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THE EFFECTS OF LOW LEVELS OF RADIATION ON HUMANS

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*Operated by Union Carbide Corporation under contract W-7405-eng-26 with the U. S. Department of Energy

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Humankind has been exposed to radiation from natural sources throughout the ages. The major source of human exposures is, of course, the natural radiation environment. Although the world average dose-equivalent rate is 125 mrem/year, there are areas with levels ten times this [1]. Figure 1 shows some of these areas of high natural background radiation and the populations living there; the populations range from tens of thousands to hundreds of thousands and are obvious groups in which to look for adverse health effects, both genetic and somatic. Though these levels are significantly higher than are allowable for the population from human-made sources, they are less than the maximum allowable levels for occupational exposure. There have been numerous epidemiological studies of these high background populations but no adverse effects have been found. Because people have lived in these areas for centuries without discernable effects, it appears reasonable to assume that these levels are "safe" and that if the effects are not zero, that the data do not exclude the possibility that the effects are zero. It is equally clear, from data discussed below, that if the lifetime natural exposure was received all at one time, especially in areas where it would total more than 50 rem, that some effects might be detectable. Therefore, the dose range of interest here is that between one rem per year and 50 rem per year. In order to assess the probability of effects in this range, we must first analyze what is known at higher doses and dose rates because there is so little data available in the "low but not negligible" range of less than 50 rem.

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One of the first large groups of exposed humans to be studied extensively was those persons who worked with radium during the first forty years of the Twentieth Century. Evans [2] reported on luminous dial painters (radium) and radium chemists. These workers ingested large quantities of radium, the chemists over a long period, sometimes decades, and the dial painters acquired large quantities generally in a shorter time. As radium is deposited primarily in bone, the relationship of radium dose and bone cancer is of great interest. Figure 2, from Evans, shows a summary graph of the incidence of bone cancer as it relates to radium dose to bone. The shaded region corresponds to the mean occurrence, 28±6%, between 1000 and 50,000 rads. The effect of absorbed doses to bone of 1000 rads or more is pronounced, the mean value for cancer incidence being about 30%. However, controversy has centered on the dose range below 1000 rads wherein Evans found no cancers, and concluded that, for radium alpha rays in bone, there is a dose threshhold of about 1000 rads.

Later, in the 1950's, there were reports of increased leukemia incidence in persons treated with x-rays for ankylosing spondylitis [3,4]. Extensive studies were conducted in the United Kingdom of both the estimated exposures and the increased incidence of leukemia. In this group of exposed persons, there was clearly an increase in leukemia, but the doses were high, totalling up to 2,000 rads or more, delivered in several exposure sessions, but to only part of the body, chiefly the torso. Obtaining the average dose to the red bone marrow was difficult and subject to significant errors. Consequently, the usefulness of the data in extrapolating to the occupational exposure range is limited rather severely with the usual dose averaging technique. Another approach will be used below.

By far the most important source of data on humans is the group of people who survived exposure to the nuclear bombings of Hiroshima and Nagasaki. Because the range of absorbed doses ranged from zero to hundreds of rads, because the Hiroshima bomb yielded significant doses of fast neutrons while the Nagasaki bomb did not, and because there has been long term medical studies made in both cities, the survivors represent a unique population from which to learn a great deal about the effects of acute exposure to ionizing radiation.

Figure 3 shows our estimates of the radiation fields as a function of distance from ground-zero, i.e., the point on the ground above which the bombs exploded. There are four curves, one for neutrons and one for gamma rays for each city. Within 2000 meters of ground zero there were 21,000 survivors in Hiroshima and 10,000 in Nagasaki. In Figure 4, the incidence of leukemia in Nagasaki is given as a function of the free-field tissue kerma in air. Nagasaki is used because of its simplicity, the neutron component of dose being inconsequential. This curve has little scientific significance except for two points: (1) because it is a semi-logarithmic plot, it indicates the futility of attempting to extrapolate to the low dose range, and (2) it shows clearly that the highest levels of leukemia that can be caused by gamma rays is about one percent, even for doses in the lethal range.

To get a better understanding of the radiobiology, the doses to specific organs of the body must be used. In the case of leukemia, the red bone marrow is the organ of interest. But, before comparing dose and effect let us consider a model from which to work. Figure 5 shows the marrow cell killing as a function of absorbed dose for fission neutrons and gamma rays. The curves were developed by Jones [5] from the model of Katz [6]. Note that the two radiations have greatly different

effectiveness for cell killing, that the gamma curve has a broad shoulder initially, and that the neutrons have little or no shoulder. Note especially, that neither of these curves would approximate a straight line on linear graph paper. With this in mind, consider Figure 6, which shows the relationship of acute leukemia to the fraction of marrow cells killed in the Japanese survivors in both cities. This is a linear relationship, and from the preceding slide, if cell killing is directly proportional to the incidence of leukemia, then certainly absorbed dose cannot be linearly related to leukemia incidence. However, it is more complicated for chronic leukemia, as seen in figure 7. This set of curves shows that for chronic leukemia, though the linear relationship holds between cell killing and incidence rate, the slope of the curve in Hiroshima is steeper. As the only difference in the exposure conditions in the two cities was the presence of neutrons in Hiroshima, the high Linear Energy Transfer (LET) is the most likely cause of the increased slope.

In figure 8, there is a depiction for total malignancies other than leukemia, which resembles the graph for acute leukemia.

Now that there appears to be a model which fits the Japanese survivors of nuclear bombings, let us examine some other human data, using the model. The populations studied are generally much smaller, the doses are less well known, and, consequently, the uncertainties in the data much larger. However, figure 9 shows a surprisingly good fit to the model, well within the range of uncertainties. In particular, the data on the ankylosing spondytitus cases were analyzed as follows: (1) calculate the fraction of the total red marrow within the collimated beam; (2) assume that the dose is so high as to kill essentially all the

marrow cells, and (3) take as the surviving marrow cells the fraction outside the collimated beam. A similar approach was used for other data for which it seemed most appropriate.

Finally, figure 10 shows the incidence of leukemia in mice as a function of marrow cells killed, again using the Katz cell killing model as applied by Jones. This gives some added measure of confidence that studies of effects in rodents may be of assistance in estimating effects on humans.

It is clear that we know more, quantitatively, about the effects of radiation on humans than we know about any other harmful agent. Further, by using the approach by Jones, extrapolations to low doses can be made with some confidence for cancers caused by neutrons and gamma rays. For different dose regimens, such as protracted or fractionated doses, the results are less clear but would almost certainly be less than predicted by the acute exposure model. The U. S. National Council on Radiation Protection and Measurements (NCRP) has published a report by Committee 40 [7] which indicates the Committee's consensus opinion that repair mechanisms for protracted and fractionated doses decrease the effectiveness of the doses by up to 80%. We are left with strong differences of opinion concerning alpha emitters, such as plutonium-239, in bone and lung, but there is sufficient human data, in the case or radium workers, and considerable "negative data" from plutonium workers that it is unlikely that effects could ever exceed those based on the linear dose model generally used to predict health effects.

In summary, though we know more about radiation effects on humans than about effects of any other agent, we need answers to several questions about low level effects. The most important relate to dose thresholds,

or lack of thresholds for several types of cancer and high LET radiations and to the effect of dose protractions and fractionation. However, for acute exposures, such as is usual in accidental exposures, the method of Jones provides a convenient way to estimate risks. Though it is based on a different concept, the effectiveness for neutrons in inducing leukemia and other cancers is not greatly different than the estimates using the linear dose model and for gamma rays, it results in risks which approximate those calculated from the quadratic model, i.e., where the effect varies as the square of the dose.

REFERENCES

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[6] Katz, R., and S. C. Sharma, *Phys. Med. Biol.* 20, No. 3, 410 (1975)

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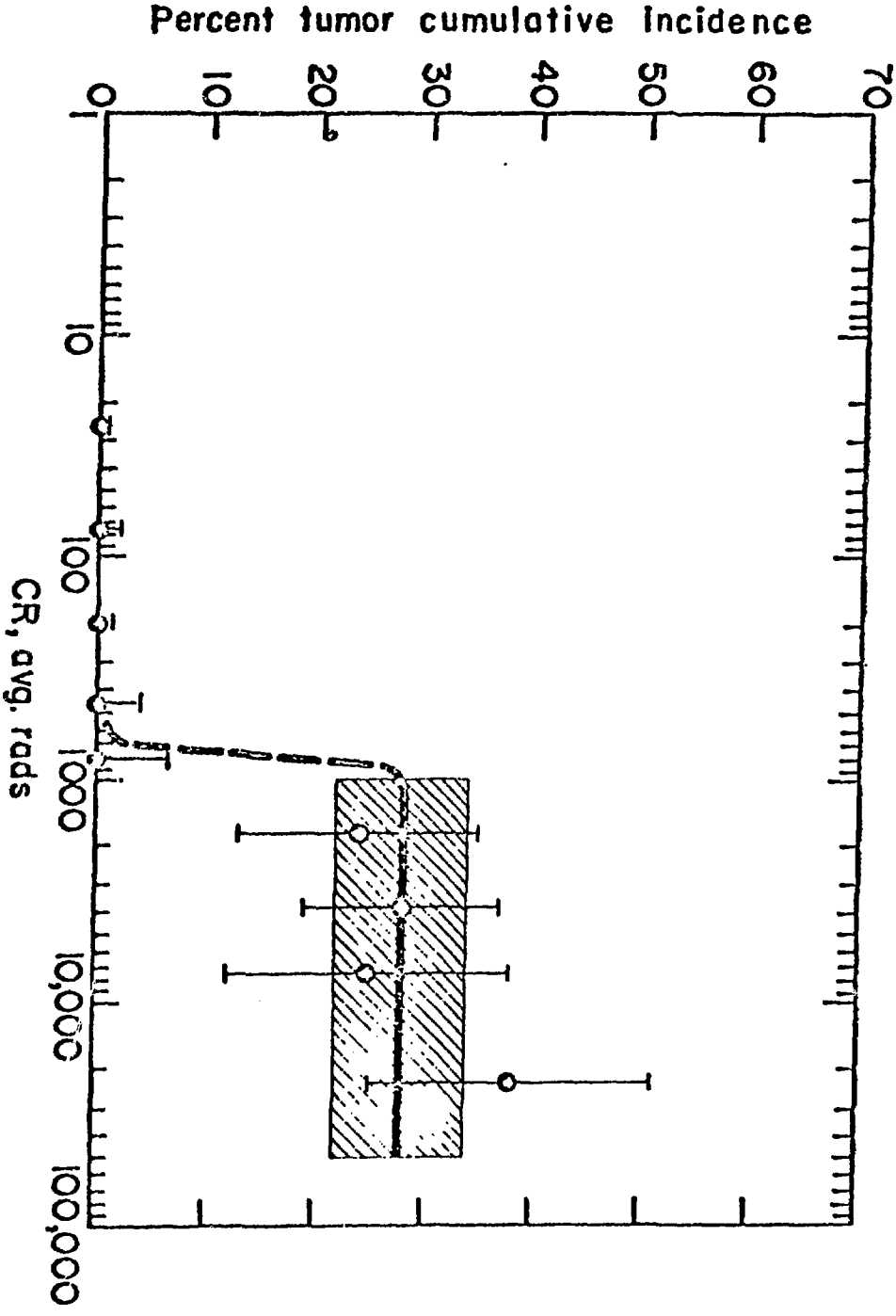
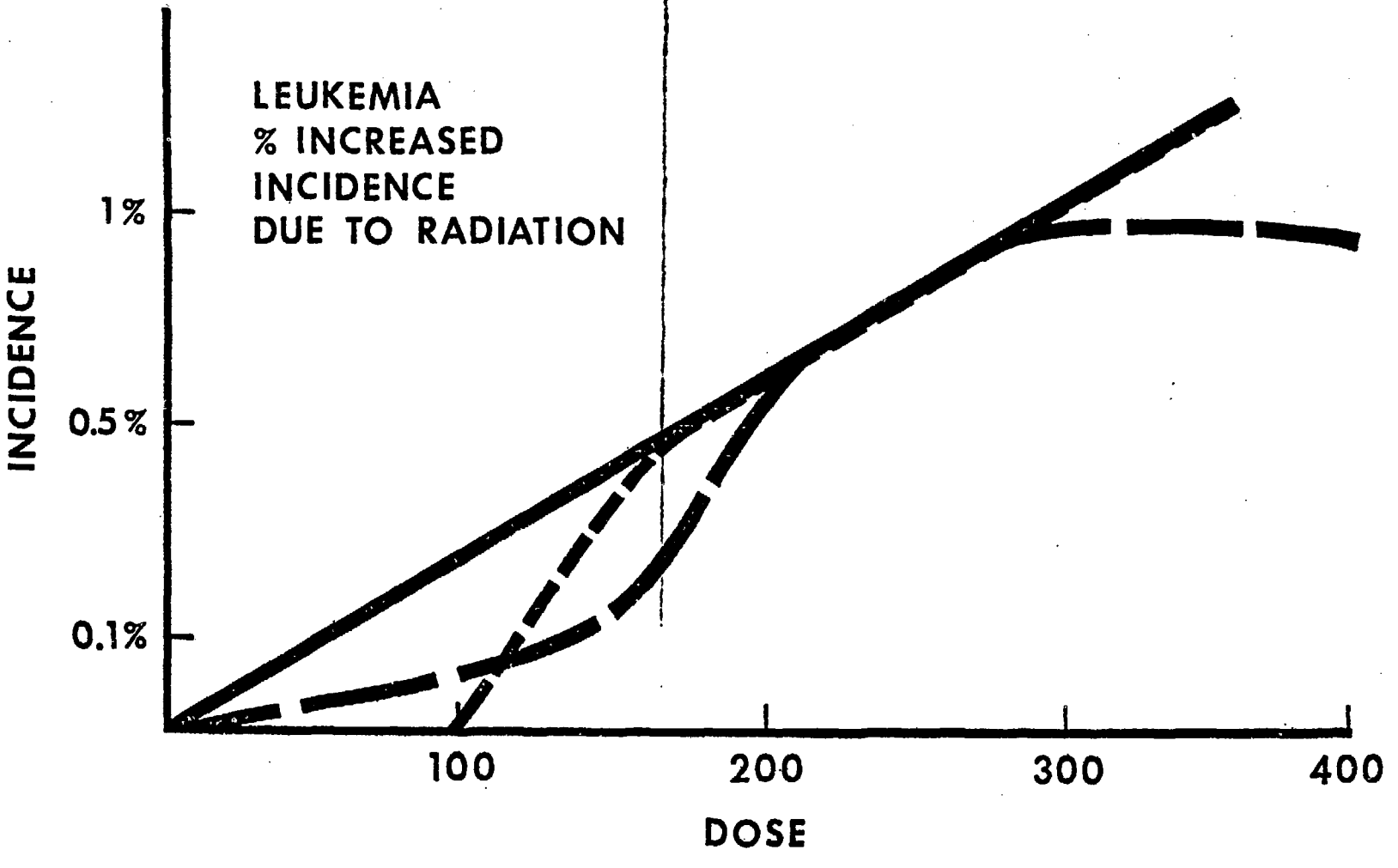
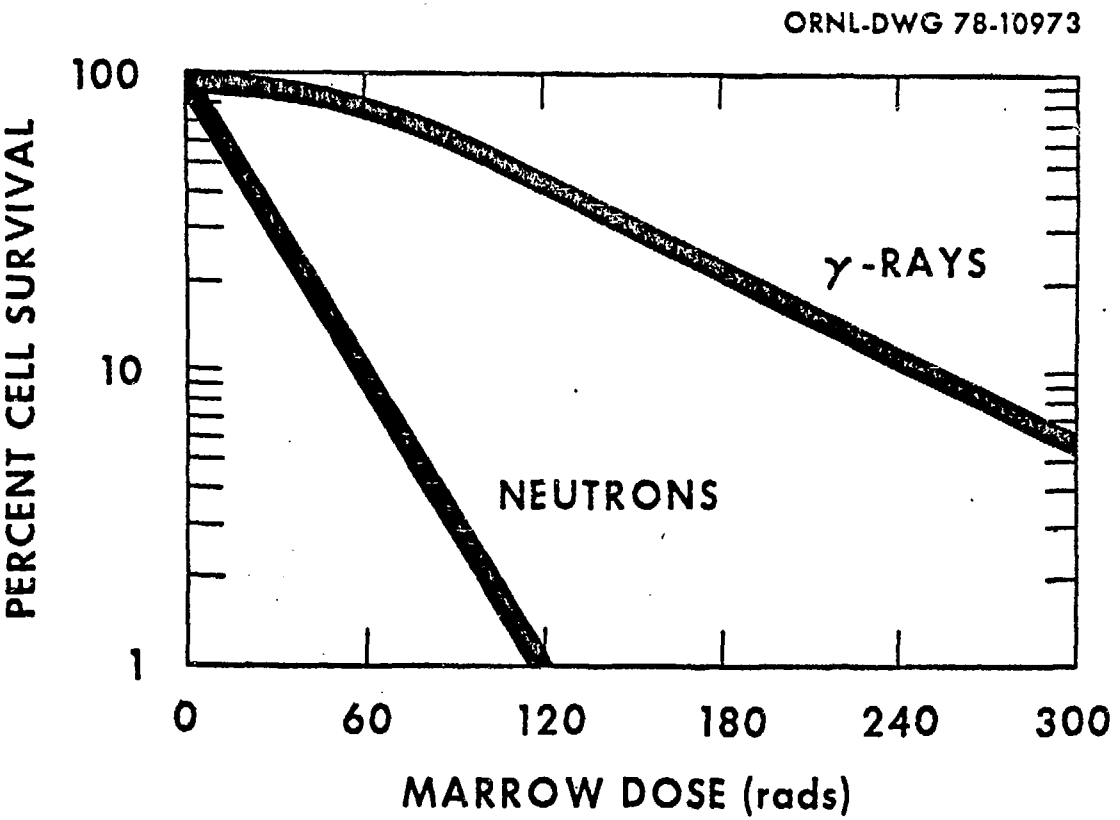
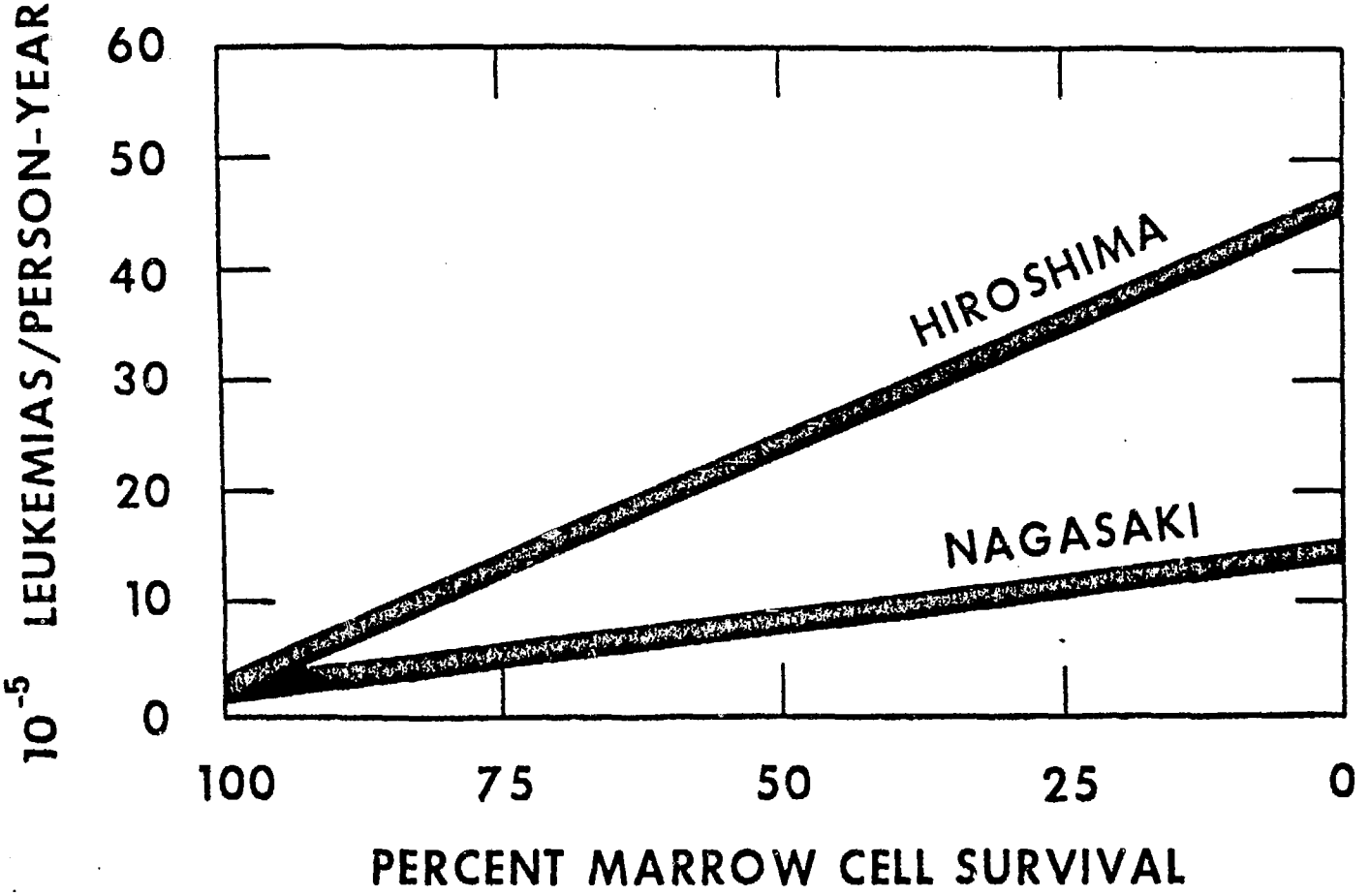

Percent tumor cumulative incidence
0
10
20
30
40
50
60
70
CR, avg. rads
1
10
100
1000
10,000
100,000


LEUKEMIA
% INCREASED
INCIDENCE
DUE TO RADIATION
INCIDENCE
1%
0.5%
0.1%
100
200
300
400
DOSE


ORNL-DWG 78-10973
PERCENT CELL SURVIVAL
100
10
1
γ-RAYS
NEUTRONS
0
60
120
180
240
300
MARROW DOSE (rads)

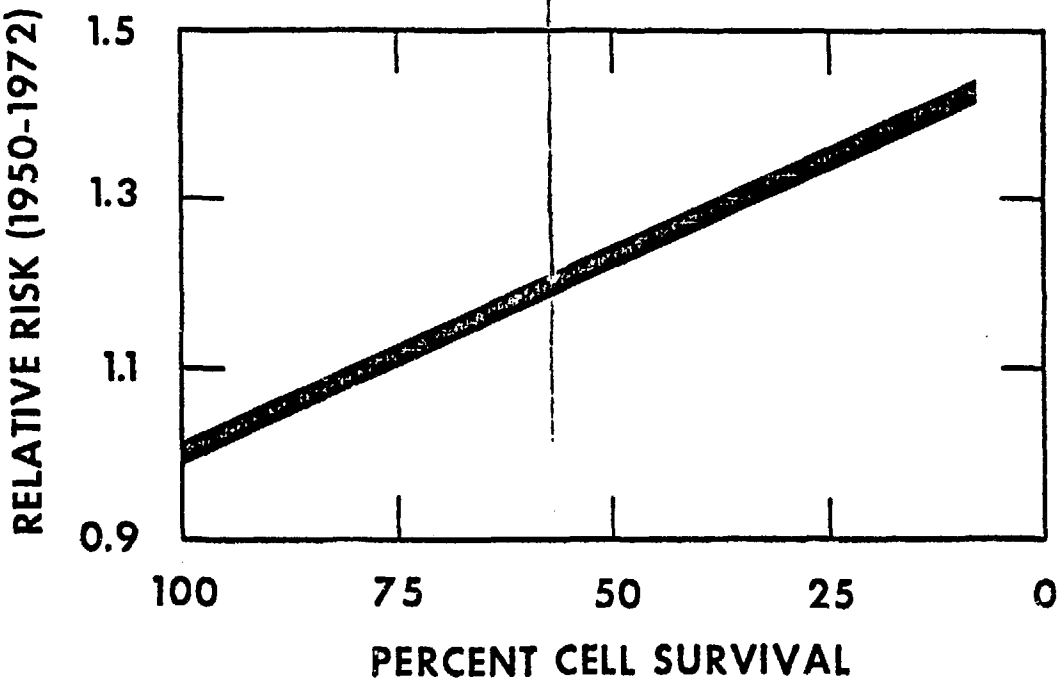
ORNL-DWG 78-10976

INCIDENCE OF CHRONIC LEUKEMIA IN A-BOMB SURVIVORS (BEIR)

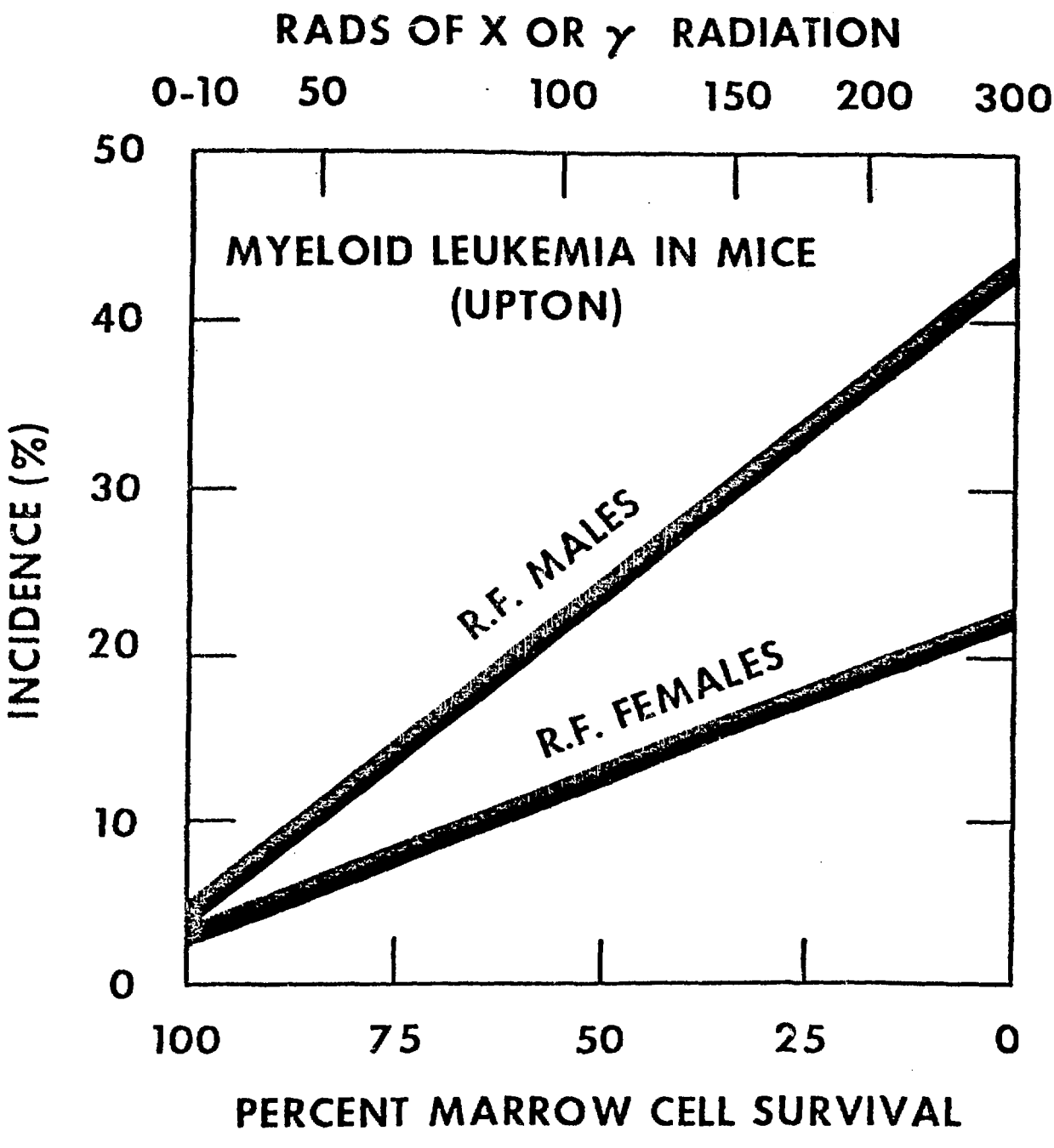


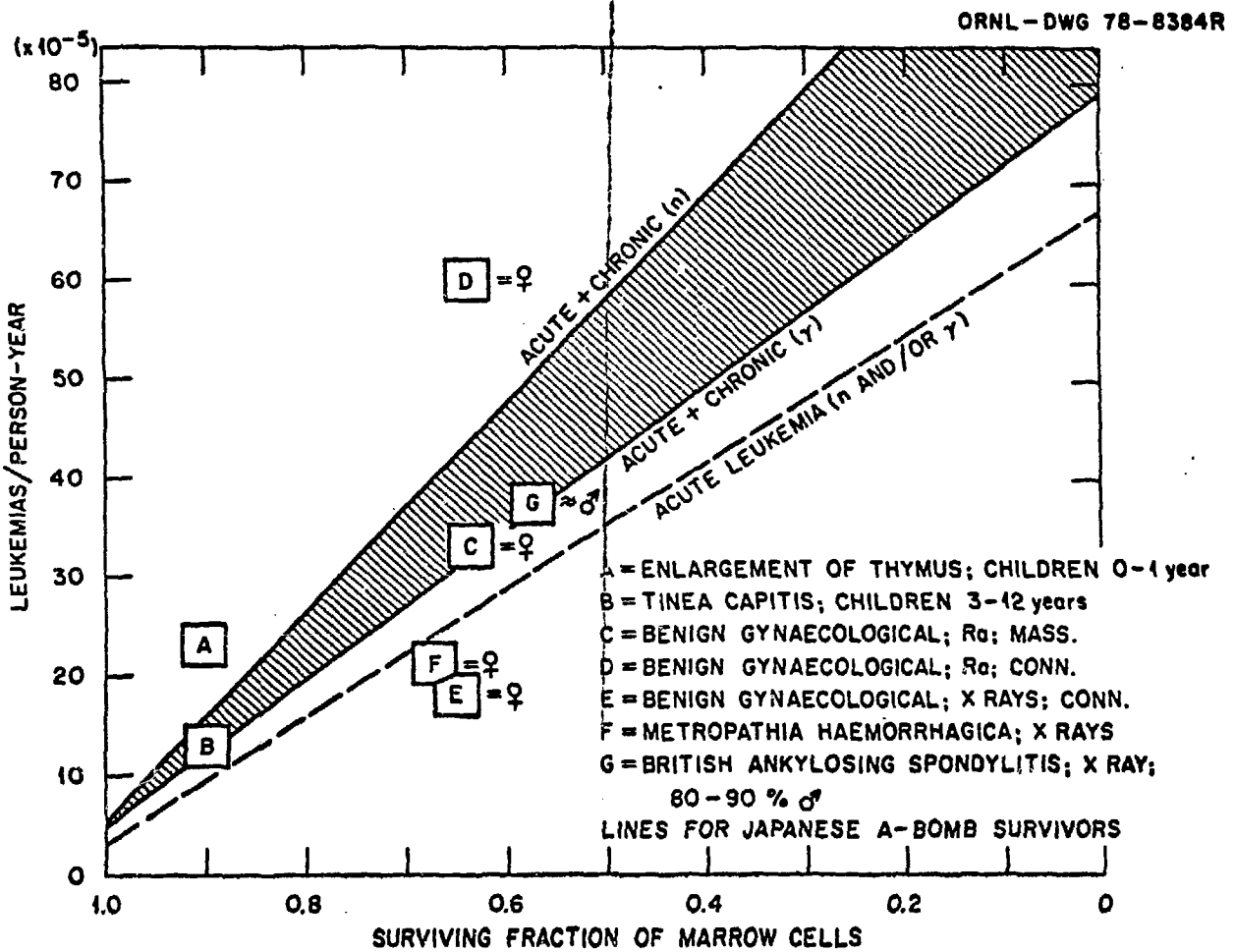
ORNL-DWG 78-10975

FATAL MALIGNANCIES IN HIROSHIMA AND NAGASAKI A-BOMB SURVIVORS (LEUKEMIAS NOT INCLUDED)



ORNL-DWG 78-10977




ORNL-DWG 78-8384R
(x10⁻⁵)
80
70
60
50
40
30
20
10
0
LEUKEMIAS/PERSON-YEAR
1.0
0.8
0.6
0.4
0.2
0
SURVIVING FRACTION OF MARROW CELLS
ACUTE + CHRONIC (n)
ACUTE + CHRONIC (γ)
ACUTE LEUKEMIA (n AND/OR γ)
A
B
C =♀
D =♀
E =♀
F =♀
G ≈♂
A = ENLARGEMENT OF THYMUS; CHILDREN 0-1 year
B = TINEA CAPITIS; CHILDREN 3-12 years
C = BENIGN GYNAECOLOGICAL; Ra; MASS.
D = BENIGN GYNAECOLOGICAL; Ra; CONN.
E = BENIGN GYNAECOLOGICAL; X RAYS; CONN.
F = METROPATHIA HAEMORRHAGICA; X RAYS
G = BRITISH ANKYLOSING SPONDYLITIS; X RAY;
80-90 % ♂
LINES FOR JAPANESE A-BOMB SURVIVORS