An overwhelming amount of data show that there is no safe level of exposure and no dose of radiation is so low that the risk of a malignancy is zero. The question is—how great is the risk?

KARL Z. MORGAN

Cancer and low level ionizing radiation

Reports of a significant increase in the risk of cancer from exposure to low levels of ionizing radiation and evidence suggesting that federal agencies had attempted to squelch these reports resulted in the congressional hearings held last winter on this subject being conducted at a rather hot pace. What prompted these heated discussions were the two letters sent in November of 1977 by several organizations—the Environmental Policy Center; Friends of the Earth; Oil, Chemical and Atomic Workers; Environmental Defense Fund; Public Interest Research Group; Sierra Club; Union of Concerned Scientists and the National Resources Defense Council—to government officials questioning actions taken by their respective departments.

The letter to James Schlesinger, Secretary of the Department of Energy, stated that the Energy Research and Development Administration (now DOE) had made a serious mistake in transferring the study by T. F. Mancuso, A. Stewart and G. Kneal at the University of Pittsburgh on the "Lifetime Health and Mortality Experience of Employees of ERDA Contractors" to the federal government's Oak Ridge, Tennessee, operations under the direction of E. A. Tompkins. The letter suggested that this move was part of "a well-defined pattern of harassment and intimidation of scientists who do not agree with the position of promoters of radiation technologies that there are no adverse effects associated with exposures to low-level ionizing radiation."

The letter to Joseph Califano, Secretary of Health, Education and Welfare, questioned the discontinuance of the Tri-State Study by I.D.J. Bross at the Roswell Park Memorial Institute in Buffalo, New York, on the risk of cancer among children who had been exposed to diagnostic X ray in utero. The Califano letter also criticized the peer review system of the National Cancer Institute, specifically an incident where radiologists were "embarrassed and angered with the findings" of effects of low-level exposure to radiation which had then been used as evidence for limiting mass mammography programs in the United States. The Bross program was canceled following peer review of the study.

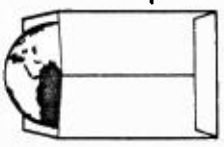
Both studies referred to in these letters have shown a significant increase in cancer incidence following exposures that were far below the maximum permissible exposure, MPE (that is, far below 5 rem per year to the total body of a radiation worker). The Mancuso study had confirmed the earlier findings in 1974 of Samuel Milham [1] that the cancer risk for radiation workers at the government's plutonium production facilities at Hanford, Washington, had increased. The Bross study had shown a 5,000 percent increased risk of cancer among the children who had been exposed to diagnostic X ray in utero, and who later developed certain respiratory diseases.

The hearings were held last January and February in the Rayburn Building by Representative Paul Rogers, chairman of the Subcommittee on Health and the Environment of the Committee on Interstate and Foreign Commerce. Testimony from many witnesses was presented on a variety of subjects such as the Mancuso and Bross programs, Test Smokey, petitions to reduce the maximum permissible exposure by a factor of 10, mechanisms of radiation damage, the peer review system as it applies to awarding of government research contracts, etc.

The congressional hearings were followed by a conference on low level radiation, held last February and conducted under the chairmanship of the author, which was sponsored by the Environmental Policy Institute, the Atomic Energy Forum and the Environmental Study Conference. The latter is a bipartisan caucus of more than 280 members of the House and Senate who have a shared interest in staying informed on environmental and related energy developments; Senators Gary Hart and James Jeffords are the co-chairmen. The panelists represented most of the organizations, government and private, that are keenly concerned with the question of tumor formation (oncogenicity) and low level radiation. Given the wide range of views and interpretation of exposure data, these conference proceedings (available from Senator Hart's office) should be interesting reading.

This article presents in some detail my views regarding the risk of cancer from low-level exposure to ionizing radiation.

During the first years of the Atomic Age (1942-1960) a large number of scientists—perhaps most who were knowledgeable in health physics and radiobiology—accepted the threshold theory that there is a safe level of exposure to ionizing radiation, and that as long as a person does not exceed this threshold or safe level no harm will result or the radiation damage on the average will be repaired as fast as it is produced. From 1960 to the present, an overwhelming amount of data have been accumulated that show there is *no safe level* of exposure and there is no dose of radiation so low that the risk of a malignancy is zero. Therefore, the question is not: Is there a risk from low level exposure? Or, what is a safe level of exposure? The ques-



tion is: How great is this risk? Or, how great may a particular radiation risk be before it exceeds the expected benefits, such as those from medical radiography or nuclear power.

It is obvious to all scientists in the field, as well as to diehards for the threshold hypothesis, that at least for some types of radiation damage and for some kinds of radiation exposure (especially from low LET radiation, that is, X, gamma and beta radiations) there is some repair of the radiation damage going on in the body. The diehards, however, do not seem willing or able to accept the evidence that for man there is never a complete repair of the radiation damage, since even at very low exposure levels there are many thousands of interactions of the radiation with cells of the human body. For example, one rad of X rays of 1 million electron volts corresponds to 2.2 billion photons per square centimeter acting on the body. It is inconceivable that all the billions of irradiated and damaged cells would be repaired completely or replaced.

There are undoubtedly many mechanisms of radiation injury such as damage to cell membranes, damage to the body repair mechanisms, indirect damage (for example, damage to cell blood supply and formation of harmful chemicals such as hydrogen peroxide in cell cytoplasm) and impairment of efficiency to lung clearance mechanisms. Each of these mechanisms may contribute to the development of a malignancy. However, perhaps the most significant damage from low-level exposure results from direct interaction of the stream of ions with the nucleus of one of the billions of irradiated cells that may in the rare event survive and continue to divide but fail to repair the radiation damage.

There are 46 chromosomes in the nucleus of each normal somatic cell of the human body, and along each chromosome are coded millions of bits of information like an immense library which enables or instructs the cell to function properly and to divide or stop dividing at the appropriate time. When radiation passes through the human body, four principal events can occur:

- the radiation passes through or near the cell without producing any damage,
- the radiation kills the cell or renders it incapable of cell division,
- the radiation damages the cell but the damage is repaired adequately, or
- the cell nucleus (or library of information) is damaged but the cell survives and multiplies in its perturbative form over a period of years (5 to 70 years) and forms a clone of cells that eventually is diagnosed as a malignancy.

Only this last event relates to somatic damage such as cancer from low-level exposure. It seems obvious that if the cell nucleus is damaged and some information is lost or if a similar series of events leads to the development of a malignancy, there can be no dose so low that the risk is zero. Thus the risk of induction of cancer from radiation exposure increases more or less with the increase or accumulation of radiation exposure. The risk is simply one of chance, just the same as the risk of chance of an accident everytime a trip is made in a taxi.

It is evident also that all persons do not run the same risk of developing a malignancy from a given radiation exposure and that the risk of some types of cancer is greater for certain people than it is for others. Burch [2, 3] has shown, for example, that the final onset of a malignancy or other disease may require a series of events and a given type of leukemia may require as many as three successive events (like throwing three electrical switches connected in series). For example, if one identical twin dies of a particular type of leukemia (one switch thrown genetically), the other twin has a high probability of eventually suffering a similar fate. Some of these switches may be thrown by viruses, bacteria, chemicals, mechanical insults or by radiation.

Studies by Bross [4, 5] lend support for the series of events hypothesis suggested by Burch, and suggest synergistic relationships between them. He has shown, for example, that children (ages 1-4) with allergic diseases such as asthma or hives have a 300 to 400 percent increased risk of dying of leukemia compared with other children (that is, allergic diseases throw one switch). Children who received in utero diagnostic X ray exposure have a 40 to 50 percent increase in risk of dying of leukemia [6] but children with two switches thrown (that is, in utero exposure and later developing an allergic disease) have a 5,000 percent increase in risk of dying of leukemia.

Students of Stewart and Kneale [6], MacMahon [7], the BEIR Committee [8], Bross [4, 5] and others suggest that children have a higher risk of dying of radiation-induced leukemia than do middle-aged persons, Hempelmann [9], Albert and Shore [10], Modan, et al., [11], Silverman and Hoffman [11, 12], and others have shown that radiation-induced thyroid carcinoma presents a higher risk in children than in an adult population and, as with leukemia, this risk decreases linearly as the dose decreases.

There are studies also which indicate that sex is a factor in the type of a malignancy which may develop. Mancuso et al. [13] report that older

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and younger men have a higher risk of radiation-induced malignancies than do men of middle age. After examining data of Saenger and Tompkins [14], E. B. Lewis [15] pointed out that they had failed to note the significant increase in leukemia among persons between ages 50 and 79 who received iodine-131 treatments. Najarian [16] in his studies of the Portsmouth (N.H.) Navy Yard nuclear submarine workers reported a 450 percent higher death rate from leukemia among the radiation workers than the general population, and noted that the cancer incidence was especially high among workers aged 60 to 69. Thus, because of genetic inheritance, various diseases, age, sex, eating and smoking habits and, perhaps, many other individual characteristics, certain members of the general population have a higher risk of radiation-induced malignancies than others.

The cancer risk from exposure to ionizing radiation is much greater than was thought to be the case some years ago. Following the deaths of the Japanese survivors of Hiroshima and Nagasaki from radiation sickness, many scientists believed that the principal chronic risk from radiation exposure was only an excess of cases of leukemia, which r ached a peak about six years after the bombing and then slowly declined. Many persons concluded that the only chronic risk among the survivors was leukemia. Unfortunately, however, as the study of these survivors continued other forms of cancer (bone, breast, lung, salivary gland, prostate, thyroid, etc.) showed a significant increase [17, 18]. Probably with the passage of time we will find that this exposure has resulted in an increase of statistical significance in many or most kinds of malignancies that are common among human populations.

It should be emphasized here that although this paper treats only the formation of tumors by ionizing radiation, the genetic risks and especially those associated with recessive mutations may be as harmful and debilitating to the human race as the increase in risk of cancer. Therefore, I wish to pause and sound a warning that I'm sure my long-time friend, the geneticist H. J. Muller, would urge me to make were he alive today:

The BEIR [8] report only treated the long-term recessive mutation question in a superficial way. It may well be that many and perhaps most of our human diseases, including cancer, are related to a genetic factor and especially to Muller's 10,000 non-visible or "small" mutations that result from each observed mutation. As Muller emphasized, it may be that in the long run these small mutations that result in a lack of vigor, susceptibility to disease, a slight reduction in mentality and physique, etc., will be a far greater

Table 1
Changes in Levels of Permissible Exposure to Ionizing Radiation[a]

FOR RADIATION WORKERS

Recommended Values		*Comments*
0.1 erythema dose/y (~1R/wk for 200 kV X ray	52 R/y	1925: Recommended by A. Mutscheller and R. M. Sievant 1934: Recommended by ICRP and used worldwide until 1950
0.1 R/day (or 0.5 R/wk)	36 R/y	1934: Recommended by NCRP
0.3 rem/wk	15 rem/y	1949: Recommended by NCRP. 1950: Recommended by ICRP for total body exposure
5 rem/y	5 rem/y	1956: Recommended by ICRP 1957: Recommended by NCRP for total body exposure

FOR MEMBERS OF THE PUBLIC

Recommended Values		*Comments*
0.03 rem/wk	1.5 rem/y	1952: Suggested by NCRP for any body organ
0.5 rem/y	0.5 rem/y	1958: Suggested by NCRP 1959: Suggested by ICRP for gonads or total body
5 rem/30y	0.17 rem/y	1958: Suggested by ICRP for gonads or total body
25 mrem/y	0.025 rem/y	1977: Suggested by EPA [20] for any body organ except thyroid[b]
5 mrem/y	0.005 rem/y	1974: Suggested by ERDA for persons living near a nuclear power plant[c]

R = roentgen. 1 R ≃ 0.88 rem
rem = roentgen equivalent man
mrem = millirem
NCRP = National Council on Radiation Protection and Measurements
ICRP = International Commission on Radiological Protection

[a]For additional information, see Morgan [21].
[b]The limit set by the Environmental Protection Agency for the thyroid was 0.075 rem per year.
[c]Present radiation protection guide of the Nuclear Regulatory Commission.

Most of us recognize that the risk of inducing cancer at low doses of radiation is far greater than we once thought it to be, and it may be as great or greater for the human race than the genetic risk.

burden to society than the easily identifiable dominant mutations. This is because the small mutations are eliminated so slowly from the gene pool.

During the first years of the Atomic Age (1942-1960) almost everyone assumed that the genetic risk from low-level radiation exposure far exceeded the risks of chronic somatic damage such as cancer or life shortening. However, it has become increasingly clear that this assumption may be unwarranted and untenable. The BEIR [7] committee pointed out "until recently, it has been taken for granted that genetic risks from exposure of populations to ionizing radiation near background levels (about 100 millirems per year) were of much greater import than were somatic risks. However, this assumption can no longer be made if linear non-threshold relationships are accepted as a basis for estimating cancer risks." The committee then went on to supply many pages of data, most of which support the linear hypothesis. In 1971 the International Commission on Radiological Protection (ICRP) [19] made a similar observation: "It could be concluded that the ratio of somatic to genetic effects after a given exposure is 60 times greater than was thought 15 years ago."

Table 2
Cancer Risk and Known Range of Linearity

Linearity of Dose down to	*Risk per Person per rad*	*Comments*
< 10 rad	$0.3 - 1.0 \times 10^{-4}\ell$ $0.5 - 1.7 \times 10^{-4}C$	Hiroshima and Nagasaki atomb bomb survivors [8, 30, 31]; see also Moriyama and Kato[a]
Av. 370 rad	$0.2 - 0.3 \times 10^{-4}\ell$	arthritis of spine (ankylosing spondylitis) patients [6, 27]
0.2 – 0.8 rad	$3 \times 10^{-4}\ell$ $6 \times 10^{-4}C$	pelvimetry exposures—Stewart and Kneale [6, 27]
~1.0 rad	$3 - 30 \times 10^{-4}\ell$	pelvimetry exposures—Bross [4, 5]
20 rad	$0.5 - 1.1 \times 10^{-4}T$	X-ray therapy—Hempelmann [9]
6.5 rad	$1.2 \times 10^{-4}T$	X ray for ringworm (tinea capitis)—Modan et al. [11] and Silverman and Hoffman [12]

ℓ = leukemia risk per person.rad
C = total cancer risk per person.rad
T = thyroid cancer risk per person.rad
rad = 100 ergs per gram of tissue

[a]I.M. Moriyama and H. Kato, "Mortality Experience of A-Bomb Survivors 1970–72," JNIH-ABCC Life Span Study Report, Tech. Report 15-73 (Washington, D.C.: Atomic Bomb Casualty Commission, 1973).

The emphasis of this article on cancer risk is not to depreciate the seriousness of genetic risks from exposure to ionizing radiation but rather to point out that the scientific community was rather smug 15 years ago (as some scientists still are today) in the belief that somatic risk is far less than genetic risk and that somatic risk is almost negligible at low doses. Now most of us recognize that the risk of inducing cancer at low doses of radiation is far greater than we once thought it to be and it may be as great or greater for the human race than genetic risk.

There has been a number of reductions in the permissible exposure levels for occupational workers and for the public during the past 35 years. Some of the quantum drops in permissible exposure levels during this period are presented in Table 1. The occupational maximum permissible exposure level has dropped by a factor of 10 and the level for the public by a factor of 300.

Much of what has been said about the risks of exposure to low levels of ionizing radiation would have considerably less weight if it could be shown that although the linear hypothesis holds at intermediate to high levels of exposure it provides a very large element of conservatism at low doses and dose rates. Unfortunately, in most cases of human exposure there is no evidence of a safety factor at low doses if we assume that the linear relationship between radiation dose and cancer induction at high doses applies also at low doses. We have a large amount of data—much of it human—showing a statistically significant increase in a number of types of malignancies as a consequence of exposure to low doses of ionizing radiation and the number of malignancies increases progressively as the dose accumulates. These doses in some cases are considerably lower than the present levels of maximum permissible annual exposure of the radiation worker. In fact, many researchers [22-25] have shown that in some cases the linear hypothesis actually underestimates the risk.

Table 2 indicates the magnitude of the cancer risk and shows that this risk increases linearly with the accumulated dose down to very low value, that is, down to 0.8 rad for leukemia or other forms of cancer (especially central nerve system tumors) resulting from pelvimetries, and to 6.5 rad for thyroid carcinoma resulting from X ray therapy of the scalp for ringworm (tinea capatis). It must be pointed out that these doses (0.8 to 6.5 rad) are not the doses below which the linear hypothesis breaks down, but the lowest points on the human exposure curves for these two malignancies. And we have every reason to believe the linearity of these curves continues on down

to zero dose and that there is a similar linearity for other types of cancer that simply have a longer incubation period or have not been studied over a wide range of doses to a human population.

It should be emphasized also that this 0.8 rad is only 2 percent of the 42 rad permitted by the International Commission on Radiological Protection (ICRP [26]) each year to the active bone marrow of the radiation worker and that the 6.5 rad is only 13 percent of the 50 rad permitted each year to his thyroids. (The maximum permissible concentration (MPC) values given by the International Commission and the National Council on Radiation Protection (NCRP) for members of the public are calculated on the basis of 10 percent of these dose rates, that is, 4.2 rem per year for bone marrow and 5 rem per year for thyroid.)

If a million children each received 1 rad from in utero exposure, we might expect 300 to 3,000 leukemias, depending upon whether or not the child had certain respiratory diseases, some of which, as indicated by Bross [4, 5], act synergistically with radiation exposure (see Table 2). There is not as much data available on the effects of low level exposure of adults as for children but, as seen from recent data of Mancuso et al. [13], the risk of radiation-induced malignancies other than leukemia may be as great or greater for adults than that for children (perhaps as high as 70×10^{-4} cancers per person.rem). Furthermore, studies of Stewart [6, 27], MacMahon [7, 28] and many others indicate that following in utero exposure the incidence of focal cancers (such as central nervous system tumors) is about the same as of leukemia, so that the total number of fatal malignancies might be twice the number of leukemias given in Table 2: 600 to 6,000 cancers for a million children exposed to only 1 rad.

In 1970, Jablon and Kato [29] pointed out that their data on the survivors of the atomic bombings who were exposed in utero do not support some findings [6-7, 27-28]. They indicated that on the basis of findings of Stewart and Kneale, and upon the corresponding linear hypothesis, they should expect 36.9 excess cancers in this group during the 10 years following exposure, but only one case of liver cancer was reported. As a consequence many persons were quick to proclaim that there is something wrong with the retrospective studies of cancer induction by diagnostic in utero X ray as reported by Stewart, MacMahon and others and that now we can relax. Unfortunately (for in utero exposed children), this is not the case.

Stewart and others [22, 30, 32-34] have published reports which support the studies of MacMahon, etc., of cancer induction by low level (< 1 rem) diagnostic in utero X ray and there is little doubt that the Japanese studies greatly underestimate this cancer risk. In fact, Jablon and Kato [8, 29], commenting on the unusually low cancer rate among children who had received in utero exposure at the time of the bombing, stated that "conceivably such a result might follow if there were an excessive spontaneous abortion rate for fetuses by large doses." Thus the fetuses which were most likely to have developed into cases of radiation-induced leukemia received such high doses and were subjected to so much trauma that they failed preferentially to survive. In fact, there is reason to believe that an unusually high incidence of abortions and high rate of infant mortality followed the atomic bombings.

Many studies [35] have shown that during periods of stress and community disasters it is the fetuses, infants and young children that suffer the most. It is known also that during such periods of suffering and unrest incipient cancers can easily be mistaken for acute infections. Also, it seems likely that the Japanese control group may have had a greater cancer risk than normal.

Rotblat's recent publications [36] seem to confirm the above explanations of why the cancer risk as determined from survivors of Hiroshima and Nagasaki atomic bombings is too low. He compared the cancer risk in two groups: one that entered Hiroshima during the first three days following the explosion and were exposed to the residual neutron-induced activity and radioactive contamination from the fallout; and the other group that entered Hiroshima at a later date and received negligible radiation exposure. Neither of these groups was subjected to the trauma of blast, fire, burial under debris, etc. The leukemia risk to the first group exposed to residual radiation was 1.6×10^{-4} leukemias per person.rad. Rotblat was conservative in several of his assumptions so this risk estimate must be considered as a lower estimate. This value for adults is, therefore, in agreement with the leukemia risk estimate in Table 2 of 3×10^{-4} which applies to children that received in utero exposure from medical diagnosis. Rotblat points out that this leukemia risk estimate is eight times the estimate of 2×10^{-5} leukemias per person.rad as given by the International Commission on Radiological Protection (ICRP) [26].

At this point we might make the interesting conjecture that perhaps Rotblat has provided a clue to the puzzle of why no genetic mutations have been observed among the offspring of the survivors of the bombings of Hiroshima and Nagasaki. Thus, there is little doubt the Japanese data greatly underestimate the risk of radiation-induced cancer and the genetic risk is also underestimated.

Some of the reasons [22] why in many cases use of the linear hypothesis to estimate risk at low doses is not conservative are as follows.

• *Overkill at high doses.* Most estimates of risk from radiation exposure are based on linear extrapolation of effects at high doses down to zero dose. Often with such extrapo-

More often than not extension of data from animals to man results in an underestimate of risks, especially at low levels of exposure.

lation insufficient account is taken of overkill at high doses and in no case can more than 100 percent of the animals be killed by radiation. Sometimes one simply determines the best least-squares line which will pass through the (0,0) point. Some points used in determining the slope of this line may be on the upper bend of the curve where the animals are injured by large doses of radiation and do not survive long enough to die from the malignancy under study.

• *Short follow-up period of human studies.* Most studies [8] of effects of ionizing radiation on man extend over only a small fraction of his life span. If, for example, one determines the slope of the curve of thyroid carcinoma risk vs. X-ray dose and the followup period is only seven years, studies of the population until all have died would increase the slope of the curve and the risk estimate especially at the low dose end of the curve.*

• *Fractional life span animal studies.* Sometimes comparisons are made between fetal damage during the first trimester of a mouse and fetal damage expected during the first trimester of a woman, or a comparison is made between animals having a life span of 20 years with expected effects over the life span of man. Since in most cases damage from radiation exposure relates to what happens in a given number of years following the exposure rather than what happens over a certain fraction of the animals' life span, such extrapolations to man can only lead to underestimates of risk.

• *Radiosensitivity differs among animal species.* Many studies have emphasized the risk of extrapolating data on effects of radiation exposure from one animal to another or to man. Differences in metabolism, turnover rate, GI tract uptake, skin perspiration, blood circulation, mitotic index, etc., can have a marked effect on animal response to a given dose of ionizing or non-ionizing radiation. An examination of data leads me to conclude that more often than not this kind of extension of data from animals to man results in an underestimate of risks, especially at low levels of exposure.

• *Heterogeneity of human population.* The vast majority of studies of effects of radiation exposure are carried out with disease free inbred animals. Radiation ecology programs must be extended to animals in the wild, if we are to simulate effects we expect from low doses to human populations.

In assessing population risk of low levels of exposure we need to know dose response for young and old, male and female, sick and well, fat and slim, different eating habits, etc. When we have such data, undoubtedly our estimates of risk to certain groups of the population from low level exposure will be much greater than the risk to the so-called average man.

• *Cell sterilization.* It is well established that as humans grow old, the percent of abnormal cells in the body increases; for example, the percent of chromosomal aberrated cells increases with age of an animal. Using our earlier analogy we might say the body has accumulated more cells in which one or more switches are thrown or which present a larger cross-section for cancer production from radiation exposure.

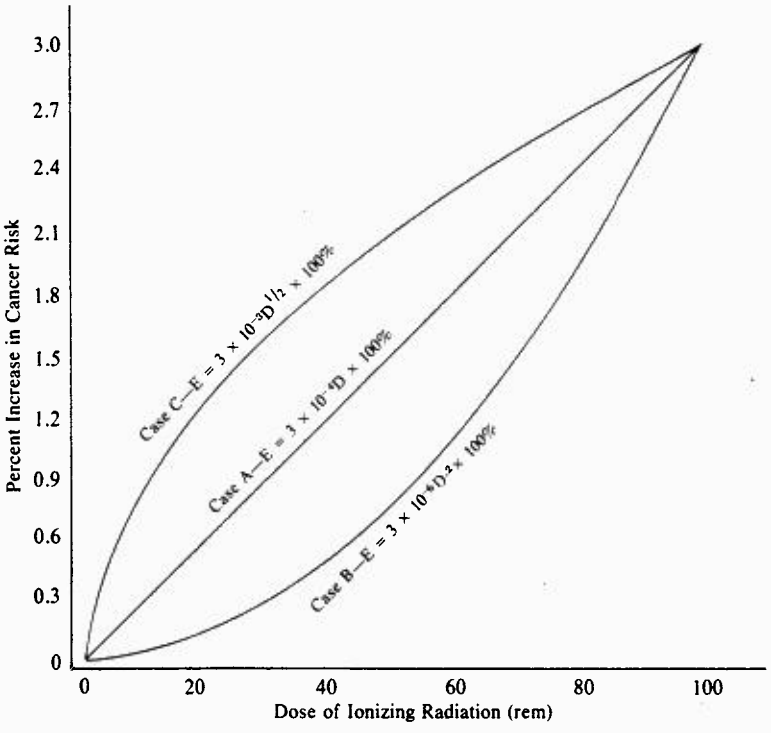
As indicated above it is commonly believed that some type of malignancies develop as a result of a series of changes that take place in the 46 chromosomes that comprise the nucleus of a normal somatic cell in man. Thus, as man ages he has a scattering of cells and clones of cells which have one or more abnormalities and are highly susceptible to be triggered in the development of a malignancy. When studies are conducted on animals exposed to high doses of radiation, cell sterilization may take place such that many of these cells that are likely targets for development of a malignancy are preferentially destroyed. Thus, such data points at high exposure levels would tend to reduce the slope of the curve that is extrapolated to zero dose and may result in an underestimate of risk at low levels of exposure.

There is no question that some animal studies of exposure to X or gamma radiation have shown that the cancer risk per rem is less at low doses when the dose is protracted or fractionated. This seems, definitely, not to be so for high LET radiation (alpha or neutron exposure) where there is little or no repair of damaged cells and where only a single particle (alpha or heavy recoil ion) passing through the cell is required to initiate the damage (for example, the rare events in which a surviving precursor malignant cell is produced. However, for some types of radiation damage (leukemia induction among middle-aged persons), following exposure to low LET (X or gamma) radiation, it may require two or more close encounters of the photons with the nucleus of a cell before the damage can become a precursor of a malignancy. In such a special case the risk per rad would be less at low doses than at high doses and the linear hypothesis would be conservative (see figure).

The average radiation dose of the 442 Hanford workers who died of cancer during the study period (1944-1972) was only about 1 rem [13]. Mancuso, Stewart and Kneale estimate only 6 to 7 percent of the cancer deaths (26 to 31 cancers) were induced by this radiation. The total number of deaths in the study group was 3,520 so their cancer risk was 7 to 8 $\times$ 10^{-3} or about 25 times the 0.03 percent risk given by curve A in the figure, and 10 to 25 times the commonly accepted total risk of radiation-induced malignancies. (This suggests perhaps that maybe

*Some of the published papers on the induction of thyroid cancer have been of studies which involved a follow-up period of only 5 to 10 years after X-ray exposure or after radioiodine was given. If the exposed populations had been followed over the life of these persons, it is certain additional cancers would have developed. At low doses cancers take longer to develop.

Cancer Induction as a Function of Dose of Ionizing Radiation from 0 to 100 rem



This figure is a plot of equation

$$E = kD^n\% \qquad (1)$$

in which E = cancer risk (percent of persons with cancer) as a result of exposure to a dose D (rem) of ionizing radiation.

Case A, in which n = 1, illustrates the *linear hypothesis* in which one would expect 3×10^{-4} cancers per person.rem.

Case B, in which n = 2, illustrates the old *threshold hypothesis* where the cancer risk becomes negligible or statistically insignificant at low average dose per person. Perhaps it typifies the low leukemia risk of middle-aged persons that are exposed to low LET (linear energy transfer) radiation.

Recent human studies suggest that Case C, or some other curve for which n < 1, applies to leukemia among the young and the old and perhaps to most other forms of cancer irregardless of the age of the person. In such cases the risk per person.rem is greater at low doses than at high doses.

Curves A, B and C are given primarily for illustration, but each curve appears to be applicable in certain cases. Perhaps it is of interest to note that for a dose of 1 rem the cancer risk is 0.03 percent by the linear hypothesis (curve A) and 3×10^{-4} percent (negligible) by the threshold hypothesis (curve B).

the power of D in curve C should be less than one-half. But I believe a more likely explanation is that the majority of Hanford exposures were more than the average of 1 rem. Baum [23] found that the power of D which gave the best fit in a number of studies of cancer induction by ionizing radiation was n = .5.)

The estimate by Mancuso et al. of a 6 to 7 percent increased risk of cancer associated with radiation exposure among the workers at Hanford created considerable controversy. This is partly because the Hanford cancer risk coefficient is 10 to 25 times higher than assumed in the past. I believe, however, that controversy develops because many persons in the nuclear industry and in the federal agencies have been inadvisably proclaiming that there is *no* radiation risk. There probably is no occupation that is free from some risks to its workers. So if the proponents of nuclear energy had been more reasonable in their claims about radiation safety, they would not now be trying desperately to save face.

The forces in support of and in opposition to the findings of Mancuso et al. have now had their day in Congressional hearings, as mentioned above. In addition, Stewart reported at the February 1978 meetings of the American Association for the Advancement of Science in Washington on further analysis of the Hanford data which greatly strengthens the arguments in the earlier paper. Gilbert [37] of the Hanford operations made a careful study of the Mancuso et al. data to determine if, in fact, their findings were accurate. At the Conference on Low-Level Radiation on February 10, 1978, Gilbert presented a report which I interpret as supporting Mancuso et al., for at least two types of malignancies that seem to have a high risk from low level radiation exposure.

All these findings confirm the 1974 Milham report that there is an increased incidence of radiation-induced cancer among Hanford workers.

In converting from absorbed dose (given in rad in which 1 rad corresponds to an energy deposition of 100 ergs per gram) to the quantity, dose equivalent (given in rem) we use the relationship,

$$\text{Dose Equivalent (rem)} = \text{Absorbed Dose (rad)} \times Q \times N \qquad (2)$$

Q is a physical correction factor related to stopping power ($-dE/dx$) or linear energy transfer (LET). N is a biological correction factor.

As a general simplification (especially for internal dose calculations), we set $Q = 1$ for X, gamma, electron and beta radiations and $Q = 10$ for alpha radiations when they are emitted by internally deposited radionuclides. It is easy to see why Q for

The real culprit of unnecessary population dose is not the nuclear industry but the medical profession.

alpha should be much greater than the Q for X rays or for the electrons produced by X rays because the specific ionization is much greater along an alpha-track than an electron-track. For example, $S_a \simeq$ 8,000 ion pairs per micron of tissue for alpha particles while $S_e \simeq 8$ ion pairs per micron of tissue for an electron when both particles have energies of about 1 megavolt.

Thus, the difference in damage to a living cell in the two cases is like the difference in damage from a bulldozer or a rabbit running through a cornfield. Many rabbits may have to step on the same corn sprout over a short period of time to damage it (or many secondary electrons may be required near the cell nucleus in a short time to cause damage). And, thus, it is understandable why for some types of X or gamma radiation damage (for example, leukemia among middle-aged persons) curve B in the figure provides the best fit to experimental data or $n = 2$ in Equation (1) (see figure). It is easy also to see why $n < 1$ for curve C in the figure applies in the case of all forms of chronic damage from internal alpha emitters. This is because at high doses or dose rates there is "overkill," that is much of the alpha-energy is wasted, as would be the case were we to try to kill a squirrel with a cannon rather than with a rifle.

The other modifying factor, N, in Equation (1) is not as well understood as Q. When I first began using N in 1947, I thought we needed a biological correction factor to account for additional biological damage from certain internally deposited radionuclides and that this factor was related mostly to non-uniform deposition or "hot areas or spots" of radiation of select parts of critical organ tissue (for example, the endosteal or perosteal tissue of the bone). However, it was soon recognized that N related to other things as well, among which were (1) the essentialness of the tissue at risk in terms of proper body function, and (2) the relative radiosensitivity of the radiated tissue. Thus N may be an important factor also in determining whether the radiation damage function behaves like curve A, B or C in the figure and why there are marked differences in dose response curves for various types of radiation, for various animals (including man) and as a function of age, sex, genetic factors, certain diseases, etc.

This N factor may in time explain why the very young and the very old persons are most susceptible to radiation damage, and why even for X or gamma radiation curve C probably gives the best fit for cancer induction from fetal exposure and for exposure of the old. The difference in the applicable curve in the figure for some animals and for man as a function of age may be due to the fact that skeletal and bone marrow development continue rather uniformly throughout the life of some animals but not in man. In the very early life of man bone turnover is rather uniform and all the bone marrow is active, but later in life much of man's bone is less active and more and more marrow becomes inactive (yellow marrow).

Radiation biologists have conducted thousands of experiments with various types of animals in order to determine the dose effect relationships and in many cases have extrapolated these data to man (perhaps brazenly or at best with some misgivings). Some ecologists and health physicists have warned that much of this animal data may not be applicable to man for many reasons, a few of which are:

- Studies have shown that the dose response of various kinds of animals can differ by orders of magnitude in going from one species to another (for example, fly to fish to mouse to monkey to man).
- Studies have shown that even slight differences in species or strains can cause a marked change in dose response. For example, Warren and Gates [38] found very large differences in leukemia induction and in life shortening between studies with different kinds of mice. Yet, the standards are based on observations of carefully controlled inbred, healthy animals. And it is these standards which are used in animal experiments to determine carcinogenicity data for man. But man is a wild or heterogeneous animal living in many types of environment with various eating and drug habits, with many diseases and eccentricities, of various ages, etc.

It is little consolation to a mother to know that the average risk to the persons living in her community is 3×10^{-4} cancers per man rem, or 0.003 percent from an environmental dose of 100 millirem accumulated over a 10-year period from a nuclear power plant, when she learns that in fact her child with asthma has a risk of 50 times this or 0.15 percent chance of developing cancer from this exposure. It helps very little to tell the mother that natural background radiation is 100 millirem each year and this gives her child a 1.5 percent risk of radiation-induced cancer over the same 10-year period. Neither does it help to tell her that if a coal burning power plant (even an unusually clean one) were to replace the nuclear power plant, the risk from the power plant probably would go up from 0.15 to 5 percent and the primary risk would then become one of chronic bronchitis and emphysema rather than cancer. It is difficult for this mother to understand why she should risk the life of her child so that the power plant can be located at a particular river site or, as she may rationalize, so the stockholders can expect a better return on their investments.

Many perceive the solution to be reduced levels of maximum permissible exposure (MPE) for occupational workers and for the public by a factor of 10. In fact, petitions have been submitted to the Environmental Protection Agency and Nuclear Regulatory Commission by a number of citizens' organizations. However, although sympathetic, I am not convinced this would be an acceptable solution. To me this seems like putting a finger in the hole of a

leaking dike. I see it this way primarily for three reasons:

1. Our goal should be a radiation exposure that approaches zero and especially one that reduces the population dose (man.rem dose) as low as reasonably achievable (ALARA),

2. The real culprit in respect to unnecessary population dose is not the nuclear industry but rather the medical profession, and

3. A smaller reduction of occupational maximum permissible exposure—for example, from 5 rem per year to 2.5 rem per year rather than the proposed reduction to 0.5 rem per year—probably could be accomplished without threatening the option of nuclear power.

With regard to the *first* reason, it is partly a matter of education and acceptance of a moral obligation by those responsible for human exposure. For decades the average occupational exposures at the government's national laboratories (such as at Oak Ridge, Argonne, Brookhaven and Savannah River) have been kept in accordance with ALARA (as low as reasonably achievable), and the average dose has been less than 10 percent of the maximum permissible exposure (that is < 0.5 rem per year), and accidents involving large individual exposures have been extremely rare events. This, of course, does not rule out the possibility of mistakes in exposure estimates and especially the risk of greater internal dose than was measured with techniques available at the time; but at least a sincere effort was made to keep all exposures ALARA. This precautionary practice was applied to individual doses (rem) and man.rem doses of the radiation workers as well as those of members of the general public. Unfortunately, however, this is not the uniform practice throughout the nuclear industry.

I was particularly unhappy, for example, with what went on at the West Valley, New York, reprocessing plant and the Kerr McGee Oklahoma fabrication plant. I believe in these instances it was a matter of wanton disregard of ALARA and of good health physics practices. I am very much concerned about the growing practice of "burning out" temporary employees: the fact that many nuclear power plants are finding it necessary to solve the individual exposure problem of repair work in persistently high radiation exposure areas of the plant by hiring temporary employees to spread out the dose on "hot" operations. This has increased the man.rem dose or the overall cancer and genetic risks to the population, and I believe this is exactly what we should strive to avoid.

I cannot be certain of the effect of the proposed lowering of the occupational maximum permissible exposure to 10 percent of its present level (that is, down to 0.5 rem per year). Certainly, it would reduce individual exposure levels; but I fear in many cases it would just mean the hiring of more people, each to receive small doses of less than 0.5 rem per year with a marked increase in the total man.rem dose. The man.rem dose would increase for the same radiation job for two reasons: (1) inexperienced persons always get more exposure, and (2) much of the exposure on a "hot" job is received going into and away from the hot operation. Golden [39] has made some estimates of the increased man.rem dose that would result in a nuclear power plant were the occupational maximum permissible exposure to be reduced to 0.5 rem per year.

The *second* reason for my hesitation in relying on solving this problem by simply lowering the occupational MPE to 0.5 rem per year is because at present the medical professions are exempt from the recommendations suggested by the ICRP for the MPE from ionizing radiation—even though they are de-

Table 3
Values of Modifying Factors Suggested by the International Commission on Radiological Protection

Organ	*Present Value of MPE or R (rem/y)*	*Values of W_1*[a]	*New Values of MPE or R (rem/y)*[b]
Total body	5	1.0	5
Gonads	5	0.25	20
Breast	15	0.15	32
Red marrow	5	0.12	42
Lung	15	0.12	42
Thyroid	30	0.03	167
Bone	30	0.03	167
Skin	30		
Remainder	15	0.3	17

MPE = maximum permissible exposure

[a]Values in ICRP [26].

[b]Values given in this column are not given specifically by ICRP but are obtained by dividing the ICRP's value of 5 rem per year by those in column 3. It is probable also that the ICRP will set an upper limit of 50 rem per year for each of these values in making internal dose calculations.

Only New York, New Jersey, Kentucky and California require X-ray technologists to have training and certification in the proper use of X-ray equipment.

livering over 90 percent of the man-made dose.* I have shown that the dose delivered by medical diagnostic X rays could be reduced to 10 percent of its present value, which could at the same time increase the quality and amount of diagnostic information from medical radiography [40]. Only New York, New Jersey, Kentucky, and California require X-ray technologists to have training and certification in the proper use of X-ray equipment. Only California requires questions on the State Board Examinations on the effects of X rays and health physics.

Is it a wonder that those who are responsible for over 90 percent of the man-made dose from ionizing radiation ignore almost completely the principle of ALARA?

Is it surprising that studies show that the skin dose from a chest X ray from one medical facility may be 10 milliroentgens while at another it may be 3,000 milliroentgens, and yet far more useful medical information is provided by the 10 milliroentgens?

Is it surprising that less than one percent of the dentists are using long open-ended cones with rectangular collimation to conform with the rectangular dental film while the rest use a circular X-ray field for a rectangular film and most of the dentists are using a short cone?

Why didn't responsible government agencies correct these medical abuses 20 or 30 years ago, instead of carrying on endless discussions with the American Medical Association, the American College of Radiology, the American Dental Association, etc., to determine how improvements can be made without cost or inconvenience to the medical professions?

When these questions are answered. When we have stopped the unnecessary exposure of the American public to ionizing radiations—which is 90 percent of our problem. Then, perhaps, I can see that the next step might be to reduce the maximum permissible exposure to 0.5 rem per year for the occupational worker and to reduce the corresponding value for members of the population at large. Just a reduction of one percent of the present unnecessary medical diagnostic exposures in the United States would reduce the population dose of man-made sources of radiation more than the elimination of the nuclear power industry to the year 2000.

I have fought to reduce unnecessary medical exposure for over a quarter of a century but progress has been very slow. The only marked successes have been abandonment of mass chest X-ray programs, restraints on mass mammography programs and the recent limits set [41] for doses delivered in the more common X-ray diagnostic procedures. It is unfortunate, however, that the most important and effective step remains to be taken: that we require all members of the medical profession to obtain education, training, and certification in health physics.

Finally, were we to reduce the present maximum permissible exposure by a factor of 10, I seriously doubt that many of our present nuclear power plants could continue in operation. It would especially be difficult for the pressurized water reactors because of the high background radiation in the vicinity of the steam generator due to the accumulation of cobalt-58 and cobalt-60. I believe the solution to this problem is to redesign the pressurized water reactors in such a way that the precursor elements do not enter the high neutron flux region of the reactor or are prevented from circulation in the primary cooling water. Also, more room must be provided in certain areas for shielding and the use of remote control equipment, remote TV cameras, etc. There should be some tightening of the measures to reduce occupational exposures in the nuclear power plants that are now in operation; but the major effort in this regard should be with those power plants that are now in the design stage.

The Nuclear Regulatory Commission took the bold and commendable step of setting the dollar cost of man.rem at $1,000 at a time when others [42] were suggesting a value as low as $10 per man.rem. Although most of us probably recoil from the thought of setting a dollar value on a human life, in the practical world we must recognize there may be no other alternative. Using an overall risk coefficient of 6×10^{-4} cancers per man.rem, we recognize this $1,000 per man.rem corresponds to $1.7 million per cancer. In other words, the Nuclear Regulatory Commission presumably applies some pressure to have the nuclear power industry eliminate a source of one man.rem of occupational exposure to ionizing radiation if the cost does not exceed $1,000. To put this more bluntly, the plant should spend as much as $1.7 million to prevent an employee from developing cancer.

One of the most unfortunate recent developments in the setting of standards for exposure to ionizing radiation is a recommendation of the International Commission on Radiological Protection, which was published in 1977 [26]. Their report recommended weighting factors, W_i, which I interpret may result in large increases in the present ICRP values of maximum permissible exposure (MPE) and in all values of total body burden and maximum permissible concentrations (MPC) in air, water and food for radionuclides except where they are rather uniformly distributed throughout the body. Table 3 summarizes these values.

I consider this report a retrograde step of the Commission because it comes at a time when their own internal reports [19] emphasize that the cancer risk is many times what we considered it to be 15 years ago. This change was made understandably in an effort to remove the inconsistency that the maximum permissible exposure for total body has been the same as that for gonads and red

*The Commission does not suggest any MPE's for diagnostic procedures. It says the physician must weigh the benefits against the risks from an X ray for the patient, and yet most doctors could not define the rad or explain the risk of radiation- induced cancer.

Many nuclear power plants solve the problem of repair work in persistently high radiation exposure areas by hiring temporary employees to spread out the dose on "hot" operations.

marrow. What the Commission should have done is set the maximum permissible exposure at 5 rem per year for gonads and red marrow and the maximum permissible exposure for total body at some value less than 5 rem per year, for example, 2.5 rem per year.

Given that we are beginning to recognize that radiation risks are greater than we used to consider them, I sincerely hope the National Council on Radiation Protection, the BEIR (Biological Effects of Ionizing Radiation) Committee, the Nuclear Regulatory Commission, the Environmental Protection Agency, etc., in this country will strongly object to this move of the Commission and will reject these new values which would tend to increase the internal dose from radionuclides deposited within the body. In conclusion I suggest the following actions:

- Reject proposals at this time to reduce the maximum permissible exposure by a factor of 10; but consider the possibility of reducing it by a factor of 2.
- Consider the feasibility of reducing the maximum permissible exposure by a factor of 10 at some later date if it can be shown that all unnecessary exposure (especially medical) can be reduced and that there will be a net benefit to mankind by such action.
- Take immediate measures to reduce the man.rem dose. This could be accomplished in several ways. For example, in the nuclear energy industry a limit of 500 man.rem per 1,000 megawatt (electrical) years might be set for presently operating plants and those under construction, and 200 man.rem per 1,000 megawatt (electrical) years for plants now on the design board.
- Take bold steps to reduce unnecessary exposure from medical sources of ionizing radiation. In February 1978 the Environmental Protection Agency and the Bureau of Radiological Health (FDA) made some encouraging progress [41] in this area; but we still have a long way to go.
- Apply the principle of ALARA—as low as reasonably achievable—in all areas of exposure to ionizing radiations and apply it to all hazardous agents, including, for example, non-ionizing as well as ionizing radiation, and chemical agents.
- In making the choice of fuel for a central power station consider all the risks and all the advantages of each type of fuel. In this evaluation keep in mind that exposure to ionizing radiation is only one of the risks, and in many cases the risks of chemical exposure may be far greater than those of radiation. (Don't forget there is a serious exposure from radiation in the burning of coal, radium-226 and 228; radon-222, etc.)
- Give adequate support to research programs designed to define more accurately the risks from human exposure to ionizing radiation.

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