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Publication Date
1976-05-01
REDUCING PATIENT EXPOSURE TO IONIZING RADIATION — IS IT REALLY NECESSARY?

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May 1976

Prepared for the U. S. Energy Research and Development Administration under Contract W-7405-ENG-48
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The thesis that medical radiation exposures to the general population in the United States are excessive and that urgent steps need to be taken to reduce them is examined. At the present time, no evidence is found that deleterious effects result from radiation exposures at the level of a few rads or less. Nevertheless it is prudent that we assume the possibility of harmful effects of radiation exposure. However, because medical radiology is clearly so beneficial, and the harmful effects are problematical, it is doubtful whether the public


† The opinions expressed are those of the authors.
should be alarmed by the suggestion of any crisis. A balancing of risks and benefits in the medical uses of radiology supports this latter contention.
INTRODUCTION

In the early 1950s, the assumption originated that any radiation exposure may carry with it some detriment. At that time, it became necessary to obtain quantitative estimates of the maximum influence on public health of radiation from fallout resulting from the testing of nuclear weapons in the atmosphere.

"A linear non-threshold model was specifically chosen on a basis of mathematical simplicity and prudence to represent the upper limit of risk in the low-dose domain, for somatic radiobiological effects which had been observed only in a higher-dose domain. The linear non-threshold model was not based on radiobiological data for somatic effects in the low-dose domain.

"As originally introduced, care was always taken in protection committee reports to point out that the true risk in the low-dose domain would be expected to lie between zero and the upper limit given by the linear non-threshold approximation."

Unfortunately there is an increasing tendency to cite risk estimates based on these assumptions as actual deaths. When it is believed that deaths actually result from the radiation exposure of large populations at low levels, then it may appear desirable to reduce all sources of human radiation exposure including natural background.
Figure 1 shows a summary of the major sources of human radiation exposure, at the present time and as predicted by the Environmental Protection Agency until 2000 A.D.\(^2\) The dominant roles of natural radiation and the medical uses of ionizing radiation are evident. It is frequently pointed out that diagnostic radiology contributes the most to man-made radiation exposures.\(^3\) However, "Not only is this true, . . . but it would be surprising if it were not true. Medical radiology is the only legitimate situation in which radiation exposure is purposefully given to human beings for their own benefit. It would be more remarkable, if not alarming, if any other man-made source exceeded it in human exposure."\(^4\)

Although it has not yet been definitely established that harmful effects will result from the uses of ionizing radiations in diagnostic radiology, there are some who have suggested there is an urgent need to reduce medical radiation exposures.

Such arguments have recently been given credence by the support of Professor K.Z. Morgan\(^5\) — a scientist of international stature, whose contributions to the development of health physics are well known — and, therefore, deserve serious consideration.

Morgan's argument is based on five points:

a. Medical exposures in the United States are higher by a factor of 2 to 10 than in most advanced countries in the world.

b. There is no threshold to the deleterious effects of radiation.

c. Linear extrapolations of biological effects observed at high radiation doses may underestimate radiation effects at low doses.
d. Despite current concern as to the somatic effects of radiation, the genetic risks may still be the limiting form of radiation damage.

e. Low level exposures such as those in medical diagnosis may be harmful.

It is certainly true, as Morgan says, that medical exposures in the United States are higher than in any advanced country in the world. However, the mere fact of exposure does not of itself establish any detrimental health effects. In fact, it is true that we have no generally agreed evidence to demonstrate that exposure to x and γ radiation below tens of rads are harmful to humans. Furthermore, it is unlikely that epidemiological studies will convincingly demonstrate the existence of detrimental effects because of their low probability and the large populations that would consequently have to be studied.6

In the absence of human radiobiological data at low dose, a linear, non-threshold model for radiation effects has been used to obtain a crude upper-estimate of radiation risks for radiation protection purposes. However, over the years there has been a subtle metamorphosis, in the minds of many, from the use of this model for public health purposes, to the unqualified use of this model to calculate actual deaths.7 An example of such a calculation is the assumption by Rowe, of the Environmental Protection Agency, that natural background causes 13,000 "health effects" per year in the United States.8
Unfortunately, such a model is often used to justify administrative policies controlling the uses of radiation, or to compare the risks of alternative procedures which result in radiation exposure and others which do not. A caution is to be urged in properly interpreting such analyses — particularly when there is little or no knowledge of the risks resulting from alternative procedures.

Morgan's thesis that medical radiation exposures should be reduced does not attempt in any way to balance the possible risks that may result from radiation exposure with the benefits that do occur from the medical uses of radiation. There are undoubted benefits from medical radiology, and great social harm might follow if the general public became unduly alarmed over its possible risks without due consideration of its advantages.

In this paper we will discuss briefly the question of whether there is a threshold to radiation effects, the dose-effect relationship at low doses, and the genetic risks of radiation exposure. Where possible we will employ the data used by Morgan. Finally, we will attempt a crude risk-benefit analysis of the medical uses of radiation.
THE DOSE EFFECT RELATIONSHIP

If there were indeed a threshold dose below which deleterious effects did not occur, our discussions would be greatly simplified.

Thresholds. We know that some radiation effects do exhibit a threshold, for example, skin erythema, cataractogenesis, and the symptoms of the acute radiation syndrome. The pertinent question is, do all radiation effects exhibit a threshold?

Morgan suggests, "During the past 25 years, however, there has accumulated a preponderance of evidence which indicates there is no safe threshold dose and in fact both experimental and theoretical evidence seem to indicate there is no dose or dose rate of ionizing radiation so low that the risk of radiation damage is zero." In support of this view Morgan cites data of Rowland\textsuperscript{9,10} on the incidence of bone sarcomas and carcinomas in radium dial painters (Figure 2). The curves drawn through the experimental points cannot — as Morgan suggests — indicate any radiation effects at low doses, because the curves should properly pass through the origin. (There is, in fact, a point at \(\sim 50\) rad indicating zero incidence of bone sarcoma.)

Morgan's suggestion that these data do not support evidence for a threshold for radiation effects at an absorbed dose of \(\sim 800\) rads is not supported by Rowland\textsuperscript{9,10} nor by Evans\textsuperscript{1,11} who has also made an extensive study of the incidence of cancer in the radium dial painters.
These studies represent one of the few pieces of chronic human exposure data we have. Figure 3 shows the observed tumour cumulative incidence in epidemiologically suitable cases as a function of average skeletal cumulative rad dose as reported by Evans et al.\textsuperscript{1,11} Evans, in fact, hypothesises that his data may show evidence of an effective threshold. Figure 4 shows a plot of tumour appearance time versus average skeletal cumulative rads. There is a suggestion that the time for tumor appearance increases as the dose decreases. When the tumor appearance time is larger than the life expectancy for an individual, an "effective threshold" exists. Jones and Grendon\textsuperscript{12} have given some theoretical support for such an observation. In the analysis of data on dose of a carcinogen versus the latent period between exposure and incidence of cancer, they find that, for all carcinogens examined, including radiation and a number of chemicals, and in a variety of mammalian species, including man, the relationship between latent period, \( L \), and dose, \( D \), can be expressed as: 

\[
L = cD^{-x},
\]

where \( x \) is generally close to 3. A biological model has been developed to account for this similarity of behavior among such divergent carcinogenic agents. If the generalization proves valid, it will justify the use of "practical thresholds" in estimating the effects of radiation and other carcinogens at low levels. Dinman\textsuperscript{13} has persuasively agreed that stochastic determinants impose a lower limit on the dose-response relationship between cells and chemicals and one would assume similar arguments applied to cellular-radiation effects.
The observation of what appears to be a practical threshold for the radium dial painters does not, of course, settle the issue for all radiation effects. In public health matters prudence is necessary, but — contrary to Morgan's view — our experience with the radium dial painters tends to suggest that we have indeed been prudent. These data will again be referred to in the discussion of the dose effect relationship.

Form of the Dose-Effect Relationship at Low Absorbed Doses. Generally in internal organs the absorbed doses from medical exposures are much lower than ten rads. There are no convincing data to show that exposures to x- and γ-rays at this intensity are harmful to humans. Two sets of human exposure data — the Hiroshima and Nagasaki victims and the in utero exposure of children — are claimed by some workers to indicate harmful effects at doses below 10 rads but there is no general agreement here.

Nagasaki and Hiroshima Bomb Victims. It is claimed that the data on incidence of cancer and leukemia in those exposed to radiation from the Hiroshima and Nagasaki nuclear weapons explosions give support to the linear non-threshold model of radiation effects and that a significant elevation in the incidence of leukemia is observed at doses as low as 20 rads. Such claims are not, however, supported by reference to the original sources.

Analysis of the Japanese survivor data is difficult, and this difficulty is compounded by the difference in radiation fields produced by the two weapons. At Hiroshima the influence of a substantial absorbed dose caused by neutrons has made dose estimates and
interpretation difficult. At Nagasaki, however, the situation is somewhat simpler in that the neutron contribution to absorbed dose was smaller and "wide ranges in the assumed neutron potency factor cause only a small variation (± 10%) in the calculated risk rate coefficient for Nagasaki."\(^\text{16}\)

Figure 5 shows the estimated annual leukemia deaths per \(10^6\) persons for the exposed population at Nagasaki during the period 1950-1970. Mays et al. conclude:

"The non-linear appearance of the plotted dose-response curve for Nagasaki raises reasonable doubt on whether the dose-response is really linear (Fig. 5). Among the 4931 persons exposed at Nagasaki to \(10^{-99}\) rads, 7.2 total cases of leukemia are predicted (4.7 natural plus 2.5 induced according to the "preferred" linear estimate), whereas only 2 leukemia cases were actually observed. A linear relationship predicting 7.2 cases when only 2 were observed is rejected significantly (\(P = 0.03\)). An excellent fit to the Nagasaki incidence rate is made by the fitted dose squared relationship of 0.003 induced leuk. per year/\(10^6\) person rem\(^2\), starting at a natural incidence rate of 52 leuk. per year/\(10^6\) persons, and assuming an average neutron potency factor of 9. This dose squared relationship will be used to provide alternative estimates of risk.

"Now, the lifetime risks will be estimated for leukemia induced by total body \(\gamma\)-ray irradiation at high dose-rates (10-1000 rem/min) such as received by the A-bomb survivors. Assuming the average death rate from induced leukemia was the same
in the unobserved interval 0 to 5 years after irradiation as
in the observed 5 to 25 year interval, the total incidence
during the first 25 years following irradiation based on the
preferred linear model would be \((25 \text{ yr})(0.8 \text{ leuk per yr/10}^6 \text{ person rem}) =
20 \text{ leuk/10}^6 \text{ person rem}\). Based on present trends about 80% of the
lifetime leukemia risk should be expressed at 25 years. Therefore,
the preferred linear estimate for the lifetime risk from leukemia
is \((20 \text{ leuk/10}^6 \text{ person rem})/0.8 = 25 \text{ leuk/10}^6 \text{ person rem}\).
The higher and lower linear estimates and the dose squared estimate
were calculated similarly, and are shown in Table 1.\(^\text{16}\)

In Utero Exposures. Stewart\(^\text{17,18}\) and other authors\(^\text{19-22}\) have
published studies on the incidence of leukemia and other cancers in
children who were irradiated in utero incidentally to diagnostic
procedures performed on the mother. Some of these authors interpret
their data as suggesting an elevation in the incidence of leukemia
and cancer in the irradiated group. For example, the Oxford study of
Stewart and Kneale\(^\text{18}\) found an increase of \(\sim 50\%\) per rad in the
exposed group as when compared with the non-exposed group used for
control. They also suggest their study is consistent with a linear
dose-effect relationship.

These suggestions, if confirmed, would be of great importance
in public health because they would provide evidence of deleterious
effects to humans at dose levels of a few tenths of a rad. However,
the conclusions drawn from these studies are not generally agreed upon.
Stewart herself has drawn attention to the fact that several prospec-
tive studies\(^\text{17}\) do not show such an effect. Jablon and Kato\(^\text{22}\) have
indicated inconsistencies between the Stewart data and those of the Atomic Bomb Casualty Commission (ABCC) studies of the Hiroshima and Nagasaki bomb victims, where no elevation in leukemia or cancer in children irradiated in utero is found. Jablon,\textsuperscript{23} in a later paper, speculated that "some of the relationships that have been reported reflect, in reality, selection of gravida for x-ray." More recently Oppenheim et al.\textsuperscript{24} in a prospective study of 1000 children irradiated in the course of routine pelvimetry found no conclusive radiation effect. However, they found several possible sources of error in such studies — particularly in the improper choice of control groups. They suggest that the reported radiation effects may "have been due to biases introduced in the selection process, rather than to the radiation itself." In addition, Shore et al.\textsuperscript{25} have disputed Stewart and Kneale's claim that their data are consistent with a linear dose relationship. When the Stewart data are divided into five year periods, the average case/control ratio fluctuates. For the most recent period (1960-1965) the case/control ratio was $-0.05 \pm 0.26$, indicating no significant elevation of cancer incidence.

There is, then, no general agreement as to whether irradiation of the human foetus to $\sim 1$ rad does produce an elevation in observed cancer or leukemia rates. This is due to the intrinsic difficulty in drawing conclusions from epidemiological data:
"As it happens, it is almost always possible to find flaws in surveys (not excluding the Oxford survey). By the very nature of the thing, in a survey we do not have experimental control. We cannot guarantee that two groups of people are wholly comparable save with respect to the one factor that we wish to study. We cannot regard any single survey, no matter how carefully done, as the equivalent of the large, well-controlled experiment that, ideally, we would prefer. We therefore invoke what is, I suppose, a new principle: If many surveys, none perfect, but conducted in diverse contexts, all point to the same conclusion, then that conclusion is likely to be right. So it has been with the issue of smoking and lung cancer; and similarly, after the first report from the Oxford Survey, a number of investigators, working in differing settings and using varying materials, have studied the question, but here the results have not been unanimous."^26

**Bone Cancer Data.** Morgan states that Rowland's bone sarcoma data show "no suggestion of a deviation from the linear hypothesis." We have already suggested that the data of Rowland are clearly non-linear and may even suggest evidence for a threshold.

Gofman and Tamplin^27 have suggested that the bone cancer data of Evans are consistent with linearity, but the chance that this is so is $100,000,000$ to $1$ against."^11
Morgan claims in his paper that "when all is done, it may be that the genetic risk as claimed by Muller is far greater than the somatic risk from exposure to ionizing radiation."\(^5\)

"Well established genetical theory requires that the net effect of induced random mutations in an outbreeding population must be to reduce the average fitness. Such an effect has not proven to be demonstrable in mammalian populations, perhaps in part due to the difficulties in measuring parameters of fitness."\(^{28}\)

"Radiation does indeed cause mutation. But at very low dose rates (and sometimes at high acute doses), the mutant alleles can be absorbed or eliminated from the pool of already existant genes. The rates of elimination or adjustment are higher than previously thought; the extent of the genetic damage is lower than previously thought. Most of the mutant individuals we see arise from genes maintained in the population by forces independent of the classical mutation-input, selection-output balance. Increases in the radiation-induced mutation rate, within fairly large limits, might be expected to have little overall effect on the human population, averaged over the entire population and averaged over time. Transient effects would still be seen, sometimes severe in isolated cases."

"It has not been difficult to demonstrate recessive mutational damage in mice or flies. Dominant lethal mutation — inferred from the reduced fecundity of irradiated individuals or from the
rate of early death among their immediate offspring — has also been observed. The class of mutational damage that has not been observed in accordance with expectation is heritable net dominant deleterious damage. The 1961 symposium "The Effects of Radiation on the Hereditary Fitness of Mammalian Populations," published as Vol. 50, No. 5, Part 2 of Genetics, November 1964, consisted almost entirely of reports of negative findings." 28 Newcombe 29 has recently discussed estimates of genetic risks to man from radiation exposure.

"Conventional risk estimates have tended to assume that a rather large fraction of the normal load of hereditary disease is maintained in the population by repeated mutations, and would increase in direct proportion to any artificial elevation of the mutation rate. It is proposed here, however, that this assumption leads to an over-estimation of the risks by more than 10-fold. The numerous and important irregularly inherited diseases, like diabetes and cleft palate, are probably not mutation-maintained. Only the simple dominant diseases would be expected to increase in direct proportion with the mutation rate, but these appear from new data to be rarer than has been supposed, affecting only about 0.1 individual per 100 born alive instead of 1 per hundred as previously believed. A revised estimate of risk suggests that a population exposure of a million man-rem probably results in no more than 20 additional causes of hereditary defects."
Thus, Morgan's claim that "when all is done, it may well be that the genetic risk as claimed by Muller is far greater than the somatic risk from exposure to ionizing radiation" is perhaps contrary to the most recent genetic thinking.

SUMMARY AND CONCLUSIONS

It is not clear from his paper whether Morgan believes that the incidence of cancers in man is proportional to absorbed dose or whether the linear hypothesis underestimates dose effects. In support of the linear hypothesis, Morgan cites the bone cancer data of Rowland and the foetal irradiation data of Stewart. That the Stewart data support the linear hypothesis, or even that they show any real effect at all, is — as we have already discussed — disputed by several workers.

In an analysis of the measured incidence of mammary neoplasms in Sprague-Dawley rats, Rossi and Kellerer find that although a linear relation between incidence and x-ray dose might be consistent with available data, such a relation would be fortuitous and linear extrapolation to lower doses is unjustified. They conclude:

"With the complexity of the tumor induction process established, there remains little justification for linear extrapolations, and this conclusion, in turn, removes apparent inconsistencies between the dose-effect relation postulates and histological evidence to the effect that carcinogenesis requires the transformation of several contiguous cells. There is, however, at present insufficient evidence for numerical estimation of tumor incidence based on linear or other extrapolations."
Morgan implies in his paper that as our knowledge of radiation effects increases it will be shown that our present estimates of the risks of radiation exposure are not upper limits but may not be sufficiently conservative. Morgan claims, "During the past few years, however, many studies have indicated that this probably is not true in general and that at very low doses and dose rates somatic damage per rad probably is usually greater than would be assumed on the linear hypothesis." This claim is contrary to the data of Evans, Rowland, and Rossi and Kellerer already discussed in this paper. Current thinking tends to support the view that estimates of cancer incidence at low doses obtained by linear extrapolation of data taken at absorbed doses of 100-500 rads will overestimate the low dose incidence by a factor of at least 2-3. However, as we have attempted to show, there is no generally agreed upon evidence that directly indicates detrimental effects to humans following x- and y-ray exposures of a few rads. The best data that we have on chronic radiation exposure of humans (the radium dial painters) suggest the existence of an effective radiation threshold for the incidence of bone cancers. From study of the Nagasaki survivors we infer a non-linear dose-effect relationship for leukemia incidence. Current evidence suggests that the genetic hazards of radiation exposure are lower than has been hitherto assumed. All these are encouraging signs suggesting that, as Morgan has written elsewhere:
"Perhaps there has never before been an enterprise that was planned so carefully for its safety and never before a risk that has been so thoroughly studied and guarded against as has been the case with the nuclear energy industry and its concern to avoid unnecessary exposure to ionizing radiation."31

It is useful to remind ourselves from time to time that:

"... we need to come down to earth at this point. These exercises in risk writing and risk estimating and balancing against benefits are very useful exercises for determining what might be the upper limit of risk, but I am afraid that we are getting to the stage now where we are believing that the risks actually occur. We must remind ourselves that the risks of radiation at the dose levels that we are talking about have not in fact been demonstrated at the present moment. They have been extrapolated from high levels and we use them as a basis for estimating what might be the maximum risk, but the actual risk might be zero."7

It would indeed be unfortunate if our concern over the hypothetical detriments were to result in public abstinence from the undoubted benefits of medical radiology. We may obtain a lower limit to the benefit-risk ratio of the diagnostic uses of ionizing radiation by computing the ratio of lives saved to deaths caused, n, given by:

\[ n = \frac{f}{RD} \]

where \( f \) = fraction of x-rays taken which result in life saving.

\( R \) = risk of producing a radiation death per rad.

\( D \) = average annual whole-body dose due to diagnostic procedures.
Chamberlain has given data for hospitals where he is Director of Radiology in which 200,000 diagnostic radiological examinations are performed per year. These examinations, in his view, are "either the vital factor, or a major one, in saving tens of thousands of lives per year." This would put the value of $f$ at $\sim 10^{-1}$. Taking the risk of cancer induction from radiation exposure as $\sim 10^{-4}$ per rad and an upper value of 0.1 rad/yr per person due to diagnostic radiology, we then find $n \sim 10^4$. There are few activities in life that are ten thousand times more beneficial than harmful! There is no indication of urgency here for a program of exposure reduction.

Nevertheless, we do not wish to leave the impression that we are opposed to reductions in patient exposure where these may be achieved at no significant loss of benefit to the patient and at reasonable cost. Many of Morgan's suggestions for patient exposure are eminently sensible provided they may be achieved at reasonable costs and without causing public apprehension.

The best view we can take of Morgan's thesis is — in terms of Scottish Law — "Not Proven."
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This work was done with support from the U.S. Energy Research & Development Administration.


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to Plutonium and other Transuranium Elements," American Industrial
<table>
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<td>Higher Linear Estimate</td>
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<tr>
<td>Dose Squared Estimate</td>
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TABLE I

Lifetime Risk from Leukemia at High Dose-Rates
(from a total body γ-ray dose "D")
FIGURE CAPTIONS

Figure 1 - A summary of the major sources of human radiation exposure predicted by the Environmental Protection Agency until 2000 A.D.

Figure 2 - The incidence of bone sarcomas and carcinomas plotted as a function of the median value of the total skeletal dose in rads. (From Rowland, Ref. 9.)

Figure 3 - The observed tumour cumulative incidence or occurrence in an epidemiologically suitable group of radium dial printers (from Evans, Ref. 1.).

Figure 4 - Log-log presentation of tumor appearance time versus average skeletal cumulative rads (CR) for radiogenic tumours (from Evans, Ref. 1.).

Figure 5 - The leukemia deaths (expressed as deaths per million people) observed among the Nagasaki survivors, plotted as a function of kerma equivalent (rem). (A neutron RBE of 9 has been assumed.) The error bars show ± one standard deviation. The darkened line shows a linear dose-effect model and the solid line a dose squared model. (From Mays et al., Ref. 16.)
Fig. 1
Fig. 2
Fig. 5

NAGASAKI LEUKEMIA RATES (1950-1970)

ANNUAL LEUKEMIA DEATHS PER 10^6 PERSONS

KERMA EQUIVALENT (REM)
ASSUMING NEUTRON POTENCY = 9

Dose Squared
Linear Model

0.003 LEUK./YR.
10^6 PERSON REM^2

0.8 LEUK./YR.
10^6 PERSON REM

XBL. 7512-9837
This report was done with support from the United States Energy Research and Development Administration. Any conclusions or opinions expressed in this report represent solely those of the author(s) and not necessarily those of The Regents of the University of California, the Lawrence Berkeley Laboratory or the United States Energy Research and Development Administration.