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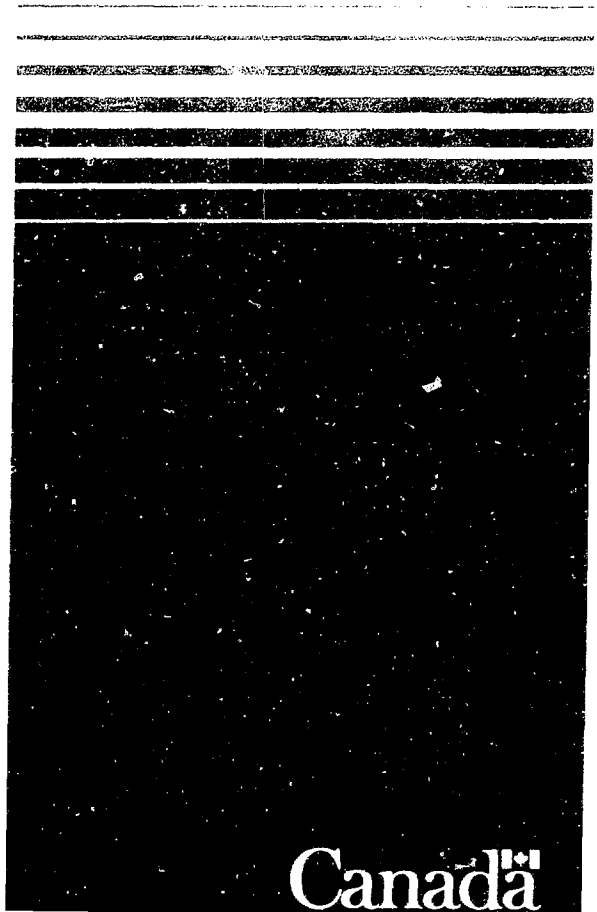
REVIEW OF THE RADIOLOGICAL SIGNIFICANCE OF REVISED DOSE ESTIMATES FOR THE HIROSHIMA-NAGASAKI BOMB SURVIVORS

by

D.K. Myers and R.V. Osborne
AECL-CRNL



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A research report prepared for the
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March 1988

Canada

Research report

REVIEW OF THE RADIOLOGICAL SIGNIFICANCE OF REVISED DOSE ESTIMATES FOR THE HIROSHIMA-NAGASAKI BOMB SURVIVORS

Prepared by D.K. Myers and R.V. Osborne, Atomic Energy of Canada Limited Research Company, Chalk River Nuclear Laboratories

ABSTRACT

Recently, the International Commission on Radiological Protection (ICRP) has indicated that new dosimetric and epidemiological data on Japanese bomb survivors will "raise the risk estimate [for fatal cancers] for the exposed population by a total factor of the order of 2. This change is for a population of all ages, whereas for a worker population of ages 18-65 the changes will be smaller". The present report has reviewed the available scientific literature that is relevant to this statement.

The topics reviewed in this report include: (a) the methods used in previous reports by scientific committees to calculate estimated lifetime risks of radiation-induced fatal cancers; (b) recent revisions of the dosimetry for Hiroshima-Nagasaki survivors; (c) updates on the epidemiological data on the Hiroshima-Nagasaki survivors, and (d) revised estimates of fatal cancer risk from the Hiroshima-Nagasaki data.

The revised dosimetry has produced little change in cancer risk estimates based on absorbed organ doses in grays for the Hiroshima-Nagasaki bomb survivors. Because the neutron component of the revised organ doses is small, the relative biological effectiveness of the neutrons cannot be estimated directly from the epidemiological data. If the quality factor of the absorbed neutron doses were assumed to be 20, as recommended by the ICRP in 1985, the calculated risk coefficients for leukemia would be increased by about 95% and for all other fatal cancers by about 38%. Depending upon the type of theoretical models used to extrapolate from the observed epidemiological data to estimated lifetime risks, the predicted lifetime risks of excess fatal cancers in the exposed population of Hiroshima-Nagasaki survivors would probably be 1.1 to 14 times greater than the previously accepted general estimates given by the ICRP Publication 26 in 1977. In our opinion, an increase of twofold is probable. The information derived from the survivors of the atomic bomb blasts in Hiroshima and Nagasaki is only one of the sources used by scientific committees in arriving at general estimates of radiation-induced fatal cancers. The impact of revised Hiroshima-Nagasaki data on overall estimates of excess fatal cancers induced by ionizing radiation is being considered by various national and international scientific committees; a final scientific consensus from the ICRP is anticipated within 2 or 3 years.

Revisions of the Hiroshima-Nagasaki dosimetry are not expected to have an appreciable effect on quantitative risk estimates for radiation-induced curable cancers or heritable genetic effects. Most of the information on which these latter estimates are based derives from other sources. The impact of these dosimetric revisions on estimates of the risk of severe mental retardation following in utero irradiation is still uncertain.

RÉSUMÉ

La Commission internationale de protection radiologique (CIPR) a indiqué récemment que les nouvelles données dosimétriques et épidémiologiques sur les survivants japonais de la bombe atomique feront approximativement doubler l'estimation de risque de cancer mortel chez la population irradiée. Ce changement s'applique à une population de tous les âges, tandis que, pour la population active de 18 à 65 ans, les modifications seront moindres. Le présent rapport a examiné toute la documentation scientifique pertinente disponible.

Le rapport examine les domaines suivants : a) les méthodes utilisées dans les rapports précédents de comités scientifiques pour évaluer le risque pendant toute une vie de mourir de cancer dû aux rayonnements; b) les révisions récentes des données dosimétriques des survivants d'Hiroshima et de Nagasaki; c) les mises à jour des données épidémiologiques sur les survivants d'Hiroshima et de Nagasaki, et d) les estimations révisées de risque de cancer mortel à partir des données d'Hiroshima et de Nagasaki.

Les données dosimétriques corrigées ont amené très peu de changement par rapport aux estimations de risque de cancer à partir des doses (exprimées en grays) absorbées au niveau des organes par les survivants des bombes d'Hiroshima et de Nagasaki. Parce que l'élément neutronique des doses révisées au niveau d'un organe n'est pas important, l'efficacité biologique relative des neutrons ne peut être évaluée directement à partir des données épidémiologiques. Si le facteur de qualité des doses de neutron absorbées se situait à 20, comme l'a recommandé la CIPR en 1985, les coefficients de risque augmenteraient de 95 pour 100 pour la leucémie et de 38 pour 100 pour les autres formes de cancer mortel. Selon le type de modèle théorique utilisé pour extrapoler à partir des données épidémiologiques observées jusqu'à l'estimation de risque pour toute une vie, le risque accru de cancer prévu pour la population irradiée parmi les survivants d'Hiroshima et de Nagasaki, serait probablement de 1,1 à 14 fois supérieur aux estimations précédentes généralement reconnues qui figurent dans la publication n° 26 de la CIPR, publiée en 1977. À notre avis, une augmentation du double est probable. Les données recueillies à partir des survivants des deux explosions atomiques d'Hiroshima et de Nagasaki ne sont qu'une des sources utilisées par les comités scientifiques pour en arriver aux estimations générales de cas de cancer mortel causés par les rayonnements. Divers comités scientifiques nationaux et internationaux examinent actuellement les répercussions des données corrigées d'Hiroshima et de Nagasaki sur les estimations générales d'incidence accrue de cas de cancer mortel dû aux rayonnements ionisants; un consensus scientifique définitif de la CIPR est prévu d'ici deux à trois ans.

On ne prévoit pas que les révisions des données dosimétriques d'Hiroshima et de Nagasaki aient des conséquences significatives sur les estimations quantitatives de risque de cancer curable dû aux rayonnements ou d'effets génétiques héréditaires. La plupart des renseignements sur lesquels ces estimations sont basées proviennent de d'autres sources. Les répercussions des révisions des données dosimétriques sur les estimations de risque d'aliénation mentale grave à la suite de l'irradiation de l'embryon demeurent incertaines.

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REVIEW OF THE RADIOLOGICAL SIGNIFICANCE OF REVISED DOSE ESTIMATES FOR THE HIROSHIMA-NAGASAKI BOMB SURVIVORS

D.K. Myers and R.V. Osborne

A. INTRODUCTION.

Estimates of the risk of radiation-induced stochastic effects published by the International Commission on Radiological Protection (ICRP) between 1977 and 1986 are summarized in Table 1. These quantitative estimates have an important influence on recommended limits for exposures of radiation workers and of the general population to ionizing radiation.

A recent statement from the September 1987 meeting of the ICRP in Como [1] dealt with assessment of the risk of cancer induction after exposure of humans to low doses of ionizing radiation. This statement referred specifically to the 1987 paper by Preston and Pierce [2] on effects of reassessment of doses received by survivors of the 1945 atomic bomb blasts at Hiroshima and Nagasaki, and to mortality data obtained in the follow-up of these survivors to 1985 [2-5]. The ICRP statement indicated that these recent data

> "raise the risk estimate for the exposed population by a total factor of the order of 2. This change is for a population of all ages, whereas for a worker population of ages 18-65 the changes will be smaller" [1].

The September 1987 ICRP statement further indicated that "the risk data are yet far from conclusive", and that the Commission would await the results of ongoing comprehensive evaluation of epidemiological data by UNSCEAR, the BEIR V committee and ICRP Committee I before judging the consequences for revision of the ICRP system of dose limitation. Two other factors that might result in larger changes in the ICRP's present risk estimates were noted, namely, adoption of a "relative risk" model for the estimation of total lifetime cancer risks from observations over a shorter period of time, and the shape of the dose response relationship. [1]

The new epidemiological data referred to in this recent ICRP statement are cancer mortality data, i.e. they are most relevant to the first line shown in Table 1. The present report will therefore concentrate on recent data (to February 1988) that are considered relevant to assessment of the risk of radiation-induced fatal cancers in humans. Brief comments on the impact of revisions of estimated doses received by the Hiroshima-Nagasaki survivors will be noted for the other stochastic or potentially-stochastic risks listed in Table 1.

B. PREVIOUS ESTIMATES OF LIFETIME RISK OF RADIATION-INDUCED FATAL CANCERS.

Table 2 summarizes the estimates of lifetime risk of radiation-induced fatal cancers that were derived by the UNSCEAR, ICRP and BEIR-III committees in 1977-80. Review of the methods used to derive these risk estimates is critical to an understanding of current scientific discussions concerning revision of these estimates.

Table 1. Previous ICRP estimates of probability of radiation-induced stochastic or potentially-stochastic effects [6-8].

Biological effect	Risk per 100 person-Sv* to exposed persons	Risk per 100 Sv* averaged over a whole population with an average life expectancy of 75 years
Fatal cancers	1.3	1.3
Curable cancers	1.3 - 2.3	1.3 - 2.3
Genetic disorders in children and grandchildren	1.0	0.4
Severe mental retardation after irradiation in utero	10.0	c. 0.13

[* Although the data are expressed per 100 person-Sv for ease of comparison, it should be noted that use of the term Sv is restricted to dose equivalents accumulated at dose rates below 50 mSv per year to the whole body or below 500 mSv per year to any given tissue in the body [9].)

Table 2. Estimates of probability of radiation-induced fatal cancers in previous reports of scientific committees

Report	Risk per 100 person-Sv*
UNSCEAR 1977 [10]	about 1
ICRP 1977 [6]	1.3
BEIR III 1980 (low LET radiations only) [1]	0.7 - 2.3 (range from less than 0.1 to 5.0)

[* See footnote to Table 1.]

Annex G in the 1977 UNSCEAR report [10] reviewed the epidemiological and dosimetric data that were available at that time for various groups of persons who had in the past been exposed to high radiation doses. These groups included the Hiroshima-Nagasaki bomb survivors, persons exposed to medical X-rays for the treatment of various diseases, radium dial painters, thorotrast patients, uranium miners and others. Two major conclusions were derived: (a) At moderately high doses of low-LET radiation above 1 Gy, the risk of radiation-induced fatal cancers appeared to be about 0.5×10^{-2} per Gy for leukemia plus 2×10^{-2} for other fatal cancers, for a total risk of $2.5 - 3 \times 10^{-2}$ per Gy of low-LET radiation (para. 317 in Annex G of UNSCEAR 1977). (b) At lower doses of low-LET radiation, the lifetime risk of radiation-induced leukemia was estimated to be about $0.15 - 0.25 \times 10^{-2}$ per Gy of low-LET radiation. Evidence was cited to indicate that increased incidence of leukemia had ceased by 30 years after exposure; thus the observed risk was essentially identical to the lifetime risk of radiation-induced leukemia. It was however noted that the observed excess of other types of radiation-induced fatal cancer in the Hiroshima-Nagasaki survivors did not represent a total lifetime risk. Assuming that the lifetime risk of other fatal cancers might be 4-6 times that of leukemia ont he basis of earlier mortality studies of American radiologists and of patients exposed to high doses of X-rays for medical purposes (para. 247 in Annex G of UNSCEAR 1977), the total risk for all fatal induced malignancies was estimated at <u>about</u> 1×10^{-2} per Sv of low-LET radiation at lower doses of radiation (Table 2).

The 1977 ICRP report [6] did not give a detailed review of the epidemiological data; risk estimates were apparently based on ICRP review of the data provided in UNSCEAR 1977 [10]. The ICRP report suggested a leukemia risk of 0.2×10^{-2} per Sv and a total risk for all fatal induced malignancies of 1×10^{-2} per Sv in males or 1.5×10^{-2} per Sv in females, the difference being due to the risk of radiation-induced fatal breast cancer in females. This report further indicated that an approximate risk estimate of 10^{-2} fatal cancers per Sv could be used both for radiation workers and for the general public. The initial 1977 document [6], like the UNSCEAR report [10], was somewhat ambiguous as to whether or not risks at very low dose levels, such as might be encountered in environmental exposures to radiation, might be substantially less. This was however clarified in a 1978 ICRP statement [9] to the effect that these estimates were intended to be realistic assessments of risks of exposure to radiation levels below 50 mSv per year to the whole body. Thus the ICRP consolidated its previous suggestions that risks of fatal cancer should be considered to be directly proportional to accumulated radiation dose.

NCRP Report No. 64 [12] provided further theoretical support for the ICRP interpretation of the epidemiological data. The NCRP review of experimental data from other living organisms emphasized the value of the linear-quadratic model for interpretation of dose-response curves on biological effects of exposure to low-LET radiation. (This model, it might be noted, was used by Lea as early as 1946 to explain the dose-response curves for induction of chromosomal aberrations by X-rays.) At low doses at any dose rate, or at higher doses at low dose rate, effects appear to be directly proportional to accumulated dose of X- or gamma-rays. The effects per unit dose at higher doses delivered at high dose rate are in general 2-10 times greater than effects per unit dose at low dose rate in the case of low-LET radiation [12].

A similar decrease in effects per unit dose is not observed at lower dose rates in the case of high-LET radiation; comprehensive theories explaining these differences have been developed. It should be noted that NCRP Report No. 64 did, however, express reservations about the value of the linear-quadratic model for interpretation of epidemiological data on radiation-induced breast cancer in women. NCRP Report No. 66 [13] also suggested that a simple linear dose-response model provided a good fit to the latter data.

The 1980 BEIR III report [11] provided a detailed review of the same type of epidemiological data that were considered in the 1977 UNSCEAR report [10], but placed more emphasis on uncertainties in the theoretical models which could be used for interpretation of these data. This BEIR report was also notable, and valuable, for its inclusion of dissenting reports and comments by various members of the committee. (The BEIR III report is also notable for inclusion in Table V-4 of a misleading quotation of the cancer risk estimates that were given in BEIR I [14]. A similarly misleading citation of cancer risk estimates from BEIR III is given in Table 1-1 of the 1988 BEIR IV report [15]. These unusual peculiarities will not be considered further in our present review.)

The BEIR III report [11] continued the use of both absolute and relative risk [14] models to interpret epidemiological data. The first model reports data in terms of excess number of cancers observed; the second model reports data in terms of percent increase in normal incidence of cancers. These concepts were applied in the 1972 BEIR I report [14]; the evidence available for review in BEIR I suggested that neither model was necessarily better than the other and that the true risks might lie somewhere in between the predictions suggested by these two theoretical models. If complete lifetime follow-up of exposed groups of persons were available, a choice between absolute and relative risk models would be irrelevant. The question relating to different models for prediction of lifetime risks arises because a large proportion of the exposed cohorts are usually still alive at the time of observation; this is also true for persons who were exposed at younger ages at Hiroshima and Nagasaki in 1945. In general, the absolute and relative risk models predict similar lifetime risks of radiation-induced cancer for persons of older ages at the time of exposure; the relative risk model predicts higher values for lifetime risks for persons of younger ages at the time of exposure (Table 3). The particular numbers given in Table 3 are derived from one particular table in BEIR III; other selections of risk estimates are available but the general trend is similar. The majority of the BEIR III committee members were again unable to make a clear choice in favour of the relative or the absolute risk projection model, and consequently preferred to give the lifetime risk estimates that were calculated by both methods.

It should be noted that the difference in projected lifetime cancer risks using these two models is largely due to a difference in estimated number of radiation-induced cancers which might appear in old age, when the natural incidence of cancers is highest, after exposure at a much younger age. Differences in average loss of life expectancy are thus much smaller than the differences in estimated number of induced cancers [7,16].

Table 3. Two estimates of lifetime risk of fatal cancers (other than leukemia) for persons exposed continuously to 10 mSv of low-LET radiation per year at different ages* [11].

Age during exposure (in years)	Lifetime risk of fatal cancers (other than leukemia) per 100 person-Sv*	
	Absolute risk projection model	Relative risk projection model
0 - 19	1.68	2.94
20 - 34	0.68	1.95
35 - 49	0.54	0.74
50 - 65	0.41	0.43

[* Data were derived by the authors from Table V-19 in BEIR III [11], which is based on a linear-quadratic model for low-LET radiation. The values given are averages for males and females. Because the BEIR III report assumed a minimum latent period of 10 years for radiation-induced fatal cancers other than leukemia, radiation exposures after age 65 have little effect on the above numbers. See footnote to Table 1.]

The 1980 BEIR III report [11] also considered quadratic, linear-quadratic and linear dose-response models for induction of fatal cancers by low-LET radiation. (The dose-response relationship for high-LET radiation was assumed to be linear). The quadratic model is based largely on dosimetric considerations of the relative biological effectiveness of neutrons as analyzed by Rossi and coworkers. The linear-quadratic model provides the best interpretation of data on induction of somatic mutations in Tradescantia, of chromosomal aberrations in human lymphocytes, and of any other biological endpoints in experimental laboratory systems [12]. The linear model reflects the most simple and direct interpretation of limited epidemiological data. In themselves, the epidemiological data are not precise enough to permit a clear-cut choice between these three dose-response models [11]. The majority of the BEIR III committee expressed a preference in favour of the linear-quadratic model, but also provided risk estimates for low doses of low-LET radiation as calculated by the other two models (Table 4).

These wide variations in projected lifetime risk of radiation-induced cancer depending on dose-response model (Table 4) apply only to low-LET radiation. In the case of radiogenic breast cancer, the BEIR III committee provided estimates based on a linear dose-response model but noted that the true risks at low doses "could be as low as one-third of the risks corresponding to the linear

Table 4. Comparative estimates of lifetime risk of cancer mortality induced by chronic exposure to 10 mSv of low-LET radiation per year throughout life.

Dose-response model for low-LET radiation	Lifetime risk per 100 person-Sv by projection model*	
	Absolute	Relative
Quadratic	<0.1	<0.3
Linear-quadratic	0.67	1.7
Linear	1.6	4.0

[* Data derived from Table V-4 in the 1980 BEIR-III report [11]. See footnote to Table 1.]

absolute-risk model, and could conceivably be zero at 1 rad [10 mSv] if the models used.... are not applicable at very low doses". The 1980 BEIR III report obviously left unresolved many practical problems in the estimation of lifetime risk of radiation-induced cancer.

C. REVISIONS OF THE DOSIMETRY FOR HIROSHIMA-NAGASAKI SURVIVORS.

The first estimates of doses from the Nagasaki and Hiroshima weapons were those designated T57 (T for tentative, 57 for the year of the study) and were based mainly on experiments carried out during weapons tests [17,18]. On the basis of experimental simulations, Auxier and his colleagues at ORNL developed the T65D dose estimates [19], which with one minor revision in 1968 designated as T65DR, have been used for risk estimates since. The results of the major reassessment of doses (designated DS86) that has been carried out by groups in Japan, the US, and the UK have now been published by the Radiation Effects Research Foundation [20] and these doses are the basis for the dosimetry-related revisions to the risk estimates. Several summaries of the dosimetry revisions have already appeared [3,21]. This section summarises the changes that have occurred in the dose estimates between T65D and DS86.

1. Revisions in the bomb yields

The bomb exploded over Nagasaki was an implosion device using plutonium and was the same type as the bombs tested at Trinity in New Mexico and Bikini. Yields were determined by measuring the fireball expansion and by radiochemical evaluation of the debris in the fireball in these tests. It is thought that these observations provide the most precise estimates of yield for the Nagasaki bomb. Other estimates, based on measured thermoluminescence in roof tiles in

Nagasaki and on shock wave calculations, gave similar but more uncertain estimates. The test estimate of yield is now 21 kt (an increase of only 5% from the previous estimate) with a 10% uncertainty [20].

The Hiroshima bomb was a gun-type of device using enriched uranium and was the only detonation ever made of a device of this type. The yield has therefore been estimated from the measured variation of pressure with time, induced thermoluminescence in roof tiles, charring of cyprus wood on a roof about 670 m from the hypocentre, induced P-32 activity in sulphur used to bond insulators to utility poles, and blast damage. The yield relative to that of the Nagasaki bomb has been estimated from the relative thermal and blast damage at the two sites. The recommended best estimate for the Hiroshima bomb is 15 kt (a increase of 20% from the previous estimate) with an outside limit of uncertainty of 20%.

2. Source terms

Calculating the number and distribution in energy and angle of the neutrons and gamma rays emerging from the bomb case is extremely complex. Such "source terms" depend on the number of fissions that take place and the nature and position of materials in the bomb. The bomb at Nagasaki had near spherical symmetry and simpler design (relative to that at Hiroshima); this has been the simpler of the two calculations and there is more confidence in the calculated emission.

The source term for the Hiroshima bomb with its more complicated shape has been the more difficult to estimate. Tests with a critical assembly and many calculations now give fair confidence in the estimated source term. The main uncertainty in the source-related parameters is in the energy spectrum of the prompt neutrons from Hiroshima. Only 5% have energies greater than 1.35 MeV but they contribute over 90% of the dose at ground ranges of 950 m and more. This is the component in the estimated source term that may still change in the future. Activation of materials in the environment by neutrons has provided some verification of the calculations. The high energy part of the neutron spectrum is responsible for Co-59 activation and the observations of this activation show large discrepancies with calculated activities. Sulphur and europium activation by thermal neutrons show no important discrepancy but the sulphur data only provide good support close to the hypocentre; for europium at all distances and for sulphur beyond 1000 m, the support for the calculations is weak. If the measured cobalt activations were accepted as correct representations of the thermal fluences and the assumption is made that the calculated fluences on the ground are low by a factor that applies to all energies, then the proportion of dose from neutrons in the mixed radiation field beyond 1000 m at Hiroshima would change from insignificant to significant. The conclusion in reference [20] is that it is unlikely that the neutron estimates are seriously wrong. The review by the Advisory Committee on the Radiation Effects Research Foundation [21] notes that a quantitative analysis of the estimates uncertainty in the Hiroshima neutron dose estimates is missing. Whalen, in reference [20], estimates that the standard deviations in the source terms are about 20% plus something for thermal and hydrodynamic effects.

3. Estimates of free-in-air kerma

The object of the reassessment program is to determine the **absorbed dose** (or simply the **dose**) in certain organs of the people exposed to the bombs. For practicality, determination of a quantity often approximately equal to the dose, the **kerma**, is usually made instead. The concept of absorbed dose deals with the energy imparted by ionizing radiation to a medium per unit mass. "Energy imparted" means the difference in energy of the particles and quanta entering and leaving a small test volume. For the particles and quanta encountered in A-bomb dosimetry, the energy difference for photons and neutrons equals the energy they give to the charged particles they produce by interactions in the volume. This remainder of the energy imparted is absorbed from charged particles: electrons, protons, and alpha particles. Under circumstances known as **charged-particle equilibrium**, the total energy of the particles entering a small mass equals that of those leaving. In other words, they suffer no net loss of energy. The absorbed dose, therefore, equals the kerma (neglecting a small energy loss to bremsstrahlung). "No net loss" does not mean that the charged particles do not deposit any energy in the material; it means that the complex interplay of photons, neutrons, and charged partticles, the photons and neutrons make up the energy losses of the particles [20].

In the RERF dosimetry report [20] the initial quantity calculated is the kerma that would be produced in tissue by the radiations at a point in air. The condition is used so often that its kerma is given a special name: The kerma-in-tissue at a point in air over bare ground (i.e. no person present and not in or near a building) is called the **free-in-air (FIA) kerma or the free-field kerma.**

The FIA kerma have been calculated for both weapons from estimates of the source terms and the transport of the radiations to ground level. The residual radiations originating from local fallout of fission products and neutron activation of ground and other materials beneath the explosions have also been estimated.

The calculation of FIA kerma involves modelling the propagation through the air of radiations emitted by the bomb, and of gamma rays produced by neutron capture in the air. These are relatively easy calculations. More difficult to calculate are the emission and propagation of the fission product gamma-rays and the delayed neutrons, because of the complex behaviour of the rapidly rising fireball.

The Nagasaki neutron spectrum peaks at few tenths of a kilovolt because of neutrons being thermalised by interacting with the hot high explosive debris around the bomb. The Hiroshima spectrum did not show this peak. However, most of the neutron dose, even at Nagasaki, was due to high-energy neutrons of about 1 MeV and higher, because the low energy neutrons are absorbed by the intervening atmosphere.

Figure 1 shows the contributions of the various gamma ray sources to the DS86 FIA gamma kerma values. In both cities the prompt primary gamma ray emissions make a small contribution to the doses. Most of the gamma kerma is from the delayed primary gamma rays (from radioactive decay of fission products in the fireball) and the prompt secondary gamma rays (prompt neutron interactions in the air and the ground). These components are also the main contributors to the total FIA kerma as shown in Figure 2. The third largest contributors differed between the two cities: prompt primary gamma rays in Nagasaki, prompt neutrons at distances less than 1500 m at Hiroshima.

The estimate standard deviations for FIA total gamma ray and neutron kermas between 1000 and 2000 m are estimated [20] to be in the range 10 - 20%, excluding uncertainties in energy yields and source terms. The DS86 estimations for FIA gamma and neutron kerma are compared with the T65D estimates in Figures [3].

In Nagasaki the estimate of neutron kerma decreased to about half or one third of the T65D estimate. The reduction in the neutron kerma at Nagasaki is due to two changes: the energies of the escaping neutrons are lower, and the effect of water vapour in the air has been included. This has reduced the transmission of neutrons because of the increase in the cross section of hydrogen with decreasing neutron energy. The T65D estimates were based on scaling from measurements in Nevada in a very dry atmosphere.

Humidity has only small effect on the gamma ray kerma and the estimated gamma kerma at Nagasaki has not changed very much: 10 - 30% or less, depending on the ground range.

The most significant differences in the Hiroshima data are the larger gamma ray kerma by a factor that ranges from 2 to 3.5 (depending on the ground range), and a neutron kerma about 10% that in T65D. A small part is due to the change in yield (12.5 to 15 kt). The rest of the change (factors from 1.7 to 2.9) in the gamma ray kermas is due to the changes in the methods of determining kermas. In DS86, the calculations are from primary physical data. In T65D, the methods were based on experimental data from bomb and other tests. Since no bomb of the Hiroshima type was ever tested, the data for calculation of kerma had to be modified from that for the Nagasaki type of bomb. Unfortunately, something did not work out and it is not known just what went wrong in the T65D calculations.

The reasons for the reduction in neutron kerma in Hiroshima are the same as for Nagasaki. The reduction at Hiroshima is greater because the reduction in the energies of the neutrons penetrating the steel bomb casing was greater than at Nagasaki which l\had mainly hydrogenous materials.

For Hiroshima, the lower DS86 values are also due to the inclusion of the effects of atmospheric moisture and to the small proportion of high-energy neutrons in the source-spectrum data.

4. Residual radioactivity in soil and other materials

The doses from induced radioactivity and fallout are believed to be of little significance in the dose reassessment; the maximum doses at Nagasaki and Hiroshima are estimated to have been about 0.5 Gy. These are appreciable doses, but care is taken to exclude persons exposed in this way from the control population.

The survivors who entered the area within 1000 m from the hypocentre a few hours or days after the explosions could have received additional radiation from fallout and by irradiation from radioactivity induced in the ground. There may have been survivors who received significant doses from these sources.

For fallout, the upper limits for absorbed dose from gamma rays for persons continuously in the fallout area at Nagasaki ranged from about 0.12 to 0.24 Gy. For Hiroshima the absorbed doses ranges from 0.006 to 0.02 Gy. The region of fallout was very small at Hiroshima so the contribution to survivor dose was probably negligible. At Nagasaki, exposure up to 20% of the maximum extends over 1000 hectares, so the exposure from these sources may have been significant for a few survivors.

The activity in soil and other materials induced by neutron fluence falls off very rapidly from the hypocentre. Exposures near the hypocentre were determined from known soil analyses and from activities measured at later times; the upper limits were 0.5 Gy at Hiroshima and 0.18 - 0.24 Gy at Nagasaki. The RERF report [20] acknowledges that individuals from areas of high residual activity should not be included in any nonexposed cohort for the epidemiological studies.

5. Shielded kerma in air

In the DS86 calculations, the modifications of the neutron and gamma spectra are tracked through building materials and other shielding; scattering at each of the locations where there were survivors is also accounted for. These calculations result in the shielded kerma in air.

The main change from the T65D dosimetry has been the reduced transmission of gamma rays through building materials and local terrain. Table 5 shows the average transmission factors for Hiroshima. The values for Nagasaki are nearly identical. The greater attenuation of the delayed gammas is because they have a lower energy.

Table 6 compares the DS86 values for shielded kerma with the T65D values. The average reduction in total kerma in Gy is 28%.

The errors are estimated to arise mainly from the uncertainties in the survivors' locations. The standard deviations of the estimates are 20 - 30% [20].

Table 5. Average kerma transmission factors relative to free-field kerma, Hiroshima at 1500 m.

Source	Neutrons	Gamma rays		
		Prompt		Delayed
T65D	0.32	--	0.90	--
In house [20]	0.40	0.54		0.47
In open [20]	0.67	0.67		0.65

Table 6. Mean shielded kerma and organ-absorbed doses among survivors exposed to 0.01 Gy and over [3].

City	Organ	Dosimetry system	Number of persons	Gamma dose (mGy)	Neutron dose (mGy)
SHIELDED KERMA					
Both	--	DS86	41719	287	8
		T65DR	41316	350	64
Hiroshima	--	DS86	31044	295	10
		T65DR	26146	344	99
Nagasaki	--	DS86	10675	265	3
		T65DR	15170	362	4
ORGAN DOSE					
Both	Bone marrow	DS86	40701	239	3
		T65DR	41316	201	16
Both	Stomach	DS86	39961	226	2
		T65DR	41316	169	12
Both	Female breast	DS86	25252	236	4
		T65DR	25211	276	33

6. Organ doses

The final step was to calculate the organ doses for each of the survivors. The conditions for charged-particle equilibrium are met approximately in most of the organs of interest in A-bomb dosimetry (i.e., the kerma gives a sufficiently accurate approximation to the dose). One organ where the conditions are not met, in general, is the skin. To calculate dose to the skin would require a special and expensive computer calculation, not justified by the limited use it would have received in the RERF studies. The RERF report [20] therefore does not deal with dose to the skin. Neither are these conditions met in and around bone; because of the importance of radiation-induced leukemia, the lack of charged particle equilibrium received special attention in the RERF [20] study.

The estimates of organ doses reflect the location, position and posture of the exposed individuals whereas the calculations for T65D used constant transmission factors [22,23].

Table 6 shows the organ doses for various tissues. The tissue transmission factors for gamma rays at Hiroshima and Nagasaki are very similar for all organs because the gamma ray spectra at the two cities are very similar. However, the prompt neutron spectra at the two cities are very different, with those at Nagasaki having more high-energy neutrons at most distances from the hypocentre. As a result the transmission factor for neutrons at Nagasaki are larger than at Hiroshima at most ranges, the difference becoming less as the distance increases [20].

The transmission factors that appeared in the BEIR report [11] were calculated from a simple cylindrical phantom and quite a different incident spectrum from that used in DS86. BEIR also assumed isotropic incidence whereas DS86 estimates were made with appropriate angular incidences. The average increase in the transmission factor is about 1.5 times greater than the one used in the BEIR report.

The uncertainties in the estimates arise mainly from the uncertainties in the orientation of the individuals. The standard deviation is suggested to be about 10 - 20% [20].

Table 7 summarises the estimates of errors suggested in reference [20].

7. Medical X-ray exposure doses

Doses received between 1948 and 1982 as a result of medical X-rays have also been assessed [24]. In general, doses to the bone marrow are about 10 mGy and are relatively small compared to those received from the atomic bomb explosions in 1945. These recorded medical doses have not been added to the estimated doses for the Hiroshima-Nagasaki survivors.

Table 7. Estimates of errors in atomic bomb dosimetry.

Component in calculation	Estimated one-sigma standard deviation	Page in reference [20]
Bomb yield	5% Nagasaki 10% Hiroshima	p 15
Source term	20%	p 64
KIA	10 - 20%	p 137
KIA, shielded	20 - 30%	p 285
Organ doses	10 - 20%	p 24

D. UPDATE OF EPIDEMIOLOGICAL DATA ON HIROSHIMA-NAGASAKI SURVIVORS.

An important revision of epidemiological data on the Hiroshima-Nagasaki survivors probably resulted from the detectable excess of breast cancers which appeared by 1980 in Japanese women who had been exposed to radiation from atomic bombs in 1945 when they were less than 10 years of age [26]. This excess, which is clearly related to radiation dose (Table 8), was not seen in earlier surveys and appeared only after the women in question had reached ages at which breast cancer rates normally increase to appreciable levels [26]. A higher relative risk was observed than in any older cohort with similar exposure. This finding has provided a major impetus to adoption of the relative risk model for projection of lifetime cancer risk estimates.

The excess cancers produced in other tissues can also be fitted by a relative risk model, but the epidemiological data are not precise enough to allow a clear-cut choice of projection model. Thus "it remains uncertain for which other cancers such a relative risk model applies, and whether it applies with a constant relative factor of increase for the full remainder of life after exposure" [1]. Some relevant information may be provided by recent reports on excess lung cancer in miners who were in the past exposed to high concentrations of radon progeny. In this case, interactions with cigarette smoking and with age should provide a clear test of the relative risk model. Although a modified relative risk model has been adopted in both the 1988 BEIR IV report and ICRP Publication 50 [27], there is no clear-cut and reproducible evidence to favour either the relative or the absolute risk projection models [28,29]. The data suggest rather that the true risks probably lie somewhere in between those calculated on the basis of relative and absolute risk projection models [11]. Lung cancer risks from inhalation of radon progeny do not remain high throughout life but tend to decrease gradually over a period of years

Table 8. Breast cancer incidence up to 1980 in Japanese women who were less than 10 years old at the time of exposure to radiation from atomic bomb explosions in 1945 [26].

	Cases associated with various average doses, expressed in terms of T65D kerma in Gy				
	0	0.026	0.17	0.55	1.88
Observed	6	5	5	5	3
Expected	13.2	5.45	3.56	0.85	0.93
Ratio obs'd/exp'd	0.45	0.92	1.4	5.9	3.2

[* Note that these data refer to total incidence of diagnosed breast cancers. Deaths from breast cancer among Hiroshima-Nagasaki survivors of all ages [4] represent about one-third of the total number of 564 diagnosed breast cancers reported by Tokunaga et al [26]).

after the last exposure [15,28,29]. No evidence for a similar decrease in risk of radiation-induced fatal solid cancers has appeared in the Hiroshima-Nagasaki cohort [4].

Table 9 illustrates some of the problems involved in deriving lifetime risk estimates from the epidemiological follow-up of the Hiroshima-Nagasaki cohort. In the first place, about two-thirds of this cohort was, fortunately, still living as of 1982 [4]; thus the theoretical models used to extrapolate from observed excess deaths to projected lifetime risks are crucial, particularly for estimates of cancer risk in persons exposed at younger ages. Second, excess cancer deaths represent a small fraction of total deaths or of the total cancer deaths observed in this cohort; thus careful attention to diagnoses of the cause of death is also critical. A third complication is presented by the fact that the overall death rate for the Hiroshima-Nagasaki cohort appears to be smaller than that for the whole population of Japan [10]. This reduction in death rate from all causes, despite the increase in deaths due to cancer, is attributed to a lower rate of death from cardiovascular diseases and can be directly linked to the frequency of voluntary visits to the free medical clinics provided for the registered survivors of the 1945 atomic bomb explosions [30]. Calculations of excess cancer risks are therefore currently based on internal comparisons of groups within the Hiroshima-Nagasaki cohort who were exposed to different radiation doses in 1945.

Table 9. Number of persons and observed deaths in the epidemiological follow-up of Hiroshima-Nagasaki survivors 1950-1982 [4].

	Total number	Number exposed to more than than 0.1 Gy *	% increase per Gy *
Total persons	117,748	25,203	--
Total deaths	39,141	8,682	--
Deaths from violent causes	2,180	--	--
Deaths from non-malignant diseases	28,637	--	--
Deaths from cancer	8,324	2,106	23
- leukemia	243	122	295
- breast cancer	193	64	69
- lung cancer	847	227	33
- stomach cancer	2,832	666	11

[* Doses in Gy are T65D estimates, not the revised DS86 doses. Data for 2,384 persons with unknown doses are not included in the above table. Note that the total number of persons included in the above follow-up is somewhat larger than the groups on which analyses of 1980-85 data were based by Shimizu et al [3]. Part of the reason for these differences is that DS86 doses have only been calculated as yet for about 83% of the life span study group].

Our own calculations, based on the numbers given in Table 9, suggest about 70 excess leukemia deaths and 255 excess deaths from other types of cancer between 1950 and 1982 in the group exposed to more than 0.1 Gy (T65D estimates) in 1945. Allowing for a small number of unrecorded excess leukemia deaths between 1946 and 1950 [2], the ratio of excess deaths due to solid cancers divided by excess deaths due to leukemia would appear to be slightly greater than 3 up to the end of 1982. This ratio is increasing from the much lower values observed up to 1972, as expected [10]. Although various theoretical projections can be made, it is not yet known whether this ratio will ultimately exceed the values of 4-6 that were used as a basis for derivation of lifetime cancer risk estimates in the 1977 UNSCEAR report. Data for Hiroshima-Nagasaki survivors to the end of 1985 are being analyzed [2,3] but are not yet available in complete detail.

Trends observed in excess cancer mortality for Hiroshima-Nagasaki survivors by 4-year intervals between 1950 and 1982 are shown in Figure 4. (In this connection, it might be noted that data to 1972 were considered in the 1977 UNSCEAR report, while data to 1974 were available for the 1980 BEIR III report). Two points might be noted from Figure 4. There has been a small number of excess leukemia deaths among the Hiroshima-Nagasaki survivors since 1974. Secondly, the relative risk of all cancers except leukemia was somewhat higher in 1975-1982 than in previous years. The dashed line in Figure 5 suggests that the excess relative risk of all other cancers (except leukemia) is increasing by about 4.8% per year on average between 1959 and 1982. This increase may be due to the fact that many of the older persons in the 1950 Hiroshima-Nagasaki cohort have died by now and recent values are more strongly influenced by death rates among the younger members of this cohort.

The excess relative risk for leukemia and for all other cancers is highest in persons who were of a young age at the time of exposure to high radiation doses in 1945 (Figure 56). This was also evident in the data to the end of 1974 that was considered by the 1980 BEIR III committee. The crucial question is whether or not the high relative risk for persons who were less than ten years old at the time of the bomb will continue throughout the remainder of their normal lifespan. Assuming that this high relative risk for younger persons would in fact continue throughout the remainder of their life, Preston and Pierce [2] projected a total lifetime risk of all radiation-induced fatal cancers except leukemia which is 13 times the lifetime risk of radiation-induced leukemia in a general population of all ages at the time of exposure to radiation. It remains to be seen whether this theoretical projection will be confirmed by further follow-up of the Hiroshima-Nagasaki survivors. Other theoretical projections based on extrapolations of the Japanese data suggest that the lifetime risk of radiation-induced fatal cancers will in fact be closer to 4-6 times the lifetime risk of radiation-induced leukemia (45).

E. REVISED ESTIMATES OF FATAL CANCER RISKS FROM THE HIROSHIMA-NAGASAKI DATA.

A number of authors have attempted to derive preliminary estimates of cancer risks from the Hiroshima-Nagasaki data following the initial publication of revised kerma doses in 1981. These attempts have generally been regarded as premature. The 1982 UNSCEAR [31] report summarized the situation as follows:

> "While it is impossible yet to say exactly what influence the revisions, if accepted, will have on the risk estimates, it is unlikely that this influence will exceed a factor of 2. Indeed, improved agreement between data from Hiroshima and Nagasaki may tend ultimately to strengthen confidence in the estimates. Secondly, the information derived from the survivors of the atomic bombs in the two cities is only one of the sources of human exposure that the Committee has used in arriving at its estimates. While little change is therefore expected to result in regard to estimates for cancer induction in man by X and gamma rays, an important presumed source of information for whole body neutron irradiation will no longer be available...." [31].

In its consideration of dose-response relationships, the 1986 UNSCEAR report [32] again omitted consideration of Hiroshima-Nagasaki data because of the absence of reliable data at that time. Definitive data based on absorbed organ doses first became available in 1987 with two reports by Preston and Pierce [2] and Shimizu et al [3]. These reports formed the basis of two general statements on revised risk estimates [1,33,34] and will be reviewed in more detail below.

1. Risk coefficients per Gy

The first major conclusion is that there is little change in cancer risk estimates based on absorbed organ doses in Gy (Table 10). In general, the risk coefficients based on DS86 absorbed doses in Gy tend to be somewhat smaller than those based on T65DR absorbed doses for deep organs in the body, and to be somewhat higher for shallow organs such as the female breast (Table 10).

Secondly, apparent differences in leukemia risk coefficients derived from follow-up of Hiroshima survivors and those derived from follow-up of Nagasaki survivors have largely disappeared. Previous estimates of risk coefficients per Gy absorbed dose for induction of leukemia were approximately twice as high for Hiroshima as for Nagasaki survivors when based on T65D dose estimates [3,10]. These apparent differences were attributed to differences in the neutron component of the radiations received in these two cities and a number of articles by eminent scientists have been concerned with this problem [see for example 35]. It now appears that these apparent differences were due largely to errors in dosimetry [3]. There are still appreciable difference between cities in calculated risk coefficients for other types of cancers. Observed dose-response relationships for selected fatal cancers in the two cities are illustrated in Figure 6.

2. Risk coefficients per Sv

Although the neutron component of the absorbed DS86 doses is fairly small for both Hiroshima and Nagasaki, some assumptions concerning the quality factor (Q) or relative biological effectiveness (RBE) of neutrons must be made in order to express risk coefficients in Sv. Preston and Pierce [2] among others have attempted to derive best estimates of the neutron RBE from the revised Hiroshima-Nagasaki data. But as pointed out by Preston and Pierce [2], "very little in the way of estimation of RBE of neutrons can be done from these data, especially with the new dosimetry. However, it is important to consider the consequences of various assumed RBE values." The ICRP in 1985 recommended that Q for fast neutrons should be increased from 10 to 20 [36] and provided more extensive data on recommended Q values for neutrons of different energies in ICRP Publication 51 [37]. Shimizu et al [3] calculated cancer risk coefficients per Sv for the Hiroshima-Nagasaki survivors using various assumed values for neutron RBE's (Table 11). [Minor differences in risk coefficients per Sv at an RBE of 1 (Table 11) and per Gy (Table 10) are not explained.] In general, risk coefficients calculated with an assumed neutron RBE of 20 tend to be about 80% of those calculated with an assumed RBE of 1.

Table 10. Revised risk coefficients for Hiroshima-Nagasaki survivors, based on cancer mortality 1950-1985 and on absorbed organ doses in Gy [3].

Site of cancer	Dose system	Excess relative risk per Gy	Ratio DS86/T65DR	Excess deaths per 10^4 person years per Gy	Ratio DS86/T65DR
Leukemia	DS86	5.21	0.90	2.94	0.95
	T65DR	5.76		3.11	
All except leukemia	DS86	0.41	0.71	10.13	0.73
	T65DR	0.58		13.97	
Breast	DS86	1.19	1.31	1.20	1.33
	T65DR	0.91		0.90	
Lung	DS86	0.63	0.88	1.68	0.89
	T65DR	0.72		1.89	
Stomach	DS86	0.27	0.69	2.42	0.72
	T65DR	0.39		3.34	

More important may be the effect of assumed RBE on the risk coefficients as calculated from the new DS86 dose estimates and the old T65DR dose estimates (Table 11). Because the apparent neutron component of the radiation doses received by the Hiroshima-Nagasaki survivors has now been substantially reduced, risk coefficients per Sv have in general been increased. At an assumed neutron RBE of 10, which was the value generally used in earlier assessments prior to 1985, the calculated risk coefficients per Sv are increased by values ranging from 5% for all fatal cancers (except leukemia) to 48% for leukemia to 133% for female breast cancer (Table 11). At a neutron RBE of 20, the increases would be about 38%, 95% and 215% respectively (Table 11).

3. Dose-response relationships

Another critical factor which must be considered for estimation of cancer risks at low radiation doses is the shape of the dose-response curve. Although a trend towards increased risk at high radition doses is usually apparent, the epidemiological data from Hiroshima and Nagasaki are in themselves not precise enough to provide definitive estimates of cancer risk (if any) at low radiation doses (Figure 5). It is therefore necessary to make some assumptions about the presumed shape of the dose-response curve. Shimizu et al [3] have analyzed the data in terms of the three postulated dose-response relationships that were

Table 11. Revised risk coefficients for Hiroshima-Nagasaki survivors, based on cancer mortality 1950-1985 and on absorbed organ dose equivalents in Sv [3].

Site of cancer	Assumed neutron RBE	Excess deaths per 10^4 person years per Sv, DS86 doses	Ratio DS86 / T65DR
Leukemia	1	2.95	0.96
	10	2.67	1.48
	20	2.40	1.95
All except leukemia	1	10.1	0.73
	10	9.41	1.05
	20	8.76	1.38
Breast	1	1.22	1.36
	10	1.00	2.33
	20	0.82	3.15
Lung	1	1.80	0.95
	10	1.59	1.35
	20	1.42	1.78
Stomach	1	2.63	0.78
	10	2.36	1.17
	20	2.10	1.57

considered in BEIR III (1980). In all three cases, the dose-response relationship for neutrons is assumed to be linear. The dose-response relationships for gamma-rays were postulated to be quadratic, linear-quadratic or linear. Deviances were calculated and examined to determine, if possible, which model fitted the data best. From the results (Table 12), it was impossible to assert that one of the models is clearly better than any other. The same conclusion had been reached in an earlier analysis in the 1980 BEIR III report. The choice between dose-response models must therefore be based on other considerations such as those reviewed by NCRP [12] and by UNSCEAR [32].

4. Preliminary estimate lifetime cancer risks

One preliminary estimate of lifetime cancer risks for the Hiroshima-Nagasaki survivors was provided by Preston and Pierce [2]. Their data are summarized in Table 13. The preliminary estimates by Preston and Pierce (Table 13) suggest a lifetime risk of radiation-induced fatal cancers for a general population of all ages which is between 4.4 and 9.6 times greater than the lifetime cancer risk of 1.3 per 100 Sv suggested in the 1977 ICRP recommendations (Tables 1 and 2) for low radiation doses. Although the risk data are incomplete, these

Table 12. Computed deviance for the fit of three theoretical dose-response models to the epidemiological data for the Hiroshima-nagasaki survivors [3].

Site of cancer	Dose-response model for gamma-rays		
	Quadratic	Linear-quadratic	Linear
Leukemia	516	511	512
All except leukemia	1149	1138	1141
Breast	292	288	289
Lung	592	583	584
Stomach	866	856	857

preliminary estimates by Preston and Pierce [2] obviously warrant some further discussion.

The following points might be noted:

(a) The best estimate of lifetime risk of induced leukemias at low radiation doses is suggested to be about 0.47×10^{-2} per Sv [2]. This value is 2.35 times higher than the best estimate of 0.2×10^{-2} per Sv given by ICRP in (1977) [6]. The value derived from Table V-16 of the 1980 BEIR report [11] based on exposure to 10 mSv low-LET radiation per year throughout life is essentially identical to that given in the 1977 ICRP publication [6]. Preston and Pierce [2] attribute most of this increase in leukemia risk to the new dosimetry. Shimizu et al (1987) have added some comments on the selection of follow-up groups by Preston and Pierce [2]; their data suggest that the increase in leukemia risk attributable to the changes in dosimetry might be closer to 1.5-2.0 (Table 11) if the Q for fast neutrons is assumed to be 20.
(b) Preston and Pierce [2] used the relative risk projection model to estimate future lifetime risks for all other radiation-induced cancers, and assumed that relative risks which have been measured to date would remain constant throughout the lifetime of the remaining two-thirds of the Hiroshima-Nagasaki survivors. Their projected estimates of lifetime risk of fatal cancers other than leukemia which might be induced by exposure to low doses of radiation are $5.1 - 10.1 \times 10^{-2}$ per Sv (Table 13) assuming that Q for fast neutrons is 20. This is 4.9 to 9.6 times higher than the value of 1.05×10^{-2} per Sv suggested by the ICRP in 1977 [6], and also appreciably higher than the range $0.5 - 1.5 \times 10^{-2}$ per Sv that was calculated in 1980 on the assumption of lifetime exposure to 10 mSv low-LET radiation per year [11]. As noted in section B, the range suggested int he 1980 BEIR report [11] depends on the use of both absolute and relative risk projection models, whereas Preston and Pierce [2] relied solely on a relative risk model.

Table 13. A preliminary estimate of lifetime risks of radiation-induced fatal cancers among the Hiroshima-Nagasaki survivors [2].

Assumed neutron RBE	Site of cancer	Risk per 100 Sv *	
		Linear dose-response model	Low dose extrapolation using a dose reduction factor of 1.5 to 3.0 for gamma-rays [32]
5	Leukemia	1.3	0.4 - 0.9
	All except leukemia	16.7	5.6 - 11.1
	Total fatal	18.0	6.0 - 12.0
20	Leukemia	1.1	0.4 - 0.7
	All except leukemia	15.2	5.1 - 10.1
	Total fatal	16.3	5.5 - 10.8

[* See footnote to Table 1.]

Preston and Pierce [2] attribute roughly one-third of the average estimated increase to the effect of change in dosimetry (see Table 11), about one-third to additional epidemiological follow-up since 1972 (see Figure 5), and roughly 40% to improved statistical methods. The data provided by Shimizu et al [3] suggest that the increase for all cancers except leukemia resulting from the new dosimetry might be in the region of 38% only, depending Q is 20 for fast neutrons (Table 11).

The Hiroshima-Nagasaki data do not provide any reason to reject the relative risk projection model, or to reject the concept that the relative risks measured to date might continue to apply throughout the remaining lifespan of the survivors (see section D). However, these theoretical concepts are subject to considerable uncertainty; the most important factor in assessment of lifetime cancer risks may well be the possible two-fold increase in estimates of leukemia risk per Sv resulting from the dosimetry (Table 11) and from the new ICRP recommendations on quality factor for fast neutrons [36,37].

5. Discussion of revised lifetime cancer risk estimates

The general impression that we have received from our own consideration of the publication of Preston and Pierce [2] and the subsequent publications by

Shimizu et al [3,45] is that the lifetime cancer risk estimates of Preston and Pierce (Table 13) are conservative and are likely to be too high. This major impact of the new dosimetry and the new epidemiological data for the Hiroshima-Nagasaki survivors will probably be on risk estimates for leukemia, on projected risk estimates for persons of younger ages at the time of exposure to radiation (Figure 5; see also Table 3) and on risk estimates for radiation-induced breast cancer in females of all ages at the time of exposure (Tables 8 and 11).

The maximum value for cancer risk estimates for the Hiroshima-Nagasaki survivors, using a relative risk projection model, a Q of 5 for fast neutrons, and a linear dose-response relationship (Table 13), would be 18.0/1.25 = 14 fold greater than the 1977 ICRP estimate [6] of 1.25 fatal cancers per 100 Sv. The minimum increase, assuming a 48-95% increase in the risk coefficient of 0.2 per 100 Sv for leukemia and a 5-39% increase in the risk coefficient of 1.05 per 100 Sv for all other fatal cancers (Table 11), would be in the region of 1.1 to 1.5 fold. The most appropriate value of Q for the neutrons absorbed by the Hiroshima-Nagasaki survivors will require further examination. The recommended value of 20 [37] applies to neutrons of 0.5 - 1 MeV energy at the site of absorption in a tissue. The Q for neutrons of 0.5 - 1 MeV incident energy at the surface of the body is expected to be appreciably lower than 20 for deep tissues in the body [38]. The best value for calculating effective dose equivalent for neutrons of 0.5 - 1 MeV incident energy is probably Q = 5 [see Table 18 in reference 37]. Other values of Q would be more appropriate for neutrons of other energies [37].

It is important to recognize that estimates of the probability of radiation-induced fatal cancer do <u>not</u> depend only upon analysis of the data on Hiroshima-Nagasaki survivors. Data on other groups of persons exposed to high radiation doses in the past [see 10,11,32] must also be considered in derivation of best estimates. Consideration of these other data is beyond the scope of the present review. This task is currently being undertaken by the UNSCEAR committee, the BEIR V committee and Committee 1 of the ICRP. Other data to be considered include (a) radiation-induced leukemias in persons exposed in the past to high doses of X-rays, to radium-224 and to thorotrast for medical purposes, and on radium-226 dial painters, and (b) radiation-induced breast cancers in radium dial painters in the U.K. and in women exposed to X-rays for various medical purposes in Massachusetts, New York and Canada. Some of these data were available for the 1977 UNSCEAR and 1980 BEIR reviews, while other data have been published more recently.

Meanwhile two official statements on this topic have been published. The U.K. NRPB has suggested that estimates of the risk of radiation-induced fatal cancer might be increased about <u>three</u>-fold [34]. No explanation for the derivation of adult radiation workers and for a general population of all ages at the time of exposure. The 1987 ICRP statement suggested that risk estimates for a general population of all ages might be increased by about <u>two</u>-fold, with a smaller change for a worker population of ages 18-65 [1].

Some data which were recently made available [45] shed further light upon this discussion. Using the most reliable risk coefficients for excess cancers among the Hiroshima-Nagasaki survivors that are available at present, projecting

these risk coefficients on to the total Japanese population as of 1982, and using a linear non-threshold dose response model with no allowance for a decrease in biological effects at low dose-rate, the projected lifetime risk of radiation-induced fatal cancers would be in the region of 4.5 to 7.1 per 100 person-Gy of absorbed organ dose [45]. These estimates are in the region of 5 times that suggested by the ICRP in 1977 [6]. An overall reduction by 2.5-fold to compensate for effects of low doses at low dose rate as compared to higher doses at high dose rate in the case of low LET radiation would of course result in an average risk estimate which was 2 times greater than the 1977 ICRP value. Dose-rate reduction factors suggested in previous reviews were about 2 to 2.5 - fold [10], 2.3-fold [11], 2 to 10-fold [12] and somewhere between 1 and 5-fold [32]. It remains to be determined whether similar dose-rate reduction factors for low LET radiation will be confirmed by the scientific committees who are currently considering the best risk estimates for radiation-induced fatal cancers.

Two other interesting points emerge from these recent calculations of projected risk estimates based on Hiroshima-Nagasaki data and on Japanese vital statistics [45]. First, there is, as expected, little difference in average loss of life expectancy for projections based on the multiplicative (relative) and additive (absolute) risk projection models, despite an appreciable difference in total number of fatal cancers calculated on the basis of these two models [45]. Second, there is little difference in the calculated, theoretical numbers of radiation-induced fatal cancers for a total population of all ages and for a population age 15-64 at the time of exposure [45]. This latter result does not agree with the suggestion made in the 1987 ICRP Como statement [1] as quoted in the introduction of this review.

It is obvious that estimated risks of radiation-induced fatal cancer will probably be increased in the near future, but the final estimate of the quantitative increase will depend upon ongoing reviews of the scientific literature by various committees appointed for this purpose. For the time being, the practical impact [39] of these suggested revisions would logically appear to be a renewed emphasis on the ICRP recommendation that all exposures should be kept as low as reasonably achievable, economic and social factors being taken into account [6]. Actual revisions of current dose limits based on project revisions of cancer risk estimates might well be considered to be premature until further detailed analyses become available over the next 2 or 3 years [1].

A 2 or 3-fold increase in quantitative estimates of the fatal cancer risks for low-LET radiation would have little effect on the ratio of estimated hazards of exposures to low-LET radiation and to radon progeny. The estimated hazards of inhalation of radon progeny are also being increased by about 2-fold [15,27,28] above those recommended in ICRP Publication 32 [40].

F. EFFECT OF REVISED HIROSHIMA-NAGASAKI DOSIMETRY ON ESTIMATES OF STOCHASTIC RISKS OTHER THAN FATAL CANCER.

The 1977 ICRP report noted that radiation was likely to induce approximately equal numbers of curable, non-fatal cancers and of fatal cancers (Table 1), but

assigned a relative weight of zero to the curable cancers. Others, notably Radford [41,42], have insisted that curable cancers are just as important as fatal cancers. The ICRP has subsequently considered weighting factors of 0.1 to 0.3 for curable cancers as compared to fatal cancers [7,43], but has not yet issued any formal recommendations on this question. Sites with appreciable numbers of radiation-induced curable cancers include the skin, thyroid gland and female breast [7]. Previous reports on the Hiroshima-Nagasaki survivors have provided some information on curable breast and thyroid cancers induced by radiation [10,11], but most of the relevant data has come from other groups of persons exposed to high doses of medical X-rays. No reports on risks of radiation-induced curable cancers in Hiroshima-Nagasaki survivors using the new revised dose estimates have become available to us as yet. However, the impact of the new dosimetry on risk estimates for curable cancers is likely to be relatively small.

Genetic risk estimates (Table 1) are based primarily on the results of animal experiments [10,11,14,31,32]. Although interesting trends have been observed, there is no statistically-significant increase in disorders of genetic or partially-genetic origin in the children of the Hiroshima-Nagasaki survivors [44]. The impact of the revised Hiroshima-Nagasaki dosimetry on quantitative estimates of the genetic hazards of exposure to ionizing radiation is expected to be negligible.

On the other hand, quantitative estimates of the risk of mental retardation after irradiation in utero (Table 1) are based solely upon data from Hiroshima-Nagasaki survivors who were pregnant at the time of the atomic bomb explosions in 1945 [8,32]. Detailed analyses of the impact of revised dosimetry are in progress but have not yet been published. Two important sentences from the 1987 ICRP statement should be noted:

> "Preliminary information, however, indicates that the estimated risks may be increased somewhat with the new dosimetry, from an average of 40% to 45% per Gy in the more sensitive period [of pregnancy]. It is potentially important for radiation protection, that preliminary analyses presented to the Commission's Committee 1 on Biological Effects indicate that significant thresholds may exist."

If there were indeed a threshold dose below which in utero irradiation did not produce mental retardation in the developing child, special measures to protect the fetus against low occupational doses of radiation might not be required. Further studies are required in order to determine whether or not this supposition has any validity. Two sentences from a recent U.K. NRPB statement might be noted:

> "However, the Board [NRPB] has sought the advice of the Medical Research Council, who have formally advised that the data are not sufficiently firm to warrant a change in present standards, and ICRP now considers there may be a threshold. The Board has decided that at present no special provisions are required for this end point in the control of occupational exposure" [39].

This particular suggestion may require further reconsideration in the light of recent data on induction of mental retardation in persons who were exposed in utero to high doses of radiation at high dose rate at Hiroshima and Nagasaki in 1945. The new data on the small number of cases of severe mental retardation (in general, an IQ considerably less than 70) observed after irradiation at 8-15 weeks post-conception do suggest a threshold dose below which no effects of irradiation are observed [46]. However, the total number of cases of severe mental retardation in this group is less than 20 and is thus insufficient to define the shape of the dose-response curve accurately. Data on reduction in the average IQ of all persons who were exposed prenatally at the same period provide a better fit to a linear, non-threshold dose-response model [46]. If these two endpoints are in fact closely related, it would therefore seem prudent for purposes of radiation protection to assume a linear, non-threshold dose-response model until such time as further clarification of these biological observations is available. On this basis, any relaxation of current AECB restrictions on radiation exposure of pregnant women would not be considered prudent at the present moment.

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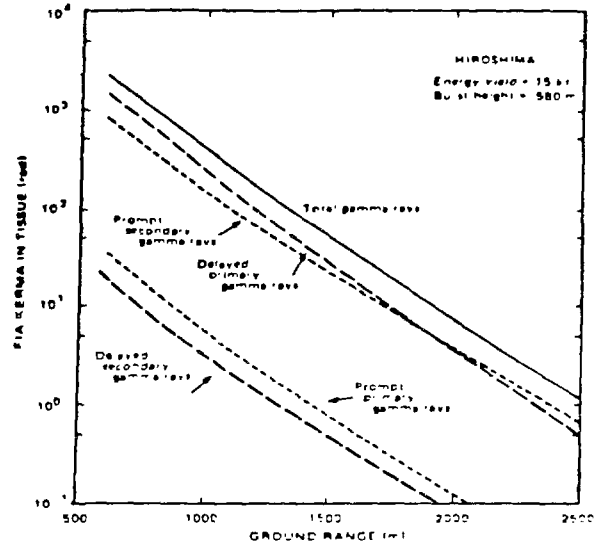

Figure 34 Contributions of various gamma-ray sources to the DS86 values for total gamma-ray kerma in Hiroshima

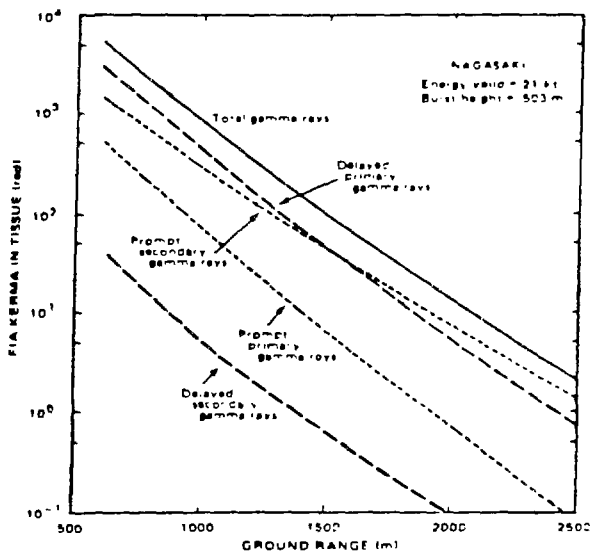

Figure 35 Contributions of various gamma-ray sources to the DS86 values for total gamma-ray kerma in Nagasaki

Figure 1. Graphs from reference [20].

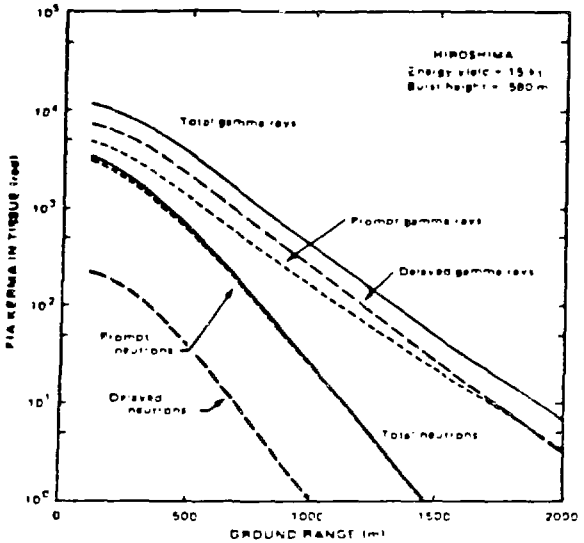

Figure 30 Prompt and delayed radiation contributions to the DS86 values for neutron and gamma-ray kerma in Hiroshima

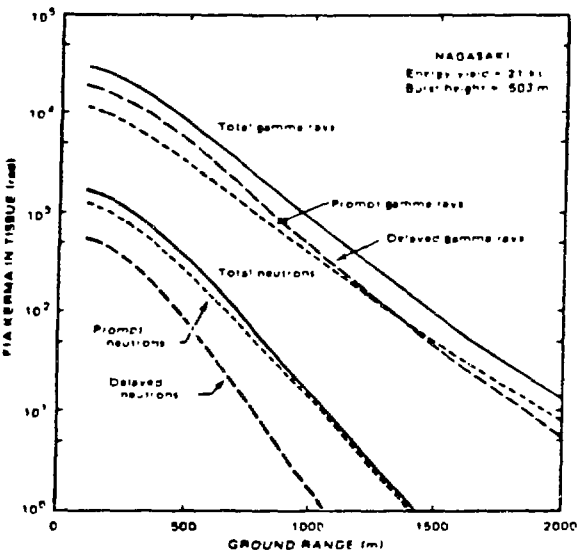

Figure 31 Prompt and delayed radiation contributions to the DS86 values for neutron and gamma-ray kerma in Nagasaki

Figure 2. Graphs from reference [20].

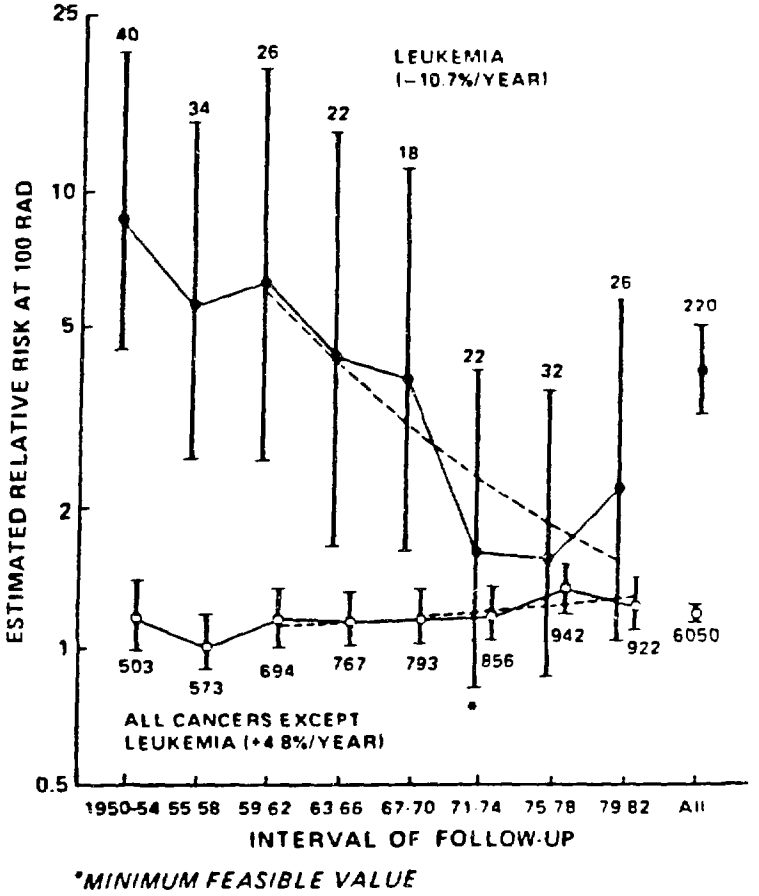

Figure 4. Variation of relative risk of mortality from leukemia and from all cancers except leukemia with time over the period 1950-1982 among the Hiroshima-Nagasaki survivors [4]. Data are averages for both cities, both sexes and all ages at the time of the bomb. Doses are T65DR estimates.

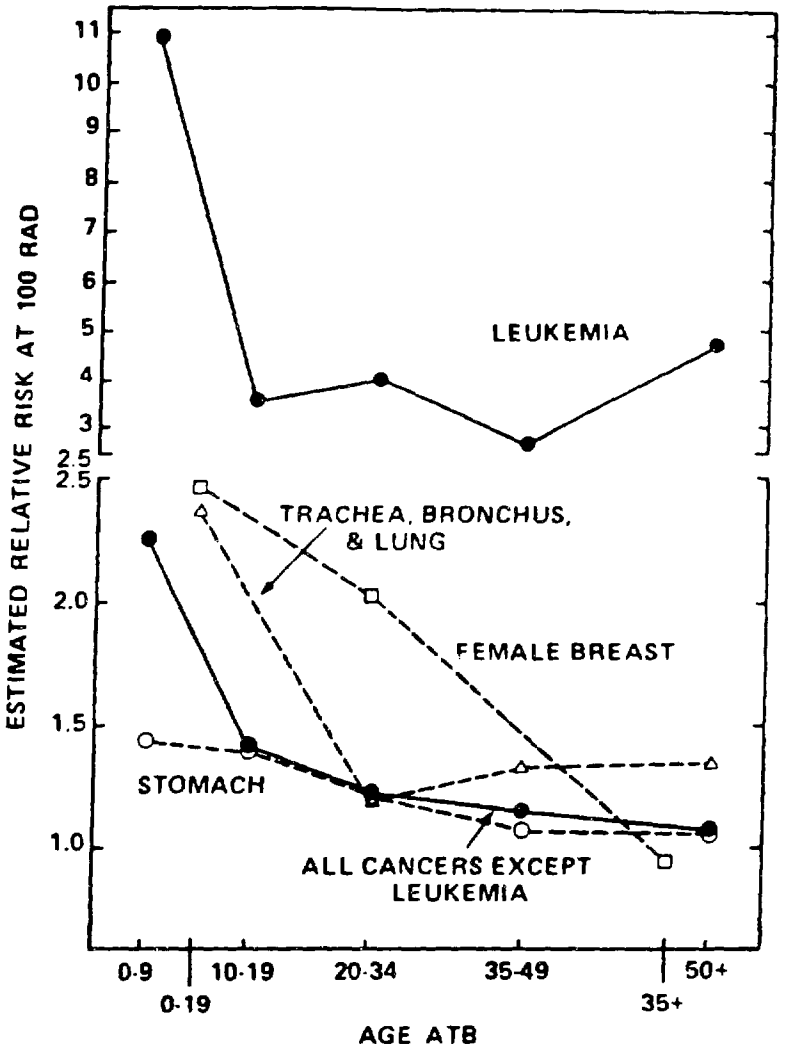

Figure 5. Variation of relative risk of mortality from different types of cancers by age at the time of the bomb (ATB) for the Hiroshima-Nagasaki survivors [4]. The vertical axis is compressed above 2.5 to accommodate the large values for leukemia. The 0-9 and 10-19 age groups are combined for lung and breast cancers; the 35-49 and 50+ age groups are combined for breast cancer. Doses are T65DR estimates. Epidemiological data are for the period 1950-1982.

(a) Leukemia

(b) All cancers except leukemia

(c) Breast cancer

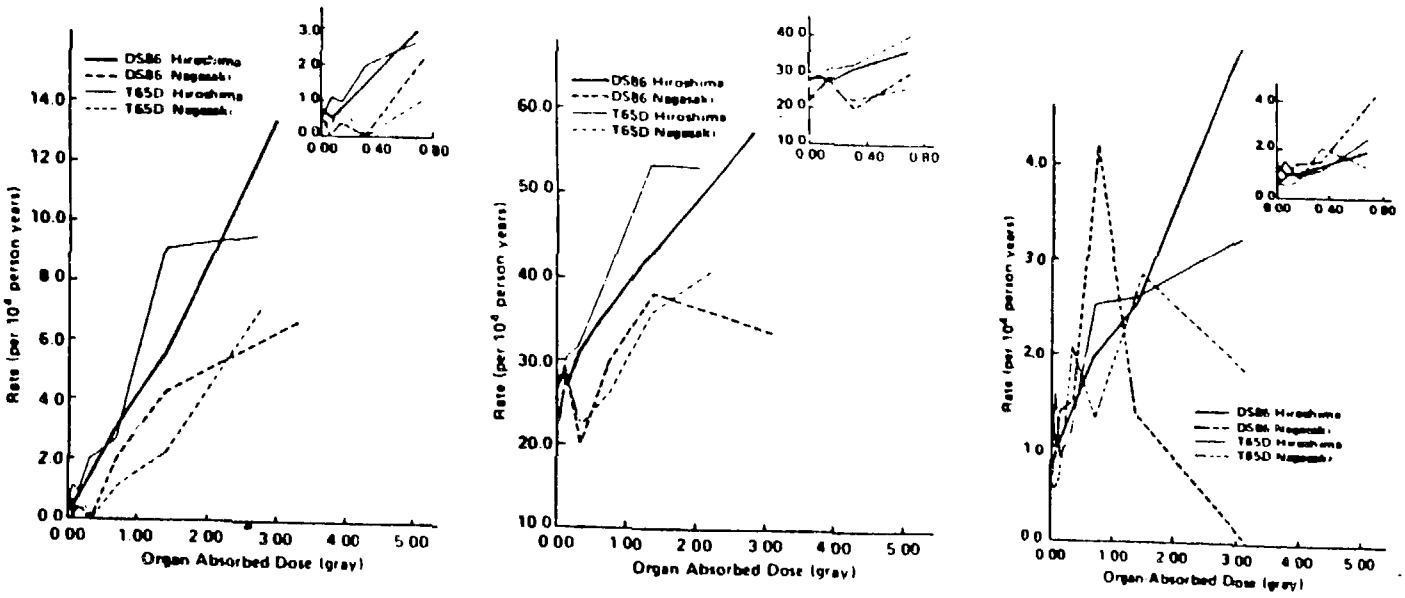

Figure 6. Dose-response curves for mortality from (a) leukemia, (b) all cancers except leukemia, and (c) breast cancer for the Hiroshima-Nagasaki survivors by city and by dosimetry system [3]. The insert in the upper right corner of each graph is an enlargement of the observed cancer rates at absorbed organ doses of less than 1 Gy. Data are averages for all ages at the time of the bomb and are based epidemiological follow-up over the period 1950-1985.