MALIGNANCY RISK TO HUMANS FROM TOTAL BODY γ-RAY IRRADIATION*

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ABSTRACT: Based on data from the A-bomb survivors and radiobiological studies on the dose-rate effect, the following estimates are made of the cumulative deaths from induced malignancies in a natural population of 1,000,000 persons of mixed ages each receiving a total body dose of 1 rad from γ-rays:

	RADIATION-INDUCED DEATHS at HIGH dose-rate (over 10 rad/min)	at LOW dose-rate (under 0.01 rad/min)
LEUKEMIAS		
Higher linear estimate	40	20
Preferred linear estimate	25	5
Lower linear estimate	14	1
Dose squared estimate	0.1	0.004
FATAL CANCERS (excluding leuk.)		
Higher linear estimate	150	75
Preferred linear estimate	100	20
Lower linear estimate	50	5
Dose squared estimate	0.4	0.016

The linear estimates are for use in radiation protection work, while the dose squared estimates illustrate the radiobiological possibility, based on the observed incidence of leukemia at Nagasaki, that the dose-response to γ-irradiation may be sigmoid, rather than linear.

Caution: These estimates are provisional and subject to future revision as more information is acquired.

INTRODUCTION

We have carefully reviewed previous estimates of risk including the BEIR Report,[1] UN Reports,[2] Marinelli,[3] Dolphin,[4] and ICRP Publications 14[5] and 8[6]. Each of these prior reviews has provided a valuable step toward the ultimate goal of a better quantification of the effects of radiation on mankind.

The most valuable information on the effects of total body irradiation of humans is from the A-bomb survivors.[1-7] At the time of burst they were a population of mixed sexes in which fetuses, children, and adults were represented. The average age at the time of burst was 29 years (BEIR Report,[1] pg. 148). We assume that an average survival time of about 40 years after exposure can be used to obtain a reasonable approximation to the average risk, with

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the realization that some of these A-bomb survivors have died sooner and were at risk a shorter time, while others will die later and be at risk a longer time. Our present risk estimates are averaged over the entire population of A-bomb survivors, which undoubtedly consists of some individuals more sensitive than average, as well as other individuals who are less sensitive.

We have analyzed the data in such a way so as to minimize interference from neutrons on the derived risk estimates for γ-rays. Our preferred estimates of lifespan risk involve future projections based on the observed trends of decreasing mortality rates from induced leukemia and increasing mortality rates from induced cancer. Yet despite the differences between our approach and that of the BEIR Report, there is reasonable biological agreement between our linear estimates and the BEIR linear estimates for high dose-rates.

To indicate the uncertainties in our "preferred" linear estimates, we have also derived "higher and lower" linear estimates. However, there is a strong possibility that the actual dose-response relationships may be sigmoid,[1,7-11] rather than linear. Therefore, alternative estimates of risk are given based on a dose-squared model.

Other reports have discussed the lesser overall effectiveness per rad expected at low dose-rates of γ-irradiation. We have now assembled sufficient radiobiological evidence to offer what may be the first realistic estimates of the actual risk at low dose-rates of total body γ-irradiation. This may be the most important contribution of the present report.

However, it must be emphasized that this is an interim report. Most of the irradiated subjects are still alive and must continue to be studied. Some uncertainties exist in the tissue doses actually received. We still lack fundamental knowledge of how malignant neoplasms are induced by irradiation. Therefore, it must be emphasized that the estimates of risk presented here are provisional and may require revision in the light of future information.

Now an outline of our analysis will be given. First, the risk for leukemia induction at high dose-rates will be estimated from the Nagasaki A-bomb data, taking advantage of the small exposure to neutrons. Next, the risk for cancer induction at high dose-rates will be obtained by multiplying the Nagasaki leukemia risk by the projected ratio of total induced cancer/total induced leukemia. Finally, the risks for leukemia and cancer induction at low dose-rates will be obtained by multiplying the risks at high dose-rates by appropriate effectiveness factors.

LEUKEMIA RISK AT HIGH DOSE-RATES

The mortality rate from leukemia in the A-bomb survivors at Hiroshima and Nagasaki from 1950 to 1970 is given in Table 1. The exposure is the "tissue kerma in air", which is the kinetic energy released per unit mass from the γ-rays and neutrons interacting with a small bit of tissue suspended 3 feet above ground.[12] The effect of shielding provided by buildings is included. The "tissue kerma in air" has also been called the "field free dose" or the "air dose," and will be referred to in this paper simply as the "kerma." The γ-ray dose within a person may be slightly less than the kerma from A-bomb γ-rays. However, the neutron dose within a person is much less than the kerma from A-bomb neutrons. This is due to a greater attenuation by the body of the neutrons than of the γ-rays. Hopefully, reliable estimates of the actual dose distributions within the human body may soon become available. In the meantime we shall assume provisionally that the γ-ray doses received by the A-bomb survivors are approximately equal to the γ-ray "tissue kerma in air." For conciseness, both the dose in rads and the dose equivalent in rems will

usually be referred to by the general term "dose" throughout this paper.

Table 1. LEUKEMIA MORTALITY IN A-BOMB SURVIVORS (1950-1970)
(Jablon and Kato, BEIR Report p. 108, 1972, corrected)

Tissue kerma in air (rads)			No. of	Person yr	Leukemia	[Leuk./yr / 10^6 persons]
Total	Gamma	Neutron	Persons	At risk	Deaths	
HIROSHIMA						
200+	269.3	93.9	1460	26700	27	1010
100-199	108.5	30.1	1677	30200	10	331
50-99	56.9	13.3	2665	48300	7	145
10-49	17.6	4.3	10707	195400	17	87
0-9	0.9	0.3	43730	795600	34	43
NAGASAKI						
200+	329.1	5.6	1310	24300	15	616
100-199	144.3	1.4	1229	23000	3	130
50-99	70.3	0.2	1231	22900	0	0
10-49	21.3	0.0	3700	67600	2	30
0-9	2.3	0.0	11404	209900	11	52

In the fitting of dose-response curves, an important constraint is that the curves pass through (or at least near) control incidence. This is because no radiation effects occur at zero dose. The lowest dosage-group at each city is the most appropriate control for that city since it was followed-up similarly to the higher dosage-groups and the average kerma to the lowest groups was negligible (a total kerma of 1.2 rads at Hiroshima and 2.3 rads at Nagasaki). For each city, dose-response curves were started at the incidence rate of the lowest dosage group (regarded as zero rads) and were given a slope such that the predicted sum of leukemias exactly equalled the observed total, using the curve-fitting procedure of Mays and Lloyd.[9] The slope for Hiroshima (2.23 leuk. per yr/10^6 person rad) was much steeper than the slope for Nagasaki (0.88 leuk. per yr/10^6 person rad), primarily due to the greater neutron component from the Hiroshima weapon and the greater potency of neutrons relative to γ-rays in inducing malignancy. By trial and error it was found that the slopes for the two cities became equal at 0.8 leuk. per yr/10^6 person rem when an average neutron potency factor* of 9 was assumed. The insensitivity of the Nagasaki risk rate coefficient to changes in the assumed neutron potency factor is shown in Table 2.

Table 2. NEUTRON POTENCY FACTOR and RISK RATE COEFFICIENT

Neutron Potency Factor	[Leukemias/year / 10^6 person rem]	
	Nagasaki	Hiroshima
1	0.88	2.23
9	0.80	0.79
20	0.72	0.42

*The neutron potency factor is defined as the ratio of γ-ray kerma/neutron kerma for equal biological effect. It is not equal to the neutron RBE which is the ratio of absorbed doses, since attenuation within the body causes the neutron dose in the body to be much less than the neutron kerma in air.

Wide ranges in the assumed neutron potency factor (1-20) cause only a small variation (± 10%) in the calculated risk rate coefficient for Nagasaki. This is due to the small neutron component of the Nagasaki weapon. The conclusion that the neutron potency factor increases as the kerma decreases[13] is probably correct, but has a negligible influence on the average risk rate coefficient for Nagasaki.

The preferred linear estimate will be taken as 0.80 leuk. per yr/10^6 person rem for the years 1950-1970 (5 to 25 years after irradiation). However, there is some statistical uncertainty due to the small number of 20 leukemia cases in the Nagasaki population exposed to 10 rads and over. From Poisson statistical tables,[14] a 10% chance of having 20 or fewer cases corresponds to an expectation value of 27.1 cases (higher limit), whereas a 10% chance of having 20 or more cases corresponds to an expectation value of 14.5 cases (lower limit). In the exposed Nagasaki population the 20 cases at 10 rads and over, correspond to 12.8 induced cases plus 7.2 natural cases. The higher limit corresponds to 27.1-7.2 = 19.9 induced cases, while the lower limit corresponds to 14.5-7.2 = 7.3 induced cases. The higher linear estimate is (19.9/12.8)(0.80) = 1.25 leuk. per year/10^6 person rem, while the lower linear estimate is (7.3/12.8)(0.80) = 0.45 leuk. per yr/10^6 person rem.

The non-linear appearance of the plotted dose-response curve for Nagasaki raises reasonable doubt on whether the dose-response is really linear (Fig. 1). Among the 4931 persons exposed at Nagasaki to 10-99 rads (Table 1), 7.2 total cases of leukemia are predicted (4.7 natural plus 2.5 induced according to the "preferred" linear estimate), whereas only 2 leukemia cases were actually observed. A linear relationship predicting 7.2 cases when only 2 were observed

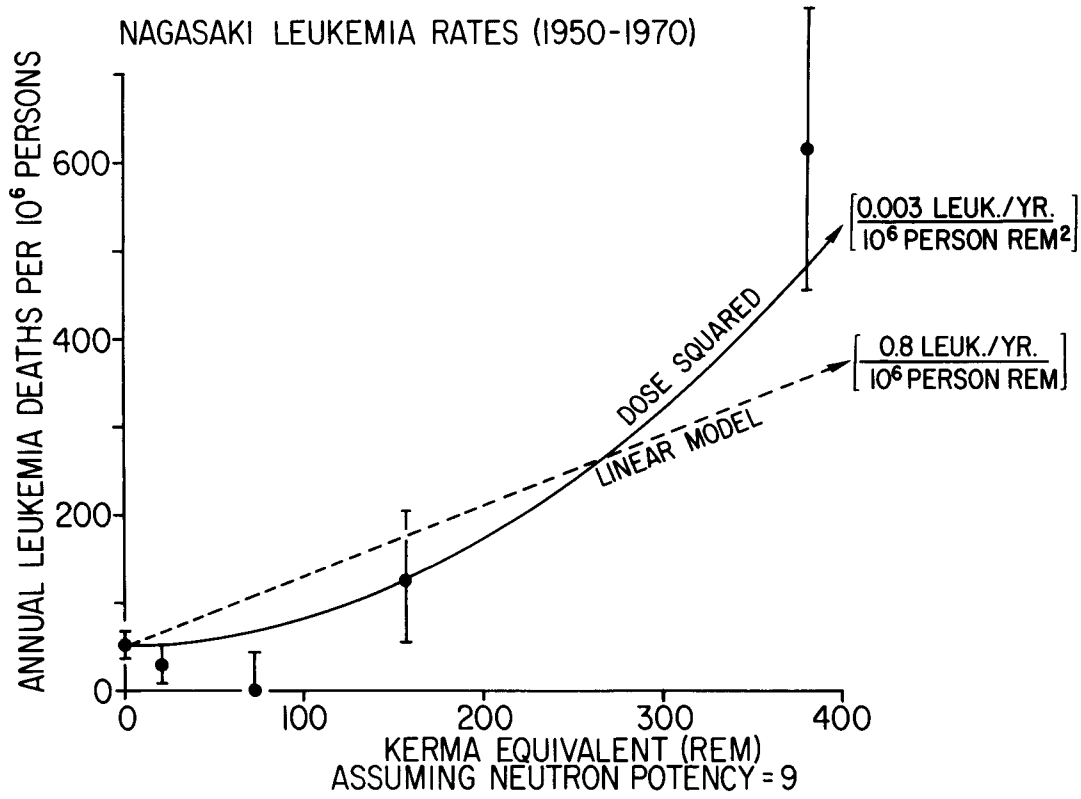

Fig. 1. Linear and dose-squared models fitted to the leukemia rates observed at Nagasaki (1950-1970). The dose-squared model makes a much better fit. The error bars are ± 1 standard deviation.

Table 3. INDUCED MALIGNANCIES IN HIGH DOSE A-BOMB SURVIVORS EXPOSED TO 200 RADS AND OVER AT HIROSHIMA AND NAGASAKI*
(Estimates in parentheses are for unavailable data)

Years After Irrad.	Years In Interval	Leukemia		Cancer	
		Deaths/yr 10^6 persons	Deaths 10^6 persons	Deaths/yr 10^6 persons	Deaths 10^6 persons
0 to 5	5	(800)	(4000)	(0)	(0)
5 to 10	5	1300	6500	600	3000
10 to 15	5	900	4500	200	1000
15 to 20	5	500	2500	300	1500
20 to 25	5	500	2500	2000	10000
25 to 30	5	(400)	(2000)	(3700)	(18500)
30 to 35	5	(300)	(1500)	(5400)	(27000)
35 to 40	5	(300)	(1500)	(7100)	(35500)
Total			25000		96500

*The net rates of induced leukemia, and cancer excluding leukemia, within the observed intervals of 5-25 years were scaled from Fig. 16 of Jablon and Kato.[7]

Induced leukemia mortality before the start of the ABCC study (0 to 5 yr) is unavailable but is assumed reasonably close to the average rate during the observed intervals. Assumed leukemia mortality rates after 25 years are based on present trends. For this heavily irradiated population, exposed to a tissue kerma in air of 200 rads and over from γ-rays and neutrons at Hiroshima and Nagasaki, 25000 total deaths from induced leukemia/10^6 heavily irradiated persons is predicted (2.5% mortality from induced leukemia).

Induced cancer mortality during 0 to 5 years is assumed virtually equal to zero, due to the long latent periods typical for non-leukemic malignancy. Beyond 25 years, the mortality rates for induced cancer are tentatively assumed to increase by roughly the same amount in each successive 5-yr interval as the observed increase of 1700 cases per yr/10^6 persons which occurred between the intervals 15 to 20 yr and 20 to 25 yr. Under this provisional assumption 96500 total deaths from induced cancer/10^6 heavily irradiated A-bomb survivors is predicted (about 10% mortality from induced cancer excluding leukemia).

If the mortality rate from induced cancer remained constant at 2000 cases per yr/10^6 persons, then 45500 total deaths from induced cancer/10^6 heavily irradiated A-bomb survivors would be predicted. Conversely, if after 25 years the increase in each successive 5-year interval were 3400 cases per year/10^6 persons (or double the observed increase between 15 to 20 yr and 20 to 25 yr) 147500 total deaths from induced cancer/10^6 heavily irradiated A-bomb survivors would be predicted.

is rejected significantly ($P = 0.03$). An excellent fit to the Nagasaki incidence rate is made by the fitted dose squared relationship of 0.003 induced leuk. per year/10^6 person rem^2, starting at a natural incidence rate of 52 leuk. per yr/10^6 persons, and assuming an average neutron potency factor of 9. This dose squared relationship will be used to provide alternative estimates of risk.

Now, the lifetime risks will be estimated for leukemia induced by total body γ-ray irradiation at high dose-rates (10-1000 rem/min) such as received by the A-bomb survivors. Assuming the average death rate from induced

leukemia was the same in the unobserved interval 0 to 5 years after irradiation as in the observed 5 to 25 yr interval, the total incidence during the first 25 years following irradiation based on the preferred linear model would be (25 yr)(0.8 leuk per yr/10^6 person rem) = 20 leuk/10^6 person rem. Based on present trends (see Table 3 and Fig. 2.) about 80% of the lifetime leukemia risk should be expressed at 25 years. Therefore, the preferred linear estimate for the lifetime risk from leukemia is (20 leuk./10^6 person rem)/0.8 = 25 leuk./10^6 person rem. The higher and lower linear estimates and the dose squared estimate were calculated similarly, and are shown in Table 4.

Table 4. LIFETIME RISK FROM LEUKEMIA AT HIGH DOSE-RATES
(From a total body γ-ray dose "D")

Higher linear estimate	= (40 leuk./10^6 person rem) D
Preferred linear estimate	= (25 leuk./10^6 person rem) D
Lower linear estimate	= (14 leuk./10^6 person rem) D
Dose squared estimate	= (0.1 leuk./10^6 person rem^2) D^2

Our linear estimates compare favorably with linear estimates derived from other sources. When we made a similar analysis on the excess leukemias in British patients given x-ray therapy for the treatment of ankylosing spondylitis,[5,15] we obtained 21 leukemias/10^6 person rem averaged over the total marrow. (The average dose to the spinal marrow was taken as 880 rads,[4] and since about 40% of the active bone marrow was irradiated, the mean dose to the total

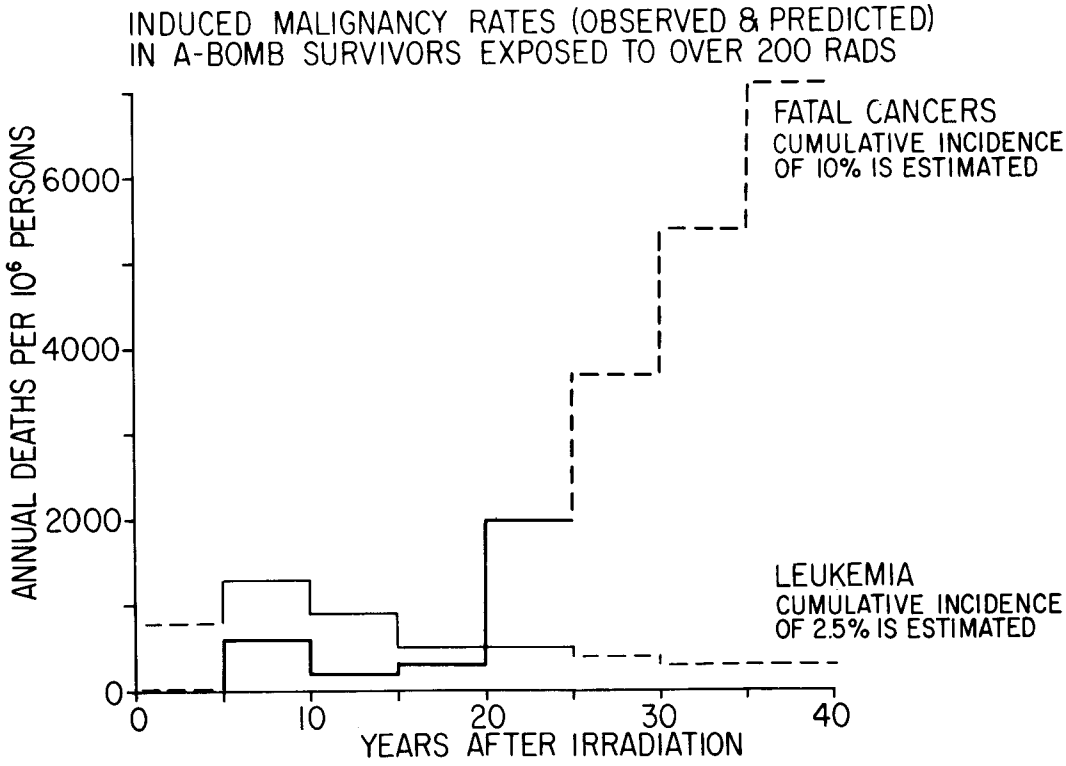

Fig. 2. Death rates from induced malignancy in the A-bomb survivors exposed to 200 rads and over (observed rates are shown as solid lines; predicted, as dashed lines). The rates from induced leukemia are decreasing, while those from induced cancer are increasing. See Table 3 for details.

marrow was taken as 350 rads). The BEIR Report[1] estimate (p. 169) of 516-738 leukemias/yr in 200,000,000 persons receiving 0.1 rem/yr corresponds to 26-37 leuk./10^6 person rem. Dolphin and Marley[4] estimate a lifetime risk of 20 leuk./10^6 person rem. Our preferred linear estimate of 25 leuk./10^6 person rem seems a reasonable estimate (presuming linearity) for the induction of leukemia at high dose-rates, and will be used in deriving linear risk estimates for cancer induction.

CANCER RISK AT HIGH DOSE-RATE

The lifetime risk from fatal cancers (fatal malignant neoplasms excluding leukemia) induced at high dose-rates will now be estimated as the projected ratio of fatal cancers/fatal leukemias, multiplied by the lifetime risk from leukemia. The combined high dosage groups at Hiroshima and Nagasaki exposed to 200 rads and over are of special interest because their incidences of leukemia and cancer are clearly elevated above normal values.[7] As detailed in Table 3 and Fig. 2, about 2.5% of the people in this highly exposed group are expected to die of radiation-induced leukemia, whereas if the mortality rate from induced cancer continues to increase at the present trend, a cumulative total of about 10% of these highly exposed persons are predicted to die of radiation-induced cancer. Assuming 4 induced cancer deaths per induced leukemia, the preferred linear estimate of the lifetime risk from fatal cancer becomes (4 cancers/leuk.)(25 leuk./10^6 person rem) = 100 fatal cancers/10^6 person rem.

However, it is uncertain whether or not the death rate from induced cancers will continue upward exactly according to the present trend. If it increases at twice the present trend, a cumulative induction of about 6 fatal cancers/leukemia is projected, for which the lifetime risk would be (6 fatal cancers/leuk.)(25 leuk./10^6 person rem) = 150 fatal cancers/10^6 person rem, which tentatively we regard as a plausible higher linear estimate. On the other hand, if the death rate from induced cancer will plateau at its 1965-1970 level, a cumulative induction of about 2 fatal cancers/leukemia is indicated, for which the corresponding lifetime risk would be (2 fatal cancers/leuk.)(25 leuk./10^6 person rem) = 50 fatal cancers/10^6 person rem, which we regard as a provisional lower linear estimate. There is much uncertainty in these projections since they involve not only the applicability of the models used, but what fraction of the future cancers will become fatal, considering new advancements in medical treatment. The next follow-up should be of exceptional interest.

For our dose squared model, the lifetime cancer risk is taken as (4 fatal cancers/leuk.)(0.1 leuk./10^6 person rem^2) = (0.4 fatal cancers/10^6 person rem^2).

Risk estimates from fatal cancers at high dose-rate are shown in Table 5.

Table 5. LIFETIME RISK FROM FATAL CANCERS AT HIGH DOSE-RATES
(From a total body γ-ray dose "D")

Higher linear estimate	= (150 fatal cancers/10^6 person rem) D
Preferred linear estimate	= (100 fatal cancers/10^6 person rem) D
Lower linear estimate	= (50 fatal cancers/10^6 person rem) D
Dose squared estimate	= (0.4 fatal cancers/10^6 person rem^2) D^2

Corresponding linear estimates converted from pp. 168-169 of the BEIR Report[1] range from 60 to 420 with a best estimate of about 140 fatal cancers/10^6 person rem, excluding leukemia. Dolphin and Marley[4] estimate 80 fatal cancers/10^6 person rem. Unfortunately, the existing cancer results from ankylosing spondylitic patients receiving partial body x-ray therapy directed at selected

regions of the skeleton are of limited usefulness in deriving numerical estimates of cancer risk because of uncertainties in soft-tissue doses, and because of the possibility that spondylitic disease may enhance the incidence of certain forms of cancer.[5,15] The risk from induced cancer is more uncertain than that from induced leukemia.

CANCER AND LEUKEMIA RISK AT LOW DOSE-RATES

γ-rays, x-rays, and β-particles interact with matter so as to transfer nearly all of their energy to electrons. These moving electrons produce a relatively sparse distribution of ionizations and excitations along their paths, and therefore, γ-rays, x-rays, and β-particles are known as radiations of low LET (linear energy transfer). The cell culture work of Elkind and Sutton[16] showed quite conclusively that considerable repair of the damage from low LET radiation was possible if sufficient time was allowed between successive irradiations. The implication from the "Elkind effect" is that in general, the residual damage from a given dose of low LET radiation should decrease as the dose-rate is lowered, due to increased available time for biological repair between successive local radiation events.

Table 6 presents ten comparisons of the dose-rate effectiveness factor (dose at high dose-rate/dose at low dose-rate for equal biological effect) for life shortening and the induction of neoplasms by low LET radiations.[17] The

Table 6. $\left\{\frac{\text{Effectiveness at LOWER dose-rate}}{\text{Effectiveness at HIGHER dose-rate}}\right\}$

Life shortening in beagles, Andersen,[18] Casarett[19] (0.006-0.06 R/min vs. 8 R/min)	0.08
Life shortening in RF male mice, Upton[20] (0.004-0.06 rad/min vs. 80 rad/min)	0.07
Life shortening in RF female mice, Upton[20] (0.0004-0.07 rad/min vs. 7 rad/min)	0.45
Leukemia in RF male mice, Upton[20] (0.004-0.06 rad/min vs. 80 rad/min)	0.14
Leukemia in RF female mice, Upton[20] (0.0004-0.07 rad/min vs. 7 rad/min)	0.26
Leukemia in CBA and C57 Bl mice, R. H. Mole[21] (0.02 rad/min vs. 1.35 rad/min)	0.15
Leukemia in LAF_1 female mice, Grahn[22] (0.01-0.06 R/min vs. 2-20 R/min)	0.2
Bone sarcomas in CF1 female mice, Finkel[23,24] (0.0001-0.01 rad/min vs. 0.02-0.09 rad/min)	0.05
Mammary tumors in S.D. female rats, Shellabarger[25] (0.03 R/min vs. 10 R/min)	0.68
Thyroid tumors in Lister and Long-Evans rats, Doniach[26] (∿ 1 rad/min vs. 150 rad/min)	0.1
Normal mean ± standard deviation	0.22 ± 0.20
Log-normal mean ± std. deviation	$0.16 \left\{\begin{matrix}+ 0.20 \\ - 0.09\end{matrix}\right.$

levels of effect ranged from slight to severe. Usually the low dose-rates were below 0.1 rad/min, and usually the high dose-rates were above 1 rad/min. Assuming a normal distribution of effectiveness ratios, the mean ± std. dev. was 0.22 ± 0.20. Assuming a log-normal distribution, the corresponding mean was 0.16 with std. deviations of + 0.20 and - 0.09.

Tentatively, a preferred estimate for the overall effectiveness of low vs. high dose-rates from sparsely-ionizing radiation is taken as 0.2 for the summed impact of delayed somatic effects in humans, with somewhat arbitrary bounds of 0.1 to 0.5 for this overall effectiveness factor. The individual effectiveness factors will of course vary with biological endpoint and species (for example, 0.05 for bone sarcomas in CF1 mice vs. 0.68 for mammary tumors in Sprague-Dawley rats), and may vary with the incidence level at which comparison is made.

The estimated risk coefficient at low dose-rates is taken as that at high dose-rates multiplied by the indicated effectiveness factors shown in Table 7.

Table 7. POPULATION RISK FROM SPARSELY IONIZING RADIATION
(Deaths per 1,000,000 persons receiving 1 rem)*

	at HIGH dose-rate (over 10 rem/min)	Eff. Factor	at LOW dose-rate (under 0.01 rem/min)
LEUKEMIA			
Higher linear estimate	40	0.5	20
Preferred linear estimate	25	0.2	5
Lower linear estimate	14	0.1	1
Dose squared estimate	0.1	0.2	0.004**
CANCER (Exc. Leuk.)			
Higher linear estimate	150	0.5	75
Preferred linear estimate	100	0.2	20
Lower linear estimate	50	0.1	5
Dose squared estimate	0.4	0.2	0.016**

* At extremely low doses from γ-rays, multiple ionizations within microscopic volumes of tissue are infrequent. Therefore, as the dose approaches zero, the effectiveness at high dose-rate should approach that at low dose-rate.

** In the dose squared estimates, the doses are squared. Therefore, to convert a dose squared risk at high dose-rate to a dose squared risk at low dose-rate, the dose-rate effectiveness factor (which is a ratio of doses) must also be squared. It is uncertain which dose-rate effectiveness factor is most appropriate for the dose squared model. Tentatively, the average factor of 0.2 obtained from linear intercomparisons[17] has been used.

The following example illustrates the numerical calculation of risk. From Table 7, the predicted number of leukemias plus fatal cancers induced in a population of 10^6 persons receiving 10 rem of γ-irradiation to the total body at low dose-rate would be:

Preferred linear estimate

$$\left[\frac{5 \text{ leuk.} + 20 \text{ fatal cancer}}{10^6 \text{ person rem}}\right] [10^6 \text{ persons}][10 \text{ rem}] = 250 \text{ cases}$$

Dose squared estimate

$$\left[\frac{0.004 \text{ leuk.} + 0.016 \text{ fatal cancer}}{10^6 \text{ person rem}^2}\right] [10^6 \text{ persons}][10 \text{ rem}]^2 = 2 \text{ cases}$$

DISCUSSION

For purposes of radiation protection, the use of the preferred linear estimates is recommended. For radiobiological predictions of actual effects, it might be desirable to give some consideration to the possibility that the dose-response relationship may be curvilinear.

For the purpose of applying these risk estimates to humans, we somewhat arbitrarily consider a "low" dose-rate to be below 0.01 rem/min and a "high" dose-rate to be above 10 rem/min. Thus, γ-ray exposures from background radiation, from properly operating nuclear reactors, and from most routine occupational situations can be regarded as occurring at low dose-rates; whereas high dose-rates typically apply to the A-bomb survivors, to patients exposed to medical x-rays, and to persons acutely exposed in radiation accidents. With more data it may be possible to estimate the dose-rate effectiveness in the "gap" between 0.01 and 10 rem/min.

These risk estimates apply to the "average" person in a general population of mixed ages, and may require modification to be applied to special groups, such as fetuses or patients with diseases, such as polycythemia vera, which can alter the susceptibility to radiation-induced malignancy.[3,11,27] The risk to populations in other parts of the world may differ somewhat from that to the A-bomb survivors. As a future refinement, it may be desirable to analyze the mortality and survival times of each age group separately rather than assuming an average post-irradiation survival time of 40 years for the total exposed population.

The lifespan risk estimates given for low dose-rates apply to uniform total body irradiation from γ-rays, x-rays, β-particles, but not for radiations of high LET, such as neutrons and α-particles, since at least under some conditions the effectiveness of high LET radiation increases as the dose-rate is lowered.[28,29]

In the future, a better understanding of the dose-response relationships and the dose-rate effect is expected which should permit more reliable estimates of the actual risks from radiation. Until then, it is hoped that the risk estimates in this report may provide interim guidance.

DEDICATION

This article is dedicated to the memory of John C. Bugher, M.D., at whose urging this analysis was undertaken.

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