Note

HIGH-ENERGY GAMMA RAYS IN HIROSHIMA AND NAGASAKI: IMPLICATIONS FOR RISK AND w_R

Tore Straume*

Abstract—**Based on the DS86 dosimetry system, nearly all of the dose to survivors of the atomic bombings of Hiroshima and Nagasaki was due to unusually high-energy gamma rays, predominantly in the 2- to 5-MeV range. These high energies resulted in part from neutron capture gamma rays as the bomb neutrons penetrated large distances of air. Because of the inverse relationship between energy and biological effectiveness, these high-energy gamma rays are expected to be substantially less effective in producing biological damage than the radiations commonly used in radiobiology and risk assessment. This observation has implications for radiation protection and risk assessment.**

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INTRODUCTION

RISK ESTIMATES for radiation-induced cancer (e.g., BEIR 1990; ICRP 1991), which serve as the foundation for radiation dose limits, are based principally on data from atom-bomb survivors exposed in 1945 in Hiroshima and Nagasaki. A fundamental assumption implicit in the use of these data for the setting of dose limits is that the atom-bomb gamma rays were of identical effectiveness to other low-LET radiations such as orthovoltage (i.e., 250 kVp) x rays for which the radiation weighting factor (w_R) is defined as unity (ICRP 1991). Here this assumption is evaluated and it is demonstrated that the survivors were actually exposed to unusually high-energy gamma rays, which at low doses or low-dose rates are expected to be less effective than common x rays and beta rays. This has important implications for both risk assessment and the setting of dose limits.

RADIATIONS IN HIROSHIMA AND NAGASAKI

The atom bombs dropped on Hiroshima and Nagasaki emitted gamma rays and neutrons (RERF 1987). Gamma-ray-energy spectra for Hiroshima and Nagasaki are plotted in Fig. 1. The free-in-air DS86 spectra are kerma-weighted at 1,000 m ground range and include contributions from "early gammas," which include prompt fission gamma rays and neutron capture gamma rays, and from "late gammas," which include delayed fission gamma rays and fission product gamma rays. It is observed that the gamma-ray spectra for Hiroshima and Nagasaki are similar.

Early gammas, which are principally from neutron capture reactions in the air and in bomb materials, have kerma-weighted mean energy of 4.7 MeV in Hiroshima and 4.2 MeV in Nagasaki. These are substantially higher energies than those commonly used in radiobiological studies from which w_R and dose-rate effectiveness factors (DREFs) are obtained and much higher than the 250-kVp x rays used as the "standard" for radiation protection purposes. These high-energy early gammas contributed about half of the dose to the survivors (RERF 1987).

Late gammas, with mean energy of 1.8 MeV in Hiroshima and 1.7 MeV in Nagasaki contributed nearly all of the rest of the dose. Thus, based on the DS86 dosimetry system, high-energy gamma rays contributed about 98 to 99% of the absorbed organ dose in Hiroshima and essentially all of the dose in Nagasaki (RERF 1987).

The small remainder of the DS86 dose was contributed by neutrons, i.e., less than 1 to 2% in Hiroshima and an even smaller amount in Nagasaki. The DS86 neutrons were accounted for in BEIR 1990 using a quality factor of 20. It should be noted that the DS86 neutron contribution is presently uncertain in Hiroshima (Straume et al. 1992) and requires resolution before accurate risk estimates are possible using the Hiroshima survivor data (Straume 1993). In contrast, DS86 neutrons have now been confirmed in Nagasaki (Straume et al. 1994), and thus those data can be used confidently in risk assessment.

In either city, the mean energy for all gamma rays (early and late gammas combined) at 1,000 m ground range is calculated using DS86 to be ~3 MeV outside in the open and slightly higher in the active bone marrow of a survivor located inside a standard wood frame Japanese house (RERF 1987).

* Health and Ecological Assessment Division, Lawrence Livermore National Laboratory, University of California, P.O. Box 808, Livermore, CA 94550.

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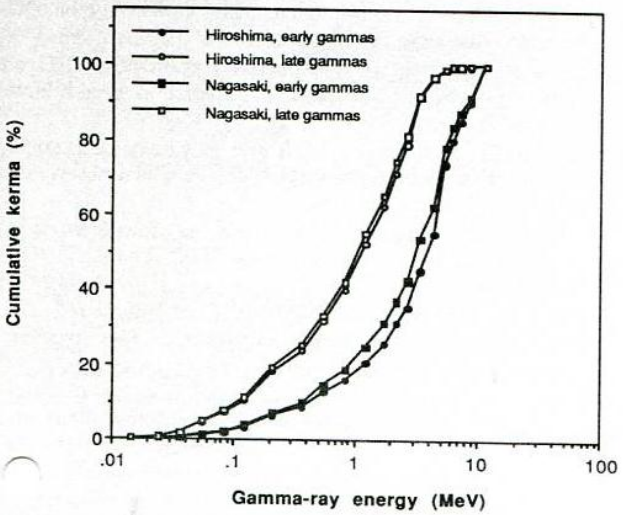

Fig. 1. Cumulative energy distributions for gamma rays in Hiroshima and Nagasaki at 1,000 m ground range. These are kerma-weighted distributions obtained by multiplying the gamma-ray fluence per energy interval by the respective kerma factor. The free-in-air energy distributions are for the DS86 dosimetry system (RERF 1987). A slight increase in the mean gamma-ray energy is observed when the gamma-rays are transported inside a wood-frame Japanese house and into the active bone marrow of a standard a-bomb survivor (see text).

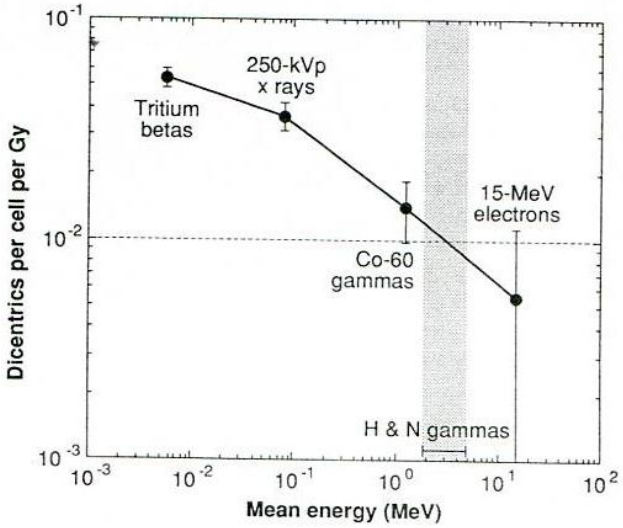

Fig. 2. The biological effectiveness of low-LET radiations. The data are the low-dose linear slopes of the linear-quadratic dose-response curves for chromosome dicentrics induced *in vitro* in human lymphocytes exposed to the radiations indicated and evaluated at the first cell division. Data for tritium beta rays are from Prosser et al. (1983); data for 250-kVp x rays and ^{60}Co gamma rays are from Lloyd et al. (1986); and data for 15-MeV electrons are from Purrott et al. (1977). Note that all of the data are from the same laboratory, the National Radiological Protection Board, UK.

BIOLOGICAL EFFECTIVENESS VS. ENERGY

It is well known that biological effectiveness decreases as radiation energy increases, i.e., becomes less densely ionizing (Dobson and Kwan 1976; Bond et al. 1978; NCRP 1980; Borek et al. 1983; ICRU 1986; Brenner and Amols 1989; NCRP 1990). This is illustrated in Fig. 2 where the measured biological effectiveness is plotted against mean radiation energy. For this illustration, we used data for dicentric aberrations in human lymphocytes irradiated *in vitro*. The data are the linear slopes of the linear-quadratic dose-response curves for the four different radiations. This slope, if adequately measured, provides a quantitative comparison of the effectiveness of the different radiations at low doses or low-dose rates (NCRP 1980). All of the data in Fig. 2 are from the same laboratory, the National Radiological Protection Board, Harwell, UK, and should therefore not exhibit the variability usually associated with interlaboratory comparisons.

A factor of ~10 decrease in effectiveness is observed in Fig. 2 between tritium beta rays (0.0057 MeV, mean) and 15 MeV electrons. The relative slopes within this energy range (assuming 1.0 for the atom-bomb gamma rays) are about 0.5 for 15-MeV electrons, ~2 for ^{60}Co gamma rays, ~4 for 250-kVp x rays, and ~5 for tritium beta rays. These are substantial differences.

The dependence of human cancer dose-response relationships on radiation energy has not been established and therefore may or may not be equivalent to that for the model endpoint (dicentrics) used here. It is, however, established that the energy dependence of dicentrics compares well with those of a broad range of other biological endpoints (NCRP 1990), including that for malignant cell transformation (Borek et al. 1983), and is a convenient endpoint that has been well characterized and widely used for similar purposes (e.g., see ICRU 1986).

Because of the scarcity of radiobiological data for Hiroshima-like gamma rays, it is strongly recommended that robust low-dose or low dose-rate curves be obtained for 3- to 5-MeV photons.

IMPLICATIONS FOR RISK AND w_R

If the induction of human cancers responds to radiation quality in a manner similar to that observed for dicentrics, Fig. 2 suggests that a large discrepancy could exist between the low-dose risk coefficients estimated from the atom-bomb survivor data and risks from low-level exposure to 250-kVp x rays and tritium beta rays. However, the actual impact on risk estimates of the unusually high-energy gamma rays in Hiroshima and Nagasaki depends on the method used to estimate risk at low doses and low doses rates. If risk coefficients obtained from the high dose and dose-rate exposures received by atom-bomb survivors are reduced using a DREF (or obtained directly from the low-dose linear slope of a linear-quadratic fit to the data), the resultant risk estimates would be for high-energy gamma rays.

Based on Fig. 2, these risk estimates would be expected to substantially underestimate the risks for low energy medical x rays and tritium beta rays.

In contrast, if the atom-bomb data are used without making allowances for reduced effectiveness at low doses and dose rates (as was the case in BEIR 1990 for non-leukemia cancers), then the risk estimates for high-energy gamma rays may be substantially overestimated.

The high-energy gamma rays in Hiroshima and Nagasaki also impact w_R. The current standard is to use $w_R = 1$ for photons and electrons of all energies (ICRP 1991). However, if dose limits are based on the atom-bomb data (as was the case in ICRP 1991), then more appropriate values for w_R may range from ~0.5 for 15-MeV electrons to ~5 for tritium beta rays. The large range in biological effectiveness between 15-MeV electrons and tritium beta rays is not a new observation (ICRU 1986). However, the unusually high gamma-ray energy in Hiroshima and Nagasaki is a new finding and was not known prior to DS86. Because the atom-bomb data are so widely used in radiation protection and risk assessment, the important differences in effectiveness values are no longer those between, e.g., ^{60}Co and orthovoltage x rays, but rather those between Hiroshima/Nagasaki gamma rays and orthovoltage x rays or tritium beta rays. That difference would appear to be substantially larger, perhaps in the 4 to 5 range.

CONCLUSION

The unusually high-energy gamma rays in Hiroshima and Nagasaki demand a careful evaluation of their biological effectiveness and of their implications for risk assessment and radiation protection standards. It would appear clear now that the use of a single risk and w_R or Q value for all low-LET radiations may be inappropriate and can lead to large errors, particularly for tritium beta rays and medical x rays. A practical solution may be to use ^{60}Co gamma rays instead of 250-kVp x rays as the "standard" radiation for radiation protection purposes and then develop the data needed to scale to the other energies. Although ^{60}Co gamma rays may be somewhat more effective than Hiroshima and Nagasaki gamma rays, ^{60}Co sources are readily available and can be used for both chronic and acute exposures.

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