Authors' Misrepresentations of their Data in Attempts to Support The Linear No Threshold Hypothesis

Myron Pollycove*

School of Medicine, University of San Francisco, San Francisco, CA USA

The current status of LNT theory is summarized in National Council on Radiation Protection and Measurements Report 121 on Collective Dose[1]:

> "...essentially no human data can be said to prove or even to provide direct support for the concept of collective dose with its implicit uncertainties of nonthreshold, linearity and dose-rate independence with respect to risk. The best that can be said is that most [sic] studies do not provide quantitative data that, with statistical significance, contradict the concept of collective dose.
>
> Ultimately, confidence in the linear no threshold dose-response relationship at low doses is based on our understanding of the basic mechanisms involved.. [Cancer] could result from the passage of a single charged particle, causing damage to DNA that could be expressed as a mutation or small deletion. It is a result of this type of reasoning that a linear nonthreshold doseresponse relationship cannot be excluded. It is this presumption, based on biophysical concepts, which provides a basis for the use of collective dose in radiation protection activities."

The LNT hypothesis was proposed tentatively more than 40 years ago and has since become firmly established, though still without any supporting low-dose data and contradicted by statistically significant epidemiologic and biologic data. Nevertheless, a *biophysical* presumption is considered sufficient justification for using LNT as the basis for current policy of protecting against levels of radiation far below the *variations* of natural background. Studies initiated with the expectation of demonstrating statistically significant increased risk of cancer at low doses of radiation have failed to do so; some even have shown statistically significant *decreased* risks. Consequent efforts to support the LNT have led to suppression and misrepresentation of their own contradictory data by authors of several studies:

Nuclear Shipyard Worker Study

This thirteen-year occupational study of the health effects of low-dose radiation was performed by the Johns Hopkins Department of Epidemiology, School of Public Health and Hygiene, reported to the Department of Energy in 1991[2] and in UNSCEAR 1994.[3] Professor Arthur C. Upton, who concurrently chaired the NAS BEIR V 1990 Committee on "Health Effects of Exposure to Low Levels of Ionizing Radiation," chaired the Technical Advisory Panel that advised on the research and reviewed results.

*Correspondence: Myron Pollycove
E-mail: pollycove@comcast.net

The results of the study contradict the conclusions of the NAS BEIR V[4] report that small amounts of radiation have risk, the LNT hypothesis. From the database of almost 700,000 shipyard workers, including about 107,000 nuclear workers, two closely matched study groups were selected, consisting of 28,542 nuclear workers (NW) with working lifetime doses ≥5 mSv (many received doses well in excess of 50 mSv) and 33,352 non-nuclear workers (NNW). Deaths in each of the groups were classified as due to: all causes, all malignant neoplasms, leukemia, lymphatic and hematopoietic cancers, mesothelioma, and lung cancer. Increased standard mortality ratios (SMRs) for mesothelioma and lung cancer are related to inhalation of asbestos to which all workers were exposed. NW SMRs for leukemia and lymphatic and hematopoietic cancers were lower, but not significantly, than those of the NNW (Figure 1). Compared to the NNW SMRs of 1.02 for death from "all causes" and 1.12 for death from "all malignant neoplasms," both showing no "healthy worker effect", the highly significant corresponding decreased NW SMRs are 0.77 (16 standard deviations below NNW SMR 1.02) and 0.95 (4 standard deviations below NNW 1.12, $P < 0.0001$). As shown in Figure 1 the SMRs for death from "all malignant neoplasms" were omitted from the Summary of Findings Table 4.1.A and not reported in UNSCEAR 1994. These risk decrements are inconsistent with the LNT hypothesis and do not appear to be explainable by the constantly invoked "healthy worker effect" (Figure 1). The NNW and the NW were similarly selected for employment, were afforded the same health care thereafter, and except for exposure to Co gamma radiation, performed the identical type of work, with a similar median age of entry into employment of about 34 years. This provides evidence with extremely high statistical power that low levels of ionizing radiation are associated with *decreased* risks.

The NCRP SC 1-6 Committee, established to evaluate the LNT model and chaired by Professor Upton, in 1998 discounts this highly significant data: "This interpretation [that there was lower total mortality in the NW than in the NNW] ignores the likelihood of occupational selection factors that led some to qualify for radiation work while others did not. The fact that there was a difference for total mortality, and not just for radiosensitive cancers, supports the interpretation that selection factors were operative." The highly significant SMRs for death from "all malignant neoplasms" shown in Table 3.6.B on page 328 of the DOE report are unmentioned, only the insignificant SMRs for leukemia and lymphatic and hematopoietic cancers are alluded to as "radiosensitive cancers." The committee does not consider that the adaptive responses to radiation that stimulate prevention, repair and removal of metabolic DNA alterations, thereby decreasing DNA mutations, also decrease the risk of death from many other diseases in addition to the risk of death from "all malignant neoplasms."

The 10 million dollar 437 page report[2] was never published. An inquiry to DOE elicited the response, "It wasn't in the contract." The author G.M. Matanoski did publish a one page abstract[5] beginning with, "The Nuclear Shipyard Workers Study (NSWS) was designed to determine whether there is an excess risk of leukemia or other cancers

associated with exposure to low levels of γ radiation. The study compares the mortality experience of shipyard workers who qualified to work in radiation areas to the mortality of similar workers who hold the same types of jobs but who are not authorized to work in radiation area." Again, only the statistically insignificant SMRs for deaths from leukemia and lymphatic and hematopoietic cancers are included: "The data clearly indicate that both nuclear worker groups have a lower mortality from leukemia and lymphatic and hematopoietic cancers than does the nonnuclear group. All three groups have lower rates than the general population." The last sentence implying a "healthy worker effect" is incorrect. The SMRs of the NNW for leukemia and lymphatic and hematopoietic cancers, 95% confidence intervals shown within ( ), are 0.97 (0.65, 1.39) and 1.10 (0.88, 1.37), respectively. The significantly lower NW SMRs for deaths from "all causes" and from "all malignant neoplasms" are unmentioned.

This study with internal comparison of NW with carefully matched NNW is designed by the technical advisory panel to eliminate any "healthy worker effect" from the comparison. Even the NNW did not demonstrate "healthy worker effect." Nevertheless, the September 1991 DOE press release, "A Study of Mortality in Shipyard Workers involved in Work on Naval Nuclear-Powered Ships" states, "The results of this study indicate that the risk of death from all causes for radiation-exposed workers was much lower than that for U.S. males. These results are consistent with other [sic] studies showing that worker populations tend to have lower mortality rates than the general population because workers must be healthy to be hired, and must remain healthy to continue their employment."

- Cancer Mortality among Nuclear Industry Workers in Three Countries

This analysis of nuclear worker mortality is based upon studies and nationally combined analyses performed in the U.S., the U.K. and Canada[6]. Seventeen authors present the results of internationally combined analyses of mortality data on 95,673 nuclear workers. The U.S. Naval Shipyard Worker Study with 106,851 nuclear workers is omitted.

The authors conclude, "There was no evidence of an association between radiation dose and mortality from all causes or from all cancers. Mortality from leukemia, excluding chronic lymphocytic leukemia (CLL)-the cause of death most strongly and consistently related to radiation dose in studies of atomic bomb survivors and other populations exposed at high dose rates–was significantly associated with cumulative external radiation dose (one-sided *P* value = 0.046; 119 deaths)."

The data presented of deaths from all leukemias except CLL contradicts this statement of significant association of these leukemia deaths with cumulative external dose (Figure 2). The authors state that their analysis is based upon 119 deaths though only

36 deaths were selected. Since fewer deaths than expected were observed in 4 of the 7 dose categories, these 86 of 119 deaths are discarded by using one-sided *P* values. Justification of the use of one-sided *P* values is stated in Statistical Methods: "As there was no reason to suspect that exposure to radiation would be associated with a decrease in risk of any specific type of cancer, one-sided tests are presented throughout." Yet the authors were aware that exposure to radiation *was* associated with a decrease in risk of at least one specific type of cancer, namely "CLL". It was for this reason that the classification "leukemia, *excluding* chronic lymphocytic leukemia (CLL.)" was used for analysis.

After application of one-sided *P* values only 33 statistically insignificant deaths are distributed among the remaining 3 categories. Another statistical method is used to simulate statistical significance: "....for leukemia excluding CLL, multiple myeloma and all cases where the test statistic exceeded 1.28 (corresponding to a one-tailed *P* value of 0.10) and the number of deaths was less than 30, the *P* value presented was estimated using computer simulations based on 5000 samples, rather than the normal approximation."

This well-funded International Agency for Research on Cancer widespread amalgamation of very heterogeneous monitoring and health policy data that lacks internal comparisons with non-nuclear workers, was able to generate only a single spurious association between non-CLL leukemia deaths and cumulative external radiation. This was accomplished by using the small fraction of these deaths selected by one-sided *P* values and then amplifying these 33 deaths to 5,000 in order to simulate a statistically significant trend *P* value of 0.046.

- Canadian Breast Cancer Mortality between 1950 and 1980 of Patients Fluoroscoped During Treatment for Tuberculosis

The mortality from breast cancer was examined in this medical cohort study of 31,710 women treated for tuberculosis in Canadian sanatoriums between 1930 and 1952[7]. More than 26% had received radiation doses to the breast of 10 cGy or more from repeated fluoroscopic examinations during therapeutic pneumothoraxes. The standardized mortality rates are related to breast radiation doses and presented only in a table (Table I). The authors compare linear and linear-quadratic dose-response models fit to the data and conclude, "that the most appropriate form of dose-response relations is a simple linear one, with different slopes for Nova Scotia and the other provinces."

On the basis of this linear model that includes only non-significant data and excludes the data with the highest confidence limits (Figure 3), the authors predict the lifetime excess risk of death from breast cancer after a single exposure at age 30 to 1 cGy(1r) to be approximately 60 per million women or 900 per million women exposed to 15 cGy.

The observed data, however, demonstrate with high statistical confidence, a reduction of the relative risk of death from breast cancer to 0.66 (P=0.01) at 15 cGy and 0.85 (P-0.32) at 25 cGy. The study actually predicts that a dose of 15 cGy would prevent 7,000 deaths from breast cancer in these million women. Lauriston S. Taylor, past president of the National Council on Radiation Protection and Measurements (NCRP), considered application of LNT hypothesis for calculations of collective dose as, "deeply immoral uses of our scientific knowledge."[8]

- Canadian Breast Cancer Mortality between 1950 and 1987 of Patients Fluoroscoped During Treatment for Tuberculosis

This medical cohort study of 31,917 women treated for tuberculosis in Canadian sanatoriums between 1930 and 1952[9] is a revision of the initial study[7] by the second author of the initial study, G.R. Howe. The relative risks are related to breast radiation doses and presented only in tables. The authors conclude, "There is strong linear trend of increasing risk with increasing dose *(P<0.0003)."*

This conclusion is based upon the high dose studies shown in Table II. High doses up to more than 10 Gy are used to extrapolate linearly to risks incurred by routine diagnostic doses to the breast, about 0.002 Gy for current mammography. The introduction attempts to justify this approach: "However, the breast tissue doses of current concern are primarily those associated with routine diagnostic procedures, particularly mammographic screening. Such doses are substantially lower than the average breast tissue dose received by women in the atomic bomb and medical cohort studies [sic]. This necessitates the development of mathematical models for risk projection, based on observations in the high-dose studies, which can then be used to extrapolate to the low doses of current interest."

Mammographic screening doses are not, "substantially lower than the average breast tissue dose received by women in the ...medical cohort studies." Current mammography doses to the breast are about equal to the 0.002 Gy breast doses delivered in the Canadian medical cohort study[7] by each postero-anterior (PA) fluoroscopic examination in all provinces except Nova Scotia. In Nova Scotia the dose to the breast from each antero-posterior (AP) fluoroscopic examination was increased by a factor of 25 to 0.05 Gy.

Aware of his 1989 medical cohort study finding of reduced relative risks of death from breast cancer following mean cumulative doses of about 15 and 25 cGy, Howe in this 1996 revision attempts to suppress this contradictory data by including them with higher dose data to create a lowest dose category of 0.01-0.49 Sv(Gy). The relative risk of this new category is 1.05, marginally larger than the 1.04 relative risk of the next

0.50-0.99 dose category that has a mean dose about 3x larger; neither relative risk is statistically significant since their 95% confidence levels are 0.84-1.30 and 0.80-1.36, respectively.

- Studies of the Mortality of Atomic Bomb Survivors. Cancer: 1950-1990

The mortality from cancer was examined in this cohort study[10] of 86,572 subjects of which 36,459 received DS86 or T65D weighted colon doses of <5mSv or <~20mSv, respectively. These were considered to have 0 dose and used as controls. The remaining 50,113 subjects had an estimated 420 excess cancer deaths of which about 86 were due to leukemia and 324 due to solid cancers. The authors conclude, "Excess risks for solid cancer appear quite linear up to about 3 Sv, but for leukemia apparent nonlinearity in dose results in risks at 0.1 Sv estimated at about 1/20 of those for 1.0 Sv. Site-specific risk estimates are given, but it is urged that great care be taken in interpreting these, because most of their variation can be explained simply by imprecision in the estimates." However, the least squares best fit to data of another Radiation Effects Research Foundation (RERF) study, "Dose-Response Analysis of Atomic Bomb Survivors Exposed to Low-Level Radiation,"[11] demonstrates relative risk *decrements* of leukemia mortality of 0.80, 0.77, 0.60, and a risk of 1.0 at DS86 doses of 15, 30, 75, and 150 mSv (0.15 Sv), respectively; none had P<0.13 (Figure 4).

This report has been cited widely as providing additional new data that shows a statistically significant increased solid cancer mortality at doses as low as 5 cSv. The summary of the distribution of solid cancer deaths by dose category during 1950-1990 is shown in Table 3. This table includes the observed numbers of cancer deaths, the expected background deaths, and the resultant excess numbers of deaths. The authors did not present the usual statistical analysis of this data even though, "the question of 'the lowest dose at which there is a statistically significant excess risk' is of interest to some". This analysis of the data was omitted, "Because of the tendency for the failure to find a significant affect to be equated to 'no effect,' this does not reflect a very cogent approach to inference about low-dose risks."

Most readers, however, are interested in knowing the lowest dose at which there is a statistically significant excess risk. For these readers who prefer to make their own inferences, the standard statistical analysis of the authors' data (Table 3) is shown in Figure 5. Not only are the observed excess deaths in the 5 cSv category insignificant (P=0.25), but the observed excess deaths in the 15 cSv category are even less significant (P=0.56). The lowest DS 86 dose at which there is statistically significant observed excess risk of solid cancer mortality is 35 cSv (0.2-0.5 Sv) (P=0.03).

The authors' "very cogent approach" does not use the *observed* excess solid cancer deaths, but substitutes *estimated* excess deaths derived from a model fit that *assumes*

linearity. Only these estimated excess deaths were presented by the authors at the Annual Meeting of the National Council for Radiation Protection in April 1996 (Table 4)[12], 3 months before publication of their report[10]. The *observed* excess deaths and the corresponding *observed* fraction of deaths are included in Table 4 for comparison with the *estimates* presented by the authors.

This 1996 RERF Life Span Study Report 12[10] was used in November 1996 to mobilize support for the LNT theory. The International Commission on Radiation Protection (ICRP) under Chairman Roger Clark and the French Society for Radioprotection reviewed this Life Span Study which includes the 1985-1990 mortality data[10, 13]. The ICRP claimed that analysis of this new data shows a statistically significant increased solid cancer mortality at doses as low as 5 cSv. According to Warren Sinclair, President Emeritus of the NCRP and Chairman of the ICRP Committee 1 which analyses results of health-effects studies, the new results "vindicate" previous recommendations to lower permissible dose limits to 2 rem/year for occupational workers and to 0.1 rem/yr for the general public. "The combination of more data points and a more precise analysis," Sinclair said, "allowed the RERF researchers to state with confidence that excess cancer risk due to radiation was observed at doses as low as 50 mSv"[13]. The "more precise analysis" does not use the *observed* excess solid cancer deaths but substitutes *estimated* excess deaths derived from a model fit that assumes linearity.

Using DS 86 dosimetry and the observed excess deaths, the lowest dose at which there is a statistically significant excess risk is 35 cSv. This dose is still erroneously low. For more than a decade numerous reports have concluded that measurements of neutron activation at Hiroshima are significantly greater than predicted by DS 86, especially at the 1.0-2.0 km ground range which is of most interest for low-dose radiation risk estimation[14-26]. The ratio of measured to DS 86 calculated neutron-induced chlorine activation is 15 ± 2[24]. This is in close agreement with the T65D neutron dose. Though this neutron dose is still about half the gamma kerma, using a neutron RBE of 10-20[27-28], the corresponding neutron dose equivalent is at least 5 times greater than the gamma dose equivalent. This is consistent with direct biological evidence that the majority of the effective dose at Hiroshima was delivered by neutrons. Differences between dose responses of mental retardation[29] (T65D), head size[29] (T65D), and cataract prevalence[30] (DS86) at Hiroshima and Nagasaki indicate that significantly higher doses of neutrons than predicted by both T65D and DS86 dosimetry dominated the total effective dose at relevant locations in Hiroshima.

The most direct biological evidence is provided by the radiation induction of stable ratios (F) of interchromosomal (pericentric inversion) to intrachromosomal (translocation) interarm aberrations[31]. The F ratio of random chromosomal breaks is ~90[32] Gamma rays produce a bias toward intrachromosomal compared with interchromosomal aberrations that reduces the F ratio to ~15.[31,32] High LET neutron

radiation produces a marked increase of intrachromosomal aberrations that further reduces the F ratio to ~6[31,33-35]. The F ratios obtained by three independent measurements of peripheral blood lymphocytes from exposed survivors in Hiroshima and controls are 6.8± 0.4[36] (1968-1969), 5.7 ± 0.4[37] (1977-1992), and 6.2 ± 0.7[38] (1989-1990). These values are consistent with the conclusion that neutrons provided the predominant radiation exposure in Hiroshima producing an F ratio of~6, not 15 as would have resulted from predominant gamma ray exposure predicted by DS86 dosimetry[39].

Both the physical measurements of neutron activation[40,41] and the biological dosimetry of atomic bomb radiation at Hiroshima contradict DS86 dosimetry and are more consistent with the initial T65D dosimetry. Some of the biological dosimetry suggests that neutron radiation at Hiroshima was considerably greater than predicted by T65D dosimetry[29]. Using T65D dosimetry and the statistical analysis (Figure 5) of the 1950-1990 data of cancer mortality shown in Table 3[10], we may conclude that the lowest dose at which there is a statistically significant observed excess risk for solid tumors is >1 Sievert.

The following quotes are excerpted from the Science Times section of the New York Times, 1992[42]:

> In the early 1980's Dr. Loewe and a Livermore colleague, Dr. Edgar Mendelsohn, used the laboratory's increasingly powerful computers to re-evaluate the Hiroshima explosion and found the results sharply at odds with the conventional wisdom [T65D dosimetry]. The new calculations [DS86 dosimetry] showed that neutrons at Hiroshima played almost no role at all, with the bomb's radiation output being primarily gamma rays. While both neutrons and gamma rays cause biological damage by upsetting the delicate machinery of the human cell, the findings suggested that gamma rays (and their close cousins xrays) were much more dangerous than previously believed.
>
> Enter Dr. Straume, a 17-year veteran of Livermore and the field of radiation effects research. Around 1988, he hit upon a novel approach to measuring low-energy neutrons at Hiroshima, using a technique he saw being applied to the dating of rocks. The main tool was an accelerator mass spectrometer, a device perfected in the 1980's that could measure in great detail the subtle differences between chemical isotopes. Dr. Straume used the new tool to measure low concentrations of the isotope chlorine-36, which is formed when natural chlorine-35 captures a slow-moving neutron.

First he tried the technique on local California rocks that he exposed to a calibrated neutron source, and found the technique very accurate. Then, beginning in 1989, he started obtaining samples of concrete and granite from Hiroshima, especially in the area between one and two kilometers from ground zero, which was far enough away for there to be survivors. To date he has measured dozens of Hiroshima samples, with striking results.

"There is a discrepancy of up to 10 times between the numbers of low energy neutrons that have been measured and those that are estimated by the current dosimetry system," Dr. Straume said. "This is a discrepancy that we have to resolve."

Experts now agree that the data are nearly indisputable, but disagree over its significance. At the center of the debate is the question of what is the link, if any, between the slow neutrons found at Hiroshima and their fast, biologically damaging cousins, which have not been measured.

Dr. Straume's guess is that the slow neutrons were originally fast ones that were slowed down just before being captured in a chlorine nucleus a few inches inside a concrete wall, for instance. That would mean the citizens of Hiroshima were mainly hit by fast neutrons.

But Dr. Loewe of Livermore, the revisionist leader of the 1980's, said that assumption could prove false. Some of the neutrons might have been slowed in the atmosphere or near to the bomb itself, before reaching humans, weakening them as a damaging force. There was probably a spectrum of neutron energies at work, he said.

But Dr. Loewe conceded that "somewhere there is something wrong" because of the gap between calculated and measured neutrons. If it turns out that fast neutrons were indeed widely present, he added, it would mean the old dose estimates of the 1960's [T65D] were "almost right by accident."

Dr. Warren Sinclair, Chairman of the Board on Radiation Effects Research at the National Academy of Sciences, said he was closely monitoring the research.

"There's mystery here," he said. "There's no doubt about it. My hunch is that its effect on risk estimates is not going to be that large. But we are pressing forward as fast as we can on this problem."

REFERENCES

1. National Council on Radiation Protection and Measurements. Principles and Application of Collective Dose in Radiation Protection. NCRP Report No. 121, NCRP Bethesda, MD; 1995:45.

2. G.M. Matanoski, Health Effects of Low-Level Radiation in Shipyard Workers Final Report. Baltimore, MD: DOE DE-AC02-79 EV10095; 1991.

3. United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and Effects of Ionizing Radiation; UNSCEAR 1994 Report to the General Assembly, with Scientific Annexes. New York, NY: United Nations, 1994; Annex B. Adaptive Responses to Radiation in Cells and Organisms: 185-272.

4. National Academy of Sciences, Committee on Biological Effects of Ionizing Radiation. Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIER V). Washington: National Academy Press, 1990.

5. G.M. Matanoski, Nuclear Shipyard Workers Study. *Radiat. Res.* **133,** 126-127 (1993).

6. E. Cardis, E.S. Gilbert, L. Carpenter, G. Howe, I. Kato, et.al. Effects of low doses and low dose rates of external ionizing radiation: cancer mortality among nuclear industry workers in three countries. *Radiat. Res.* **142,** 117-132 (1995).

7. A.B. Miller, G.R. Howe, G.J. Sherman, J.P. Lindsay, M.J. Yaffe, P.J. Dinner, H.A. Risch and D.L. Preston. Mortality from breast cancer after irradiation during fluoroscopic examination in patients being treated for tuberculosis. *N. Engl J Med* **321,** 1285-1289 (1989).

8. L.S. Taylor, Some non-scientific influences on radiation protection standards and practice. *Health Phys* **39**, 851-874 (1980).

9. G.R. Howe and J. McLaughlin, Breast cancer mortality between 1950 and 1987 after exposure to fractionated moderate-dose-rate ionizing radiation in the Canadian fluoroscopy cohort study and a comparison with breast cancer mortality in the atomic bomb survivors study. *Radiat Res.* **149**, 694-707 (1996).

11. Y. Shimizu, H. Kato, W.J. Schull and K. Mabuchi, Dose-response analysis of atomic bomb survivors exposed to low-level radiation. RERF Update **4**, (3): 3-4 (1992).

12. D.L. Preston, K. Mabuchi, D.A. Pierce, and Y. Shimizu, Mortality among atomic bomb survivors, 1950-1990. In *Proceedings of the Thirty-Second Annual Meeting of the National Council on Radiation Protection and Measurements (3-4 April, 1996).* Implications of New Data on Radiation Cancer Risk (J.D. Boice, Jr., Ed.). Proceedings No. 18, pp. 31-39. National Council on Radiation Protection and Measurements. Bethesda, MD, 1997.

13. Nucleonics Week. Linear nonthreshold model said vindicated by new data. McGraw-Hill; 1996; **37**(46):1-3.

14. T. Nakanishi, T. Imura, K. Komura and M. Sakanoue, Europium-152 in samples exposed to the nuclear explosions in Hiroshima and Nagasaki. Nature **302,** 132-134 (1983).

15. T. Hamada, Measurement of ^{32}P activity induced in sulfur in Hiroshima. In *U.S.-Japan Joint Workshop for Reassessment of Atomic Bomb Radiation Dosimetry in Hiroshima and Nagaski* (D. J. Thompson, Ed.), pp. 45-54. Radiation Effects Research Foundation, Hiroshima, 1983.

16. M. Sakanoue, K. L. Tan and K. Komura, *In-situ* measurement and depth profile of residual ^{152}Eu activity induced by neutrons from the atomic bomb in Hiroshima. In *U.S.-Japan Joint Reassessment of Atomic Bomb Radiation Dosimetry in Hiroshima and Nagasaki, Final Report* (W. C. Roesch, Ed.), Vol. 2, pp. 261-265. Radiation Effects Research Foundation, Hiroshima, 1987.

17. T. Hamada, Measurements of ^{32}P in sulfur, In *U.S.-Japan Joint Reassessment of Atomic Bomb Radiation Dosimetry in Hiroshima and Nagasaki, Final Report* (W.C. Roesch, Ed.), Vol. 2, pp. 272-279. Radiation Effects Research Foundation, Hiroshima, 1987.

18. M. L. Gritzner and W. A. Woolson, Sulfur activation at Hiroshima. In *U.S.-Japan Joint Reassessment of Atomic Bomb Radiation Dosimetry in Hiroshima and Nagasaki, Final Report* (W. C. Roesch, Ed.), Vol. 2, pp. 283-292. Radiation Effects Research Foundation. Hiroshima, 1987.

19. Nakanishi, K. Kobayashi, T. Yamamoto and K. Miyaji, Residual neutron-induced radioactivities in samples exposed in Hiroghima. In *U.S.-Japan Joint Reassessment of Atomic Bomb Radiation Atomic Bomb Radiation Dosimetry in Hiroshima and Nagasaki, Final Report* (W.C.Roesch, Ed.), Vol. 2, pp. 310-319. Radiation Effects Research Foundation, Hiroshima, 1987.

20. M. Hoshi, K. Yokoru, S. Sawada, K. Shizuma, K. Iwatani, H. Hasai, T. Oka, H. Morishima and D. J. Brenner, Europium-152 activitivy induced by Hiroshima atomic-bomb neutrons. Comparison with the ^{32}P, ^{60}Co and ^{152}Eu activities is in Dosimetry System 1986 (DS86). *Health Phys.* **57**, 831-837 (1989).

21 T. Kimura, N. Takano, T. Iba, S. Fujita, T. Watanabe, T. Maruyama and T. Hamada, Determination of specific activity of cobalt ($^{60}Co/Co$) in steel samples exposed to the atomic bomb in Hiroshima. *Radiat. Res.* ***31,*** 207-213 (1990).

22. G. D. Kerr, F. F. Dyer, J. V. Pace, III, R. L. Brodzinski and J. Marcum, *Activation of Cobalt by Neutrons from the Hiroshima Bomb.* Report ORNL-6590, Oak Ridge National Laboratory, Oak Ridge. TN, 1990.

23. T. Nakanishi, T. Ghtani, R. Mizuochi, K. Miyaji, T. Yamamoto, K. Kobayashi and T. Imanaka, Residual neutron-induced radionuclides in samples exposed to the nuclear explosion over Hiroshima: comparison of the measured values with the calculated values. *Radiat. Res.* **32** (Suppl.), 69-82 (1991).

24. T. , Straume, S. D. Egbert, W. A. Woolson, R. C. Findel, P. W. Kubik, H. E. Gove, P. Sharma and M. Hoshi, Neutron discrepancies in the DS86 Hiroshima dosimetry system. *Health Phys.* **63**, 421-426 (1992).

25. K. Shizuma, K. Iwatani, H. Hasai, M. Hoshi, T. Oka and H. Morishima, Residual ^{152}Eu and ^{60}Co activities induced by neutrons from the Hiroshima atomic bomb. *Health Phys.* **63**, 421-426 (1992).

26. D. C. Kaul, W. A. Woolson, S. D. Egbert and T. Straume, A brief summary of comparisons between the DS86 A-bomb survivor dosimetry system and *in-situ* measurements in light of new measurements, revised nuclear data and improved calculational methods. In *Proceedings of the Eighth International Conference on Radiation Shielding,* pp. 232-237. American Nuclear Society, New York, 1994.

27. D. J. Brenner, Significance of neutrons from the atomic bomb at Hiroshima for revised radiation risk estimated. *Health Phys.* **60** 439-442 (1991).

28. RP, 1990 Recommendations of the International Commission on Radiological Protection, Publication 60. *Annals of the ICRP* **21**. Pergamon Press, Oxford, 1991.

29. W. J. Blot, Growth and development following prenatal and childhood exposure to atomic radiation. *Radiat. Res.* **16**, Suppl., 82-88 (1975).

30. M. Otake and W. J. Schull, Radiation-related posterior lenticular opacities in Hiroshima and Nagasaki atomic bomb survivors based on the DS86 dosimetry system. *Radiat. Res.* **121**, 3-13 (1990).

31. D. J. Brenner, Direct biological evidence for a significant neutron dose to survivors of the Hiroshima atomic bomb. *Radiat. Res.* **145**, 501-507 (1996). [Commentary]

32. L. Hlatky, R. K. Sachs and P. Hahnfeldt, The ratio of dicentrics to centric rings produced in human lymphocytes by acute low-LET radiation. *Radiat. Res.* **129**, 304-308 (1992).

33, J. Pohl-Ruling, P. Fisher, D. C. Lloyd, A. A. Edwards, A. T. Natarajan, G. Obe, K. E. Buckton, N. 0. Bianchi, P. P. W. Buul, B. C. Das, F. Dashil, L. Fabry, M. Kucerova, A. Leonard, R. N. Mukherjee, U. Mukherjee, R. Nowotny, P. Palitti, Z. Poliakova, T. Sharma and W. Schmidt, Chromosomal damage induced in human lymphocytes by low doses of D-T neutrons. *Mutat. Res.* **173**, 267-272 (1986).

34. A. Bender and P. C. Gooch, Persistent chromosome aberrations in irradiated human subjects. *Radiat Res.* **16**, 44-53 (1962).

35. M. A. Bender and P. C. Gooch, Persistent chromosome aberrations in irradiated human subjects, II. Three and one-half year investigation. *Radiat. Res.* **18**, 389-396 (1963).

36. A. A. Awa, T. Sofuni, T. Honda, M. Itoh, S. Neriishi and M. Otake, Relationship between the radiation dose and chromosome aberrations in atomic bomb survivors of Hiroshima and Nagasaki, *Radiat. Res.* **19,** 126-140 (1978).

37. K. Ohtaki, G-banding analysis of radiation-induced chromosome damage in lymphocytes of Hiroshima A-bomb survivors. *Jpn. J. Human Genet.* **37**, 245-262 (1992).

38. R. K. Sachs, A. Awa, Y. Kodama, M. Nakano, K. Ohtaki and J. N. Lucas, Ratios of radiation-produced chromosome aberrations as indicators of large-scale DNA geometry during interphase. *Radiat. Res.* **133,** 345-350 (1993).

39. W. H. Roesch, Ed., U. *S.-Japan Joint Reassessment of Atomic Bomb Radiation Dosimetry in Hiroshima and Nagasaki.* Radiation Effects Research Foundation, Hiroshima, 1987.

40. T. Straume, L. J. Harris, A. A. Marchetti and S. Q. Egbert, Neutrons confirmed in Nagasaki and at the Army Pulsed Reactor Facility: Implications for Hiroshima. *Radiat.Res.* **138**,193-200 (1994).

41.. W. A. Rhoades, J. M. Barnes and R. T. Santoro, An explanation of the Hiroshima activation dilemma. In *Proceedings of the Eighth International Conference on Radiation Shielding,* pp. 238-244. American Nuclear Society, New York, 1994.

42. W.J. Broad, New study questions Hiroshima radiation. In *Science Times,* pp C1, C10, C11. The New York Times, New York, October 13, 1992.

A:/nonlinearmisterpretations.wpd

Nuclear Worker Cumulative Dose: 0.5 – >40 cSv (rem)
SUMMARY OF FINDINGS: SMR Ratios Table 4.1.A
*OTHER CAUSES OF DEATH: SMR Ratios Tables 3.6.B (NW), 3.6.D (NNW)

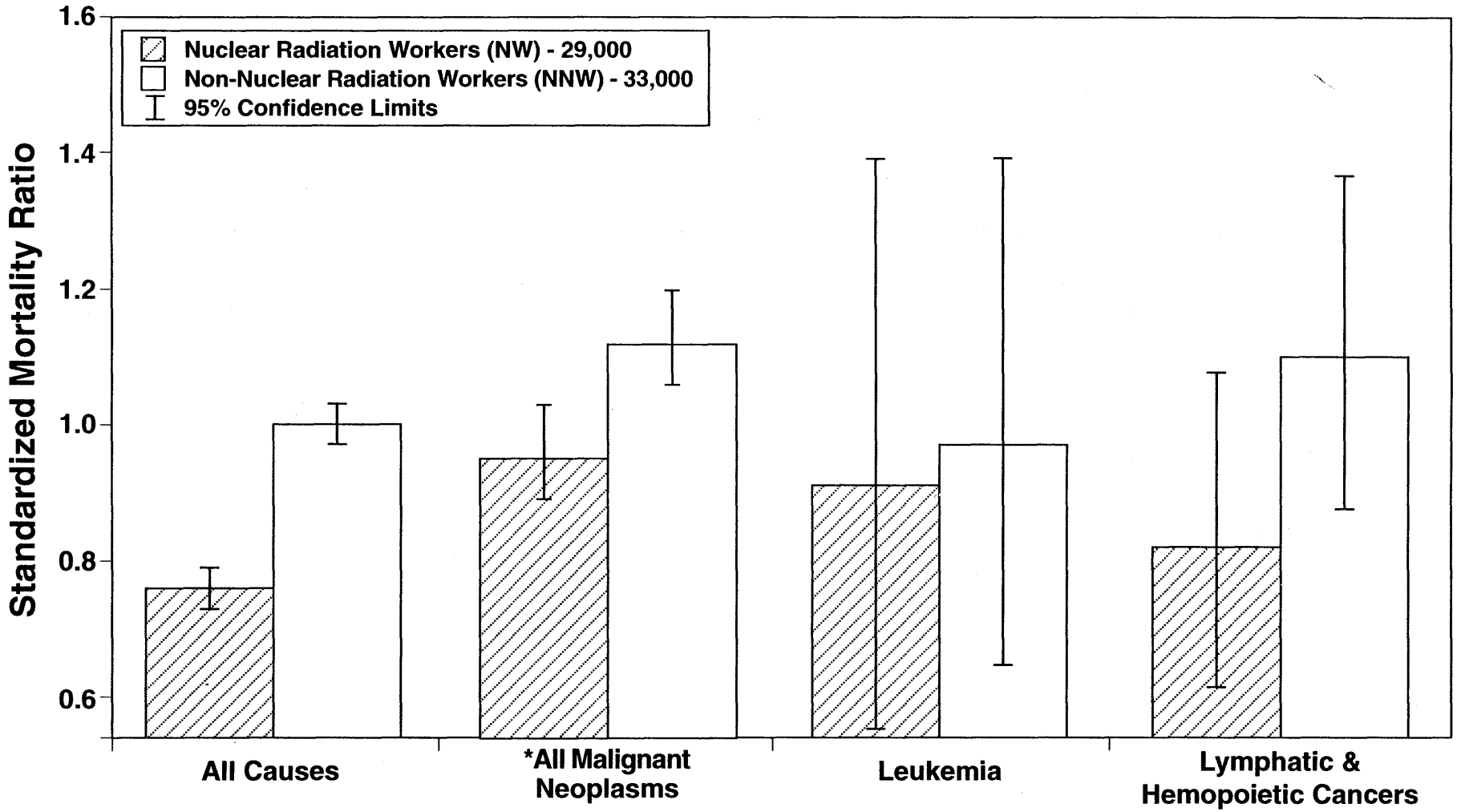

Figure 1. Standardized mortality ratios for selected causes of death among shipyard workers in the U.S. Matanoski GM. (1991)

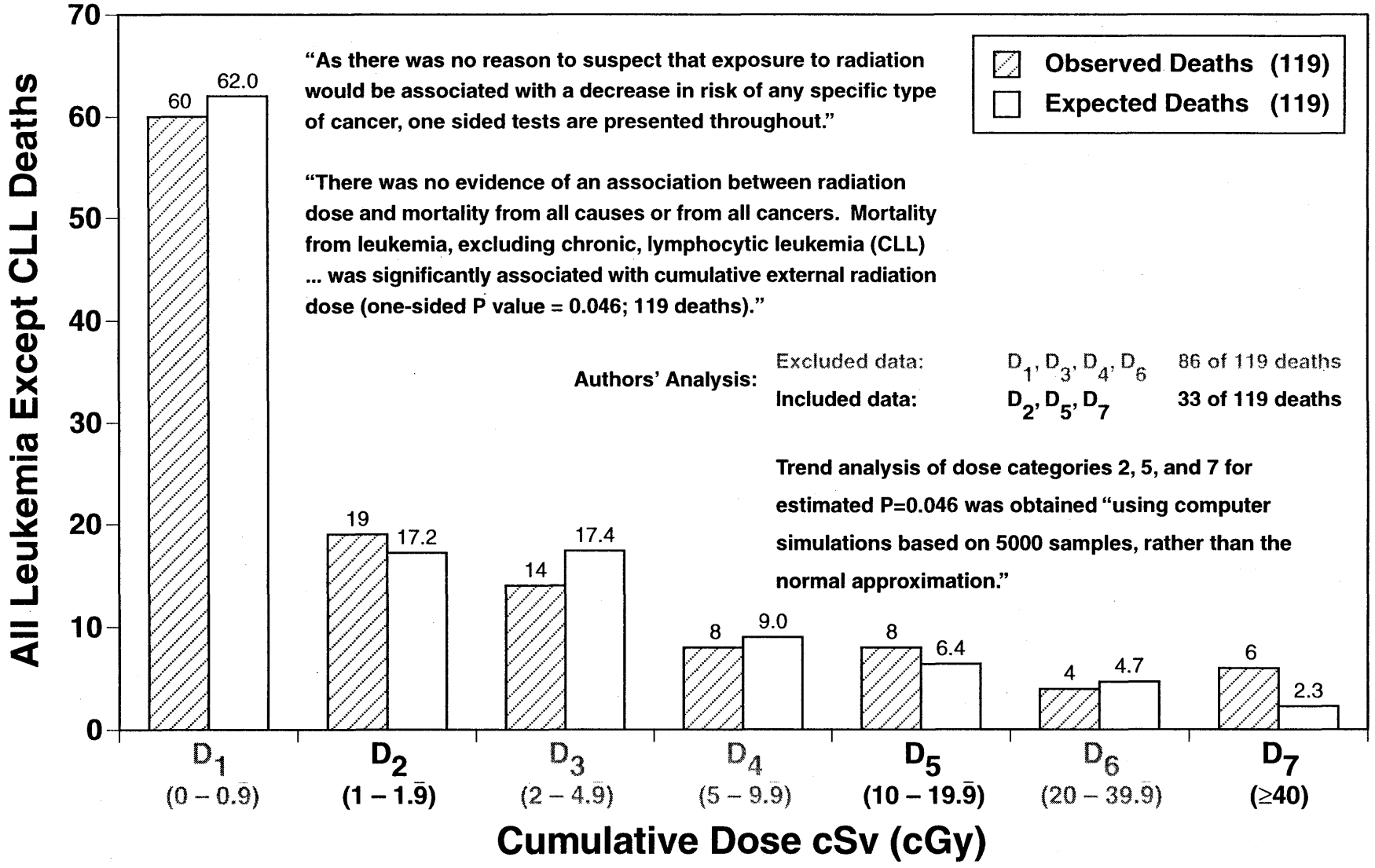

Figure 2. Cancer mortality among nuclear industry workers in three countries. Cardis E, et al. (1995)

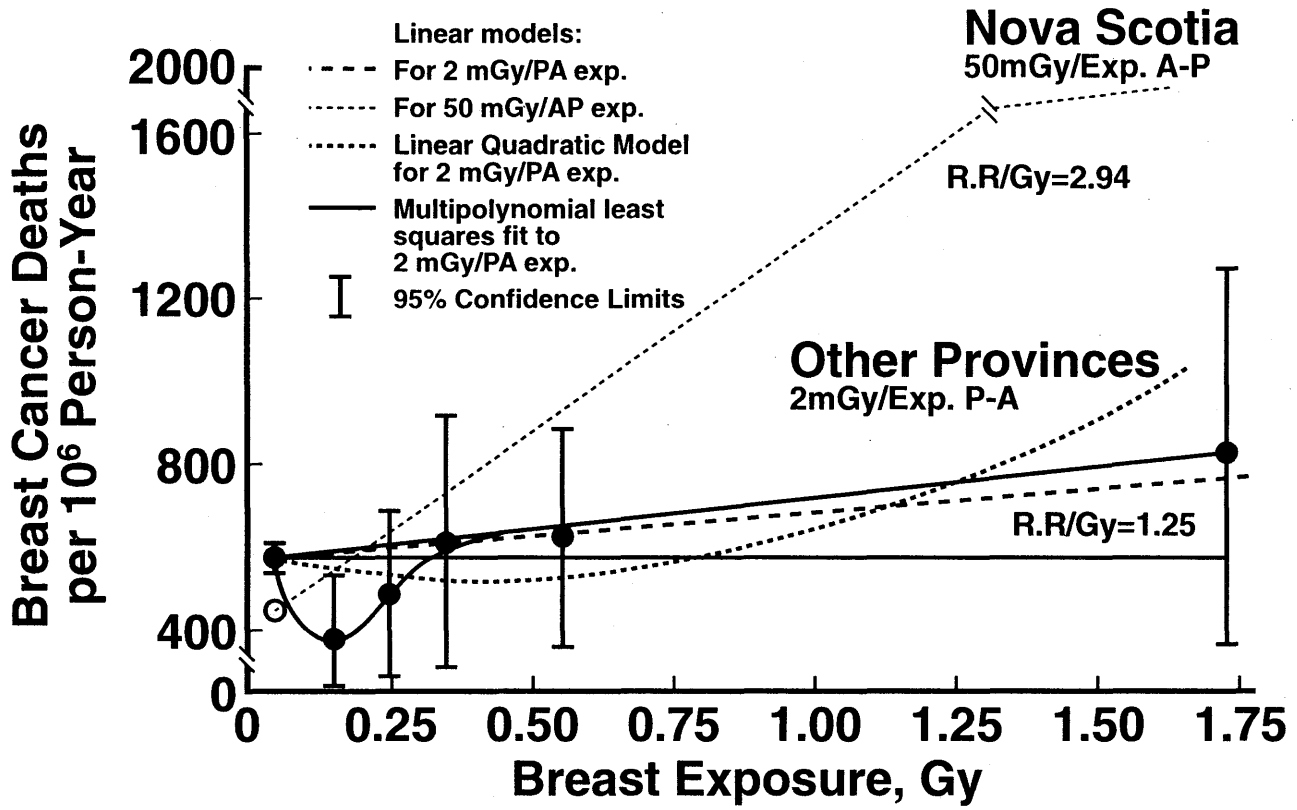

Figure 3. Canadian breast fluoroscopy study. Adapted from Miller AB, et al. (1989)

Leukemia Mortality

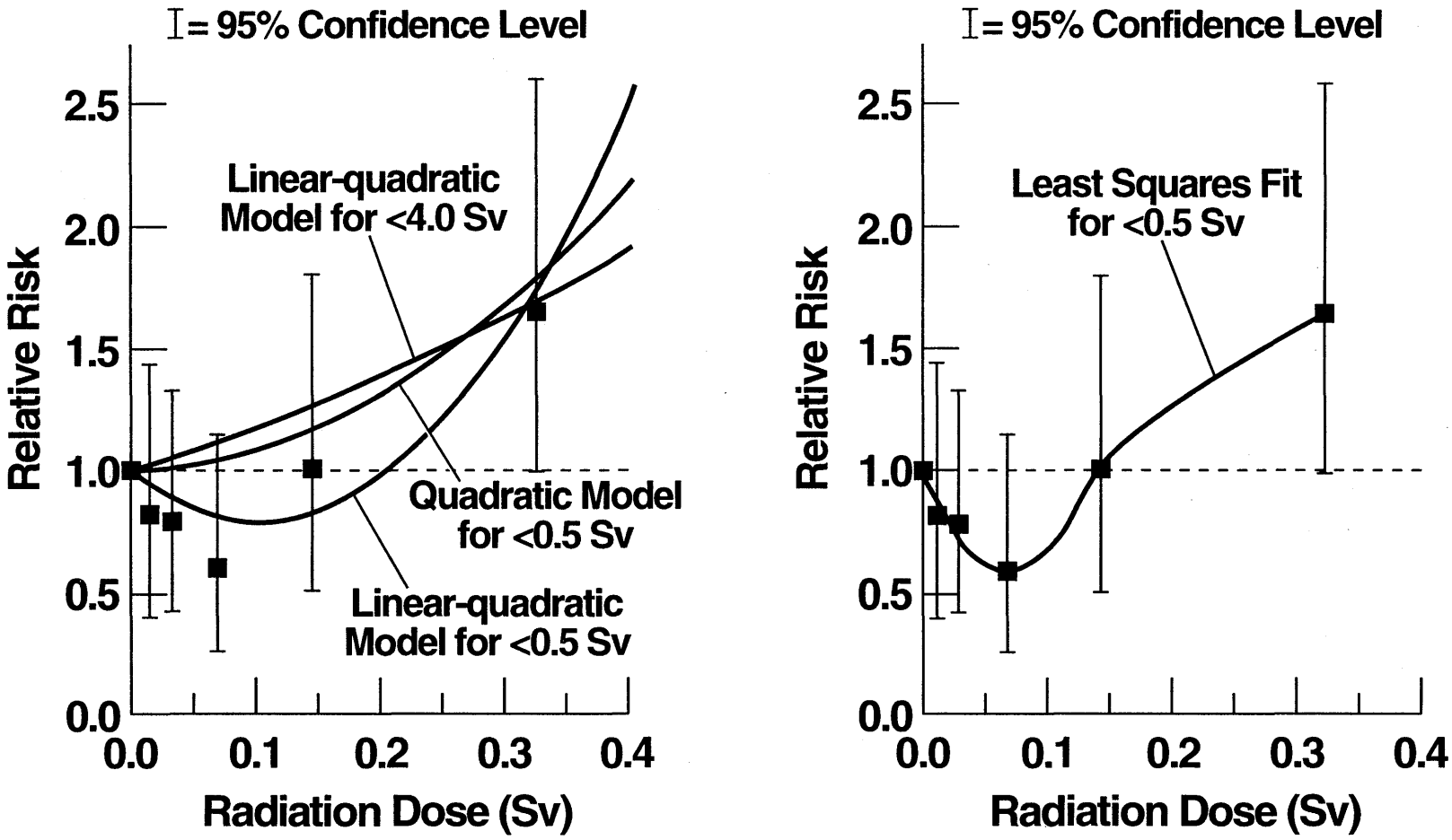

Figure 4. Dose-response analysis of relative risk of leukemia mortality of atomic bomb survivors exposed to low-level radiation. The left figure shows the best fit models of the authors to their data. The right figure shows the least squares fit to their data.

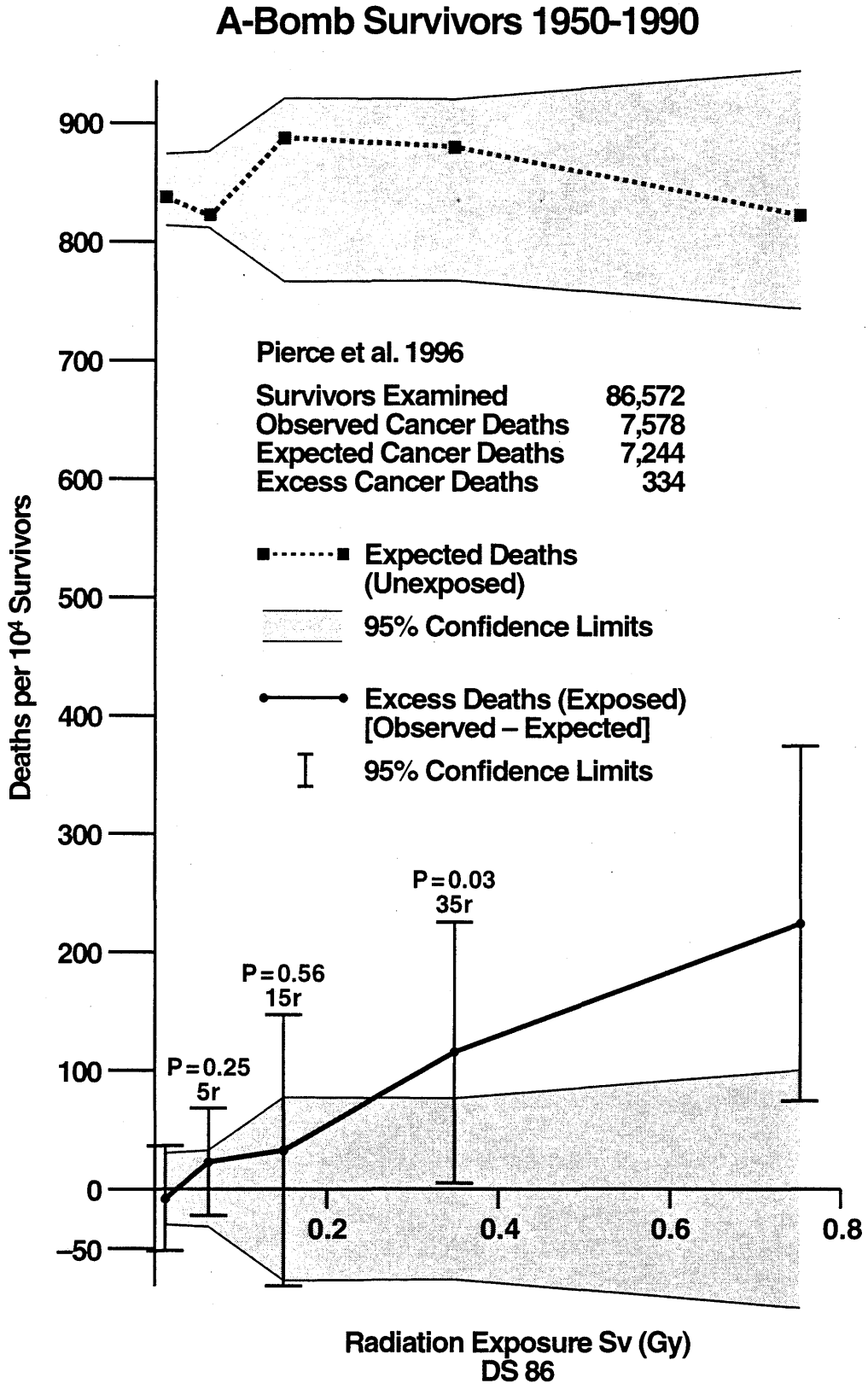

Figure 5. Statistical analysis of solid cancer mortality data of atomic bomb survivors 1950-1990: Expected Deaths and Excess Deaths. Adapted from Pierce DA, et al. (1996)

TABLE I
Breast Cancer Mortality in the Canadian Fluoroscopy Cohort Study (1950–1980) Standardized Mortality Rates by Dose Category

	Nova Scotia		Other Provinces	
Dose (Gy)	Deaths	SMR	Deaths	SMR
0-0.09	13	455.6	288	585.8
0.10–0.19			29	389.0
0.20–0.29			24	497.8
0.30–0.39	11	1709	17	630.5
0.40–0.69			19	632.1
0.70–0.99				
1.00–2.99	14	2060		
3.00–5.99	13	2811	14	873.1
6.00–10.00	8	7582		
≥10.00	12	21.810		

Adapted from Miller AB, Howe GR, et al. (1989)

TABLE II
Breast Cancer Mortality in the Canadian Fluoroscopy Cohort Study (1950–1987) Relative Risks by Dose Category

	Nova Scotia		Other Provinces	
Dose (Sv)	Number of Deaths	Relative Risk (95% Confidence Interval)	Number of Deaths	Relative Risk (95% Confidence Interval)
<0.01	23	1.00	309	1.00
0.01–0.49	8	1.50 (0.68, 3.27)	112	1.05 (0.84, 1.30)
0.50–0.99	6	1.99 (0.82, 4.82)	67	1.04 (0.80, 1.36)
1.00–1.99	14	3.15 (1.66, 5.98)	61	1.22 (0.93, 1.61)
2.00–2.99	12	4.15 (2.11, 8.16)	15	1.24 (0.73, 2.08)
3.00–3.99	10	3.59 (1.75, 7.38)		
4.00–6.99	15	2.82 (1.33, 5.94)		
7.00–9.99	7	13.9 (5.97, 32.2)	14	2.24 (1.31, 3.83)
≥10.00	8	43.7 (19.2, 99.5)		

Adapted from Howe GR, McLaughlin J. (1996)

TABLE III
Observed and Expected Deaths for Solid Cancers

Dose (Sv)	Sujects	1950-1990			1986-1990		
		Observed Deaths	Expected Background	Excess Deaths	Observed Deaths	Expected Background	Excess Deaths
0 (<0.005)	36,459	3013	3055	-42	489	496	-7
0.005-0.1	32,849	2795	2710	85	443	428	15
0.1-0.2	5467	504	486	18	90	74	16
0.2-0.5	6308	632	555	77	106	85	21
0.5-1.0	3202	336	263	73	48	42	6
1.0-2.0	1608	215	131	84	40	22	18
>2.0	679	83	44	39	11	7	4
Total	86,572	7578	7244	334	1227	1153	74

From Pierce, et al., (1)

TABLE IV
Excess Solid Cancer Deaths in the Japanese Life Span Study Cohort by Dose, 1950-1990*

Dose (Sv)	Subjects	Observed Deaths	Excess Deaths		Fraction (%)	
			Obs	Estimated**	Obs	Attributable**
0 (<0.005)	36,459	3,013	-42	0.0	-1	0
0.005-0.1	32,849	2,795	85	33.7	3	1
0.1-0.2	5,467	504	18	28.7	4	6
0.2-0.5	6,308	632	77	74.6	12	12
0.5-1.0	3,202	336	73	77.9	22	23
1.0-2.0	1,608	215	84	70.0	39	33
>2.0	679	83	39	48.9	47	59
Total	86,572	7,578	334	333.9		

* Pierce, Shimizu, Preston, Vaeth, Mabuchi. Rad. Res. July 1996

** Preston, Mabuchi, Pierce, Shimizu. NCRP, April 1996
Observed Excess Deaths and Observed Fraction were not shown.