appear to be the result of neutron radiation [this was also found for the earlier T65 doses (*5*)].

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4. *International Commission on Radiological Protection Publication 26* (Pergamon, Oxford, 1977), p. 10; *United Nations Scientific Committee on the Effects of Atomic Radiation 1977 Report* (United Nations, New York, 1977), p. 413; *Report of the Advisory Committee on the Biological Effects of Ionizing Radiations* (National Academy of Sciences, Washington, D.C., 1977), p. 171.
5. H. H. Rossi, *Radiat. Res.* **84**, 395 (1980).

Marshall generally associates those named in his article on Japanese A-bomb survivor dose estimates with the proposition that the likely outcome of dose reassessment will be to increase the risk of cancer ascribed to exposure to low LET radiation. I wish to disassociate myself from this thesis.

The reanalysis of the Japanese dosimetry is a complex task, affecting not only the neutron dose but the gamma ray dose as well. Evidence presented to date suggests that, while estimates of neutron doses at both cities may decrease, the estimate for the Hiroshima gamma ray dose may increase relative to that at Nagasaki. The degree to which these variations may occur has not yet been established and depends on the device radiation output, atmospheric conditions, and local shielding. To date, only the effects of the former two parameters have been investigated by Science Applications, Inc., Oak Ridge National Laboratory, and Lawrence Livermore National Laboratory.

It may well be asked why the T65D estimates are being questioned now, 15 years after their publication. The reason for this is that the T65D values were derived almost wholly from measurements made during field tests of weapons and other radiation sources in the 1950's and early 1960's. As such, claims of substantial accuracy could be made for them at a time when purely analytical methods of predicting device radiation output, transport and shielding effects were in their infancy and their results subject to considerable uncertainty. Since that time considerable advancement has been made in the development of analytical techniques and the physical data required for their use. Within the last 5 years this advancement has reached the stage at which the dose measurements made during the atmospheric tests can be reproduced analytically to within the uncertainty of the measurements themselves. The claim by Rossi and Mays (*1*) that the risk of leukemia from neutron exposure should be raised by a factor of 10 based on current A-bomb survivor epidemiology and the T65D estimates provided the impetus for the reanalysis of survivor dosimetry, using state-of-the-art analytical techniques, and precipitated the current controversy.

State-of-the-art analytical methods and data may be used successfully to produce highly accurate dose reconstructions for Japanese A-bomb survivors. However, current results from such efforts must be considered incomplete and preliminary. Many effects, such as those of radiation free-field variations on the character of local shielding, have yet to be determined. The scientific community will require substantial proof of the accuracy of the analytical techniques before accepting results of such methods in lieu of the largely empirical T65D values. The programs to produce this information are just how beginning.

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