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THE BEIR-III REPORT AND THE HEALTH EFFECTS OF LOW-LEVEL RADIATION[1,2]

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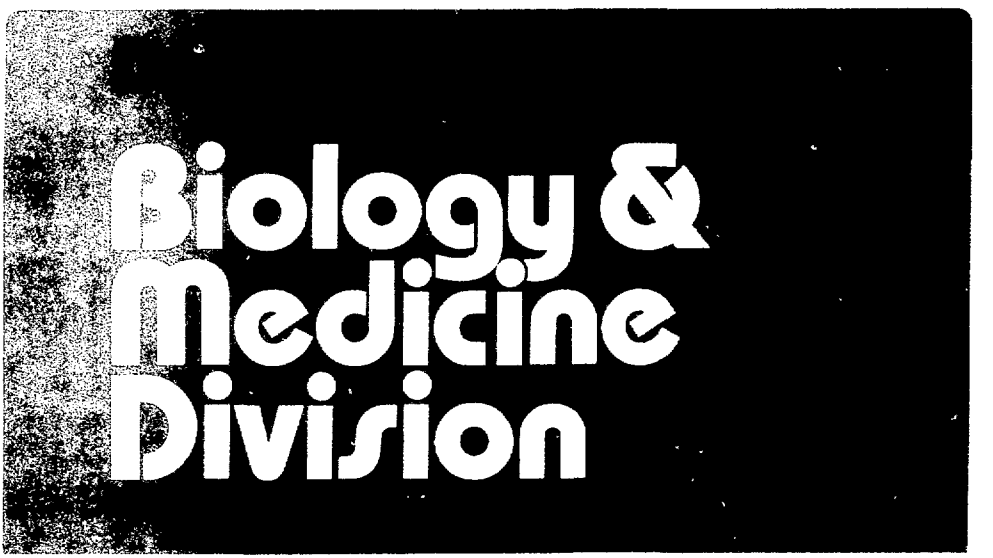

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The BEIR-III Report and the Health Effects of Low-Level Radiation[1,2]

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The BEIR-III Report and the
The Health Effects of Low Level Radiation

Introduction

The question of the health hazards of low-level radiation has become so critical to public acceptance of nuclear energy, it is considered by many to be possibly among the most important scientific issues underlying the nuclear controversy.[1] It was in this setting---not on the probabilities of malfunction in nuclear reactors, but rather on the probabilities and health consequences of a reactor accident with the release of considerable amounts of radioactivity---that Three Mile Island created such extreme concern.[2] The possible fear of radiation-induced cancer or genetically-related ill-health goes deep into the public consciousness. Public acceptance of the risk of radiation is very different from the risk of other noxious substances that are products of our industrialized technological society, particularly agents such as mercury or polychlorinated biphenyls that persist in our environment. But to the public and to the media, it appears that radiation is different; it is mysterious. It cannot be sensed; it cannot be seen, and it can kill you.

During this discussion, I should like to share with you some of the things we know, and some we do not know of the health hazards of low-level radiation, and why the estimates of these hazards continue to be clouded by scientific dispute. I shall use as my setting not the health hazards of the accident at Three Mile Island,[2] but rather the scientific evidence---epidemiological studies and laboratory animal experiments---for estimating numerical risk coefficients for health hazards to human populations exposed to low-level

radiation. I shall try to present, however briefly, the areas of agreement and disagreement among scientists as to the health effects of very low levels of radiation, even levels as low as our natural background. And finally, I shall take as my text not the Report of the President's Commission,[2] or of any Regulatory Commission associated directly or indirectly with nuclear energy, but rather the current Report of the Committee on the Biological Effects of Ionizing Radiation---The BEIR-III Report---of the National Academy of Sciences.[3] Whatever I may consider important in these discussions, I speak only as an individual, and in no way do I speak for the BEIR Committee whose present deliberations are soon to become available. It would be difficult for me not to be somewhat biased and directed in favor of the substance of the BEIR Reports,[3-5] since as an individual, I have been sufficiently close to the ongoing scientific deliberations of agreement and disagreement as they developed over the past 10 years.

What are the Biological Effects of Low-Level Radiation?

My remarks will be restricted primarily to those so-called delayed or late health effects in humans following exposure to low-LET x-rays and to gamma rays from radioactive sources, since these are the ionizing radiations most often encountered in medicine and in nuclear industries. Briefly, low-level ionizing radiation can affect the cells and tissues of the body in three important ways. First, if the macromolecular lesion occurs in one or a few cells, such as those of the hematopoietic tissues, the irradiated cell can occasionally transform into a cancer cell, and after a period of time there is an increased risk of cancer developing in the

exposed individual. This effect is called carcinogenesis. Second, if the developing embryo or fetus is exposed during gestation, injury can occur to the proliferating and differentiating cells and tissues, leading to developmental abnormalities in the newborn. This effect is called teratogenesis. Third, if the injury is in the reproductive cell of the testis or ovary, the hereditary structure or genome of the cell can be altered, and the injury can be expressed in the descendants of the exposed individual. The effect is called mutagenesis or a genetic effect.

There are a number of other possible biological effects of ionizing radiations, such as cataracts of the lens of the eye, or impairment of fertility, but these three important effects---carcinogenic, teratogenic and genetic---are of greatest concern. This is because a considerable amount of scientific information is known from epidemiological studies of exposed human populations and from laboratory animal experiments. Most scientists believe that any exposure to radiation, even possibly at very low levels of dose, carries some risk of such deleterious effects. Furthermore, as the dose of radiation increases above very low levels, the risk of these deleterious effects increases in the exposed populations. It is these latter observations that have been central to public concern about the possible health effects of low-level radiation, and to the task of determining risk estimates for establishing standards for protecting the health of exposed human populations. Scientific reports of almost all expert advisory committees on radiation---the ICRP,[6] the UNSCEAR,[7,8] the BEIR Committee,[3,4] the NCRP[9,10] and others---are in close agreement on the broad and substantive issues of such health effects.

What are the Health Effects of Low-Level Radiation?

Based on (1) careful statistical analyses of epidemiological surveys of exposed human populations, (2) on research in laboratory animals, (3) on dose-response relationships of carcinogenic, teratogenic and genetic effects, and (4) on mechanisms of cell and tissue injury, there are a number of very important conclusions on the health effects of ionizing radiation about which we are quite certain.

1. In regard to radiation-induced cancer, solid cancers arising in the various organs and tissues, such as the female breast and the thyroid gland, rather than leukemia, are the principal late effects in individuals exposed to radiation. The different organs and tissues vary greatly in their relative susceptibility to radiation-induced cancer. The most frequently occurring radiation-induced cancers in man include primarily in decreasing order of susceptibility, the female breast, the thyroid gland, especially in young children and females, the hematopoietic tissues, the lung, certain organs of the gastrointestinal tract, and the bones. There are influences, however, of age at the time of irradiation, of sex, and of the radiation factors and types---LET and RBE---affecting the cancer risk.

2. The effects on growth and development of the embryo and fetus are related to the gestational stage at which the radiation exposure occurs. A threshold level of radiation dose may exist below which gross teratogenic effects are not observed. However, these levels vary greatly depending on the particular developmental abnormality.

3. The paucity of human data from exposed populations has made it necessary to estimate genetic risks based mainly on laboratory mouse experiments. Knowledge of fundamental mechanisms of radiation injury at the

genetic level permits greater assurance for extrapolation from such laboratory experiments to man. Mutagenic effects are related linearly to radiation dose. With new information of the broad spectrum and incidence of serious genetically-related ill-health in man, such as mental retardation and diabetes, the risk of radiation-induced mutations affecting future generations takes on a new and special meaning.

What is Not Known about the Potential Health Effects of Low-Level Radiation?

However, there is still very much that is not known about the potential health hazards of low-level radiation.

1. It is not known what the carcinogenic, teratogenic, and mutagenic health effects are at radiation dose rates as low as a few hundred millirem per year. It is probable that if health effects do occur at such very low levels of radiation, they will be masked by environmental or other factors that produce similar effects.

2. The vast epidemiological data on exposed human populations are still highly uncertain in regard to the forms of the dose-response relationships for radiation-induced cancer, and this is especially the case for low dose levels. Therefore, it has been necessary to estimate human cancer risk at low doses primarily from observations at relatively high doses. To do this, various forms of no-threshold linear-quadratic dose-response relationships are now most frequently used, recognizing the lack of our scientific understanding of fundamental mechanisms of radiation-induced cancer in man. In considering the many forms of the dose-response relationships applied to the epidemiological data, it is not known whether the cancer incidence observed at high dose levels applies also at low levels.

3. As yet, there are no reliable methods for estimating the repair of injured cells and tissues of the body exposed to very low radiation doses and dose rates. And, further, there are no methods of identifying those persons who may be particularly susceptible to radiation injury.

4. From the epidemiological surveys of irradiated populations exposed in the past, there is only limited information on the precise radiation doses absorbed by the tissues and organs of the body. Furthermore, the complete cancer incidence in each population studied still is not known, since new cases of cancer cont'nue to appear with the passing of time. Thus, any estimation of risks to health based on such limited dose-response information must be incomplete until the entire study population has died of natural causes.

5. Finally, little is known of the role of competing environmental and other host factors---biological, chemical or physical factors---existing at the time of radiation exposure, or following exposure, which may affect and influence the carcinogenic, teratogenic, or genetic effects of low-level radiation.

What are the Problems of the Dose-Response Relationships for Radiation-Induced Human Cancer?

The present BEIR-III Committee recognized early that there was great uncertainty in regard to the shapes of the dose-response curves for cancer induction in humans by radiation, and especially at low doses. Estimates of risk at low doses appear to depend more on what is assumed about the mathematical form of the dose-response function than on the epidemiological data themselves. Wherever possible, in estimating the cancer risk from low

doses of low-LET radiation, the BEIR-III Committee has chosen to use a linear-quadratic dose-response model that was felt to be consistent with epidemiological and radiobiological data in preference to more extreme dose-response models. In this regard, the present BEIR-III Report[3] differs substantially from the 1972 BEIR-I Report.[4] I should like to examine this matter more closely.

In recent years, a general hypothesis for estimating the excess cancer risk in irradiated human populations, based on theoretical considerations, extensive experimental animal studies and epidemiological surveys, suggests that complex dose-response relationships exist between radiation dose and cancer incidence.[11,12] Perhaps the most widely accepted model, based on the available information and consistent with both knowledge and theory, takes the complex linear-quadratic form: $I(D) = (\alpha_0 + \alpha_1 D + \alpha_2 D^2)\exp(-\beta_1 D - \beta_2 D^2)$, where I is the cancer incidence in the irradiated population at dose D in rad, and α_0, α_1, α_2, β_1, and β_2 are non-negative constants (Figure 1). This multicomponent curve contains an initial upward-curving linear and quadratic functions of dose which represent the process of cancer induction. This is modified by an exponential function of dose which represents the competing effect of cell-killing at high doses. The dose-response function encompasses all these parameters and is necessarily complex, but certain of the parameters can be theoretically determined. α_0, the control or natural incidence of cancer in the population, is the ordinate intercept at 0 dose of the dose response curve. α_1 is the initial slope at 0 dose, defining the linear component in the low dose range. α_2 is the curvature near 0 dose at the upward-curving quadratic function of dose. β_1 and β_2 are the slopes defining the cell-killing function, that is, the downward-curving function in

the region of high dose.

Review of a large number of the available dose-incidence curves for cancer in irradiated populations has demonstrated that for different radiation-induced cancers, whether in man or in experimental animals, the extent of variation in the shapes of the curves does not permit determination of any of these parameter values with precision, or of assuming their values, or of assuming any fixed relationship between two or more of these parameters. In the case of the available epidemiological data on irradiated populations, this general dose-response mathematical form cannot be universally applied. It has become necessary to simplify the model by reducing the number of parameters or by eliminating those parameters which will have the least effect on the form of the curve in the dose range at low levels of radiation. Such simpler models with increasing complexity are the linear, quadratic, linear-quadratic, and finally, the linear-quadratic form with an exponential modifier due to the effect of cell-killing similar to the general form (Figure 2).

There has been much concern among radiation scientists centering on one particular form of radiation-dose cancer-incidence relationship, generally a linear, no threshold dose-response relationship, that is, where the effect observed was linearly related to dose. There was no reason to assume that the linear form, or any form of dose-response relationship, was the inflexibly correct, or the appropriate function either for cells in tissue culture, or for animals in cages, or for man in his society, to warrant universal application in determining public health policy on radiation protection standards. The lack of our understanding of the fundamental mechanisms of radiation-induced cancer in man, and the recognition that the dose-response information from human data is highly uncertain, particularly at low levels of dose, does

not relieve decision-makers of the responsibility for determining public health policy based on radiation protection standards. The BEIR-III Committee was well aware of the experimental and theoretical considerations which suggest that various and different mathematical forms of dose-response relationships may exist for different radiation-induced cancers in exposed human populations, indeed for different somatic and genetic mutations. It was therefore essential that very precise explanations and qualifications of the assumptions and procedures involved in determining such risk estimates are to be provided, and this has been done explicitly in the present BEIR-III Committee Report containing the estimates of excess cancer risk. The Committee recognized that some experimental and human data, as well as theoretical considerations, suggest that, for exposure to low-LET radiation at low doses, the linear model probably leads to overestimates of the risk of most radiation-induced cancers, but can be used to define upper limits of risk. Similarly, the Committee believes that the quadratic model may be used to define the lower limits of risk from such radiation. For exposure to high-LET radiation, linear risk estimates for low doses are less likely to overestimate risk and may, in fact, underestimate risk.

What is the Controversy over Low-Level Radiation?

The estimate of the cancer hazard of low-level radiation is said to be clouded by scientific dispute. In particular, there appears to be strong disagreement among some scientists as to the effects of very low levels of radiation, even levels as low as our natural radiation background. Most scientists would generally agree that low-level radiation is that which falls within the dose range considered permissible for occupational exposure.

There is, at present, only one set of standards for radiation exposure accepted throughout the world.[13] According to these standards, 5 rem to the whole body per individual radiation worker per year would be the allowable upper limit of low-level radiation. In this context, most of the estimated delayed cancer deaths which may be associated with a so-called hypothetical nuclear reactor accident are therefore considered by some scientists to be caused by exposures well below the occupational limits. If it is assumed that any extra radiation above natural background, however small, causes additional cancer, then if millions of people are exposed, some extra cancers will result. Other scientists strongly dispute this, and firmly believe that low-level radiation is nowhere near as dangerous as their adversarial colleagues would insist. Unfortunately, since the health effects, if any, are so rarely seen because the exposures are so small, the issue may never be resolved---it may be beyond the ability of science and mathematics to decipher. However, there is one standard---natural background radiation---with which to compare additional radiation exposure. At Three Mile Island, for example, the total radiation dose to the population was about 1 percent of natural background---a level where no health effects can be seen.

It is just this type of controversy that is at the root of the division within the present BEIR-III Committee. The Committee's most difficult task has been to estimate the carcinogenic risk of low-dose low-LET whole body radiation. As the earlier BEIR-I study[4] in 1972 had done, some members of the present BEIR-III Committee wished to adopt a linear hypothesis of dose-response to estimate the cancer hazard at very low levels of radiation where no human epidemiological data are available. Here, it is assumed that the same proportional risks are present at low levels as at high levels of radiation.

This finding---that even very small doses are carcinogenic---could force the Environmental Protection Agency to adopt stricter health standards to protect people against radiation. Others of the Committee believe this to be an alarmist approach. When there is *no human* epidemiological evidence, these scientists preferred to assume that the risks of causing cancer by radiation are proportionally lower.

Let us look at some of the problems. In its deliberations, the present BEIR-III Committee concluded two important points: (1) It is not yet possible to make precise low-dose estimates for cancer induction by radiation because the level of risk is so low that it cannot be observed directly. (2) There is great uncertainty as to the dose-response function most appropriate for interpolating in the low-dose region. In studies of exposed animal and human populations, the shape of a dose-response relationship at low doses may be practically impossible to ascertain statistically. This is because the population sample sizes required to estimate or test a small absolute cancer excess are extremely large; specifically, the required sample sizes are approximately inversely proportional to the square of the excess. For example, if the excess is truly proportional to dose and if 1,000 exposed and 1,000 control persons are required to test the cancer excess adequately at 100 rads, then about 100,000 in each group are required at 10 rads, and about 10,000,000 in each group are required at one rad. Experimental evidence and theoretical considerations are more likely than empirical data to guide the choice of a dose-response function. In this dilemma, the BEIR-III Committee has chosen to adopt as a working model for low-LET radiation the linear-quadratic dose-response form with an exponential term to account for the frequently observed turndown of the curve in the high-dose region. However, only derivatives of this model, including the linear,

linear-quadratic and pure quadratic, could prove practical.

The cancer risk estimates presented in the 1972 BEIR-I Report[4] for whole-body exposure were derived from average excess risk per rad observed at doses generally of a hundred or more rads. These linear-model estimates have been criticized on the grounds that the increment in cancer risk per rad may well depend on dose and that the true risk at low doses may therefore be lower or higher than the linear model predicts.[10] In animal experiments, it has been shown, often with considerable statistical precision, that the dose-effect curve for radiation-induced cancer can have a variety of shapes. As a general rule, the curve has positive curvature for low-LET radiation, i.e., the slope of the curve increases with increasing dose. However, at high doses, the slope often decreases and may even become negative. Dose-effect curves may also vary with the kind of cancer, with animal species, and with dose rate. On the basis of experimental work and current microdosimetric theory, the present BEIR-III Committee could therefore quite reasonably adopt as the basis for its consideration of dose-response models the linear-quadratic with an exponential term to impart negative slope in the high-dose region (Figure 1).

For the most part, the available human data fail to suggest any specific dose-response model and are not sufficiently reliable to discriminate among a priori models suggested by theoretical and experimental work. However, there are exceptions (Figure 3); for example, cancer of the skin is not observed at low doses,[15] and dose-response relationships observed in the Nagasaki leukemia data appear to have positive curvature.[16]. The incidence of breast cancer seems to be adequately described by a linear dose-response model.[12,14]

Simplification of any linear-quadratic model was required to obtain

statistically stable risk estimates in many cases. It is now well known that some members of the BEIR-III Committee strongly favor the linear model, others the quadratic form. A further modification of the linear-quadratic form can be assumed with the linear and quadratic components to be equivalent at some dose, which is consistent with epidemiological and radiobiological data, and avoids dependence on either of the extreme forms.[11]

What are the Uncertainties in Estimation of the Carcinogenic Risk in Man of Low-Level Radiation?

The quantitative estimation of the carcinogenic risk of low-dose, low-LET radiation is subject to numerous uncertainties. The greatest of these concerns the shape of the dose-response curve. Others include the length of the latent period, the RBE for fast neutrons and alpha radiation relative to gamma and x radiation, the period during which the radiation risk is expressed, the model used in projecting risk beyond the period of observation, the effect of dose rate or dose fractionation, and the influence of differences in the natural incidence of specific types of cancer. In addition, uncertainties are introduced by the biological risk characteristics of humans, e.g., the effect of age at irradiation, the influence of any disease for which the radiation was given therapeutically, and the influence of length of observation or follow-up. The collective influence of these uncertainties is such as to deny great credibility to any estimates of human cancer risk that can be made for low-dose, low-LET radiation. It is for these reasons, the present BEIR-III Committee has placed more emphasis on the methods of risk estimation than on any numerical estimates derived thereby.

What are the Risk Estimates of Radiation-Induced Cancer in Man?

The chief sources of epidemiological data used in the BEIR-III Report[3] are the Japanese populations exposed to whole-body irradiation in Hiroshima and Nagasaki,[16] patients with ankylosing spondylitis[17] and other patients who were exposed to partial-body irradiation therapeutically,[18] or to diagnostic x-rays[19] and various occupationally exposed populations,[20] such as uranium miners and radium-dial painters. Most epidemiological data do not systematically cover the range of low to moderate radiation doses for which the Japanese atomic-bomb survivor data appear to be fairly reliable. Analysis in terms of dose-response therefore rely greatly on the Japanese data. The substantial neutron component of dose in Hiroshima and its correlation with gamma dose limit the value of the more numerous Hiroshima data to the estimation of cancer risk from low-LET radiation. The Nagasaki data, for which the neutron component of dose is small, are less reliable for doses below 100 rads.

For its illustrative computations of the lifetime risk from whole-body exposure, the present BEIR-III Committee chose three radiation exposure situations:

(1) a single exposure of a representative (life-table) population to 10 rads;

(2) a continuous, lifetime exposure of a representative (life-table) population to 1 rad per year;

(3) an exposure to 1 rad per year over several age intervals exemplifying conditions of occupational exposure.

The three exposure situations were not chosen to reflect any circumstances that would normally occur, but embrace the areas of concern---general population and occupational exposure and single and continuous exposure. These were substantially different from the exposure situation chosen for illustrative computation by the 1972 BEIR-I Committee, where 100 mrem per year was selected.

Below these dose levels chosen for the current report, the uncertainties of extrapolation of risk to very low levels were strongly felt by some members of the present Committee to be too great to justify risk estimation. The selected annual exposure, although only one-fifth the maximal permissible dose for occupational exposure, is nevertheless consistent with occupational exposures in the nuclear industry. The U.S. 1969-1971 life-table was used as the basis for the calculations, and all results are expressed in terms of excess cancers per million persons throughout their lifetime after exposure. The expression time was taken as 25 years for leukemia and the remaining years of life for other cancers. Separate estimates were made for cancer mortality and for cancer incidence.

The resulting cancer mortality risk estimates for all forms of cancer differ by as much as an order of magnitude. The uncertainty derives chiefly from the range of dose-response models used, from the alternative absolute and relative projection models, and from the sampling variation in the source data. The lowest estimates are derived from the pure quadratic model; the highest, from the linear model. The linear-quadratic model provides estimates intermediate between these two extremes.

In the absence of any increased radiation exposure, among one million persons of life-table age and sex composition in the United States, about 164,000 persons would be expected to die from cancer, according to present cancer mortality rates. For a situation in which these one million persons are exposed to a single dose increment of 10 rads of low-LET ratiation, the linear-quadratic model predicts increases of about 0.5% and 1.5% over the normal expectation of cancer mortality, according to the projection model.

For continuous lifetime exposure to 1 rad per year, the increase in cancer mortality, according to the linear-quadratic model, ranges from about 5% to 10% over the normal expectation, depending on the projection model.

To compare these estimates with those of the 1972 BEIR-I Report[4] and the 1977 UNSCEAR Report,[8] it was convenient to express them as cancer deaths per million persons per rad of continuous lifetime exposure. For continuous lifetime exposure to 1 rad per year the linear-quadratic dose-response model for low-LET radiation yielded estimates 25% to 50% below the comparable linear estimates in the 1972 BEIR-I Report,[4] depending on the projection model. Although the present BEIR-III Report[3] uses much more scientific information not available for the earlier 1972 report, the differences mainly reflect changes in the assumptions made by the two BEIR Committees almost a decade apart. The present Committee preferred a linear-quadratic, rather than linear, dose-response model for low-LET radiation, and preferred not to assume a fixed relationship between the effects of high-LET and low-LET radiation. The present risk estimates do not, as in the 1972 BEIR-I Report,[4] carry through to the end of life very high relative-risk coefficients obtained with respect to childhood cancers induced <u>in utero</u>

by radiation. The present BEIR-III risk estimates do not differ appreciably from those in the 1977 UNSCEAR Report.[8]

Cancer-incidence risk estimates were less firm than mortality estimates. The present BEIR-III Committee used a variety of dose-response models and several data sources. The dose-response models produced estimates that differed by more than an order of magnitude, whereas the different data sources gave broadly similar results. For the linear-quadratic model and for continuous lifetime exposure to 1 rad per year, for example, the increased risks expressed as percent of the normal incidence of cancer in males were about 2% to 6%, depending on the projection model. Risks for females were substantially higher than those for males, due primarily to the relative importance of radiation-induced thyroid and breast cancer.

Estimates of excess risk for individual organs and tissues depend in large part on partial-body irradiation and use a wider variety of data sources. Except for leukemia and bone cancer, estimates for individual sites of cancer were made only on the basis of the linear model and were stated in terms of excess cancer cases per year per million persons exposed per rad. For leukemia, the linear-quadratic model yielded about 1.0 to 1.4 excess leukemia cases (or deaths) per year per million persons exposed per rad, for females and males, respectively. For solid cancers, linear-model estimates were, for example: for thyroid in males, about 2, and in females, about 6; for female breast, about 6; and for lung, about 3.5 to 4. These risk coefficients derive largely from epidemiologic data in which exposure was at high doses, and these values may, in some cases, overestimate risk at low doses.

What Conclusions can be Drawn from the BEIR-III Experience?

The present scientific evidence and the interpretation of available human data can draw very few firm conclusions on which to base scientific public health policy for protection standards for low-level radiation. However, based on the radiation risk estimates derived, any lack of precision does not minimize either the need for setting public health policy standards nor the conclusion that such risks are extremely small when compared with those available from alternative options, and those normally accepted by society as the hazards of everyday life. When compared with the benefits that society has established as goals derived from the necessary activities of energy production and medical care, it is apparent that society must establish appropriate standards and seek appropriate controlling procedures which continue to assure that its needs and services are being met with the lowest possible risks.

I do not believe that the potential health hazard of low-level radiation is central to survival of nuclear energy. I do believe that a substantial part of the nuclear controversy has been mounted on the question of low-level radiation and linked to public acceptance of nuclear energy. In a third of a century of inquiry, embodying among the most extensive and comprehensive scientific efforts on the health effects of an environmental agent, certain practical information necessary for determination of radiation protection standards for public health policy is still lacking, and may remain so. It is now assumed that exposure to radiation at low levels of dose carries some risk of deleterious effects. However, how low this level may be, or the probability, or magnitude of the risk, still are not known. Our best scientific knowledge and our best scientific advice are essential for the

protection of the public health, for the effective application of new technologies in medicine and industry, and for guidance in the production of nuclear energy. Man cannot dispense with those activities which inevitably involve exposure to low levels of ionizing radiation in medicine, where he readily recognizes some degree of risk to health, however small, exists. In the evaluation of such risks from radiation in nuclear energy, as is done in medicine, it is also necessary to limit the *radiation exposure to a level at which the risk is acceptable both to the* individual and to society.

The present BEIR-III Committee has not highlighted any controversy over the health effects of low-level radiation. In its evaluation of the experimental data and epidemiological surveys, the Committee has carefully reviewed and assessed the value of all the available scientific evidence for estimating numerical risk coefficients for the health hazards to human populations exposed to low levels of ionizing radiation. Such devices require scientific judgment and assumptions based on the available data only, and has led to disagreement not only outside the committee room, but among committee members, as well. But such disagreement centers not on the scientific facts or the epidemiological data, but rather on the assumptions and interpretations of the available facts and data.

Responsible public awareness of the possible health effects of ionizing radiations from medical and industrial radiation exposure, and from the production of nuclear energy has called for expert advice and guidance. And, advisory committees on radiation of national and international composition have for many years met and served faithfully and effectively to report on three important matters of societal concern: (1) to place into

perspective the extent of harm to the health of man and his descendants to be expected in the present and in the future from those societal activities involving ionizing radiation; (2) to develop quantitative indices of harm based on dose-effect relationships; such indices could then be used with prudent caution to introduce concepts of the regulation of population doses on the basis of somatic and genetic risks; and (3) to identify the magnitude and extent of radiation activities which could cause harm, to assess their relative significance, and to provide a framework for recommendations on how to reduce unnecessary radiation exposure to human populations. To a greater or lesser extent, each advisory committee on radiation---such as the UNSCEAR, the ICRP, the NCRP, and the BEIR---deal with these matters, but the reports of these various bodies are expected to differ because of the charge, the scope, and the composition of the committee, and public attitudes existing at the time of the deliberations of that committee, and at the time of the writing of that particular report. The main difference of the BEIR Committee Report[3] is not so much from new data or new interpretations of existing data, but rather from a philosophical approach and appraisal of existing and future radiation protection resulting from an atmosphere of constantly changing societal conditions and public attitudes.

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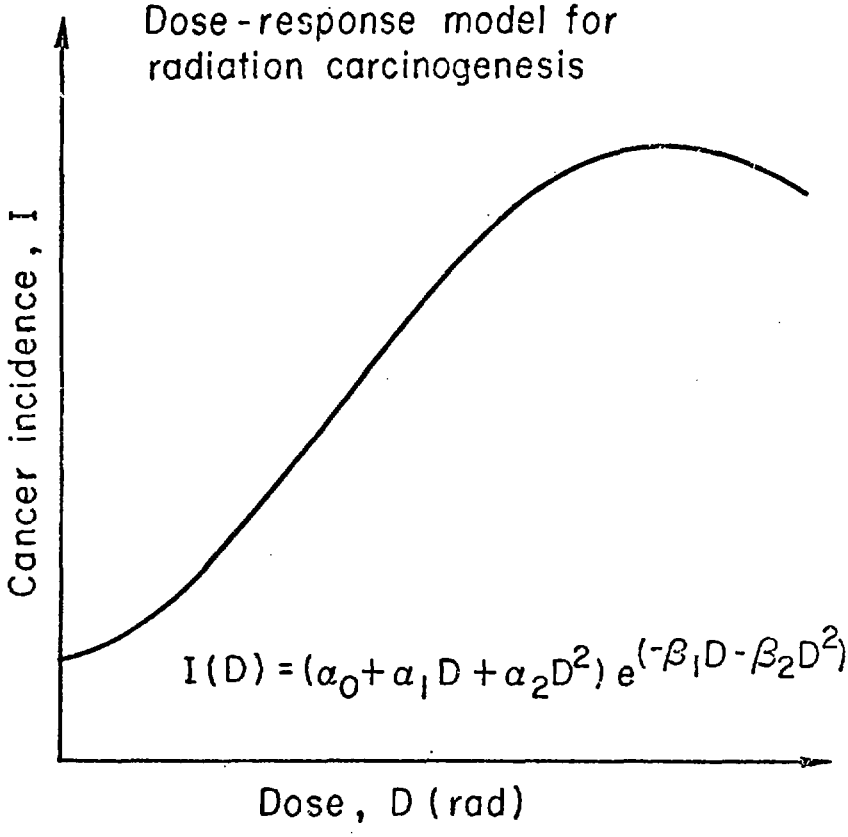
Dose-response model for
radiation carcinogenesis
Cancer incidence, I
$I(D) = (\alpha_0 + \alpha_1 D + \alpha_2 D^2)\, e^{(-\beta_1 D - \beta_2 D^2)}$
Dose, D (rad)
XBL791-3029

Figure 1

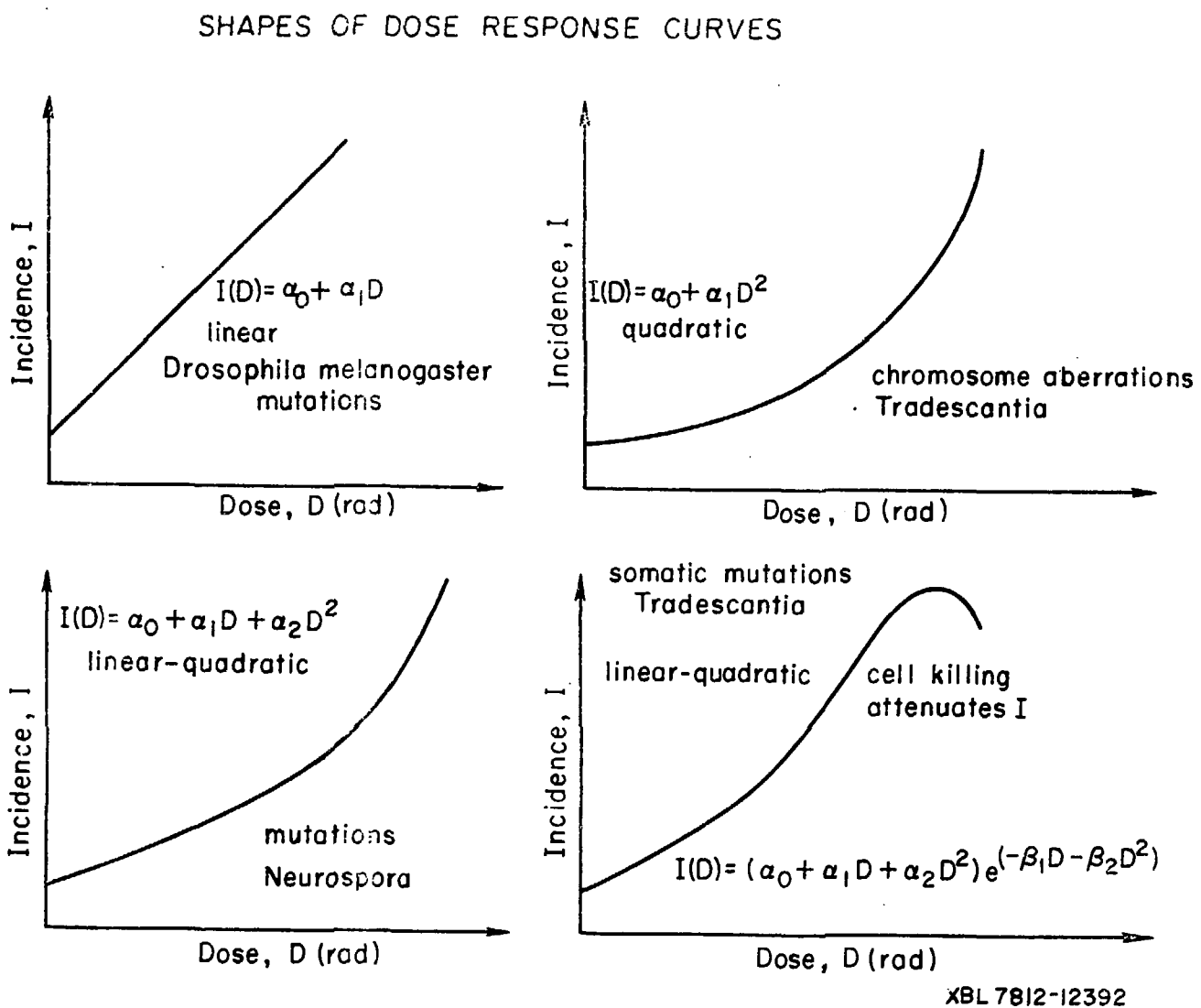
SHAPES OF DOSE RESPONSE CURVES
Incidence, I
$I(D) = \alpha_0 + \alpha_1 D$
linear
Drosophila melanogaster
mutations
Dose, D (rad)
Incidence, I
$I(D) = \alpha_0 + \alpha_1 D^2$
quadratic
chromosome aberrations
Tradescantia
Dose, D (rad)
Incidence, I
$I(D) = \alpha_0 + \alpha_1 D + \alpha_2 D^2$
linear-quadratic
mutations
Neurospora
Dose, D (rad)
somatic mutations
Tradescantia
linear-quadratic
cell killing
attenuates I
Incidence, I
$I(D) = (\alpha_0 + \alpha_1 D + \alpha_2 D^2) e^{(-\beta_1 D - \beta_2 D^2)}$
Dose, D (rad)
XBL 7812-12392

Figure 2

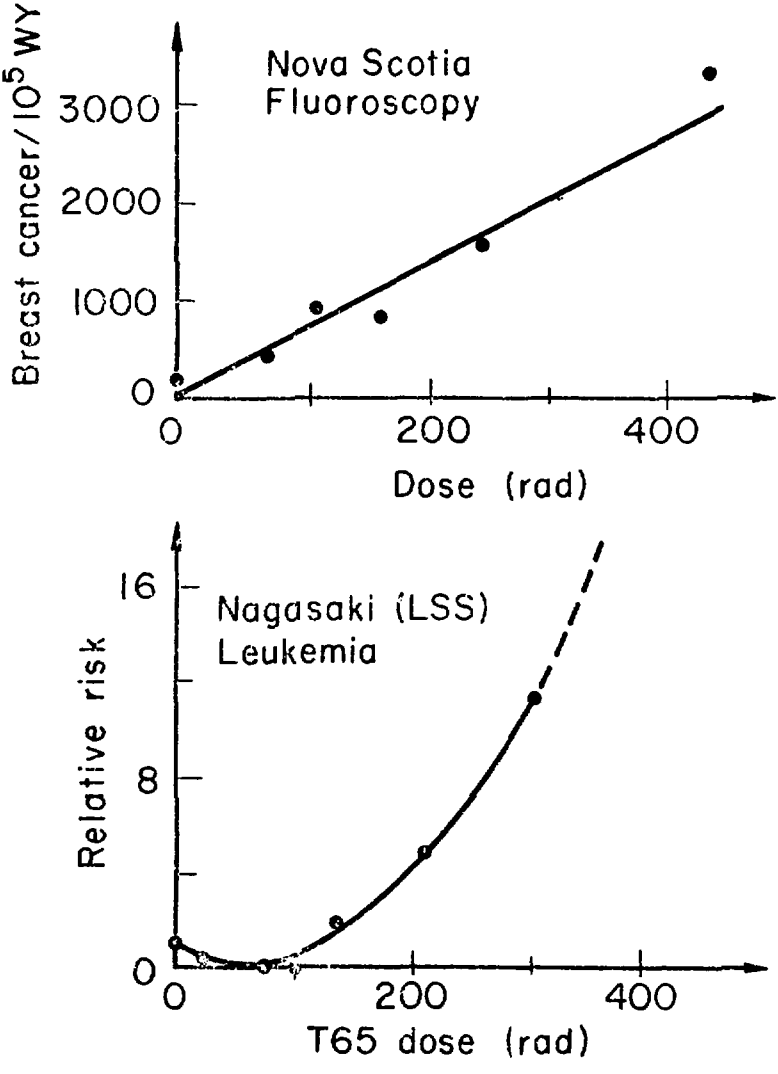
Breast cancer/10^5 WY
3000
2000
1000
0
Nova Scotia
Fluoroscopy
0
200
400
Dose (rad)
Relative risk
16
8
0
Nagasaki (LSS)
Leukemia
0
200
400
T65 dose (rad)
XBL801-3027

Figure 3