Distribution Category:
General Reactor Technology
(UC-520)

ANL-92/23

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IS THERE A LARGE RISK OF RADIATION? A CRITICAL REVIEW OF PESSIMISTIC CLAIMS*

by

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July 1992

Work performed
under Memorandum of Agreement
between Argonne National Laboratory
and Harvard University

*A short version of this report will be published in Environment International, Vol. 18 (1992).

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I. INTRODUCTION

From the earliest times men have studied the world around them and tried to find causes for their successes, their ailments and their tragedies. In the 20th century, many technological triumphs have been based upon scientific knowledge that is not widely shared. The misunderstanding, often deliberate, of the scientific bases for technology, can lead to foolish predictions: on the one hand excessive optimism in the technological triumph itself, and on the other, predictions of catastrophe that cannot, in fact, occur.

In this report we address one particular misunderstood concept--radiation. We now know that radiation is ubiquitous. But since it was first discovered, progressively by Huygens, Maxwell and Roentgen, mankind has learned to observe it, measure it, control it, and use it. It is a natural background, a necessity of life, a pollutant when in excess, a cure for disease, a cause of disease.

Some persons make a sharp distinction between natural and man-made radiation. But in practice this distinction is fuzzy. The natural background can be reduced or increased by our actions. We can build houses to avoid radon gas or to trap it; by being careless with fluorocarbons we can allow excessive amounts of ultraviolet light from the sun to penetrate the earth's atmosphere and reach its inhabited surface.

To those who understand the physics of radiation and begin to understand the biology, this is now commonplace; it certainly excites wonder, as do all of nature's works; it engenders caution, but rarely fear. But among those who have not understood, fear is a common response--an irrational fear that can prevent rational action to achieve the desired benefits and reduce hazards while introducing a minimum of new hazards.

X-rays have been with us since the 1890's and radioactivity was discovered soon thereafter, and while there was some fear of the usual X-rays, the widespread public fear did not arise until 1945 when the first atomic bomb exploded.

When fear exists, there will, in a free society, be those who exploit the fear for their own ends, who feed it and nourish it. Those who search for truth and believe that in truth lies future prosperity (and act thereon, whether for their own ends or otherwise) usually try to ignore such exploitation. The exaggerated claims and predictions of doom appear in the newspapers (or the Congressional Record), but rarely in scientific journals. This whole issue of fear has been discussed by Weart (1988).

One of the skills of a scientist is to decide which information to collect and understand and which to ignore; which scientists consistently produce work that is worth reading and which scientists can be safely ignored. But occasionally a correct idea can thereby be missed. Nor does the ignoring of the claims by scientists prevent them from having considerable influence on public policy, and we note that public policy is only partially based on established science.

In this report, we deliberately search out some of these claims. We attempt to discover what, if anything, that is useful these claims tell us. In

a search of this sort, non-medical people such as ourselves have sometimes suggested effects (such as a linear dose-response) which medical men and women would, in light of their experience, not usually consider. Among the possible results we search for are:

(1)

(2) Places where the description of the important ideas has been inadequate, so that new descriptive methods must be found to ensure that they are widely understood.

We collect a number of papers and studies and comment thereon

II FROM INDIVIDUAL CASE TO CONTROLLED STUDY

When a physician notices an unusual problem among his patients, he looks for a pattern. The literature is, properly, full of such case reports by observant physicians. It was the observation by Percival Pott that most chimney sweeps died prematurely of cancer of the scrotum that led to the realization that the soot causes cancer. This observation was so clear that no fancy epidemiological procedures were necessary. However, when effects are small, more elaborate procedures are needed.

We note here the importance of using words in ways that are widely understood. In this instance, they must be understood outside the particular discipline. Sir Austin Bradford Hill (1965) uses the word "association" to describe a situation when two phenomena are known to occur at the same time or place. A statistician often refers to a "correlation" between two observables in the same sense and insists that a correlation may not always be "causal"; However, this distinction between a "causal" and a "non-causal" correlation is not always realized, and "correlation" is often automatically exaggerated into "causal correlation". We here use the word "association" instead of correlation in order to emphasize this distinction, and reject any implication of causality, although an association may sometimes be a causal correlation.

Hill (1965) outlined nine criteria that have to be considered when attempting to attribute a cause to an effect. Hill emphasizes that they need not all be simultaneously necessary. For example, the strength of the association observed by Percival Pott was so great that the association forced attention even though there was little biology to make the causality plausible and nothing with which to make an analogy. The nine criteria are as follows:

1 The strength of the association. If the strength of the association is large, then common sense usually makes it outweigh other considerations. Nonetheless, cigarette smoking gives a large effect, but

the delayed nature of the effect meant that 50 years passed before it was generally accepted that most lung cancers are caused by cigarettes.

2. The consistency of the results. If the same data set is analyzed by different people, they should all find similar results.

3. The specificity of the results. If a specific health condition is associated with the claimed cause, it is usually more believable than a general claim of increased mortality.

4. Temporality. The effect must follow the claimed cause and never precede it. If there is a delay (latency period) it must be plausible and understood.

5. Existence of a biological gradient. The effect should increase as the pollutjon increases.

6. Biological plausibility. The effect should be plausible biologically. This need not mean that there is a detailed explanation, but that the effect should not violate known biological laws.

7 Coherence. Various studies should be correlated in a coherent picture; one isolated study is hard to believe if it seems to contradict others.

8. Experimentation. In some cases, the epidemiological study can be supported by experiments on animals where doses are given in a controlled way. It is such experiments, for example, that led to the Linear Quadratic model of BEIR (1980).

9. Analogy. Sometimes we can make an analogy between two carcinogenic agents. For example benzene causes acute myeloid leukemia with a short latent period. Thus, one might reasonably expect a short latent period for radiation induced leukemia.

Each of these nine criteria are here considered in conjunction with the unusual claims of effects of radiation.

These may seem sophisticated criteria, but a close examination shows that they are reasonably simple logical requirements. Although Hill emphasizes that the attribution of cause to an effect does not need all the items to be present, it is clear that there must be no disproof. Even here we must be careful. Biological plausibility depends upon the biologist; some ideas that are now generally accepted such as lack of a threshold for some effects met with much resistance when first promulgated. We must be alert to the new, but demand careful and reasoned argument.

As one looks at the successful discoveries of causes of diseases that scientists have made over the last century, it is impressive that the first step often seemed illogical at the time and were a bold flight of imagination. The first step can therefore only be accepted as a correct step when many verifiable

deductions have been made from it. It is important to address inexplicable observations, but equally important not to make immediate public policy conclusions therefrom. This was discussed in another connection in an editorial in Nature (1988).

It must be remembered that if a phenomenon does not fit with existing scientific understanding, it requires more, rather than less, evidence to prove its reality. If for example, it was claimed that a dog ran down 5th Avenue in the city of New York at noon, not many people would be surprised. But if it was claimed that a lion ran down 5th Avenue at noon, there would be considerable proof required. The required proof would be less if other information made it more plausible, if it were known, for example, that a lion had escaped from the Bronx Zoo in New York City. However, if it were claimed that a pterodactyl ran down 5th Avenue at noon, most auditors would be skeptical because pterodactyls are extinct. They would believe that the claimant was, at least, mistaken. So it is with science.

Anyone who claims that low doses of radiation give large effects must overcome a weight of prior evidence; this demand might be reduced if it could be shown that the instruments measuring the dose or the calculations thereof were faulty and the dose might not be low after all. In most of the cases we discuss here, the evidence provided is less than would be required if the claims fit established science.

Associated with this need for increased proof in unusual situations, is the need to create a plausible model to describe the event. This model, which presumably should be valid at other places and times, should be tested to see whether it indeed makes such valid predictions.

For example, if occupational exposure to radiation is claimed to cause an excess of cancer, and a background of environmental and medical exposures gives 10 times the radiation dose, one should easily be able to find an excess of cancers from these environmental and medical exposures. If one cannot, then the model must be incorrect.

Any claim of unusual association which does not go on to describe a plausible model is incomplete; it will, however, be seen that few authors make such models. (The exception being Dr. J. W. Gofman whose work we describe below, and whose model is capable of proof or disproof).

A scientist often makes 99 false steps for every successful one; the 99 false steps are rarely recorded in detail. In this field in particular, an imaginative case report can be a statistical artifact; however, if it stimulates other physicians to look for similar effects, and other reports of similar cases follow, it can be the beginning of a serious epidemiological investigation which eventually proves the cause of disease. If one finds a cause, one may eventually find a cure for a disease. We must be alert to the imaginative idea--but not so much or in such a way that unnecessary panic is caused, and one unnecessarily stops worthy endeavor. An argument similar to this was made by Cehn and Sagan (1988).

There is a fine dividing line here, and for the particular case of radiation it is this dividing line we are exploring.

III. STATISTICAL TRAPS

Hill did not state the two most elementary criteria--and the criteria most frequently ignored. There must be a statistically significant effect to consider. Secondly, the statistical analysis must not be biased.

Many errors in the analyses of publications considered in this paper are statistical. The most important of these is biased selection of initial data. Errors associated with such data selection are also some of the hardest to explain to those unacquainted with statistical methods.

The late Richard Feynman had a dramatic way of demonstrating that a biased selection of data can invalidate standard statistical tests. Coming into class, he said, "You know, the most amazing thing happened to me tonight. I was coming here, on the way to the lecture, and I came in through the parking lot. And you won't believe what happened. I saw a car with the license plate ARW 357! Can you imagine? Of all the millions of license plates in the state, what was the chance that I would see that particular one tonight?" (Goodstein 1989) We can easily work it out: 3 is one out of 10 numbers, 5 is one out of 10 numbers, 7 is one of 10 numbers, A is one of 26 letters, R is one out of 26 letters, and W is one out of 26 letters. If we multiply these numbers together we find a low probability of 1 in 18,000,000. Yet Feynman saw it. This commonplace experience does not seem that improbable. What is the answer to this paradox?

As presented, the answer to this paradox is obvious: Feynman did not ask the question about the particular license plate until he knew the answer. However, in epidemiological studies the paradox is often disguised. Even competent persons have been caught at times. The less expert falls into this trap frequently. This trap is far from unique to epidemiology, nor is it unusual. Physicists of all sorts fall into it with surprising regularity. In honor of our friend, the late Professor Richard Feynman, we call it the Feynman Trap.

The importance of using unbiased data in any statistical study, and epidemiological studies are only one of many, is so great that it can hardly be overemphasized. The ideal procedure in epidemiology would be to select a cohort (group of persons) for study while they are young and follow them into the future. Such a study can only be complete after the proverbial lifespan of 3 score years and 10 (70 years), and even then is not immune from genetic bias or bias due to pre-existing environmental effects.

In practice, what is called a prospective study does not do this; the epidemiologist defines a cohort of interest that existed in the past and then goes through records to find out what happened to the members of the cohort. He must make every effort to be sure that he is not influenced by any prior knowledge of the final result in selection of the cohort. This is hard to do; it is not sufficient that the investigator not have prior knowledge. His boss and his funding agency may have such knowledge and have an influence upon the choice of cohort.

This is so difficult, yet so important, that it is preferable that every prospective epidemiological paper starts with a discussion of this point, especially if the numbers are small and the effect of bias most serious. Unfortunately, this is not done in many epidemiological studies, even by some of

the best authors and even in some studies using small numbers upon which major societal decisions depend.

For example, if a small, possibly unusual, cluster of cancer cases is found in a certain location, concerned citizens will properly search for possible causes. They might find an abandoned well or dump site containing some chemical known to be toxic, but with no specific known adverse chronic health effects. It is proper to postulate this chemical as a possible cause. This is sometimes called "the hypothesis-generating event". This can be related to the automobile in the Feynman example.

The hypothesis-generating event can then trigger an epidemiological study; the epidemiologist must search for other similar wells or dump sites also containing the chemical of concern. The people must be similar to the general population in all respects except their proximity to the well or dump site and possess no other difference in common with the people around the original well. Having found such a cohort, and not before, (or he might be influenced in his choice by the result) he can then search the records to find out whether the same type of cancer appears at the new location.

Finally, in establishing statistical significance, the epidemiologist must omit the original group of people, with their cancer cases, that brought the subject to his attention in the first place. We see that this then will satisfy the requirements of reproducibility and specificity outlined by Hill. In many of the discussions below of the claims of large effects of radiation, the first requirement seems to be met, but the others are not.

Statistical significance is a technical term used in statistics to deal with the fact that the outcome of an experiment that measures the occurrence of certain stochastic (random) events (e.g., the outcome of a throw of a die), when repeated many times under identical conditions, is not sharply deterministic, but assumes a distribution around a mean value. Consequently, we quantify this by reporting the mean value plus the standard deviation within a certain probability or confidence limit. For normal distributions, if the mean value is N, then the standard deviation is $\sqrt{N}$. The 95% confidence limit corresponds to the range of values not exceeding $(N + 1.64 \cdot \sqrt{N})$. Therefore, if the expected number of cancers among a group of residents is N and the number observed exceeds $(N + 1.64 \cdot \sqrt{N})$, then one can claim that a cluster is observed and there is less than 5% chance that the observed excess is due to a statistical fluctuation above the normal rate.

One should be careful in applying these statistical uncertainties when there is a constraint upon the data; when this occurs the statistical error is less than the $\sqrt{a}$. For example, if there are exactly 250 cancer deaths in a group of 1000 people, the statistical error of the number of dead will be less than $\sqrt{250}$ and the statistical uncertainty in the number of those alive will be less than $\sqrt{750}$ because the sum of those dead and those alive is fixed. The standard deviation σ_p of the *proportion* p is given by $\sigma_p = \sqrt{p(1-p)/n}$, which in the example gives $\sqrt{0.25 \cdot 0.75/1000} = 0.014$, so that the ratio is 0.250 ± 0.014, and the standard deviation of the *absolute* number of cases is 14, instead of $\sqrt{250} \approx 16$. If p=0.5 this constraint would divide the standard deviation by 1.4 (the square root of 2); however, in most cases the difference is small and can be neglected.

There are numerous, well-established, epidemiological studies that show that large radiation doses to people cause an increase in leukemia rates, and we know roughly, how much. Moreover, radiation-induced leukemias appear after a moderately short latent period, so that they are easier to identify than radiation-induced cancers with a long latent period. It seems obvious, therefore, to search for possible increases in leukemia near nuclear power plants, or any other known radiation sources. It seems especially appropriate to use leukemia as a marker for chronic effects of radiation. Thus, it would appear that the hypothesis has already been generated. However, this is only true if we have enough radiation from the source to cause a statistically significant increase in the leukemias. In several of the cases below, we are discussing a new hypothesis: "radiation causes leukemias at several hundred times the rate expected from the known and published radiation measurements assuming linear biological gradient." This could happen either because the actual radiation levels are several hundred times the "known and published" ones, or because of a new, and most scientists would say unlikely, biological phenomenon.

The most systematic and complete test of this new hypothesis is the study of cancers near nuclear facilities in the UK discussed below where all nuclear facilities in the UK were studied. By taking all such facilities, bias in selection of facilities for study is avoided.

One of the most common temptations for any epidemiologist or other student of statistics, is to decide upon groups of data, or decide upon statistical tests, <u>after</u> the preliminary results of the study are known. It must always be remembered that if 20 independent biological endpoints (such as cancer in 20 separate organs) are studied, and each tested according to separate statistical tests, then one will appear to be statistically significant with $P < 0.05$, by chance alone.

Again, in practice, it is rarely possible to be absolutely "pure" in this regard. When a new idea for a test arises after the study has started and the data collected, some correction can be made by increasing the level of statistical significance demanded. In the case above, where 20 tests are examined, and it is not known in advance which test is to be examined, one should demand $P < 0.05/20 = 0.0025$ instead of the usual $P < 0.05$. A failure to do this is sometimes called Tippett's trap, because the well-known statistician Tippett called attention to this problem (Tippett 1937).

The reader can often tell whether basic statistical errors such as these have been made. If an author of a paper has data which are just significant, and does not discuss these potential problems, it can usually be assumed that he was unaware of them and may have fallen into one of the traps. We know of few exceptions.

Before discussing this UK work in detail, we study first a much bolder claim: that low-level nuclear radiation causes an increase in infant mortality rates. This is rarely explicitly stated with its details made clear. It is not clear whether it is external radiation, or ingested radionuclides, that are claimed to cause the increase in infant mortality. It is not clear whether the effect is claimed to be immediate or delayed, and if so with what time delay. At a first reading of such claims it appears that the claimed effect has a time delay of a few months; radiation damage to the fetus could make it subject to a large number of causes of death. It is, of course, essential that any subsequent confirming study at other locations address the identical claim.

IV. DOES RADIATION CAUSE INFANT MORTALITY?

Dr. Ernest Sternglass, Professor Emeritus in the School of Public Health, University of Pittsburgh, published a paper (Sternglass 1963) alleging a link between fallout from nuclear bomb tests and the infant mortality rate.* This was based on the experimental evidence by Stewart and Kneale (1970) and by MacMahon (1963) that X-rays given to pregnant women increased the incidence of childhood leukemias. Fitting these data to a linear dose-response relationship, he argued that fallout from bomb tests should increase childhood leukemias, and then extended the argument to other infant mortality. This paper made a number of arbitrary assumptions which were criticized by Dunham (1963), Bennett (1963) and MacMahon (1963). In 1969 Sternglass produced a number of other papers and reports (Sternglass 1969a, 1969b, 1969c, 1969d, 1969e, 1969f). In these papers he made a number of suggestions that fallout from nuclear bomb tests was responsible for a number of infant leukemias. These claims were made on the basis of a plot of infant mortality versus time (see Fig. 1).

It was tempting at the time for scientists to believe Sternglass' claims without looking carefully at them. By 1963, a majority of scientists had successfully persuaded the major countries of the world to stop testing of nuclear bombs in the atmosphere. Sternglass appeared to provide extra ammunition to justify this. Rotblat, a leader in urging nuclear test bans, asked that this temptation be rejected; sooner or later, he argued, the acceptance of bad science, even for a good reason, would backfire (Rotblat 1970). He was particularly concerned that it would be used against peaceful uses of nuclear energy.

These claims met with a storm of criticism (Graham and Thro 1969; Boffey 1969; Stewart 1969; Wrenn 1969; Sagan 1969; Eisenbud *et al.* 1969; Heller 1970). This then led to an unprecedented statement read by the current and signed by all living past presidents of the Health Physics Society (Moeller 1971). "We, the President and Past Presidents of the Health Physics Society, do not agree with the claim of Dr. Sternglass that he has shown that radiation exposure from nuclear power operations has resulted in an increase in infant mortality."

Sternglass then extended the arguments about fallout from nuclear bomb tests to study infant mortality (and sometimes leukemia) near nuclear power plants. A number of persons have reviewed various of his claims; one of the most specific is that of Hull and Shore (1971). Sternglass has since produced a string of about 10 reports a year, none of which has been accepted in the community as having any validity.

An example of one of these is his claim that infant mortality increased near Indian Point I Nuclear Power Plant just after it began operation in 1961. Figure 1 shows how these claims, made for one specific pair of years, show selection bias. The top figure (1a) shows Sternglass' two points. They look less significant when statistical errors are shown (1b). When the whole graph is shown

*Infant mortality rate is usually expressed as: (number of deaths of infants <1 year old) x 1000/(number of live births during same year) [MacMahan 1980, p. 68].

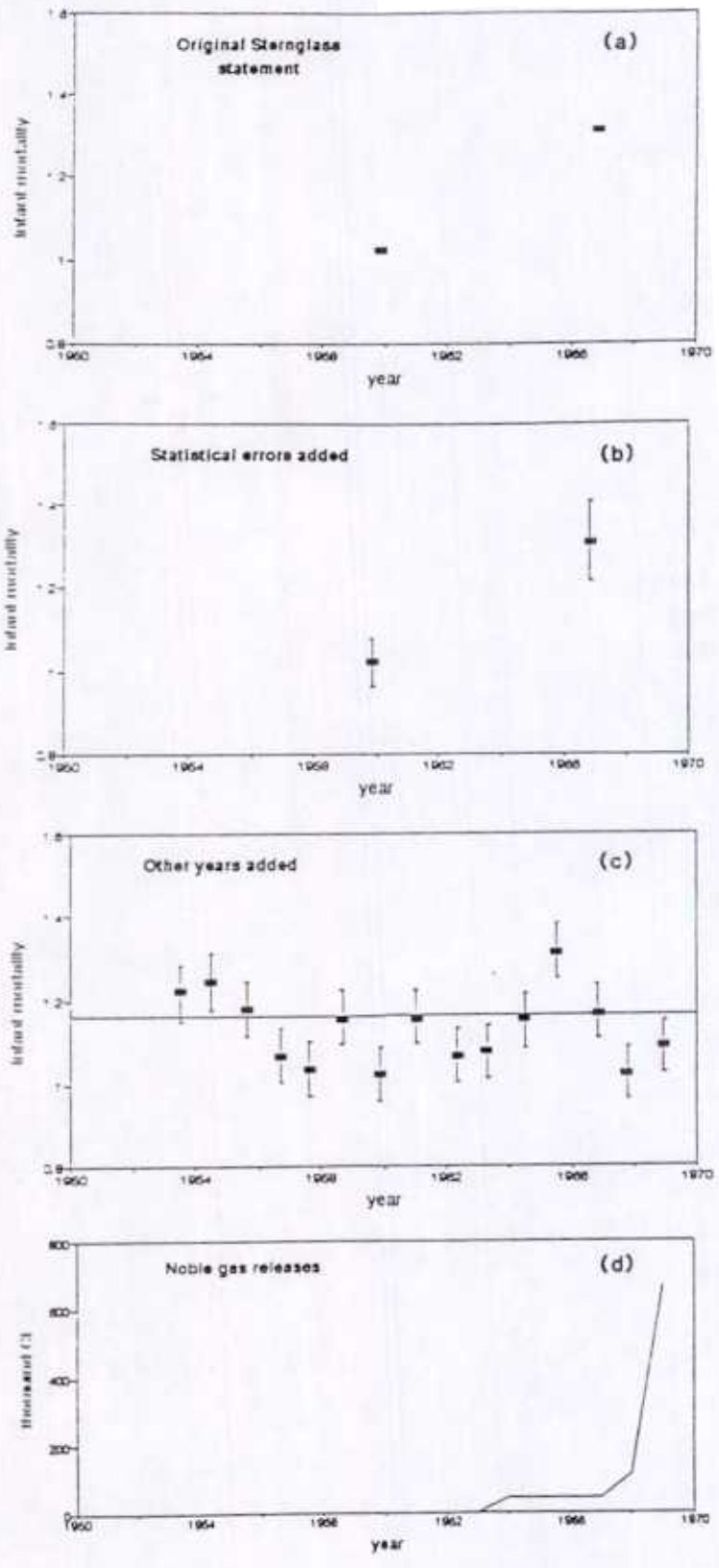

Fig. 1. Infant Mortality Near Indian Point, New York

(Hull and Shore 1971) it is clear that the points were arbitrarily selected in time. Figure 1d shows that the increase was not correlated with radioactivity releases as originally claimed, but preceded them. We note that this was a selection bias in time. We can also have a biased selection of place.

In one of the more recent reports, Sternglass (1986) claims that a release of radioactive material to the environment from the Pilgrim Nuclear Power plant in Plymouth, MA, in June 1982 caused an increase in infant mortality in the counties nearby. As reported to the Nuclear Regulatory Commission, the release was a solid material, and was confined to the power plant property. Nonetheless, it is, of course, plausible to look for effects near the power plant. Sternglass claimed an increase in infant mortality from 1981 to 1982.

In Fig. 2, we show the full data on infant mortality for various years collected by the Massachusetts Department of Public Health (DPH) (Mais 1987). In Fig. 2a are Sternglass' two points for the town of Plymouth for 1981 and 1982. These indeed suggest an increase. When the statistical errors are added in (Fig. 2b) the claim already looks less impressive. In Fig. 2c, the data for many years are included, showing that the overall trend is opposite to that implied by Sternglass. When the data are collected for the whole county and the whole state, in Figs. 2b and 2c, the fluctuations are reduced because of the larger statistical sample. Finally, we note that the measured radioactivity releases from the power plant were larger during the early years of operation--before a graphite filter was installed and while there was a period of leaking fuel pins. However, at no time would these releases have suggested a large excess of cancers, and indeed no such excess has been found. We call attention to the similarity of the claim of infant mortality around Indian Point, and its refutation, to the claim of infant mortality around Pilgrim. Figure 3 shows the same argument for the recent low birthweight around Pilgrim Power Plant.

Not content with the claim that there was increased infant mortality near Pilgrim in 1982 caused by the 1982 release, Sternglass attributed an increase in infant mortality in southwest New Hampshire, 100 miles away, to a combination of Pilgrim and two other nuclear power plants--Vermont Yankee and Yankee Rowe. The smog in Boston is closer, thus providing a more likely potential culprit to study.

V. LEUKEMIA CLUSTERS

It is self-evident that people dying of infectious diseases do not die uniformly throughout the world, but in clusters, either in space or in time, where the infection has taken hold. Diseases which are not infectious are not expected to cluster, except insofar as there might be exogenous causes. Cancer is generally believed to be a non-infectious disease.

Only 3% of cancers are leukemias; but about 20% of cancers that are induced by radiation in the first 30 years after exposure seem to be leukemias. This is because of the relatively short latent period for leukemia. This suggests looking for leukemias--particularly acute myeloid leukemias--as an indicator or

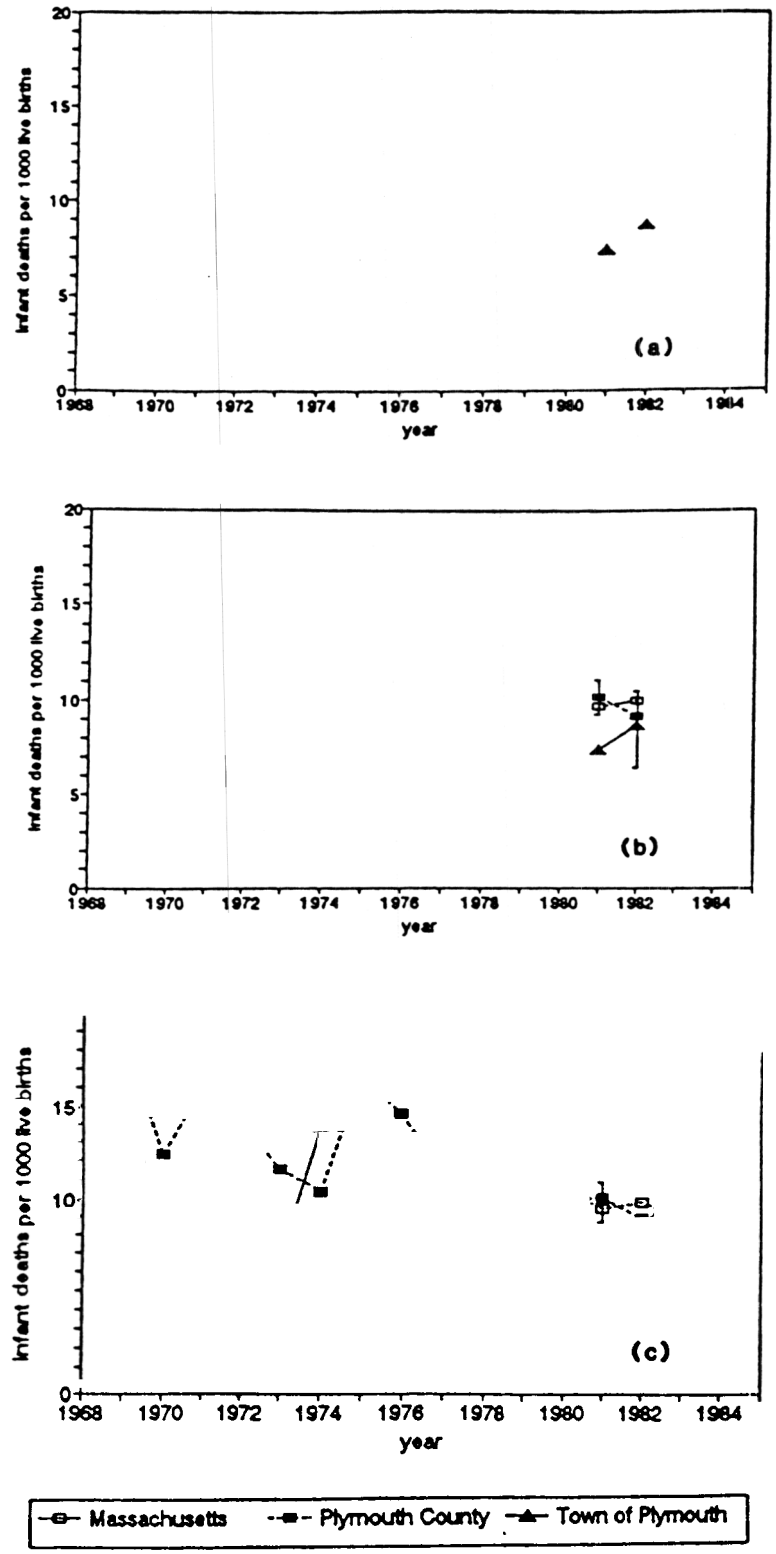

Fig. 2. Infant Mortality Rate in Plymouth, Plymouth County, and Massachusetts 1969-1984

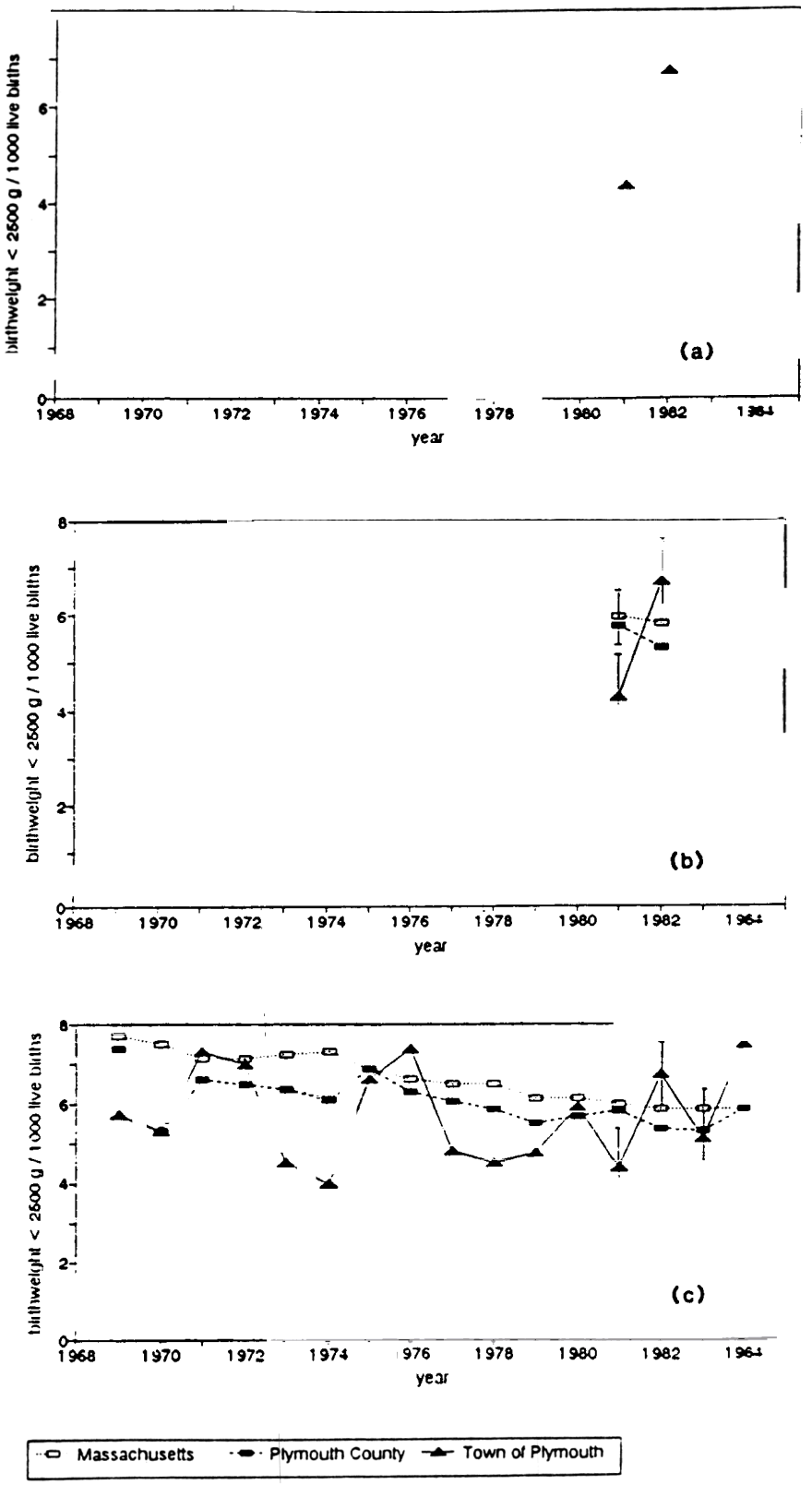

Fig. 3. Percent of Low Birthweight in Plymouth, Plymouth County, and Massachusetts 1969-1984

marker of radiation exposure. Moreover leukemia has a short latent period and a casual association with an event becomes easier to prove than for other cancers. But there are several other causes of leukemia; such as benzene and possibly other solvents. Leukemias are believed to cluster in such a way that deviations from expected rates exceed the standard deviation (Glass *et al.* 1968).

We note here that there are four major types of leukemia that are hematologically distinct: acute lymphocytic (ALL), chronic lymphocytic (CLL), acute myelogenous (AML) and its variants, and chronic myelogenous (CML). Of these, CLL is not known to be caused by radiation. Indeed the progression of the disease is slow, as evidenced by a doubling time of white blood cells of two to three years after diagnosis. Extrapolating back to a single cell division suggests that CLL is caused early in life, and perhaps has a genetic origin. Therefore in studies of leukemia caused by an external agent such as radiation, it is usual to exclude CLL.

However, there have been many searches for clusters, particularly of leukemia, from a suggestion that leukemia, and in particular childhood leukemia, might have a viral origin (Smith 1982). Darby and Doll (1987) also addressed this idea. For a long time, leukemias have been known to cluster without an obvious cause, an effect that suggests that the origin might be an infectious disease. For example, the first child in a family is much more likely to get childhood leukemia than later ones. A particularly interesting phenomenon was noted by Smith *et al.* (1985). One way of curing leukemia is to destroy blood cells and bone marrow by heavy radiation exposure. Then, new blood can be provided by a blood transfusion, preferably from a twin. Smith *et al.* (1985) noted the occurrence of leukemia in a patient with new bone marrow well after the treatment by whole body irradiation. This is consistent with a viral origin for the leukemia. Some earlier suggestions that clustering occurred are usually attributed to biased post hoc selection of boundaries for the grouping of leukemias (Glass *et al.* 1968). We will return to the suggestions that childhood leukemia might have a viral origin, and cause clusters, when we discuss the cancers around British nuclear facilities.

We emphasize that few clusters of cancer or leukemia survive as real (i.e., not due to statistical fluctuation) clusters when the data was subjected to careful screening and analysis. Still, a few real clusters exist, for example, at Sellafield in the U.K. In a later section we discuss attempts to establish a causal correlation between clusters in the U.K. and radioactivity releases from nearby nuclear facilities. We note here also Jablon et. al. (1990) of the National Cancer Institute (NCI) of the U.S. have carried out a comprehensive analysis of leukemia and cancer incidence at the county level around all nuclear plants in the U.S. and found no significant effect. They noted a deficit of leukemias in Plymouth county which contains the Pilgrim Nuclear Power Plant which we will discuss next.

Finally, we reiterate that even real (nonstatistical fluctuations) leukemia or cancer clusters can occur randomly without an apparent cause. Such random clusters, it appears, do not discriminate between nuclear or non-nuclear facilities. In a blind attempt to study leukemia clusters, leukemia around 14 military sites in England was studied. Clusters were found around two of them. When the identity of the two military sites were released to the study group, it turned out that the sites were medieval castles (Cehn and Sagan 1988). It is unclear whether the study group was influenced by the statement that they were military sites.

VI. LEUKEMIAS NEAR PLYMOUTH, MASSACHUSETTS

Cobb (1987) noted that the number of leukemias in certain counties in SE Massachusetts was larger than expected. He asked whether they could have been caused by the Pilgrim Nuclear Power Plant. Cobb postulated a certain pattern of coastal circulation of the air within 2-4 miles of the coastline (Clapp 1987). In his testimony in front of the Joint Committee of Energy of the Commonwealth of Massachusetts, he stated that, "It is easy to imagine how an injection of pollutants to the middle of such a pattern might be contained and carried along the coast." However, detailed measurement shows that winds do not follow the postulated pattern (Stone and Webster 1988). A more detailed listing of leukemias in Plymouth county has been carried out by Rothman *et al.* (1988). (Tables 1 and 2). In these tables, the expected number is based upon state-wide statistics.

Table 1 shows a small excess of leukemia (excluding CLL which, as we noted, is not caused by radiation) for the years 1982-84 in the five coastal towns closest to Plymouth. This is barely statistically significant and the significance vanishes when more years are included. This is shown more clearly in Table 2 from Rothman et al. Moreover, we know of no postulated reason, other than the impossible one that they are due to the windborn radioactivity. However, an interesting fact emerges upon which Rothman *et al.* did not comment. If we add a fourth group of three columns to Table 2, for Plymouth County less the five towns close to Plymouth, a marked deficit appears after 1977. For the period 1977-86, 168 leukemias were observed with 207 expected. The deficit of 39 is over twice the standard deviation of $(207)^{\frac{1}{2}} = 14$ and therefore significant (Wilson 1991). In a nationwide study of leukemias near nuclear power plants, carried out at a country level, Jablon *et al.* (1990) also noticed the deficit of leukemias in Plymouth County.

In 1990 a report (Morris and Knori 1990) was released. These authors did a case control study of leukemias near Plymouth, using a complex "score" of closeness to Pilgrim as a surrogate for exposure level. The report issued by DPH emphasizes the first of these descriptions. Table 3 shows the data for cases diagnosed between 1978 and 1986. Since these are the same cases already discussed, a similar difference between close to Plymouth and far from Plymouth is expected. A statistically significant difference is indeed found. Since the previous data and reports already suggested an effect of the same magnitude as found in the DPH study, it is hard to understand the statement on page (vi) of the summary of Morris and Knori, "These (earlier) findings are somewhat inconsistent with those of this investigation".

Morris and Knori further subdivided the data into the periods 1978 to 1981, 1982 and 1983, and 1984 to 1986, and find an effect only in the first two. This is strange, because our simple calculation in Table 2 shows an effect persisting in 1984-86. Moreover, the Pilgrim plant only began operating after 1973. If it is hypothesized that if the radiation from the plant immediately after startup caused leukemias, they would be expected to continue to occur from 1978 through 1993; and there is no valid reason for excluding the years 1984 to 1986 in this analysis. To make such an "exclusion" without a valid reason makes the statistical calculations invalid.

TABLE Observed and Expected Incidence of Leukemias Other Than Chronic Lymphocytic Leukemia

TABLE 1. Observed and Expected Incidence of Leukemias Other Than Chronic Lymphocytic Leukemia in Three Groups of Massachusetts Towns, 1982-1986.[a] Data from Rothman *et al.* (1988)

	Five Coastal Towns[b]				Five Towns Closest to Plymouth[c]				Plymouth County[d]			
Years	Obs.	Exp.	SMR	95% CI	Obs.	Exp.	SMR	95% CI	Obs.	Exp.	SMR	95% CI
1982-84	27	17.0	1.59	1.05-2.31	13	12.2	1.06	0.59 1.78	63	73.8	0.85	0.66-1.09
1985-86	6	11.8	0.51	0.21-1.06	6	8.6	0.70	0.28-1.45	36	47.5	0.76	0.53-1.05
1982-86	33	28.8	1.14	0.79-1.61	19	20.8	0.91	0.57-1.40	99	121.3	0.82	0.66-0.99

[a]Abbreviations: Obs., observed cases; Exp., expected cases; SMR, standardized mortality ratio (Obs./Exp.); CI, confidence interval (by exact method).
[b]Duxbury, Kingston, Marshfield, Plymouth and Scituate.
[c]Carver, Duxbury, Kingston, Plympton and Plymouth.
[d]27 towns, including all those in the other two groups.

TABLE 2. Observed and Expected Mortality from Leukemias Other Than Chronic Lymphocytic Leukemia in Three Groups of Massachusetts Towns, 1969-1986.[a] Data from Rothman *et al.* (1988)

	Five Coastal Towns[b]				Five Towns Closest to Plymouth[c]				Plymouth County[d]				Plymouth County Less 5 Towns	
Years	Obs.	Exp.	SMR	95% CI	Obs.	Exp.	SMR	95% CI	Obs.	Exp.	SMR	95% CI	Obs.	Exp.
1969-72	17	17.3	0.98	0.59-1.54	10	10.9	0.91	0.47-1.64	86	87.1	0.99	0.79-1.22	76	76.2
1973-76	14	19.3	0.72	0.41-1.19	7	12.9	0.54	0.24-1.07	80	90.7	0.88	0.70-1.10	73	77.8
1977-80	18	21.2	0.85	0.52-1.32	13	14.6	0.89	0.50-1.48	79	94.2	0.84	0.66-1.05	66	79.6
1981-86	34	35.5	0.96	0.66-1.34	26	25.0	1.04	0.68-1.52	128	152.0	0.84	0.70-1.00	102	127.0
1977-86													168	206.6

[a]Abbreviations: Obs., observed cases; Exp., expected cases; SMR, standardized mortality ratio (Obs./Exp.); CI, confidence interval (by exact method)
[b]Duxbury, Kingston, Marshfield, Plymouth and Scituate.
[c]Carver, Duxbury, Kingston, Plympton and Plymouth.
[d]27 towns, including all those in the other two groups.

TABLE 3 Results of Matched Case-Control Analyses: Estimated Relative Risks[a] of Leukemia by Exposure Level--Both Sexes Combined--Cases 1978-1986

Exposure Score	Cases	Controls	O.R. (CI)
low (<0.030)	18	56	1.00 (0)
medium (0.030-0.199)	50	106	1.97 (0.99, 3.95)
high (0.2+)	37	46	3.89 (1.74, 8.68)
Total	105		

chi square trend = 11.38, p = 0.001

[a]Odds ratios presented are controlled for age, sex, vital status, year of death, SES, smoking status, occupation and industry.

Table 2 of Morris and Knor , 1990)

Even if it is accepted that there is an association between leukemias and something in Plymouth, a causal connection can only be accepted if there is a cause. The reported release of radioactivity materials from Pilgrim were never enough to cause measurable radiation levels above the natural background radiation level and could not therefore have caused measurable cancer increase above background cancer levels. This is a robust conclusion and is independent of any particular relationship that is assumed between radiation dose and leukemia incidence. Anyone suggesting that Pilgrim was the cause of any of these leukemias must therefore postulate unreported and unmeasured release of radioactivity far exceeding the reported levels. Indeed, an examination of the BEIR V report (BEIR 1990), suggests that the exposure must be 200 rem* to each individual to quadruple the leukemia rate. If such unreported releases occurred (and that is very doubtful) they should be stopped. But they would not be stopped by the DPH recommendation to reduce the regulatory limit from its present value of 25 mrem. They must also postulate another reason for leukemia to be decreased overall (independent of location) so that the releases appear to leave the number of leukemias near Plymouth unchanged, while reducing them further away.

In this example, Dr. Sydney Cobb should be praised for raising the question, and postulating an explanation even though this explanation was subsequently shown to be invalid. However, the report by Morris and Knori was publicly released by the Massachusetts Department of Health in a press conference and television appearances by the Deputy Commissioner of Health (not the authors) just after his budget was cut. The budget was quickly restored.

*Absorbed dose is measured in Sievert (Sv); 1 rem = 10 mSv. Exposure is measured in Gray (Gy). For gamma-radiation the absorbed dose is numerically equal to exposure; in this case 1 rem = 10 mGy.

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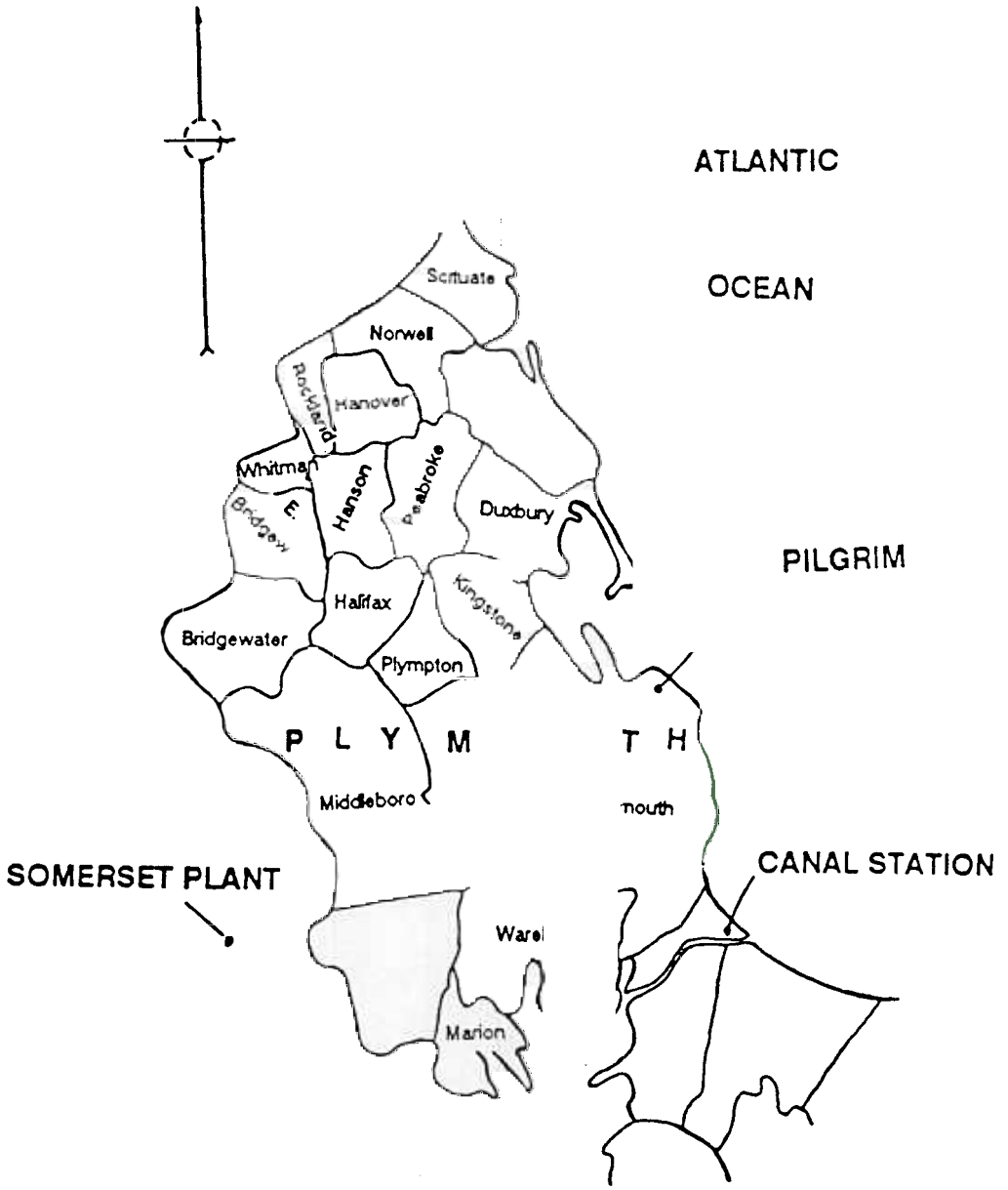


Fig. 4. Map of Coastal Area Around Plymouth

VII. DID THE CHERNOBYL ACCIDENT INCREASE U.S. MORTALITY?

Two reports by Gould (1986, 1988) have been widely publicized.

In the first of these reports, Gould *et al.* (1986) endeavor to see whether increases in overall mortality, total cancer mortality, and changes in fetal or infant mortality can be related, firstly to the presence of nuclear power plants in the state, and secondly to the radioactivity releases from these power plants. As an exploratory study, this is appropriate; but the words imply that the study is more than exploration. We shall assume that the arithmetic calculations are correct, and discuss whether or not they make their case. A statement such as "it is clear that emissions in the nuclear counties have an adverse effect on mortality particularly among the very young and very old" implies causality. We believe that neither this statement, nor the title "Nuclear emissions take their toll" is close to being justified.

Gould *et al.* first compare "Infant Mortality Ratio (IMR)", "Total Mortality Ratio (TMR)" and "Cancer Mortality Ratio (CMR)"* for nuclear states and non-nuclear states both for the years 1965-69 and 1975-82. They suggest, reasonably, that effects of nuclear plants would not be present in the earlier period. These are summarized in their Tables 3 and 4. They then note that the infant mortality ratio has fallen less in nuclear states (-3.95% annual rate) than in non-nuclear states (-4.33%) although the infant mortality ratio was still less in 1975-82 in nuclear than non-nuclear states. This is also true of total mortality. Cancer mortality increases in the nuclear states more than in the non-nuclear states and is larger in both time periods. They claim, and we have not checked, that these differences are statistically significant. (Here we note a point of presentation; these would be seen more clearly by means of a graph, with "error bar" corresponding to the $\sqrt{N}$ standard deviation, superimposed.)

Gould *et al.* do note that "there is no clearly defined tendency evident in Table 2 (of Gould *et al.*) among each of the so-called nuclear states to have increases in mortality that exceed those of the nation." (Gould *et al.* 1986, p. 5, first column). Another way of saying the same thing would be to say that the infant mortality declines are not distributed about the mean in a statistical manner and this, therefore, calls into question their use of the statistical criteria based solely on the number of persons and cases. One crude way of correcting for this would be to use the observed fluctuations in these parameters among nuclear states and the observed fluctuation in non-nuclear states instead of the square root of the number of cases. Then the statistical significance probably vanishes. Thus the only valid conclusion from the data that make up their Tables 3 and 4 is that while the data are consistent with the assumption made, they are very far from proving it.

*The "Standard Mortality Ratio (SMR)" in a given group is the number of deaths expressed as a percentage of the number of deaths that would have been expected if the age-and-sex-specific rates in the general populations had obtained. The "Cancer Mortality Ratio (CMR)" is the same, with "death" replaced by "cancer deaths."

Presumably because they recognize this, Gould *et al.* go on to look in closer detail at counties within 30 miles of a nuclear power plant. Again a slight difference is found. It is just significant (the probability that it is due to chance is less than one in 20), but Gould *et al.* do not ask how consistent this difference is among the various counties and we must again ask whether there are other causes of fluctuation than the square root of the number of cases. Thus the statement "it is clear that emissions in the nuclear counties have an adverse effect on mortality" (our underlining) is patently false.

Gould has been selective in his choice of items to consider. Just one illustrates a fluctuation in the opposite direction from Gould's argument. Boiling water reactors (BWR) release more xenon than do pressurized water reactors (PWR), as noted in Gould's Table 5. Yet the increase in cancer mortality from 1965-69 to 1975-82 (1.140) is less than that for PWRs (1.230) and less than the increase for non-nuclear counties.

In the report Gould *et al.* mention the noble gas releases, but do not discuss them or use them in a correlation. Yet in any assumed relation of health effects to nuclear power plants, the releases must be more directly related to the health effects than the mere existence of the power plant itself.

Even if consistency and statistical significance were clear, all the other issues in Hill's list would have to be addressed. We might still have a real correlation between one of the public health parameters and nuclear power plant location, but it is not necessarily a causal correlation.

If, for example, we compare the number of nuclear power plants in the country with expectation of life in that country, it is obvious that the expectation is higher in the U.S. with its many power plants than in Africa which has none. A priori, this increase of life expectancy near nuclear plants is as likely to be a direct causal relationship as the one Gould *et al.* propose. Few people believe that the nuclear power plants are a direct cause of the longer life expectation, however, and attribute the causal relationship to nutrition and good health care. These are related to prosperity, just as are nuclear power plants are related to prosperity, and prosperity is closer to being the true cause.

As one delves more deeply, Gould's case becomes even less. Although not explicitly stated by Gould, it seems that he is endeavoring to attribute the cause of mortality to an assumed radiation dose to human organs. Ideally, therefore, one would correlate cancer incidence with radiation dose. This information is hard to get, but one can imagine using human exposure, and calculate the dose to various human organs from the exposure. At this point we note that radioactivity releases have been measured. We know how to calculate exposure from releases. It is then easy to see that the radiation exposure will in all cases be much less than the natural background and less than the fluctuation and changes in natural background. Unless Gould *et al.* are prepared to claim and substantiate that the radioactivity releases have been grossly understated, or that we do not know how to calculate exposure from release, any case for causality stops at once.

Having shown that the statistical case Gould *et al.* present is weak and inconsistent and that it is not plausible based upon the comparison of dose and

background dose, we now complete the picture by suggesting a number of other possible causes for the effects which are much more plausible than radiation.

1 For infant mortality, fetal mortality and total mortality, Tables 4 and 5 of Gould *et al.* show that the rates in non-nuclear counties and states are now close to those in nuclear counties. This could be due to medical care "catching up" in rural states.

2. The larger cancer rates in nuclear states can be due to general industrialization.

In the second report, Gould (1988) was even less specific. He noticed that 33.06% of the 1986 deaths occurred in the U.S. during the months of May to August 1986 compared to 31.97% in earlier years. The difference claimed is small (although statistically significant). It might have any of a number of causes. Gould chose to suggest iodine releases from Chernobyl Nuclear Power Plant.

Taking Gould's specific suggestion of the cause first, we note that this suggestion satisfies almost none of Hill's requirements. The only one satisfied is temporality; the suggested cause does precede the effect.

Taking just one other requirement, we note that the iodine doses and doses from other radionuclides around the world from the Chernobyl plant release have been measured. The average first year dose to the U.S. was about 1.3 mrem compared with 60 mrem average in Italy and 40 rem for the 24,000 between 3 and 15 Km from the power plant (excluding Pripyat) (Goldman, Catlin and Anspaugh, 1987). The difference of the 1986 mortality in the US (33.06%) and the 1985 mortality (31.97%) is about 3%. If this was due to radioactivity from Chernobyl, and we assume linearity with dose, there would have to be a 415% (5.2 times the natural rate) effect in Italy and 2770 times bigger (2770 times the natural rate) in the area immediately downwind of the Chernobyl power plant. As shown in Table 4, these have not been seen. Thus the claim fails completely on the question of "existence of a biological gradient." This argument by itself should be enough to discredit the whole discussion. However, it was not enough to stop the Wall Street Journal dignifying Gould's claim by saying, in a column, that it had caused scientific controversy.

TABLE 4. Consequences of a Linear Biological Gradient in Gould's Predictio

	First Year Dose	Factor to Multiply by if Effect Proportional to Dose	% Increase
United States	1.3 mrem (Gould)	09	9
Italy	60 mrem (Calcul. here)	5.2	420
Persons 3-15 km from plant (not including Pripyat)	40 rem* (Calcul. here)	2770	277,000

cuation

A Seattle newspaper was better (News Tribune 1987). They discussed a part of this claim--that cancers in the state of Washington were caused by Chernobyl and clearly made the above point. Dr. Patricia Starzyk of the Washington State Department of Social and Health Sciences (Starzyk 1987) noted that mortality only rose 2% in summer 1986, not 9% as was alleged. This was not an unusual increase. Moreover, five traditional medical causes for summer increases have been identified: infectious disease; arteriosclerosis; chronic lung disease, suicide and diabetes.

However a more direct refutation of Dr. Gould's claim came from a Los Angeles Times reporter (Steinbrook 1988) who noted that Gould had used incomplete numbers. The 33.06% that Gould had stated as the fraction of U.S. deaths between May and August 1986 was incorrect. A more precise number is 32.2%, which is "identical to the data for the summer of 1984, and consistent with normal seasonal mortality patterns. The 1985 rate was 31.6%."

Another study (Brancker 1988) found no effect in Canada, although the effect on Canada should have been similar to that on the U.S. if Gould *et al.* were correct. In Canada deaths from infectious diseases remained steady while death rates among 25-34 year olds and among infants fell.

VIII. THE PORTSMOUTH SHIPYARD PROBLEM

In 1977, a Boston physician became concerned that there was an unusual number of cases of leukemia among workers from the Portsmouth Naval Base and suspected that radiation might be the cause. With the help of reporters from the Boston Globe, he searched through over 100,000 death certificates. He concluded that there were 22 leukemia deaths, whereas five should be expected using ordinary death rates. In a later scientific report (Najarian and Colton 1978), he changed this to 18 cases of leukemia and other neoplasms of lymphatic and hematopoietic tissue with 10 expected. Dividing these into cases among nuclear workers and non-nuclear workers on the basis of whether the worker wore a radiation badge, the number of "nuclear" cases is 10 with 2.9 expected (see Table 5).

TABLE 5 Observed and Expected Cancer Deaths Among Nuclear and Non-nuclear Workers by Type of Cancer

Malignancy	Nuclear			Non-nuclear		
	O	E	O/E	O	E	O/E
Leukemia	6	1.1	5.62	2	2.8	0.71
Other neoplasms of lymphatic and hematopoietic tissues	4	1.8	2.26	6	4.3	1.41
All other malignant neoplasms	46	28.6	1.61	80	72.6	1.10
Total	56	31.5	1.78	88	79.7	1.10

O = Observed cases; E = Expected cases.
(from Table II of Najarian and Colton, 1978)

Later it appeared that of the 10 nuclear cases, two had no radiation exposure. The effect was getting smaller as the data collection improved. Finally, Greenberg *et al.* (1985) showed that there was considerable under-reporting and misreporting of cases.

Najarian's observation was published in the medical literature (Najarian and Colton 1978), as is appropriate, even for case reports where statistical relevance has yet to be determined. But, he also published in the public press (Najarian 1978) in a way to arouse anxiety rather than information, and in Congress in a way that aroused disapproval even of liberal representatives. Congress requested a study by the National Institute for Occupational Safety and Health (NIOSH). A detailed study was made (Rinsky, Zumwalde and Waxweiler 1981) which found no statistically significant increase of leukemia among the shipyard workers. No effect was found in a subsequent "case-control" study either (Stern *et al.* 1986).

A number of possible sources of bias were discussed in a ater paper by Greenberg *et al.* (1985). These include:

(1) The healthy worker effect. Workers are more healthy than the average member of the population, so that comparing the deaths with those expected can understate the effect.

(2) Selection bias--which could occur in the selection of cases

(3) Measurement bias--which could result from a misclassification of the occupational exposure of those who died.

A more recent follow-up (Rinsky, Melius and Homung 1988) found a slight increase of lung cancer among the workers that was not statistically apparent in the first study. Many questions still arise. Can the increase be attributed to the Portsmouth shipyard? If it can, what about the shipyard could have caused the effect? Ninety percent of lung cancers are attributable to cigarette smoking, and cigarette smoking history is not detailed on death certificates, so that corrections for variation are hard to make. Rinsky, *et al.* concluded "This... suggests that radiation workers were more heavily exposed to asbestos and/or welding fumes than were other workers and that these exposures confounded the observed association between radiation and lung cancer."

Radiation *per se* is not known to be a major cause of lung cancer (although inhaled radon gas is), so that the original suggestion that radiation releases caused the cancers is not biologically plausible. Asbestos exposure does cause lung cancer, especially synergistically with cigarette smoking, and asbestos is common around ships and shipyards, so that asbestos is a likely cause of the increase. The increase was among electrical workers who were often exposed to asbestos. However, we make this statement without any specific knowledge of asbestos exposures at Portsmouth; it does, however, seem a cause more worthy of exploration than radiation exposure. This raises a question; why did Najarian immediately claim radiation as a cause of lung cancer when there were other, more plausible, causes?

Najarian has not accepted the criticisms implied in the NIOSH reports, nor those explicitly made by Hamilton (1983) (Najarian 1983). His last comment there suggests a reason for the concern which led to the article. "One wonders also how these risk estimates (if confirmed with other studies on similarly exposed people) might alter the thinking of those who are planning survival from nuclear war with similar product exposures."

We have commented on the way concern about these leukemias was brought dramatically to public attention. After the Boston Globe article, there was testimony in Congress and the NIOSH investigation which cost over $1,000,000. When the results of this became known, Senator Kennedy, not known for his support of either military or civilian uses of radiation, publicly condemned Dr. Najarian for unduly alarming shipyard workers and their families (Wermiell 1979). Other scientists were also critical (Hamilton 1983).

One scientist (Cohen 1983) has discussed the way in which this case was discussed in the press. He noted that in 1977-1978 there were 14 articles in the New York Times (several on the front page), mostly reiterating that there were a large number of excess cancers among the shipyard workers. In 1981, after the first NIOSH study was published, the New York Times published just one article, on page 32.

IX. LEUKEMIA AMONG THE HANFORD WORKERS

For many years, there have been studies of the health of workers at the Hanford Atomic Energy laboratory at Richland, Washington. No significant effect was found. However, in three papers, Mancuso, Stewart and Kneale (1977) and Kneale, Mancuso and Stewart (1981, 1984) claimed that there was an increase in leukemia and other cancers among those workers exposed to radiation (See also Stewart and Kneale 1991).

They compared the estimated (occupational) radiation dose which had been accumulated for patients who died of cancer, with the radiation dose who died of other causes. The "null hypothesis" that these doses are the same was tested. They found that the mean radiation dose for those dying of cancer was 1.38 rad and that for those dying of other causes was 0.99 rad. The implication was that the increase of 0.39 rad over about 10 years was the cause of cancer. This held for eight categories of malignant cancers, namely: multiple myeloma, pancreas cancer, brain tumors, kidney tumors, lung tumors, tumors of the large intestine, myeloid leukemia and lymphomas. This increase was said to be statistically significant. (The probability is less than 0.05 that it could occur by chance.) From these data they derived very small doubling doses for these cancers.

This work was reviewed by Gilbert and Marks (1979, 1980) Hutchinson *et al.* (1979), Hamilton (1980), BEIR (1980), Kleitman (1978), Mole (1977), Sanders (1978) and Speirs (1979). For example, Hutchinson *et al.*, found a statistical bias in the estimation of doubling dose; and made several important corrections to the data for various associated variables; calendar year of exposure, interval between beginning employment and exposure, interval between exposure and death and age at exposure to age at death. When this was done, there were two significant effects left; for myeloma, and for pancreas cancer, but not for other cancers thought to be radiogenic.

Unfortunately the description of the Mancuso, Stewart and Kneale paper was sufficiently obscure that they found a detailed line-by-line criticism was hard. This is important, because Mancuso, Stewart and Kneale use an unconventional method of analysis, and their results need, therefore, more than a cursory justification. Further analysis, using a different method, were then made by Kneale *et al.* (1981, 1984). They still claim a radiation related effect. This in turn was criticized by Gilbert *et al.* (1989) who also studied mortality over an extended period 1945 to 1981.

We emphasize here the statistical importance of making the corrections for associated variables. If they are properly made, the statistical fluctuations will become the only fluctuations of importance.

We look carefully at the 1984 paper of Kneale *et al.* They grouped the cancers into two groups; group A which are claimed to be cancers in tissues where previous studies had found that radiation produces cancers (radiosensitive tissues), and group B in tissues where radiation is not known to cause cancer (non-radiosensitive tissues). In Tables 6a, 6b, and 6c the observed and expected cancers are tabulated for several dose groups. The observed number of cancers were less than the expected at high doses for group B (Table 6a) and more than expected at high doses for group A. Does this mean that radiation is sometimes

TABLE 6a. Comparing Cancers of Radiosensitive Tissues (Group A) With Survivors After Control for Job Risks and Obvious Factors With No Allowance for Latency or Age Effect

Dose (rem)	Observed	Expected	t-value
0.00	112	119.0	-0.79
0.01-0.07	70	72.0	-0.27
0.08-0.31	102	104.4	-0.27
0.32-0.63	89	78.9	+1.26
0.64-1.27	94	88.6	+0.64
1.28-2.55	70	80.4	-1.33
2.56-5.11	39	38.0	+0.18
5.12-10.23	28	25.0	+0.65
10.24-20.47	17	17.6	-0.14
>20.48	22	19.2	+0.71
Summary t-values	Rank weighted +0.95 Dose weighted +0.77		

From Kneale et al. (1984).

Table 6b. Comparing Cancers of Nonradiosensitive Tissues (Group B) With Survivors After Control for Job Risks and Obvious Factors

Dose (rem)	Observed	Expected	t-value
0.00	96	83.7	+1.71
0.01-0.07	39	45.0	-1.00
0.08-0.31	62	66.3	-0.60
0.32-	57	49.3	+1.23
0.64-	53	54.1	-0.17
1.28-	45	42.2	+0.49
2.56-	21	19.1	+0.47
5.12-	7	12.7	-1.70
10.24-	6	8.3	-0.83
>20.48	4	9.4	-1.93
Summary t-values	Rank weighted -2.33 Dose weighted -2.69		

From Kneale et al. (1984).

TABLE 6c. Comparing Cancers of Radiosensitive Tissues (Group A) With Survivors After Control for Job Risks and Obvious Factors With Allowance for Latency and Age Effect*

Dose (rem)	Observed	Expected	t-value
0.00	114	125.4	-1.31
0.01-0.07	95	100.6	-0.67
0.08-0.31	126	110.9	+1.70
0.32-0.63	43	56.2	-1.93
0.64-1.27	54	53.5	+0.08
1.28-2.55	47	44.4	-0.44
2.56-5.11	32	34.2	-0.41
5.12-10.23	26	20.5	+1.31
10.24-20.47	19	12.1	-2.09
>20.48	16	14.3	+0.52
Summary t-values	Rank weighted +2.44 Dose weighted +1.88		

*Allowance for age effect by increasing dose by 10% for each year after age 40.
From Kneale et al. (1984).

good for you? This unlikely conclusion is obviated by noting that there are several biases which can be collected together and are called the "healthy worker" effect. It is well-known that employed people are healthier and have a lower mortality rate than unemployed people. Obvious reasons for this are numerous and include:

employers only employ healthy workers
someone with a job eats better than someone without a job

but on the other side:

executives have more strain

It was plausibly suggested (but without proof) that those who had high radiation doses were often professionals with higher income and probably better health. Then, it is the difference in the trend with dose between the A cancers and the B cancers that is important. Kneale *et al*. related the reductions in group B (shown in Table 6b) with increased radiation, to a similar, more significant reduction in total death rate. In Table 6c are presented the data of Table 6a corrected for lateness and length of employment.

We note here another possible reason for finding spuriously significant results. The radiation exposure was measured by dosimeters and film badges, which were worn only at work, and therefore, exclude most of the natural background exposures. If we omit radon exposure, and ignore any discussion of the lung cancer that radon might produce, the average radiation exposure at sea level is about 100 mrem at sea level plus 95 mrem X-ray exposure (see Table 11 in Sect. IXX). In a typical 10-year period, this is 2 rem (200 mSv); comparable to the typical occupational radiation exposure and greater than the 0.39 rem difference between cancer victims and others. If proper correction is not made for this, spurious results can ensue. In principle, the comparison of exposed with non-exposed workers, corrects for this, if the background and medical exposures are the same in each group. But the variations of the medical and background exposures still persist, and reduce the statistical significance of any answer.

One obvious conclusion exists. Lawyers and bureaucrats have often insisted on extra medical checks for radiation workers. One of us (RW) for example, was asked to take an extra chest X-ray for a summer job involving radiation. His film badge (deliberately worn during the X-ray) showed the highest reading for anyone in that laboratory. It is not possible to correct for effects such as these now. But an estimate can be made that in the early days of Hanford photofluorographic exposures of about 600 mrem per year were given. This exceeds 15 fold the radiation difference of Mancuso, Stewart and Kneale. In such circumstances it would seem mandatory to discuss whether these background environmental and medical exposures can bias the data or increase the fluctuations and therefore statistical significance. Mancuso, Stewart and Kneale did not discuss this; it must be presumed that they had not thought about it, and little credence can therefore be given to the small barely significant effect found in their analysis.

Fig

Nonetheless we examine their data further by plotting them in Figs. 5 and 6. Figures 5a and 5b show the ratio O/E (observed cancers/expected cancers) of Table 6a and 6b. The statistical uncertainty is also plotted. The computer fitted

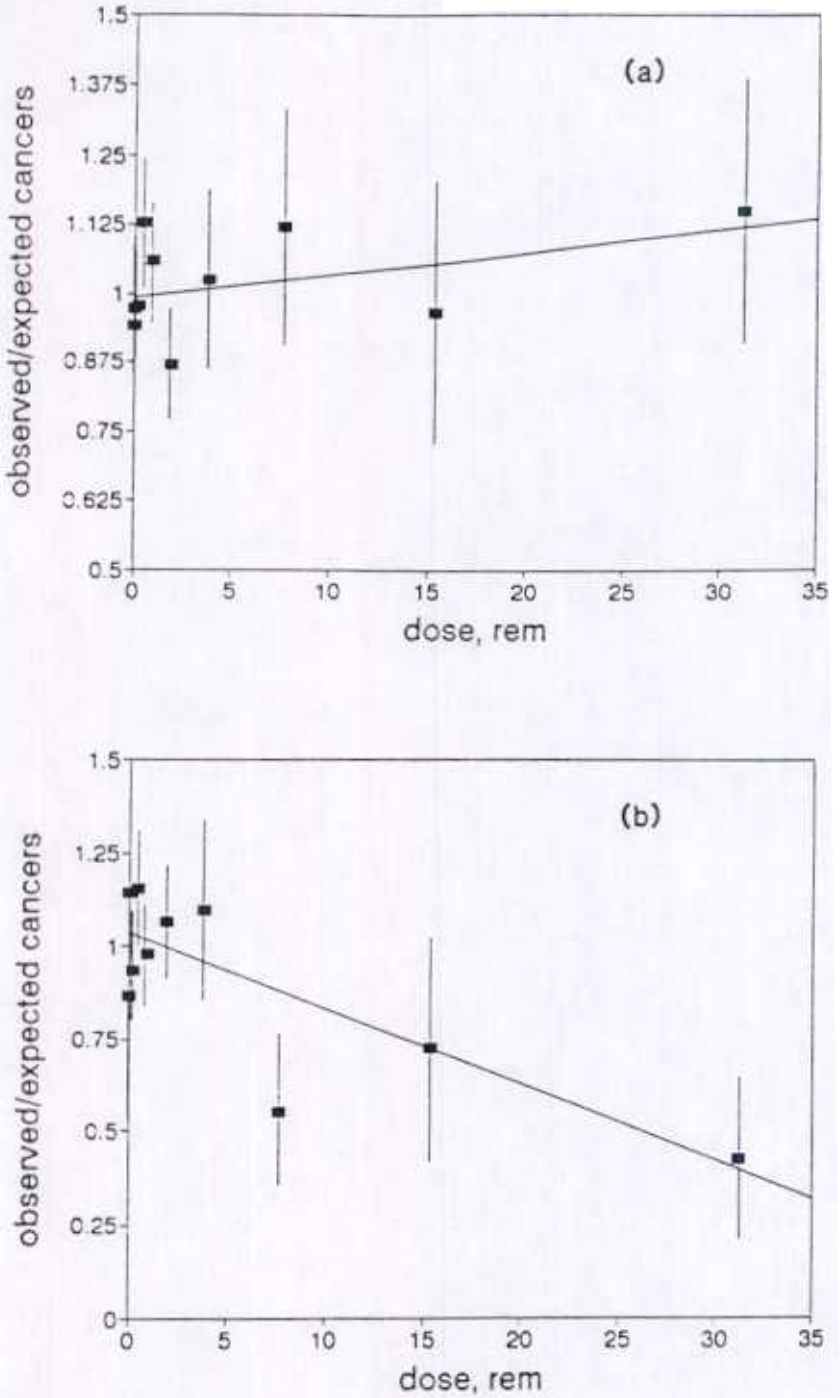


Fig. 5. Cancers among Hanford Workers. a) Ratio of Observed Cancers of Radiosensitive Tissue and Expected vs Dose and b) Ratio of Observed Cancers of Nonradiosensitive Tissue and Expected vs Dose

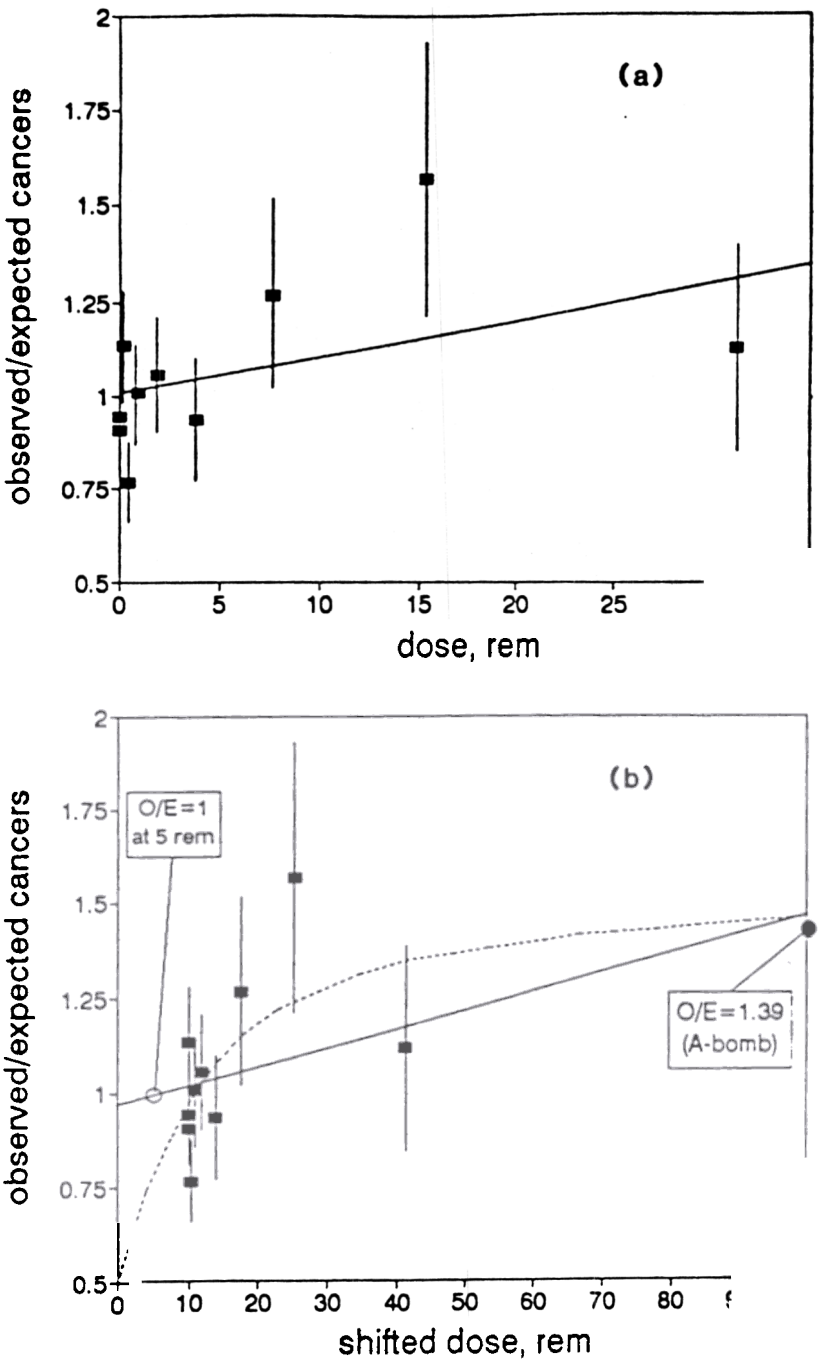


Fig. 6. Ratio of Observed Cancers of Radiosensitive Tissue to Expected After Correction. a) vs Additional Dose and b) with Dose Scale Shifted.

line was calculated without considering these error bars, and assuming that all points are equally weighted--which is approximately true. Although this line goes through more than 2/3 of the error bars (which is all that is required of an adequate fit) we can see clearly the suggestion of Kneale *et al.* that the data rise faster with dose at low doses for Fig. 5a. We note that our plotting of "error bars" and their use, is similar to the use of "t-statistics" used by Kneale *et al.*

Figures 6a and 6b plot the data from Table 6c which are corrected for Figs. 6a and 6b latency and other factors. Again Fig. 6a shows a possible rapid increase at low doses. But on Fig. 6b, we replot the same data against total dose, and not merely the occupational dose. The origin is shifted to 10 rem, being 5 rem extra medical X-rays and 5 rem lifetime environmental background. Since the expected numbers come from people with similar environmental backgrounds, the fitted curve should go through (or at least close to) O/E = 1 at 5 rem. Also on the plot is a point with O/E = 1.39 ± 0.04 from a fit to the data for all malignant neoplasms in atomic bomb survivors (Shimuzu *et al.* 1988 Table 2A). The fitted line is not a bad fit to the data, but Kneale and others' rapid increase starting at 10 rem (shown in a dotted line) now seems less plausible because a simple plot would imply that half of all cancers are caused by radiation. However, we should consider this dotted line as a postulate for further study. Are other data consistent with this line? We return to this when we consider variatins of cancer rate with natural background in Fig. 19.

There is one more feature of the Mancuso, Stewart, and Kneale analysis that deserves mention. The differences in Figs. 5a and 5b between cancers of radiosensitive tissue and non-radiosensitive tissue used an old, inaccurate, ICRP classification. If the effect is really due to radiation, this difference should increase when a more modern classification is used. Oral statements have been made at conferences that the effect vanishes. This should be documented.

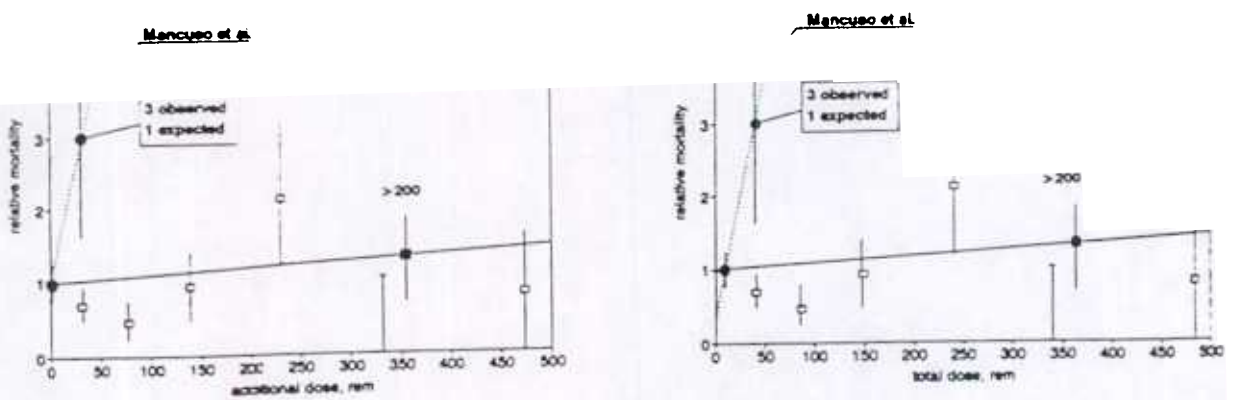


Fig. 7. Mortality from Cancer of the Pancreas Among Japanese A-bomb Survivors. a) Among Hanford Workers and b) According to Mancuso Analyses.

X. DOES PLUTONIUM FROM ROCKY FLATS CAUSE EXCESS CANCER?

The Rocky Flats facility, 15 miles NW of Denver, is used to machine plutonium for manufacture of U.S. nuclear weapons. As plutonium metals are machined, fragments can catch fire and vaporize. Extreme care must be, and is, therefore, taken. However, two fires broke out in 1957 and 1969, and although they were contained, plutonium was found to have contaminated the soil in regions SE of the facility towards, and including, Denver from an oil cleanup in 1968. Figure 8 shows the distribution of this contamination.

Johnson (1981) and Chinn (1981) examined cancer rates in these areas for the years 1969-1971, and found that total cancer rates in the areas closest to the plant (area 1) were 24% higher for males and 10% higher for females than in areas of the Denver area further away. He attributed the increase to plutonium.

Plutonium is an alpha emitter, and the cancers should, therefore, arise close to where the plutonium is absorbed--the lung, if it is inhaled, and the liver and bone if it is absorbed. One should expect more plutonium in the bodies of those with cancer than in others. Also, we should expect the trends to be found at other time periods.

Crump *et al.* (1987) examined all of these questions. Firstly, they confirmed the statistically significant trend found by Johnson for total cancer, digestive cancer, respiratory cancer, and cancers normally considered radiosensitive (for whole body radiation). However, they found less of a trend for the years 1979-1981. This is the opposite to what one would expect. 1979-81 is after the

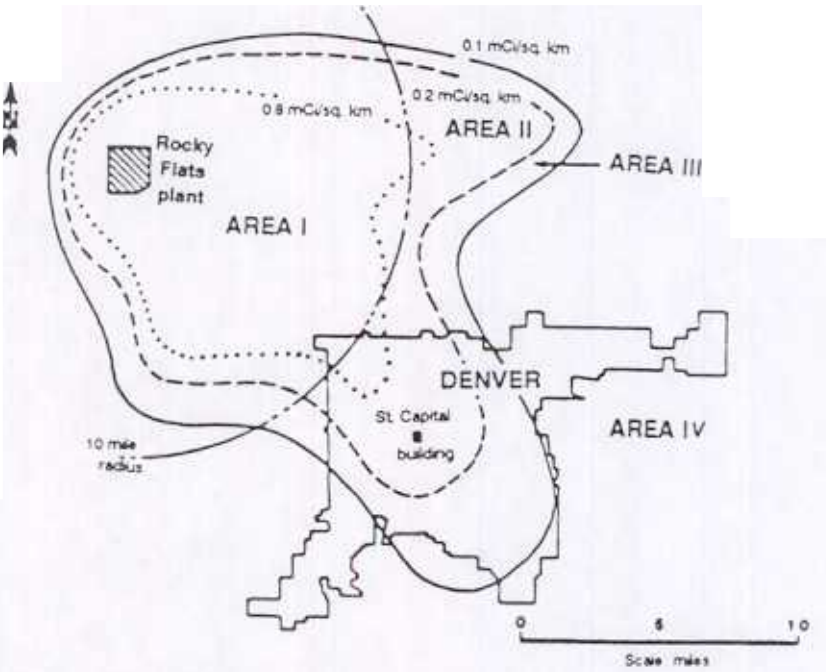


Fig. 8. Map of Area Around Denver and the Rocky Flats Plant Showing Plutonium-in-Soil Isoconcentration Areas. Source: Cohen (1980)

latency period for all cancers, whereas 1969-71 is in the latency period for some of them if the initiating event was plutonium. No excess of bone cancer was found, contrary to the presumption.

Finally Cobb *et al.* (1982) found no increase of plutonium in an autopsy of some (but not all) of the cancer victims. None of these fit the hypothesis that plutonium from Rocky Flats was the cause of the cancer increase. However, another, much more plausible cause for the cancer excess can be found. Crump *et al.* (1987) noted that there is an increased rate of many cancers in urban areas (Goldsmith 1980). This is called the urban factor. Crump *et al.* corrected the data for the "urban factor" by looking at the distance from the Colorado State Capitol in Denver. Many persons in Group I are closer to the state capitol than persons in Group IV.

Johnson (1987), in response, called into question each one of Crump and others' arguments. He pointed out that the autopsy results were only from a selection of the cancer victims and perhaps a biased selection. Crump found fewer cancers during 1979-81 in area I than area II; but Johnson noted that this was probably due to a large influx of new population into area I who had not been exposed.

But Johnson failed to describe an effective and complete model for the cause of the cancers and its relationship to other knowledge as Crump et al. have done. Therefore Crump and others' explanation must be preferred.

XI. IS THERE A PRECURSOR TO LEUKEMIA?

It is common to believe that the cause-effect relationship in disease etiology is unique; the effect will always be an outcome of the cause. When people are given a large dose of a strong poison like strychnine, they will always die. If they are given a small dose, they will always live. In between, some will live and some will die, and the difference is assigned to a variation of individual sensitivities.

It is tempting to try to find the same behavior with cancer-causing agents. But in general, it does not seem to work. Of heavy cigarette smokers, one out of five will develop cancer due to their habit; but four will be unaffected, and we do not know which. Does that mean that one of the five is especially susceptible, and the others are not? If so, diligent search might find the cause of susceptibility. However, we have, so far, not uncovered these reasons for especial susceptibility, and we do not know whether these reasons are unknowable or merely unknown. However, for practical purposes this distraction makes no difference.

This may appear callous in that it seems to ignore the need of the susceptible individuals. But an illustration shows that it is, in fact, in accord with a common sense approach to risks that society often has. Suppose we consider the chance of being killed in an automobile accident. We observe one accident where a car of Ontario license 423 KBT kills a pedestrian. If we knew in advance that this might happen, we would stop the car at the Canadian border--

and avert the accident. But we have no way of knowing in advance, which car (if any) will cause an accident. Yet if we knew enough detail, and were able to calculate the consequences of this detail, it might in principle be knowable. We therefore describe the possibility as a risk, and society accepts the risk, because prevention is not possible without draconian measures such as stopping all cars.

Physical scientists who have gotten used to the Heisenberg uncertainty principle, have little trouble in accepting this argument. Medical scientists more often have problems and continue to search for precursors to these seemingly random events--such as the occurrence of cancer.

There are some precursors to cancer that can be taken into account. There is a synergistic relationship between cigarette smoking and asbestos; the probability of getting lung cancer (at high doses) is proportional to the product of number of cigarettes smoked and the asbestos exposure. Therefore it is possible that anyone exposed to asbestos can reduce the chance of developing lung cancer if he stops smoking. Retinoblastoma, a rare cancer of the eye, runs in families and presumably is genetically caused.

Whether some objective ailments are precursors to cancer has been discussed both for asbestos and benzene. This, however, is usually considered to give suggestions about the shape of the dose-response relationship. Thus the U.K. chief inspector of factories Dr. Merriman, (1938) asked "Does silica, or asbestosis or the fibrosis of the lung they produce tend to inhibit cancer of the lung or to produce it? If the latter, do either of these substances act as specific carcinogenic agents like tar, or is it that the disease they produce only prepares the soil for the occurrence of cancer? With asbestosis, among 103 fatal cases in which asbestosis or asbestosis with tuberculosis were present, cancer of the lung was associated in 12 cases (11.6%)." If asbestosis is necessary for lung cancer incidence, the dose response relationship might show a threshold. This question is still largely unanswered today.

In studying leukemias produced by benzene, Goldstein (1977) commented upon the fact that pancytopenia often precede leukemia, although some cases of leukemia have occurred without a preceding diagnosis of pancytopenia. But because of the limited medical information in the individual cases, undiagnosed pancytopenia could always have preceded it. (See also Lamm *et al.* 1989).

In a series of papers, Bross and Natarajan (1977) Bross *et al.* (1979) and Bross and Natarajan (1980) make a pioneering attempt to identify persons especially susceptible to leukemia. They choose as a database the Tri-State Survey, carried out in certain specified areas of New York, Maryland and Minnesota. (Graham *et al.* 1963, Gibson *et al.* 1968). They first concentrated on childhood leukemias.

Other authors have found an association between childhood leukemias and X-rays during pregnancy of the mother (Stewart and Kneale 1970, MacMahon 1963). This association does not, in itself, tell us whether X-ray radiation causes these leukemias, or whether another agent, which caused the leukemia made the X-ray more likely. Even now, this is disputed (MacMahon 1989). Such an effect was also found in the Tri-State Study (Gibson *et al.* 1968).

Assuming that the cause of these leukemias was intrauterine radiation, Bross and coworkers set out to discover whether there were precursors. They found that several ailments were associated with the leukemias; a virus (red measles or chicken pox); bacteria (whooping cough or dysentery); and allergy (asthma or hives). This is displayed in Fig. 9. This is a highly suggestive finding. But it was initially and properly addressed with caution by Bross *et al.*

The existence of an association in this dataset, between two apparently unconnected end points such as virus and leukemia, does not prove causation; the correlation may not persist to other data sets. Moreover, even if it does, one cannot infer unequivocally that viruses cause leukemia, or make people more susceptible; it might be that a latent leukemia makes one especially susceptible to viruses. These points were brought out by Rothman *et al.* (1988). It is also unclear that this association, even if a causal correlation, has any predictive ability. The other associated ailments seem only discoverable after the intrauterine radiation had already taken place.

The argument is very similar to that of Feynman's example. There was an association (and as noted, some call it a correlation) between the particular license plate and the parking lot. Few believe that whenever one has a parking lot, one will see that license plate; or whenever one sees that license plate, it will shortly be in a particular parking lot. In Feynman's example, we can easily repeat the observation on other days and other places--and our informal checking of this kind, is what convinces us that the association is unique to this particular parking lot or the particular time.

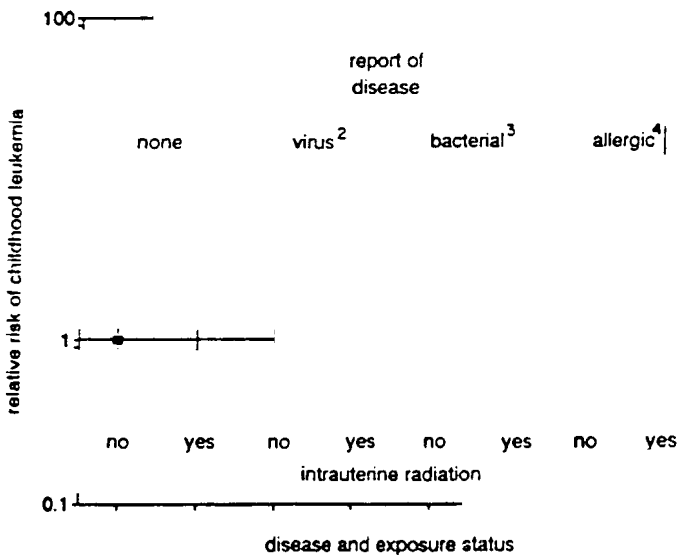


Fig. 9. Approximate Confidence Intervals on the Relaxative Risk of Childhood Leukemia (Age-Adjusted Risks in Relation to Children Not Exposed to Intrauterine Radiation and Without Report of Specified Childhood Disease). According to Exposure to Intrauterine Radiation and History of Disease. 1) No Report of the Specified Diseases, 2) Report of Red Measles or Chicken Pox, 3) Report of Pneumonia or Whooping Cough or Dysentery, and 4) Report of Asthma or Hives.

Source Bross and Natarajan (1980)

Bross and Natarajan must have been aware of these arguments when they stated "a formal objective test of the 'susceptible' hypothesis requires exclusive information on medical history and exposure to potential hazards on a large series of cases of leukemia and controls representatives of the general populations." But Bross *et al.* did not, unfortunately, look at other situations themselves, nor did they put their effort into encouraging others to do so. Instead, they put their effort into arguing for a change in radiation safety regulations, which most scientists regard as premature. To the best of our knowledge and belief no one else has tried to extend these studies to other cohorts.

Bross, Ball and Fallen (1979) claim that the Tri-State Study also shows that diagnostic X-rays affect adult leukemia and heart disease. They write down a model to evaluate a dose-response curve for those persons most "affected" by radiation. In one figure they show the number of persons "affected" as a function of dose. It is not clear how this is derived since details are not provided. It seems likely that this is merely a plot of excess leukemias verses dose, with the ordinate changed by an arbitrary assumption that only a small fraction of persons are "affected" by radiation.

Even here, however, their claim that these demonstrate a response relationship that is very nonlinear near the origin, in the direction that there are more leukemias at low dose than calculated, cannot be sustained by the data, and they themselves comment that a linear fit cannot be excluded.

Boice and Land (1979) specifically review the work of Bross *et al.* (1979). They point out that conventional analyses find of that radiation, and presumably X-rays, can cause adult leukemia, a causal connection with heart disease has not been established. Such an association could be due to leukemia and heart disease patients receiving more intense clinical examination.

Bross also looked at other radiation issues and reached conclusions that differ from the usual ones. Bross and Natarajan (1980) and Bross (1983) reanalyzed the work of Schull, Otake and Neel (1980) on genetic effects of the atomic bomb explosions in Japan. Schull *et al.* had concluded that "in no instance is there a statistically significant effect of parental exposure." Bross and Natarajan claimed the data shows that there is. Bross' claim was looked at in its turn by Hamilton (1983) and Hamilton et al. (1983). Hamilton shows that Bross was guilty of post hoc grouping of data--a variant of the Feynman trap. In particular, he included a zero dose group in among a group exposed to 0-9 rad.

We also note that all these authors discuss excess cancers due to X-ray doses. As noted in the preceding sections, the X-ray dose is superimposed upon a natural background, and the full biological dose response curve must include the effect of natural background. A kink in the curve just above the dose that corresponds to the natural background does not, in this context, seem very plausible.

XII. CANCERS NEAR THREE MILE ISLAND

After the accident on 28 March, 1979 at the second unit of the power plant at Three Mile Island (TMI) near Harrisburg, Pennsylvania, there was considerable public concern about a possible increase of cancer because of radioactivity releases. This concern was not allayed by the official report, agreed to by six federal agencies, that radioactivity releases were primarily noble gases, and that the radiation doses were very small (NUREG 1979). The biological plausibility of an effect due to radiation is small.

However, an accident of this nature causes unusual stress and stress has often been claimed to be a cause of cancer. This is, for example, found in animal bioassays where such "trivial" matters as size of cages, or possibly lighting, seems to affect the background cancer incidence (Crouch and Wilson 1987). The Kemeny Commission (Kemeny *et al.*, 1979) suggested that if any extra cancers appeared near Three Mile Island stress would be the most likely cause. There is, therefore, a plausible reason for a search for cancers near Three Mile Island.

Most of the studies were anecdotal (Wasserman, 1987). We comment here on one which was more detailed. Two persons, Aamodt and Aamodt (1985) claimed an excess of leukemias around Three Mile Island. They claim 20 cancers from 1979-1984 and 19 between 1980 and 1984 in a population of 443 (433 listed but this was an addition error) for a ratio of cancer mortality to expected of 6.57 (corrected from their 7.13) with an uncertainty of ±1.5. This claimed effect is large enough that it led to a more detailed study by Public Health for the Commonwealth of Pennsylvania (Tokuhata and Dignon 1985). They showed that Aamodt and Aamodt fell into the Feynman trap. Aamodt and Aamodt surveyed an area of Newbery Township, but arbitrarily selected 4 out of 14 streets. They failed to show, and could not show, that these streets were selected before there was knowledge of leukemias, or that there was some objective way of selecting them (such as being all the streets within a given distance from the plant). In the 10 streets not included, there were no cancers. This gave an artificially large ratio. Tokuhata showed that if a proper selection of an area was made, then there was no excess of leukemia at all.

That the Aamodts found there are more cases in these streets than average then becomes a logical tautology and no more surprising than the fact that Feynman's car had the particular license plate it happened to have. It is a lot of work to discover biases such as this; it often involves redoing the study completely, but properly.

We also note that radiation cancers manifest themselves with a 5-20 y latency after exposure, so that cancers so soon are doubly implausible. On the other hand, the absence of extra cancers also tells us little because they would not be expected for 25 y.

A review of health effects around TMI has been prepared by Behling and Hildebrand (1986).

XIII. DID ATOMIC TESTS INCREASE CANCER IN UTAH?

Between the years 1950-1960 there were many atomic bomb tests in Nevada, and there was some exposure of communities downwind in Utah. Lyon et. al. (1979) studied leukemia in children between 0 and 14 years of age, who lived in Utah between 1959 and 1967. They compared the leukemia rate with that expected in the general U.S. population. They particularly looked at those children born between 1951 and 1958 (which they called a high exposure cohort) and who lived in counties where they claimed that the fallout was the greatest. Low exposures were defined as those born between 1944 to 1950 (before the tests), and 1959 to 1975 (after the tests were over).

Their analysis compared leukemia in the exposed and the control group. They chose two control groups; the pre-exposure cohort whose members were born between 1944-1950 (and were therefore unaffected by the later tests) and post-exposure cohort born, 1959-1975 (and therefore unaffected by the earlier tests). They chose to compare with these control groups rather than with average U.S. incidence, because "for reasons unknown, leukemia mortality among the low-exposure cohort in the high-fallout counties was about half that of the United States and of the remainder of the state." The data for the three cohorts for the various counties is shown in Fig. 10 drawn from their data (Table 3 and Fig. 1 of Lyon *et al.*, 1979). The data for the high fallout counties show a marked increase (doubling) for the high exposure cohort. We added error bars to their figure (corresponding to the square root of the expected number); these make the uncertainties evident and the data far less convincing. We note that the fluctuations down from 14 cases expected to 7 observed is more likely than a fluctuation upward from 7 expected to 14 observed.

In their Table 4, Lyon *et al.* produce a single summary statistic as follows. They compare the leukemias in a "high exposure" cohort with those for the "low exposure" cohort (defined as above by the time of leukemia), by deriving a standardized (leukemia) mortality ratio. For the high fall-out counties, SMR =

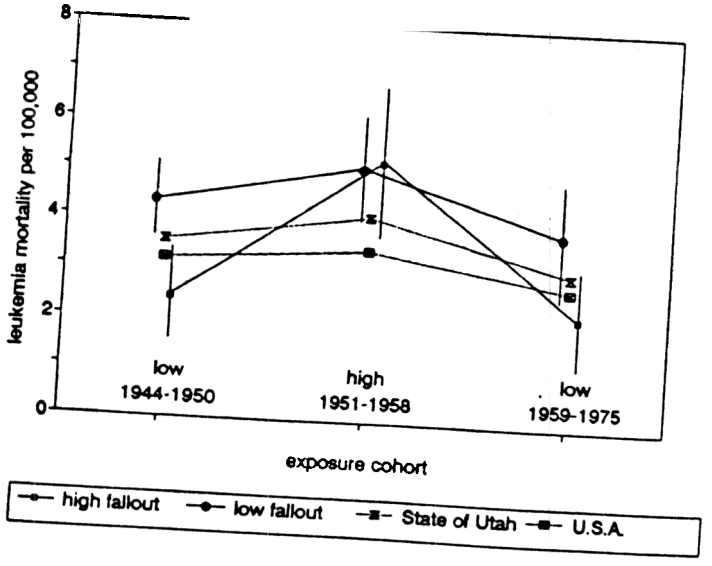


Fig. 10. Adjusted Leukemia Mortality Rates per 100,000 Males and Females

2.44 with 95% confidence limits 1.18 to 5.03. This, then, was their evidence for an effect due to some difference between the two group of counties.

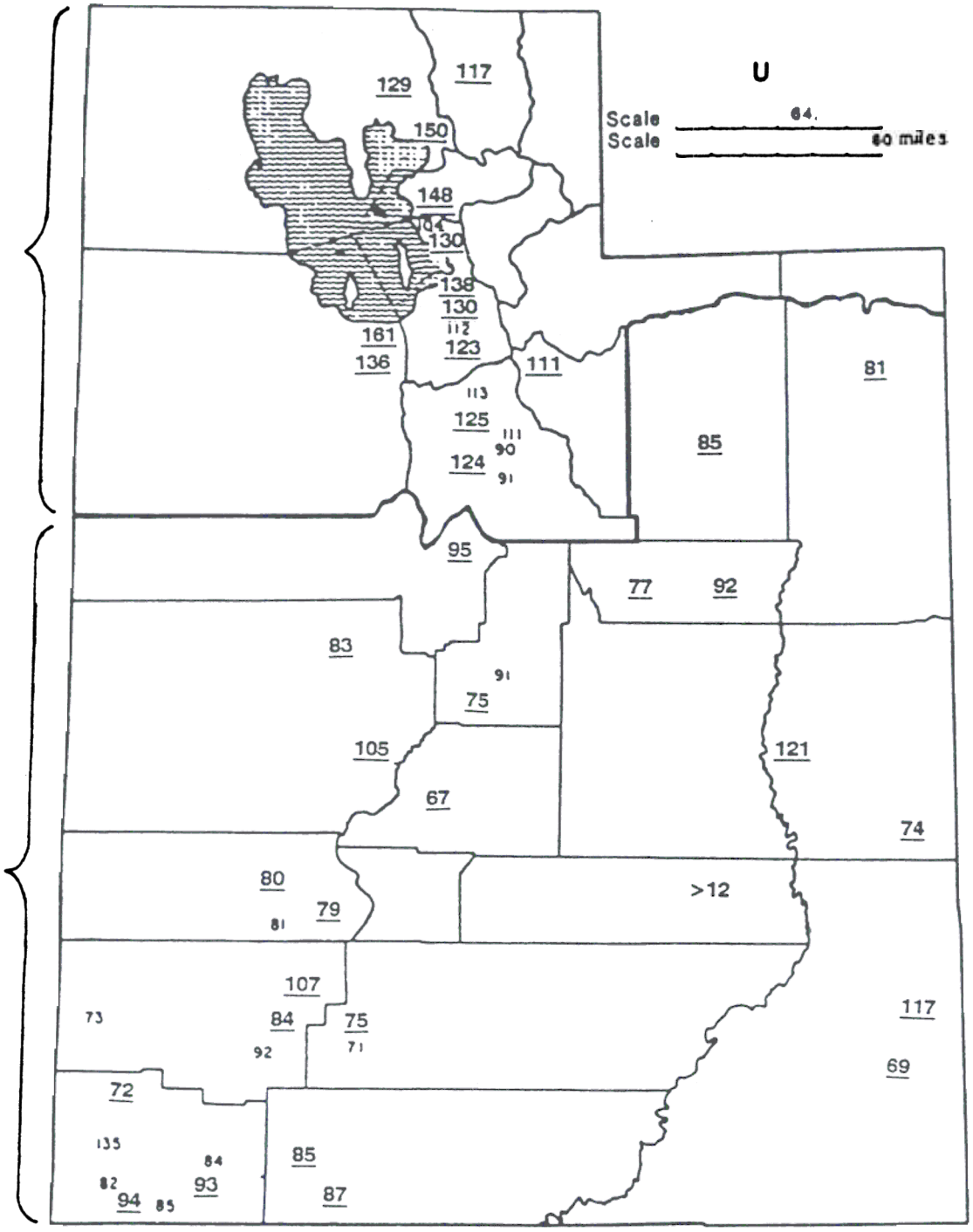
This procedure would, formally, be statistically valid if this combination had been chosen in advance and if we were absolutely sure that there were no other confounding effect or fluctuation. Why not compare the leukemia incidence only to the U.S. incidence? Indeed, Hamilton (1983), Land (1979) and Engstrom (1979, 1980) all concluded that this combining of groups was arbitrary. Even if not arbitrary, it is still susceptible to two meanings. One, the final conclusion of Lyon *et al.*, is that relative excess in the high fallout counties was due to some external cause, such as radiation, another is, that the relative deficit in the controls for the high fallout counties was caused by whatever caused the reduction below the U.S. incidence (perhaps low reporting for the early time period). Nothing in the data helps us decide between these two explanations. However, the second is more plausible because it fits better into the general body of scientific understanding (Lyon *et al. 1979*; Hamilton 1983).

Another more telling argument comes from the actual measurements of fall-out (Cs-137 and Pu-239) on the ground in Utah. Figure 11 shows the results of Beck and Krey (1983). Superimposed on this map is the line separating the "high" and "low" fall-out counties of Lyon *et al.* (from their Fig. 3). It appears that this was based on the single "smoky" shot of 31 August, 1957). It is clear that some of Lyon's low fall-out counties actually had a higher fall-out than many of the high fall-out counties! Any assignment of the effect to radiation from fall-out becomes harder to sustain.

This, however, is not the end of the story. Johnson (1984) looked at Washington county in SW Utah which is the closest to the test site, (and includes the largest town of St. George, Utah). He found 19 leukemias in 1958-1966. This was more than expected and gave a risk ratio of 5.28 (95% confidence 3.18-8.24). Machado *et al.* (1987) repeated this study and found a smaller effect; 62 leukemias between 1955 and 1980, and a smaller risk ratio of 1.45 (95% confidence 1.18-1.79). Johnson noted in an oral report that Washington county had the lowest leukemia rate in the state.

It appears, therefore, that there is a small cluster of childhood leukemia cases in SW Utah for the period 1951-1960 which was the cause of the original claim. This conclusion comes out clearly in a most careful case-control study by Stevens *et al.*(1990). They considered 1177 victims of leukemia, who (a) died between 1952-1981, (b) were born before 1959, (c) were Mormons (members of the Church of Jesus Christ of Latter-day Saints) or spouse or one parent were Mormons, so that church records could be used. These cases were compared with 5330 controls. Total bone marrow dose was computed from residence information and deposition on external surfaces (primarily Cs^{137}) as measured by Beck and Anspaugh (1990) following the earlier work by Beck and Krey (1983). This exposure analysis found a high average bone marrow dose for those in the SW corner of the state (Wartington County containing St. George) where the dose was 19 mGy (1.9 rem) between 1952-58.

The bone marrow rate by county is shown in Fig. 12. We note that this seems inconsistent with a naive look at the map of Fig. 11 from Beck and Krey (1983). This deserves further explanation. The principal result is that for 17 leukemia cases (except CLL) in this high exposure region, there was a risk ratio of 1.72 (95% confidence 0.94-3.12). Five were cases of acute leukemia between 0-10 y,



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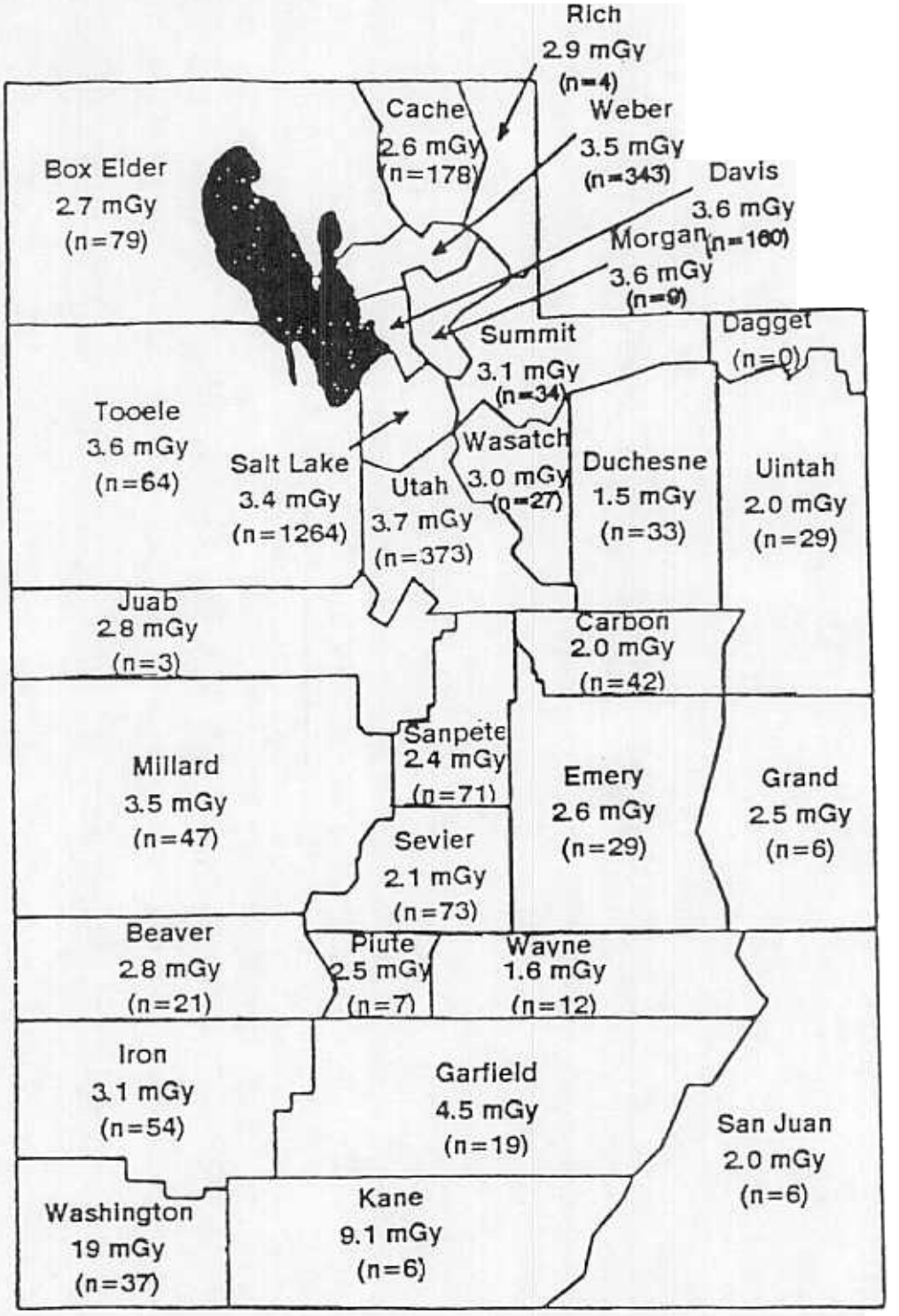

Fig. 12. Fall-out Map (Fig. 1) from Stevens et al.

and for them the risk ratio was 7.82 (95% confidence 1.9-32), which is significant (p=0.02). The significance increases (p=0.009) when there is a restriction to acute lymphocytic leukemia. There was no elevated risk ratio for doses up to 5.9 Mgy (0.59 Rem).

At this altitude and in this general area, background doses are high. The average background bone marrow dose is 70 mrem/y in SW Utah. Over a 20-y period, this gives as much radiation as the addition from the bomb tests. Fluctuation in background should not affect the results so long as they are not correlated with the study group. Stevens *et al.* looked for plausible reasons for higher background in Washington County than the rest of Utah, but found none.

Moreover, case-control studies, by themselves, do not prove causation. A cohort study seem impossible here, but a careful connection to other data is necessary. In particular, if there is a linear dose-response relation, and the risk ratio of 7.8 for acute leukemias 0-19 is to be believed, one should also find a marked increase of leukemias in those western states with a high background compared to eastern states, provided that other factors can be corrected. No such increase has been found, and indeed Washington County has a low background leukemia rate, but this may be due to other compensating factors of urban environment or life style (alcohol, tobacco, and coffee).

Finally, we should learn from this that Lyon incorrectly drew conclusions in his original paper; although the conclusions were not necessarily incorrect. The more careful look at the data by Stevens *et al.* pulls out a small group of people that need close examination. Such close examination might include measurement of the concentrations of 137Cs at each residence directly, and also measurement of other background doses both of radiation and chemicals.

One scientist, born and raised in the small town of St. George, noted that he was aware of most family names in that small town, and recognized none of the names of the leukemia victims (Everett 1991). This suggests a peculiarity that deserves investigation; perhaps they come from some farming group exposed to some other agent.

The conclusion that there is an association of leukemia with fallout therefore rests on the 17 cases in Washington County, and in particular the cluster of five who had acute leukemia at a young age.

XIV. LEUKEMIAS NEAR U.K. NUCLEAR FACILITIES

In this section we discuss the epidemiological reports studying the incidence of leukemia near nuclear power plants and other nuclear facilities in the United Kingdom. The most detailed report is by Forman *et al.* (1987). They discuss many different cancers. They conclude that "there has been no general increase in cancer mortality near nuclear installations in England and Wales during the period 1959-80. Leukemia in young people may be an exception, though the reason remains unclear." If the leukemias were due to radiation, why were other radiation-induced cancers not seen?

This study shows that the Standard Mortality Ratios (SMR's)* for Local Authority areas near nuclear installations are significantly less than the SMR's for control areas more often than the reverse. Only for acute lymphoid leukemia, which occurs primarily in the age group up to 20 years, does there seem to be the increase. It is hard to explain these cases by either direct radiation, or radionuclide releases. This has been studied in a detailed report by Strather, Clarke and Duncan (1988). "These cases could not be explained by radiation alone, unless the release was 300 times that known" (Forman *et al.* 1987). Another possibility is that the carcinogenic effect at low doses of radiation is much higher than thought. But then, why do not the radionuclides from bomb test fallout, many of which are similar, produce a similarly large effect?

The effect seems to be primarily a reduction in the number of leukemias in the control areas compared with the number expected from national incidence figures. This strongly suggests be a chance effect. We also note that the increase was not around nuclear power plants, but around experimental sites: Sellafield fuel processing plant, Dounreay fast breeder reactor and the Royal Ordinance Factory.

One other feature of interest comes out of this work. Usually epidemiologists, such as Forman *et al.* (1987) study cancer mortality. This is because mortality is an objective criterion, and recently has not been subject to reporting bias. Beral (1987) pointed out that there is an increase in cancer incidence near nuclear installations although no increase in mortality. This may be due to a tendency to report cases more frequently near nuclear installations but it may also be due to emigration of diagnosed cancer patients from areas with nuclear installations. The measured effect is small enough (10%) that biases are all important.

The paper of Forman *et al.* (1987) was misquoted in the U.S. press. The Boston Globe (Tye 1987) had a misleading headline, "More cancers near nuclear plants," and combined this with a discussion of Gould's work to give a confusing picture. The title was misleading, and nowhere in the text was the main conclusion quoted. Unfortunately, accurate reporting of these matters in the press is rare.

The statistically significant increase of childhood leukemias has aroused a lot of attention. Clusters of childhood leukemia were originally reported near the experimental breeder reactor in Dounreay, North Scotland. Five leukemias in the age group 0-24 were observed, whereas 1.6 ± 1.3 were expected (Heaseman *et al.* 1986). This is enough to generate a hypothesis that there is something about Dounreay that leads to childhood leukemias. Five leukemias were also found near the British Nuclear Fuel Services Chemical plant at Sellafield (Taylor and Wilkin 1988; Darby and Doll 1987) (including one who had moved out of the area and found later) with 0.5 expected (when the calculation gives a fractional expectation or a number less than one for the expectation, means that both 0 and 1 are likely). Observation near the second plant seems to confirm the hypothesis that there is something common to Dounreay and Sellafield that leads to childhood leukemia.

*SMR--"the number of deaths, either total or cause-specific, in a given occupational group expressed as a percentage of the number of deaths that would have been expected in that occupational group if the age-and-sex-specific rates in the general population had obtained." (MacMahon 1980, p. 125).

A slight excess in Berkshire, Barrington and West Hampshire where there are three nuclear establishments, Atomic Energy Research Establishment (AERE), Harwell, Atomic Weapons Research Establishment (AWRE), Aldermaston, and Royal Ordnance Factory (ROF) Burgfield (Roman *et al.* 1987) made the hypothesis even more likely. However, an examination of Roman and others' Table 8 shows that increased leukemias are only significant within 10 km of ROF Burgfield (38 cases ages 0-14 vs. 23.9 expected, 8 vs. 6.4 expected within 10 km of AWRE, and 0 vs. 0.4 expected within 10 km of AERE. Inclusive reviews of these and other cancers have been made by Cook-Mozaffari *et al.* (1987), Forman *et al.* (1987) and Strather *et al.* (1988).

Many scientists have searched for a possible cause of these childhood leukemias. Darby and Doll (1987) found higher leukemia incidence near several nuclear power plant sites, even when no nuclear power plant had yet been built! This suggests that there must be another explanation unrelated to nuclear power or radiation itself.

Since Sellafield and Dounreay are new communities, the young new population might have brought in viral diseases not common in the region from the outside. But this argument could not apply to Aldermaston which is a settled community. This hypothesis was tested by (Kinlen 1988) in another "new" community in Scotland, Glenrothes, where there were no nuclear facilities. A cluster of childhood leukemias were found--10 in the age group 0-24 (between 1951-67) versus the 3.6 expected. It is important to realize that this is a bigger cluster than either the one at Dounreay or the one at Sellafield. It also important to realize that this is not enough to <u>prove</u> a viral cause.

Still a third possibility was studied by Gardner et. al. (1990a, 1990b) who identified 52 cases of childhood leukemia and 22 cases of non-Hodgkin's lymphoma which had been diagnosed between 1950 and 1985 in the county of West Cumbria, and compared them with 1001 controls. They investigated four possible causes:

(1) prenatal X-rays (which are known to cause leukemia),

(2) infectious disease (which might have predisposed the victims to a leukemia infection),

(3) eating shellfish (which concentrate radionuclides), and,

(4) paternal occupation

The most complete information was available from birth certificates which were available for 46 cases of childhood leukemia and 16 cases of non-Hodgkin's lymphoma. For leukemia alone, Gardner et al. found nine cases whose fathers worked at Sellafield. The risk ratio was 2.62 when area controls were used (95% confidence 1.07 to 7.40) which is just statistically significant. If local controls were used, the risk ratio is reduced to 2.03 (95% confidence 0.69 to 5.93) which is not significant because risk ratio less than 1.0 cannot be excluded. When non-Hodgkin's lymphoma is added, the risk ratio drops to 2.02 even with area controls (95% confidence 0.87 to 4.67) which is insignificant.

One interesting fact that was not highlighted in press accounts, is that there were 9 leukemias and non-Hodgkin's lymphomas among children whose fathers worked in the iron and steel industry. Using local controls, this gives a risk

ratio of 3.20 (1.23 to 8.28 at 95% confidence), which is more significant than the relationship to Sellafield. Also elevated, but not significantly so, was the risk ratio for those whose fathers were farmers.

Since the results of the study by Gardner et.al., are just statistically significant by only one of the measures, overall the study cannot be considered significant by Tippett's rule. Moreover, Gardner *et al.* do not tell us whether their nine cases overlap with the five cases found in previous studies; presumably they do and the associations are not independent. Clearly, if a family moved to Sellafield because it is a new town, it is likely that the father worked at the nuclear facilities; the child could nonetheless be subject to specific viral infection as Kinlen suggests; although Gardner *et al.* looked for nonspecific infections. If the idea that parental exposure caused the childhood leukemias is correct it is also the correct at Dounreay. However, of the five children with leukemia only one has a parent working at the plant. Clearly this work raises more fascinating questions than it provides answers.

Because of concerns raised by the reports about finding some increase in mortality from leukemia among young persons, especially under 10, living nuclear facilities in the UK, a comprehensive survey of cancer rates was conducted by the US National Cancer Institute in population living near nuclear facilities in the USA (Jablon *et al.* 1990). The survey evaluated over 900,000 cancer deaths occurring between 1950 through 1984 in 107 counties with nuclear installations. This covered all 62 nuclear facilities that went into service prior to 1982, including commercial electricity-generating power plants and major DOE facilities. Each study county was matched for comparison to three similar control counties in the same region. Cancer deaths studied in the control counties over the same period amounted to more than 1,800,000 cases.

The study found no evidence to suggest higher occurrence of leukemia or any other form of cancer in the study counties than in the control counties after the start of the nuclear facilities, as can be clearly seen from Table 7. The study did reveal that some of the study counties had slightly higher ratio of cancers, and some had lower ratios. This pattern was also observed either before startup of some facilities or after startup of other facilities, and, therefore, no evidence for a cause-effect relationship between nuclear facilities and cancer occurrence in a nearby population could be established. Clearly, because the

TABLE 7. Ratio of Cancer Deaths in Counties Near Nuclear Plants and Cancer Deaths in Control Counties

	Before Startup	After Startup
Childhood leukemia	1.08	1.03
Leukemia at all ages	1.02	0.98

Jablon et al. (1990)

study was limited by the correlational approach and the large size of counties, it could not prove the absence of any effect; but such effect, if it exists, must be small or it would be detected by such a study.

Gofman raises what we call four "postulates" (Gofman 1986a).

I "All forms of cancer, in all probability, can be increased by ionizing radiation, and a correct way to describe the phenomenon is either in terms of the dose required to double the spontaneous mortality rate for each cancer, or alternatively, in terms of the (percent) increase in mortality rate of such cancers per rad of exposure."

II. "All forms of cancer show closely similar doubling doses and closely similar percentage increases in cancer mortality rate per rad (at a given age)."

III. "Youthful subjects require less radiation to increase the (cancer) mortality rate by a specified fraction than do adults."

IV. "The peak percent increase in cancer rate per rad is reached grossly earlier for such high Linear-Energy-Transfer radiations as alpha-particle irradiation in contrast to the time to reach peak percents for low LET radiation."

The postulates are reasonable and plausible, and some are suggested by the data. However, when reading Gofman's papers it is important to continually remember that these are only assumptions; as one read further the postulates become "laws"--a somewhat more grandiose description than is justified. Moreover, it is important to realize that these postulates by themselves do not lead to the high numbers in Gofman's calculations. It is therefore possible that all the four postulates are correct and yet Gofman's numbers are completely wrong! The opposite is also possible in principle: the postulates may be wrong, but the bottom line, a large number of radiation induced cancers may be correct.

XV THE RISK ACCORDING TO THE "ESTABLISHMENT"

Two methods have been used to extrapolate radiation induce excess cancer to ages where no data exists: the absolute risk method and the relative risk method. The absolute risk method assumes that the increased number of cancers is unrelated to the background rate, while the relative risk method assumes that the same multiplier applies to the background risk at all ages. Risk estimates of the "establishment" have been primarily based on the Life Span Study (LSS) of the Japanese atomic bomb survivors at Hiroshima and Nagasaki although other studies, mainly medical exposure ones, have also been investigated and used for incidence and mortality risks for specific sites in each case after a latent period. The effects of each assumption are illustrated in Fig. 13. To date, the "Japanese data" represent the largest cohort size with the largest follow up period and has been subjected to most thorough and careful investigation; only this survivor cohort contains persons of all ages at exposure.

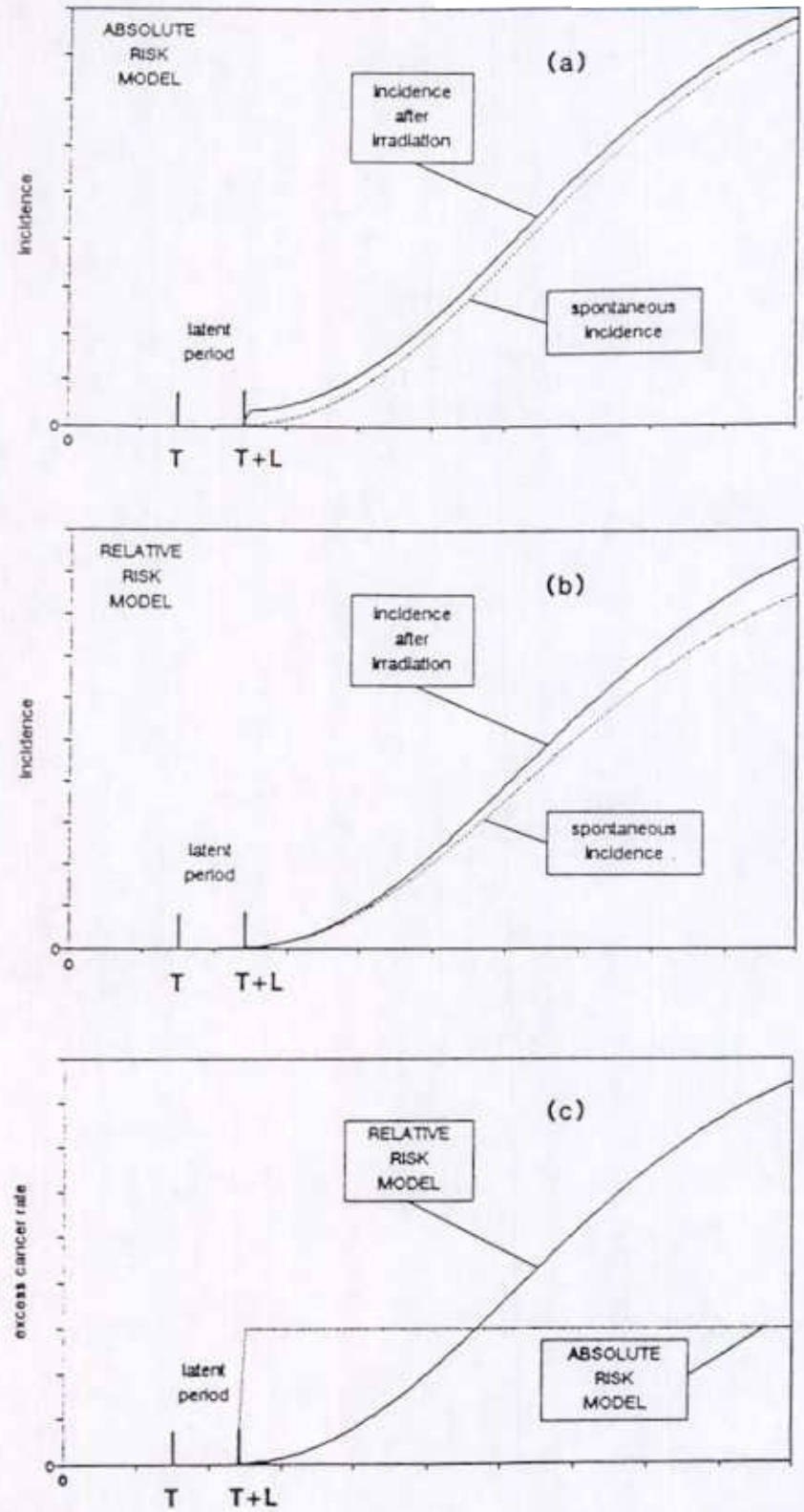

Fig. 13. Lifetime Expression, Comparison of Absolute and Relative Risk Models
Source: Gofman (1981b)
(From BEIR 1980)

In extracting estimates from high-dose epidemiological studies, one is confronted with two difficult tasks. First, one must assume a certain dose-response function to allow extrapolation of the risk to low dose. The data at high and intermediate dose do not provide definitive information about the shape of the dose-response curve. For leukemia, a linear-quadratic relationship fitted the animal data, and was adopted by the 1980 report of the National Academy of Sciences (BEIR 1980). For other cancers, the data are not definitive. As more data become available with a longer follow-up period, most recent expert studies preferred a more conservative linear relationship to describe the dose-response curve. It is prudent, and has long been advocated by the International Commission for Radiological Protection (ICRP), to assume a linear dose-response relationship for preventative public health purposes.

Furthermore, since no reliable epidemiological study (for all age groups at exposure) is available with the full follow-up period yet, one must project the lifetime risk. In the early seventies, only short follow-up periods were available for the Japanese cohort, and both the absolute and relative lifetime projection methods were consistent with the data. As time evolved and data for longer follow up periods became available, it appears that the relative risk method is more consistent with the data for cancers other than leukemia, though one must still await until the follow-up period is essentially complete (another 20 years), before making a definitive conclusion. In any case, since the relative risk method gives more conservative estimates, it is the preferred method for radiation protection, and has been adopted by most national and international regulatory bodies.

In Table 8 we present a summary of the estimates made over the last 20 years by various national and international bodies and the basis of these estimates. The table also includes Gofman's estimates (1981b), which remain unchanged in Gofman (1990). For acute doses at high dose rates the most recent risk factors recommended by UNSCEAR (1988) and BEIR(1990) range from 700 to 1100 cancer deaths per million person-rem. For low dose and slow dose rates these reports suggest that based on animal and other evidence a reduction factor of between 2-10 should be used. In the remainder of this report we adopt a "standard", and we believe conservative, risk factor of 500 cancer deaths per million person rem for estimating risk from low dose and slow dose rate exposure. One notes from the table an upward trend with time (by a factor of 2-3) in the recommended risk factors. This is mainly due to the recent reassessment of the A-bomb dosimetry at Hiroshima and Nagasaki which resulted in lowering the average dose equivalent estimated for each city (see Preston & Pierce 1987, 1988). Some previous studies (e.g., BEIR 1980) contained an implicit dose rate reduction factor of nearly 2.5. Both UNSCEAR (1988) and BEIR (1990) reports provide excellent comprehensive and up to date reviews of the subject, including all recent findings and the subsequent revisions of recommended risk factors.

We wish to emphasize that all the estimates in Table 8 were meant to quantify a possible but not proven, small harmful effect from exposure to low-level radiation. These estimates were derived primarily for preventative public health protection, and therefore must be, and are, significantly conservative without being unduly excessive or unreasonably high. One must also remember that it is possible that the risk from low level radiation below a certain threshold level will turn out to be nil. There are even some suggestions of benefits that may accrue from very small doses of radiation. This is called radiation hormesis in the literature (Wolf *et al.* 1988).

TABLE 8. Excess Cancers per Million Person-rem Assuming Low Dose Linearity

ICRP (1977)		100
BEIR 1972	absolute risk	117
	relative risk	620
Gofman 1981b	low	2636
	standard	3771
	high	5988
BEIR 1980	relative risk: linear quadratic	226
	absolute risk: linear quadratic	77
BEIR 1980	relative risk: linear	501
	absolute risk: linear	167
BEIR 1990	relative risk: linear linear (no linear quadratic number presented)	1100
UNSCEAR 1988		
(High and intermediate dose and dose rate)	relative risk:	700-1100
	absolute risk:	400-500
(Low dose and dose rate)	relative risk:	(550-70)
	absolute risk:	(250-40)
BEIR V 1990		
High dose and dose rate	relative risk:	800
(UNSCEAR 1988 and BEIR 1990 recommend that at low doses and low dose rates a reduction factor of 2-10 should be applied)		
"Standard" model used in this report (Low dose and dose rate)		500

XVI. COMPARISON OF GOFMAN'S AND BEIR ESTIMATES

Gofman's estimates in 1970 were considered very high. And although the "establishment" estimates have been revised upwards somewhat, there is still a large discrepancy (factor of 3 to 5) between Gofman's estimates and those of the latest BEIR and UNSCEAR reports. In the early seventies, when Gofman's claim of excessively high risk first came out, the follow-up period in the Japanese cohort was only 25 years or so.

It is useful now to review here the discrepancy between Gofman's estimates and those of the "establishment", especially since they rely essentially on the same data. For more detail, we refer primarily to his books (Gofman 1981b, 1990). We compare first with BEIR (1980) since Gofman himself compares to BEIR (1980), and since the difference from BEIR (1990) is mainly due to the new dose scale. We find it convenient also to go back to the data of the Radiation Effects Research Foundation (Preston and Pierce, 1987). Indeed we note that if these data were complete, then the risk of exposure to ionizing radiation of an average member of the population would be directly given by the data. However, the data are not complete; those who were under 35 at the time of Hiroshima are mostly still alive, and may yet develop additional radiation-induced cancers.

We will compare only the estimates assuming a linear dose-response relationship for carcinogenesis. Using what at first sight appear to be the same set of assumptions, we note that this set of assumptions in both cases is in complete accord with the generalization, postulates or laws listed in the preceding section. Yet, BEIR (1980) project 501 excess cancer deaths per million person·rem in the exposed population whereas Gofman (Gofman 1981a, Fig. 5, p. 272 and Table 18, p. 274) projects 2636 excess cancer deaths per million person rem in the same population, using the Japanese A-bomb survivors data only (Gofman's lower limit). The difference is due mainly to the different method adopted by Gofman to project the limited follow-up data into full lifetime, and the excessively high values assigned by Gofman to the relative risk per rad for the young groups at exposure. We show in Fig. 14 the risk ratios used in the various models for different ages at exposure.

It is widely believed, and therefore assumed, that the sensitivity to radiation varies with age, and is greater for the young. A particular problem arises for the small group that was less than 10 years old at exposure in the Japanese data. The relative risk derived for this group was large, but statistically unstable. In BEIR (1972) the large figure was taken; BEIR 1980,

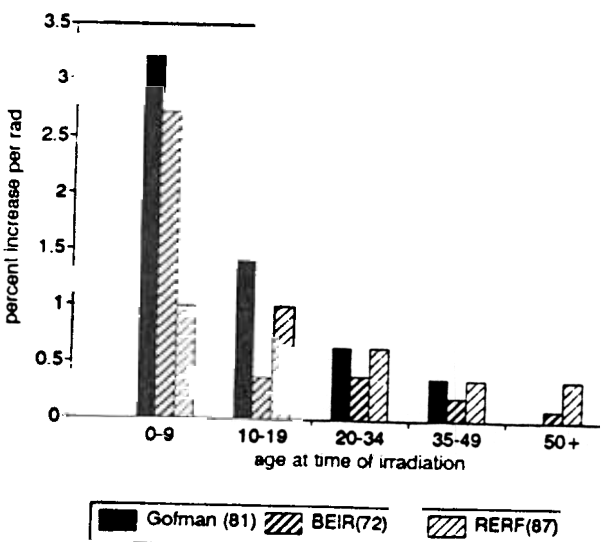

Fig. 14. The Constant Lifetime Excess Relative Risk Plotted as Present Increase Over the Natural Cancer vs Age at Irradiation

however, states that "For ages under 10 years at exposure, the relative-risk ratio thus appeared unreliable (referring to the limited follow-up period of 11-30 years after irradiation), and the ratios for ages 10-19 at exposure were substituted for them." This was a drastic departure which reduced the calculated risk below that calculated in the BEIR 1972 report. Gofman, on the other hand, chose to extract extremely high values for the relative risk of the young age group at exposure, relying on the statistically unstable data of the LSS at the time.

The additional follow-up period up to 1985 for the Japanese data has resulted in a relative-risk for the age group under 10 years at time of exposure that is more stably estimated. Using the new data increases the BEIR (1980) risk estimate by about 30% (Preston and Pierce, 1987, 1988, BEIR 1990). But Gofman's very high estimates of the risk factors for the young at exposure are clearly not borne out.

Furthermore, relying on the observation that excess incidence for radiation-induced cancer of the lungs and female breast cancer, within the first 30 years after exposure in the LSS data, which appeared to follow the same temporal patterns as the natural age-specific incidence or mortality. BEIR (1980) made a judgement to use a constant relative risk ratio throughout lifetime. They were careful to note that "this may not apply to all radiation-induced cancers, or it may apply only to individual cancers and not to groups of cancers." Indeed, we know that the relative risk for leukemia does fall after 20 years and even the absolute risk model can overstate the risk for leukemia.

Gofman, on the other hand, made a very different assumption from the "establishment" about the way relative risk varies with time after exposure (see Fig. 15). Instead of a constant relative risk after a latent period, he assumed that the relative risk, which is the ratio of excess cancer to normal cancer, is zero during a latency period of about 10 years. After that it increases shortly to reach a peak, which he calls "maximum peak percent increase, x," 40 years after irradiation. The ratio then decreases symmetrically, in a bell-shaped curve, reaching zero around the year 70 following irradiation. He also claims,

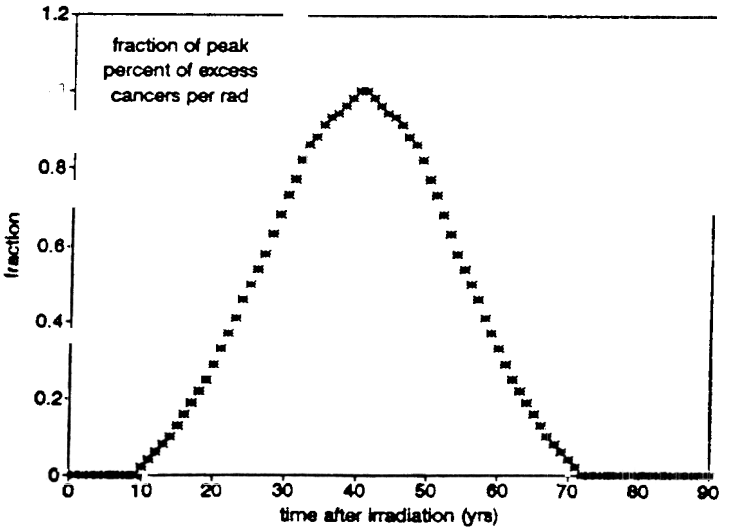

Fig. 15. [redacted]

the shape and magnitude of this relative curve is valid for all cancers and all ages. At the time of this assumption data existed only for a small portion of the rising part of the curve and such a projection was consistent. To support his assumption, Gofman incorporated data from studies other than the atomic bomb survivors--but these studies were less reliable. In doing this Gofman while in his book pointing out the problems of deriving data from small numbers, falls into the trap himself. Nowhere in his book is there a proof of the validity of this bell-shaped relative risk curve; nor, to be fair, has there been a clear discussion of why the more conventional curve is superior. One fact seems certain; the curve does not apply to radiation induced leukemia, because leukemia is well-known to display a short latent period (BEIR 1972, 1980). Finally Gofman, while in his book pointing out the problems of deriving data from small numbers, falls into the trap himself.

In fact, continued follow-up of the A-bomb survivors in the Japanese data (Preston *et al.* 1987, 1988, BEIR 1990, UNSCEAR 1988) shows that the risk of radiogenic cancer relative to spontaneous incidence remains comparatively constant for all age groups at exposure, and is therefore compatible with risk estimates based on use of a constant "relative risk ratio for projection". Recent data do not support Gofman's proposed "bell-shape" model for the expression of lifetime radiogenic cancer.

However, as pointed out by BEIR (1990), the availability of longer follow-up time data from the LSS study and the Ankylosis Spondylitis study suggest, at least for some solid cancer that a variable relative risk model is more consistent with the data. In this model, the relative risk increases slightly for about 20 years following a latency period, then it declines. This possible model is far different than Gofman's.

We can conclude that Gofman's excessively high estimates for the lifetime risk from low level ionizing radiation can be attributed to two key flaws:

(i) his choice of a different model for the time behavior of the Risk Ratio (RR) (which we believe to be incorrect); and

(ii) the use of excessively high initial Risk Ratio, especially for the young age groups, extracted incorrectly from the data.

Figure 16 illustrates these points clearly. In it we show the annual excess cancer deaths for a male cohort of 100,000 at birth, which were exposed to a dose of 1 rad at age 13. The curve A shown by asteriks is taken directly from Gofman's 1986b Table 20 (page 281), based on 1.4% relative risk extracted by Gofman from the available Japanese data in 1974, combined with his bell-shaped lifetime relative risk model. The solid curve B is based on a constant lifetime relative risk of 1.4%. The total lifetime cancer death for this cohort from curve A is 3.4 larger than for curve B.

In addition it must be noted that the risk estimates of Gofman (1981) were based upon a choice of a Radiobiological Equivalent (RBE) of 1.0 for the neutron component of the dose. (The dose was given by the T65DR50 dosimetry system.) His justification, (Gofman 1981b, p. 246) for this choice at that time was based upon the work of McGregor *et al.* (1977) who stated "that there is no significant difference in the breast cancer incidence between the Hiroshima and Nagasaki survivors," and "that there is no evidence to suggest that an RBE value for neutrons other than 1.0 is needed."

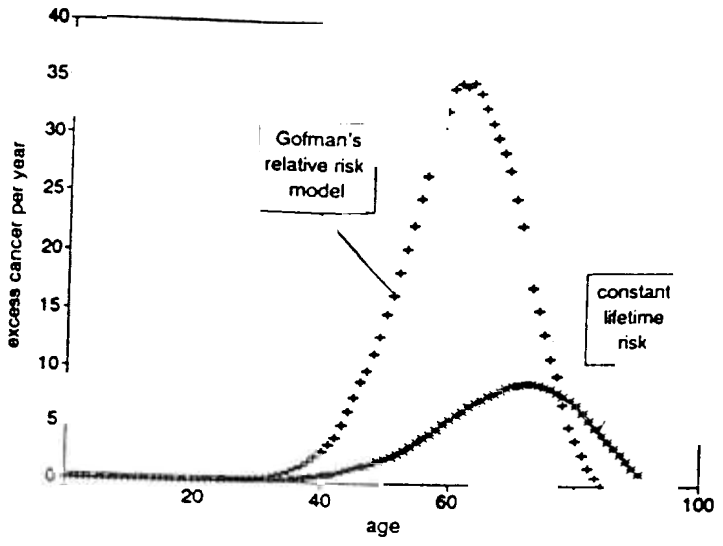

Fig 16 Annua Excess Cancer per 1 rad Exposure of 97,710 Males at Age 13

In his most recent book (Gofman 1990) Gofman used an RBE factor of 20 for the neutron component of the revised DS86 dosimetry system for the A-bomb survivors in the Japanese data. In the same book, he also used an RBE value 2.0 when referring to the older T65DR50 dosimetry. His stated reason was to equate the neutron dose equivalent component in the two dosimetry systems. Had Gofman used a high (20) RBE value for neutrons, as is usually accepted, in conjunction with the T65DR50 dosimetry in his analysis, his 1980 risk factors would have been lower by a factor of 2 to 3, and would have only been a factor of 2-3 higher than those of BEIR (1980).

XVII. DOES GOFMAN'S MODEL AGREE WITH RECENT DATA?

We can see the comparison of Gofman's model more directly by returning to the data themselves from the Japanese (LSS DS86) subcohort of 75,991 people followed up to 1985 and described by Preston and Pierce (1988) and ask whether these two life-time projection models, Gofman's bell-shaped risk ratio and the BEIR constant relative risk, supposedly developed from detailed analysis of the same data, properly represent the totals. In Table B1, page 462, of Preston and Pierce (1988) the observed cancer rate for the period 1950-85, is tabulated. This is summarized in Table 9 classified by age at exposure, sex, time since exposure and intestinal dose. They also give the expected cancer mortality for the same period. We must add to these totals any cancers yet to develop. Then dividing by the number of persons and the dose, should give the final numbers as shown in the table.

We assume that the mean absorbed dose for this subcohort is the same as for the full DS65 cohort, which is given by Gofman (1981b) in his Table-9, (Chapter 6, page 169), ranging from a low of 23 rad to a high of 31 rad for the exposed

TABLE 9. Cancers Appearing in the Japanese Cohort up to 1985

Age at Exposure	Observed	Expected	Difference
<20	539	489.7	50 ± 22*
20-34	1002	929.7	72.3 ± 30*
35+	4193	4052.7	140.3 ± 64*
Totals	5734	5472..	262 ± 73*

This difference is to be compared to the prediction for the difference at end of life (relative lifetime risk model only):

Gofman model	Japanese curve - low	2800
	Standard - central	4010
	High	6365
BEIR 80	linear-quadratic central	240
	linear-linear	533
UNSCEAR 88	high dose (upper limit)	1170
	high dose (lower limit)	745
Total cancers expected at end of ife		15000

*These stochastic uncertainties are the square root of the expected number ($\sqrt{N}$). This quantity ($\sqrt{N}$) is one standard devitation (σ). A deviation greater than two times the standard deviation will occur with probability (P) < 0.05.

group. When we include the unexposed group for the DS65 cohort we obtain a mean absorbed dose for this cohort of about 14 rad per person. The mean absorbed dose for the DS86 subcohort can be extracted directly from Table 3 and appendix Table 2 of Shimizu et al. (1987). This yields a total of approximately 1 million person-rem for the DS86 subcohort. This DS86 subcohort is made up of a mixture of all age groups and although this mixture is not identical with a representative group, it is not far from it. Accordingly, using Gofman's lower value for lifetime risk factors, we expect an additional excess cancer mortality in the DS86 subcohort of approximately 2800.

Now the lifetime cancer mortality rate is known to vary between countries so that we must be careful how we use the Japanese rate. According to Preston and Pierce (1988), the natural cancer mortality rate has been about 19.3%. Therefore, over the lifetime of this subcohort a total of approximately 15,000 normal background cancer deaths is expected. Over the first 40 years of follow-up, the total observed cancer mortality was 5734, while the expected is 5472. The excess is thus 262 cancer deaths over that period. Therefore, if Gofman's risk values are correct, more than 2500 excess cancer deaths should materialize over the remaining lifetime of this subcohort in addition to another approximately 10,000 normal background cancer deaths. Put differently, only 9% of the 2800 total

additional cancer excess predicted by Gofman for this subcohort over its lifetime have materialized over the first 40 years after exposure. This represents also only 5% of the normal cancer deaths that have materialized so far. To make Gofman's predictions come true, this ratio must jump dramatically in the remaining period of the lifetime of the subcohort to an average value of 30%! A most unlikely proposition indeed.

In fact, according to Gofman (1981b) (see Tables 23 and 24, page 295), about 10% of the total excess cancer deaths will occur in the age group 35+ years at exposure. Therefore in DS86 subcohort this represents about 280 out of the 2800 total excess cancer deaths. Most of these deaths should have been observed by 1985, as the youngest of this age group was 75 years old by then. This is twice what is observed for this age group and more than the total excess cancer deaths for all age groups observed in this subcohort by the year 1985! If we took Gofman's "standard" or "high" estimates, for the lifetime risk from low-level radiation, the outcome of the comparison will be much worse.

On the other hand, using BEIR (1980) risk values we get the alternate predictions shown in Table 9. It should be noted first that the downward revision of the LSS dosimetry by a factor of about 2 would not change these predictions as this would be balanced by an upward revision by a similar factor in the risk factors. When compared with the 262 observed excess cancer deaths in this subcohort over the period 1950-1985, both BEIR predictions appear to be consistent with observations so far though the linear-quadratic model would appear to somewhat underestimate the lifetime excess cancer in this subcohort. It is to be remembered, however, that the linear-quadratic model was derived for use in low dose exposure and is therefore not applicable for this situation. Nevertheless, the data over time till 1985 agrees much more with the most recent of BEIR (1990) and UNSCEAR (1988) than with those of Gofman. We can think of this as a continuation of an experiment to verify the models that were derived to explain the data obtained in the first part of the experiment, which comprised the cancers diagnosed between 1950 and 1974.

XVIII THE CHELYABINSK COHORT

This section is different from others in this report. First, it describes a recently unclassified, database (which we optimistically call a cohort), that has a potential of becoming a major source of data on occupational radiation risks. Second, the doses were extremely high. Therefore, there is evidence of increased radiation-related cancer mortality that meets all of Hill's criteria and the numbers are well beyond statistical uncertainty. These data provide clear-cut evidence of radiation effects on workers. We present here our own rough estimates of lifetime cancer risk (Shlyakhter and Wilson, 1991). The rough results apparently are considerably lower than the claims of Gofman and tend to support the "establishment" position. They also address the suggestion of Stewart and Kneale that the Japanese data gives too small a value.

In February 1990 some data on the radiation doses and cancer rates to the workers in the first Soviet atomic bomb facility near Chelyabinsk were released and published in the Soviet popular science magazine Priroda (Nikipelov *et al.* 1990). As of May 1991, this is the only piece of unclassified information about

occupational doses at Chelyabinsk and the effects on health for the 40 years since irradiation.

The Chelyabinsk installation is actually located near the town of Kyshtym, 40 miles north of Chelyabinsk, in South Urals. It was the site of the first large-scale plutonium-production reactor (Facility A) and radiochemical plutonium separation plant (Facility B). Here plutonium was produced for the first Soviet nuclear explosion in August 1949.

Nikipelov *et al.* present the radiation doses for each year, averaged over the workforce, and the cancer rates for high and low dose groups. Doses were extremely high, especially during the first five years of operation 1948-1953 (see Fig. 17). Facility B appeared especially complex both in technology and in radiation conditions. It appears that in their haste to build a bomb, the Soviets did not wait for remote control devices. Nor were they short of volunteers to work at hazard pay in a patriotic duty. That is why, in 1950-1951 almost half the employees got more than 100 rem in one year (see Fig. 18). According to Nikipelov *et al.*, Table 10 demonstrates the undisputable increase in cancer mortality.

The mortality level in the groups with lower doses does not differ appreciably from the cancer mortality level of the adult population of the U.S.S.R. (about 200 cases per 100,000 persons/year or approximately 6% for 30 years). The authors conclude that the "clear increase of mortality in the groups with large radiation doses can be considered to be the result of radiation e xposure."

Unfortunately Nikipelov *et al.* did not tell us some important numbers. In particular, we do not know the absolute number of workers exposed to radiation and their age. Pending ultimate release of full data, we have deduced an estimate of the total number of workers n, and hence the workforce, from the errors σ assigned by Nikipelov *et al.* to the cancer rates π. We assume that they used

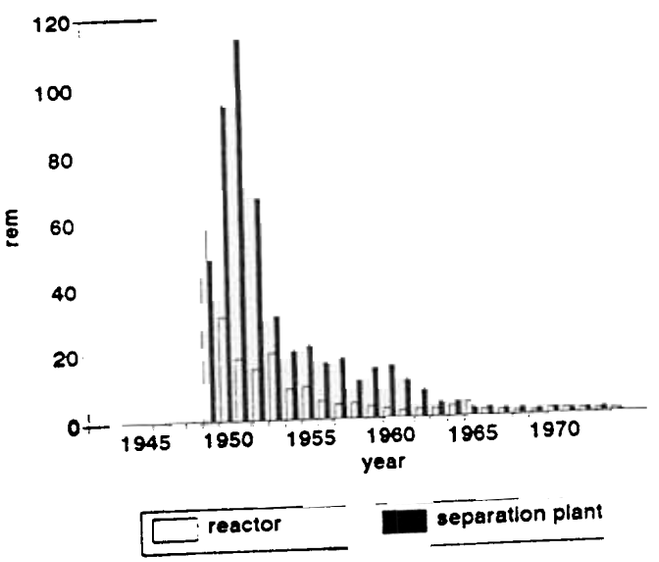

Fig. 17. Occupational Irradiation of Soviet Nuclear Workers at Chelyabinsk (yearly averaged doses from Nikipelov et al. 1990)

Fig. 18

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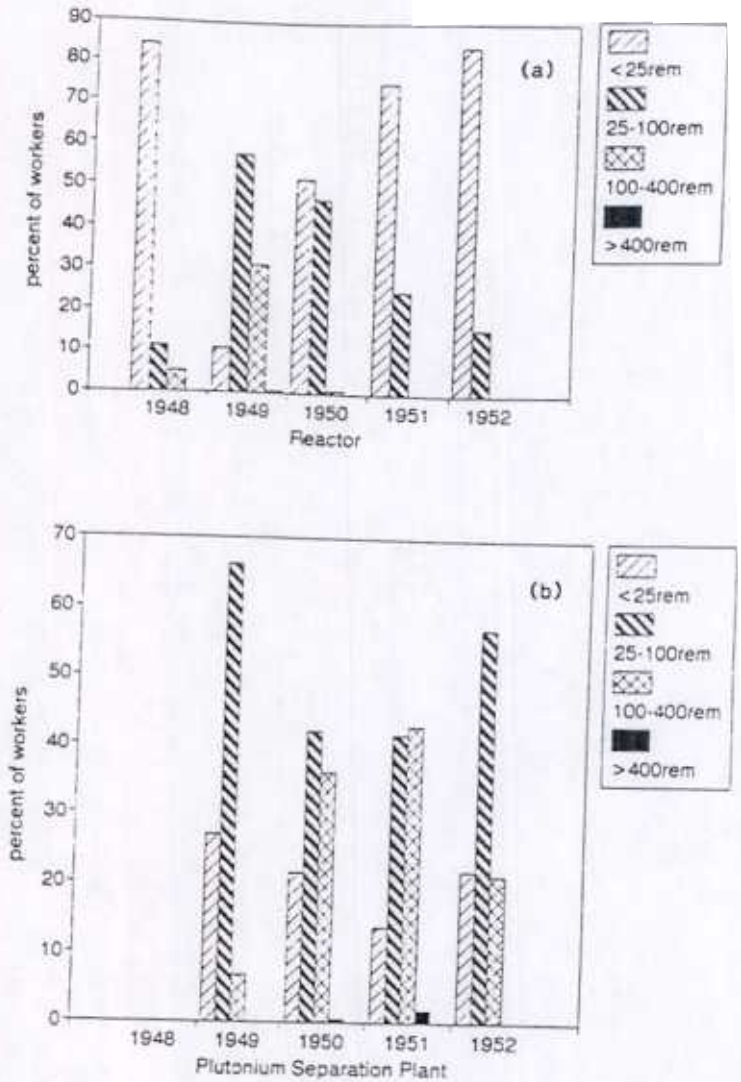

Fig. 18. Dose Distribution at Chelyabinsk in 1948-1952 (Nikipelov et al. 1990) (a) Reactor workers; (b) Separation plant workers.

the standard statistical formula $n = \pi \cdot (1-\pi)/\sigma^2$. For example, an entry in their Table 3 which we reproduce here as Table 10, gives a cancer mortality of 5.7% ± 0.6% for those workers at the reactor, employed before 1958, who received less than 100 rem. From π=0.057 and σ=0.006 we estimate that the workforce at the reactor must have been at least 1500 persons. Calculating such estimates for every entry we find that the numbers agree within 10% accuracy; combining several estimates we determine that the number of people employed before 1958 at the reactor was 2000 persons, and 4600 at the plutonium separation plant. This is the total number of people. The number of people employed at any one time is a little less because some workers were replaced.

The sum of the annual doses is high--over 300 rems. The evaluated collective dose is simply the estimated workforce multiplied by the sum of the yearly average radiation doses to which the workforce is exposed. If no workers were ever replaced, and our estimate of workers exposed was the total number exposed at one time then

TABLE 10. Chelyabinsk Cancer Mortality (% Incidence in Workers Employed Prior to 1958)

Facility	Total Gamma Dose, rem		Maximum Year Dose, rem	
	<100	>100	<25	>25
A	5.7 ± 0.6	9.4 ± 1.2	5.9 ± 0.7	8.7 ± .1
B	4.3 ± 0.4	8.1 ± 0.6	4.2 ± 0.5	7.7 ± 0.5
Total	4.8 ± 0.4	8.4 ± 0.5	4.9 ± 0.4	7.9 ± 0.5

Mortality due to neoplasms in blood and lymphatic tissues is included.

(from Nikipelov et al. 1990)

$$collective\ dose = (workforce) \times \sum_{1948}^{1976} coverage\ annual\ dose$$

The evaluated collective dose would be less if the irradiated workers were promptly replaced because the yearly averaged doses refer only to those actually exposed. If we allow for an average work period of five years out of the 10 years, we should halve these numbers to get the workforce at any one time. This work period of five years may be short, because about 70% of the dose was accumulated by 1953, while the first regulations requiring transfer of the overirradiated workers to "clean" jobs were enforced only in 1954. If we assume 50% exposure reduction factor, collective doses are: 260,000 person·rem for the reactor, and 1.2 million person·rem for the processing plant.

To estimate the number of cancers that were caused by these doses, we take from Table 10 the 3.6% difference of the cancer rates for two dose groups: greater and less than 100 rem, multiplied by the number of people that, we estimate, received over 100 rem, which is 3100. In order to estimate the number of excess cancer deaths during lifetime, we must know the workers' age at the time of irradiation (ATR). We estimate it by comparing cancer mortality in the lower dose group during the 40 years of observation with the lifetime cancer mortality in the USSR.

XCD(life)=XCD(40 years)*XCD(ATR to ATR+40)/XCD(ATR to 100)

If we assume the age of 25 years, the background cancer rate will be 15%, in agreement with the Soviet cancer mortality, which is lower than in the US because of lower life expectancy. We did not have Soviet data on cancer mortality in different age groups, so we use the U.S. data (U.S. Statistical Abstract, 1989) and obtain the factor that projects the observed XCD to the whole lifetime:

XCD(25-65)/XCD(25-100) = 3.1. This factor is very sensitive to the assumed age; our choice is supported by the data: if we assume, workers were 35 years old rather than 25 years, they would be 75 years old by now and the lifetime cancer mortality for the lower dose group would be 7.5% instead of 15%.

Multiplying these factors (3.6% difference in cancer rates from Table 10; 3100 workers total and 3.1 lifetime correction factor gives a total number of 310 with a standard deviation error of ±52. This can be broken down into 67 ± 24 lifetime cases from reactor exposures, and 242 ± 46 from the reprocessing facility. This should still be increased by adding the smaller number of cancers among the equal number of persons who accumulated less than 100 rem. Assuming that these received an average of 30 rem, and that linearity applies, probably another 1/4 or 80 cases should be attributed to the lower exposures for a total of 400. Dividing by the collective dose, we find 300 "excess cancer fatalities" per million person·rem. Our estimates suffer from many uncertainties; still, it seems that most of them could only make the estimates of radiation risk lower. For example, if it appears that workforce was replaced at a slower rate. Workers were older than we assumed, neutron irradiation and internal doses from radionuclides prove to be important, the risk would be decreased.

Therefore the extra cancer deaths will be far easier to distinguish from background. The estimated risk of excess cancer fatalities is smaller than the risk for A-bomb survivors with similar doses (about 800 XCD per million person·rem in BEIR 1990 report). However, it is found from animal data and generally accepted that spreading the doses over time can reduce the risk by a factor between 2 and 10. A-bomb doses were almost instantaneous, whereas the doses at Chelyabinsk, were spread out over several years. This comparison of the effects from Chelyabinsk and the A bombs, confirms that there is some reduction factor.

Our preliminary analysis of the new Soviet data suggest that the "establishment" might be too conservative in estimating the occupational radiation risk. If low dose linearity is assumed, our estimate falls three times below BEIR (1990) (without a dose-rate reduction factor) and 10 times below Gofman (1986b).

With this new data we also get a severe constraint on claims about large effects of occupational exposures. The Chelyabinsk installation was a counterpart of Hanford Atomic Energy Laboratory in Richland, Washington where Mancuso *et al.* claimed to find effects of radiation. But doses at Chelyabinsk were about hundred times higher than at Hanford. Even at Chelyabinsk, the increase in cancer mortality is *only* about five times the statistical error. Were the doses just five times less, no effect could be proved!

Stewart and Kneale (1990) have suggested that data derived from the Hiroshima-Nagasaki cohort (Japanese data) understates the risk, because the cohort is one of healthy survivors; those who were especially sensitive to acute effects would not be present. This argument would not apply to the Chelyabinsk cohort, at least, for occupational radiation risk analysis. For nonoccupational applications we must discuss the "healthy-worker" effect, but this is unlikely to be a factor of more than 1.5.

Although all the desired information about the cohort may not be available, the size of the dose makes it a most important cohort.

XIX. CANCER FROM NATURAL BACKGROUND RADIATION

Other data can also address this question. In Wilson and Jones (1974), modified here as Table 11, is a list of activities giving various radiation doses. Attached to that list is the number of cancers that would be found if all the U.S. were exposed, on the assumption that the slope of the dose-response is 500 cancers per million person rem (2000 person·rem/cancer). Gofman uses 3771 cancers per million person rem for a population of mixed ages. (We note here that although 3771 seems an accurate number because four figures are quoted, most scientists would "round off" the number to 3800 to stress that the accuracy is not that high).

Can we find situations where the method of Gofman "obviously" overpredicts the cancers? Since Gofman is suggesting a large risk, it should be easier to find such situations than if Gofman was suggesting small risks. In order to find such a situation it is necessary to study a large enough population, and to find places where there is enough increase from background exposure to give an appreciable effect (perhaps 50% increase). In view of the very large risk ratio that Gofman predicts for young people, a population of young people would be especially interesting, although one again has the problem that one has to wait a longer time until death.

The size and variation of the natural background suggests that there should be changes in cancer incidence associated with changes in the natural background. This has been looked at by Frigerio and Stowe (1980) who compared the vital statistics by state with the natural background. They found that the cancer rate was lower ($132/10^5$) in the states with the highest background (170 mrem/yr) compared with that ($147/10^5$) in the states with the lowest background (118 mrem/yr) and $155/10^5$ in states with 130 mrem/y. The data are shown in Fig. 19 taken from Goldman (1989). The fitted line is a decrease with increasing radiation dose. The statistical accuracy of such a comparison is excellent. Naively one would say that radiation at these low doses reduces the cancer probability. But there was no discussion, by Frigerio and Stowe, of possible alternative explanations; absence of major industry or confounding effects of major lifestyle contributors to cancer such as cigarette smoking. A real decrease is probably not likely. We suggest here, that a comparison of lung cancer incidence can tell us whether smoking plays a major role, and a look at bladder cancer might tell us the role of industrial emissions. Cohen (1980) notes that a refusal to accept the data as indicative that radiation is good for you depends upon preconceptions, whether correct or not. He comments "The fact that states with high natural radiation have considerably lower cancer rates than average is generally discussed as indicating only that radiation is very far from being the principal cause of cancer, and this point is logically correct. However, this author (Cohen) is highly skeptical over whether that attitude would be accepted if states with high natural radiation happened to have somewhat higher than average cancer rates."

We also note that there is no indication of the steep increase with radiation dose suggested by the dotted curve of Fig. 6b.

Other studies of natural background, with a smaller statistical sample, exist. For example in an area of Guandong Province, Peoples Republic of China, there exists a region with three times the normal level of exposure to radium and thorium products; yet the lung cancer incidence is actually less in these areas

TABLE 11. Some Typical Radiation Doses

Source	Dose (mrem/yr)	Radiation Cancers/yr if all U.S. Pop. (250 million) So Exposed (Assuming 500 Cancers Source per Million Person-rem)
Potassium 40 naturally occurring in body	20	2,500
Potassium 40 naturally occurring in neighboring body	2	250
Gamma rays from neighboring soil and rocks (average)	50	6,250
Gamma rays inside brick or stone buildings	30-500	2,200-37,000
Cosmic rays at sea level	30	3,700
Background dose at sea level (average)	100	12,500
Background dose at sea level in Kerala, India (average)	500-2,000	37,000-150,000
Cosmic rays at Denver, CO	67	8,300
Three-hour jet plane flight	2	250
60 hour/month of jet plane flight	500	62,500
Medical diagnostic X-rays in U.K. (average)	14	1,750
Medical diagnostic X-rays in U.S. (average)		
1964	55	6,875
1970	95	11,875
Weapons tests "fall-out"	3	375
AEC "design criteria" for reactor boundary (upper limits for actual use)	5	625
Within 20-mile boundary of BWR with 1-day hold-up but leaky fuel (gaseous emission) (average)	0.1	12.5
Within 20-mile boundary of PWR with leaky fuel (average)	0.002	0.25
Within 20-mile boundary of coal plant (average)	0.1	12.5

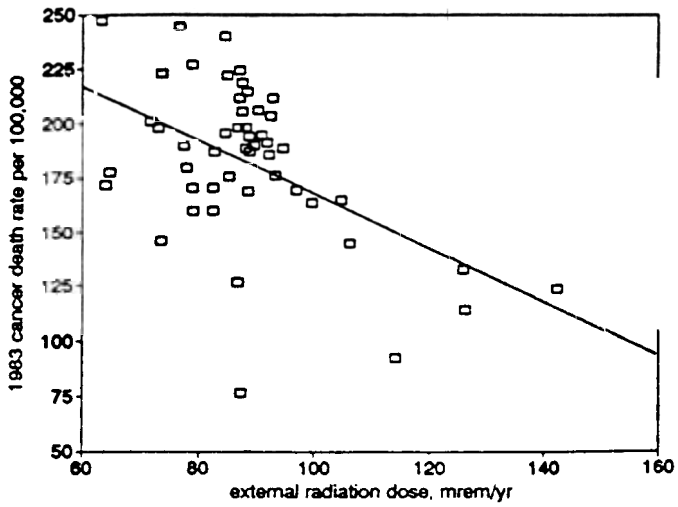

Fig. 19. Cancer Mortality vs Natural Radiation by State (Goldman 1989)

than in nearby areas with normal exposure (Hoffman, Katz and Chungxian 1985)! In this instance the increased dose is primarily to the lung and would be expected to cause an increase in lung cancer incidence. Instead, a small decrease was found.

XX. CANCER AMONG AIRLINE PERSONNEL

The radiation dose from an airplane flight comes from cosmic radiation. The cosmic ray dose increases with altitude. A part of this dose is from neutrons, leading to about 0.3 mrad/hr (UNSCEAR 1977) at 11 km (approximately 35,000 ft). This suggests that there is a significant exposure of the over 100,000 airline crews, including pilots, stewards and stewardesses. Air crews typically fly 70 hrs per month at 35,000 ft, for a total dose of 29 mrad per month, or about 350 mrad per year based on data for a typical New York-London flight from Table 33 (UNSCEAR 1977 annex E). For a pilot working from age 25 to age 65 this gives 14 rad in a lifetime, or among all air crews, 1.4 million rad. The dose in rem may be a little higher because of the cosmic ray neutrons which give a larger effect for a given dose than minimum ionizing particles. We neglect this here. According to the standard model with about 500 cancer cases per million person rem, there will be 700 "extra" cancer deaths in this group in addition to the 20,000 (±140) expected by natural incidence. Although it would be a four standard deviation effect and formally statistically significant, a 3% effect would be somewhat small to believe. However, if Gofman's figures are correct, we would expect, using Tables 21 and 22 of Gofman's book (Gofman, 1981b) to exclude children, over 2200 extra cancers, or an increase of 11%, mostly due to exposures before age 40. This number should be easily detectable. If such an epidemiological study is undertaken, these rough calculations would have to be refined; and the doses well-estimated. This should be comparatively easy, because airlines keep records of hours of flight and altitude of flight from which one can calculate the cosmic ray dose.

XXI. CANCER FROM DIAGNOSTIC X-RAYS

We note also the average dose for medical diagnostic X-rays in the U.S. of 95 mrem in 1970. This leads to about 10,000 radiation cancers per year with 500 cancers per million person rem. This would be 70,000 or so if Gofman's assumed slope were correct. Seventy thousand seems large, and certainly large enough to be identified if true. Strangely enough, although the whole of Chapter 19 of Gofman (1981b) is devoted to medical X-rays, this conclusion of a huge effect is not mentioned. We also note, that the radiation doses from diagnostic X-rays are given over a short time, and dose rate effects are less likely than in other low dose exposures.

XXII. CANCER AMONG MEDICAL TECHNICIANS

The occupational X-ray doses to medical technicians are also appreciable and are also calculable from the UNSCEAR report (see Table 12). The average dose of U.S. medical technicians is about half that of airline personnel, and the number is comparable to the number of airline personnel, and therefore one would also expect, according to the establishment view, about 350 extra cancers in addition to the 20,000 expected according to the average in the population, and 1100 extra according to Gofman. In the "bad old days" of 1920-1940, radiologists and physicians were much more careless than they are in 1988, and lifetime X-ray doses of 20,000 rem were recorded (Warren, 1956). Warren found that the life expectancy of a diagnostic radiologist was five years less than that of other physicians, BUT NOT ALL DIED OF CANCER, whereas upon the Gofman model, cancer death would be early and almost certain. It would seem worth while to reexamine these data.

XXIII. CANCER IN NUCLEAR INDUSTRY (BRITISH NUCLEAR FUEL SERVICES)

The doses in the nuclear industry, including the nuclear power and the defense nuclear industry, are approximately known. In an earlier section we discussed claims that radiation exposure caused an unusual number of cancers at Hanford. For nuclear power plants 500 mrem/yr is typical, although in exceptional cases, higher doses are found. For example, the workers in Chernobyl units 1 and 2 for the first year of operation after the accident received 1.4 rem (Umanetz, 1987). There are about 100,000 exposed nuclear workers, so once again a study should show conclusively whether Gofman can be right or not. We have a preliminary idea from a study of the workers at British Nuclear Fuels Services (BNFS) (Smith and Douglas, 1986). Of 10,083 radiation workers at BNFS, the average lifetime dose was 12.4 rem. The expected number of cancers from the standard model is 30 to 40. So far, there have been 396 cancers, with 419 expected by chance. This is a difference of -23±20. The reduction could be a "healthy worker effect." The 419 expected is 1/5 of the 2000 that are eventually

TABLE 12. Occupational Exposures from Diagnostic X-ray Examinations (mrem)

	Average Dose Equivalent			
	1974-76	1978-79	1980-81	1984
Medical				
Radiologists				
Canada		40		25
Japan		30		
Norway			270	
Switzerland		60		
United Kingdom			51	
United States			170	
Technologists				
Canada		20		12
Japan		50		
United Kingdom			37	
United States			50	
Nurses				
Canada		40		15
Japan		20		
United Kingdom			35	
Aides, porters				
Canada		40		8
United Kingdom				
Physicists				
Canada				36
Norway			74	
United Kingdom			14	
All medical workers				
Japan		50		
Poland	50-100			
United Kingdom			14	
Dental (all workers)				
Australia		10		
Canada	4	5		2-5
France	50	40		
Switzerland		20		
United Kingdom				10
United States			20	

From UNSCEAR (1988)

expected to manifest themselves by other causes, so of the 30-40 possible radiation cancers, only 8 to 10 should have manifested themselves so far. Under Gofman's model, however, 80 should have been seen already, and they were not.

We add a cautionary word here that was noted earlier; if the other radiation doses to which workers are exposed are as large as the occupational doses, and if there are correlations in these doses with these occupational doses, spurious effects can be seen. In the UK the medical X-rays among this group were probably about 40 mrem/yr (see Table 12) leading to a background over a 50 year period of 2 rem. This probably fluctuates by an amount at least as large as itself. Any correlation between medical x-rays and occupational dose is likely to be of the order of the fluctuation. This, 2 rem, is smaller than the average occupational dose of 12 rem, so the study is not invalidated. But a study in the USA might be. The average diagnostic X-ray dose in the USA is larger than that in the UK (95 mrem/yr, Table 11) and its fluctuation perhaps also about 100 mrem/yr. Then it would be impossible to find reliable epidemiological effects of doses below 4 rem without detailed correction for medical x-rays. The fluctuation in the background of radon gas exposures is greater still. However, radon gas is expected to affect only the lung, so that a valid study could still be undertaken if lung cancers are excluded.

Also not mentioned by Gofman, is that his model would predict that about 140,000 annual cancer deaths, 1/3 of all annual cancer deaths in the USA, would be caused by background radiation. Added to the 35% generally believed to be caused by cigarettes, this would lead to a knowledge of the cause of 70% of all cancers. Knowing the cause is the first step towards the reduction or even elimination of the cancers. This would be good news indeed. Alas, it is unlikely to be true.

XXIV. CANCER FROM CHERNOBYL

The accident at the unit IV of Lenin Atomic Energy station near Chernobyl, Ukraine, gives an opportunity for further study. There are three particularly interesting groups of exposed people. The first (group I) are those who were at the plant during the accident, and helped to put out the fire. They developed acute radiation sickness, but most of them recovered. However, they received large doses. We here estimate these doses to be an average 250 rem each. If the doses were much higher, more firemen would have died; if much less, they would not have had radiation sickness. There are conflicting reports, but there are probably about 250 persons in this group.

The second group (group II) is those persons who lived within 15 km from Chernobyl, but not including the town of Pripyat. For various reasons, late evacuation, being in a direct line of the initial plume, or living in wooden houses, they received higher doses than the others in the region. Dr. Pavlowski estimated that these 24,200 people accumulated 1,080,000 person rem, or an average of 44.6 rem each (Table 7.2.3 of Legasov, 1986).

A third group, (group III) consists of those evacuated from Pripyat (45,000) and those who lived between 15 km and 30 km from Chernobyl, (original estimate

65,700, revised downwards to 41,000) to make an adjusted total of 86,000 in group III and a total of 110,000 evacuees. Group III received an average dose of 4 rem.

In Table 13 we show the number of cancers expected during the present lifetimes of those exposed according to the estimates above. In this, we assume that the population has an average age distribution, so that the average figures are applicable.

We also show the rate expected on the basis of US statistics; that 20% of all persons will die of cancer, and assuming that these numbers will also apply to the Ukraine. The expected uncertainty in this number from statistics alone is just the square root of this number. We note that any claimed excess must be compared with this uncertainty in a test for statistical significance.

By comparing the number of expected cancers from any one of the models with the statistical uncertainty of the expected number, we see that each group is as sensitive as the other; group I has more exposure, but group II has more people. An increase over the lifetime of the persons concerned will just be discernable

TABLE 13. Cancers Expected near Chernobyl upon Various Assumptions

	Group 1	Group II	Group II
Number of Persons	250	24,200	86,000
Natural Number of Cancers	50 ± 7	4,800 ± 69	17,200 ± 131
Collective Dose Commitment	62,500	1,080,000	344,000
Low dose slope cancers/10^6 person·rem			
100 (ICRP 1977)	63	108	34
117 (BEIR 1972)	7.3	126	40
2,636 (Gofman 1981)	129*	2,847	907
3,771 (Gofman 1981)	153*	4,073	1,297
5,998 (Gofman 1981)	194*	6,467	2,063
501 (BEIR 1980)	31	541	172
226 (BEIR 1980)**	14	244	78
1,100 (UNSCEAR 88)	69	1,180	378
800 (BEIR 1990)	50	864	275

if the "standard" model is correct, easily discernable if Gofman is correct, and not discernable if the more optimistic models are correct. To be discernable, the excess should be two to three times the standard deviation.

We take for leukemia the central estimate in an NRC report (Evans, Cooper and Moeller 1985) without and with adjustment for dose rate reduction. We simplify by ignoring the difference between "kerma" dose and tissue dose. For group II we expect 52 leukemias "without" and 26 "with". For group III we expect 21 "with" and 7 "without". The "expected" number in the absence of the Chernobyl accident is 0.7% in a lifetime; this must be adjusted downwards a little to 1/2% to allow for the fact that the evacuees do not have a full lifetime. This gives an expected level in the absence of Chernobyl of about 121 ± 11 cases in group II, and 430 ± 21 in group III. A similar emphasis on leukemia, without the numbers noted here, has been made by Goldman (1987).

The significance can be further enhanced by noting that most (and we here assume 75%) of the cases will appear between 4 and 19 years after the accident (1990 to 2005) The expected number in the absence of Chernobyl becomes about 24 for group I. The first few cases should already be beginning to appear. Therefore we can expect a reliable preliminary answer to these questions in 15 years or so.

There are many other possibilities. All depend upon the fact that the radiation exposures can be estimated much more accurately than the effects on health can be estimated. We emphasize here, however, the importance of asking the relevant questions before the study is undertaken and demonstrating that even in the presence of a fluctuating background of medical X-rays and radon gas exposures, an answer can, in principle, be found.

XXV. CHERNOBYL'S CANCER CONSEQUENCES--GOFMAN'S ESTIMATES

Very soon after the accident, Gofman made public some estimates of the collective dose commitments resulting from the accident which seemed excessively high. For example, he estimated that there would be 9000 leukemia deaths in Finland alone as a consequence of the fallout from Chernobyl (Metzenbaum 1986). This would imply at least 10 million person-rem in Finland alone even on Gofman's high dose-response relationship.

TABLE 14. Leukemias Expected from Around Chernobyl

	Group	Group II	Group III
"Expected" Total	1.2 ± 1	121 ± 11	430 ± 21
"Expected" 1990-2005	1 ± 1	24 ± 5	86 ± 9
No Dose Rate Adjustment			
Total from Chernobyl	2.7	52	21
1990-2005 Chernobyl	2	39	16
With Dose Rate Adjustment			
Total from Chernobyl	n/r	26	7
1990-2005 Chernoby	n/r	20	5

Gofman shortly afterwards published revised estimates of the exposure of people, mainly Europeans, to radioactivity from Chernobyl (Gofman 1986a), using various early reports. These may be compared to the more careful study in a report to DOE (Goldman *et al.* 1987a, 1987b) here presented as Table 15. Note that Gofman has reduced his early estimates for Finland and the USSR. The figures for USSR given in the table are from Ilyin and Pavlowski (1988). These external doses are revised downwards by a factor 1.75 from Pavlowski (1986) to account for an underestimate of the decay of the radiation due to absorption of Cs by the soil (Wilson 1987). The internal, ingestion, doses are reduced over a factor of 10 to account for cleanup and a better estimate of the takeup of radioactivity by plants and subsequent ingestion thereof.

Gofman's revised estimates of doses is only slightly higher than the best estimates available now. Goldman *et al.* did a global study, accounting for all the iodine and caesium emitted (and even assuming that more was emitted than the Soviets reported) so that on a global basis it is unlikely that they underestimated. Many Europeans believe that Goldman overestimated. For example, Frey (1987) suggests only 300,000 person-rem for the U.K. compared with Goldman's 1,500,000. In fact, the latest data coming out of the USSR indicate that the total collective dose commitment for the central part of the European USSR should be reduced further from 57 to 31 million man-rem (Ilyin *et al.* 1990). Goldman took the dose from ingestion equal to the external dose. The usual figure is about 30% and the preventive measures taken in the Ukraine and Byelorussia probably reduced this to 18%.

According to Gofman estimates, the additional cancer fatalities in many European countries will be significantly larger than the standard deviation of the normal cancer fatalities in those population, and thus should, in principle, be detectable over the next few decades. For example, Gofman estimates an additional 212,000 cancer fatalities over the lifetime of the population of the central part of the European USSR, a region with a population of about 85 million. This region accounts for most of the collective dose commitment from

TABLE 15. Lifetime Dose Commitment from the Chernobyl Accident As Calculated by Gofman and by Goldman et al.

Country/Region	Population	Dose Commitment Gofman	Person-rem Goldman
Albania	2,500,000	30,000	600,000
Austria	7,600,000	1,322,400	1,400,000
Belgium	10,000,000	20,000	90,000
Canada	22,125,000	8,850	10,000
Czechoslovakia	15,500,000	806,000	1,000,000
Denmark	5,100,000	76,500	80,000
Finland	4,800,000	1,195,200	400,000
France	54,540,000	3,163,320	1,200,000
Germany, Fed.	61,400,000	10,560,800	6,000,000
Germany D. Rep.	17,100,000	3,437,100	1,300,000
Greece	9,700,000	29,100	400,000
Hungary	10,600,000	434,600	1,300,000
Ireland	3,100,000	4,030	180,000
Italy	56,200,000	1,629,800	6,000,000
Japan	119,500,000	95,600	120,000
Luxembourg	350,000	4,200	8,000
Netherlands	14,400,000	172,800	400,000
Norway	4,130,000	355,180	170,000
Poland	36,900,000	9,557,100	15,000,000
Romania	22,900,000	17,633,000	9,000,000
Spain	38,200,000	99,320	0
Sweden	8,300,000	4,116,800	900,000
Switzerland	6,500,000	1,534,000	400,000
Turkey	48,000,000	4,800,000	1,700,000
United Kingdom	56,000,000	3,640,000	1,500,000
United States	235,000,000	11,750	110,000
USSR	85,440,000	56,900,000	35,000,000*
Yugoslavia	23,000,000	4,255,000	8,000,000
TOTAL	978,885,000	127,400,000	92,268,000

*European Part, Ilyin and Pavlowski 1988.

Chernobyl accident in the USSR. The normal cancer fatalities expected for this population is approximately 12.75 millions with a standard deviation of about 3600 (based on the present rate of 15% for cancer deaths in the USSR). If Gofman cancer risk factors are correct and indeed there is no need for further reduction factors due to low dose and low dose rate, and the collective dose commitment estimates remain significantly unchanged, then within few decades a 2% increase in the normal cancer fatality rate should materialize, assuming of course that all other factors affecting the public health in this population remain unchanged.

If we use the standard cancer risk factor of 500 cancer deaths per million person-rem, combined with the latest estimate of 31 million person-rem for the collective dose commitment (Ilyin *et al.* 1990), we calculate 15,500 additional cancer death for the same population. While this number is still more than four times the standard deviation of the normal cancer deaths, it represents only 0.0012 of the total normal cancer deaths expected for the population based on present rates. Given the fact that total cancer deaths in industrial countries have risen by about 25% over the past few decades, it is doubtful, if not impossible, that even the large additional cancer deaths estimated by Gofman as a result of the Chernobyl accident can be ascertained among the population at large of the central part of the European USSR.

XXVI. EFFECTS OF CHERNOBYL

It is very likely that the media in the coming years will often carry reports or claims of excess cancer observations, which will be attributed to Chernobyl, especially for the population near the accident site. Already some claims of excess cancer have been published in some avant-garde Soviet publications and were quoted in the Western press. They have, however, been formally denied by the Scientific Center for Radiation Medicine of the USSR (Pyatek 1989). Such reported claims must be examined with extreme care to verify whether they are the result of careful scientific studies or simply "claims" that lack scientific basis and scrutiny. For in the new Soviet environment of Glasnost and free speech, some baseless claims will undoubtedly flourish (Kapitza 1989).

For example, V. Kolin'ko (1989), describes the grave effects of radiation in Narodichi, Zhitomir district of Byelorussia. In particular, Kolin'ko claims that 140 calves and piglets with visible anomalies were born at the Petrovsky collective farm. This claim was investigated by a team of experts from the Institute of Agricultural Radiology. First, it appeared that the number of abnormal calves was much less than claimed: only 8 calves instead of 62. Second, this farm had the *lower* radioactive contamination level than two nearby farms where no abnormal animals were born. Third, evidence was found that concentration of nitrates in the food, that animals were fed with, exceeded the allowed limit by 20 times, while some necessary nutrients were deficient. The experts concluded that the anomalies observed could be attributed to malnutrition and, possibly, to existing breeding practices when closely related animals are mated (Maslov 1989).

Even when there are obvious effects, they are not necessarily directly caused by radiation (Aleinikova 1990), a Minsk pediatric hematologist warns about the increase in childhood leukemia in Minsk and the surrounding district. This is surprising, because Minsk district was not noticeably contaminated. We plot the data in Fig. 20 which shows the observed rates of leukemia together with statistical uncertainties. The rate before 1986 was 2/3 of the Western European rate, suggesting significant underreporting. It was already on the rise when Chernobyl accident happened in 1986. This trend could explain at least some of the observed increase; the other possibility is that some children from more contaminated areas were probably brought to Minsk hospitals after the accident. We also note that a similar plot for the Gomel oblast (district) of Byelorussia shows no increase in 1989.

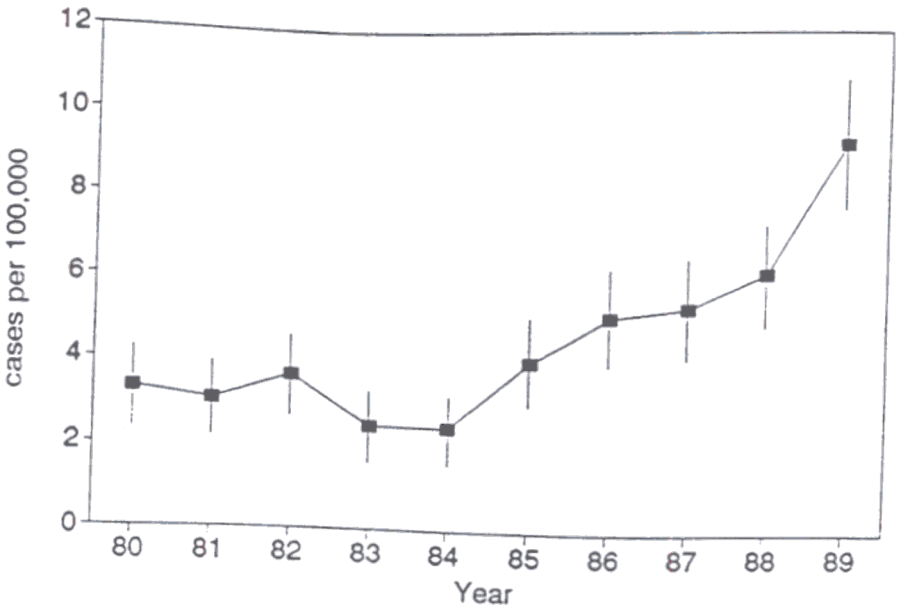

Fig. 20. Childhood Leukemia in Minsk (Aleinikova 1990)

XXVII. COMPARISON OF COHORTS

In examining claims of effects of low levels of radiation, it is useful to get a preliminary perspective by considering how many persons were at risk; how big was the average dose; and how big was the product--the collective dose. For excess cancers to be clearly visible, the average dose must be high; for statistical accuracy to be adequate the collective dose must be high. In Table 16 we show these for the various cohorts discussed in this report.

We plot in Fig. 21 the needed cohort size to find a significant result for a 1 rem dose per/person, assuming various cancer slopes. We note the large cancer slope needed to get a significant effect for a 1 rem/person dose in a reasonable size cohort.

We conclude this section by noting that in his most recent book (Gofman, 1990), Gofman now asserts that substantial agreement exists between his estimates and those of UNSCEAR 1988 and BEIR 1990 with respect to cancer-risk per rad from moderate doses acutely delivered; this in spite of the fact that his estimates are still a factor of 3-5 higher than the "establishment."

He further asserts that his independent analysis, based exclusively on human evidence, "proves beyond reasonable doubt that no safe dose or dose rate exists with respect to radiogenic cancer;" he also claims that, again based exclusively on human evidence, his higher risk factors are applicable with certainty to acute low and slow low doses, and any use of a reduction factor is proven wrong."

Unfortunately, all what Gofman's detailed analysis demonstrate is, as is well-known, that no direct human-based exists (or is likely to become available

TABLE 16. Comparison of Cohorts

Cohort	Number of Persons	Average Dose Rems	Collective Dose Persons Rems	Excess Cancers Observed
Chelyabinsk	6,600	~200	1,460,000	310 ± 52
Japanese (Table 9)	75,991	14*	1,064,000	262 ± 73
Chernobyl (Table 13)	24,000	43	1,080,000	?
European Region of the USSR	75,000,000	0.415	31,100,000	?
British Nuclear Fuel Service	10,083	12.4	125,000	-23 ± 20
Hanford Workers	23,704	3.32	76,700	?
SW Utah (Washington County)	48,500	1.9**	92,000	7 ± 4***

*30 for exposed group of 35,000.
**Average for leukemia victims.
***Leukemia.

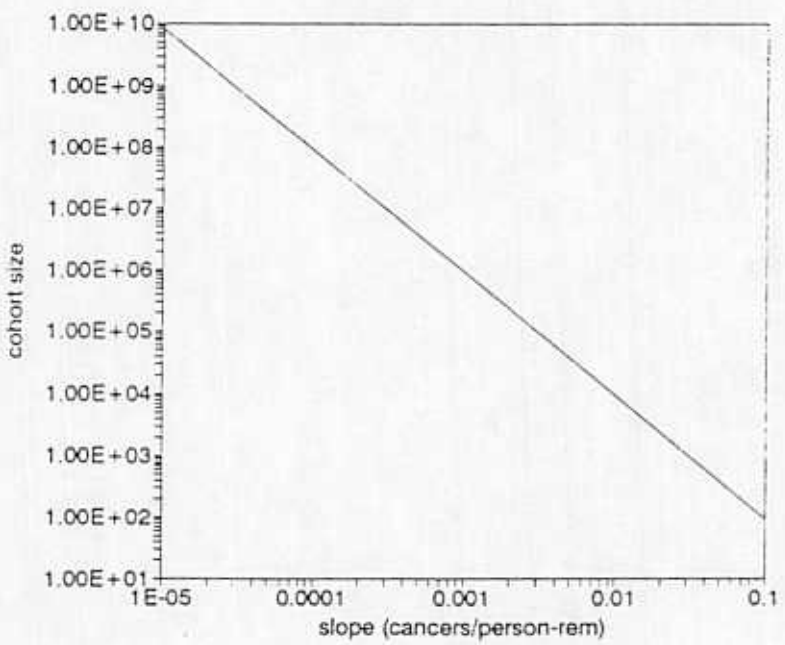

Fig. 21. Cohort Size vs Slope. A plot (log-log) of the minimum necessary cohort size for an epidemiological study to show the excess cancer with statistical significance at the 2-sigma level above the natural cancer vs the slope of the dose response for 1 rem average dose per person.

in the near future) that will conclusively prove/disprove the threshold or no-threshold dose response theories. The validity or refutation of self-repair mechanism leading to reduction of risk factors for slow dose rates might be proven by a more careful look at the Chelyabinsk cohort, but it cannot be disproved at present or the near future, based on human evidence alone. Belief in a threshold or in its absence, must depend upon indirect evidence. No conclusive proof is not a conclusive disproof. Ruling out all other evidence, animal-based and other indirect methods, as Gofman insists, will just make it much harder to settle these important issues in the foreseeable future.

XXVIII. EXPRESSING THE RISK

The way the risk is expressed can, and usually will, influence the way that it is perceived. The issue in this paper is how to express the risk in such a way that the public realizes, as much as possible, the nuances that are well-understood by the expert. Newspapers often express the results in the most sensational manner possible. For example, Dr. Ilyin's careful statement in the official report (Legasov 1986) that the increase of cancer rate will be less than 0.06% (based on pessimistic dose estimates and a linear dose-response curve but a somewhat smaller number of cancers per person-rem than that used here) was translated in a Washington Post headline to mean that 45,000 persons <u>will</u> get cancer. The verb <u>will</u> is clearly wrong. Replacing it by <u>may</u> makes the statement accurate but still, by itself, confusing. Dr. Ilyin held a press conference in Vienna to explain the distinction.

For an individual, the possible increase of 0.06% in cancer probability is probably most important. Only for someone making decisions for the whole country is the total number relevant.

However, we argue, as did Crouch and Wilson (1987), that to be fully understood, the risk must be looked at in all its facets. One of the most important, however, is to compare it to risks we regularly accept such as these from background radiation.

XXIX. RADIATION REVIEWS

In addition to the official "establishment" reports (BEIR, 1972; BEIR, 1980, UNSCEAR 1988) there are a number of other review papers and books by distinguished people. Some of them address the issues here and we list them for convenience (Yalow 1986; Webster 1980; Bond 1970, 1981; Hamilton 1983; Cohen 1980, 1981, 1986; Pochin 1983; Archer 1980; Goldman 1989).

There are also a number of books and papers which are written in a less restrained manner by various persons (McCracken 1982; Grant 1988). These are useful as sources of information, but are in general, too partisan to present a proper case.

XXX. DISCUSSION AND CONCLUSION

Any discussion and review of the effects of radiation on health is necessarily incomplete. It has been estimated that there are over 100,000 references on the subject. In making this review, we have only begun to address many of the claims and have only read a fraction of the papers. However, we hope, and believe that we show how to address the main issues.

XXXI. ACKNOWLEDGMENTS

This project was stimulated by the late Dr. William Preston, who kept asking one of us how to respond to claims of radiation damage that seemed superficially plausible. The project began with a short report for, and funded by, DOE. It was extended and distributed with funds from U.S. Council for Energy Awareness (USCEA) and gifts from Stone and Webster Corporation. This updated and corrected version was made possible by a contract from Argonne National laboratory, and a gift from General Electric Company. To all of these, we are grateful for their help. We gratefully acknowledge helpful comments and suggestions from many people and in particular Dr. Ralph Lapp, Dr. Arthur Upton, Mr. John Landis, Dr. Robert Budnitz, Dr. Victor Bond, Dr. Stan G. Prussin, Dr. R. Yalow and Dr. E. E. Pochin. We have benefitted greatly from many conversations upon the general subject with many of the authors to whose work we refer, and in particular Dr. Ilyin and Dr. Pavlowski. In conclusion the detailed help of Ms. S. Wey and Mr. R. McDonald in assembling the final report and graphs is much appreciated.

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