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A 1980 REASSESSMENT OF THE HEALTH HAZARDS OF LOW-LEVEL IONIZING RADIATION

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Abstract

A decade ago the risks of leukemia from exposures to low levels of ionizing radiation were estimated by linear extrapolation from data on persons exposed to much higher levels. In recent years, however, a number of scientific studies have reported excess risks where the data was on persons actually exposed to low-level radiation. The new findings are incompatible with the estimates based on the Linear Hypothesis although these estimates continue to be used in public health. Fifteen studies involving low-level nuclear radiation and ten studies involving diagnostic radiation are listed and briefly described. Most of these studies have positive qualitative findings but a few also have quantitative estimates of risk such as doubling doses. The qualitative findings would be extremely unlikely at the estimated exposure levels (which represent average exposures well under 5 rads or rems) if the extrapolative estimate of over 100 rads of the Federal Interagency Task Force Report were correct. The quantitative estimates from the data on persons exposed to low-level radiation give doubling doses in the vicinity of 5 rads and are also incompatible with the extrapolative estimates. The failure of the Linear Hypothesis to fit the new facts seems to reflect a greater efficiency-per-rad in producing genetic damage for the low-dose range than for the high-dose range.

1. INTRODUCTION: THE REASSESSMENT OF RISKS IN 1980

In the past, the assessment of the hazards of low-level ionizing radiation has been carried out by large, federally-sponsored committees or task forces. Hence this might appear to be too formidable a task for one person without federal funding to carry through. However, in 1980 there are several reasons why such a reassessment is both feasible and desirable. It is desirable because official panels funded by the government are in a conflict of interest situation since findings on radiation hazards would have immediate impact on federal agencies. Some agencies have actively promoted radiation technologies and others are involved in legal claims such as those of servicemen at the Big Smoky nuclear weapons tests. Under these circumstances, some recent official reports lack credibility.

While the reassessment is not an easy task for one person, there are several factors that make such a review feasible in 1980 when it might not have been feasible earlier. The main reason why the task has become feasible is the that there are now a series of scientific studies which are directly relevant to the crucial public health issue, the health effects of exposures in the vicinity of 5 rem or less. For the first time there are facts on the occurrence of leukemia and other diseases in populations actually exposed to these low levels of ionizing radiation. The new facts complicate the assessment since they contradict the earlier findings but they greatly simplify the task in other ways.

When there are reliable facts that can give direct answers to questions about low-level radiation hazards without guesswork, there can

be no scientifically valid reason for bringing in obsolete, less relevant data and using extrapolations that are mostly guesswork. Most of the evidence that was the basis for the earlier assessments, the animal data or the high-dose human data, can be omitted from a 1980 assessment without any serious loss. While this facilitates the assessment here, it creates difficulties for the official panels by creating another kind of conflict of interest. No panel scientist can easily acknowledge that his area of expertise or his lifework has become irrelevant to a 1980 reassessment of radiation hazards.

Finally, a consensus of opinion of a large panel may be one way of striving for objectivity when the facts are lacking but when there are directly relevant facts at hand, objectivity is achieved by looking at these facts and by disregarding subjective opinions or interpretations. This is what will be done with for more than a score of new biostatistical-epidemiological reports of health effects in populations exposed to low doses of nuclear radiation or medical x-rays.

Yet another reason why assessment is easier today is that there have been major scientific advances in our understanding of the causes of human cancer, in the area of carcinogenesis, in the past 20 years. Despite the impression created by the traditional mystique of cancer research, the fact is that we now know the immediate cause of radiation-induced cancers and probably all human cancers. The first event in the long evolutionary biological process that ends with death from leukemia or other cancer is the occurrence of a biochemical lesion or a break-point in the complex chemical structure of the DNA in the

genetic material of a human cell. This break-point may be inherited from a parent as genetic damage, or it may be produced by radiation, chemicals, or biological materials in the environment. We now know that this genetic degradation is the cause of cancer and some other chronic diseases. Hence, although the type and circumstances of the radiation exposures are different in the score of positive reports, the underlying process of carcinogenesis is the same in all of them.

Finally in 1980 it is possible to narrow the question to a specific quantitative evaluation of the health hazards. The issue today is not whether there is a hazard but how much of a hazard there is. While various measures have been used, the technical concept that is probably most easily grasped is the doubling dose. The health effect that shows up most clearly is the occurrence of leukemia. Hence, the crucial question in the reassessment reduces to a very specific one: What is the doubling dose for leukemia in men?

While this focus may seem overly narrow, the official position of the federal agencies stands or falls on the answer to this question. Moreover, the bulk of new factual evidence directly relates to the doubling dose of leukemia. The doubling dose estimate can also be directly related to official standards such as the 5 rem per year permissible exposure to nuclear workers set by the Nuclear Regulatory Commission. Thus if, as was claimed in recent federal reports, the doubling dose were over 100 rem, the standard is defensible. On the other hand, if the doubling dose is less than 5 rem then NRC is permitting a dangerous exposure. No other carcinogen is permitted at levels close to a doubling dose for cancer in humans.

Finally, one might ask: Why focus on leukemia instead of some other disease? Other cancers and other diseases can be considered. However, there are three strong scientific reasons for the focus on leukemia. First, leukemia is generally acknowledged to be a radiogenic disease. Second, it is often our earliest warning, since it can start occurring in around seven years whereas the solid cancers tend to take twenty years. Third, it is probably our clearest indicator of genetic damage. Therefore other health risks can be predicted from the leukemia risks. Leukemia is, of course, a serious health hazard in its own right, but it is particularly important as our best early warning system for other hazards.

2. THE RIVAL RISK HYPOTHESES: THREE THEORIES OF LOW LEVEL RISKS

Putting the question in the form "What is the doubling dose for leukemia?" allows a relatively clear and simple statement of the three hypotheses that are involved in the current controversy. The doubling dose can be calculated from the relationship between, say, dose in rems and relative risk of leukemia for a given dose, from what is generally called a dosage response curve. The rival hypotheses can be represented as three curves on the graph for the dosage response curve. The three theoretical curves are shown in Figure 1.

INSERT FIGURE 1

The three rival theories are shown as curves A, B, and C in Figure 1. They are:

(A) The original threshold hypothesis which was probably the most popular view in 1960 and which supported the official doctrine that "Low-level radiation is harmless". This curve is shown as a heavy dotted line that goes down to the x-axis at some point, say above 5 rem. According to this theory there would be no risk at dosages below the point where the curve intercepts the horizontal axis.

(B) The linear hypothesis which was probably the most popular view in 1970. It is the theory adopted in the 1972 BEIR report and in the 1979 update which takes the same approach. This curve is a solid straight line in Figure 1. When the dosage response curve plots excess radiation (in addition to background) versus excess risk of leukemia, the straight line should go through the point where the x-axis and y-axis intercept. The linear hypothesis is an irreplaceable assumption for all of the estimates in the BEIR report since the actual data is on persons exposed to higher dosages of radiation, generally over 100 rads. Linear extrapolation must be used to estimate the risks at the low levels, generally under five rads, which are the critical levels for the public health problems from both nuclear and medical radiation.

(C) From a public health standpoint the worst possible curve is the one which arises with what might be called a genetic degradation hypothesis. This curve is the light dotted line that bends off above the straight line at the lower doses. It will be argued that this is the hypothesis that fits the facts that are available in 1980. We now

have information on leukemia risks in groups which were actually exposed to low-level radiation. Hence, estimates of risk can now be made directly from the data without the strong assumption of the linear hypothesis.

The difference between the three rival hypotheses can be expressed very simply in terms of the notion of excess risk-per-rad. The linear hypothesis assumes that there is a constant risk-per-rad--the risk being the same at high doses as at low doses. The threshold hypothesis assumes that the risk per rad is less (or vanishes entirely) at low doses. The genetic degradation hypothesis assumes that the risk per rad is greater at low doses than at high doses. The rationale for this hypothesis is that at low doses, chances are that there will be one break-point produced or none at all. At high doses, however, multiple break-points are produced. This heavy damage blocks the cellular reproduction needed to produce the cancer. It therefore "wastes" the break-points and results in a lower risk per rad at higher doses.

3. TESTING THE HYPOTHESES: QUALITATIVE TESTS

Modern science began with the Galilean Rule: A theory must fit the facts. So the first step in the 1980 reassessment of radiation risks is to determine how well each of the three rival theories fits the epidemiological facts that are now available. In principle, the best test would be a quantitative one: A dosage response curve for the range around five rads would be constructed from actual data on persons exposed to radiation in this range and this actual curve compared

directly with the theoretical curve. This will be done in a later section. However, the quantitative tests are more complicated, and we may start with the simpler qualitative tests of the three hypotheses.

The reason that qualitative tests are feasible here is that there is an enormous difference between the estimates from the linear hypothesis and the estimates from the genetic degradation hypothesis. The latter, as will be seen later, gives an estimate of the doubling dose that is probably less than five rads (25). The official estimates, such as those in the latest Federal Interagency Task Force Report (2), put the doubling dose at over 100 rads. With one estimate more than 20 times another, even a qualitative approach can indicate which estimate fits the facts and which does not. The threshold hypothesis is easily distinguished from both other hypotheses since it implies an infinite doubling dose at low doses.

If the doubling dose were over 100 rads or if it were infinite, then the effects of doses between 100 millirads and 10 rads, in what will be called the <u>one rad range</u>, would be negligible. My testimony of March 6, 1979, to the Senate Government Affairs Subcommittee on Energy, Nuclear Proliferation, and Federal Services in Washington, D.C. (3), began by noting this point:

"Three years ago it was widely believed by the self-styled radiation protection community that it would be impossible to detect any health effects in studies of people exposed to dosages in the one rad range. At that time, Tom Mancuso and I were the only ones doing large-scale epidemiological studies to look at these hazards. Two years ago I

predicted that if scientists would only try to look at populations with exposures in the one rad range they would find, as we did, that there are serious health hazards. Since that time more than half a dozen new studies have looked at what happened to persons exposed to nuclear radiation in the one rad range and have reported positive results. These are the studies that I want to try to put together.

In ten minutes I cannot hope to go into details on all the studies, the criticisms of these studies that have been made by the members of the radiation protection community who wrote the interagency report, or the answers to these criticisms. Very briefly, there are three kinds of studies of nuclear radiation hazards at the one rad level. The first kind deals with persons who were exposed to fallout from the nuclear weapons testing of the cold war era. This includes studies of the servicemen at Big Smoky and other tests. There are also the after-effects on adults and children in the areas of Utah downwind from the tests. The second kind of study involves occupational exposures. This includes studies of the workers at the Hanford reprocessing plant and at the Portsmouth Naval Shipyard. The third class of study involves exposures to nuclear wastes such as the uranium tailings or releases from power plants. Depending on what is counted, there are now between half a dozen and a dozen positive reports of hazards to persons exposed to nuclear radiation in the one rad range. It is virtually impossible that they are all false alarms."

This testimony involved an early draft of the Interagency Report, commonly called the Libassi Report, but the bibliography of the final version (2) will be used here.

More specifically, the final version of the Libassi Report cites five references for the hazards of nuclear radiation from fallout if thyroid cancer is also considered (4-8). However, this list only includes publications in the technical literature. It omits the reports on fallout from Dr. Ernest Sternglass and others even when they appear in a Congressional report (9). It omits media reports entirely, for instance the reports on the marines at Nagasaki (10). The coverage of hazards to workers at nuclear installations is better and seven references with positive results are cited (11-16). There are three positive reports on hazards of nuclear wastes or emissions or areas of high natural radiation (17-19), but none of the studies of populations in the vicinity of nuclear power plants (20) is cited. The Rocky Flats and uranium tailings hazards are given only cursory mention. Despite the omissions, it can be seen that there are well over a dozen positive studies which were cited in the Libassi Report, disparaged, and then disregarded.

There are eleven reports of positive findings for diagnostic x-rays cited (21-31), all of which find excess leukemia among patients exposed to this low-level radiation. A negative study of occupational hazards of radiologists is cited (32) but not the positive studies. An important study of the children of radiologists (33) is omitted as are some important diagnostic x-ray studies (34).

One might wonder why in 1980 there are so many positive studies on groups exposed to low-level radiation when in 1960 or 1970 there were so few. Basically what has happened is this: Time is

running out on both the threshold hypothesis and the linear hypothesis. The nuclear exposures started in the 1950's and 1960's but because of the long latent period for the malignant diseases the health effects are only now coming to light.

These are the qualitative facts. How well do the three rival theories fit the facts? The long list of positive reports cited above is about what would be expected if the genetic degradation hypothesis were correct and if the doubling dose for leukemia were less than 5 rads. They would be extremely unlikely if the linear hypothesis were correct. They would be impossible if the threshold hypothesis were correct. Or putting it another way: In accordance with the Galilean Rule that a theory must fit the facts, the threshold hypothesis would have to be rejected completely and the linear hypothesis almost as strongly rejected on the basis of these facts. This does not absolutely prove the genetic degradation hypothesis but it makes it the only tenable hypothesis of the three.

4. TEST OF THE THEORIES: A QUALITATIVE EXAMPLE

This brief survey will not attempt to review all of the dozen or more of studies that now show positive effects in groups exposed in the one rad range. Instead it will focus on one such study which, characteristically, has been reported in the media rather than in a scientific journal. As I noted in a letter to Health Physics (35), the report appeared in the New York Times for June 17, 1979:

"Under the headline 'Study Casts Doubt on A-Test Fears' was a report from the Defense Nuclear Agency that retrospective reconstruction of the troop exercises at Big Smoky gave an estimated mean exposure of 970 millirems--'less than a third the exposure now permitted nuclear workers over three months'. The implication was that this 'could help determine the Government's liability in veterans' claims' and, as the headline suggest, minimize the pay out. The underlying notion here was that 970 millirem was too small a dose to have any serious health consequences, the same thing some health physicists have been saying for many years.

In the same news story, it is noted that studies of leukemia in the troops at Big Smoky by the Center for Disease Control found 'about double the statistical expectation'. The significance of this result is in no way changed by the dosimetry estimate."

If exposure to about one rem produces a doubled risk of leukemia, the logical inference is that the doubling dose for leukemia is probably under five rem.

Indeed, the CDC study is rather strong evidence in its own right and even the Libassi Report (2) admits "an expected incidence of between two and four cases" of leukemia from the radiation. This would be a statistical impossibility with a 100 rad or an infinite doubling dose.

All of these nuclear radiation studies might be called "fragile" statistically when considered <u>separately</u>. The main reason for this fragility is that there are a relatively small number of leukemias.

Therefore, unless a good statistical analysis is used, the hazard is likely to be missed. A poor analysis can easily cover up the positive effects. This makes it quite easy to "discredit" the positive findings. As I noted in my 1979 Senate testimony (3):

"The radiation protection community has used a divide and conquer strategy to deny or discredit these reports, treating each as if it is separate and unrelated and attacking each in turn. The main thrust of the criticisms is that the numbers of leukemias or cancers in the critical series that give positive findings is generally small. The numbers range from 6 in the Portsmouth Shipyard study (with one expected) to 32 in the Utah children (with 13 expected). It is argued that this is too few to be sure of the hazard. It is also claimed that even if there was a hazard, the casualties would be unimportant and not worth worrying about. The attitude of the radiation protection community has been that we should take a few civilian casualties for the sake of nuclear power or nuclear deterrents."

While it is relatively easy to fault the qualitative results of each study and relatively hard to argue that any one study is conclusive, the cumulative evidence cited in these more than 20 references, all of which show excess risks of leukemia or other diseases among persons exposed to doses of ionizing radiation in the one rad range, is not so easy to shake. Thus, even if the chances of spurious positive results in these studies were as high as 50-50, the chances that all of the results are spurious would be far less than one in a thousand.

It has been argued that the types of radiation and circumstances of exposure are different in each of the the different studies so that evidence cannot be combined. However, all of the studies involve the same critical event--the production of a break-point in the DNA of human genetic material. Hence, all of them are directly relevant to the question of whether or not there is an appreciable risk of genetic damage occurring at low doses of radiation. All provide positive evidence of such damage.

5. TESTING THE THEORIES: QUANTITATIVE ANALYSIS

While there are numerous epidemiological studies which provide qualitative evidence of serious hazards at low levels of ionizing radiation, there are fewer that provide quantitative results. The main reason for this is the relatively large number of leukemia cases needed for a quantitative analysis. Leukemia is such a rare disease that even if risks are doubled or tripled there will only be a handful of cases in most studies. Quantitative studies are also much more demanding with respect to the design of the study, the methodologies used in collecting the data, and the amount of detailed and verified information on each person. The two main quantitative studies are those of Mancuso, Stewart, and Kneale on the Hanford workers (11-13), and those of Bross, Ball, Natarajan, Falen, et al on the Tri-State Survey (21-25).

The kind of extensive and detailed data that is needed for quantitative studies is illustrated by Table 1. Table 1 shows the observed numbers of men in the Tri-State Survey who were 65 years or

older tabulated by three factors. One factor was a report of non-lymphatic leukemia or no leukemia. The second factor was a report of heart disease or no heart disease. The third factor was the dosage of medical x-rays estimated in rads from verified reports of exposures. The table also shows expected numbers which are numbers predicted under a genetic degradation hypothesis. Similar tables can be constructed for men 15-44 and 45-64 years of age (25).

INSERT TABLE 1

An inspection of Table 1 indicates some of the strengths of the Tri-State Survey data for quantitative analysis. There are more than 100 leukemia cases in this one table. For purposes of comparison, there are also 68 "controls" without leukemia. These are not the "pick-up" controls that are so often used in epidemiological studies. The controls are persons in a stratified random sample of households in the general population that was carried out concurrently with the leukemia survey. Random samples are ideal (but too expensive for most epidemiological studies) and they allow further methodological refinements such as "double-blind" interviewing. In other words, the person interviewed in the household was told only that this was a health survey while the interviewer was given an address and not told whether it was a leukemic or a random sample household. Other precautions were taken to avoid interviewer biases such as validation of all reports of x-rays against hospital or other records.

Speaking informally, the basic idea of the mathematical model for the genetic degradation hypothesis that was used here to calculate the expectations is this: The x-ray produces genetic degradation, break-points in the DNA of genetic material of the human cells. This concept leads, in turn, to what can be called a co-occurrence hypothesis. In other words if a clone of defective cells develops, the breakpoint is likely to have a spectrum of health effects rather than the single effect of producing leukemia. This is because we are dealing with non-specific break-points and the actual biological end result of putting this misinformation into the genetic code is likely to be a loss or reduction of some enzyme. As Dr. B.N. Ames has noted, "Damage to DNA appears to be the major cause of most cancers and genetic birth defects, and it may contribute to aging and heart disease." (36)

Such a deficiency, in turn, affects the operations of the complicated host defense system in a variety of ways. One result may be impairment of the feedback controls for the white cell system and clinical symptoms of leukemia. Another result may be difficulties with the circulatory system and clinical symptoms of heart disease. Thus one cause, a given break-point, can therefore produce more than one effect. In this data, we are looking at co-occurrence of two effects, heart disease and leukemia. Bringing in heart disease may seem odd since it is not generally considered to be radiogenic, but if it were not radiogenic the co-occurrent analysis would fail. Recently, new and independent evidence of the radiogenity of heart disease has been reported in a study of risks of radiologists over seven decades (37).

By using the co-occurrence hypothesis, it is possible to confront the three theories directly with the facts. What does the dosage response curve actually look like in the dosage range of about 5 rem? Figure 2 shows the results from one of our studies of men who received diagnostic x-rays with dosages in this range. The X axis shows the estimated trunk dosage in rads for the men in the various age and exposure categories. These are calculated from verified medical x-rays for each individual and then averaged over the category. The Y axis shows the percentage increase in the risk of non-lymphatic leukemia and confidence intervals on the individual estimates. Note that the percentage increase has already adjusted out the background risk of leukemia so that this dosage response curve should go through the origin. The graph shows separately the results for three age groups and this turns out to look like three replications of an experiment.

What does this graph tell us about the health effects of low-level radiation? There are several points that can be seen directly from the data here. First of all, there is clearly a coherent dosage response curve coming out of this analysis. As the dosage increases, the percentage excess risk of leukemia goes up. Not shown on this graph are data on a few persons at dosages averaging over 30 rads, but these show still higher excess risk. The pattern in this data is clear and reasonably consistent and it is evident that the 100% excess risk of leukemia, the doubling dose, is well down in this low dose range.

What else do these facts tell us? For one thing, they suggest that the worse case from a public health standpoint, the genetic degradation

hypothesis, seems to be right. The threshold hypothesis and the linear hypothesis are wrong. The diagonal lines shown on the graph make this point in another way. One of the lines, the steeper one, is the line for a doubling dose of 5 rem while the other pictures a doubling dose of 100 rem. The 5 rem line fits fairly well although it is possible to do a little better. The 100 rem line doesn't fit at all and obviously lies well below the confidence intervals.

INSERT FIGURE 2

6. QUANTITATIVE ESTIMATES OF DOUBLING DOSE

The mathematical model that successfully predicted the Tri-State Survey data in Table 1 and gave the dosage response curve in Figure 2 can be readily extended to provide a relatively precise estimate of the doubling dose for non-lymphatic leukemia in men. In Figure 2, each estimate of the "percent increase in the risk of leukemia" is separately determined by the data for a given age and dosage category. If an additional parameter is introduced, the doubling dose, then the simple mathematical relationship between this parameter and the original parameters of the model permits the calculation of the expectations for the entire body of data. Providing that there is a coherent dosage-response pattern to the overall data, the numerical value of the doubling dose that minimizes the total Chi-Square will predict (or explain) the whole of the data.

The Minimum Chi-Square procedure that has just been described in words can be reduced to algorithmic form (e.g., to a completely mechanical procedure) that can then be programmed on an electronic computer. Details are given elsewhere (38). When this has been done, the basic data can be typed in at a terminal, a button pushed, and an estimate of the doubling dose will be printed out that is determined solely by the data and is uncontaminated by opinions, expert or otherwise. This has in fact been carried out and the results are shown in Figure 3. On the x-axis of Figure 3 are different values of the doubling dose parameter and on the y-axis the corresponding values of Chi-Square. The estimate of doubling dose and its confidence interval can be read off directly from Figure 3.

INSERT FIGURE 3

Two curves are shown in Figure 3. The solid curve shows the push-button results for all 13 age-dosage categories. The dotted curve shows the corresponding results obtained by omitting the most divergent category. The horizontal lines indicate the critical level for the confidence intervals (e.g., the minimum Chi-Square plus the 95% tabular value for one degree of freedom). The intersection of the horizontal line with the corresponding curve for Chi-Square is shown by arrows and gives the confidence limits on the estimates. Thus for the full data the minimum occurs for a doubling dose of about five rads and the confidence interval is 3.6 to 7.6 rads. For the dotted curve the estimate is 3.3 rads and the interval from 2.2 to 4.4 rads.

There are now other estimates of doubling dose which serve to reinforce the Tri-State Survey results. The Mancuso, Stewart, and Kneale studies of Hanford find excess blood cancers although they do not find excess leukemia, for reasons probably related to the small number of cases. For the blood cancers, the doubling dose reported in Vienna was 3.6 rem (12). The Hanford data also provides estimates of doubling doses for solid tumors such as breast cancer in women and lung cancer in men. These values are higher than for the blood cancer but are generally in the 5 to 10 rem range.

Dr. Thomas Najarian and Dr. Theodore Colton have redone their original study using the badge doses for the individual workers that were finally released by the Portsmouth Naval Shipyard. As reported in congressional testimony (39), they have largely confirmed their original findings by what amounts to an independent study. The excess risks of blood cancers and of leukemia are double or triple the expected values but the overall cancer risks are about what would be expected. From the average exposures (40), it is possible to make rough estimates of the doubling dose. These turn out to be about 3.5 rem for blood cancers. So it seems fair to say that all of the quantitative estimates of doubling dose that are based on data on persons actually exposed to low-level radiation are in agreement that the doubling dose for leukemia is probably less than the five rem exposure that the Nuclear Regulatory Commission currently permits for nuclear workers each year.

7. IMPLICATIONS FOR PROTECTING THE PUBLIC HEALTH

On the basis of present facts, the best 1980 estimate for the doubling dose for leukemia (or for blood cancers) would seem to be about 3.5 rads or rems. However, in view of the historical trends in the estimates of risks from ionizing radiation, the present estimate should be viewed with some caution. The hazards have consistently been underestimated and the estimates have been drastically revised every generation. Improvements in the data or the biostatistical techniques for analyzing the data might well result in the lowering of the estimate of the doubling dose to 1.0 rad or less. Hence in cost-benefit evaluations for the deployment of new radiological technology the 3.5 rad estimate should be regarded as a minimum cost.

The 1980 scientific evidence on radiation risks indicates that these risks are more than 30 times greater than official estimates made in 1979. This drastic revision in the risk estimates should in theory require major changes in the way in which radiation technology is currently deployed and used. In practice, however, the standards set by the Nuclear Regulatory Commission and other official agencies or by the quasi-official organizations (e.g. ICRP, NCRP) reflect the state of the art in the technologies rather than health statistics. Unfortunately, this situation is not likely to be changed by the current scientific evidence on health hazards.

Perhaps public and judicial awareness that compliance with the present standards does not adequately protect the health and safety of nuclear workers or of the general public may compel changes in the

present promiscuous and sometimes dangerous uses of radiation technologies. Litigation involving low-level radiation exposures is rapidly increasing in the United States. Lawsuits involving compensation, malpractice, or environmental protection may eventually make it unprofitable to misuse radiation technologies even if the official standards continue to permit such abuses.

LEGENDS FOR FIGURES

Figure 1: Three Theoretic Hypotheses for the Shape of the Dosage Response Curve for Ionizing Radiation

Figure 2: Confidence Intervals of the Percentage Increase in Excess Leukemia Risk by Average Trunk Dose (Rads) and Age (solid bars, 65+; short dash, 46-64; long dash, under 45). Theoretic Lines for Doubling Doses of 5 and 100 Rads shown for Comparison.

Figure 3: Minimum Chi-Square Estimation Procedure for Doubling Dose for Non-Lymphatic Leukemia in Men for Range 0-9 Rads.

Table 1

Observed and Expected Numbers of Men Over 65 Years (Tri-State Survey) by Disease Status (Non-Lymphatic Leukemia, Heart Disease) and Number of Rads

Less than 1 rad		Heart Disease	No Heart Disease
Leukemia	Observed	9	14
	Expected	8.27	17.43
No Leukemia	Observed	5	17
	Expected	4.98	17.92
1-5 rads		Heart Disease	No Heart Disease
Leukemia	Observed	9	19
	Expected	9.35	17.43
No Leukemia	Observed	4	17
	Expected	4.88	16.98
5-10 rads		Heart Disease	No Heart Disease
Leukemia	Observed	7	9
	Expected	6.56	12.38
No Leukemia	Observed	5	10
	Expected	3.47	12.14
10-20 rads		Heart Disease	No Heart Disease
Leukemia	Observed	10	13
	Expected	11.76	11.68
No Leukemia	Observed	4	4
	Expected	2.74	6.62
20 rads or more		Heart Disease	No Heart Disease
Leukemia	Observed	5	6
	Expected	6.40	4.66
No Leukemia	Observed	1	1
	Expected	0.93	1.15

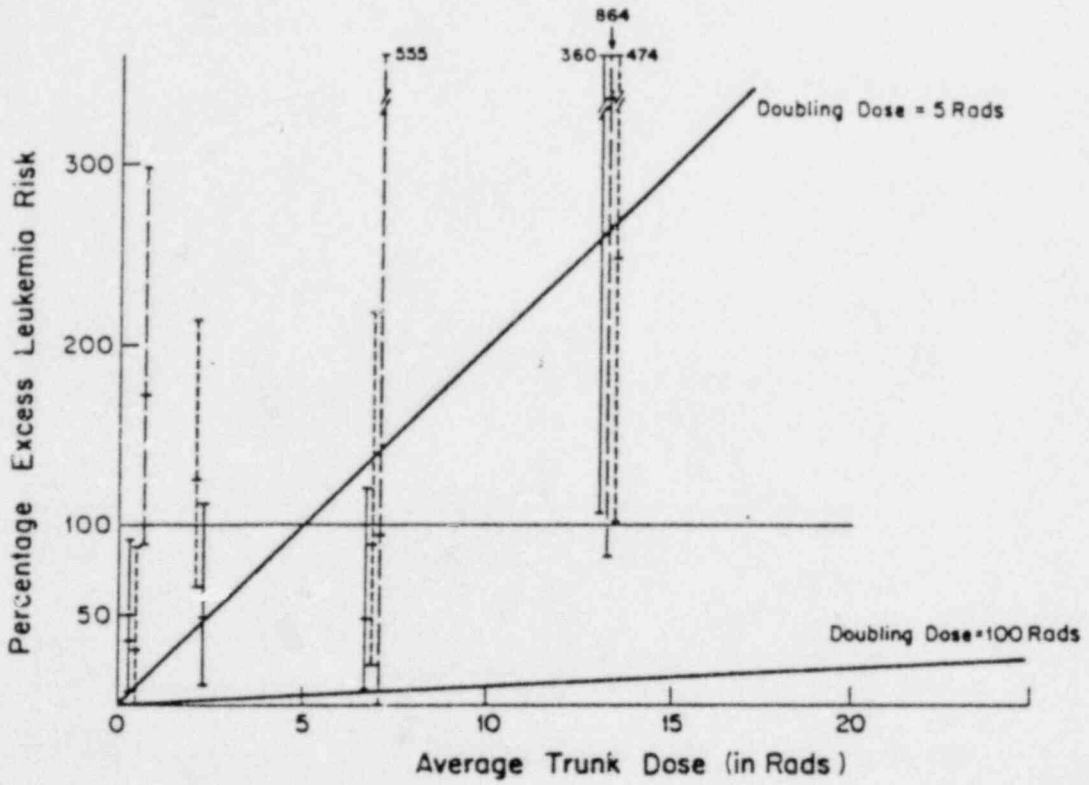
864
555
360
474
Doubling Dose = 5 Rads
Doubling Dose = 100 Rads
Percentage Excess Leukemia Risk
300
200
100
50
0
5
10
15
20
Average Trunk Dose (in Rads)

FIGURE 2

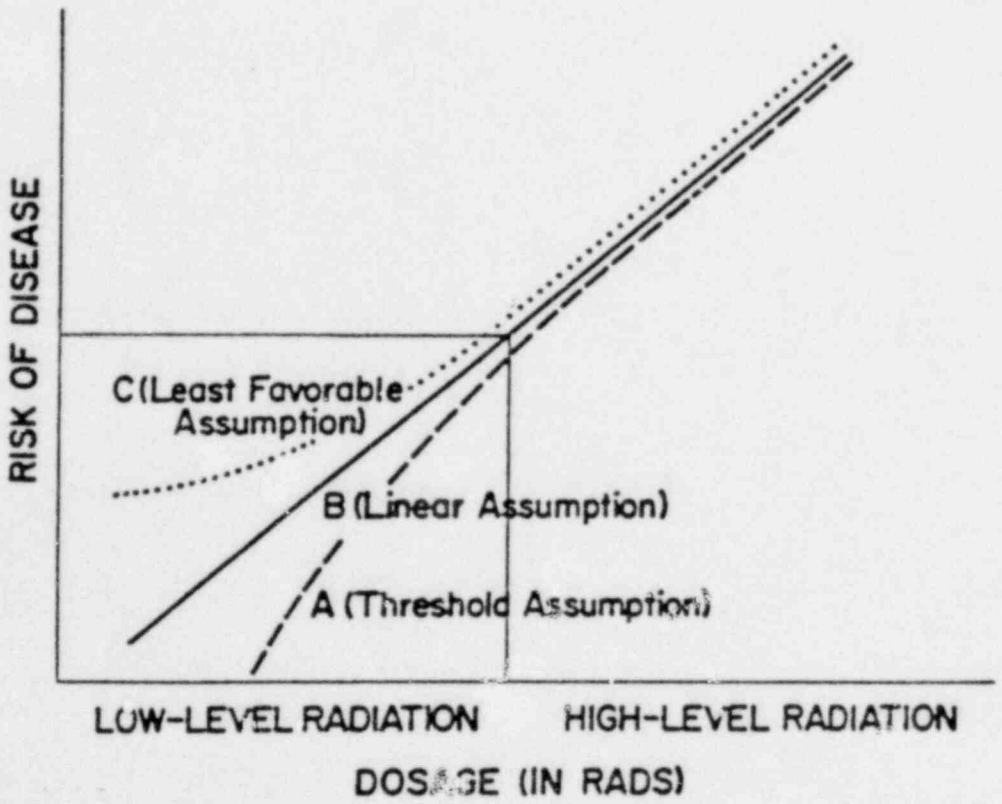
RISK OF DISEASE
C(Least Favorable Assumption)
B (Linear Assumption)
A (Threshold Assumption)
LOW-LEVEL RADIATION
HIGH-LEVEL RADIATION
DOSAGE (IN RADS)

FIGURE 1

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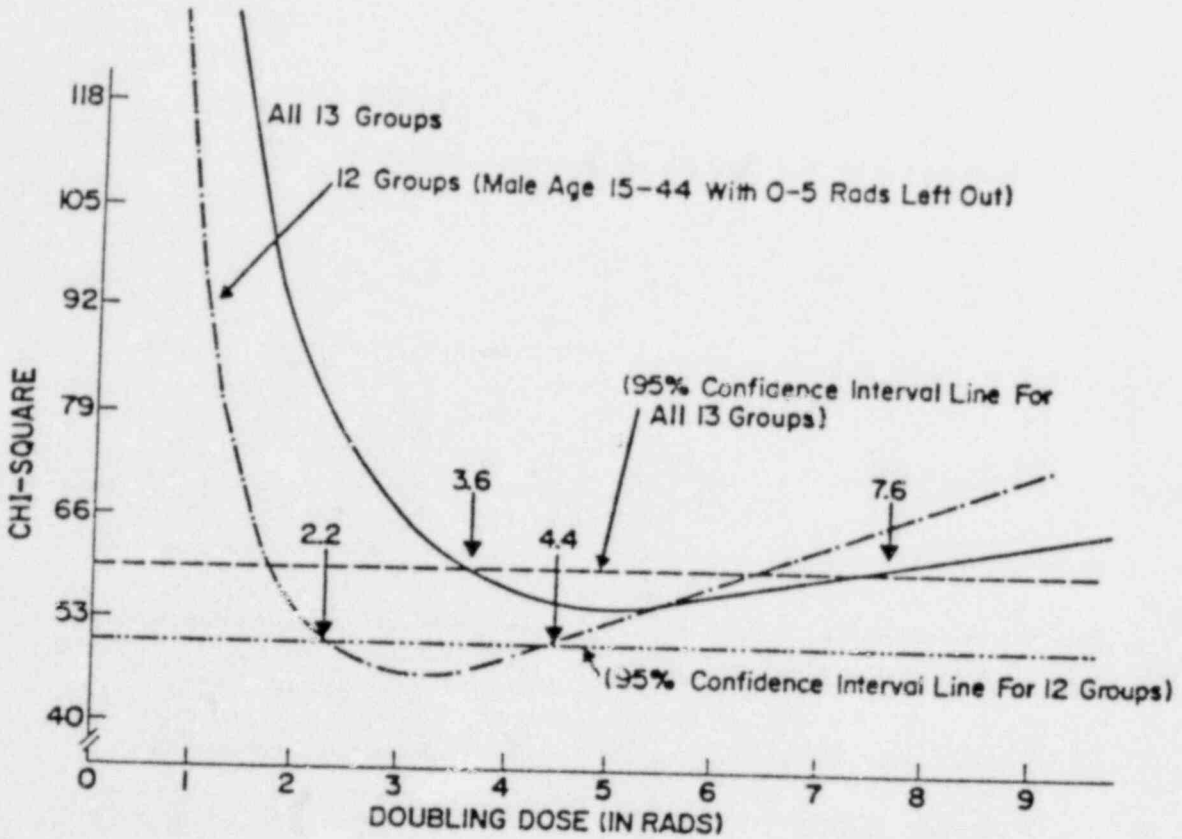
118
105
92
79
66
53
40
CHI-SQUARE
All 13 Groups
12 Groups (Male Age 15–44 With 0-5 Rads Left Out)
(95% Confidence Interval Line For All 13 Groups)
3.6
7.6
2.2
4.4
(95% Confidence Interval Line For 12 Groups)
0
1
2
3
4
5
6
7
8
9
DOUBLING DOSE (IN RADS)

FIGURE 3

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