RISK ASSESSMENT OF EXPOSURE TO IONIZING RADIATION

ANOTHER VIEW*

by

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In the early period, up to about 1950, essentially all the scientific community subscribed to the threshold hypothesis; namely, there is a safe, low level of exposure to ionizing radiation below which no harm will result to the exposed individual. During this period, most of the concern was directed toward preventing large occupational exposure where there might be early manifestations of the radiation syndrome in the form of a threshold erythema, changes in the differential blood count or a general rundown in health requiring an extended vacation for the radiation worker as had been the assumption and practice in several European countries. Very little consideration was given during this period to maximum permissible exposure (MPE) levels for members of the public, but data on fruit flies studied by Muller[1] suggested that these MPE levels should be based on prevention of excessive radiation-induced genetic mutations. During the past three decades there has been a large ongoing program of research in many countries of the world on the induction of malignancies by intermediate and low levels of radiation exposure to animals and man. As the follow-up periods in these studies have been extended, malignant tumors (especially those with long latent periods in man) have shown a surprising incidence even at low exposure levels. As a consequence, levels of MPE have, from time to time, taken downward quantum jumps. For example, the MPE for occupational workers has dropped approximately by a factor of ten since 1950, and the MPE for members of

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the public has dropped by a factor of sixty (from 1.5 rem/y to the present EPA level of 25 mrem/y) since 1952. This increasing concern for the risk of cancer from low-level exposure to ionizing radiation is exemplified by a statement of the International Commission on Radiological Protection in 1971,[2] "It could be concluded that the ratio of somatic to genetic effects after a given exposure is sixty times greater than was thought fifteen years ago."

Perforce, during the early period most of the studies on the effects of low-level exposure were conducted on mice, rats and other animals rather than on man. These animal studies in many cases grossly underestimated the cancer risk to man because of the greater radiosensitivity of man and because many types of cancer have incubation periods of ten, twenty, and fifty years, i.e., longer than the life span of most experimental animals, and cancer incidence relates more to time since a given exposure than to the fraction of life span under observation since the exposure. In recent times it has been possible to conduct a limited number of epidemiological studies of humans exposed to low levels of radiation (Oxford in utero x-ray exposure studies of Stewart and Kneale;[3] studies of Modan et al.[4] of persons whose scalps were x-rayed for ringworm; studies of Hanford radiation workers by Mancuso, Stewart and Kneale;[5] etc.). These studies have revealed a cancer risk that is ten to fifty times the risk suggested from many of the animal studies or as indicated by studies of survivors of atomic bombings of Hiroshima and Nagasaki and of patients treated therapeutically with x-rays, especially the x-ray treated patients with ankylosing spondylitis. Unfortunately, the standards setting bodies have, for the most part, accepted these two human studies (i.e., Japanese survivors and spondylitis patients) as though they were gospel truth and have not attempted to examine the serious biases they introduce which cause them to grossly underestimate the radiation risk. The most significant of these biases were the fire, blast and traumatic situation faced by

the Japanese survivors of a catastrophic experience and the large radiation doses they received, which caused them (and especially the weaker members who had a large cross section for developing cancer) to die early of common diseases long before they had time to manifest a malignancy. A somewhat similar bias existed in the case of patients with ankylosing spondylitis. These were sick persons suffering with a painful and serious disease such that studies of Radford et al.[6] indicated they too died early of common diseases--pneumonia, chronic bronchitis, influenza, peptic ulcer, gastrointestinal disease, tuberculosis, cerebrovascular disease, etc.--during the usual latency period of most cancers. Kneale and Stewart[7,8] have shown that persons with in situ cancer (a malignancy in early stages of development) have a propensity, a large cross section for, or are in grave danger of dying from secondary infections and accidents before malignancies are diagnosed clinically. This is shown to result from the fact that the precancer state is associated with lowered immunological competence. In the case of survivors of bombings of Hiroshima and Nagasaki and of patients suffering from a fatal disease such as spondylitis, the insults of fire, blast, deprivation and radiation exposure along with disease further weakened the reticuloendythelial system of the likely candidates of a radiation-induced malignancy such that they could no longer fight off successfully the ravages of common diseases. In the case of the Japanese, many of those who avoided or survived these diseases succumbed to cancer, the leukemias reaching a peak of incidence during the period of six to eleven years after exposure. Later, and even now, all other types of malignancy with longer latency periods have been on the increase. The amount of increase is related to the magnitude of the radiation exposure.

One may ask why is it that the standards setting bodies (ICRP, NCRP, UNSCEAR, BEIR Committee, etc.) have been so reckless in their willingness to

accept data from the Japanese survivors and the spondylitis patients as hallmark references to the exclusion of other data and to use these data without attempts to correct for the above mentioned serious biases. I, of course, cannot answer this question. One interpretation would be that consciously or subconsciously many members of these bodies wanted to find that the risk of low-level exposure is very small or completely negligible. I, however, would like to render the more charitable explanation that when the above needed corrections to the Japanese survivor data and the spondylitis patient data were not made, there was a reasonably good fit to the data on short-lived animals for which there is a large amount of data, so they did not attempt to solve the extremely difficult question of how one could reasonably go about making corrections for these biases.

This paper is concluded by pointing out that although the standards setting bodies have discarded the threshold hypothesis in favor of the linear hypothesis, many of their individual members have expressed the opinion that the linear hypothesis greatly exaggerates the risk of low-level exposure to ionizing radiation. This may be true for some types of radiation exposure, but there is very strong evidence that in many (if not in all) cases of human exposure to low-level radiation, the linear hypothesis seems to underestimate the radiation risk, and the best fit to the data is a super linear relationship.

In the simple case, the risk of cancer from low-level exposure to ionizing radiation may be given by the relation $P(d) = a + bd^k$ in which $P(d)$ is the probability of succumbing to a malignancy from a dose d(rem), and a, b and k are constants. When $k = 1$ we have the linear hypothesis, when $k > 1$ we have the threshold hypothesis, and when $k < 1$ we have the superlinear hypothesis. Baum[9] was one of the first of a number of researchers to show that $k < 1$, or the superlinear relation gave the best fit for a number of malignancies among the

survivors of Hiroshima and Nagasaki bombings (i.e., k = 0.5 for all malignancies at Hiroshoma, k = 0.8 for acute leukemia at Nagasaki, k = 0.86 for leukemia at Hiroshima; and for the combined cities k = 0.19 for lung cancer, k = 0.35 for stomach cancer and k = 0.5 for female breast cancer. A series of papers[10-12] strongly suggests that the induction of thyroid carcinoma at low doses of ionizing radiation is more serious than was thought a decade ago and that $k < 1$, or it too is best represented by a superlinear relation to dose.

In their analysis of the ankylosing spondylitis data the GAO[13] concluded, "All mixed models tested did much better than the linear model, and the unusual square root-cubic model did the best of all." Since at doses less than 100 rem their cubic term contributed $<<$ 1% to the cancer risk P(d), this means that at low doses the best fit related to k = 0.5 or $P(d) \propto \sqrt{d}$. The GAO[13] concluded that for the Japanese survivors, "Dose-response curves that were square root, linear, quadratic or cubic at low levels all gave acceptable fits for at least one set of data" and that "highly sensitive groups at low doses could lead to dose-response curves for the entire population that shows larger effects per rad at low than at high doses", i.e., a superlinear relationship. They went on to point out that, "The shape of the radiation dose-response curves would not be affected by the response of subgroups that are moderately sensitive or highly resistant to radiation." The BEIR-80[14] Committee stated, "the existence of exquisitely sensitive subgroups of suitable size conceivably would produce a dose-response curve that showed a greater effect per rad at very low doses than at high." I believe there is evidence for the existence of such subgroups in a heterogeneous population of humans that may not be apparent in a group of the usual homogeneous animals that are studied to find dose-effect relationships and that the results of such animal studies can and have led to false assumptions about human populations.

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