

Public Health Hazards from Electricity-Producing Plants

The hazards from both conventional and nuclear plants should be compared in an epidemiological study.

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The current energy crisis makes it necessary to build new electricity-producing plants. These may be coal- or gas-burning plants or nuclear generators. Whatever type is chosen for a given locality, the normal operation of the plant will contribute to local pollution in its own way. The effects on public health will depend upon the combination of the pre-existing pollution and the contribution of the new plant. In this connection a reliable methodology is needed to estimate how many more cancer cases and how many more other important ailments should be expected as a result of the normal functioning of any electric generator proposed for the given locality. This problem is different from that, now widely discussed, of a variety of disasters feared in connection with proliferating nuclear facilities.

The problem of estimating deleterious health effects of additional pollution of one kind or another may be solved through a large, interdisciplinary, multi-pollutant, and multilocality epidemiological study (1). One of the prerequisites for success is statistical competence sufficient to avoid the many threatening statistical pitfalls (2). The current methods of approaching the same societal problem are different. Particularly with reference to irradiation hazards, they consist in efforts to establish the so-called safe levels of exposure to irradiation. The determination of such levels is based on extrapolations of two kinds: from results of experiments on lower animals and from ex post facto studies of certain cases of human experience, exemplified by studies of atomic bomb survivors at Hiroshima and Nagasaki.

The purpose of the present article is to

bring out evidence that the above two extrapolations cannot be considered as sources of reliable information. Hopefully, this will serve as convincing argument in favor of the recommended multi-pollutant and multilocality epidemiological study.

Phenomenon of Competing Risks

One of the greatest difficulties in comparing health hazards from pollutants of different kinds is the phenomenon of competing risks. While the term may appear puzzling, the phenomenon itself is omnipresent and familiar. Street traffic and cancer are two of the many factors which compete for our lives. If I were run over by a car and killed tonight, I could not die later from cancer, and subsequently this would be reflected in public health statistics. Similar situations occur in technology (3) where they have generated a special field of investigations labeled reliability theory.

Both situations involve predictions of mortality rates to be expected after specified changes in the environment. In the problem of electric generators the mortality in question is that of humans, and the changes in the environment are the predictable changes in local pollution. In technology the "mortality rates" may be those of airplane engines with changes in "environment" represented by some contemplated changes in the engines' important parts. In both cases a satisfactory solution of the problem depends upon the mechanism of competition of the various factors being understood. As illustrated below, such mechanisms may be of great complexity and our understanding them requires both delicate experimentation and the special development of statistical theory.

Table 1, reproduced from (4), refers to an important experiment performed by Hoel and Walburg at Oak Ridge (5). It illustrates the competition for the lives of irradiated mice of the following factors (or causes): (i) germs as they occur in ordinary laboratory conditions, (ii) thymic lymphoma (a cancer), (iii) reticulum cell sarcoma (another kind of cancer), and (iv) all other causes.

In the presence of germs, sarcoma and other causes appear as very strong competitors of lymphoma. But, when the germs are removed, the victims of lymphoma are almost double those of sarcoma. The statistical problem consists in disentangling this (and other) complicated knots of competitions. A closer look indicates that for this purpose the experiment discussed is not quite sufficient. A different design, with the so-called serial sacrifices [see Upton *et al.* and Kohn (6)] offers a real possibility.

If the two sets of mice in the experiment were really samples from the same population, then Table 1 would be of considerable significance. The change from ordinary laboratory conditions to "germ-free" conditions can be described as a change in the environment of the animals subjected to irradiation. Table 1 thus illustrates that what some persons might consider a rather minor environmental change can result in very profound changes in the pattern of death rates.

Currently, there are two different theoretical approaches to the problem of competing risks: the "potential survival time" approach and the "Markov chain" approach. The first is comparatively easy to use and has a substantial literature. The second is very messy and I am not sure that it was ever used in practice. Unfortunately, the potential survival time methodology is now known (7) to be unreliable. The theory of the Markov chain approach is given by Chiang (8) and work is in progress to simplify its use.

Competing Risks in Real Life

While Table 1 illustrates the complexity of competing risks in an experimental situation, in real life these complexities are much greater. In conditions of real life only observational-epidemiological studies are possible (1). Here one of the great difficulties is the necessary complete inclusiveness of important pollutants. If, in an effort to identify the causes of, say, an increase in the frequency of cancer, one omits from an

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observational study a really important agent, then the effects of this agent will be ascribed to some other agent, possibly quite innocent. Yet, the published studies tend to concentrate on pollution of just one kind (for example, radioactive pollution) (4) with complete neglect of others (say from big coal-burning plants).

Here, then, before embarking on an epidemiological health-pollution study, we face the problem of identifying *all* the important causative factors, or agents, which have to be carefully observed (monitored) to be later related to frequencies of this or that human ailment. Currently, there is a substantial list of *kinds* of radiation and of chemicals, all of which are suspected of being carcinogenic. To include all of them into the advocated epidemiological study would be a practical impossibility and some selection is unavoidable. This is one of the difficulties requiring an interdisciplinary approach.

Whichever pollutants are chosen, their concentrations in the environment (or doses) must be carefully monitored. Here, the variability may be expected to be more impressive than that in the Walburg experiment discussed above.

Dose-Rate, a Neglected Health-Affecting Agent

Experiments with animals indicate the necessity of monitoring an agent of a different kind, not just the total dose of an agent (whether radioactive or non-radioactive like DDT or defoliants) but also the rate at which the total dose is received by the population. The techni-

Table 1. Percentages of death among irradiated mice. [Data from (5); reproduced from (4)]

Cause of death	Germs	Germ-free
Thymic lymphoma	22	35
Reticulum cell sarcoma	38	18
Other causes	39	46

cal term of this special kind of agent is "dose-rate."

Some manifestations of dose-rate effects on humans are familiar. A single sleeping pill taken now and then appears harmless and serves its purpose. These are the low-rate effects of a chemical agent. However, a bottle of sleeping pills consumed at one time may cause death. Experiments with animals illustrate less dramatic, but very important occurrences. Figure 1 summarizes the results of three independent experiments with mice (9). All the mice received the same total dose of a chemical called urethane, namely, 1 milligram per gram of body weight. However, each of the three experiments included two groups of mice. One group received the whole dose at a single injection (high dose-rate), while the other group received the chemical in 12 or in 16 fractions, extended roughly over a whole month (low dose-rate). Figure 1 shows a striking difference between crops of lung tumors in mice depending on the high or low dose-rate. With the high dose-rate, there are many more tumors.

Similar dose-rate experiments have been performed with irradiation. With gamma rays (but, curiously, not with neutrons) the results were similar to the above: the higher the dose-rate the more

cancer was generated. However, these dose-rate effects were observed in experiments with animals: mice, rats, dogs, pigs, and monkeys. But what about humans? One is tempted to make extrapolations from animals to humans, but, as convincingly illustrated by Rall (10), such extrapolations are not reliable. In addition, there is this question: Does real life produce differences in the dose-rate experience with a variety of agents that are sufficient to produce real effects on humans? With reference to radiation, the following section suggests that it might.

Geographical Variability of Atmospheric Radiation

Figure 2 and Table 2 [from Grahn (11)] illustrate the change in the average annual whole body exposure to irradiation as a function of the distance from a nuclear electric power generator. These are estimates computed by Grahn using the in-plant data on releases of the radioactive noble gases. The two charts correspond to two different meteorological sectors in the vicinity of a nuclear electric power generator, and each has two different scales of the vertical axes, one for 1966 and the other for 1972. One of the two curves in each chart corresponds to effluents at ground level and the other to those "elevated" to the atmosphere.

The two charts illustrate a dramatic decrease in the annual exposure to irradiation as one moves away from the release point in one direction or another. The same phenomenon is illustrated in Table 2. Here, the last column, giving 8-year averages for eight counties considered by Grahn, shows variability from

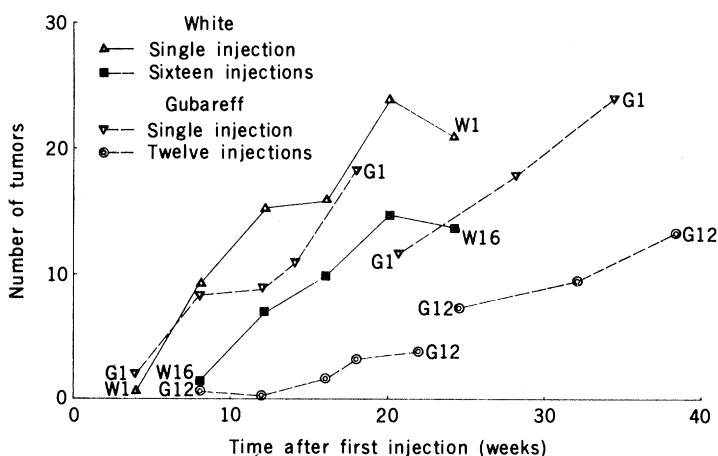


Fig. 1 (left). Comparison of lung tumors in mice induced by the same total dose of urethane, administered at high or low dose-rate. Data from three independent experiments, two by Gubareff and his co-workers and one by White *et al.* [see (9)]. Fig. 2 (right). Total body dose-rate from noble gas exposure for meteorological sectors 1 and 2 as a function of distance from the point of release. [Redrawn from Grahn (11)]

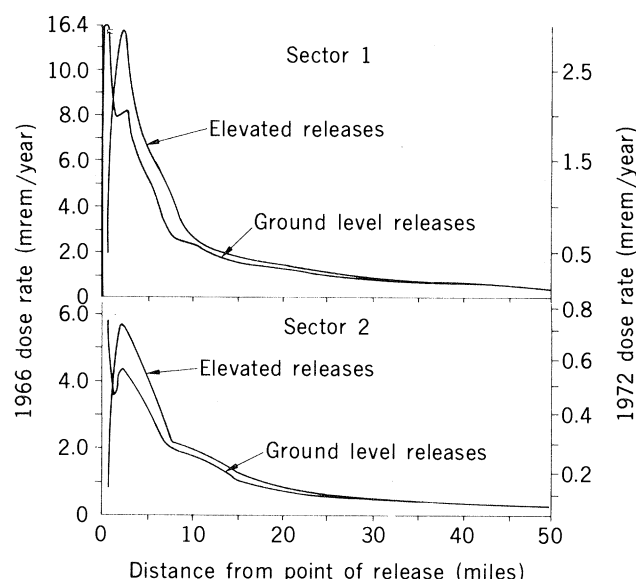


Table 2. Estimated whole body exposures to county populations in Michigan derived from the annual releases of radioactive noble gases. [Data from Grahn (11)]

County	Estimated exposure (mrem/year)								Annual average 1965 to 1972*
	1965	1966	1967	1968	1969	1970	1971	1972	
Antrim	0.15	1.20	0.40	0.40	0.40	0.40	0.50	0.30	0.47
Charlevoix	0.40	3.00	1.10	1.10	1.10	1.10	1.30	0.60	1.21
Cheboygan	0.05	0.40	0.15	0.15	0.15	0.15	0.15	0.20	0.18
Emmet	0.15	1.00	0.35	0.35	0.35	0.35	0.45	0.40	0.43
Grand Traverse	0.10	0.50	0.20	0.20	0.20	0.20	0.20	0.10	0.21
Kalkaski	0.10	0.80	0.30	0.30	0.30	0.30	0.35	0.20	0.33
Leelanau	0.10	0.50	0.20	0.20	0.20	0.20	0.20	0.10	0.21
Otsego	0.10	0.80	0.30	0.30	0.30	0.30	0.35	0.20	0.33

*Population-weighted mean individual annual exposure: 0.4 mrem.

0.18 millirem per year to 1.21 for Charlevoix County where the power plant is located. But these figures represent average annual exposures and the reality of the dose-rate effect discussed above raises the question of whether the annual rate is all that is necessary for estimating public health hazards. Here it is important to remember the sequences of smoggy days in a city and also other sequences of clear days. The appearance of smog depends on combinations of meteorological factors such as inversions and winds which occur everywhere with varying frequencies. The lowest annual exposure calculated by Grahn for Charlevoix County for a recent year (1972) is 0.60 mrem. The question is, what could have been the local exposure over a few really smoggy days in the same year in the same county, say within 5 or 10 miles of the release point? Even more relevant is the question of whether such dose-rate variability in radiation exposure as occurs in real life does or does not affect human population.

Regrets About the NAS-NRC Report

This brings us to the next section which is concerned with a report of the National Academy of Sciences-National Research Council on radioactive pollution and public health (12), the so-called BEIR report.

First published in 1972 and then republished in 1974, the BEIR report is concerned with "an evaluation of present radiation protection guides." Information on the various kinds of possible biological dangers comes from experiments with animals. Because of recognized uncertainties of extrapolations, the actual human safety standards are based on analyses of several experiences with humans, mainly on Hiroshima-Nagasaki studies.

The report contains much useful infor-

mation. My personal regret refers to a contrast between the confident and comforting tone of the first page of the "Summary and Recommendations," on the one hand, and the expressions of doubts and uncertainties in certain passages in the main body of the report, on the other. A few quotations must suffice. On page 1 the following passages appear relevant.

In anticipation of the widespread increased use of nuclear energy, it is time to think anew about radiation protection. . . . In the foreseeable future, the major contributors to radiation exposure . . . will continue to be natural background with an average whole body dose of about 100 mrem/year, and medical applications. . . . Exposures to man-made radiation . . . will produce additional effects that are less in quantity and no different in kind from those which *man has experienced and has been able to tolerate throughout his history.* [Emphasis added.]

Upon reading these passages, one may be inclined to think that the important societal problem mentioned at the outset is essentially solved; no harm worth talking about is to be expected from the proliferation of nuclear power plants. However, this overall comforting confidence of page 1 of the BEIR report appears to contrast with the following passage on page 97:

The available data on radiation-induced cancer in man are relatively scanty, the conditions of exposure nonuniform and uncertain, the irradiated samples highly heterogeneous, the controls uncertainly or crudely matched, the observations confined to limited (high) or ill-defined ranges of dose, dose rate, and fraction of total possible post-exposure risk time, and the effects of variables other than radiation incompletely known.

With particular reference to Hiroshima-Nagasaki studies, a passage in small print on page 101 states:

Homes were destroyed; food was short; living patterns were profoundly disrupted. The influence of this concatenation of disasters upon the subsequent health of the survivors is unknowable.

Although the term competing risks is not used, it is obvious that the author of these statements was aware of the phenomenon and visualized cases in which the post-bombing "traffic" must have prevented many survivors from dying later from cancer or other diseases. However, some other sentences, possibly written by a different person, tend to suggest that the observed death rates may be overestimating the true effects of irradiation. Who knows? This is not impossible. But the competing risk problem is there, complete with all the unresolved statistical difficulties.

The study of survivors of the atomic bomb attacks on Hiroshima and Nagasaki continues (13), but recently it has been reorganized. It is now conducted by the Radiation Effects Research Foundation (RERF) funded equally by Japan and by the United States. The possibly radiation-related clinical disorders and abnormalities detected in atomic bomb survivors are tumors, leukemia, and chromosome aberrations. Among the individuals exposed to irradiation in utero, microcephaly and mental retardation have been noted. All these findings are interesting and important. But it may be hoped that the reports of RERF will not include extrapolations to what might be expected from normal operation of nuclear electric generators. Because of the differences in dose-rate and in competing risks, the reliability of such extrapolations would be illusory.

The Obsolescence of the BEIR Report

With energetic studies being conducted in many centers, it should not be surprising to find that the BEIR report, first published in 1972 and probably written in 1971, is now somewhat out of date. Indeed, specific indications of this being the case are easy to find. One example is the following passage from page 23 of the

BEIR report. It deals with methods of calculating curves such as those in Fig. 2 herein, particularly those relating to "elevated" effluents:

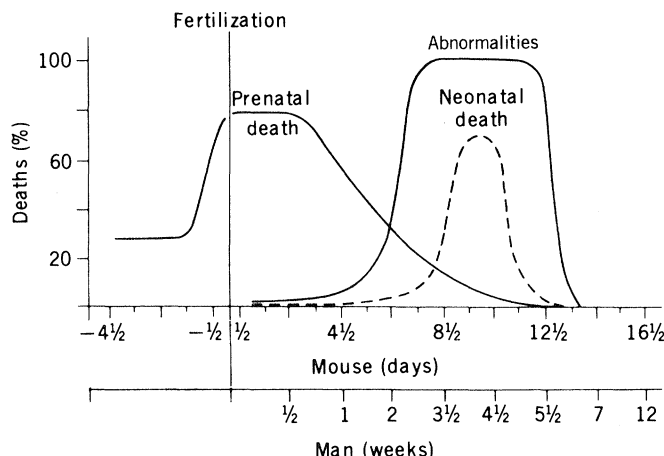
A comprehensive document in this area is *Meteorology and Atomic Energy* edited by D. H. Slade. The relationship of air quality to environmental impact requires an assessment of the amount and nature of radionuclides released, of air concentration integrated over the time of interest, and of deposition or contamination patterns determined from atmospheric and geographical conditions [references cited]. Mathematical simulation of concentration distributions has been more helpful generally than actual measurements of air quality because the requirements of time and space sampling for the latter are difficult to fulfill adequately. Efforts of modeling have been reviewed in detail [references cited]. *In the future, more attention may need to be given to development and implementation of sampling procedures.*

Air quality measurements are normally required to survey distribution of pollutants in anticipation of control strategies, to collect data for research purposes—environmental models, etc., or to document air quality conditions for public record [reference cited]. It is useful to have measurements of both air quality and meteorological parameters. These measurements can be used for radiation-dosage calculations [reference cited]. *The mode of calculation depends upon whether the major effect is from the total radiation received from the cloud, instantaneous peak concentrations or a combination of radiation dose from the passing of the cloud and from contaminated surfaces following the passage.* [Emphasis added throughout.]

This passage deserves special attention. First published in 1972, it provides evidence that scholars at the Atomic Energy Committee were aware of the dose-rate problems in the vicinity of a nuclear reactor sometime before 1968 when Slade's document was published. Also, there is evidence that the authors of the passage just quoted had regrets about the unavailability of "actual measurements" of air quality and hoped that such measurements would become available in due course. Finally, there is evidence that these authors had suspicions about major effects of "peak concentrations" of radioactive effluents from nuclear facilities, either by themselves or, synergistically, with the material on the earth's surface. To this we might add synergisms with nonradioactive pollutants.

Since the passage quoted was written, instruments to monitor continuously the air pollution, both radioactive and non-radioactive, have been constructed (2) and, presumably, perfected. Also, several studies have been published. One example is the three-volume study of the Environmental Protection Agency (14), published in 1973. Another example is

Fig. 3. Incidence of prenatal and neonatal deaths and of abnormal progeny of mice subjected to x-irradiation at varying times, some before and some after fertilization. [Redrawn from Totter (16); data from Russell and Russell (17)]



the set of papers presented at the conference in 1976 on the origins of human cancer (15). This conference included a great variety of contributions relevant to the problem of pollution and health.

The BEIR report thus requires a fundamental revision. The whole problem of pollution and public health ought to be reviewed from beginning to end, taking into account the laboratory and observational findings that became available. The phenomena of competing risks and synergisms indicate that realistic environmental protection guides could be attained only through simultaneous coordinated studies of both radioactive and nonradioactive pollutants. Again, dose-rate effects indicate that, to begin with at least, attempts to formulate radiation protection guides in terms of averages over areas or over time per individual population member would be imprudent.

A Not Very Difficult but Interesting Problem

The organization of the advocated large multipollutant and multilocality epidemiological study can hardly be expected soon (1, 2). First, there must be a broader recognition that such a study is really necessary. Then it will take some time to set up the study. In the meantime a much smaller but still very interesting study seems desirable.

As the authors of certain chapters of the BEIR report are careful to point out, their estimates of genetic and somatic radiation effects on humans are based on extrapolations from experiments with animals, frequently mice. An extrapolation of this kind involves not just one, but two steps. One step is an extrapolation from a small mammal to a much more complicated and larger mammal, man.

The other step is an extrapolation from controllable laboratory conditions to conditions of an uncontrolled environment. As already pointed out with reference to the Walburg experiment, a change in environment may result in a very impressive change in the patterns of radiation effects.

Figure 3 illustrates the effect of irradiation of mice on the frequency of malformations of the progeny and on the frequency of neonatal deaths (16). If pregnant mice are irradiated at any time during a roughly 1-week period some days after conception, then almost 100 percent of their progeny can be expected to be malformed. Also, Fig. 3 shows a high frequency of neonatal deaths. Finally, it will be seen that the last line of the diagram gives a time scale of similar effects for humans. Naturally, this time scale is an extrapolation.

Figure 3 refers to experiments performed under laboratory conditions with certain chosen doses of irradiation. The very interesting and important question is whether any similar phenomena occur in conditions of real life with such doses of the noxious agents as occur in our present environment. Are there any signs of frequent malformations at birth and of neonatal deaths of any mammals, and particularly humans exposed to some recorded agents, whether radioactive or not?

A study which might provide answers to such questions, at least with reference to domestic animals, now appears possible. This possibility is connected with the current happenings in the vicinity of Denver, near Rocky Flats. Frequent articles in the *Denver Post*, published over a period of more than a year now, have referred to complaints of ranchers about unexpected malformations at birth among the domestic animals they raise. Also, the local public health authorities

are alarmed to the point that the governor of the state has appointed a special task force to investigate the matter, and there are public hearings with spirited discussions.

The increased frequency of malformations among domestic animals in the locality in question is not disputed. It is attributed to the presence of selenium, a toxic chemical entering the food chain through certain plants. The alternative hypotheses about causes of malformations include radioactivity from the nuclear facility at Rocky Flats and also certain nonradioactive effluents from an arsenal.

Here, then, we have a situation in which frequent malformations have been recognized and several more or less specific hypotheses have been proposed regarding possible causes. A competently executed epidemiological study, perhaps including a few other areas, some marked with abundant presence of selenium and some not, might produce very interesting results.

Summary

When a new electricity-producing plant is to be built in a given locality it is natural to take into account the public health consequences of the normal operation of each type of plant contemplated. Here, the fossil-burning plants and nuclear facilities come under consideration. I have attempted to show that, in spite of the many important studies performed, there is currently no reliable methodology to estimate how many more cancer cases, and how many more heart attacks and other diseases have to be anticipated

as a consequence of the normal operation of this or that type of electric generator. In part, this is because the currently available estimates of radiation effects on humans are based on extrapolations from studies of two kinds. Those of one kind may be exemplified by studies of atomic bomb casualties in Hiroshima and Nagasaki. The other kind are laboratory experiments with lower animals, frequently mice. The unreliability of both kinds of extrapolations is connected with the following circumstances: (i) The omnipresent troublesome phenomenon of competing risks. (ii) The dependence of health effects of a given noxious agent on the preexisting local pollution. (iii) The dependence of health effects not only on the "dose" of an agent, but also on the rate at which the agent is administered. (iv) The noted difficulties of making extrapolations from one mammal to another.

Our obtaining reliable estimates of the public health effects of extra pollution from new industrial plants would seem to depend on a large multipollutant and multilocality epidemiological study being conducted—one requiring the cooperative effort of several governmental agencies. However, a much easier study of certain developments in the vicinity of Rocky Flats, Colorado, might provide important direct information on health phenomena as they occur in real life.

References and Notes

1. J. Neyman, in *Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability*, L. LeCam, J. Neyman, E. L. Scott, Eds. (Univ. of California Press, Berkeley, 1972), vol. 6, p. 561.
2. ———, in *ibid.*, p. 575. Many published studies are marked with blunders, including "spurious correlations." The results, found statistically significant, include the startling "discovery" that air pollution with copper dust is helpful against breast cancer.
3. F. Proshan and R. J. Serfling, *Reliability and Biometry, Statistical Analysis of Life Length* (Society of Industrial and Applied Mathematics, Philadelphia, 1974).
4. J. Neyman, *Int. Stat. Rev.* **43**, 253 (1976).
5. D. G. Hoel and H. E. Walburg, *J. Natl. Cancer Inst.* **49**, 361 (1972).
6. A. C. Upton *et al.*, *Radiation Induced Cancer* (International Atomic Energy Agency, Vienna, 1969), p. 425; see also H. I. Kohn *et al.*, *Radiat. Res.* **7**, 407 (1957).
7. A. Tsatis, *Proc. Natl. Acad. Sci. U.S.A.* **72**, 20 (1975); A. Peterson, *ibid.* **73**, 11 (1976).
8. C. L. Chiang, *Introduction to Stochastic Processes in Biostatistics* (Wiley, New York, 1968).
9. J. Neyman and E. L. Scott, *Proceedings of the Fifth Berkeley Symposium on Mathematical Statistics and Probability*, L. LeCam, J. Neyman, E. L. Scott, Eds. (Univ. of California Press, Berkeley, 1967) vol. 4, p. 745; M. B. Shikkin, R. Wieder, D. Marzi, N. Gubareff, V. Suntzeff, in *ibid.*, pp. 707 and 710; M. White, A. Grendon, H. B. Jones, in *ibid.*, p. 721.
10. D. Rall, *J. Wash. Acad. Sci.* **64**, 63 (1974).
11. D. Grahn, *Analysis of Population, Birth and Death Statistics in the Big Rock Point Nuclear Power Station, Charlevoix County, Michigan*, Report 8149 (Argonne National Laboratory, Argonne, Ill., 1975).
12. Advisory Committee on Biological Effects of Ionizing Radiations, *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation* (National Academy of Sciences, Washington, D.C., 1974).
13. S. C. Finch and H. B. Hamilton, *Science* **192**, 845 (1976).
14. *Environmental Analysis of the Uranium Fuel Cycle*, EPA-520/9-73-003-D (Environmental Protection Agency, Washington, D.C., 1973).
15. *Abstracts of Papers Presented at the Meeting on Origins of Human Cancer*, 7 to 14 September 1976 (Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y., 1976).
16. J. R. Totter, in *Proceedings of the Sixth Berkeley Symposium on Mathematical Statistics and Probability*, L. LeCam, J. Neyman, E. L. Scott, Eds. (Univ. of California Press, Berkeley, 1972), vol. 6, p. 71.
17. L. B. Russell and W. L. Russell, *J. Cell. Comp. Physiol.* **43**, 103 (1954).
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