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Jacob I. Fabrikant

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THE CONTRIBUTION OF MODERN MEDICAL IMAGING TECHNOLOGY TO
RADIATION HEALTH EFFECTS IN EXPOSED POPULATIONS^{1,2}

Jacob I. Fabrikant, M.D., Ph.D.³
Biology and Medicine Division
Lawrence Berkeley Laboratory
University of California, Berkeley⁴

and

Department of Radiology
University of California School of Medicine
San Francisco

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- 2 Supported by the Office of Health and Environmental Research of the U.S. Department of Energy under Contract W-7405-ENG-48.
- 3 Professor of Radiology, University of California School of Medicine, San Francisco
- 4 Mailing Address: Donner Laboratory, University of California, Berkeley, California 94720

THE CONTRIBUTION OF MODERN MEDICAL IMAGING TECHNOLOGY TO RADIATION HEALTH EFFECTS IN EXPOSED POPULATIONS

Jacob I. Fabrikant, M.D., Ph.D.
Donner Laboratory
University of California, Berkeley

The introduction of technically-advanced imaging systems in medicine carries with it potential health hazards, particularly from ionizing and nonionizing radiation exposure of human populations. This paper will discuss what we know and what we do not know about the health effects of low-level radiation, how the risks of radiation-induced health effects may be estimated, the sources of the scientific data, the dose-response models used, the uncertainties which limit precision of estimation of excess health risks from low-level radiation, and what the implications might be for radiation protection in medicine and public health policy. Research supported by the U.S. Department of Energy under Contract W-7405-ENG-48 and the Environmental Protection Agency.

Introduction

The clinical uses of radiological imaging invade almost all medical specialties and are frequently changing; to keep abreast with them is a constant challenge to investigators and clinicians. In the past decade a wide variety of sophisticated, costly, and potentially hazardous diagnostic imaging procedures are becoming available, and at the present time we still do not know how to assess what is to be learned and how to examine selectively those imaging contributions of greatest significance and promise. In many instances, comparatively simple laboratory tests and radiographic procedures will adequately supplement the patient's history and physical examination and will supply the additional data required for diagnosis and effective management. We are nevertheless continuing to investigate and develop the potential of a host of new static and dynamic imaging technologies, such as computed tomography and fluoroscopy, positron emission tomography, heavy ion radiography, nuclear magnetic resonance, microwave imaging, fluorescent-excitation imaging, and many more in addition to new developments in radiography, nuclear medicine, ultrasonography, and thermography. All these imaging technologies, and more, are designed to achieve two important goals: to improve medical diagnosis and to decrease the radiation dose.

These new imaging technologies promise potential benefits, but are not without potential risk. The introduction of ionizing radiations in medical imaging has brought with it concomitant health hazards of low-dose radiation exposure. During the present discussion, I should like to share what we know, and what we do not know of these health

hazards of low-level radiation and why the quantitative estimates of these risks continue to be clouded by scientific dispute and controversy. I shall use as my setting 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 radiation scientists as to the health effects of very low levels of radiation, even levels as low as our natural background, and particularly levels to be expected in diagnostic medical imaging.

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 gamma rays from radioactive sources, since these are the ionizing radiations most often encountered in diagnostic radiological 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,¹ the UNSCEAR,^{2,3} the BEIR Committee,^{4,5} the NCRP^{6,7} 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 statistical analyses of epidemiological surveys of exposed human populations, on research in laboratory animals, on dose-response relationships of carcinogenic, teratogenic and genetic effects, and on mechanisms of cell and tissue injury, there are a number of important conclusions on the health effects of ionizing radiation about which we are now certain.

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.

The effects of 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.

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
Health Effects of Low-Level Radiation?

However, there is much we do not know about the potential health effects of low-level radiation. We do not know 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.

The 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.

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.

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 continue 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.

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?

There is still 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, current scientific opinion suggests the use of 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 current 1980 BEIR-III Report⁴ differs substantially from the earlier 1972 BEIR-I Report.³ 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, suggest that complex dose-response relationships exist between radiation dose and cancer incidence.^{8,9} 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 of 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 cancer, 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 is no reason to assume that any form of the dose-response relationships is 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. We are now becoming 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 is therefore essential that very precise explanations and qualifications of the assumptions and procedures involved in determining such risk estimates are provided in any calculations resulting in estimates of excess cancer risk. It is now generally 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, there is general agreement 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 risk 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, and of diagnostic radiological exposure. 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.¹⁰ 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 or with

diagnostic radiological exposure levels 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, as in the case of diagnostic radiology, 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 has been highlighted recently by some radiation scientists. It is a most difficult task to estimate the carcinogenic risk of low-dose low-LET whole body radiation. As the earlier studies, such as the BEIR-I Report⁵ in 1972, had done, some current radiation scientists adopt a linear hypothesis of dose-response to estimate the cancer risk 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 Bureau of Radiological Health or the Environmental Protection Agency to adopt stricter health standards to protect people against radiation. Other radiation scientists believe this to be an alarmist approach. When there is no human epidemiological evidence, these scientists prefer to assume that the risks of causing cancer by radiation are proportionally lower.

Let us look at some of the problems. There are two important points to consider: (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 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 dose-response function. In this situation, it is not unreasonable 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, prove practical.

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; for example, cancer of the skin is not observed at low doses,¹² and dose-response relationships observed in the Nagasaki leukemia data appear to have positive curvature.¹³ The incidence of breast cancer seems to be adequately described by a linear dose-response model.^{9,11}

Simplification of any linear-quadratic model is required to obtain statistically stable risk estimates in many cases. It is now well known that some radiation scientists 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.⁸

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.

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

The chief sources of epidemiological data used in current analysis and calculations of the carcinogenic risk of low-level radiation are

the Japanese populations exposed to whole-body irradiation in Hiroshima and Nagasaki,¹³ patients with ankylosing spondylitis¹⁴ and other patients who were exposed to partial-body irradiation therapeutically,¹⁵ or to diagnostic x-rays¹⁶ and various occupationally exposed populations,¹⁷ 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 for 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 illustrative computations of the lifetime risk from whole-body exposure, the estimates calculated in the 1980 BEIR-III Report should be considered. Here, three radiation exposure situations were established: (1) a single exposure of a representative (life-table) population to 10 rads; (2) a continuous, lifetime exposure of 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 do not reflect any circumstances that would normally occur, but embrace the areas of concern—general population and occupational exposure and single and continuous exposure.

Below these dose levels chosen for the current report, the uncertainties of extrapolation of risk to very low levels were considered 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 medical and nuclear industries. 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 cancer incidence.

The resulting cancer mortality risk estimates calculated 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 radiation, the linear-quadratic model predicts increases of about 0.5 percent and 1.4 percent 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 3 percent to 8 percent over the normal expectation, depending on the projection model.

Cancer-incidence risk estimates are less firm than mortality estimates; a variety of dose-response models and several data sources are used. The dose-response models produce estimates that differ by more than an order of magnitude, whereas the different data sources give 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 are about 2 percent to 6 percent, depending on the projection model. Risks for females are substantially higher than those for males, due primarily to the relative importance of radiation-induced thyroid and breast cancer.

Should Radiation Risks be Compared with Other Risks?

These estimated figures remain unreliable, but they do provide a basis for comparison with other estimates of avoidable risks, or voluntary risks, encountered in everyday life. This comparison can be approached in an objective way in regard to occupational risks.¹⁸ In 1975, the U.S. Government report on Accident Facts published by the National Safety Council¹⁸ (Table 1) indicated that the estimated risk from the occupational exposure to radiation would be at the very lowest end of the scale, indeed very much less than 100 fatal cancers per million persons per rad lifetime risk. If occupational exposure in industries listed in Table 1 is considered for workers 20 to 65 years of age, the safety margin for lifetime occupational risk from radiation carcinogenesis would approach a safety factor of over 50 to 100 compared with that expected each year in the hazards of dying in government and service occupations.¹⁹ A lifetime safety factor of over 500 to 1,000 obtains in comparison with that of fatal mining accidents each year.¹⁹

Table 1
Occupational Accident Rates - U.S. (59)¹
(Deaths/10⁶/year in 1975)

Industry	Deaths	Mean (1955-75)
Trade	60	83
Manufacturing	80	103
Service and Government	115	131
Transport and Public Utilities	330	373
Agriculture	580	613
Construction	610	717
Mining and Quarrying	630	994
All Industries	150	200

¹ Modified from References 18 and 19.

In regard to the risk of radiation carcinogenesis in the general population, even very crude quantitation of radiation risk estimates provides some method for intercomparison with other objective risks, both voluntary and involuntary experiences. The evidence, for example, for the magnitude of risk of dying from malignancy induced by radiation as a result of exposure from nuclear power generation providing 1 kilowatt of energy per person year in the general population has been estimated at less than 0.5 excess cancer deaths per million persons per year.¹⁹ If one wishes a familiar comparison for a comparable risk of dying in the population, this has been estimated to be equal to smoking one cigarette every two years.¹⁹

It is tempting to establish qualitative or quantitative levels of risk for various societal activities, then make those comparisons which would appear appropriate in an attempt to develop a method for comparative indices of risk and benefit.¹⁹⁻²⁰ However, such comparisons are easily made, and such comparisons must be biased, since they assume both, that no avoidable risk is acceptable, and that acceptability of unavoidable risk depends on comparisons with existing alternatives or with other existing risks accepted by society. Both assumptions can be proven spurious. Comparison could be justified, however, not on the basis of existing risks, but on the basis of existing alternatives provided the activity were desired or could be abandoned. It is just not possible in the complexities of modern medicine to dispense with medical radiation exposure in diagnosis and treatment of disease at the present time, and it is not possible nor practical to lower the population dose by the delivery of fewer exposures to each individual radio-diagnostic or radiotherapeutic patient. It is possible, on the other

hand, not to build a nuclear energy plant or a coal-burning plant, and thereby providing less energy to the population by delivering less, or making available less energy to each individual.

It follows, therefore, that health protection standards do not necessarily have to be related directly to risk. Two natural boundary conditions of comparative population risks in the United States can be discerned. The high risk boundary of 1:100 is the statistical risk of death from all causes (risk of death per year, 1:100 to 1:1,000). The medium level risk of death per year would encompass the range of 1:1,000 to 1:100,000. This would include the risk of death per year from exposure to 170 mrem per year. The low risk boundary of 1:1,000,000 is the risk of death from natural disasters and catastrophes such as floods, earthquakes, lightning and snakebites.²⁰ These negligible risks tend to be readily accepted by society without much that can be done to avert them, so-called "acts of God" (risk of death per year, 1:100,000 to 1:10,000,000). This would include the risk of death per year from exposure to 1 mrem per year. However, the high risk boundary can often be controlled by society in most circumstances, since they invariably involve individual decision-making, such as mode of transportation (e.g., auto travel, plane travel) and sports (e.g., hunting, skiing, mountain climbing).¹⁸⁻²⁰ And finally, the perception of risk does not necessarily lead to a logical response to avoid them. At the height of the radioactive fallout deliberations in the early 1960s, it was estimated that the removal of traces of radiostrontium from milk at the cost of a few pennies per quart would ultimately cost some 20 million dollars for each case of bone cancer averted.²⁰

Any attempt at assessing a cost-benefit relationship for any societal activity involving ionizing radiation implies that an identifiable benefit exists, and can be identified.^{18,20} This is much more discernable for radiation and medicine, than for radiation and nuclear energy. Furthermore, unless society needs the associated benefit, any associated man-made risk may be regarded as avoidable. Thus, if one benefit of nuclear energy lies in the avoidance of injurious health from fossil fuel combustion, it does not necessarily follow that the benefits from nuclear energy exceed those of alternative options, or that the risks are less. And finally, an assessment of cost-effectiveness is necessary not only to determine how avoidable a defined risk can be, but it provides insights into decision-making on how societal resources may be allocated to decrease existing risks at an increased financial cost.^{18,20}

What Conclusions can be Drawn?

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 and particularly for the medical uses of ionizing radiations. 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 hazards of everyday life. When compared with the benefits that society has established as goals derived from the necessary activities of medical care and energy production, as two important examples, 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 believe that the potential health hazards of low-level radiation are central to the development of new imaging technologies in medicine. I believe that a substantial part of the controversy of government regulation and control has been mounted on the question of low-level radiation and linked to public acceptance of rigid and inflexible radiation protection standards in matters of radiation and health. 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, and for the effective application of new technologies in medicine and industry. 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 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.

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