A 1981 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 1981

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 1981 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 1981 when it might not have been feasible earlier. The main reason why the task has become feasible is 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 rems 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 for 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 1981 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 1981 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 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, we almost certainly 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 radiogenesis is the same in all of them.

Finally, in 1981 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 reassessment can focus on very specific questions such as: 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 such questions. The doubling dose estimate is directly related to official standards such as the 5 rems 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 rems, this standard is defensible. On the other hand, if the doubling dose is around 5 rems then NRC is permitting a dangerous exposure. No other carcinogen is permitted at levels close to a doubling dose for cancer in humans.

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 rems. 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 (1) and 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 (or some variant) is an irreplaceable assumption for all of the estimates in the BEIR report since the actual data used is on persons exposed to higher dosages of radiation, generally over 100 rads. Extrapolation over log orders of magnitude must be used to estimate the risks at the low levels, generally under 5 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 1981. 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. One 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. Another factor is "repair" at low doses (now known to often be misrepair).

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 1981 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 5 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. 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>1-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 1-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 1-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 1-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 1-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 1-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.

In the final version of the Libassi Report, there are five references for the hazards of nuclear radiation from fallout when thyroid cancer is also considered (4-8). However, this list is largely limited to publications in the technical literature. It omits all reports on fallout from Dr. Ernest Sternglass and others even when they appear in Congressional publications (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 somewhat better. 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 mentioned without citation. Despite these omissions, it can be seen that well over a dozen positive studies 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 on radiologists. An important study of the *children* of radiologists (33) is omitted as are some of the important diagnostic x-ray studies (34).

One might wonder why in 1981 there are so many positive studies on groups exposed to low-level radiation when in 1960 or 1972 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 (35).

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. Combined Weight of Scientific Evidence From Fragile Studies

Because the studies of nuclear radiation hazards are likely to involve relatively few cases of leukemia or myeloma or other radiogenic diseases under study, they are not "robust" (in the technical statistical sense). Therefore, it may not take much to change a study which is "positive" (i.e., achieves the traditional 5% probability level) into one which is "negative" (i.e., fails to achieve this level). Critics have only to change an underlying assumption, exclude a few cases directly or indirectly (by analytical decision), or simply use a less powerful statistical method in the analysis. Hence, in a technical sense, the studies tend to be "fragile" and <u>individually</u> they are vulnerable to critical attacks such as those in the official reports. 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."

Although it is relatively easy to fault the positive findings of each study separately and difficult to argue that any one study is conclusive, with so many positive studies it is now necessary for critics to deal with the cumulative evidence of excess risks of leukemia and other diseases in persons exposed to low-level radiation. This they have not done. Indeed, there are difficult technical questions involved in assessing the combined weight of evidence for any series of fragile studies. Although more than 20 studies have been cited here, no 2 of them are similar enough in all respects to simply pool the data.

Scientific guidelines for assessing the combined weight of evidence are needed here. The casual and subjective "expert opinions" that have been offered so far are no substitutes for such guidelines. As might be expected, such opinions depend on preconceived opinions on the hazard issue. Those who wish to discredit the low-level data argue that any number of "iffy" studies add up to an "iffy" conclusion. Their opponents argue that while an individual study might be called a "frail reed", the analogy to a bundle of frail reeds suggests the combined evidence is strong.

More adequate guidelines can be obtained by applying well-known general statistical principles and procedures to the specialized problem of combining the information in a set of fragile studies. A mathematical derivation using what is called the "likelihood ratio" approach is too lengthy to present here. However, a brief outline can be given. Starting with a minimal mathematical model for an individual case history in a study of low-level radiation hazards, the scientific structure can be characterized in terms of observed quantities and parameters. The radiation exposure, z, and the health effects, x (e.g., leukemia, no leukemia), are observed. The age-sex-disease specific risk, π, and the inverse of the doubling dose, θ, are parameters. The probability of a leukemia death, $P(x|\theta,\pi, z)$, can then be written as:

$$P(x|\theta,\pi, z) = (1 + \theta z)\pi \qquad (4.01)$$

from the definition of a doubling dose and a linear interpolative assumption for the low-dose range. Note if the exposure z equals the doubling dose the risk is doubled ($\theta z = 1$).

All of the hypothesis tests commonly used are related to the likelihood ratio of a series of case history reports in a given study. Full details on likelihood ratio methods can be found in Chapter 24 of Kendall and Stuart's, The Advanced Theory of Statistics. Here, the purpose is to test the null hypothesis that low-level radiation is harmless (or nearly so), $\theta = 0$, against the counterhypothesis that it is a serious hazard (i.e., the doubling dose is in the vicinity of 5 rems or $\theta = 0.20$). The likelihood ratio, l_i, for a given fragile study contains all or almost all of the information relevant to the hypothesis under test and can be used directly or indirectly for a standard significance test. The strength of an individual study is usually measured by what statisticians call its "power", (i.e., the probability of detecting a radiation effect when the counterhypothesis is true). This power is a function of a ratio (i.e., the ratio of the estimate θ to its standard error). For a fragile study, the power would be somewhere in the vicinity of 0.50 and detection of a real radiation effect if it exists would be "iffy."

To assess the strength of the combined information in a set of fragile studies, we can apply the concept of "power" to the set of studies rather than to the individual studies. To make the results more conclusive, the more stringent 1% (or 99%) probability level will be used here instead of the usual 5% level. At this more stringent level, the odds would be heavily against any one fragile study being statistically significant. By a straightforward use of the likelihood ratio approach, it is possible to combine the information by using the product, l, of the individual likelihoods ($l = \Pi\ l_i$). An asymptotic significance and the power of this test comes directly from likelihood ratio theory (40). The results are shown graphically in Figure 2 and they provide the desired guidelines for assessing the combined weight of evidence in a set of fragile studies.

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As would be expected from common sense, the power increases as the number of studies, m, increases and as the average strength of an individual study in the set, A, increases. What common sense alone does not provide is the quantitative relationship between factors m and A and the power of the combined test. However, this is shown in Figure 2. As might be expected, when an estimate is no larger than its standard error (A = 1), the cumulative evidence is relatively weak and the power increases very gradually. In this situation one would expect most of the fragile studies would turn out negative (e.g., at least 5 out of 6). However, when the studies are strong enough to have a 50-50 chance of a positive result (e.g., A = 2.7), the strength of the combined evidence rises rapidly as the number of studies increases. When (as for the cited studies of low-level radiation) the studies are predominantly positive, this corresponds to the region of Figure 2 where A is greater than 4. Here the power of the combined evidence is high even for as few as half a dozen fragile studies.

INSERT FIGURE 2

What Figure 2 suggests is that the evidence from the more than 20 studies cited is conclusive. The null hypothesis that low-level radiation is harmless ($\theta = 0$) must be rejected. The official estimates of doubling doses over 100 rems (where $\theta \leq 0.01$) must likewise be rejected. Indeed, the evidence is probably decisive for three separate subseries of studies. These are the series of studies on (1) nuclear workers, (2) fallout from nuclear weapons tests, and (3) diagnostic medical x-rays.

The analogy with the strength of a bundle of "frail reeds" seems to hold.

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 cases of leukemia or other radiogenic diseases 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 <u>co-occurrence</u> <u>hypothesis</u>. 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 rems? Figure 3 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 rems while the other pictures a doubling dose of 100 rems. 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 3

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 3 can be readily extended to provide a relatively precise estimate of the doubling dose for non-lymphatic leukemia in men. In Figure 3, 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 4. .On the x-axis of Figure 4 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 4.

INSERT FIGURE 4

Two curves are shown in Figure 4. 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 5 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 low-dose 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. The CDC/NIOSH follow-up of the Portsmouth Naval Shipyard workers inspired by the Najarian-Colton studies has now been completed. It was hoped that this massive study of more than 25,000 PNS workers would settle these questions, but only 6 relevant leukemia cases were found. The estimates of doubling dose are therefore imprecise. On the basis of average dose in the leukemics, an estimate of 9 rems is obtained. A slightly more precise non-parametric procedure gives an estimate of 3 rems. About all that can be said with any assurance is that the doubling dose is somewhere in the vicinity of 5 rems. However, this indicates that the exposures permitted by NRC on an annual basis are hazardous to nuclear workers.

7. Implications for Protecting the Public Health

In the time interval between the first presentation of this report as an invited lecture in Heidelberg in October 1979 and the present, the list of low-level radiation studies was twice updated, but new reports have been appearing in the literature and in the media and quickly make any list out of date. However, a few recent items will be noted here and for balance let's start with two negative studies.

A negative study on diagnostic x-rays from Mayo Clinic has appeared in the New England Journal of Medicine (41) and my critique will appear in the same journal (42). Science has published a negative report on background radiation in China (43), but for reasons noted in a Health Physics letter (44) this class of studies is of doubtful relevance.

Among the positive reports was a retrospective study of myeloma in the New England Journal of Medicine (45). Media reports indicated 5 malignant myelomas in about 1000 servicemen involved in the [illegible] operations after the Japanese A-bombs (46). There are also reports that updated studies of the Big Smoky bomb tests confirm a relative risk of leukemia of about 2.5. Excess melanoma has been reported among workers at the Livermore facility by biostatisticians using the excellent population-based cancer registry that operates in California (47). Many scientists have a misleading impression of the available evidence because of biased reporting in the technical literature (48).

On the basis of present facts, the best 1981 estimate for the doubling dose for leukemia (or for blood cancers) would seem to be about [illegible] 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 them might well result in the lowering of the estimate of the doubling dose to 1.0 rads or less. Hence, in cost-benefit evaluations for the deployment of new radiological technology the 5-rad estimate should be regarded as a minimum cost.

The 1981 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.

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

			Heart Disease	No Heart Disease
Less than 1 rad				
	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

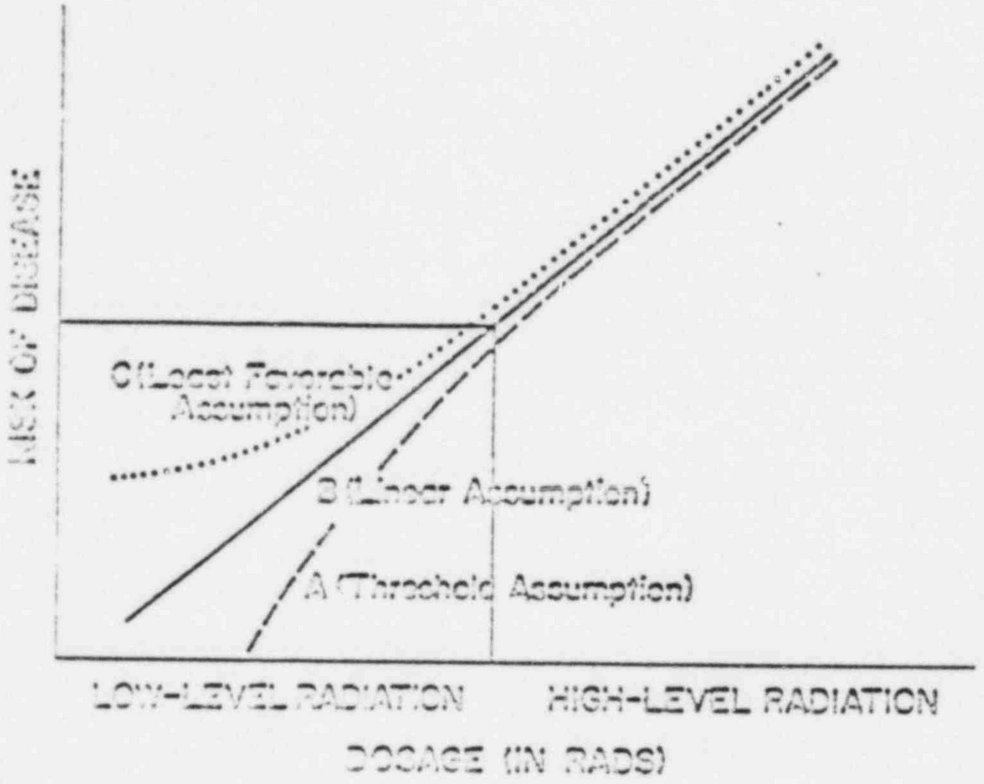
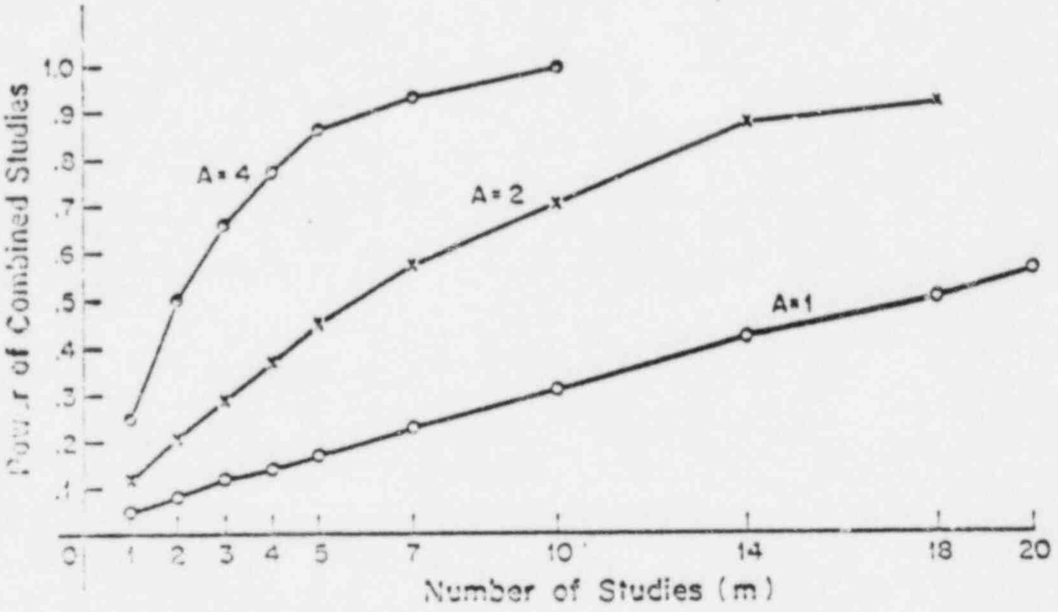

RISK OF DISEASE
C (Least Favorable Assumption)
B (Linear Assumption)
A (Threshold Assumption)
LOW-LEVEL RADIATION
HIGH-LEVEL RADIATION
DOSAGE (IN RADS)

FIGURE 1



THE POWER OF COMBINED STUDIES

Note: The power curves are for the 1% probability level, $\alpha = 0.01$, according to the number of studies, m, and the average strength, A.

FIGURE 2

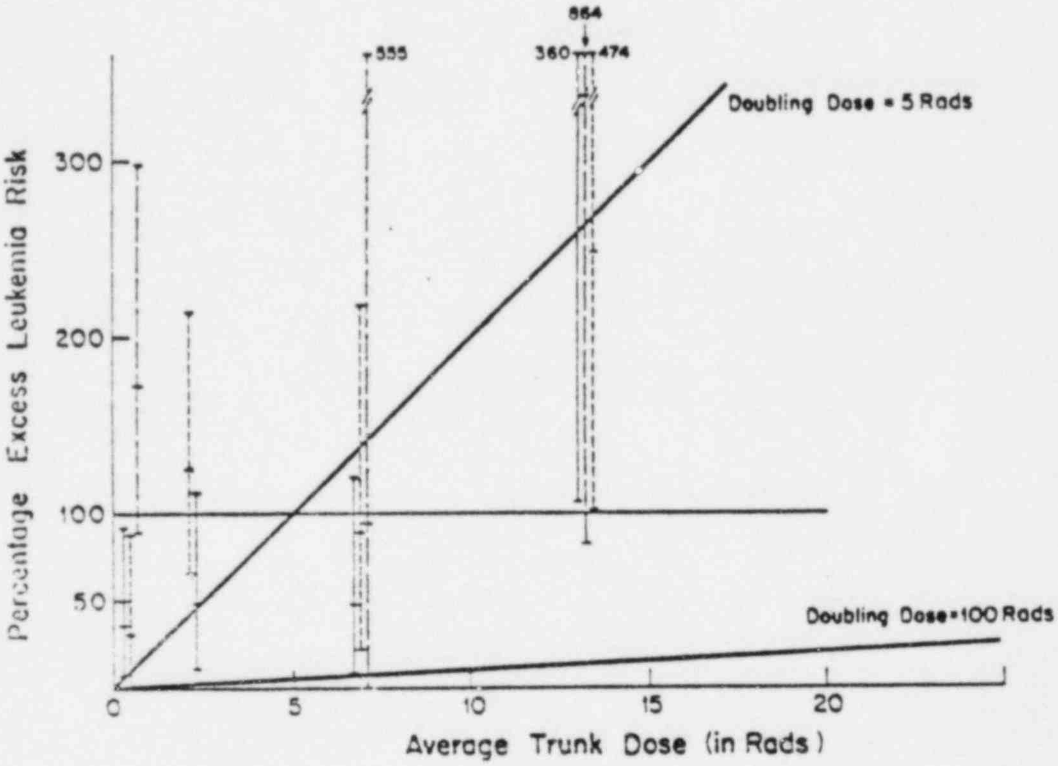

864
555
360
474
Doubling Dose = 5 Rads
Doubling Dose=100 Rads
Percentage Excess Leukemia Risk
300
200
100
50
0
5
10
15
20
Average Trunk Dose (in Rads)

FIGURE 3

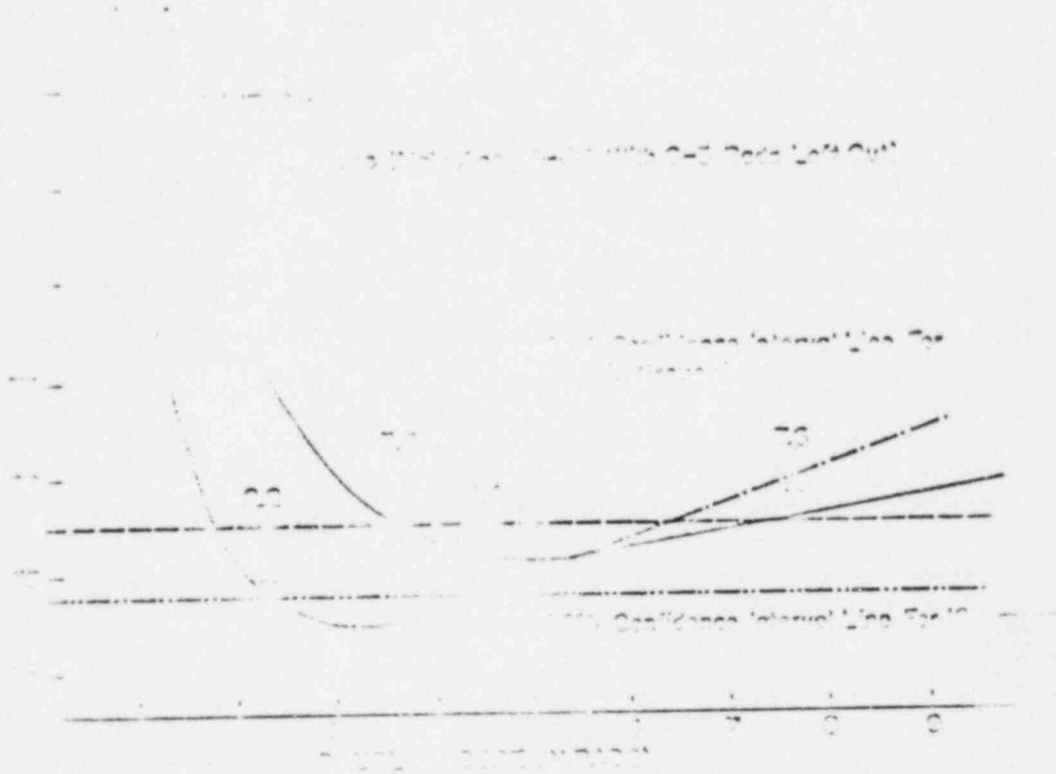

Data Left Out
Confidence Interval Line For
Confidence Interval Line For
75

FIGURE 4

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AFFIDAVIT

The accompanying material, "A 1981 Reassessment of the Health Hazards of Low-Level Ionizing Radiation", was prepared by me and has been submitted to a technical journal for publication. I swear that the facts are true and correct to the best of my knowledge, information, and belief.

Jessie D Davis
(Name)

12/8/80
(Date)

State of New York
County of Erie

Signed before me this 8 December 1980

Grace E Bueckle

GRACE E. BUECHLE
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Qualified in Erie County
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Society for Epidemiologic Research
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UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

BEFORE THE ATOMIC SAFETY AND LICENSING BOARD

In the Matter of §
§
HOUSTON LIGHTING & POWER COMPANY § Docket No. 50-466
§
(Allens Creek Nuclear Generating §
Station, Unit 1) §

CERTIFICATE OF SERVICE

I hereby certify that copies of ANSWER OPPOSING NRC STAFF'S MOTION FOR SUMMARY DISPOSITION OF CONSOLIDATED CONTENTION (CUMINGS 9, GRIFFITH 1, JOHNSTON 1, LEMMER 5) in the above-captioned proceeding have been served on the following by deposit in the United States mail, first class, this 23rd. day of December, 1980.

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