Rational Risk Estimation in Relation to
Atomic Bomb Radiation

SOHEI KONDO

Atomic Energy Research Institute, Kinki University,
Kowaka, Higashiosaka, Osaka 577, Japan
(Received September 29, 1989)
(Revised version, accepted April 2, 1990)

Atomic bomb radiation/Model of radiation carcinogenesis/Tumor progression/Threshold effect/Risk at 1 cGy

This paper summarizes genetic and somatic data on persons exposed to low doses of atomic bomb radiation in Hiroshima and Nagasaki. Compared with experimental estimates, the new dosimetry system proposed in 1986 underestimates neutron doses, supporting qualitatively the conclusion by the 1965 dosimetry system that Nagasaki A-bomb emitted predominantly gamma rays whereas Hiroshima A-bomb emitted both gamma rays and fast neutrons. A theory based on two recessive mutations in hemopoietic stem cells is proposed to explain radiation leukemogenesis. The theory can explain, at least partly, the actual dose-response curve for incidences of acute leukemia in Hiroshima but cannot explain chronic leukemia in Nagasaki. Existence of a large threshold dose in the latter's dose relationship supports the hypothesis that A-bomb radiation at high doses above a threshold value was a promoter and/orgressor of leukemia. Various lines of evidence that support this hypothesis are presented. Hence, it is not warranted to assume that risk of death from cancer at a high dose, say, 1 Gy can be divided by 100 to obtain the risk at 1 cGy. Risk at low doses should be assessed by direct scrutiny of actual data at low doses in spite of their large statistical uncertainty. Actual data show that A-bomb survivors at 1–9 cGy had apparently lower incidences of tumors than unexposed persons.

INTRODUCTION

Reactor accidents at Three-Mile Island and at Chernobyl convinced most of the public that radioactive fallout from reactor accidents is so dangerous to them and their offspring that the generation of atomic power should be abandoned in the near future. Many lines of evidence have been accumulating, however, to show that a crisis of greenhouse effects is approaching and that its major causes are excessive use of fossil fuels and the burning of natural forests. The best alternative energy source is solar energy, but it will take us a long time to adapt to a solar energy civilization. For the time being, the generation of atomic energy will serve as the major energy source in our daily lives.

This paper reviews actual data on persons who were exposed to low doses of atomic bomb (A-bomb) radiation. The results indicate that low doses of radiation are not as hazardous to human
survival as the public believes.

GENETIC EFFECTS ON CHILDREN OF A-BOMB SURVIVORS

In painstaking, long-term studies carried out between 1948 and 1987, no significant increase in the frequency of genetic abnormalities was seen in the children of A-bomb survivors.

Untoward pregnancy outcomes
The incidence of untoward outcomes of pregnancy (still births, major congenital defects, deaths during the first postnatal week) was 4.78% (408/8537) in children whose parents had received an average dose of about 50 rem, and 4.75% (2924/61545) in control children\(^1\).

Deaths of live-born children
The frequency of death before the age of 17 years was 6.3% (737/11736) in children whose parents had received an average dose of about 50 rem, and 6.4% (2494/38953) in control children\(^1\).

Chromosomal abnormalities
The frequency of aneuploidy and structural rearrangements was 0.52% (30/5762) among children whose parents received an average dose of 45 rem, and 0.49% (25/5058) among control children\(^3\).

Mutations resulting in altered electrophoretic mobility of blood proteins
The mutation frequency was \(4.5 \times 10^{-6}\) (3/670000) per locus per generation among children whose parents had received an average dose of 24 rem, and \(6.4 \times 10^{-6}\) (3/470000) among control children\(^3,4\).

GENOTOXIC EFFECTS ON A-BOMB SURVIVORS

For three somatic effects, an approximately linear or linear-quadratic dose-response relationship with radiation was seen, although a threshold dose was found in some cases.

Mental retardation\(^5,6\)
The incidence of mental retardation among children in Hiroshima who had been exposed in utero and tested at the age of 17 years increased significantly above control level, at a rate of about \(10^{-3}/\text{cGy}\) after a threshold dose of 10–30 cGy. Radiation from the A-bomb dropped in Nagasaki caused less severe teratogenic damage than that from the Hiroshima A-bomb. This difference can be explained by the fact that only the radiation from the Hiroshima A-bomb contained a high level of neutrons (see below for details).
Chromosomal aberrations of stable types in peripheral lymphocytes\textsuperscript{7–9)}

Chromosomal aberrations in peripheral lymphocytes from A-bomb survivors have been examined extensively to investigate the genotoxic effects of A-bomb radiation. The frequencies of aberrations were scattered widely in individual donors within subgroups exposed to equal doses; however, clear dose-response relations were found when the mean frequencies for subgroups at different dose intervals were plotted against dose.

The average frequency of aberrations in samples obtained in 1968–80 increased linear-quadratically with increasing dose of A-bomb radiation, as follows\textsuperscript{9)}:

\[
y = c + dx + ex^2,
\]

where \( y \) is the \% frequency of aberration-bearing cells, \( c \) the spontaneous rate, \( d \) the coefficient of the linear term, \( e \) the coefficient of the quadratic term and \( x \) (Gy) the radiation dose; \( c = 1.3 \) (Hiroshima) or 1.4 (Nagasaki), \( d = 5.4 \) (Hiroshima) or 1.1 (Nagasaki) per Gy, and \( e = 1.4 \) (Hiroshima) or 3.3 (Nagasaki) per Gy\(^2\).

In survivors of both the Hiroshima and Nagasaki A-bombs, the frequency of aberrations increased with increasing dose only above an apparent threshold dose of about 10 cGy\textsuperscript{9)}.

Aberration frequencies are not significantly different in cells sampled in 1968–71 and those sampled in 1978–81\textsuperscript{7,8). This finding supports the notion that most of the lymphocytes with aberrations were replicas of ancestral mutant stem cells, which had been produced among the hematopoietic stem cells shortly after exposure to A-bomb radiation and thereafter continued to produce new cells with specific chromosomal aberration markers.

Somatic mutations at the glycophorin A locus in A-bomb survivors\textsuperscript{10)}

Blood samples from Hiroshima A-bomb survivors who were heterozygous for codominant alleles M and N of glycophorin A (a cell-surface glycoprotein on erythrocytes) were used to detect the following erythrocytes of variant types ('mutants'): M/O (having the M form but lacking the N form), N/O (N without M), M/M (expressing the M form twice), and N/N. The results of Jensen and coworkers\textsuperscript{10)} can be approximated by the linear dose-response relationship:

\[
y = a + bx,
\]

where \( y \) is the frequency of mutant cells in the samples from persons exposed to dose \( x \) (cGy), \( a \) is the frequency in controls, and \( b \) is an average rate per cGy of increase in frequency. On the basis of a reassessment of samples from the same donors using the new dosimetry system, DS86, and a modified technique with a single beam flow-cell sorter\textsuperscript{11)}, \( a = 24 \times 10^{-6}/\text{locus} \) for mutation M/O or N/O and \( 15 \times 10^{-6}/\text{locus} \) for mutation M/M, and \( b = 3.3 \times 10^{-7}/\text{locus/cGy} \) for mutation M/O or N/O and \( b = 1.4 \times 10^{-7}/\text{locus/cGy} \) for mutation M/M. These values are slightly different from the corresponding values originally reported\textsuperscript{10)}. 

NII-Electronic Library Service
CANCERS IN A-BOMB SURVIVORS

I describe here a classification of dose-response relationships and cancer incidence at low doses.

Dose-response relationships for cancer mortality

Using the DS86 dosimetry system, relationships between mortality rates from cancers in 1950–85 and radiation dose have been reassessed\(^2\).

Apparently beneficial effects of low doses of A-bomb radiation on the risk of death from cancer

As seen from Figure 1 for death rates at low doses\(^2\), the dose-response curves for most cancers have troughs at the low-dose intervals, 1–5, 6–19 or 20–49 cGy. In other words, appropriately low doses of radiation apparently reduced cancer incidence – an indication of beneficial effects of low doses of radiation on human survival\(^3\).

Whether low doses of radiation really did have beneficial effects in A-bomb survivors cannot be concluded from the epidemiological data alone because of the large statistical uncertainty at each trough. With this reservation in mind, apparently beneficial effects are expressed in terms of an apparent threshold dose \(D_{th}\) given in Table 1 (see footnote c to Table 1 for the definition

![Graphs of cancer rates vs. time](image.png)

**Fig. 1.** Dose-response curves for rates of deaths from five types of cancer in survivors of A-bomb irradiation at low doses in Hiroshima (——) and Nagasaki (-----) (reproduced by extracting the inserted figures only from Shimizu et al. (1987))
Table 1. Characteristics of dose-response relationships for cancer mortality\textsuperscript{a)}

<table>
<thead>
<tr>
<th>Cancer site</th>
<th>Spontaneous rate (per $10^4$ person.years)</th>
<th>Induced rate at 300 cGy</th>
<th>Apparent threshold dose (cGy)$^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Nagasaki</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukemia</td>
<td>0.4</td>
<td>6</td>
<td>36</td>
</tr>
<tr>
<td>Colon</td>
<td>0.8</td>
<td>-0.7</td>
<td>54</td>
</tr>
<tr>
<td>Stomach</td>
<td>6.3</td>
<td>8.5</td>
<td>Nonexistent</td>
</tr>
<tr>
<td>Breast</td>
<td>1 (2)$^b)$</td>
<td>(2)$^b)$</td>
<td>5</td>
</tr>
<tr>
<td>Lung</td>
<td>2.6</td>
<td>(1)$^b)$</td>
<td>28</td>
</tr>
<tr>
<td><em>Hiroshima</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leukemia</td>
<td>0.8</td>
<td>12.5</td>
<td>12</td>
</tr>
<tr>
<td>Colon</td>
<td>1.2</td>
<td>7</td>
<td>31</td>
</tr>
<tr>
<td>Stomach</td>
<td>10</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>Breast</td>
<td>1.0</td>
<td>3.5</td>
<td>Nonexistent</td>
</tr>
<tr>
<td>Lung</td>
<td>2.4</td>
<td>2.5</td>
<td>Nonexistent</td>
</tr>
</tbody>
</table>

\textsuperscript{a)} Extracted from Shimizu et al. (1987, 1989).
\textsuperscript{b)} Average of a value at 3 Gy, which happened to be close to zero, and a maximal value at about 1 Gy (see ref's 12-14).
\textsuperscript{c)} Upon survey of the dose-response curves in Fig. 1, if death rates from particular cancers at doses between 0 and $D_{th}$ are below the spontaneous level, we conclude that there exists an apparent threshold dose $D_{th}$ for induction of that particular cancers by radiation.

of $D_{th}$). Apparently beneficial effects of low doses with $D_{th}$ value 28 cGy or larger are seen for three of five types of cancer in Nagasaki and for one of five in Hiroshima. Furthermore, at 3 Gy, radiation-induced rates of death from four of the five types of cancer were higher in survivors in Hiroshima than in those in Nagasaki, as shown in Table 1.

These two types of differences between the two cities may be, at least partly, explained by a higher biological effectiveness of the Hiroshima A-bomb radiation. In fact, the Nagasaki A-bomb emitted practically only gamma rays whereas the Hiroshima bomb emitted a considerable amount of fission neutrons in addition to gamma rays, as described in the next section.

**DIFFERENCE IN QUALITY OF RADIATION BETWEEN HIROSHIMA AND NAGASAKI A-BOMBS**

*Physical dosimetry of A-bomb radiation*\textsuperscript{15)}

The new DS86 dosimetry system proposed in 1986 provides larger and smaller estimates,
respectively, for the emissions of gamma rays and fast neutrons from the Hiroshima A-bomb than the old T65D dosimetry system proposed in 1965; however, the two systems give similar estimates for the doses of gamma rays and fast neutrons from the Nagasaki A-bomb (Fig. 2).

The DS86 doses are much closer to experimental estimates of the dose of gamma rays from the Hiroshima A-bomb than the T65D doses, although, at ground distances of 1.4–2 km from the hypocenter (the center of the A-bomb radiation), DS86 doses are still 20–70% smaller than experimental estimates\textsuperscript{16,17}.

With regard to the emission of fast neutrons from the Hiroshima A-bomb, the DS86 system gives an estimated dose that is lower by an order of magnitude than that by the T65D system. For example, at 1.2–1.4 km from the hypocenter, the relative ratio of fast-neutron dose to gamma-ray dose is about 50% in terms of T65D and about 3% in terms of DS86, as shown in Figure 2; however, for neutron doses at 0.8–1.3 km from the hypocenter, DS86 estimates tend to be underestimates by factors of 2 to 10 with increasing distance, in comparison with experimental estimates based on induced radioactivities of $^{32}$P\textsuperscript{18–20} (see Fig. 3), $^{60}$Co\textsuperscript{15,21} and $^{152}$Eu\textsuperscript{22–25}. These calculations strongly suggest that the DS86 system gives underestimates of the neutron dose emitted by the Hiroshima A-bomb. Estimates of the amount of fast neutrons emitted by the Nagasaki A-bomb were negligibly small compared with those for gamma rays (Fig. 2). Hence, as concluded by use of the T65D system, it is true that the Nagasaki A-bomb emitted predominantly gamma rays, whereas the Hiroshima A-bomb emitted both gamma rays and fast neutrons.

**Biological dosimetry of A-bomb radiation based on chromosomal aberrations**

Estimations of biological doses of radiation emitted by the Hiroshima and Nagasaki A-bombs on the basis of chromosomal aberrations support the conclusions reached by physical dosimetry.
Fig. 3. Comparison of measured and calculated $^{32}$P activities. ▲, Arakatsu (1953); △, Yamasaki and Sugimoto (1953). (reproduced with modification from ref. 25)

The relative biological effectiveness of $^{235}$U fission neutrons for induction of chromosomal aberrations in vitro in human lymphocytes was about 5–10, in comparison with $^{60}$Co gamma rays for the dose range of 80-20 cGy. These values were practically independent of changes in the neutron energy spectrum achieved by filtering fission neutrons with 5-cm blocks made of plastic, bismuth or iron$^{26}$.

The dose-response relationship of chromosomal aberrations in Hiroshima survivors is more nearly linear than that for Nagasaki survivors, and the aberration frequency in Hiroshima survivors was higher over the whole dose range than that in Nagasaki survivors, as indicated by the derived values for Eq. (1)$^{9}$.

**MECHANISM OF RADIATION CARCINOGENESIS**

*Three-step model of carcinogenesis*

I assume the following three-step model of carcinogenesis (Fig. 4) as recently proposed by Pitot$^{27,28}$.
A-BOMB RADIATION AT LOW DOSES

Fig. 4. Three-step model of carcinogenesis. Initiation (-----), is an irreversible step by mutation, promotion (-----) a reversible step and progression (-----) an irreversible step by continued alterations in the genome and the cellular phenotype.

(i) The first step in carcinogenesis is called 'initiation'. We assume that tumor initiation is an irreversible event induced by somatic mutation in immortal, multipotent stem cells in a target tissue. Initiated mutant stem cells are called here precancerous cells.

(ii) The second step in carcinogenesis is called 'promotion'. This step occurs when endogenous or exogenous factors promote a reversible increase in the rate of replication of the progeny of an initiated stem cell.

(iii) The third step in carcinogenesis is called 'progression'. This is the stage in which initiated cells acquire the "characters" of a neoplasm. Neoplastic characters include increased growth rate, invasiveness, metastatic potential and capability and abnormal hormone responsiveness and morphological characteristics. They are thought to result from irreversible, continued alterations to the cell genome in the progeny of initiated stem cells.

In experimental cancers produced with appropriate carcinogens, dose-response curves exhibit a threshold for both promotion and progression\textsuperscript{28,29}, but the dose-response for tumor initiation caused by radiation-induced somatic mutation is assumed to be linear because the frequency of mutant cells among red blood cells from A-bomb survivors increases linearly with increasing dose of radiation, as shown in Eq. (2) and derived values. Hence, scrutiny of the shapes of the dose-response curves for cancers produced in A-bomb survivors\textsuperscript{28,29} can show whether A-bomb radiation acted as an initiator or as a promoter-progressor in human carcinogenesis. Leukemia in A-bomb survivors is considered as an example in the next section.

Theory of radiation leukemogenesis by two recessive oncogenic mutations\textsuperscript{13}

Leukemias arise from hematopoietic cells. Using the model of tumor initiation by stem-cell mutation, we can assume that the frequency $F$ of an initiated, preleukemic stem cell induced
in hematopoietic stem cells in an A-bomb survivor exposed to dose $D$ (cGy) is as follows:

$$F = s(a' a'' + a' b'' D + b' D a') + s(a' a' + a' b' D)/2,$$

(3)

where $a'$ is the spontaneous and $b'$ the radiation-induced rate of recessive leukemogenic deletions, $a''$ is the spontaneous and $b''$ the induced rate of mitotic recombination, and $s$ is the number of hematopoietic stem cells per human body (see 13 for details). As previously reasoned$^{13}$, the parameters used in Eq. (3) can be approximated by the numerical values given for Eq. (2), as follows: $s = 5 \times 10^6$ hematopoietic stem cells/person (from 10); $a' = 2 \times 24 \times 10^{-6}$; $a'' = 15 \times 10^{-6}$; $b' = 2 \times 3.3 \times 10^{-7}$ and $b'' = 1.4 \times 10^{-7}$, where the meaning of the multiplication factor 2 was given previously$^{13}$. We can thus calculate that:

$$F = 9.3 \times 10^{-3} + 1.62 \times 10^{-4} D \text{ (preleukemic cells/person).}$$

(4)

If we assume that the production of one preleukemic cell in the body eventually leads to leukemia, then Eq. (4) can be rewritten in terms of an annual rate $f$ of the incidence of leukemia, by dividing by 29 years, for comparison with the results of epidemiological studies of leukemia in A-bomb survivors from 1950 to 1978 carried out by Ichimaru et al.$^{20}$ as follows:

$$f = 3.2 + 0.056 D \text{ (leukemia/10^4 person.years).}$$

(5)

Figure 5 shows a comparison of the theoretical curve based on Eq. (5) with the observed dose-response curves for annual rates of incidence of chronic myelogenous leukemia and acute leukemia. Since T65D doses were used in the studies of leukemia, use of DS86 doses would change the shapes of the dose-response curves shown in Figure 5 to some extent, but not greatly. Hence, we may conclude that the slope of the theoretical curve shown in the upper panel of Figure 4 is fairly close to that of the observed dose-response curve for the incidence of acute leukemia in Hiroshima. This agreement is compatible with the assumption that A-bomb radiation acted, at least partly, as an initiator of acute leukemia by inducing recessive, leukemogenic deletions and somatic crossing-over in hematopoietic stem cells.

Tumor progression induced by high doses of radiation

As shown in the lower panel of Figure 5, the dose-response curves for chronic myelogenous leukemia in both Hiroshima and Nagasaki deviate widely from the theoretical curve. Furthermore, the observed curve for chronic leukemia is approximately linear for Hiroshima A-bomb survivors but indicates a large threshold effect for Nagasaki A-bomb survivors. The existence of a large threshold effect means that radiation from the Nagasaki A-bomb, i.e., mostly gamma rays, acted as a promoter and/or progressor in chronic leukemogenesis.

As shown in the lower panel of Figure 5, the Hiroshima A-bomb radiation was a much more effective inducer of chronic leukemia than the Nagasaki A-bomb radiation. Since fission neutrons occurred at a high level only in the Hiroshima A-bomb radiation, the previous proposal$^{31}$ that fission neutrons were responsible for the higher incidences of chronic leukemia in Hiroshima than in Nagasaki remains valid. Now, this proposal is elaborated to suggest that fission neutrons were more effective as promoter and/or progressor of chronic leukemia than gamma rays.
A-BOMB RADIATION AT LOW DOSES

Fig. 5. Crude annual incidence rates for leukemia among A-bomb survivors in the Radiation-Effect-Research-Foundation sample by total marrow dose, type of leukemia, and city, 1950-78. Closed and open circles are values for Hiroshima and Nagasaki, respectively (reproduced with minor modification from Ichimaru et al. [1986]). The theoretical curve A is obtained from Eq. (5) in the text.

Why should fission neutrons be more effective in tumor promotion and progression than gamma rays? One answer may lie in the well-known fact that fission neutrons have a greater relative biological effectiveness in inducing chromosomal aberrations and in cell killing\(^{32}\). In an A-bomb survivor who was exposed to an intermediate dose of fission neutrons or to a high dose of gamma rays, many cells would have been destroyed and various parts of his bone marrow would have been depopulated, resulting in degradation of the ordered cell-cell structure of the tissues that supply new blood cells (see Fig. 6).

As pointed out previously\(^{13}\) in relation to leukemia induced by X rays in mice\(^{33}\), granulocytic progenitor cells in the bone marrow in humans were depressed for more than two decades after exposure to 2–6 Gy of radiation from hydrogen-bomb fallout\(^{34}\). Such persistent disturbance in the cell society of blood-forming tissues would partly free its component cells from territorial restraints on their proliferation (Fig. 6), enabling them to undergo continued evolution of their cellular functions towards more advanced autonomy from the influence of the host\(^{13,27,28}\). Progression of preleukemic cells to advanced autonomy may be achieved by continued epigenetic changes, in combination with continued DNA rearrangements, to lead to gross karyotypic and phenotypic changes.
**Dose-dependent shortening of the latent period as evidence of radiation-induced progression of leukemia**

Leukemia appeared two to three years after exposure to A-bomb radiation in Hiroshima, reaching a peak in 1950–51 and then slowly decreasing, but remaining above the control level even in 1979–82\(^{30,35}\). In Nagasaki, the incidence reached a peak in 1959–62 and decreased thereafter rather rapidly, reaching the control level in 1971–74\(^{30,35}\).

The mean interval from A-bomb irradiation to appearance of either acute or chronic leukemia was shorter in the group exposed at over 100 cGy than in those exposed at 1–99 cGy or in the control group\(^{30}\). This dose-dependent latent period of leukemogenesis implies that an increase in radiation dose shortened the time required for progression of preleukemic cells to advanced leukemic cells in A-bomb survivors, which may be explained as follows: higher doses of radiation induce more chaotic disturbance in the cell society of the bone marrow, resulting in circumstances more favorable for acquisition by the component cells of the neoplastic characters by rapid changes in the phenotype and genotype. If this is the case, the data on leukemia given above indicate that the radiation from the Hiroshima A-bomb, and especially its fast-neutron component, induced a longer disturbance of the cell society than that produced by gamma rays, the major component of the Nagasaki A-bomb radiation.

**EFFECTS OF LOW DOSES OF RADIATION ON HUMAN SURVIVAL**

*Rational estimation of the risk for death from cancer induced by A-bomb radiation at 1 cGy*

Tumor incidences in A-bomb survivors (1950–85) were classified using the DS86 system into doses from 1–4 cGy to over 400 cGy by Shimizu *et al.*,\(^{12,14}\). Table 2 summarizes an essential part of that report; the data seem to indicate that persons exposed to 1–9 cGy had lower death
rates than unexposed persons from either leukemia or all other cancers, indicating the possibility of no harmful effect at 1 cGy.

As discussed in previous sections, a high dose of radiation above a threshold value can act as a promoter and progressor, whereas low doses of radiation do not. Therefore, it is not warranted to assume that the risk of death from cancer at the high dose of 1 Gy can be divided by 100 to obtain the risk of death from cancer at 1 cGy. The risk after exposure to 1 cGy can only be assessed by direct scrutiny of the data on A-bomb survivors exposed to low doses, say, 1–9 cGy, as mentioned above. This method may provide a quantitatively important basis for rational risk assessment, in spite of the large statistical uncertainty in epidemiological data.

Apparent beneficial effects of low doses of A-bomb radiation on human survival

Since 1970, data on $10^5$ survivors of the Nagasaki A-bomb have been maintained at the Scientific Data Center for the Atomic Bomb Disaster at Nagasaki University School of Medicine. We selected information on 3456 persons exposed to known T65D doses. Mortality (1970–88) in this small but carefully selected group (observed) was compared with that of an age-matched control group (expected) who were living far from the center of the A-bomb radiation in Nagasaki. The observed: expected ratios show that the mortality of exposed persons was slightly lower than or equal to that of unexposed persons at all four low doses, 1–49, 50–99, 100–149 and 150–199 cGy, and that radiation-induced mortality was apparent only in the group exposed to 200–599 cGy.

The apparent absence of harmful effects of low doses of radiation was analyzed by determining the observed to expected ratios of persons classified according to cause of death, sex and dose. As shown in Figure 7, in males, doses of 50–99 cGy caused significant reduction in deaths from all causes except cancer to 64% of the control value; however, deaths from cancer increased at all levels of doses except at 1–49 cGy. Thus, low doses of radiation had two opposing effects – beneficial and harmful – on human survival in Nagasaki after the A-bomb.

---

Table 2. Number of subjects and cancer deaths in 1950-85 among A-bomb survivors classified by DS86 dose

<table>
<thead>
<tr>
<th>Dose (cGy) (shielded kema)</th>
<th>Number of subjects</th>
<th>Leukemia deaths</th>
<th>All other cancer deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. Frequency (%)</td>
<td>No. Frequency (%)</td>
</tr>
<tr>
<td>0</td>
<td>34,272</td>
<td>58 0.17</td>
<td>2443 7.13</td>
</tr>
<tr>
<td>1–9</td>
<td>23,321</td>
<td>38 0.16</td>
<td>1655 7.10</td>
</tr>
<tr>
<td>100–199</td>
<td>1,946</td>
<td>23 1.2</td>
<td>221 11.4</td>
</tr>
</tbody>
</table>

a) from Shimizu et al. (1989).
Fig. 7. Relative risk for deaths from all causes, all causes except cancer and cancer alone in male survivors in Nagasaki plotted against T65D doses. The values of relative risk are ratios of the numbers of deaths in exposed groups at indicated doses to those in number- and age-matched unexposed groups (reproduced with minor modification from Mine et al. (1989). * P<0.05.

ACKNOWLEDGEMENTS

I am grateful to Elisabeth Heseltine and Y. Shimizu for comments and suggestions during the preparation of the manuscript, to Drs. M. Akiyama, S. Fujita, A. Awa, M.S. Sasaki and S. Abrahamson for providing materials and to S. Hisanaga and T. Ito for preparing the manuscript. This work was supported by funds from Shigaku Shinko Fund, Kinki University and the Central Research Institute of Electrical Power Industry.

REFERENCES


