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10 June, 1996

US Nuclear Regulatory Commission
Attn: Noel Dudley
ACRS M/S T-2 E 25
2 White Flint North
11545 Rockville Pike
Rockville MD 20852-2738

Dear Mr. Dudley:

Enclosed is a copy of a presentation that Mr. James B. Muckerheide made recently to the Massachusetts' Governor's Advisory Council on Radiation Protection. This presentation contains information that bears directly on the subject of the health effects of low-levels of ionizing radiation. I understand that subject will be discussed during the June 14, 1996 meeting of the ACRS. I offer this document as testimony providing evidence that the effects of low-levels of ionizing radiation are not harmful to humans.

Mr. Muckerheide gave me permission to provide this document with the caveat that distribution is as "a preliminary summary of a partial list of significant data" produced without clerical or editorial report-writing support." Mr. Muckerheide is currently seeking government or private support to prepare a more complete and adequately edited and summarized report. If, as a result of perusing this document, the ACRS is inclined to provide such support, it can contact Mr. Muckerheide at the Massachusetts Emergency Management Agency, 400 Worcester Road, P.O. Box 1496, Framingham MA 01701-0317, phone 508-820-2039.

I, as a health physicist certified by the American Board of Health Physics, have studied and worked in the field of radiation protection for over 40 years. I find the information in Mr. Muckerheide's document persuasive in demonstrating that the costly work performed by NRC licensees in reducing ionizing radiation exposures to their employees and to the environment is providing no benefit to the American people, to the workers, or to the environment. Moreover, the design requirements for decommissioning and decontamination work and waste disposal facilities (both high and low level) that require reducing exposure to ionizing radiation to natural background levels are, to me, far too stringent. The Commission's regulations in 10 CFR 20 are too stringent when they demand no exposure of the public outside of restricted or controlled areas above 100 mrem per year and impose costly evaluations and actions to keep doses as low as reasonably achievable below applicable limits to both workers and the environment.

The National Council on Radiation Protection and Measurements (NCRP) has established a subcommittee chaired by Arthur Upton to examine the current state of knowledge about the effects of low levels of ionizing radiation. This work is funded by the NRC. I trust that the ACRS will take whatever action it can to ensure information such as that in Mr. Muckerheide's document is included in the deliberations of the NCRP in this matter. It appears to me that the NCRP has become somewhat one sided in its insistence on adherence to the linear no-threshold hypothesis as the basis for setting radiation protection standards in light of current information on the effects of low-levels of exposure to ionizing radiation. ALL of the currently-available information on the effects of low-levels of ionizing radiation must be taken into account by the NCRP, including information that demonstrates no harmful effect and that indicates a beneficial effect if the United States is to have a rational and efficient radiation protection program.

I trust that this information will be useful to the ACRS and will be happy to provide additional information if necessary (Phone: 208-524-3800).

Sincerely,

Al N. Tschaeche, CHP

cc: M. Goldman w enclosure
J. B. Muckerheide w/o enclosure
A. Upton w enclosure

# PRESENTATION AND REPORT TO MASSACHUSETTS' GOVERNOR'S ADVISORY COUNCIL ON RADIATION PROTECTION

JAMES MUCKERHEIDE, MASS. STATE NUCLEAR ENGINEER
Massachusetts Emergency Management Agency
Governor's Advisory Council on Radiation Protection

David Balfour, Research Assistance, Worcester Polytechnic Institute

December 9, 1994
March 10, 1995
June 2, 1995 (Dr. Mark Johnson - CARS present "linear model" sources)
Sep 8, 1995 (Response to CARS sources)
March 29,1996 (compiled data source materials)

# LOW LEVEL RADIATION HEALTH EFFECTS: A COMPILATION OF DATA AND PROGRAMS

J. MUCKERHEIDE
MASS. STATE NUCLEAR ENGINEER

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MASS. GOV. ADVISORY COUNCIL ON RADIATION PROTECTION

# SOURCES OF LOW LEVEL RADIATION EXPOSURE DATA

## Human Populations Exposed to Low Level Radiation

1.0 Japanese Atomic Bomb Survivors
2.0 Occupationally Exposed (Radiologists, Military, & Industry)
3.0 Medically Exposed ($I^{131}$, X-rays, etc.)
4.0 Radium Ingestion (Dial Painters, Medical, Nostrums)
5.0 Weapons/Facilities Releases
6.0 Natural Background Radiation Sources

## Radiobiology Research Data

7.1 Non-human Biological Populations
7.2 Cellular, Molecular Biology and Genetics Research

# EXECUTIVE SUMMARY

## ACTUAL DATA SHOW NO ADVERSE HEALTH EFFECTS

**Compiled data on low to moderate radiation health effects confirms no adverse health effects at doses <10s of rem (<10,000s of mrem).**

Radiation "protection" assumptions of low-dose health effects applies dose-response at very high doses projected over orders of magnitude to very low doses. Such projections are scientifically invalid, and contrary to actual radiation health effects data. Public fear is the result of misrepresenting scientific data, ignoring actual data, suppressing the publication of valid contrary data, eliminating programs that document actual data, and supporting scientifically invalid programs that enhance public fears.

Such government and industry costs are passed through to the public.

Health effects data exist for the following significantly exposed human population groups: **1. Japanese atomic bomb survivors; 2. Occupationally exposed; 3. Medically exposed; 4. Internal radium exposed; 5. Weapons and facility releases; and 6. Natural background radiation.**

In addition, research data comes from **non-human biological populations**, and from **cellular and molecular biology and genetics research.**

## SIGNIFICANTLY EXPOSED POPULATIONS

Significant actual data quantifying low to moderate radiation health effects, ie, radiation doses below about 50 rem (0.5 Gy, 50,000 mrem), with chronic doses to 500 rem (5 Gy, 500,000 mrem) are identified in the following significantly exposed populations:

### 1. Japanese atomic bomb survivors

A population of ~75,000 persons exposed to the atomic bombings of Hiroshima and Nagasaki in 1945, comprising the "exposed group" of persons, estimated to have received >1 rem (0.01 Gy, 1000 mrem), exposed to mean radiation doses of 16-18 rem, and the "unexposed group" of ~35,000 persons in the area exposed to <1 rem, also carrying the health cards of the survivors.

**In this population there is:**

- **No excess total mortality at <200 rad (<200,000 mrad)** in 20,777 deaths from 1950-1985, for radiation exposures <200 rad. Excess mortality risk from 200-400 rad is ~15%, statistically non-significant! Further, 15% excess risk is not normally considered to have epidemiological health effects consequence. The excess risk at >400 rad is 38%.
- **No excess cancer mortality <20 rad (<20,000 mrad)** Excess cancer is only above 20 rad. At 20-50 rem, for "all cancers except leukemia" there is a 12% increase, not statistically significant and of no epidemiological consequence, and for leukemia a 79% increase. From 50-200 rem, "all cancers except leukemia" have increases of limited consequence, and double at >200 rem. For leukemia, there are consequential increases of 4 times control rates at 50-100 rem, 8 times at 100-200 rem, and 18 times at >200 rem.
- **Lower cancer rates than controls at 1 - 9 rem (1,000 - 9,000 mrem)** Total cancer mortality is >100 fewer cancer deaths in the 0.5-5.0 rem population. Similar analyses finds lower cancer than controls in the 1-5 and 1-9 rem range, and lower rates in specific radiogenic cancers, especially leukemia, with a significant reduction in colon cancer.
- **Actual cancer data vs. arbitrary models, reduced effects at low doses.** When models are not artificially constrained to linear/linear quadratic, applied only to high dose effects, polynomial relationships are generally found to be the "best fit."

- **Note: BEIR V (p.242) states: "For the combined data (Hiroshima and Nagasaki), the rate of mortality is significantly elevated at 0.4 Gy (40 Rad) and above, but not at lesser dose."** However, BEIR then uses high-dose data to arbitrarily quantify effects as "per-person-year-Sv", which projects dose effects linearly to zero dose, contrary to (and misrepresenting) the actual data.
- **No teratogenic effects <10 rem, no significant effects <50 rem.** Mental retardation from effects on the fetus was limited to doses >50 rem in Hiroshima, and greater than 300 rem in Nagasaki. Mental retardation was most significant for fetuses exposed at 8-15 weeks gestation. A threshold for severe mental retardation is 55 rem, with IQ or school performance reduced for exposures >10 rem.
- **No genetic effects in children of Japanese survivors.** There is no difference in genetic effects children of exposed survivors and the control population (with parental doses that average 36-60 rem).
- **Exposed survivor population is outliving non-exposed population.** Longevity of the exposed survivors is greater than the controls (hypothesized to be affected by the loss of weaker members at the time of the bombing, but refuted by the data).

## 2. Occupationally exposed workers

This group includes various populations generally identified and monitored as "radiation workers", with a wide range of doses.

Primarily radiologists (to an estimated 500 rem lifetime), early nuclear materials facility workers, with doses >10 rem with moderate dosimetry, and 10,000s of workers have later experience at low doses with good dosimetry.

This group is represented by:

- **Marie Curie, at 1000s of rem, died at age 66, pos: 'ble aplastic anemia.** From 1898, 4 years in a shed, from her early 30s, to separate radium, with "a warm glow in the evening", sufficent to read by, continuing for 12 years; she then developed and applied radiology in WW I, working often 16-18 hour days, days at a time, manually manipulating the x-ray devices, "with the apparatus in action surrounded by a mysterious halo". She fitted 20 "radiologic cars", started 200 field hospital x-ray rooms, and trained 100s of technicians, receiving 1000s rem. After the war she then continued her work at the Curie Insitute of Radium, til her death in 1934.
- **No excess cancers in British radiologists, est. 500 rem (500,000 mrem).** British radiologists before 1921 had 75% excess cancers vs. other physicians (in a 1950s study), but radiologists that started practice after 1921, with enhanced radiation protection practices, had no excess cancer vs. other medical professionals (in a 1981 study).
- **No excess cancer in US Army radiologic technicians, est. 50 rem dose.** In WW II ~6,500 radiological technicians received an estimated 50 rem dose in training (practicing x-rays on each other) have no excess cancer at a 29-year follow-up vs. other Army medical, laboratory, and pharmacy technicians.
- **Mortality and cancer lower in shipyard nuclear workers.** In a $10M study, 1978-1987, ~70,000 of ~700,000 US nuclear shipyard workers were studied. The 28,542 workers at >5 rem dose had 24% less total age-adjusted mortality (statistically significant) than the 33,352 non-nuclear workers; with the nuclear workers at <5 rem dose at 19% less mortality(statistically significant).
- **Mortality and cancer is lower in US nuclear weapons plant workers**
- **Cancer and leukemia are lower in British nuclear weapons plant workers**
- **Cancer is lower in Canadian nuclear plant workers** vs thermal plant workers

## 3. Medical patient exposures

Medical patient groups can be followed to assess radiation health effects to populations at moderate doses far exceeding normal exposures of the public, or even radiation workers. Medical radiation includes x-rays and radioisotopes, in diagnostic and therapeutic applications, with internal and external exposures. Uses include relatively high doses to relatively young and otherwise healthy patients, vs the much higher radiation therapy doses for cancer in life-threatening conditions, with significant doses to older patients. Early use exposed large populations to doses that are relatively high compared to current acceptable practices.

These groups include:

- **No excess leukemia at 10-15 rem WB/bone from $I^{131}$ hyperthyroid therapy.** In 36,000 hyperthyroid patients, 22,000 treated with $I^{131}$, and most others with surgery, at 7-year and 10-year follow-ups, sufficient to see the peak excess leukemia, the $I^{131}$-exposed population had lower (13 ± 3, vs 16 ± 4, not statisically significant) incidence, with BEIR predicting a large increase, to 36 leukemias at 10 rem (46 at 15 rem). In a similar study in Sweden, another 10,000 patients were followed for 15 years, also demonstrating no leukemia increase.
- **No excess thyroid cancer in 50 rem $I^{131}$ diagnosis, not for potential cancer.** Mean thyroid doses of ~50 rem (est 1-3 million US patients before 1968, with no follow-up). In Sweden, a 20-year follow-up of 35,000 patients, ~5% <20 years old, finds less thyroid cancer (0.62 of normal, statistically significant, a much more scientifically valid "protective effect" vs no data indicating an adverse health effect), in patients not diagnosed for possible thyroid cancer,.
- **No excess leukemia from 300 rad of x-rays from normal medical care.** In a case-control study of 138 leukemias in patients of Mayo Clinic and another small clinic for Olmstead County, MN, with accurate x-ray exposure records, no excess leukemia for small doses administered over long time periods from medical care.
- **Fluoroscopy doses below 30 rem (30,000 mrem) suppress breast cancer** in Canadian women with high doses from fluoroscopy for tuberculosis, with lower cancer rates at 15 and 25 rem. Linear extrapolation per BEIR, predicts 900 excess cancers in 1,000,000 women exposed to 15 rem at age 30; contradicting the actual data (highly statistically significant, p=<.01, >16 standard deviations below normal) of 0.66 SMR, ⅓ less than normal breast cancer mortality, reflecting 10,000 fewer breast cancer deaths in 1,000,000 women exposed to 15 rem.

## 4. Internal radium from ingestion and injection

After the discovery of radium and its separation in 1902, its use and its stimulative properties, led to substantial internal body burdens from industrial and medical uses. Bone necrosis was recognized early, and long term effects, especially bone sarcomas and head carcinomas were known in the 1920s. From 1930s human studies, a conservative body burden of 0.1 uCi was set in 1941. Threshold evidence was ignored by BEIR (1972), and a linear assumption imposed on the data.

Dr. Robley Evans, MIT radium program 1932-1970, proved BEIR and other linear no-threshold models scientifically invalid, from 508 MIT cases <1000 rad, and constant 28% cancers in 108 cases at 1000-50,000 rad. US radium cases (before 1950) were consolidated at the Center for Human Radiobiology at Argonne in 1970, eventually >4000 cases, finding no change in these conclusions (1983), the program was defunded and terminated with >1000 cases still alive.

In this population there is:

- **No radiogenic cancer at <1000 rad bone dose in >50 years** Gofman-Tamplin linear model "goodness-of-fit" to MIT data <1/200,000,000, and "full-range" model 1/220,000, with BEIR in between; all data (>4000 cases) "has continued to show no radiogenic tumors, or other effects, in hundreds of persons whose effective initial body burden was less than about 50 uCi of $Ra^{226}$, and whose cumulative skeletal average dose is less than about 1000 rad." These conclusions are again confirmed with updated data another decade later.
- **Log-normal projection to a minimum ~400 rad threshold** (65 tumors in 1545 cases, in 154 at >1000 rad) in the homogeneous group of young women luminizers/dial-painters, demonstrated to be "best-fit" to log-normal distributions.
- **Significantly lower mortality from all causes** in young US and UK white, female dial painters with very high radiation doses. Only breast cancer is minimally elevated, noting large internal body burdens and radiation doses, and work at benches with radium compounds with substantial external exposure to the chest, neck and head areas. Without breast cancer there is no increase in cancer for this high-dose population. Reduced circulatory/cerebrovascular diseases are the most significant effects.
- **Far lower non-cancer mortality in the high-dose group** than the general population, especially for the first 20 years following initial exposure, indicating the need to assess the benefits of radium as a dietary supplement, and/or the effect of a "booster" to continue the documented beneficial effects.
- **Radium cancers have identical dose-response form, latency, and threshold** for people, dogs, and mice.
- **Radithor, 1 uCi $Ra^{226}$ + 1 uCi $Ra^{228}$, 3.5 uCi (3.5 million pCi) $^{226}Ra$ - equivalent.** Eben Byers died from from drinking 3-4 vials/day for 3 years (~10 Billion pCi) vs US drinking water limit of ~2000 pCi/yr, (~2 Million uCi systemic uptake vs 50 uCi / 1000 rad threshold in radium-exposed population).

## 5. Nuclear weapons and facility releases

- **"Atomic veterans" of above-ground tests have no adverse effects in 46,186** US persons exposed to nuclear weapons tests. Operation Smoky, had 3200 participants, 10 leukemia deaths, vs. 3.97 statistically expected, only one >3 rem, no dose effect; Operation Greenhouse, 3000 participants, 1 leukemia death vs. 4.43 expected; both are typical in applying statistics to small numbers.
- **No increased cancers or all causes mortality in 22,347 British participants** in weapons tests and experimental programs in Australia and the Pacific Ocean.
- **No difference in mortality nor trends by dose in 954 Canadian** military personnel involved in clean-up operations after nuclear reactor accidents at Chalk River or observed weapons tests.
- **Utah population downwind of above-ground tests show no adverse effects.** Counties with higher health effects were not the counties with higher radiation doses.

## 6. Natural background

- **Cancer mortality ~15% less than US average at ~3 times US average doses** in 7 US Colorado Plateau states.
- **No effect in stable, equivalent, Chinese populations at 3 times higher doses.** Equivalent, 60,000-70,000 Han peasant populations to 6 generations. Uranium, and radium (plus 14 α and β decay chain radionuclides) are >4 times (~7 ppm vs. ~8 ppm); and thorium (and 17 α and β decay chain radionuclides) is >6 times (~8 ppm vs. ~50 ppm). Area radiation monitoring and personnel dosimetry were conducted for over 5 years. Slightly lower mortality in high dose area for all cancers (including leukemia). Equivalent hereditary diseases and congenital defects except higher Down's syndrome, largely from abnormally low incidence in low dose area vs the region and China, plus significantly high birth rate to women >35 years old in the high dose area (a known association to Down's syndrome).

- **No discernible health effects in Kerala India, 4 times normal** in 12,918 people in Kerala vs a neighboring town with a control population of 5938, with no discernible health differences, except 12 cases of Down's syndrome in the Kerala population, and none in the control population. However, Down's syndrome in India is 1:1215 in 58,325 live births, equivalent to (higher than) the rate in Kerala.
- **Guarapari Brazil, 6 times background, no adverse health effects.** A small population in Guarapari Brazil has 6 times the radiation exposure of other areas. No difference in health effects is demonstrated associated with this very large increase in environmental radioactivity and human radiation doses.
- **No adverse health effects from indoor radon.** Base non-smoker lung cancer is 2-3/100,000. In 1985, males were 75/100,000, and females 27/100,000. In 1930, lung cancer was 4/100,000 male, and 2/100,000 female, with male lung cancer from increased smoking after WWI (with 20 year lag). Lung cancer data contradicts EPA claims that 15% of lung cancer is from radon (20,000 of 140,000 deaths/yr).
- There is no lung cancer in non-smoking uranium miners at <1000 times 70-year indoor radon levels. EPA erroneously predicts 1000-5000 lung cancer deaths/100,000 (1-5 deaths/100) so exposed.
- There are 2.7/100,000 lung cancer deaths in the high dose area in China; and 2.9/100,000 in the low dose area.
- Uranium miners and smokers have different prevalent lung cancer type than in non-smokers. Lung cancer in non-smokers can not be associated with radon projected from radon exposure in uranium miners.
- Soils and rocks have a high variation in radon (to 10 times in Massachusetts, higher elsewhere), with detectable health effects if EPA predictions were valid. Much higher concentrations exist, with no detectable health effects. EPA proposes 10 Bq/l limits, while 100,000s of people seek radon waters in health spas with radon to 12,000 Bq/l, with competent independent medical literature confirming positive long term effects; and workers in these high radon environments with occupational exposure studies that confirm no adverse health effects.
- **Lung cancer by US county has negative correlation with radon.** From 272,000 home radon measurements, in 1601 US counties (covering >90% of the US population, eliminating retirement states) a strong negative correlation of lung cancer with radon is demonstrated (for males and females, with and without a correctionfor smoking), and resolving all potential confounding factors, contrary to and disproving BEIR and EPA projected health effects.
- **Radon Spa areas confirm lower health effects with higher doses** from detailed health effects studies of the Misasa Japan radon spa area and others.

# NON-HUMAN RADIATION EFFECTS DATA

**A century of research on plant and animal species demonstrates positive biological responses to low- to moderate-doses in animal, plant and micro-organism populations.** No adverse health effects have been consistently demonstrated ; with substantial significant evidence, and extensive non-significant indication, of beneficial, hormetic, effects in biological populations and biology.

## 7.1 NON-HUMAN BIOLOGICAL POPULATIONS

- **Mice and guinea pigs exposed to 0.11, 1.1, 4.4, and 8.8 rad/day, show 0.11 and 1.1 rad/day, had normal life spans, litters, health conditions (with longer mean lifespans in the 0.11 rad/day group) over 5 to 6 generations.**
- **Organisms in lead-shielded space, high altitude space, deep mine space, and other experimental radiation-response conditions, with controlled radiation above and below ambient conditions, shows improved health and growth conditions with moderate radiation dose** (putting plants under a 'grow-light').
- **Plant and small animal organisms studies show that radiation plays an essential role to biological life.**
- **Replacement of natural potassium, including $K^{40}$, with $K^{39}$ results in negative health effects in small organisms.** ($K^{40}$ contributes a significant fraction of the natural radiation dose to biological organisms, and is in substantial homeostasis in mammals to maintain an essential effective potassium level, possibly essential to biological functioning).

## 7.2 CELLULAR, MOLECULAR BIOLOGY AND GENETICS

**Work on cancer research and genetics has established that radiation can not initiate cancer as a stochastic, linear, process.** Radiation is not carcinogenic in accordance with current knowledge of multi-stage tumorigenesis. Radiation stimulates DNA repair mechanisms. New research confirms underlying biological mechanisms of radiation stimulation of repair of the high rate of natural DNA damage events (improving repair of ~240,000 events/cell/day, while 1 rad produces 20 damage events/cell), explaining the beneficial effects of low- to moderate-radiation doses on essentially all biological organisms, including humans. Such work is also showing positive effects of low-level radiation in treating and preventing cancer, and treating other immune system related diseases.

## 1.0 Japanese Survivors

Dr. R.C. von Borstel states in his review (1995), "Kondo (1993) presents the case in detail of the apparently beneficial effects of low-level atomic bomb radiation on lifespan, mutation induction, and mortality from most types of cancer for survivors. He points out that a hormesis-like effect may have been induced by the radiation that lasted for 20 years."

### 1.1. Japanese Survivors/Cancer - BEIR V

BEIR V, p. 242, states "In the atomic bomb survivors of the Life Span Study Cohort, a total of 202 deaths from leukemia were recorded for the period from 1950 to 1985, during which there were an estimated 2,185,335 person-years of follow-up. For the combined (Hiroshima and Nagasaki) data, the rate of mortality is signifigantly elevated at 0.4 Gy and above but not at lesser doses (Figure 5-1)."

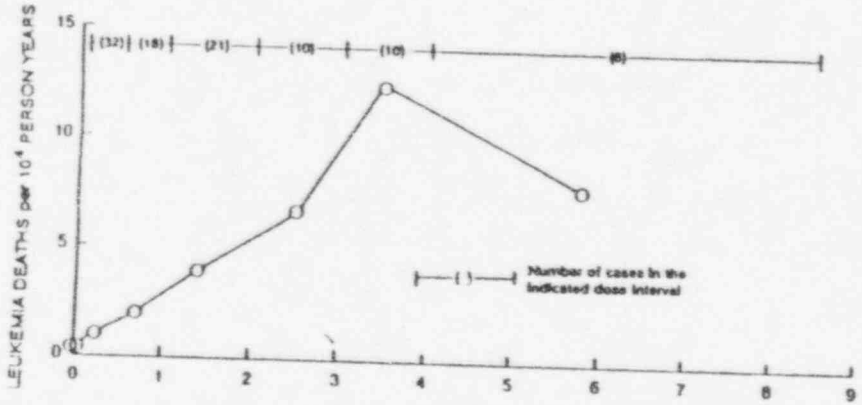


FIGURE 5-1 Cumulative leukemia mortality in Hiroshima and Nagasaki as a function of the estimated dose equivalent to the bone marrow under DS86. By 1985, there were 51 cases in the 0 Sv category and 31 cases in the 0.01-0.1 Sv stratum.

### Japanese Survivors/Cancer - Jaworowski 1995b

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Zbigniew Jaworowski states (1995b) that,

"The UNSCEAR (1994) report states that among survivors from Hiroshima and Nagasaki who received doses of <200 mSv (<200 times higher than the proposed EPA annual limit) there was no increase in the number of total cancer deaths. In fact, mortality caused by leukemia was less in this population at doses <100 mSv than among the nonirradiated inhabitants of these Japanese cities, which is not statistically significant."

### Japanese Survivors/Cancer - Hattori 1994

Sadao Hattori, Vice President of CRIEPI reports (1994) that, "The follow up data of people who received radiation from the Atomic Bomb show us an interesting feature especially in the low dose range. Figs. 1 and 2 show that about 8 cGy, is the optimum dose for the suppression of leukemia through the surveys of the people of Hiroshima and Nagasaki exposed to the radiation of the Atomic Bomb."

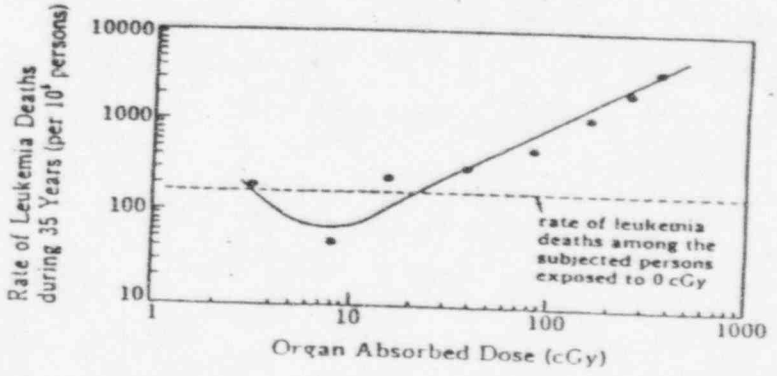


Figure 1 Dose-response relation of leukemia deaths among A bomb survivors

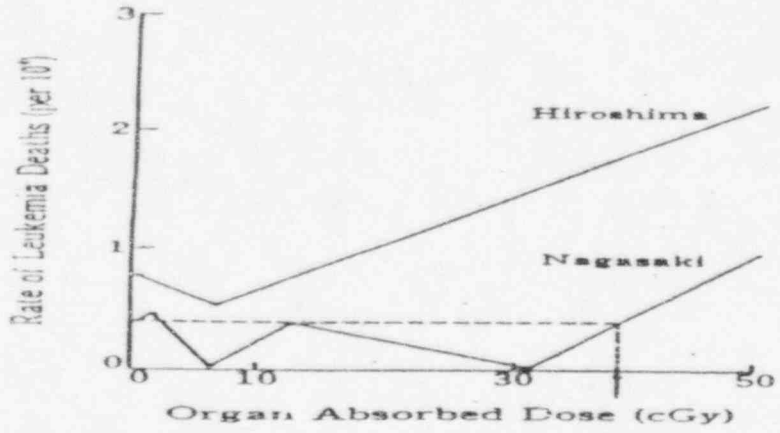


Figure 2 Threshold-like dose ---------- estimated from dose-response relation curve of leukemia deaths among A-bomb survivors.

Table 3.5 Numbers of subjects and cancer deaths, 1950-85, among atomic bomb survivors classified by DS86 dose

| Dose (rad)a | Number of Subjects | Leukemia No. | Leukemia Frequency(%) | All other cancers No. | All other cancers Frequency (%) |
|---|---|---|---|---|---|
| 0 | 34,272 | 58 | 0.17 | 2,443 | 7.13 |
| 1-9 | 23,321 | 38 | 0.16 | 1,655 | 7.10 |
| 100-199 | 1,946 | 23 | 1.2 | 221 | 11.4 |

(constructed from data of Shimizu et al., 1989)

Estimates of excess cancer deaths (%) at low doses of radiation by no-threshold linear extrapolation from data on high doses

| Dose (rad)a | All cancers | Leukemia | Other cancers |
|---|---|---|---|
| 1 | 0.035 | 0.007 | 0.029 |
| 5 | 0.17 | 0.03 | 0.14 |

a Shielded kerma values

## Japanese Survivors/Cancers - Kondo 1993

*Professor Emeritus Dr. Sohei Kondo reports on apparently beneficial effects of low doses of atomic bomb radiation with regard to induction of cancer.*

Professor Emeritus Dr. Sohei Kondo (Kondo 1993, Section 3.2) reports that "When tumor incidence (1950 85) among survivors of the atomic bomb in Hiroshima and Nagasaki is classified by dose (Shimizu et al., 1989: Table 3.5), people who were exposed to 1 9 rad appear to have lower death rates from leukemia and from all other cancers than unexposed people, indicating that radiation at these doses has no harmful effect (Kondo, 1990)."

"The dose response curves for most cancers seen after exposure to atomic bomb radiation at Nagasaki, which consisted mainly of gamma rays, have troughs at the low-dose intervals 1-5, 6-19 and 20-49 rad (Fig. 1-updated). In other words, low doses of gamma rays apparently reduce cancer incidence—an indication of beneficial effects. Whether low doses of radiation really did have beneficial effects on atomic bomb survivors cannot be concluded from the epidemiological data alone, because of the large statistical uncertainty at each trough. With this reservation in mind, apparently beneficial effects are expressed in Table 3.6 in terms of apparent threshold dose Dth (see also footnote a to Table 3.6)."

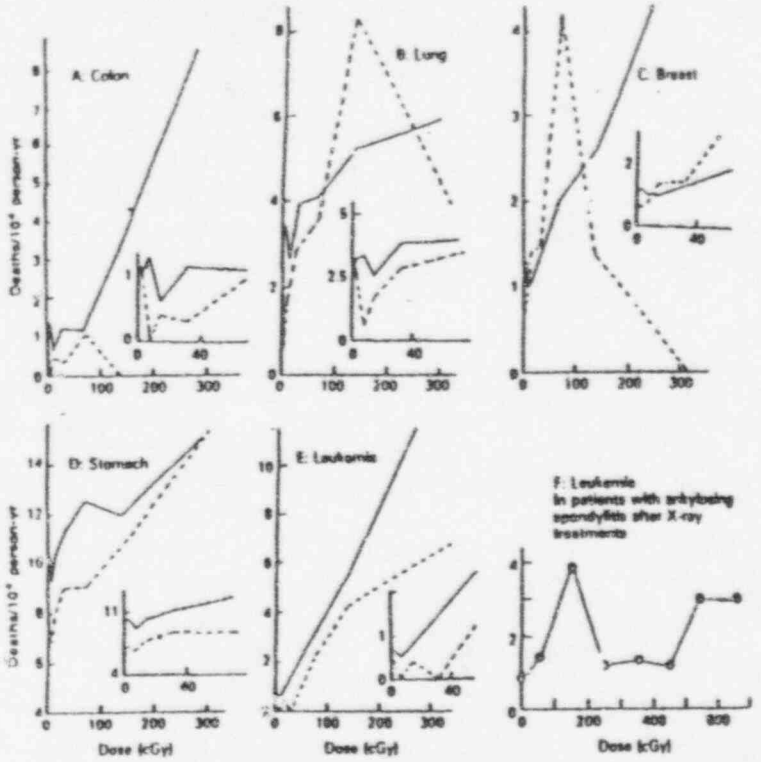


Fig. 1. Dose response curves for death rates from five types of cancer in bomb survivors at Hiroshima (solid line) and Nagasaki (broken line) (panels A through E) (Ref. 3) and from leukemia in ankylosing spondylitis patients treated with X rays (panel F) (Ref. 4).

Table 3.6 Characteristics of dose response relationships for cancer mortality among people exposed to the atomic bombings at Nagasaki and Hiroshima

| Cancer site | Spontaneous rate/ 10^4 person-years | Induced rate at 300 rad/ Apparent threshold 10^4 person-years Dth | (rad)a |
|---|---|---|---|
| Nagasaki | | | |
| Leukemia | 0.4 | 6 | 36 |
| Colon | 0.8 | 0.7 | 54 |
| Stomach | 6.3 | 8.5 | Nonexistent |
| Breast | 1 | (2)b | 50 |
| Lung | 2.6 | (1)b | 28 |
| Hiroshima | | | |
| Leukemia | 0.8 | 12.5 | 12 |
| Colon | 1.2 | 7 | 31 |
| Stomach | 10 | 5 | 12 |
| Breast | 1.0 | 3.5 | Nonexistent |
| Lung | 2.4 | 2.5 | Nonexistent |

Extracted from Shimizu et al. (1987, 1989); see Kondo (1990) for details

## Japanese Survivors/Cancer - Pollycove 1994

Prof Emeritus, Myron Pollycove, MD, reports (Pollycove 1994) that, "A recent article by Shimizu, et al (1992) concerning the effects of low level radiation in atomic bomb survivors concluded that analysis of dose response 'in the less-than-0.5Sv region fails to indicate the presence of hormesis.' They did not observe any significant decrease in the relative risks (RR) of (a) leukemia, (b) all cancers except leukemia, (c) lung cancer, (d) thyroid cancer, or (e) noncancer mortality. This conclusion is in agreement with the data shown for the three cancer groups (b,c,d), but appears inconsistent with the data presented for the RR of the leukemia and noncancer mortality groups.

"The upper half of Figure 11.1 shows the data for these two groups as analyzed by the authors with a variety of models. The discussion of leukemia states that though the RR is less than 1 for the three groups with doses less than 0.1 Sv, since all had p>0.10 they did not differ statistically from unity and thus, were within the range of random variation. In clear contradiction to least square fits, the quadratic model for <0.5 Sv was considered to better fit the data than the linear-quadratic model for <0.5 Sv that demonstrated a RR of 0.78 at 0.11 Sv. The lower half of Figure 11.1 shows analysis of the data with models that provide a better fit. The five data points for leukemia are fitted by an empirical polynomial function. The RR for the 0.010 to 0.019, 0.020 to 0.049, and 0.050 to 0.099 Sv dose categories appear consistently related to one another, not varying randomly. The RR of 0.6 plotted at 0.075 Sv is 1.5 SD less than 1 (p<0.15). This study of atomic bomb survivors is in agreement with the decreased leukemia mortality seen in the nuclear shipyard worker study. In both studies the very low incidence of leukemia makes it difficult to obtain sufficient numbers for high statistical power.

"Desired statistical power is present, however, for mortality rates. In the upper half of Figure 11.1 the RR data for noncancer mortality after low-level radiation are ignored and fitted with a threshold model derived from a prior study of survivors in the <4.0 Sv high-level dose range, assuming the threshold dose is 1.5. Though the mortality RR of 0.83 in the 0.200 to 0.499 Sv dose category is 3.2 SD below 1 (p = 0.001) and is the most statistically significant data point of the entire study, nevertheless, this highly significant decreased RR is rejected with the statement, 'The RRs for the subgroups within the low dose group (<0.5Sv) when compared with the 0-Sv group did not differ and were close to unity...' If the only mathematical models used for analysis are those that a priori exclude a U-shaped dose-response relationship, it is not surprising that such analysis 'fails to indicate the presence of hormesis.' The lower half of Figure 11.1 fits a linear model down to, but no farther than, the noncancer mortality RR of 0.83. This decreased mortality risk associated with acute low-level radiation is consistent with the highly significant (-16SD and -8SD) decreased standardized mortality rates observed in prolonged very low level exposures of the nuclear groups of shipyard workers. (Cameron 1992)."

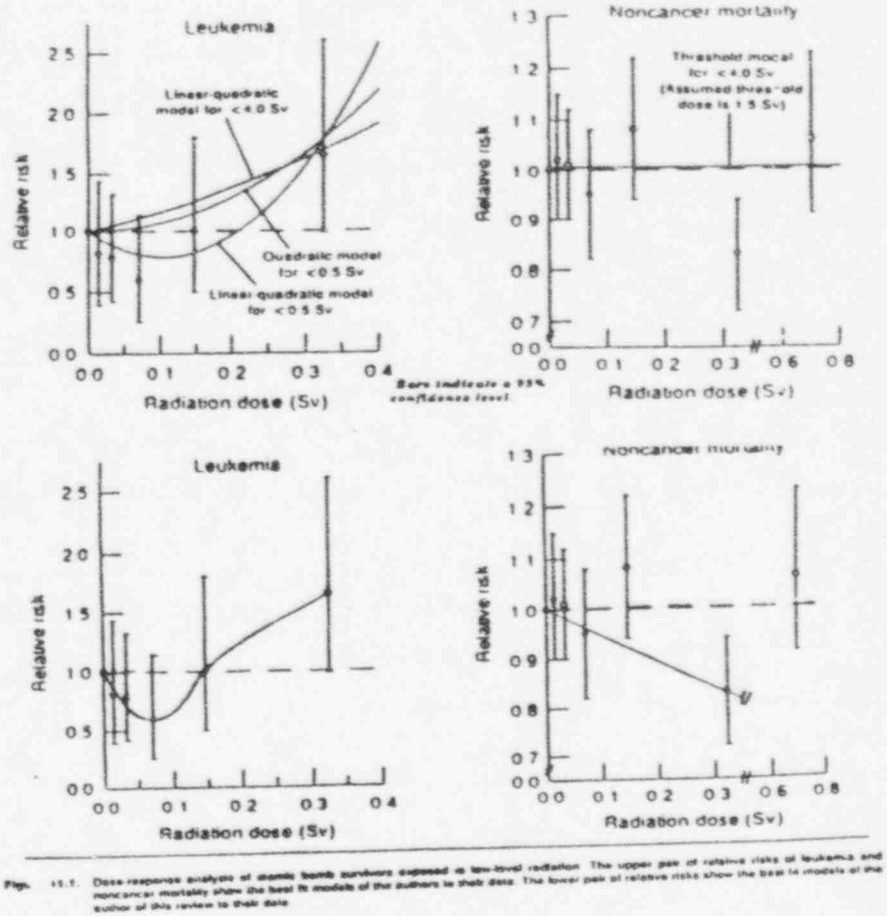


Fig. 11.1. Dose-response analysis of atomic bomb survivors exposed to low-level radiation. The upper pair of relative risks of leukemia and noncancer mortality show the best fit models of the authors to their data. The lower pair of relative risks show the best fit models of the author of this review to their data.

## Japanese Survivors/Cancer - Brodsky 1996

Dr. Alan Brodsky reports (1996) that, "The Shimizu analysis (Shimizu et al. 1988) was indicated to be based on 5,930 total cancer deaths occuring in the years 1950-85 in the 'DS86 sub-cohort'(then denoted as the 75,991 persons for whom dose data was available).

"The summary table of measures of dose-response *for shielded kerma* is shown in Table 2-33. The numbers in parentheses in the columns for estimated relative risk at 1 Gy (100 rad), excess risk per 10,000 person-y Gy, and attributable risk, are estimated 90 % confidence intervals about the indicated respective values. It is apparent that if 95% confidence intervals had been used, only the bladder cases, and possibly the liver cases (which also included cancers of other primary sites), would have been considered significant in providing a trend with increasing dose. This number of significant subcategories could itself have easily appeared by chance for eleven subcategory tests. Since the statistical precision of estimation is so poor, even 1 Gy, it can be seen that estimation of risks from this data at levels below 0.01 Gy (1 rad) based on linear dose-response assumption cannot be supported on the basis of the Japanese data alone.

Table 2-33. Summary measures of radiation dose response for the Japanese survivor population, analyzed by shielded kerma (as presented by Shimizu et al., 1988, Table 2-B)

TABLE 2-B Summary Measures of Radiation Dose Response for Cancer Mortality by Site, for Certain Specific Cancers, Both Cases, Both Sexes, All Ages ATB, 1950-1985 (Shielded Kerma)

| Site of Cancer | No. of deaths | Statistical test (P)* | Estimated RR at 1 Gy | Excess risk per $10^4$ PYGy | Attributable risk (%)* |
|---|---|---|---|---|---|
| Liver including not specified as primary | 590 | 0.02 | 1.34 (1.06, 1.67) | 0.65 (0.17, 1.18) | 7.02 (1.87, 13.2) |
| Kidney | 58 | 0.18 | 1.58 (0.81, 2.94) | 0.09 (-0.03, 0.26) | 15.7 (-3.37, 45.3) |
| Bladder | 90 | 0.003 | 2.13 (1.40, 3.28) | 0.43 (0.16, 0.70) | 23.6 (9.31, 40.8) |
| Tongue | 28 | 0.40 | 0.83 (-, 1.49) | -0.02 (-, 0.06) | -3.35 (-, 14.1) |
| Pharynx | 33 | 0.61 | 0.83 (-, 2.04) | -0.02 (-, 0.09) | -4.14 (-, 31.4) |
| Nose | 44 | 0.18 | 0.84 (-, 1.47) | -0.03 (-, 0.12) | -4.04 (-, 14.5) |
| Larynx | 46 | 0.14 | 1.51 (0.93, 3.68) | 0.10 (-0.01, 0.29) | 13.4 (-1.45, 37.1) |
| Skin cancer except melanoma | 21 | 0.89 | 1.17 (-, 2.47) | 0.02 (-, 0.13) | 5.40 (-, 38.7) |
| Brain | 37 | 0.61 | 1.23 (-, 2.76) | 0.02 (-, 0.16) | 6.36 (-, 42.9) |
| Brain tumors | 43 | 0.97 | 1.03 (0.51, 2.09) | 0.01 (-0.12, 0.20) | 1.0 (-13.0, 22.5) |
| Tumors of central nervous system (CNS) except brain | 14 | 0.08 | 3.09 (1.06, 9.74) | 0.10 (0.00, 0.25) | 33.9 (1.4, 82.2) |

Numbers in parentheses indicate a 90% confidence interval

* Test for trend in the risk for increasing level of radiation dose

Based on 61,168 subjects exposed to 0.01+ Gy, through 8,305 deaths

"In Table 2-34, brain tumors and CNS tumors other than brain are regressed versus dose groupings. Although the relative risks (RR) are indicated to be above one for all positive doses, it should be noted that this is so because of the fitting of the linear model to all of the data. It can be seen that the observed numbers of cases are below the expected numbers for the lowest dose groups, below 0.09 Gy (9 rad, or 9,000 millirad). Also, the expected totals were inappropriately constrained to equal observed totals (see parenthetical totals). This constraint could cause some of the excess of RRs of higher dose ranges, and invalidate the entire analysis.

*Table 2-34. Mortality from CNS tumors by type (from Shimizu et al., 1988, Table 3)*

| | | DS86 dose (shielded kerma, Gy) | | | | |
|---|---|---|---|---|---|---|
| | Total | 0 | 0.01-0.09 | 0.10-0.49 | 0.50+ | Test† |
| | | *Brain Tumors* | | | | |
| Observed | 47 | 16 | 15 | 13 | 3 | |
| Expected | (47) | 21.42 | 13.96 | 7.49 | 4.13 | |
| RR | | 1.00 | 1.44 | 2.32 | 0.97 | p>0.10 |
| | | *CNS Tumors other than brain* | | | | |
| Observed | 14 | 4 | 3 | 4 | 3 | |
| Expected | (14) | 6.12 | 4.27 | 2.32 | 1.29 | |
| RR | | 1.00 | 1.08 | 2.63 | 3.56 | 0.05<p<0.10 |

† Test for increasing trend in radiation dose

"The last two exhibits abstracted here from Shimizu *et al.* (1988) are shown in Tables 2-36 and 2-37. These tables show the variations of all malignant neoplasms, and leukemia only, versus dose groupings in shielded kerma. For the total time period, and for doses in the range less than about 0.5 Gy (50 rad) it can be seen that there is no statistically significant increase of total cancer in each table, for the exposed groups compared to controls. In fact, the actual numbers of cases of cancer and leukemia are below the expected numbers in the dose groups below 0.06 Gy (6 rad) for all cancers, and below 0.2 Gy (20 rad), for Tables 2-36 and 2-37, respectively.

*Table 2-36. All malignant neoplasms versus dose group for various time periods of RERF study (from Shimizu et al., 1988, Table 2-2)*

ALL MALIGNANT NEOPLASM: HIROSHIMA - NAGASAKI - MALES - FEMALES, ALL AGES ATB

| Follow-up Interval | Total Deaths | SHIELDED KERMA DOSE IN GRAY | | | | | | | | | | Excess RR per Gray Std Error (P-Value) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0 | .01-.05 | .06-.09 | .10-.19 | .20-.49 | .50-.99 | 1.0-1.99 | 2.0-2.99 | 3.0-3.99 | 4.0+ | |
| 1950-85 OBS | 5936 | 2501 | 1358 | 335 | 427 | 558 | 349 | 244 | 93 | 32 | 37 | 0.3895 |
| TOTAL EXP | | 2680.68 | 1414.57 | 335.03 | 429.44 | 538.88 | 300.81 | 161.37 | 44.90 | 13.34 | 17.69 | 0.0478 |
| OBS REL RISK | | 1.00 | 1.03 | 1.07 | 1.07 | 1.11 | 1.24 | 1.62 | 2.26 | 2.57 | 2.24 | (0.0000) |
| FITTED REL RISK | | 1.00 | 1.01 | 1.03 | 1.05 | 1.12 | 1.26 | 1.52 | 1.93 | 2.26 | 2.97 | |

*Table 2-37. Leukemias vs. dose group for various time periods of RERF study (from Shimizu et al., 1988, Table 2-3)*

2-3 LEUKEMIA: HIROSHIMA - NAGASAKI - MALES - FEMALES, ALL AGES ATB

| Follow-up Interval | Total Deaths | Shielded Kerma Dose in Gray | | | | | | | | | | Excess RR per Gray Std Error (P-Value) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 0 | .01-.05 | .06-.09 | .10-.19 | .20-.49 | .50-.99 | 1.0-1.99 | 2.0-2.99 | 3.0-3.99 | 4.0+ | |
| 1950-85 OBS | 202 | 58 | 36 | 2 | 13 | 21 | 19 | 23 | 15 | 9 | 6 | 3.921 |
| TOTAL EXP | | 87.53 | 49.05 | 12.19 | 15.16 | 19.20 | 10.34 | 5.67 | 1.67 | 0.56 | 0.63 | 0.7764 |
| OBS REL RISK | | 1.00 | 1.11 | 0.25 | 1.29 | 1.65 | 2.77 | 6.12 | 13.53 | 24.40 | 14.31 | (0.0000) |
| FITTED REL RISK | | 1.00 | 1.08 | 1.27 | 1.55 | 2.22 | 3.67 | 6.25 | 10.33 | 13.67 | 20.80 | |

"Moreover, if the observed and expected numbers of cases are totalled horizontally for all dose groups, then in each table, the expected numbers of cases is exactly equal to the given observed numbers, 5,936 for all cancers and 202 cases for leukemias. As in Table 2-34, the total of expected numbers in each dose group were improperly normalized to the total observed numbers of cancer deaths. Expected numbers should be calculated independently for each population dose group from natural cancer data; i.e., by using the age and sex distributions, and natural risks, of the control populations to calculate the expected cases of each disease category for each dose grouping in the exposed populations. Thus, the total excess number of cases cannot be obtained from this data. Worse yet, the indicated excess cancers and higher relative risks at the higher dose groups can be, at least in part, attributed to the fact that, when normalizing the total sizes of cancer deaths to be equal between observed and expected groups, the numbers of expected cases in each of the higher dose groupings would be constrained to be smaller for the expected cases to make up for the higher expected number in the lower dose goupings of expected cases. This type of tabular presentation of the basic Hiroshima/Nagasaki data is grossly misleading in showing the higher relative risks for the higher dose groups.

"Ron *et al.*, (1994, 1995a, 1995b) have carried out a further analysis of the RERF data, and have again found no increase in cancer at doses less than 0.2 Gy (20 rad). They also have evidently computed expected cases in an appropriate manner; they did not fix the total number of expected cases to equal the total number of observed cases within each group comparison, as in Shimizu, *et al.* 1988."

## Japanese Survivors/Cancer - Luckey 1994

Professor Emeritus Dr. Don Luckey reports (Luckey 1994) that "Japanese survivors of atomic-bomb explosions at Hiroshima and Nagasaki provide the best available data for cancer mortality rates following acute exposures in humans (Shimizu et al., 1992). When dissociated from cancer and blood diseases, the death rates of exposed and control groups were comparable.

"When exposed to 1-1.9 cSv, the total cancer mortality rate of surivors of atomi-bombs in Hiroshima and Nagasaki appeared to be lower than that of the control group, estimated to be 0.2 cSv (Figure 4) (Shimizu et al., 1992). The difference was not statistically significant. However, if ten times more people had been involved, a)

The total cancer mortality rate of the 74,000 survivors exposed to less than 2 cSv would be lower than that of the 450,000 controls, $p < 0.001$; b) the total cancer mortality rate of 179,000 survivors exposed to 1-4.9 cSv would be the same as the controls, and c) only when those exposed to 1-50 cSv were considered would the exposed population have a higher total cancer mortality rate than controls, $p < 0.01$. The data of Figure 4 suggest that the ZEP for total cancer mortality from acute exposures was about 3 cSv. This defines an acute dose for tiage considerations in nuclear disasters.

"The leukemia mortality rate of Japanese bomb survivors who received less than 50 cSv was not statistically different from that of controls (Figure 5) (Shimizu et al., 1992). However, if ten times more people had been exposed, the decreased leukemia mortality rates for survivors at doses less than 20 cSv would be lower than controls, $p = 0.06$ to 0.01. For acute eposures the ZEP for leukemia mortality was about 25 cSv.

"The decreased leukemia and total cancer mortality rates observed in Japanese bomb survivors were echoed by the cancer and leukemia mortality rates of 46,425 United States army observers of atmospheric nuclear explosions (Figure 6) (Robinette et al., 1985). Note that the lowest dose of Shimizu et al., 1.0-1.9 cSv, is near the highest dose noted for army observers, 2.5 cSv. These results were comparable with those found in 22,325 British observers and a similar number of Canadian observers (Luckey, 1991).

"Cumulative lung cancer mortality rates for Japanese atomic-bomb survivors exposed to less than 20 cSv was not greater than that of controls (Figure 7) (Shimizu et al., 1992). If ten times more people had been involved, there would be no difference for those receiving less than 2 cSv; persons exposed to greater than 2 cSv would have a higher rate than controls, $p<0.001$. The ZEP for lung cancer mortality vas about 1.4 cSv.

"Stomach cancer, endemic in Japanese people, was not increased by low-dose irradiation (Figure 8) (Shimizu et al., 1992). Cumulative stomach cancer mortality ates of exposed and control persons vere comparable from 1 to 99 cSv, p = NS. If the population were ten times larger, exposure to 1-1.9 cSv would have resulted in a lower cancer mortality rate than the controls, $p=0.001$. The 280,000 who received less than 20 cSv would show no increased stomach cancer rate.

"A summary of the Japanese data (Table 1) shows the effects of acute, whole-body exposure to low-dose irradiation. Exposures to greater than 2 cSv reduce cancer mortality rates. Excepting lung cancer, cancer mortality in persons exposed to 1-4.9 cSv is negligible."

### 1.2 Japanese Survivors/Non-cancer Mortality and Morbidity - Kondo 1994

Professor Emeritus Dr. Sohei Kondo reports (1994) that, " slight but insignifigant decreases in noncancer deaths in bomb survivors exposed to 6 to 19, 20 to 49, 50 to 99, and 100 to 199 cGy occurred as early as 1950-1955; these seemingly beneficial effects of radiation were greater in men than in woman (Table 1)."

TABLE I

Relative Risk for Noncancer Mortality in Bomb Survivors[1]

| | Number of Deaths | Relative Risk at Doses (cGy) [a] | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | 1-5 | 6-9 | 10-19 | 20-49 | 50-99 | 100-199 | 200-299 | 300-399 | ≥ 400 |
| Total | 20,777 | 1.03 | 0.97 | 0.97 | 0.97 | 1.15 | 0.96 | 1.13 | 1.18 | 1.38 |
| Sex | | | | | | | | | | |
| Males | 9,344 | 1.05 | 0.94 | 0.95 | 0.96 | 0.98 | 0.95 | 1.05 | 1.17 | 1.80 |
| Females | 11,433 | 1.05 | 0.99 | 0.99 | 0.97 | 1.04 | 0.97 | 1.21 | 1.13 | 1.07 |
| Period | | | | | | | | | | |
| 1950-55 | 2,901 | 1.07 | 0.89 | 0.91 | 0.92 | 0.99 | 0.74 | 0.97 | 1.49 | 1.46 |
| 1956-60 | 2,899 | 0.87 | 0.83 | 0.9. | 1.08 | 0.71 | 0.94 | 0.88 | 1.08 | 1.10 |
| 1961-65 | 2,969 | 1.12 | 0.98 | 1.01 | 1.00 | 1.20 | 0.87 | 1.07 | 1.34 | 1.82 |
| 1966-70 | 2,958 | 1.03 | 0.88 | 0.87 | 1.04 | 0.94 | 1.18 | 1.23 | 0.98 | 1.21 |
| 1971-75 | 2,988 | 0.97 | 0.95 | 0.95 | 0.92 | 1.16 | 1.02 | 1.13 | 1.04 | 1.45 |
| 1976-80 | 3,057 | 0.94 | 1.01 | 1.08 | 0.82 | 0.96 | 0.98 | 1.34 | 1.00 | 1.34 |
| 1981-85 | 2,905 | 1.11 | 1.11 | 0.84 | 1.05 | 1.17 | 0.97 | 1.30 | 1.27 | 1.89 |

### 1.3 Japanese Survivors/Health Effects on the Unborn Fetus - Kondo 1994

Professor Emeritus Dr. Sohei Kondo reports (1994) in Section 3 of the report:

"*birth defects*: children with small head size, mental retardation, and reduc-tion in IQ scores and school performance were born to pregnant mothers exposed to high doses; there were threshold doses of ~50 and 10 cGy, respectively, for severe mental retardation and reduction of IQ scores and school performances.

*"Small Head Size*

"The frequency of small head size among children exposed prenatally to atomic bomb radiation in Hiroshima increased with doses above 10-19 rad, whereas in Nagasaki an increased prevalence of small head size was found only among children exposed *in utero* to more than 150 rad of atomic bomb radiation. It should be noted that when exposure occurred later than 17 weeks after fertilization, the incidence of small head size was low even after high doses of radiation.

*"Mental Retardation*

"The frequency of severe mental retardation after prenatal exposure to atomic bomb radiation at Hiroshima increased significantly with increasing doses above 50-99 rad, whereas in Nagasaki severe mental retardation occurred only at doses over 300 rad.

"When the IQ scores and school performances of prenatally exposed survivors of the Hiroshima atomic bomb were compared with those of suitable comparison groups, the period 8-15 weeks after fertilization was again the period of greatest vulnerability to mental injury after exposure to bomb radiation. The threshold dose for a reduction in either IQ or school performance, after exposure to radiation 8-15 weeks after fertilization, was estimated to be ≥ 10 rad."

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**Japanese Survivors/Health Effects of the Unborn Fetus - Jaworowski - 1995b**

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that,

"Part of the information on (unexpected) positive genetic effects of ionizing doses of radiation comes from Hiroshima and Nagasaki...(where data) shows that acute irradiation with moderate doses does not produce any major negative effect on the health of the following generation.

"What can be demonstrated, on the other hand, are the positive effects. Among the children of parents who survived the atomic bombings in Hiroshima and Nagasaki there were about 4% fewer deaths between 1946 and 1958 than among the children of parents unexposed to radiation from atomic bombs, 28% less aneuploidy, 29% fewer chromosomal aberrations, and 30% fewer mutations in blood proteins."

**1.4 Japanese Survivors/Genetic Effects - Kondo 1993**

*Genetic effects*

Professor Emeritus Dr. Sohei Kondo reports (Kondo 1993, Section 3.4) that "the indicators of genetic effects in the children of survivors that have so far been studied are: congenital defects, stillbirth, death among liveborn children through 1985, sex ratio, growth and development during childhood, sex chromosomal aneuploidy and reciprocal translocations of chromosomes, cancer occurrence prior to the age of 20, and mutations affecting the character of erythrocyte and serum proteins (Neel et al., 1990). The essential results of the 40 year follow-up studies are summarized in Table 3.9."

Table 3.9 Genetic effects of radiation in children of atomic bomb survivors in Hiroshima and Nagasaki

| Indicator | Frequency (no.abnormal/no. studied) | | Parental dose^a (rem) | References |
|---|---|---|---|---|
| | Control | Exposed | | |
| Untoward pregnancy outcome b | 4.99% (2,257/45,234) | 5.00% (503/10,069) | 36 | Unto Otake et al. (1990) |
| Deaths of liveborn children c | 7.35% (2,451/33,361) | 7.08% (989/13,969) | 40 | Yoshimoto et al. (1991) |
| Stable chromosomal aberrations | 0.31% (25/7,976) | 0.22% (18/8,322) | 60 | Awa et al. (1989) |
| Aneuploidy | 0.30% (24/7,976) | 0.23% (19/8,322) | 60 | Awa et al. (1989) |
| Mutations in blood proteins | 6.4 x 10-4 (3/4.7 x 10[illegible]) | 4.5 x 10 -6 (3/6.7 x 10[illegible]) | 41 | Neel et al. (1988) |
| Leukemia | 0.05% (21/41,069) | 0.05% (16/31,159) | 43 | Yoshimoto et al. (1991) |

a Sum of average doses to mothers and to fathers
b Congenital malformations, stillbirths and deaths in the first 14 days of life
c Birth years from 1946 to 1958

"As seen in Table 3.9, after the long-range project, carried out over nearly half a century, Neel and his coworkers (1988, 1990) found no statistically significant effect of parental exposure to radiation on any of the indicators."

"Tumor incidence during the first 20 years of life among 31,150 children of atomic bomb survivors exposed to an average total gonadal dose of 43 rem was compared with that among 41,066 controls. The incidence of all malignant tumors was 0.14% (43/31,150) for the offspring of the exposed people and 0.12% (49/41,066) for controls; the incidences of leukemia plus malignant lymphoma were 0.05% (16/31,150) for the offspring of the exposed people and 0.05% (21/41,066) for controls. There was thus no significant difference in tumor incidence between the two groups (Yoshimoto et al., 1991)."

Dr. Kondo states: "I like the simple (and at the same time somewhat sophisticated) statement of Neel et al. (1990):

'The children of the most highly irradiated population in the world's history provide no statistically significant evidence that mutations were produced in their parents. Absence of statistically significant findings does not deny the possibility that exposed survivors sustained an increased mutation rate undetected by the method employed. On a more positive note, these studies have produced an extensive body of data against which to evaluate empirically both past and future surmises concerning the genetic consequences of exposure to ionizing radiation. In particular, the studies should prove reassuring to that considerable group of exposed Japanese and their children, without whose magnificent cooperation these studies would have been impossible and who have over the years been subjected to a barrage of exaggerations concerning the genetic risks involved.'"

### 1.5 Japanese Survivors/Longevity - Hattori 1994

Japanese Survivors/Longevity Sadao Hattori, Vice President of Research at CRIEPI, states "The exposed groups are showing longer lives through the comparison of the death rate of each age between exposed group and non-exposed group (Fig. 2.7)."

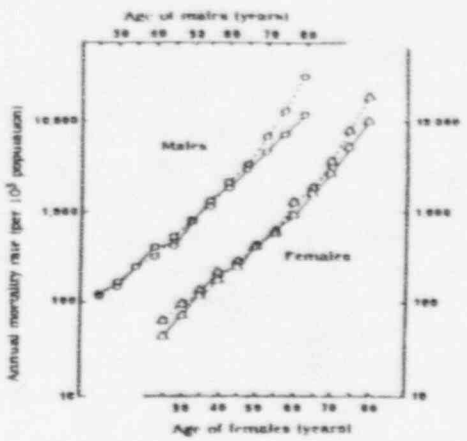


**Fig. 2.7 Comparison of age-dependent rates of mortality (1970-76) for atomic bomb survivors (solid lines) and controls (broken lines) in Nagasaki**
Mortality rates are averages for 1970-76 at five-year intervals; e.g., the rate at age 30 is the average for ages 30-34 years [constructed from data of Mine et al., 1981]

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Japanese Survivors/Longevity - Kondo 1993

*Professor Emeritus Dr.. Sohei Kondo reports on the finding of "apparently beneficial effects of atomic bomb radiation on life span".*

Professor Emeritus Dr.. Sohei Kondo reports (Kondo 1993, Section 3.1) reports, "A total of 7782 deaths that occurred during 1970-76 among the bomb survivors registered in Nagasaki City were analyzed as seen in Table 3.1."

Table 3.1 Observed and expected annual rates of deaths (1970-76) from all causes among atomic bomb survivors in Nagasaki

| Age range (years) | Observed deaths (O) | | Expected deaths (E) | O/E |
|---|---|---|---|---|
| | No. | Rate (per 10^5) | | |
| Men | | | | |
| 25-29 | 26 | 143 | 23 | 1.15 |
| 30-34 | 69 | 201 | 49 | 1.42* |
| 35-39 | 84 | 267 | 77 | 1.09 |
| 40-44 | 149 | 436 | 112 | 1.33** |
| 45-49 | 113 | 456 | 129 | 0.87 |
| 50-54 | 87 | 770 | 90 | 0.97 |
| 55-59 | 184 | 1,327 | 164 | 1.12 |
| 60-64 | 299 | 1,896 | 346 | 0.87* |
| 65-69 | 508 | 3,004 | 578 | 0.88* |
| 70-74 | 816 | 5,006 | 1,140 | 0.72** |
| 75-79 | 825 | 7,796 | 1,416 | 0.58** |
| >80 | 869 | 12,677 | 2,264 | 0.38** |
| Women | | | | |
| 25-29 | 9 | 50 | 11 | 0.80 |
| 30-34 | 26 | 75 | 34 | 0.77 |
| 35-39 | 50 | 146 | 39 | 1.27 |
| 40-44 | 87 | 219 | 72 | 1.20 |
| 45-49 | 119 | 259 | 130 | 0.92 |
| 50-54 | 166 | 430 | 164 | 1.01 |
| 55-59 | 193 | 644 | 185 | 1.04 |
| 60-64 | 276 | 946 | 385 | 0.72** |
| 65-69 | 416 | 1,614 | 482 | 0.86* |
| 70-74 | 591 | 2,800 | 806 | 0.73** |
| 75-79 | 753 | 5,307 | 1,137 | 0.66** |
| >80 | 1,067 | 10,202 | 2,057 | 0.52** |

>From Mine et al (1981) *,p<0.05; **,p<0.01

"The age-specific rates of death from all causes (observed deaths) in people over 60 years of age were significantly lower than those for people without the health handbook (expected deaths) presumed to be unexposed (see also Fig. 2.7). The age-specific death rates for all malignant cancers were, however, not significantly different between the two groups (probably because of the small size of the samples) (Mine et al., 1981).

"The unexpected finding of a lower death rate in the exposed people was interpreted by Mine et al. (1981) as a 'healthy survivor' effect (Section 2.3.2). To exclude any such effect, Mine et al. (1990) compared mortality rates among subgroups of health handbook holders classified by dose of exposure to bomb radiation. Since 1970, data on 100,000 atomic bomb survivors with the health handbook have been maintained at the Scientific Data Center for the Atomic Bomb Disaster at Nagasaki University School of Medicine. Information was selected from this data base on 3,456 people who had been exposed to known doses, and mortality during 1970-88 in this selected group (observed) was compared with that of an age-matched control group (expected) who were given the health handbook but lived far from the hypocenter of the Nagasaki bombing (see footnote b to Table 3.2).

"The ratio of observed: expected numbers of deaths show that the mortality of exposed people was slightly lower than or equal to that of unexposed people at all four low to intermediate doses, 1-49, 50-99, 100-149 and 150-199 rad, and that a significant increase in deaths occurred only in the high dose range, 200-599 rad (Table 3.2).

"The apparent absence of harmful effects of low doses of radiation was analyzed by determining the observed:expected numbers of people classified according to cause of death, sex and dose. As shown in Figure 3.1 and Table 3.3, doses of 50-99 rad significantly reduced the number of deaths from all causes except cancer, to 65% of the control value. On the other hand, the number of deaths from cancer increased at all dose levels except 1-49 rad, although the increase was not statistically significant. Thus, low doses of radiation had two opposite effects--beneficial and harmful--on the human life span in Nagasaki after the atomic bombing."

Table 3.2 Initial numbers of subjects (1970), observed (O) and expected (E) numbers of deaths from all causes and relative risk during 1970-88 among atomic bomb survivors in Nagasaki classified by dose and sex

| T65D a dose (rad) | Initial no. of subjects | | | | Total deaths b | | Relative risk (O:E) | |
|---|---|---|---|---|---|---|---|---|
| | | | | | Observed | Expected | | |
| | M | F | M | F | M | F | M | F |
| 1-49 | 562 | 938 | 162 | 202 | 160.7 | 209 | 1.01 | 0.97 |
| 50-99 | 182 | 168 | 56 | 39 | 63.3 | 34.7 | 0.88 | 1.12 |
| 100-149 | 108 | 158 | 36 | 39 | 39.7 | 34.7 | 0.91 | 1.12 |
| 150-199 | 196 | 267 | 59 | 48 | 58.7 | 48 | 1.01 | 1.00 |
| 200-599 | 440 | 437 | 172 | 79 | 149.7 | 59.3 | 1.15 | 1.33 |

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a T65D, tentative dosimetry proposed in 1965 (see Section 3.5)

b The exposed group, shown in columns 2-5, consisted of health handbook holders who were exposed to the indicated dose, still alive in 1970 and did not move out of Nagasaki City before 1988. The control group (zero exposure) consisted of handbook holders who were > 3 km from the hypocenter at the time of the bombing. Each group was divided into 10 subgroups by dose and sex, and each exposed subgroup was matched with three control groups of the same age and sex.

c Expected numbers estimated from deaths in age-matched controls divided by 3.

Table 3.3 Observed (O) and expected (E)a numbers of deaths during 1970-88 in Nagasaki among atomic bomb survivors classified by cause of death, sex and dose Table3.3 Observed (O) and expected (E)a numbers of deaths during 1970-88 in Nagasaki among atomic bomb survivors classified by cause of death, sex and dose

| Dose (rad) | Non-cancerous diseases b | | Cancer | |
|---|---|---|---|---|
| | O | O:E | O | O:E |
| Men | | | | |
| 1-49 | 126 | 1.09 | 35 | 0.84 |
| 50-99 | 30 | 0.65* | 26 | 1.56 |
| 100-149 | 23 | 0.77 | 13 | 1.34 |
| 150-199 | 38 | 0.84 | 21 | 1.58 |
| 200-599 | 113 | 1.07 | 54 | 1.32 |
| Women | | | | |
| 1-49 | 144 | 0.89 | 56 | 1.24 |
| 50-99 | 30 | 1.11 | 8 | 1.10 |
| 100-149 | 26 | 0.96 | 13 | 1.86 |
| 150-199 | 31 | 0.84 | 16 | 1.60 |
| 200-599 | 50 | 1.11 | 28 | 2.11** |

(Mine et al., 1990. Copyrights Taylor & Francis Ltd, London. Reproduced with permission)
a Expected numbers estimated from deaths among age-matched, unexposed groups (see footnotes to Table 3. 2 for details)
b Excluding accidents, violence and other external causes *, p <0.05 (X^2 test; **, p <0.01 (X^2 test)

"If we were to take the observed expected ratios for mortality from all causes given in Table 3.2 at face value, we would be forced to conclude that whole-body irradiation with 50-150 rad (actually 40-110 rad after correction for the systematic error in the T65D dosimetry had a beneficial effect on the survival only of men--a decrease in overall mortality of about 10%. This level of radiation, however, caused about a 40% (nonsignificant) increase in deaths from cancer in men (Table and higher levels had harmful effects on both men and women.

"The slight but nonsignificant decrease in mortality among bomb survivors exposed to low and intermediate doses (6-19, 20-49, 50-99 and 100-199 rad) of radiation was seen as early as 1950-55 (Table 3.4). Table 3.4 is taken from a recent report by Shimizu et al. (1992), which is based on the follow-up studies that have been conducted since 1950 by the Atomic Bomb Casualty Commission (1950-74) and the Radiation Effects Research Foundation (RERF) (1975 ) on a fixed cohort of about

75,000 atomic bomb survivors in Hiroshima and Nagasaki and 35,000 suitable controls (referred to in this book as the RERF population). It is interesting to note from the Table that the seemingly beneficial effects of low to intermediate doses of radiation are larger in men than in women. This finding is in agreement with the conclusion of Mine et al. (1990), and reflects the experimental finding that daily whole-body irradiation of mice at 0.1 rad significantly increased the mean survival time of males, despite significant increases in the incidence of cancers, from 684 +-14 days in controls to 783 +-14 days after irradiation, whereas females irradiated in the same way showed a slight but insignificant increase in mean survival from 803 +-16 days in controls to 820 +-18 days after irradiation (Lorenz et al., 1955). Thus, the males in this experiment appeared to be more sensitive to the 'beneficial' effects of low-level radiation than females."

Table 3.4 Relative risk for mortality from all diseases except neoplasms and hematological conditions among atomic bomb survivors, 1950-85 by sex, age at the time of the bombings and period of follow-up

| Number of deaths | Relative risk at doses (rad in DS86) of | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1-5 | 6-9 | 10-19 | 20-49 | 50-99 | 100-199 | 200-299 | 300-399 | >400 |
| Total 20,777 | 1.03 | 0.97 | 0.97 | 0.97 | 1.15 | 0.96 | 1.13 | 1.16 | 1.38 |
| Sex | | | | | | | | | |
| Males 9,344 | 1.03 | 0.94 | 0.95 | 0.96 | 0.98 | 0.95 | 1.05 | 1.17 | 1.60 |
| Female 11,433 | 1.03 | 0.99 | 0.99 | 0.97 | 1.04 | 0.97 | 1.21 | 1.13 | 1.07 |
| Age (yrs) at time of bombings | | | | | | | | | |
| <10 324 | 0.80 | 0.78 | 1.20 | 1.25 | 0.75 | 0.46 | 1.46 | 1.29 | 1.35 |
| 10-19 814 | 1.06 | 1.03 | 0.73 | 0.94 | 1.07 | 1.7 | .62 | 0.81 | 2.21 |
| 20-29 929 | 0.97 | 0.80 | 0.92 | 0.98 | 0.74 | 1.07 | 0.94 | 0.72 | 2.03 |
| 30-39 2,441 | 1.12 | 1.09 | 1.05 | 0.84 | 1.26 | .94 | 1.87 | 1.47 | 1.81 |
| 40-49 5,995 | 1.07 | 0.97 | 0.99 | 1.05 | 1.07 | 1.07 | 1.22 | 0.95 | 1.28 |
| >50 10,274 | 1.00 | 0.96 | 0.95 | 0.95 | 0.96 | 0.89 | 0.95 | 1.45 | 1.10 |
| Period | | | | | | | | | |
| 1950-55 2,901 | 1.07 | 0.95 | 0.91 | 0.92 | 0.99 | 0.74 | 0.97 | 1.49 | 1.46 |
| 1956-60 2,999 | 0.97 | 0.93 | 0.93 | 1.06 | 0.71 | 0.94 | 0.88 | 1.08 | 1.10 |
| 1961-65 2,969 | 1.12 | 0.96 | 1.01 | 1.00 | 1.20 | 0.87 | 1.07 | 1.24 | 1.52 |
| 1966-70 2,958 | 1.03 | 0.86 | 0.97 | 1.04 | 0.94 | 1.18 | 1.23 | 0.96 | 1.21 |
| 1971-75 2,988 | 0.97 | 0.95 | 0.95 | 0.92 | 1.16 | 1.02 | 1.13 | 1.04 | 1.45 |
| 1976-80 3,057 | 0.94 | 1.01 | 1.05 | 0.82 | 0.96 | 0.98 | 1.34 | 1.00 | 1.34 |
| 1981-85 2,905 | 1.11 | 1.11 | 0.94 | 1.03 | 1.17 | 0.97 | 1.30 | 1.27 | 1.59 |

(from Shimizu et al., 1992. Copyright Academic Press, Orlando. Reproduced with permission)

"Between two and 18 months after the atomic bombing, Nakashima and 23 coworkers at Kyushu University School of Medicine made a follow-up study of peripheral leukocyte counts in 280 residents of Nishiyama, located 3 km from the site of the atomic bomb explosion in Nagasaki. The majority of the residents had a prolonged increase in leukocyte count throughout the period of measurement, but did not have overt infectious disease (Nakashima et al., 1953). This population had not been exposed to the atomic bomb radiation, as Nishiyama was shielded by a mountain (Mt. Konpira); however, they were exposed to radioactive fall-out, at a cumulative dose of about 20 rad, from external gamma rays and to an unknown dose from continued exposure to radiation by ingestion of radioactive materials. The percentages of males and females who had leukocyte counts over 30,000 at least once were 19 and 15% aged 1-10 years, 29 and 16% aged 11-20 years, 33 and 7% aged 21-50 years and 35 and 6% aged 51-73 years. The mean leukocyte counts of adult males (aged 21-50 and >50 years), but not young and adult females and young males, showed a sharp, high maximum during the 5-8 month period after exposure.

"These fragmentary data indicate that males may be more sensitive to stimulation by low-level radiation than females. If this sexually different response to low-level radiation was real, it might have reflected a sexual difference in the characteristics of homeostasis in that after exposure to moderate external stress (including irradiation), males responded more sensitively to up- or down-regulation of homeostasis than females.

"Stewart and Kneale (1984, 1988) originally noted the U-shaped dose response relation for non-cancer deaths in the RERF population. They proposed a selection hypothesis, as follows: 'The dose response curve has oppositely directed slopes at high and low dose levels as a result of survivors with high and low doses having different reactions to, say, infections--selection effects of early infection deaths and residual effects of marrow damage. Low dose survivors were at high risk of an infection death during the fall and winter of 1945 and for an unknown period thereafter. This general hazard was dose related and obviously greater for people with low than high levels of immunological competence. On the assumption that it took at least two years for living conditions in the two cities to revert to normal, we

"This significant difference in gender response to low and intermediate acute doses of radiation parallels the observations of Lorenz et al (1955) and Congdon (1987) regarding comparison of the survival of male and female mice exposed to 0.0011 Gy delivered in 8 hours daily from age 2 months to death. The longevity of irradiated male mice was significantly increased to 115% of irradiated controls (783 days vs 683 days). However, the longevity of female mice did not increase significantly above their control level of 803 days that was nearly matched by the

extended lifespan of the irradiated male mice. Human populations also demonstrate that female longevity is greater than that of the male. These results suggest that low level irradiation of men and mice may stimulate a physiologic process in the male, relatively unenhanced in the female, that enables male longevity to approximate that of the female."

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Japanese Survivors/Longevity - Pollycove 1994

Prof Emeritus, Myron Pollycove, MD, reports (Pollycove 1994) that, "The (reported) decreased mortality risk reported by the US-Japan Radiation Effects Research Foundation (RERF) study of Hiroshima and Nagasaki (Shimizu 1992) is also consistent with the recent article on Nagasaki survivors from Nagasaki University and the Atomic Energy Research Institute, Kinki University, Japan. Mine et al. report (1990) upon the 'apparently beneficial effect of low to intermediate doses of A-bomb radiation on human lifespan'. The decreased RR of noncancer male deaths to 0.65 ($p<0.05$) in the 0.50-0.99Gy dose range was to a large extent offset by the RR increase to 1.56 in cancer deaths (Table 11.2B). The male RR for total deaths in this dose range was 0.88 (Table 11.2A), with low statistical power ($p=0.34$). Fitting of a U-shaped dose-response relationship confirmed the significantly lower male RR for noncancerous diseases with maximum reduction to 0.76 ($p<0.02$) in the 1.00 to 1.49, average 1.08, Gy dose range (Table 11.2C). Female survivors, on the other hand, showed no significant change in RR of death from all causes until the 2.00 to 5.99 Gy dose range was reached, in which there was a rise of the RR of both cancer deaths ($p < 0.01$) and total deaths."

**Table 11.2. Total Deaths and Relative Risks of Male and Female A-Bomb Survivors in Nagasaki During 1970–1988 Classified by T65 Dose**

**A. Initial numbers of subjects (1970), observed (*O*) and expected (*E*) numbers of total deaths and relative risk in 1970–1988 in Nagasaki among A-bomb survivors classified by T65D dose and sex.**

| T65D Dose (cGy) | Initial No. of Subjects | | Total Deaths | | | | Relative Risk (O:E) | |
|---|---|---|---|---|---|---|---|---|
| | | | Observed | | Expected | | All Causes | |
| | M | F | M | F | M | F | M | F |
| 1–49 | 562 | 938 | 182 | 202 | 106·7 | 209 | 1·01 | 0·97 |
| 50–99 | 182 | 168 | 56 | 39 | 63·3 | 34·7 | 0·88 | 1·12 |
| 100–149 | 108 | 158 | 36 | 39 | 39·7 | 34·7 | 0·91 | 1·12 |
| 150–199 | 196 | 267 | 59 | 48 | 58·7 | 48 | 1·01 | 1·00 |
| 200–599 | 440 | 437 | 172 | 79 | 149·7 | 59·3 | 1·15 | 1·33 |

**B. Observed (*O*) and expected (*E*) deaths in 1970–1988 in Nagasaki among A-bomb survivors classified by natural causes of death, sex and T65D dose.**

| Dose (cGy) | Number of Deaths from: | | | |
|---|---|---|---|---|
| | Non-Cancerous Diseases | | Cancer | |
| | *O* | *O:E* | *O* | *O:E* |
| *Males* | | | | |
| 1–49 | 126 | 1·09 | 35 | 0·64 |
| 50–99 | 30 | 0·85 | 26 | 1·56 |
| 100–149 | 23 | 0·77 | 13 | 1·34 |
| 150–199 | 38 | 0·84 | 21 | 1·56 |
| 200–599 | 113 | 1·07 | 54 | 1·32 |
| *Females* | | | | |
| 1–49 | 144 | 0·89 | 58 | 1·24 |
| 50–99 | 30 | 1·11 | 8 | 1·10 |
| 100–149 | 26 | 0·96 | 13 | 1·86 |
| 150–199 | 31 | 0·84 | 16 | 1·60 |
| 200–599 | 50 | 1·11 | 28 | 2·11 |

**C. Calculated (*L*) values by the logistic function $p = 1/[1 + \exp[-a - b_1(D - \langle D \rangle) - B_2(D - \langle D \rangle)^2 - cA]]$ and observed (*O*) values for deaths from non-cancerous diseases in males in Nagasaki classified by T65D dose.**

| Dose (*D*) (cGy) | Number of Non-Cancer Deaths | | | |
|---|---|---|---|---|
| | Observed (*O*) | Corrected *O:E* | Calculated (*L*) | Corrected *L:E* |
| 27 (1–49) | 126 | 1·07 | 123 | 1·05 |
| 79 (50–99) | 30 | 0·88 | 35·3 | 0·80 |
| 108 (100–149) | 23 | 0·80 | 21·9 | 0·76 |
| 167 (150–199) | 38 | 0·88 | 35·3 | 0·82 |
| 288 (200–599) | 113 | 1·11 | 113·1 | 1·11 |

$\langle D \rangle = 130$; $a = -6.14$ ($p < 0.01$); $b_1 = 0.29 \times 10^{-3}$ (*p* NS); $b_2 = 0.213 \times 10^{-4}$ ($p < 0.02$); $c = 0.115$ ($p < 0.01$).

NS = not significant.

## 2.0 Occupational Exposure

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Zbigniew Jaworowski states (1995b) that:

"From several studies of people occupationally exposed to low radiation doses discussed in UNSCEAR (1994), data on mortality of 13,491 employees of the Atomic Energy of Canada Limited, (Gribbin et al., 1992), 5504 were not exposed to radiation. The mean radiation dose of exposed persons was 49 mSv for men and 5.5 mSv for women. As shown in Table 6 the mortality die to all leukemias in the exposed group was only 32% of that in the general Canadian population. The observed mortality among employees of AECL from all cancers and from all noncancer diseases was also less than expected."

### 2.1 Occupational Exposure/Radiologists - Yalow 1994

*No excess cancers were found in British radiologists, with estimated 100-500cGy lifetime doses.*

Nobel Laureate Dr. Rosalyn Yalow states (1994) that, "British radiologists before 1921 (including much extreme X WI exposures) had 75% excess cancer-related deaths compared to other physicians. However, those starting after 1921 (with general improved radiation protection practices) had no excess cancer deaths, with typical excess exposures estimated at 100 to 500 rem." (Smith and Doll 1987)

*No excess cancers were found in U.S. Army radiologic technicians, with estimated 50-cGy doses.*

Nobel Laureate Dr. Rosalyn Yalow states (1994) that, "In WWII, 6500 radiologic technicians had an estimated 50 rem in training, with 24 months median service. A 29-year follow-up found no increased malignancies compared to army medical, laboratory, and pharmacy technicians."

### 2.2 Occupational Exposure/Nuclear Shipyard Workers - Cameron 1994

Prof Emeritus Dr. John Cameron reports (Cameron 1994) that "the Nuclear Shipyard Workers Study (NSWS 1991)... groups were selected from a database of almost 700,000 shipyard workers, including about 108,000 nuclear workers. The three study groups consisted of 28 542 nuclear workers with a working lifetime dose equivalent (DE) equal to or greater than 5 mS (0.5 rem), referred to here as NW >5; 10 462 nuclear workers with a working lifetime DE <5 mSv, referred to here as NW <5; and 33 352 nonnuclear workers, referred to as NNW...

TABLE I

Summary of Mortality, SMR, and 95% Confidence Interval for NW > 5, NW < 5, and NNW Shipyard Workers

| RADIATION EXPOSURE: | NW>5 | NW<5 | NNW |
|---|---|---|---|
| CAUSE OF DEATH: N = | 28,542 | 10,462 | 33,352 |
| ALL CAUSES | 2,797 | 1,168 | 4,453 |
| SMR | 0.76* | 0.81* | 1.00 |
| (95% C.I.) | (0.73, 0.79) | (0.76, 0.86) | (0.97, 1.03) |
| LEUKEMIA | 21 | 6 | 29 |
| SMR | 0.91 | 0.42 | 0.97 |
| (95% C.I.) | (0.56, 1.39) | (0.11, 1.07) | (0.65, 1.39) |
| LHC[b] | 50 | 13 | 85 |
| SMR | 0.82 | 0.53* | 1.1 |
| (95% C.I.) | (0.61, 1.08) | (0.28, 0.91) | (0.88, 1.37) |
| MESOTHELIOMA | 18 | 8 | 10 |
| SMR[c] | 5.49* | 6.14* | 2.54 |
| (95% C.I.) | (3.03, 8.08) | (2.48, 11.33) | (1.16, 4.43) |
| LUNG CANCER | 237 | 98 | 306 |
| SMR | 1.07 | 1.11 | 1.15 |
| (95% C.I.) | (0.94, 1.21) | (0.90, 1.35) | (1.02, 1.29) |

*Statistically significant.
[b]Lymphatic and hematopoietic cancers.
[c]Associated with asbestos exposure.

"Both nuclear worker groups had a lower death rate from leukemia and lymphatic and hematopoietic cancers than the nonnuclear group. All three groups had lower LHC death rates than the general population. Table I summarizes the data.

"The most significant and surprising finding of the NSWS research was that the nuclear workers with the greatest radiation exposure, a cumulative lifetime occupational dose equivalent of 5 mSv or more, had a standardized mortality rate (SMR) of deaths from all causes of only 0.76 that for their age and sex in the general population, while the

nonnuclear workers had an SMR of 1.0. The standard deviation of the SMR was ~0.015; i.e., the mortality rate for the nuclear workers was ~16 standard deviations below that of the nonnuclear worker group!

"The occupational exposure to the nuclear shipyard workers was comparable to the cumulated effective dose equivalent theyreceived from natural radiation. Their total radiation, occupational plus natural, is comparable to natural radiation exposures in some parts of the world.

"This study is probably the best scientific evidence, of many scientific data sources, to show that low levels of ionizing radiation exposure are without health hazard. The results clearly contradict the conclusions of BEIR that even small amounts of radiation have risk (in BEIR V and earlier reports), which have been largely based on the data from the Japanese atomic bomb survivors, who largely received their radiation exposures in very brief, high dose rate conditions and who are also now demonstrating that effective radiation health effects thresholds exist in the range of 20 to 200 rem.

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**Occupational Exposure/Nuclear Shipyard Workers - Pollycove 1994**

Dr. Myron Pollycove reports (1994) that "A ten-year study by the Johns Hopkins Department of Epidemiology, School of Public Health and Hygiene of nuclear shipyard workers was concluded recently. The Technical Advisory Panel (TAP), chaired by Arthur C. Upton, advised on the research and reviewed results. John Cameron, a member of the TAP, summarized the study and stated, 'This study is probably the best evidence that low levels of ionizing radiation are without health hazard.'

"The results contradict the conclusions of the BEIR V report that small amounts of radiation have risk -- the linear risk hypothesis. The database of almost 700,000 shipyard workers included almost 108,000 nuclear workers witli exposures beginning in the 1960s until the end of 1981. Three study groups were selected: 33,352 non-nuclear workers (NNW), 10,462 nuclear workers with a working lifetime dose equivalent (DE) of less than 5 mSv (NW<5), and 28,542 nuclear workers with a DE greater than or equal to 5 mSv (NW>5) where 5 mSv (0.5rem) is approximately equal to the sea-level background radiation (340 mr/yr) one would receive in 1 1/2 years. Deaths in each group were classified as due to: all causes, leukemia, lymphatic and hematopoietic cancers (LHC), mesothielioma, and lung cancer. The only cancer that showed a significantly increased incidence in the exposed groups as well as the NNW was the rare malignancy mesothelioma (36 deaths), a marker for asbestos exposure that is also associated with lung cancer. The data are summarized in Cameron 1994 Table 1 above.

"The nuclear worker groups had a lower death rate from all causes, leukemia, and LHC than the non-nuclear workers. These apparently beneficial effects of low dose irradiation are consistent with the increased longevity and 15'7o lower mortality and cancer death rates seen in the seven western states with high natural background radiation averaging about 1 mGy per year above that of the other states.

"The non-nuclear workers' death rates exactly matched those of the external non-shipyard matched control population. This demonstrates absence of the external healthy worker effect ascribed to adequate income, better health care, and the presence of Health sufficient to allow maintenance of a reliable work schedule. There remains the question of an internal Healthy worker effect resulting from the possible selection of more active individuals to be nuclear workers. The NW>5 group with the greater exposure had a death rate from all causes of 0.76 the standardized mortality rate (SMR), 16 standard deviations below that of the non-nuclear worker group (NNW). The NW<5 with lesser exposure had 0.81 SMR, about 8SD below the NNW. While a possible internal healthy worker effect could contribute to the lowered SMR of nuclear workers, comparison of the NW>5 group with the NW<5 group demonstrates that the group with the greater dose had the lower SMR with even greater statistical power. This provides very strong evidence that low levels of ionizing radiation are without health hazard." See Table 1, p. 2-1.

## 2.3 Occupational Exposure/High-Dose Workers - Berry 1994

Dr. Roger Berry reports (Berry 1994) that in a study "of morbidity and mortality data in a cohort of 542 male workers, who had accumulated individual doses in excess of 500 mSv and up to -2 S%, by the end of 1983. and an overlapping cohort of 470 workers who were involved in fighting the Windscale pile fire in 1957 or in subsequent cleanup operations, having a collective occupational radiation dose of ~180 person-Sv.

"A clear correlation was seen between recorded cumulative external radiation dose and the incidence in peripheral blood lymphocyte chromosomes of translocations scored by banding, but as expected, no correlation was seen between total dose and the incidence of unstable aberrations such as dicentrics, rings, and acentric fragments...

"In the >500-mSv, cohort, overall mortality to date is not significantly different from the U.K. national average, corrected for age, sex. and social class, and the slight excess of observed over expected deaths is due not to cancer but to diseases of the circulatory, system. There is actually- a slight deficit overall against expectation to date of deaths from malignant disease, due in part to a large deficit against expectation of lung cancer deaths. However there is a nonsignificant increase against expectation in cancer deaths from haemopoietic and lymphatic tissues... Comparable data for the Windscale fire cohort show a similar deficit of cancer deaths against expectation.

"Up-to-date cancer incidence data for these cohorts are reviewed and continue to show rates below those expected in the general population. Thus, in a population of workers exposed during their occupation over many years to radiation doses that would be considered unacceptable today, and studied as a "bellwether" for predicting risks to current workers, there is evidence at a cellular level of their having received that exposure, but as yet no evidence of unpredicted harm."

## Occupational Exposure/High Dose Workers - Fry 1995

Dr. Shirley Fry reports (1995) that, "In a population of 3145 current and former civilian employees at DOE facilities and the U.S. Navy's Nuclear Reactor Propulsion Plants [exposed to > 50 mSv (5 rem) in a year] for the years 1943 through 1978, follow-up doses for the total cohort are presented in Table II.

This population comprises individuals who were among the most highly exposed to radiation in the modern nuclear industry. We estimated that the study would be able to detect an increased risk for all cancers combined that was three times that estimated for low levels of radiation based on studies of the A-bomb survivors and other high-dose, high-dose-rate populations. From this perspective, the study was able to address allegations that the risks of radiation-induced cancers to those derived from underestimated "high-dose" populations exposed to radiation at high dose rates are underestimated.



TABLE II

Distribution of the ≥50-mSv Cohort ($N$ = 3145) by Cumulative Whole-Body Penetrating Radiation Dose*

| Dose (mSv) | Number | % | Dose (mSv) | Number | % |
|---|---|---|---|---|---|
| Unknown | 1 | — | 400-499 | 140 | 4.45 |
| 50-99 | 972 | 30.92 | 500-599 | 54 | 1.72 |
| 100-149 | 558 | 17.74 | 600-799 | 44 | 1.40 |
| 150-199 | 453 | 14.40 | 800-999 | 21 | 0.67 |
| 200-249 | 335 | 10.65 | ≥1,000 | 28 | 0.89 |
| 250-299 | 260 | 8.27 | Total | 3145 | 100.00 |
| 300-399 | 279 | 8.87 | | | |

*Collective dose: 718.0 person-Sv; range: 50.0 to 44 010 mSv; median cumulative dose: 153.4 mSv.

TABLE III

Number of Deaths from All Causes and Selected Site-Specific Cancers

| Cause (ICDA-8)* | ALL* Obs | WM Obs(E)* | WM SMR* | WM 90% CI* |
|---|---|---|---|---|
| All causes (001-999) | 588 | 448 | 88 | 80,97* |
| All cancers (140-209) | 163 | 126 | 109 | 90,129 |
| Digestive (150-159) | 45 | 36 | 124 | 87,171 |
| Lung (162, 163) | 80 | 46 | 109 | 80,144 |
| Bone (170) | 0 | 0 (.4) | 0 | — |
| Skin (172, 173) | 1 | 0 (3.4) | 0 | — |
| Brain, CNS (191, 192) | 5 | 5 | 138 | 41,298 |
| Thyroid (193) | 0 | 0 (.32) | 0 | — |
| All lymphoma, hematopoietic (200-209) | 16 | 13 | 112 | 61,197 |
| Lymphosarcoma (200) | 6 | 6 | 330 | 73,527 |
| Hodgkins disease (201) | 0 | 0 (1.28) | 0 | — |
| Leukemia (204-207) | 3 | 3 | 47 | 8,148 |
| Other lymphatic (202,203,208) | 7 | 5 | 146 | 47,343 |
| Benign neoplasms | 2 | 2 | 340 | — |
| All circulatory (390-458) | 359 | 190 | 78 | 68,90* |
| All respiratory (460-519) | 33 | 28 | 97 | 64,140 |
| All digestive (520-577) | 14 | 14 | 83 | 38,87* |
| All genitourinary | 5 | 4 | 74 | 20,190 |
| All external injuries (800-999) | 50 | 43 | 79 | 54,108 |
| Suicide (950-959) | 15 | 14 | 104 | 57,175 |

*Total ≥50 mSv/yr cohort ($N$ = 3145).
*Standardized mortality ratio (SMR) for all white males ($n$ = 3393).
*International Classification of Disease, adapted for use in the United States, 8th Revision.
*Number of deaths observed (number of deaths expected where none was observed)
*Observed number of deaths/number of expected based on U.S. white males, adjusted for age per calendar year.
*Confidence intervals not calculated when less than two deaths are expected.
*Statistically significant at 90% confidence level.

Mortality due to all causes and selected site-specific cancers for the total cohort and for all white males in the cohort are given in Table III.

## Occupational Exposure/High Dose Workers - Luckey 1995

*Cancer mortality was shown to be less in workers exposed to 5 cSv per year compared to control workers who were unexposed.*

Professor Emeritus Dr. T.D. Luckey (Luckey, 1995)) agrees with studies that show "... that over 96,000 workers, exposed to about 5 cSv (5 rem) above background

levels per year, have significantly lower cancer mortality rates than 212 000 control workers in the same plants, p<.001. In each study, the standard mortality rate (SMR) for cancer deaths in all the workers was significantly less than that of the general population, p< 01." Table 1 is a review of these major studies. It shows "total cancer mortality in over 300 000 nuclear workers, (mostly white males).... The data was corrected for age and lagged 10 years for cancer deaths and 2 years for leukemia deaths. The chi square statistic was used to estimate probabilities."

Table 1. Total Cancer Mortality in Nuclear Workers (Luckey, 1991)

| Plant | Shipyard Mantanoski | Weapons Gilbert | Weapons Kendall | Weapons Abbatt |
|---|---|---|---|---|
| Number of workers | | | | |
| Control | 111,757b | 20,619 | 58,945c | 21,000 |
| Exposed | 40,774d | 15,318 | 36,272e | 4,000 |
| Years Observed | | | | |
| Total | 16 | 33 | 30 | 20 |
| Mean(f) | 8 | 17 | 15 | 10 |
| Lifetime Exposure | | | | |
| Man Sv | 1,095 | 1,140 | 3,066 | 280 |
| mSv/Worker | 27 | 74 | 85 | 70 |
| mSv/y(f) | 3.4 | 4.3 | 5.7 | 7.0 |
| Cancer Mortality | | | | |
| Control Dead | ?,086 | 718 | 584 | 463 |
| Control Rate(g) | 27.6 | 34.8 | 9.9 | 22 |
| Exposed Dead | 968 | 318 | 96 | 8 |
| Exposed Rate(g) | 23.7 | 20.8 | 2.6 | 2 |
| Ratio (h) | .84 | .60 | .27 | .09 |
| pValue | <.001 | <.001 | <.001 | <.001 |

a) More that 95% white male adults. Death were age corrected and lagged 10 years in all except the Abbatt data.
b) All workers exposed to <5 mSv, 1984.
c) All workers exposed to <10 mSv.
d) Workers exposed to >or equal 5; charts 55 and 56, 1984.
e) All workers exposed to > or equal 10 mSv.
f) Estimated at one half the total observation years.
g) Cancer mortality per 1000 workers.
h) Mortality ratio = Exposed/Control

**2.4 Occupational Exposure/Plant Workers - Luckey 1994**

Professor Emeritus Dr. T.D. Luckey finds (1994) that, "A total of 35,933 white male workers (5,546 deaths) from three United States nuclear weapons plants with lifetime exposures of 2 to 20 cSv had lower total cancer mortality rates than internal controls, p < 0.001 (Figure 15) (Gilbert et al., 1989). The continuously decreased rate, shown in the cumulative curve, and the fact that those exposed to 25 cSv had less cancer mortality than those exposed to 13 cSv, p < 0.05, strongly suggest that the optimum lifetime exposure for decreased cancer mortality is greater than 25 cSv. Since the follow-up period averaged 19 years, the optimum exposure appeared to be more than 1 cSv per year. The combined workers had a lower cancer mortality rate than that of the United States population; the SMR was 0.79."

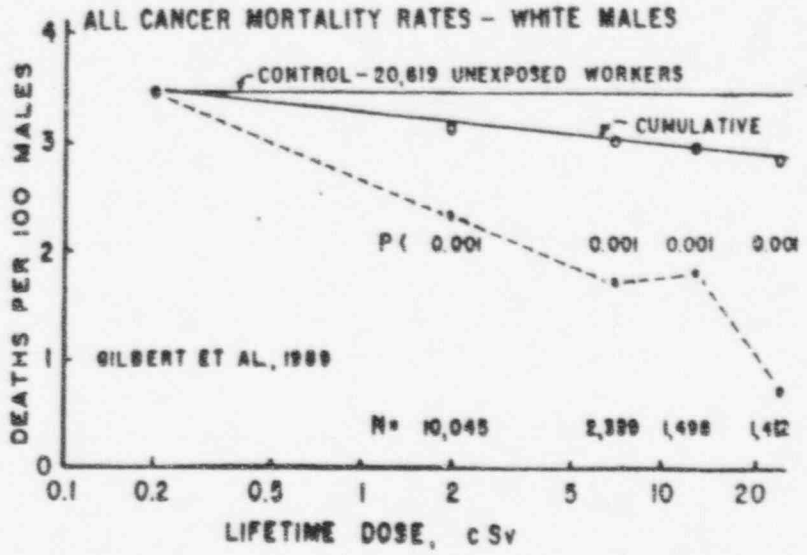


FIGURE 15. Effect of lifetime exposure upon cancer mortality rates in male nuclear weapons workers (Gilbert et al., 1989). The number of workers and p values are expressed within the figure.

When the leukemia mortality rate of exposed workers from three United States nuclear weapons plants were compared with that of unexposed workers, no statistically significant differences were found (Gilbert et al., 1989). When compared with the respective local populations, the mean SMR for leukemia mortality in all male workers of the three plants was 0.92.

levels per year, have significantly lower cancer mortality rates than 212 000 control workers in the same plants, p<.001. In each study, the standard mortality rate (SMR) for cancer deaths in all the workers was significantly less than that of the general population, p<.01." Table 1 is a review of these major studies. It shows "total cancer mortality in over 300 000 nuclear workers, (mostly white males).... The data was corrected for age and lagged 10 years for cancer deaths and 2 years for leukemia deaths. The chi square statistic was used to estimate probabilities."

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| Cancer Mortality | | | | |
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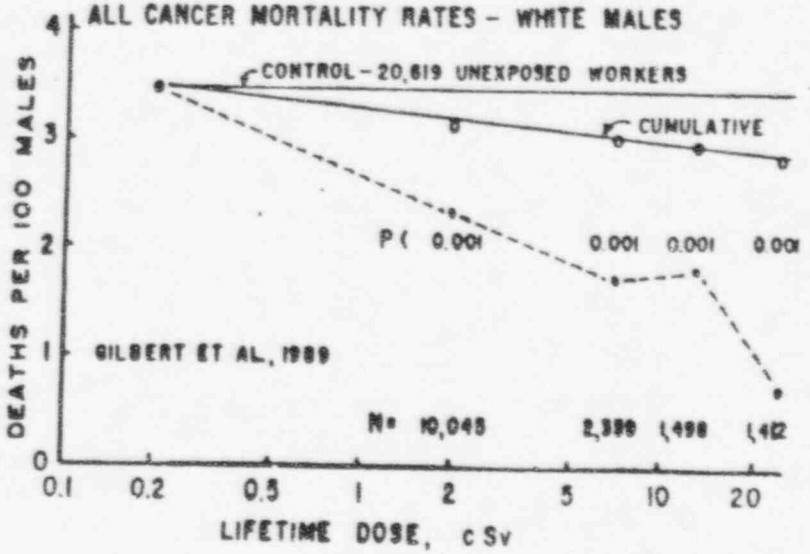


FIGURE 15. Effect of lifetime exposure upon cancer mortality rates in male nuclear weapons workers (Gilbert et al., 1989). The number of workers and p values are expressed within the figure.

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Cumulative lung cancer mortality of male workers in three United States nuclear weapons plants appeared to decrease as the dose increased (Figure 16) (Gilbert et al, 1989). Only in those with lifetime exposures of greater than 20 cSv was the decrease statistically significant, $p < 0.001$. The SMR for lung cancer mortality in all workers was 0.76.

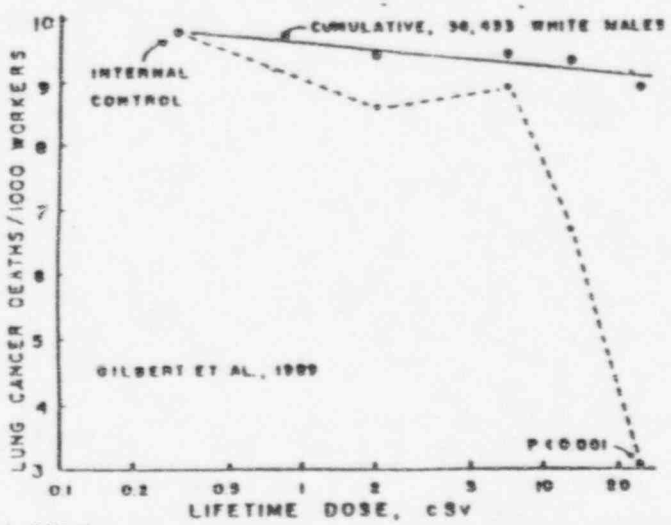


FIGURE 16. Effect of lifetime exposure upon lung cancer mortality rates in male nuclear weapon workers (Gilbert et al., 1989).

**Luckey, 1994, Intl. J. of Occ. Med. and Toxicology**

During 20 years in a Canadian energy plant, 4,000 nuclear workers with an average exposure of 70 mSv had a lower cancer mortality rate than 21,000 unexposed workers, $p < 0.001$ (Figure 17) (Abbatt et al, 1983). The cancer mortality rate of thermal workers in the plant was comparable with that of the general population of Ontario; the SMR was 0.97. There were no leukemia deaths in exposed workers during this study.

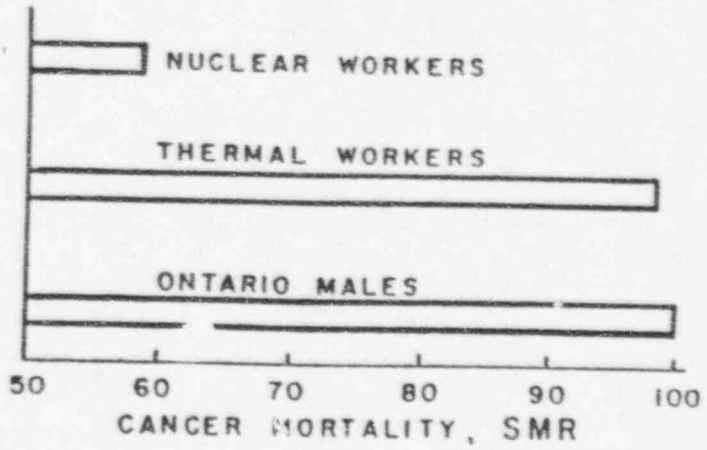


FIGURE 17. Comparison of cancer standard mortality rates of 4,000 males in nuclear energy work with 21,000 males in thermal energy work in a single Canadian plant (Abbatt et al., 1983). The SMR of each worker cohort is taken from the population of Ontario.

Deaths in another Canadian energy plant were followed from 1956 to 1985 (Gribbin et al, 1993). A comparison of over 4,000 exposed workers with 4,000 other workers in the same plant showed no significant differences in cancer mortality rates. The authors made an age adjustment without giving enough data to make an age correction. Comparison of all cancer deaths in all workers with the general population gave the following SMRs: all cancer, [illegible] prostate, 1.21; alimentary, 1.02; leukemia, 0.62; and lung, 0.86. None of these differences were statistically significant.

A study of 95,000 predominantly male workers in several British nuclear weapons plants from 1955 to 1988 involved 6,660 deaths; only 2.7% of the deaths were female (Kendall et al, 1992). The total cancer mortality rate decreased inversely with exposure, $p < 0.001$ (Figure 18). Since workers exposed to a mean of 7 cSv had a lower cancer mortality rate than those exposed to 2.4 cSv and had about the same rate as those who received 25 cSv, the optimum lifetime exposure for the 33 years appears to be at least 20 cSv, about 0.6 cSv per year. When compared with the population of England and Wales, the SMR for all cancer deaths in nuclear weapons plants was 0.86, $p < 0.001$.

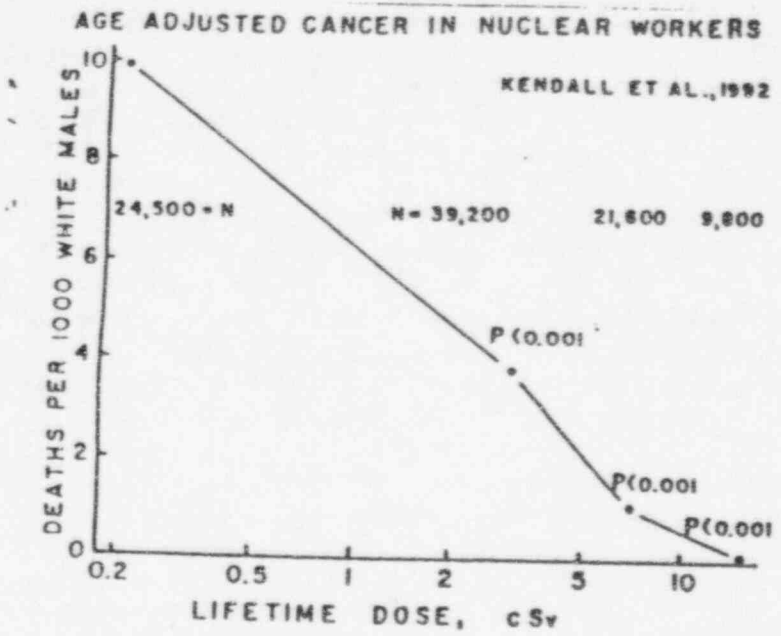


FIGURE 18. Effect of lifetime exposures upon age-corrected cancer mortality rates in several British nuclear weapons plants (Kendall et al., 1992). The numbers of workers and p values are given for each dose.

Leukemia mortality followed the pattern of total cancer mortality in the British study (Figure 19). Leukemia mortality in exposed workers was less than that of unexposed controls in the same plants, $p < 0.001$. The optimum appeared to be 10-30 cSv per 33 years. When compared with the general population, the SMR for leukemia mortality in all workers was 0.91, p = NS.

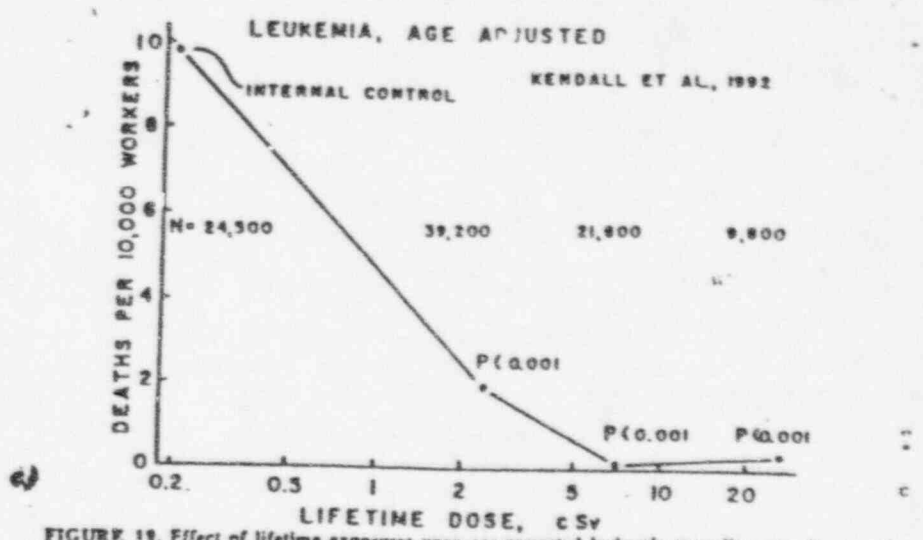


FIGURE 19. Effect of lifetime exposures upon age-corrected leukemia mortality rates in several British nuclear weapons plants (Kendall et al., 1992). The numbers of workers and p values are expressed for each dose.

Conclusions about chronic exposure of humans to low-dose irradiation are based upon almost eight million person-years (Table 2). These studies predominate over those studies which report increased cancer mortality in small pockets of workers, which are included in the larger surveys. Internal comparison with control and exposed workers in the same plant give irrefutable evidence that low-dose irradiation is beneficial. The "healthy worker effect" cannot account for the decreased cancer mortality rates in nuclear workers. The consistently decreased cancer mortality rates of exposed nuclear workers when compared with unexposed workers in the same plants are compelling evidence that the differences observed are <u>not</u> due to a "healthy worker effect." Both groups entered the plants under the same conditions and received comparable medical care. When compared with the general population, the longer average life span of workers should result in a higher cancer mortality rate. It does not. Thus, the "healthy worker effect" helps to validate radiation hormesis in cancer mortality.

TABLE 2. Major Studies of Cancer Mortality in Nuclear Workers

| Plant | Workers | Person-Yr | Reference |
|---|---|---|---|
| Shipbuilders | 70,730 | 1,591,832 | Matanoski, 1991 |
| Hanford | 44,100 | 1,675,800 | Gilbert et al, 1989 |
| Oak Ridge | 8,518 | 291,130 | Gilbert et al, 1989 |
| Rocky Flats | 5,897 | 165,116 | Gilbert et al, 1989 |
| Canada | 8,944 | 268,320 | Gribbin et al, 1993 |
| Canada | 25,000 | 500,000 | Abbatt et al, 1983 |
| Britain | 95,100 | 3,237,378 | Kendall et al, 1992 |
| Total | 258,089 | 7,729,576 | |

### 2.5 Occupational Exposure/IARC Radiation Worker Study - Pollycove 1995

Professor Emeritus Dr. Myron Pollycove reports (1995) that a recent report by the International Association for Research on Cancer (IARC) (Cardis et al 1995) similarly "misrepresents dose-response data to report a 'linear model' result. The IARC report chooses to ignore data that shows lower risk, i.e., a risk decrement.

"First, in this combined occupational exposure group it chooses to ignore the most accurate data, the Nuclear Shipyard Worker Study compared to the early weapons facility workers with their questionable dosimetry and confounding factors.

"Then, in a population of 15,825 total deaths, IARC reports on 119 leukemia deaths, excluding non-radiogenic leukemia. The data show that there are 60 deaths observed with 62.0 expected for doses of less than 1 cSv, and there are 59 deaths observed with 57.0 expected for doses greater than 1.0 cSv (applicable data extracted in Table 2). Clearly, there is no excess leukemia found in this data."

Dr. Pollycove notes that: "The IARC report states explicitly in the Statistical Methods section that they applied (they presumed) the linear model across 11 dose categories, and that 'As there was no reason to suspect that exposure to radiation would be associated with a decrease in risk..., one-sided tests are presented throughout.' This states that they effectively discount and ignore all negative data.

"For the table, the eleven dose categories were collapsed to seven, resulting in greater-than-expected leukemias in three of the seven dose groups (the * groups in Table 2). Since only positive data are allowed to be considered, only the data from the three greater-than-expected dose groups are used, even though these dose groups are not even contiguous. Since the selected data are not significant, the IARC reports that it performs a Monte Carlo calculation on 5,000 trials (effectively multiplying the data by roughly a factor of 100) to 'find' that the results show a 'significant' linear dose-response 'trend'.

"This 'result' was then the subject of a world-wide media campaign, reasonably reported even in Nuclear News, that the 'linear model' is confirmed. This report was widely distributed and accepted long before the data and analysis were published and available for review.

"IARC also reports that the 44 multiple myeloma deaths are similarly found 'significant', noting that this is 'attributable primarily to the associations reported previously ... in the Hanford and Sellafield cohorts.' This note indicates that they are aware, without so stating, that this 'association' is not found in other cohorts and is generally considered to be erroneous in the referenced studies, consistent with the weakness in the dosimetry and the confounding effects. (The study reports that cancer relative risk is 0.99 and leukemia is 1.22 at 10 cSv.)"

Clearly, if all data were considered by IARC without arbitrarily excluding contrary data, the mortality data in these combined populations do not support the 'linear model.' As Dr. Luckey has found, objectively examining all the data in each of the cohorts indicate positive/beneficial effects for the exposed populations, a result which would be reasonably expected to result in a positive (beneficial) effect in the combined populations. The IARC, consistent with BEIR, NCRP and other government data presentation, capriciously misrepresents the data to conform to the costly radiation protection policy mandate.

In an April 1996 report to the NCRP on this study, Dr. Ethyl Gilbert did not report that the study found support for the "linear model" in the data. However, the NCRP summary of the meeting explicitly attributed to Dr. Gilbert the conclusion that the IARC study had found confirming evidence of the "linear model."

**Table II. IARC Observed-Expected Leukemia (Except Chronic Lymphocytic Leukemia) Mortality (119 Deaths in 15 825 Total Deaths)**

| Cumulative dose (cSv) | Deaths (Observed/Expected) |
|---|---|
| 0-1 | 60/62.0 |
| 1-2 | 19/17.2* |
| 2-5 | 14/17.4 |
| 5-10 | 8/9.0 |
| 10-20 | 8/6.4* |
| 20-40 | 4/4.7 |
| >40 | 6/2.3* |

*Greater than expected leukemias. Note, for this table, 11 dose categories were collapsed to seven.

### 2.6 Plutonium Workers

## 3.0 Medical Patients

Dr. Sadao Hattori, Vice President of CRIEPI reports (BELLE 1994) that, "Professor Sakamoto is using radiation hormesis to cure and to suppress the reappearance of cancer in the hospital of Tohoku University. For example, he applied 10 cGy twice weekly for several weeks successfully against liver cancer and lymphatic tumors. He is successfully applying whole body, or half body low level dose combined with local high dose irradiation to treat non-hodgkin's lymphoma. The low survival rate of 36% in patients with non-hodgkin's lymphoma after five years of the therapy improved to a 90% survival rate with a low dose treatment schema. Some analytical results demonstrate an increase of the ratio of the helper T cells to suppressor T cells."

### 3.1 Medical Patients/Thyroid Cancer - Pollycove 1990

Dr. Myron Pollycove finds (Pollycove, 1995) that "ICRP (1990) agrees with UNSCEAR 1988 and BEIR V that the most current estimates of the risk to the thyroid are presented in the NCRP Report 80 (NCRP, 1985). ICRP 1990 states that the carcinogenicity of external radiation is estimated for the high dose range and extrapolated to low doses "...because of the presumed linear nature of the thyroid response to external radiation. I-131 was estimated to be about one-fourth to one-third as effective as external radiation (NCRP, 1985; UNSCEAR 1988b)."

"...The UNSCEAR report states that for "A combined analysis of nearly 47,000 Swedish patients given I-131 for thyroid cancer, for hyperthyroidism or for diagnostic purposes [Holm 1989, 1991] ....no clear association of cancer induction by radiation was evident in the analysis." The NCRP 1985 report analyses the earlier studies by Holm (1980, 1981 1984) of 14,690 I-131 administrations, including 10,133 patients (494 under age 20) with diagnostic doses and 4,557 patients with therapeutic doses for hyperthyroidism and con concludes,".... I-131 has not been shown to be carcinogenic in people..." This 'problem' was circumvented by assuming the largest number of thyroid cancer cases compatible with the data at the upper limit value of one-third is the relative effectiveness of I-131 compared to external radiation for the induction of thyroid carcinoma. A decade later the above mentioned reports by Holm continue to demonstrate no excess cancer or leukemia. These reports include a cohort of 35,074 patients given diagnostic doses, including 2000 under the age of 20, and another 12,000 patients given therapeutic doses of I-131. This much larger number of patients has reduced the upper limit of relative effective carcinogenicity of I-131 compared to external radiation from 1/3 to 1/17. To reach zero in this manner, and infinite number of patients is required."

### Medical Patients/Thyroid Cancer - Yalow 1994

*No excess leukemia is found with doses of 10-15 cGy to the whole-body dose from I-131 hyperthyroid therapy.*

Nobel Laureate Dr. Rosalyn Yalow states (1994) that, "Before 1968, 1 to 3 million US patients received I-131 thyroid diagnosis. A Swedish 20-yr follow-up of about 35,000 patients, 5% exposed at < 20 yr old, with a mean thyroid dose of 50 rem, found that patients diagnosed for reasons other than a suspected tumor, had thyroid cancers at 62% of controls (significant)."

### 3.2 Medical Patients/I-131 Leukemia - Yalow 1994

*No excess leukemia is found with doses of 10-15 cGy to the whole-body dose from I-131 hyperthyroid therapy.*

Nobel Laureate Dr. Rosalyn Yalow states (1994) that, "Hyperthyroid patients treated with I-131 have about 10 rem whole-body (bone marrow) irradiation. In a study of 36,000 patients, 22,000 received I-131, with 14,000 mostly receiving surgical treatment. At 7- and 10-yr follow-ups, sufficient for leukemia effects, no difference exists in the two groups.

"Another study of 10,000 patients followed for 15 yr is also negative.

### 3.3 Medical Patients/X-ray Leukemia

### 3.4 Medical Patients/Fluoroscopy Breast Cancer - Pollycove 1994

Prof. Emeritus, Myron Pollycove, MD, reports (Pollycove 1994) that, "The Canadian study of fluoroscoped women includes 31,710 patients admitted to national sanatoriums between 1930 and 1952 and alive on January 1, 1950. (Miller 1989) The results relate deaths from breast cancer between 1950 and 1980 that occurred 10 or more years after first exposure to fluoroscopic radiation. Fluoroscopic examination in Nova Scotia was performed AP (anterior-posterior), with the patient facing the fluoroscope. This position increased the breast dose to 50 mGy per exposure compared to 2 mGy per exposure in all the other provinces in which the examination was performed PA (posterior-anterior), with the patient's back against the fluoroscope. The standardized mortality rates from breast

cancer for various dose ranges is shown in Table 11.4 with the high dose, high dose rate data of Nova Scotia separated from the low dose rate data of the other provinces.

"Linear and litiear-quadratic dose-response models were compared w h respect to data fit. The authors concluded 'that the most appropriate form of dose response relation is a simple linear one, with different slopes for Nova Scotia and the other provinces.' On the basis of this linear model, Table 11.5 predicts the lifetime excess risk of death from breast cancer after a single exposure to 1 cGy, an amount approximately three times the average annual background radiation.

"The epidemiologic data listed in Table 11.4 and the associated fitted models were not presented graphically. The omitted graph is shown in Figure 4, together with an empirical polynomial function fitted to the data. The linear model for 2 mGy exposures discards the data at 0.15 Gy and at 0.25 Gy, the data with the best confidence limits. Compared to the controls receiving 0 to 0.09 Gy, 0.15 Gy and 0.25 Gy demonstrate relative risks (RR) of 0.66 ($p<.01$) 0.85 ($p<.38$), respectively. While the P' of 0.85 is not statistically significant, it is consistent with the significant RR of 0.66 and the zero equivalent point of 0.31 Gy indicated by the fitted polynomial function. For exposures above the zero equivalent point, the RR becomes positive after being negative in the range of 0 to 0.31 Gy. The decreased RR of breast cancer produced by low dose, low dose rate radiation were rejected a priori by the choice of mathematical models that extrapolate the dose-risk relation from high dose exposures to low dose exposures. The risks associated with low dose exposures cannot be measured, the authors state, 'because the expected small excess of breast cancers would be obscured by the much higher background rate of breast cancer.' Consequently, the unexpected was rejected since the possibility of a measurable decreased risk associated with low exposures appeared to be inconceivable. The highly significant decreased RR of 0.66 at 0.15 Gy and the RR of 0.85 at 0.25 Gy, both with the highest confidence limits of the entire study, are not shown graphically, not even discussed. Instead, the linear model for 0.002 Gy exposures is used in Table 11.5 to predict the lifetime excess risk of death from breast cancer to be approximately 60 per million women after a single exposure to 1 cGy at the age of 30. Nine hundred excess deaths from breast cancer are predicted theoretically from the exposure of one million women to 0. 15Gy. However, the quantified low dose data predicts with better than 99% confidence limits that instead of causing 900 deaths, a dose of 0.15Gy would prevent 10,000 deaths in these million women.

**Table 11.4. Canadian Study of the Incidence of Breast Cancer Following Fluoroscopic Examinations**

| Dose Gy | Standardized Rate Per $10^4$ Person Years | | |
|---|---|---|---|
| | Nova Scotia | Other Provinces | All Provinces |
| 0–0.09 | 455.6 (131) | 585.8 (288) | 578.6 (301) |
| 0.10–0.19 | | 389.0 (29) | 421.8 (32) |
| 0.20–0.29 | | 497.8 (24) | 560.7 (26) |
| 0.30–0.39 | 1709 (11) | 630.5 (17) | 650.8 (18) |
| 0.40–0.69 | | 632.1 (19) | 610.0 (19) |
| 0.70–0.99 | | | 1362 (13) |
| 1.00–2.99 | 2060 (14) | | 1382 (17) |
| 3.00–5.99 | 2811 (13) | 873.1 (14) | 2334 (14) |
| 6.00–10.00 | 7582 (8) | | 8000 (9) |
| ≥ 10.00 | 21.810 (12) | | 20.620 (13) |

*The number of deaths is shown in parentheses. The calculations exclude the values for 10 years after the first exposure and heve been standardized according to age at first exposure (10 to 14, 15 to 24, 25 to 34, and ≥ 35 years) and time since first exposure (10 to 14, 15 to 24, 25 to 34, and ≥ 35 years) to the distribution for the entire cohort.

**Table 11.5. Predicted Lifetime Excess Risk of Death from Breast Cancer per Million Women after a Single Exposure to 1 cGy**

| Age at Exposure Yr. | Additive-Risk Model | Relative-Risk Model |
|---|---|---|
| 10 | 125 | 108 |
| 20 | 95 | 89 |
| 30 | 67 | 55 |
| 40 | 42 | 27 |

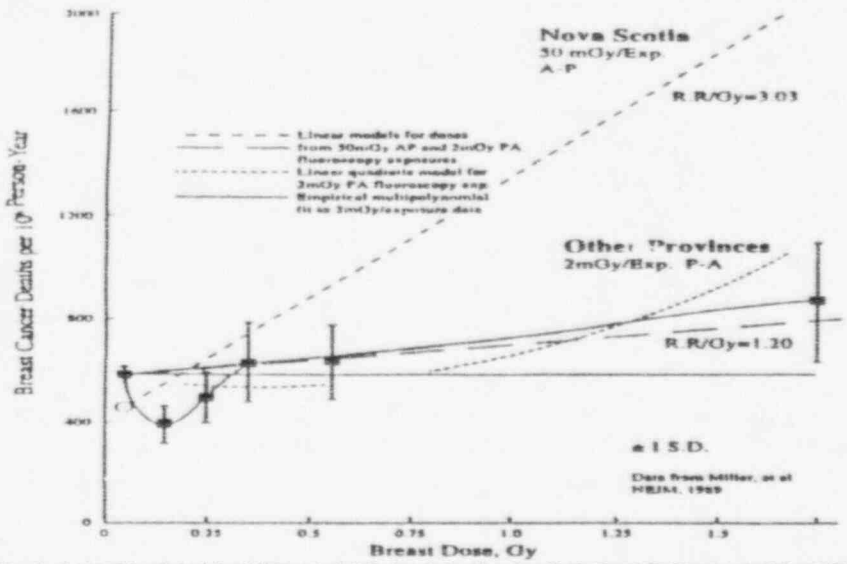


Fig. 4. A graphic plot of the Miller et al. tabular data showing their "best-fit" linear model and linear-quadratic model relationships, and the best-fit empirical polynomial function for the data.

## CONCLUSION

"Significant positive health effects associated with low level radiation have been demonstrated in a review of five epidemiologic studies: decreased mortality of nuclear shipyard workers, decreased noncancer mortality of atomic bomb survivors in both Hiroshima and Nagasaki and Nagasaki alone, decreased lung cancer mortality associated with increased radon exposure of the U.S. population, and decreased breast cancer mortality of women in Canada after having received multiple fluoroscopic examinations. The tendency to neglect or reject data that contradicts the linear-no threshold theory of radiation carcinogenesis is supported by confidence that chromosome aberration and gene mutation can be produced by a single particle of ionizing radiation and so initiate a malignancy. The number of such interactions with cell nuclei is both logically and demonstrably proportional to the dose. However, no consideration is given to biological defense mechanisms that could be stimulated further by low level increments of radiation above the background level. Such stimulated defense mechanisms could also decrease carcinogenesis by chemical and other non-ionizing agents as well as high level ionizing radiation. Multiple defense mechanisms at molecular, cellular, organ, and systemic levels involving enzymatic, hormonal, immunologic, and stress protein interactions are currently being demonstrated and confirmed by numerous investigators.10-31 Recently a human radiation repair gene has been cloned and transfected into a mutant Chinese hamster with sensitivity to both ionizing radiation and certain alkylating agents resulting from defective repair of DNA strand breaks. These trailisfected mutants demonstrate overexpression of the human DNA repair minigene with repair capacity increased above that of the wild-type Chinese hamsters."

Mounting reproducible evidence of the operation of various defense mechanisms and their stimulation by low dose ionizing radiation will provide further details of how biological defense mechanisms, nonoperative at high doses, are stimulated and enhanced by low level radiation damage so as to overcorrect and predominate. These investigations have clarified why the negative health effects observed at high levels of radiation that effectively overwhelm these defense mechanisms cannot be extrapolated to the low levels in which these stimulated defense mechnanisms predominate with decreased cancer induction, decreased mortality, and other observed positive health effects.

### 3.5 Medical Patients/Thorotrast Patients

## 4.0 Radium-burden Population

### 4.1 Radium-burden Population/Bone and Head Cancer - Evans 1974

Professor Emeritus Dr. Robley Evans states (1974) that, "We have tested a number of mathematical relationships (Evans 1966; 1967) (Evans 1969) and have found no smooth function which gives an acceptably close fit over the entire range of dosage, for either a pharmacological end point such as the so-called classical and reduced X-ray scores (Evans 1966; 1967) or for an epidemiological end point such as cumulative tumor incidence. Rather, the data of Fig. 3 seem to divide into two domains, characterized in the low-dose domain by negligible radiobiological effects where body repair mechanisms presumably keep pace with the rate of radiation injury, and in the low-dose domain by negligible radiobiological effects where body repair mechanisms presumably keep pace with the rate of radiation injury, and in the high-dose domain by a highly significant occurrence of osteoporosis, dense bone necrosis, spontaneous fracture, life-span shortening and radiogenic malignancy.

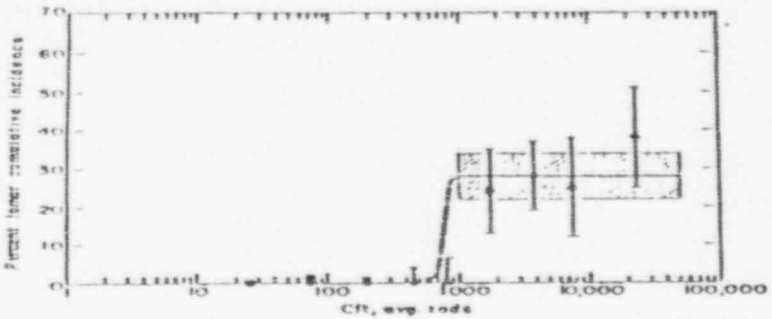


FIG. 3. The observed radiogenic tumor cumulative incidence or occurrence in the "epidemiologically suitable" cases[12] summarized in Table 2, and in classes a through e of Table 1. The shaded region corresponds to the mean occurrence 28 ± 6% between 1000 and 50,000 rads.

"We do adhere to the principles first clearly enunciated by Chamberlain (T.C. Chamberlain 1965) in 1890 of the "Method of Multiple Working Hypotheses." Several authors who like to select and massage portions of other people's data, including Hems (Hems 1968), Gofman and Tamplin, Goss (Evans 1972) (Goss 1970), Snyder (Snyder) and others have asserted that in their hands our data are in satisfactory agreement with a linear nonthreshold model. None of these authors has bothered to apply any statistical tests for goodness of fit or to offer any critique of the detailed statistical evaluations of the data in our previous publications (Evans 1969; 1972).

*Gofman-Tamplin linear nonthreshold model*

"Figure 4 shows our data plotted on a linear scale of cumulative rads from 0 to 50,000 rads. The dashed line labeled G-T is the linear nonthreshold relationship proposed by Gofman and Tamplin (Gofman 1971) for sarcoma occurrence. When the chi-square test for goodness of fit is applied to the portion of our data which they have selected as proof of their linear nonthreshold thesis one finds that the probability, *P*, that differences from the G-T linear model as large or larger than those observed could be due to chance is less than 1 in 200,000,000. These mathematical odds against this linear model are astronomical. Their claim that they can represent the radium and mesothorium data by their linear model is therefore quantitatively unsupportable.

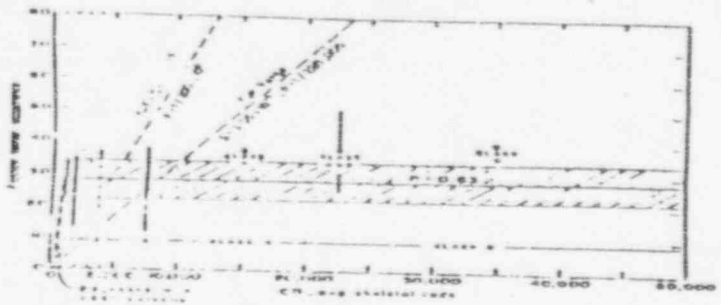

FIG. 4 The epidemiologically suitable radium and mesothorium cases of Table 2 and Fig. 3 plotted with a linear scale of dosage. On such coordinates the 500-plus cases in the region of interest below 1000 rads become compressed into a structureless blob. The linear nonthreshold model advocated for sarcoma occurrence by Cohen and Lapin[10] is the dashed line labelled "G–T", whose slope is $6 \times 10^{-6}$ sarcomas per person-rad, and for which the chi-square test for goodness of fit gives a probability, $P$, of less than 1 in 200,000,000. The "Full Range" linear nonthreshold model has a slope of $3.39 \times 10^{-6}$ tumors per person-rad, and a chi-square probability, $P$, of less than 1 in 200,000. The BEIR report's linear nonthreshold model lies between the G–T and Full Range lines, has a slope of $4.4 \times 10^{-6}$ sarcomas per person-rad, and is also strongly rejected by the chi-square test for goodness of fit.

*BEIR committee's linear nonthreshold model*

"In its consideration of the radium and mesothorium cases, the BEIR report (1972) seems brief and tightly written, treats the subject as "highly controversial," and involves what appear to be internal contradictions, omissions, and other doubtful matters. First: it discusses only bone sarcomas, and does not recognize the existence of the well-established occurrence of head carcinomas; second: it uses exclusively the CHR data (R.E. Rowland) but does not recognize that more than half of the sarcoma cases in the M.I.T. and ANL-ACRH series which are blended in the CHR data are symptom-selected cases and are epidemiologically unsuitable for constructing dose vs. response relationships; third: it compresses the CHR dosages cohorts by lumping more than 500 cases with dosages less than 500 rads into a single point plotted at zero rads on an arithmetic-scale graph, and lumps 80 cases with dosages from 5000 to 44,000 rads into a single point plotted at 12,000 rads.

"Fourth: the BEIR report notes that the resulting graph is 'more consistent with a curvilinear relationship' and that 'there appears to be a lower limit of dose at which no significant cancer effects have yet been observed,' and yet proceeds to evaluate an 'absolute risk' on a linear nonthreshold model. By introducing an assumed RBE of 10 for alpha rays, and a 40-yr burden-time for all tumor cases, it elects to represent the regrouped data by a linear nonthreshold model with a slope of 0.11 bone sarcomas per year per million person-rems. It is inadvisable to use the rem as a unit to relate skeletal average bone dose to bone tumor incidence because the target tissues are not known with certainty and the RBE of alpha particles for tumor induction in humans is unknown. Converting back to rads with their assumed 1 rad = 10 rem, and to cumulative incidence with the 40-yr burden time which they apparently introduced *ad hoc* leads to a slope for cumulative incidence or occurrence of $4.4 \times 10^{-6}$ sarcomas per person rad. In Fig. 4 this would be a line about midway between the two dashed lines marked 'G-T' and 'Full Range.' Application of the chi-square test indicates that differences from this BEIR linear nonthreshold model as large or larger than those observed could be due to chance is less than 1 in 1,000,000 repetitions. Clearly this model is unsupportable, especially in the low-dose domain.

"Fifth: this 'absolute risk' appears to have been extrapolated, along with other risks, to the domain of 5 rem per 30 yr (0.17 rem/yr), and treated in the BEIR report as an absolute basis prediction, rather than as an upper limit of risk, a caveat which has been so often emphasized by the UNSCEAR, ICRP and NCRP. Extrapolations which extend from the dosage region above 1000 rads to the region of 5 rads (a factor of more than 200) may be very much in error and must be viewed with substantial reservations."

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**Radium-burden Population/Bone and Head Cancer - Rowland 1983**

R.E. Rowland et al. report (1983) that, "In Table 1 all the known cases of potential exposure to radium before 1950 in the U.S. are summarized. Of the 4076 known cases, body burden measurements have been made for 1953. Of these measured cases, 632 (32.4%) had died by the end of follow-up. (Dec. 1979)

*Table 1. Radium cases exposed before 1950; status at end of 1979*

| Group | Sex | Measured Cases: Number of cases known | Measured Cases: Number of cases | Measured Cases: Mean year of first exposure (± S.D.) | Measured Cases: Average age at first exposure (± S.D.) | Measured Cases: Number dead | Measured Cases: Number of bone sarcomas | Detected but Unmeasured Cases: Number of cases | Detected but Unmeasured Cases: Mean year of first exposure (± S.D.) | Detected but Unmeasured Cases: Average age at first exposure (± S.D.) | Detected but Unmeasured Cases: Number dead | Detected but Unmeasured Cases: Number of bone sarcomas |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Dial workers | F | 3055 | 1468 | 1931 ± 11 | 20.3 ± 5.0 | 331 | 42 | 1077 | 1933 ± 11 | 24.5 ± 9.1 | 512 | 21 |
| | M | 272 | 79 | 1928 ± 12 | 25.9 ± 9.2 | 41 | 0 | 116 | 1926 ± 11 | 33.6 ± 12.9 | 98 | 0 |
| Medical cases | F | 164 | 96 | 1927 ± 5 | 35.4 ± 13.5 | 84 | 15 | 23 | 1926 ± 4 | 37.7 ± 15.5 | 30 | 1 |
| | M | 171 | 67 | 1927 ± 5 | 33.7 ± 14.7 | 50 | 2 | 57 | 1928 ± 3 | 45.9 ± 15.9 | 56 | 1 |
| Laboratory workers | F | 40 | 25 | 1926 ± 10 | 22.1 ± 5.1 | 8 | 0 | 4 | 1930 ± 12 | 28.3 ± 13.8 | 2 | 0 |
| | M | 309 | 188 | 1926 ± 11 | 26.3 ± 8.1 | 108 | 0 | 75 | 1926 ± 11 | 30.9 ± 10.1 | 63 | 1 |
| All other cases | F | 29 | 16 | 1923 ± 8 | 24.3 ± 5.4 | 6 | 0 | 2 | 1931 ± 0 | 23.3 ± 7.9 | 0 | 0 |
| | M | 35 | 13 | 1937 ± 13 | 29.8 ± 12.2 | 4 | 0 | 17 | 1937 ± 11 | 36.6 ± 13.1 | 11 | 0 |
| Totals | | 4076 | 1953 | - | - | 632 | 60 | 1381 | - | - | 772 | 24 |

*"Female dial workers*

"Female dial workers constitute the largest of the groups. Of the 2545 located women, body burden measurements have been performed on 1468; of these, 1137 were alive at the end of follow-up.

*"Medical exposures*

"The only other group in which bone sarcomas have been observed among the measured cases are those who acquired radium for medical reasons. These individuals either received radium by intravenous injection or orally. Evans (Evans 1966), in an informative description of the medical uses of radium, estimated that several thousand persons acquired radium via these routes. There are few mechanisms by which those who acquired radium medically can be identified. Most of the 18 bone sarcoma cases among the measured medical cases were identified as radium cases only after signs of bone sarcoma.

*"Laboratory workers*

"Some relatively large radium intakes have been measured for the laboratory workers, but only one bone sarcoma has been recorded, and that one occurred in an unmeasured case. Evans (Evans 1966) has estimated that there might have been between 500 and several thousand laboratory workers exposed to radium. Thus, like the medical cases, the sample of these cases available is only a small fraction of the total.

*"Male dial workers*

"Relatively few men, compared to women, were employed in the dial industry."

SYSTEMIC INTAKE

"The systemic intake is the quantity ($\mu$Ci) of radium that entered the blood during the period of exposure. This quantity is estimated from measurement of the body content at later times. It has been shown (Rowland 1978) that, for the induction of bone sarcomas in humans, each microcurie of (Ra-226) appears to be about 2½ times as effective as a $\mu$Ci of (Ra-228). Therefore in this report the systemic intake for each case is given, in $\mu$Ci, as the sum of $\mu$Ci (Ra-228) intake plus 2.5x $\mu$Ci (Ra-226) intake.

"The best group for a dose-response analysis is the female dial worker population. In this case the measured population contains 1,468 cases who experienced 42 bone sarcomas; age- and time-specific rates for white females indicate that 0.5 bone sarcoma was expected in this group (R.R. Monson 1974).

"To eliminate cases possibly measured as a consequence of their symptoms, any case of death or diagnosis of bone sarcoma that occurred within 2 years of first measurement was removed. Using these criteria, 1,257 female dial workers are in the measured population, but only 13 bone sarcomas remain; the expected number for this group was 0.2 bone sarcoma.

"Various logical forms of a general dose-incidence expression were fitted to the data, and subsequently tested by a $\chi^2$ statistic. Each equation was fitted to the 13 data points by a general weighted least-squares procedure for arbitrary functions (A.J. Barr) (A.R. Gallant). Acceptable fits implies that the coefficients $\alpha$, $\beta$, and $\gamma$ were positive and the $\chi^2$ analysis resulted in a $p$-value equal to or greater than 0.05. The fitting procedure yielded an excellent fit for the linear-quadratic-exponential (LQE) function for the data derived from year of first entry into the industry, but the coefficient for $\alpha$ was negative.

"In Fig. 2 the dose-squared exponential function, ± S.D., is shown on a semi-logarithmic plot of the data points. The range of values shown here overlaps the LQE function except at the lowest intake levels. These two functions differ most markedly at about 100$\mu$Ci intake; Fig. 3 shows the two functions on a semi-logarithmic plot which includes only the eight lowest intake levels where no bone sarcomas were observed. The area between the two curves is hatched, to indicate that the LQE function might fall anywhere in this region with $p > 0.05$.

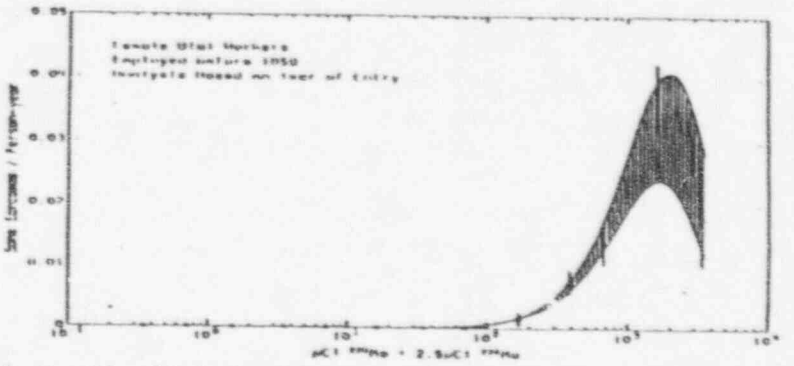


FIG. 2. A semi-logarithmic plot of bone sarcomas per person-yr at risk ($I$) vs systemic intake ($D$) for female dial workers employed before 1930 showing the dose-squared exponential fit. The shaded band indicates the range covered by the function when the fitted coefficients are allowed to vary by ± 1 S.D. The error bars represent the binomial standard errors of the observed incidences.

For the data set based on first measurement, no test of the general equation was possible, for with three fitted parameters there were no degrees of freedom left for the $\chi^2$ test. However, the situation was obviously similar to that above; the least squares fit to the LQE equation yielded a negative coefficient for the linear term while with $\alpha$ set equal to zero, the dose-squared exponential was found to be an acceptable fit.

"EFFECT OF ADDITIONAL BONE SARCOMAS

"The question is often asked, if the next bone sarcoma to appear falls in one of the systemic intake levels in which there are no bone sarcomas, will a linear or linear-exponential function then adequately fit the data? This effect was examined by adding extra sarcomas to the data set based on entry into the industry. Fits were obtained and tested with these "extra" sarcomas placed in the intake ranges where no bone sarcomas have been observed. No additional acceptable fits could be found with one or two "extra" sarcomas, but when three were added, a linear-exponential function could be fit to the data with $p > 0.05$.

"EXTRAPOLATION TO NON-DIAL RADIUM CASES

"The assumption is made that the dose-squared exponential function derived from

the analysis of female dial workers based on year of employment can be used to predict the number of bone sarcomas in various population subgroups.

"Table 4 gives the number of observed and predicted bone sarcomas by radium intake level for three population groups. The medical cases, laboratory workers, male dial workers, and miscellaneous cases have been combined into two groups, by sex, for comparison with the female dial workers.

*Table 4. Observed and predicted radium-induced bone sarcomas, by systemic intake level, for female and male radium cases*

| Systemic intake level (pCi) | Female Dial Workers | | | Female | | | All Other Radium Cases | | | Male | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Number of cases | Observed | Predicted | Number of cases | Observed | Predicted | Number of cases | Observed | Predicted | Number of cases | Observed | Predicted |
| >2500 | 17 | 4 | 5.1 | 2 | 0 | 0.5 | 5 | 0 | 1.3 | | | |
| 1000–2499 | 25 | 17 | 13.3 | 10 | 2 | 1.9 | 7 | 0 | 4.1 | | | |
| 500–999 | 19 | 9 | 9.8 | 11 | 4 | 5.3 | 5 | 1 | 2.4 | | | |
| 250–499 | 34 | 10 | 8.2 | 16 | 4 | 2.3 | 11 | 2 | 2.3 | | | |
| 100–249 | 29 | 2 | 2.2 | 21 | 4 | 1.2* | 16 | 0 | 0.4 | | | |
| 50–99 | 22 | 0 | 0.4 | 8 | 1 | 0.1 | 14 | 0 | 0.2 | | | |
| 25–49 | 49 | 0 | 0.2 | 6 | 0 | 0.0 | 15 | 0 | 0.0 | | | |
| 10–24 | 75 | 0 | 0.1 | 4 | 0 | 0.0 | 19 | 0 | 0.0 | | | |
| 5–9.9 | 72 | 0 | 0.0 | 8 | 0 | 0.0 | 27 | 0 | 0.0 | | | |
| 2.5–4.9 | 103 | 0 | 0.0 | 9 | 0 | 0.0 | 37 | 0 | 0.0 | | | |
| 1.0–2.4 | 196 | 0 | 0.0 | 11 | 0 | 0.0 | 34 | 0 | 0.0 | | | |
| 0.5–0.99 | 164 | 0 | 0.0 | 8 | 0 | 0.0 | 29 | 0 | 0.0 | | | |
| 0.25–0.49 | 152 | 0 | 0.0 | 12 | 0 | 0.0 | 20 | 0 | 0.0 | | | |
| <0.25 | 529 | 0 | 0.1 | 12 | 0 | 0.0 | 98 | 0 | 0.0 | | | |
| Totals | 1460 | 42 | 40.3 | 138 | 15 | 17.3 | 347 | 3 | 10.9* | | | |

*p < 0.05 (chi-squared, Aspelund-Nekola)

## "DISCUSSION

"The accumulation of information on persons carrying internally deposited radium isotopes has been underway in this country since the pioneering studies of the early radium dial painters (H.S. Martland 1931). However, not until the U.S. Atomic Energy Commission funded studies of the effects of radium did an intensive search for radium cases get under way.

"Since no bone sarcomas have been observed among the 1680 measured cases with systemic intakes less than 50pCi, it is evident that the life-span probability of bone sarcoma induction is very small for small doses of radium.

"The analysis based on the entire female dial worker population found only one acceptable least-squares fit.

"These results are not greatly sensitive to the occurrence of new bone sarcomas among the lower intake levels, where no sarcomas have yet been seen. It would have taken three additional sarcomas strategically located in the lowest intake ranges in order for the linear-exponential function to be considered at the $p=0.05$ level. With the passage of time, it is to be expected that a non-radium-induced bone sarcoma will appear in this population of dial workers. Such a malignancy cannot be distinguished from a radium-induced sarcoma. If a naturally occurring sarcoma appears, it is likely to appear in one of the eight lowest intake levels, for 1110 of the 1137 living cases are in these ranges. Indeed, since only 27 women remain alive in the five highest intake ranges, the number of potential new bone sarcomas in these intake ranges is limited.

"From examination of Fig. 3, it might appear that there is little difference between the dose-squared exponential and the LQE at very low intakes, but this is not the case. At the very low intakes which correspond to current standards, the predictions of these two functions appear to be markedly different. Consider the drinking water standard of 5pCi/l. as proposed by the Environmental Protection Agency, which can be shown to correspond to an annual intake of 843pCi/l. of $^{226}$Ra (R.E. Rowland 1978). After a 1-yr. intake, the LQE function with $\alpha=0$ would predict a lifetime risk from radium of $5 \times 10^{-11}$ bone sarcoma/person-yr, while with $\alpha=1.3 \times 10^{-3}$ the prediction is $1 \times 10^{-8}$ bone sarcoma/per son-yr, a factor of $2 \times 5 \times 10^{+14}$ greater. The natural incidence of bone sarcoma is a function of the age and composition of the group considered, but a reasonable overall value for adults is $10^{-5}$ bone sarcoma/person-yr. Of course, neither of these induced rates could be distinguished from the natural incidence of bone sarcoma, even with a population at risk as large as the current population of the U.S.

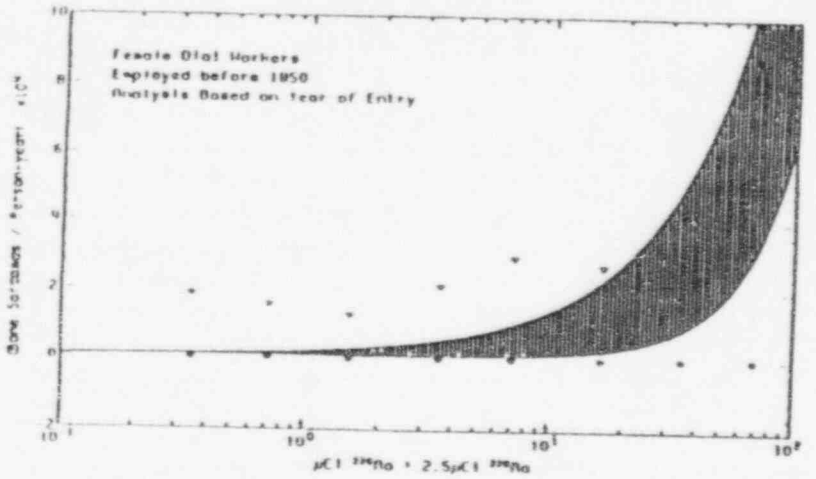


Fig. 3. A semi-logarithmic plot of bone sarcomas per person-yr at risk ($I$) vs systemic intake ($D$) over the lower end of the systemic intake range where no bone sarcomas have been observed. The eight circular points show the observed zero values for each intake interval; the triangles indicate where each point would have been plotted had one bone sarcoma been observed in the intake interval. Two functions are plotted: the lower solid curve is the dose-squared exponential fit; the upper solid curve is the limiting value of the LQE function ($\alpha = 1.3 \times 10^{-4}$, $p = 0.05$). If an LQE function is a proper description of the dose response relationship, the true relationship would be expected to lie within the shaded area.

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"The analysis based on first exposure to radium, wherein the biases present might be expected to over-emphasize the hazards of radium, contradicts the generally accepted linear relationship between insult and effect."

Radium burden Population/Bone and Head Cancer - Maletskos 1994

Maletskos, 1994 reports that, "Over the next five or six decades, about 2000 subjects were investigated. R.D. Evans (1974) plotted the Massachusetts Institute of Technology results in terms of incidence of cancer (osteogenic sarcomas and carcinomas of the paranasal and mastoid sinuses) per cumulative average skeletal dose interval. (See Evans 1974, Fig. 3, p. 4-1) The curve was a step function with incidence at zero in the first three decades of 0 to 10 Gy and essentially constant at 28% beyond the 10 Gy up to 500 Gy. Evans joined the two horizontal lines with a narrow, S-shaped dotted curve to indicate a transition between the two incidences without presuming the true shape. In combination with the observation that the latent period for cancer to develop increased as the cumulative dose decreased, Evans proposed a "practical threshold" at a cumulative dose, ~10 Gy, below which cancer would not be observed during a person's lifetime.

"R.E. Rowland (1978) showed the relationship of incidence to cumulative dose was quad-multiplied by an exponential term to explain the peak in the response at high dose due to cell killing. The quadratic response started at zero with no implication of a threshold.

"In all three studies, only Evans faced the fact that a large fraction of the subjects was symptom free below ~10 Gy.

"Recently, some new analyses have been conducted using new approaches that minimize or avoid the biases of grouping by dose intervals, that include the more recent data that have become available, and that, in effect, allow the data to speak for themselves.

"The first new analysis by O.G. Raabe (1990) is based on the linearity between the logarithm of time to radiation-induced cancer death and the logarithm of lifetime average dose rate. By scaling with dimensionless time (expressed as a fraction of life span for each species), results from different animal species and human beings show the same median risk. With the use of a three-dimensional logarithmic representation of the dose rate/time/response relationships, the combined risk of dying from causes due to natural life span, radiation-induced cancer and radiation injury (nonneoplastic injury) can be determined. The carcinogenic portion indicates that the time-to-tumor occurrence is longer as the dose rate decreases and may exceed the natural life span, yielding a threshold of ~1 Gy.

"The second approach by C.J. Maletskos (1992) uses the hazard function in which cumulative dose is used as a surrogate for time, the independent variable. Cumulative hazard is calculated by both a nonparametric method and an analytical method, the former yielding individual values for each subject with cancer and the latter yielding a continuous relation with cumulative dose. The result is a straight line that superposes very well on the points and that intercepts the abscissa at a threshold of ~11 By.

"In the third analysis by R.G. Thomas (1994), the net incidence of cancers per unit dose interval above 10 Gy, below which no cancers are observed, is plotted against cumulative dose. This net distribution (the natural bone sarcoma life-time incidence

having been subtracted) is shown to be strongly lognormal, and the intercept of the curve on the dose scale is finite at a threshold of – 4.7 Gy.

"Three different approaches, without significant restraints, show that the dose responses for exposure to internal radium do not pass through zero and, in fact, predict a threshold of cumulative dose below which cancers are not to be expected."

**Radium-burden Population/Bone and Head Cancer - Thomas 1995**

"Robert G. Thomas reports (1995) that, "Some of the most extensive epidemiological studies of the effects of ionizing radiation in humans have failed to conclude that there are health effects below whole-body equivalent radiation doses of 0.2 Gy (20 rads). This has been demonstrated in the study of survivors of the bombings in Japan; in the cases of radium dial painters (luminizers) studied in the United States, this value is 10 Gy (1000 rads).

"Dose-response data from the 1515 U.S. female workers who were exposed to radium through the painting of luminous dials and who subsequently had their skeletal burdens measured by whole-body counting and radon breath analysis are lognormally distributed. A lognormal analysis for the 65 cases of radium-induced bone sarcomas and head carcinomas allowed calculation of geometric means and standard deviations for segmented dose populations, and these were used for inter-comparisons of dose responses. The analysis of the radiation dose data from the 65 tumor cases indicates an extrapolated dose of at least 4 Gy below which no skeletal tumors could be expected in a lifetime. Only 12% of the female radium dial painters began work after the age of 20 yr. The geometric mean age at beginning of exposure (18 yr) is lower than would be expected today, but these exposures occurred in the 1910s to 1930s. The very strong correlation between latent period and age at death or diagnosis is expected.

"Most analysis of data like that from the radium dial painters use logarithmic axes to express the dose response because the dose range is very large. The data are lognormally distributed, so this makes log-graphic representation more sensible, even though lognormal presentations can be deceiving. The lognormal analysis does not reflect specific biological processes, but it does verify the existence of a previously reported threshold dose response for [226, 228] Ra in humans. The term 'threshold' in this paper refers to that dose below which no skeletal tumors have been reported. Maletskos et al. used hazard function analysis on a similar version of these data and reported a value of 11 Gy as a dose 'below which radiogenic tumors are estimated statistically not to occur, in support of a threshold model.' Evans et al. originally pointed out this no-effect dose as being ~ 10 Gy to the skeleton, and he referred to it as a 'practical threshold.'

"Perhaps more interesting than the cancer cases are those with radium skeletal burdens that never developed a related illness. The message is that there are 1391 female luminizers with average estimated skeletal doses below 10 Gy who have not shown skeletal tumors. This totals to a mean collective dose of about 850 person-Gy, this cohort would have been expected to reveal at least five cancer deaths."

**4.2 Radium-burden Population/All-cause Mortality and Longevity - Kondo 1993**

*Professor Emeritus Dr. Sohei Kondo reports ~ a there is an "apparently beneficial effect of low doses of external gamma rays on the life span of radium-dial painters."*

Professor Emeritus Dr. Sohei Kondo reports (Kondo 1993, Section 4.3) that, "Data on women who painted radium on the dials of watches early in this century are maintained at the Center for Human Radiobiology, Argonne National Laboratory. [Ed. Note: The USDOE has shut down this program and efforts to analyze and report on the health effects of this significant radiologically exposed population, of which many hundreds of subjects remain alive.] After removing 62 cases of malignancies known to have been induced by internally deposited radium, Rowland et al. (1989) surveyed the health status of the remaining 1261 cases. They were classified into three subgroups by the absorbed dose of radium gamma rays as shown in Table 4.12. No dose-dependent increase in deaths from various cancers was seen among the three groups exposed to mean doses of 2.9, 23 and 91 rad by chronic gamma irradiation (Table 4.12)."

**Table 4.12 Numbers and rates of cancer deaths among 1261 white female radium-dial painters in the USA classified into three dose groups**

| Dose (rad) | | No. of subjects | Total person-years | No. of cancer deaths (rate/$10^4$ person years) | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Range | Mean | | | Total | Breast | Colon | Ovary | Lung | Pan-creas | Sto-mach |
| 0-10 | 2.9 | 860 | 11,731 | 47 (4.0) | 16 (1.4) | 9 (0.7) | 5 (0.4) | 4 (0.3) | 3 (0.3) | 2 (0.2) |
| 10-50 | 23 | 302 | 4,956 | 38 (7.7) | 8 (1.6) | 7 (1.4) | 3 (0.6) | 5 (1.0) | 1 (0.2) | 3 (0.6) |
| >50 | 91 | 99 | 1,709 | 9 (5.3) | 2 (1.2) | 0 | 1 (0.6) | 1 (0.6) | 0 | 0 |

From Rowland *et al.* (1989). Copyright © British Institute of Radiology, London. Reproduced with permission

"When the numbers of deaths from different causes in the radium-dial painters are compared with those in the control group, the observed: expected ratio for deaths from all causes is 0.88 ($p < 0.05$ (Table 4.13). This means that the study group of radium-dial painters, which excluded the workers died of cancer due to internally deposited radium, showed significant reduction in the mortality from all causes compared with the control group."

**Table 4.13 Observed and expected numbers of deaths from selected causes among white female radium-dial painters in the USA**

| Cause | Observed | Expected | Ratio |
|---|---|---|---|
| All causes | 337 | 382.3 | 0.88* |
| All malignant neoplasms | 94 | 84.7 | 1.11 |
| Buccal cavity and pharynx | 1 | 1.2 | 0.83 |
| Digestive organs and peritoneum | 29 | 25.9 | 1.12 |
| Respiratory system | 11 | 8.9 | 1.24 |
| Bone | 1 | 1.3 | 0.77 |
| Breast | 28 | 16.0 | 1.62* |
| Genitourinary system | 10 | 14.9 | 0.67 |
| Other and unspecified sites | 6 | 11.5 | 0.52 |
| Lymphatic and hematopoietic tissue | 10 | 7.0 | 1.32 |
| All benign neoplasms | 2 | 1.1 | 1.83 |
| All circulatory system disease | 168 | 222.6 | 0.75** |
| Ischemic heart disease | 107 | 137.1 | 0.78* |
| Cerebrovascular disease | 24 | 50.0 | 0.48** |
| All respiratory system disease | 16 | 17.5 | 0.91 |
| All digestive system disease | 9 | 13.1 | 0.69 |
| All external causes | 8 | 11.3 | 0.71 |

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*, $p < 0.05$; **, $p < 0.01$ in the chi-square test

"A similar survey of 1203 radium luminizers in the UK were carried out by Baverstock and Papworth (1989), who also found significantly lower rates of death from all causes except cancer among luminizers than among controls (Table 4.14). No death from leukemia was observed among the British luminizers, although at least one case of leukemia would have been expected on the basis of the usual incidence rate."

**Table 4.14 Observed (O) and expected (E) numbers of deaths (1960-85) from selected causes among female radium luminizers in the UK**

| Cause | Observed | Expected | Ratio | p |
|---|---|---|---|---|
| All causes of death | 243 | 268.74 | 0.90 | 0.106 |
| All causes except cancer | 148 | 182.75 | 0.81 | 0.01 |
| All cancers | 95 | 85.99 | 1.10 | 0.331 |
| All cancers except breast cancer | 67 | 65.49 | 1.02 | 0.853 |
| Breast cancer | 28 | 20.50 | 1.37 | 0.097 |
| Leukemia | 0 | 1.93 | 0 | 0.276 |
| Osteosarcoma | 1 | 0.17 | 5.83 | 0.158 |

From Baverstock and Papworth (1989). Copyright © British Institute of Radiology, London. Reproduced with permission

"Mortality from breast cancer was significantly higher among radium painters in the USA (Table 4.13); however, the excess of breast cancer in the USA cannot be attributed to radiation, as no dose-dependent increase in the incidence of breast cancer was observed in three subgroups classified by exposure dose (Table 4.12). The number of deaths from breast cancer in British luminizers (Table 4.14) was also higher than the control level, although the increase is not statistically significant. This excess may in fact be real, however, because the observed:expected ratio of deaths from breast cancer steadily increased with time since first exposure and reached a maximum of 2.12 ($p < 0.023$) at 30-40 years after first exposure (Baverstock and Papworth, 1989). This temporal trend is different from that in the incidence of various types of solid tumors in survivors of exposure to atomic bomb radiation.

"Furthermore, no dose-dependent increase in the observed:expected ratio was seen. The ratios are 1.67 and 1.51 for young women (<30 years at the start of luminizing work) with low (<20 rad) and high (>20 rad) exposure, respectively, whereas these values are 2.09 and 0.45 for older women (>30 years at the start of luminizing work) (Baverstock and Papworth 1989). The observed excess of breast cancer in luminizers therefore cannot

be attributed to radiation. Hence, 50-year follow-up studies of US and UK radium luminizers suggest but fail to provide positive evidence [illegible] low doses of radiation cause breast cancer." [Ed. Note. The luminizers also worked with highly radioactive luminous paint compounds at their studio work-benches, providing significant, unmonitored, direct external radiation to the chest, head and neck areas, contributing to the significant dose received by these women relative to what is normally considered "low-dose" radiation exposure.]

"Table 4.15 summarizes the temporal trend after exposure in mortality of British radium luminizers who had worked for two or more years. In the first 20 years after exposure, the ratio of observed:expected numbers of non-cancer deaths was 0.31-0.47, indicating a beneficial effect of radiation on the life span of the workers. The ratio gradually increased thereafter, indicating diminution of the benefit of radiation with time, reaching 1.02 (disappearance of benefit) 40-50 years after exposure.

"The temporal trend in reduction of mortality in the luminizers (Table 4.15) is reminiscent of the observation that mortality from non-cancer deaths among atomic bomb survivors exposed to low to intermediate doses of radiation was reduced in the early period after the bombings, and thereafter gradually increased with time [See 1.2 Kondo 1993 Table I]. The observed:expected ratio for non-cancer deaths among women who worked for less than two years as luminizers, however, was 2.21, with p value of 0.004 for the period 0-10 years after first exposure although the overall ratio for the period 0-50 years after first exposure for this group is close to one (Baverstock and Papworth, 1989), an indication that the women had a shortened their life span during the first 10 years after first exposure. If this effect was due to radiation, we must conclude that a short period (<2 years) of exposure to radiation has harmful effects, whereas long periods (>2 years) has a beneficial effect (see discussion of Table 4.15). This conclusion is, however, hardly compatible with data of bomb survivors (See 1.2 Kondo 1993 Table I). My interpretation is that most of the women in the group that worked for <2 years retired from luminizing work due to illness shortly after they started and that a considerable number of them died within 10 years as a result of progression of their illness."

**Table 4.15 Observed (O) and expected (E) numbers of deaths from causes other than cancer in women working as luminizers for ≥ 2 years**

| Years since entry | Observed | Expected | Ratio | *p* (two-tailed) |
|---|---|---|---|---|
| 0-10 | 4 | 13.02 | 0.31 | 0.008 |
| 10-20 | 6 | 12.70 | 0.47 | 0.066 |
| 20-30 | 20 | 25.64 | 0.78 | 0.32 |
| 30-40 | 32 | 44.86 | 0.71 | 0.06 |
| 40-50 | 25 | 24.46 | 1.02 | 0.84 |
| 0-50 | 87 | 120.68 | 0.72 | 0.001 |

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"The British radium luminizer population is unique in that the women worked for a limited period under fairly uniform conditions of exposure at low external gamma dose rates (5-20 rad/year), resulting in accumulated doses of up to more than 100 rad (average, 40 rad) and 80% of them are still alive (Baverstock and Papworth, 1989). Radium-dial painters are an irreplaceable resource for elucidating important questions about the risk of low-level radiation. I hope that the follow-up surveys on the UK and USA study populations will be continued."

[Ed. Note: Such programs and this significant data and analysis have been terminated in the USA., while >$100 million are being expended on analysis of the populations around US weapons facilities sites for which small indeterminate doses to individuals and no possible dose response association can be identified, while fostering public fear.]

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**Radium-burden Population/All-cause Mortality and Longevity - Spiers (1983)**

F.W. Spiers reports (Spiers 1983) that, "The number of persons exposes to radium is known to be large; some 3500 persons have now been located and the radium burdens of more than 2000 have been measured. In this context it is clearly important to consider malignancies of the bone marrow both because as much as one third of the skeletal marrow can be irradiated by α particles arising from radium in the trabeculae (F.W. Spiers 1974) and because so little information is available on the induction of leukaemia by α-particle irradiation of human bone marrow.

"In this report the number of persons with leukaemia has been identified in a defined population of people exposed to radium and comparisons have been made (a) with the number expected in a comparable population of the same size and age distribution and (b) with predictions based on the risk factor and α-particle Quality Factor proposed for protection purposes by the ICRP (International Commission on Radiological Protection 1977) and the estimated bone marrow doses.

"The pre-1930 cases were chosen for analysis because in the later cohorts there were"

no leukaemia cases recorded and only 0.25 expected. These data are presented in Table 5

*Table 5. Leukaemia: observed and predicted by dose*

| Skeletal Dose* Range (rad) (1) | Mean (rad) (2) | Marrow** Dose (rad) (3) | Nos. of Persons (4) | Predicted Nos. of Cases Radiation (5) | Natural (6) | Observed Cases (7) |
|---|---|---|---|---|---|---|
| <10 | 1.7 | 0.09 | 230 | 0.003 | 0.60 | 0 |
| 10 - 24 | 16.6 | 0.86 | 109 | 0.016 | 0.37 | 0 |
| 25 - 49 | 35.9 | 1.86 | 77 | 0.031 | 0.53 | 0 |
| 50 - 99 | 68.4 | 3.54 | 73 | 0.030 | 0.32 | 0 |
| 100 - 249 | 166.4 | 8.61 | 73 | 0.093 | 0.32 | 0 |
| 250 - 499 | 357.9 | 18.53 | 61 | 0.112 | 0.16 | 1 |
| 500 - 999 | 755.2 | 39.09 | 33 | 0.127 | 0.05 | 0 |
| 1000 - 2499 | 1597.0 | 82.67 | 24 | 0.294 | 0.07 | 1 |
| 2500 - 4999 | 3509 | 185.9 | 25 | 0.680 | 0.07 | 0 |
| 5000 - 9999 | 6552 | 339.3 | 16 | 0.803 | 0.05 | 0 |
| >10,000 | 10966 | 981.9 | 3 | 0.436 | 0.01 | 0 |
| | Totals | | 693 | 3.63 | 3.05 | 2 |

*Based on 5 kg skeletal bone mass as recorded in the file.

**Calculated on basis of 3.4 kg for female skeletal bone mass (ICRP74).

"The sum of the cases expected naturally plus those calculated from the bone marrow irradiation is 4.7 against the 2 cases observed in the study population and this difference is only marginally significant, $p<0.1$.

"Some of the assumptions which were required for the above analysis do not have to be made for the group of 1285 located female radium workers who were exposed before 1930. This group is not totally suitable, in that it may be biased. The follow-up, however, is from the year of first exposure to death or to the end of 1979. The follow-up period is of the order of 60yr so that life-time risks can be used. In this group 4 cases of leukaemia were observed and 5.44 cases were expected in the comparable population. It is a reasonable assumption that the magnitudes and distribution of the α-particle doses were at lest approximately the same as those given in Table 5 for the 694 persons whose radium burdens were measured. On this basis it can be shown that about 13 cases of radiation-induced leukaemia could be expected in a follow-up period of 60yr (chronic lymphocytic and chronic lymphatic excluded). That is, on the assumption of a Quality Factor of 20 for α-particle irradiation, the total of natural plus radiation-induced leukaemias in 1285 persons would be expected to be about 18, as against 4 observed.

"It does not appear that the low incidence of leukaemia in this group of 1285 workers can be accounted for by the combination of a high, Quality Factor and a low value of initial dose.

*"Low Let Radiation*

"Two sources of low LET radiation can further irradiate the bone marrow of the radium dial workers. They are (1) β-particle radiation from radium daughters incorporated in bone and (2) external γ radiation from the luminizing paint being used by the dial worker herself and by those surrounding her in the work room. The risks from these two sources add to those calculated for the α-particle radiation and must be evaluated in relation to the α-particle risk.

*"The γ Irradiation*

"The dose to dial workers in a workroom from the γ radiation from the luminous paint has been variously estimated by different workers. Finkel et al. (A.J. Finkel 1969) gave 4 rad/yr for a mean body exposure dose rate; Polednak (A.P. Polednak 1980) estimated 4.8 rad/yr as the ovary dose, corresponding to about 6 rad/yr to bone marrow; Baverstock et al. (K.F. Baverstock 1981) have given dose rates based on film badge measurements on British luminisers which would be equivalent to a range of bone marrow dose rates from 3 to 13 rad/yr depending on the years when working between 1943 and 1952. We have taken an arbitrary figure of 8 rad/yr to bone marrow as sufficiently typical for U.S.A. workers. Taking this figure (as the bone marrow dose) and using the average duration of employment for the female dial workers of 145 weeks, the γ-ray dose amounts to 22 rad.

*"Conclusions*

"Among the total number, 2940, of located radium dial workers, 10 cases of leukaemia were observed and in a group of this size and age distribution 9.24 cases would be expected in the general population. This does not suggest a significant number of cases induced by the radiation exposure and bears out the earlier opinions that leukaemia was not an outstanding feature in the radium cases.

"In the smaller group of 693 dial painters for whom it was thought worthwhile to carry out an analysis by dose group, 2 cases were observed against 2.04 expected naturally. On the basis of the α-particle doses to bone marrow and the risk factor suggested by the ICRP (International Commission on Radiological Protection 1977) it can be calculated that some 2.63 cases would be expected in the exposed population additional to the natural incidence. If the same analysis is applied to the total number of 1285 located female radium dial workers followed up for 60 yr, some 13 cases of radiation-induced leukemia would be expected additional to 5.4 cases expected naturally, that is a total of about 18 as against 4 cases observed."

## 5.0 Weapons/Facility Releases

### 5.1 Weapons/Facilty Releases/ "Atomic Veterans" - Yalow 1994

*Weapons tests participants*

Nobel Laureate Dr. Rosalyn Yalow states (1994) that, "The National Academy of Sciences National Research Council analyzed 46,186 nuclear weapons-test participants, showing 10 leukemia-related deaths (3.97 expected) among the 1957 Operation Smoky participants. However, only 1 of those 10 was exposed to more than 3 rem. There wereno increases in other cancers. Conversely, three thousand 1951 Operation Greenhouse participants had 1 leukemia death (4.43 expected). These are typical statistical variations with small numbers. Of all weapons-test participants, there is no excess cancer."

### 5.2 Weapons/Facility Releases/Bikini Bomb Test - Kondo 1993

Professor Emeritus Dr. Sohei Kondo (1993) reports that, "On 1 March 1954, a hydrogen bomb test was performed on Bikini Island, and 23 Japanese fishermen, 18-39 years old, were exposed to 'lethal' radioactive fall-out (about 1 mCi/g by a crude estimate). A brief description of the event and its effects is given here because these men can be regarded as having received a level of radiation intermediate between that of the Hiroshima-Nagasaki atomic bombings and that of the Chernobyl accident (see Kumatori et al., 1980, for details).

"Estimates of whole-body doses of gamma rays from the external fall-out, which were received during the first two weeks up to 14 March when they returned to Japan, were 200-295 rad for 11 fishermen, 325-395 rad for five, 415-475 for three, 545-575 rad for three and 670 rad for one. The acute effects of chronic irradiation at these doses were estimated to be approximately equivalent to those of a single, acute irradiation with half of the doses, i.e., total acute doses of 80-320 rad. Additional doses of radiation in the thyroid, on the basis of radioactive iodine nuclides incorporated, were estimated to be 230-550 rad.

"One fisherman with hematological disturbances (anemia, leukopenia and thrombopenia) and hepatitis died 206 days after the accident, and one with ascites caused by cirrhosis died 21 years later. No malignant disease has been observed in the remaining men. Follow-up studies of peripheral leukocytes and platelets from the exposed fishermen were carried out from the time they returned to Japan. Average leukocyte and platelet counts were markedly depressed for about two years after the exposure, recovered gradually, reaching normal levels two to five years after the exposure, and then showed several excesses for about two years thereafter. More than 20 years after the exposure, the average leukocyte counts were slightly depressed. In two fishermen exposed to high doses, neutrophil counts were depressed continuously for 25 years when compared with the counts on the first day after their return to Japan."

### 5.3 Weapons/Facility Releases/Chernobyl Releases - Jaworowski 1995b

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Zbigniew Jaworowski states (1995b) that "Unexpected results were obtained in one of the best studies in human genetics carried out in Hungary before and after the Chernobyl accident. Several serious congenital anomalies occurred after the Chernobyl accident with lower frequency than before the accident."

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**Weapons/Facility Releases/Chernobyl Releases - Jaworowski 1995a**

Professor Emeritus Dr. Zbigniew Jaworowski states (1995a) that, "The 'no-threshold' arithmetic was also applied to population exposed to the local Chernobyl fallout, and lead to a decision of the Supreme Soviet to evacuate about 200,000 inhabitants of Ukraine and Belarus, which lead to unspeakable sufferings and a loss of many billions of dollars, equivalent of about 1.5% of the General National Product of the former Soviet Union . The intervention level for evacuation was a lifetime (70 years) radiation dose 350 mSv, i.e. a level only about twice as high as the global average natural lifetime dose of 170 mSv. All families with pregnant women and children under age of 12 years were relocated from areas with 137Cs contamination greater than 550 Bq per m2 . 137Cs body burden in children still living in such areas was found to range between 0.04 and 2.25 kBq, which is less than natural amount of 40K in the children's bodies (an adult body carries about 4000 Bq of 40K). Radiocesium body burdens of several thousands Bq are now common in Northern Canada and were as high as 100,000 Bq during weapons tests in the 1960's ."

### 5.4 Weapons/Facility Releases/USSR Releases - Jaworowski 1995b

Prof Emeritus Dr. Zbigniew Jaworowski states (1995b) that, "In September 1957, inhabitants of 22 villages in the Eastern Urals were irradiated with high radiation doses of up to 1500 mSv, the result of the radioactivity release from thermal explosion in a

Soviet military nuclear facility. About 10,000 people were evacuated and their cancer mortality was studied during the next 30 years.

"From this group, 7,852 of the persons studied were divided into three exposure groups: those who received average doses of 496, 120, and 40 mSv. Tumor related morality in the 496 mSv group was 28% lower than in the nonirradiated control population from the same region; in the 120 mSv group it was 39% lower, and in the 40 mSv group it was 27% lower. In the first two groups the difference from the controls was statistically significant (Kostyuchenko and Krestinina, 1994)."

## 6.0 Natural Background

Dr. Alan Brodsky reports (1996) that, "UNSCEAR reports are considered scientifically authoritative, and are often used by nations in developing their own radiation protection standards. A recent article by two United States representatives to UNSCEAR and other U.S. scientists provides a concise summary of the conclusions of the 1986 and 1988 UNSCEAR reports (Mettler et *al.* 1990; UNSCEAR 1986, 1988).

"UNSCEAR estimates the annual average effective dose equivalent per person in the world population to be 3 mSv (300 millirem). Most of this (2.4 mSv) comes from natural radiation that has always been in the environment, and 0.4 to 1 mSv is attributable to medical exposures. Other sources contribute less than 0.02 mSv (2 millirem) annually. The worldwide collective effective dose equivalent annually is between 13 and 16 million person-Sv (1.3 to 1.6 billion personrem). Table 2-43 shows the estimated annual effective dose equivalents from *natural* sources, as given in the 1988 report (UNSCEAR 1988).

"While many of the natural sources and doses vary among individuals and populations, independent of human activity (such as cosmic rays), some depend strongly on human activities. The most obvious source of natural radiation exposure that depends on human activity is the exposure to radon and thoron and their decay products. The locations of home and building construction, the amounts of ventilation provided, and the types of construction, all affect greatly the amounts of radon and thoron exposure to individuals. The estimated average annual natural radiation effective dose -- the world population is seen from Table 2-43, the current (240 mrem) average of 2.4 mSv.

"In the 1988 report, the external estimate from cosmic radiation has been increased by about 0.05 mSv (5 mrem), as a result of taking geographical distribution, as well as altitude distribution, into account. This cosmic ray component of natural exposure, as is well known, can be raised by a factor of two or more above the average of 35.5 millirem shown in Table 2-43 simply as a result of moving to a high altitude such as that in Denver, Colorado."

*Table 2-43. UNSCEAR 1988 annual effective dose equivalents from natural radiation, averaged for the world population (from Mettler et al. 1990, Table 1)*

**Annual effective dose equivalent from natural sources**

| Source of irradiation | Annual effective dose equivalent (mSv) | | |
|---|---|---|---|
| | *External* | *Internal* | *Total* |
| Cosmic rays: | | | |
| Directly ionizing component | 0.30 | — | 0.30 |
| Neutron component | 0.015 | — | 0.015 |
| Cosmogenic radionuclides | — | 0.015 | 0.015 |
| Primordial radionuclides | | | |
| Potassium-40 | 15 | 0.18 | 0.33 |
| Rubidium-87 | — | 0.006 | 0.006 |
| Uranium-238 series | 0.1 | 1.24 | 1.34 |
| Thorium-232 series | 0.16 | 0.18 | 0.34 |
| TOTAL | 0.8 | 1.6 | 2.4 |

In Table 2-44, the total natural background exposure rate is compared with other sources of manmade exposures.

*Table 2-44. UNSCEAR 1988 estimates of average annual effective dose equivalents from manmade compared to natural sources (from Mettler et al. 1990, Table 3)*

| Source or practice | Present annual individual doses (mSv) | | Collective dose commitments | |
|---|---|---|---|---|
| | *Per person (world population)* | *Typical range (for exposed individuals)* | *Million man-Sv* | *Equivalent years of background* |
| ANNUAL | | | *Per year of practice* | |
| Natural background | 2.4 | 1-5 | 11 | 1 |
| Medical exposures (diagnostic) | 0.4-1 | 0.1-10 | 2.5 | 0.2-0.5 |
| Occupational exposure | 0.002 | 0.3-5 | 0.01 | 0.001 |
| Nuclear power production | 0.0002 | 0.001-0.1 | 0.001 | 0.0001 |
| | | | (0.03)* | (0.004)* |
| SINGLE | | | *Per total practice* | |
| All test explosions together | 0.01 | 0.01 | 5 | 0.5 |
| | | | (76)* | (2.4)* |
| Nuclear accidents | | | 0.6 | |

* The additional long term collective dose commitment from Rn and °C for nuclear power production and °C for test explosions are given in parentheses

"The Chemobyl accident has been estimated in an appendix of the 1988 UNSCEAR report to produce a collective dose equivalent of 0.6 million personSv (60 million person-rem), mostly in the former Soviet States and Europe. Thirty percent of this collective dose has been delivered in the first year following this 1986 accident, and the remainder will be delivered in tens of years after the accident. This collective dose (in the first year) is about 2 percent of the annual natural background collective dose to the world population.

"The UNSCEAR 1988 report also presents (Table 2, Mettler et al 1990) collective effective doses from nuclear energy generation industries as integrated over 100 years, and over all time, the 24 person-Sv per GW over the next 100 years capacity of 500 GW estimated for the year 2000, would amount to a collective dose of 12,000 person-Sv (1.2 million person-rem), compared to the natural background integrated over 100 years, which would be about 16 million person-Sv (1.6 billion person-rem) per year times 100 years, or 1.6 billion person-Sv (160 billion person-rem) over 100 years."

"Thus, the average annual exposure per person from nuclear power production is shown in Table 2-44 to be only 0.0002 mSv per year (0.02 millirem per year). For comparison, the BEIR V report estimates that, averaged over the United States population, the natural background exposure is 3.0 mSv per year, 2.0 mSv of which is from radon. The annual medical diagnostic exposure is 0.39 mSv, the nuclear medicine exposure is 0. 14 mSv, consumer product exposure is 0. 10 mSv, the nuclear fuel cycle exposure is <0.01 mSv, and occupational exposure is (averaged over the total population) <0.01 Sv. The total of natural and artificial (manmade) exposure in the United States is estimated to be 3.6 mSv (360 mfilirem) per person (NAS 1990, pp. 18-19)."

### 6.1 Natural Background/U.S. States - Yalow 1994a

Nobel Laureate Dr. Rosalyn Yalow states (1994) a "U.S. average radiation dose of 0.1 rem/yr (ie, 5 rem/50 yr), not including radon, varies up to 10-fold locally.

"The seven Colorado plateau states have doses about twice the US average. Mean cancer death rates average 15% less than US rates (considering complicating factors). This does not prove a protective effect of radiation exposure, but the opposite result would cause some to unequivacaly declare radiation the cause..."

### 6.2 Natural Background/China - Kondo 1993

Professor Emeritus Dr. Sohei Kondo (Kondo 1993) notes that "in modern, civilized countries, citizens' health is in principle taken care of by a ministry of public health, or its equivalent, in the government of each country. If low-level radiation really presents a risk to human health, therefore, it will be handled by the ministry of public health. This is not the case, however, in most countries of the world.

"A notable exception is China. In 1972, the Chinese Government supported a national project on research into high background radiation to conduct studies on the health of people living in Tongyou and Donganling areas of Yangjiang County, Guangdong Province, in southern China, where the natural level of radiation is about three times higher than the world average. The high-background areas are located along a river system from Mt. Donganling and Mt. Ezhang and have the total area of 540 km^2, including 463 villages and the population of about 80,000. As the control areas, Wudianmeihua area in Enping County and Sanhe area in Taishan County, were selected; these areas have normal level of radiation. The locations of the high-background radiation areas and the control areas are shown in Figure 4.1."


**Fig. 4.1 Locations of areas in the follow-up study conducted by the High Background Radiation Research Group in China**
Tongyou and Dong-anling regions (dotted) in Yangjiang County, are the areas of high-background radiation. The two control areas are located in Enping and Taishan counties (striped) (from Wei et al., 1990, with minor modification)

"Locations of areas in the follow-up study conducted by the High Background Radiation Research Group in China Tongyou and Dong-anling regions (dotted) in Yangjiang County, are the areas of high-background radiation; the two control areas are cited in Enping and Taishan counties (striped). (from Wei et al., 1990, with minor modification) In the first stage of the health surveys, from 1970 to 1978, a retrospective method was used whereas, since 1979, follow-up studies of residents in the two areas have been carried out using a card enrollment system.

"Recent results have been summarized in a review article by Wei Luxin et al. (1990).

"Recently, researchers from the US National Institutes of Health have been participating in cooperative work on this and other health surveys of Chinese residents."

"The average annual absorbed doses from external gamma rays in the high-background areas is about 0.21 rad, whereas that in the control areas is 0.08 rad (Fig. 4.4)."

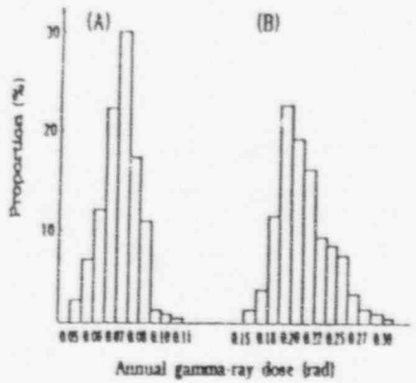


**Fig. 4.4 Distribution of absorbed doses of natural gamma rays received by inhabitants in control (A) and high-background (B) areas of radiation in southern China**
From Wei *et al.*, 1990

"If we add the doses of internal radiation from potassium-40, radon-222 and its decay products and others, the annual effective dose equivalents become 0.55 and 0.21 rem, respectively, in the high-background and control areas.

"The cumulative effective dose equivalents of residents with age of 70 years in the high-background areas are 35-53 rem, with an average of 38 rem. Therefore, if the life-time dose limit of 35 rem adopted by the ex-USSR as the intervention level for evacuation (see Section 2.2) were to be applied in China, the entire area of high-background radiation in Yangjiang County would be evacuated immediately. Chinese radiation experts have not taken such measures, however, as they have obtained epidemiological evidence that the natural radiation in this area is not harmful to residents."

"Cancer mortality is lower in areas of high background radiation than in control areas."

"Between 1970 and 1986, 74,000 people were studied in the high-background areas and 77,000 in the control areas. One of the most important characteristics of these populations is their stability; most people have lived in the same place for more than five generations"

"The two areas have similar geographical characteristics: the altitude in the high-background areas is 20-50 m and that in the control areas, 15-25 m above sea-level. Habits, customs and living conditions are very similar. The educational level is fairly similar, although that in the control areas is slightly higher. Medical care does not differ in the two areas; on average, there are one medical specialist and two county doctors for every 1000 inhabitants. Both areas are rural: 94 and 93% of inhabitants in the high-background and the control areas, respectively, are peasants.

"For the purposes of the long-term health survey, the HBRR group established the following principles for selecting the study population: of Han nationality; belong to families who have lived in the area for more than 40 years; their parents or grandparents are not closely related; and they are not occupationally exposed to ionizing radiation.

"As shown in Table 4.3, there were 467 deaths from cancer in the high-background areas in 1970-86 for about 1 million person-years, with an adjusted mortality rate of 48.8/105, and 533 in the control areas for 995,000 person-years, with an adjusted mortality rate of 51.1/105. The difference is not significant."

**Table 4.3 Site-specific cancer mortality (per $10^5$ person-years)[a], 1970–86, in high-background and control areas of radiation in China**

| Cancer site | High-background areas | | | Control areas | | |
|---|---|---|---|---|---|---|
| | No. | Mortality rate | | No. | Mortality rate | |
| | | Average | Adjusted[b] | | Average | Adjusted[b] |
| Nasopharynx | 94 | 9.32 | 9.84 | 109 | 10.95 | 10.45 |
| Esophagus | 13 | 1.29 | 1.40 | 16 | 1.61 | 1.49 |
| Stomach | 53 | 5.25 | 5.60 | 47 | 4.72 | 4.44 |
| Liver | 115 | 11.40 | 12.05 | 145 | 14.57 | 13.92 |
| Intestine | 16 | 1.59 | 1.70 | 25 | 2.51 | 2.38 |
| Lung | 25 | 2.48 | 2.65 | 35 | 3.52 | 3.29 |
| Breast | 7 | 0.69 | 0.75 | 13 | 1.31 | 1.25 |
| Cervix uterus | 13 | 1.29 | 1.37* | 5 | 0.50 | 0.45 |
| Leukemia | 31 | 3.07 | 3.02 | 33 | 3.32 | 3.39 |
| Osteosarcoma | 5 | 0.50 | 0.52 | 6 | 0.60 | 0.59 |
| Others | 95 | 9.42 | 9.91 | 99 | 9.95 | 9.44 |
| Total | 467 | 46.29 | 48.81 | 533 | 53.56 | 51.09 |

From Wei *et al.* (1990), with permission
[a] For the period 1970–86, 1,008,769 person-years observed in the high background areas and 995,070 person-years in the control areas
[b] Adjusted to the combined population in the high-background and control areas
*$p < 0.05$

"However, the cancer mortality rate becomes significantly lower in the high-background areas than in the control areas, if we compare deaths from cancers other than leukemia among people over the age of 40 years to see the possible effects of differential cumulative doses of radiation (Table 4.4)."

**Table 4.4 Annual rates (1970–86) of mortality from all cancers except leukemia among inhabitants aged 40–70 years in high-background and control areas of radiation in China**

| Area | Person-years observed | Mortality | | ß-value (%) (95% CI)[a] | *p* |
|---|---|---|---|---|---|
| | | No. | Rate (per $10^5$) | | |
| High-background (HB) | 207,900 | 299 | 144 | -14.6 (-24.8, -3.0) | 0.04 |
| Control (CA) | 224,380 | 377 | 168 | | |

From Wei *et al.* (1990), with permission
[a] The AMFIT computer program (Preston, 1987) was used to fit the Poisson regression model, $R_{HB}(S,T,A) = R_{CA}(S,T)(1 + ßA)$, where $R_{HB}$ and $R_{CA}$ are mortality rates in HB and CA, respectively, S is sex, T is age, A is area, 0 represents CA, 1 represents HB and ß is the 'excess' rate in HB over CA; CI, confidence interval

"Leukemia is the most sensitive type of cancer to induction by radiation. Furthermore, most of the cases reported in Table 4.3 were diagnosed on the basis of histopathological evidence. As shown in Table 4.5, the incidence of deaths from leukemia in the high-background areas was within the range of variation of the spontaneous leukemia incidence in neighboring countries of Asia."

**Table 4.5 Rates of mortality from malignant neoplasms and leukemia in people of all ages in some Asian countries or areas (per 100,000 population)**

| Country or area | Malignant neoplasms | | Leukemia | |
|---|---|---|---|---|
| | Males | Females | Males | Females |
| Hong Kong (1986)[a] | 173 | 117 | 3.4 | 3.3 |
| Japan (1986)[a] | 191 | 127 | 5.2 | 3.7 |
| Korea (Republic of) (1985)[a] | 95 | 55 | 2.9 | 2.4 |
| Singapore (1986)[a] | 128 | 95 | 3.1 | 3.2 |
| Sri Lanka (1982)[a] | 25 | 24 | – | – |
| China[b] | 84 | 63 | 2.8 | 2.2 |
| High-background areas[b] | 88 | 35 | 3.2 | 2.9 |
| Control areas[b] | 65 | 41 | 3.6 | 3.1 |

From Wei *et al.* (1990), with permission
[a] From World Health Organization (1987)
[b] The two areas surveyed by the High Background Radiation Research group

"The incidences of thyroid diseases in high-background and control areas of radiation were compared by examining 1,000 women aged 50-65 years from each area. The estimated average cumulative doses to the thyroid were 14 rad for residents of the high-background areas and 5 rad in the control areas. Each woman was interviewed by a trained Chinese interviewer to obtain information on relevant medications, medical and reproductive histories, specific symptoms relevant to thyroid function, smoking habits, diagnostic and therapeutic x-ray procedures and diet. Physical examinations of the thyroid were conducted by three US thyroidologists who were unaware of the exposure status of the women.

"For all nodular diseases, the prevalence was 9.5% in the high-background areas and 9.3% in the control areas. There was no significant difference between the two areas in the prevalence of any type of abnormality of the thyroid, indicating that continuous exposure to several times the normal level of natural radiation throughout life is unlikely to increase appreciably the risk for thyroid cancer."

"It should be noted, however, that chromosomal aberrations were significantly more frequent in peripheral blood from elderly women in the high-background areas than for women of the same ages in the control areas. Thus, chromosomal aberrations may be a good monitor of the dose of low-level radiation but they do not necessarily reflect the occurrence of overt disease."

**Natural Background/China - Luckey 1995**

*No excess cancer found in a Chinese population with a three times higher background radiation.*

Professor Emeritus Dr. T.D. Luckey (Luckey 1995) finds that, "Wei and Wang compared the health of 77,000 Chinese peasants living in a world average background radiation level, with 73,000 peasants living in a background radiation which was three times higher (Wei, 1995). This study involved 2 500,000 person years. They found the non-leukemia cancer mortality rate of the 40-70 years age group to be statistically lower in peasants living in the high background radiation level than in peasants of the control cohort . . ."

"An earlier summary suggests the background radiation group benefited in several parameters of health (Luckey 1991, 1992). When both populations were compared, cancer mortality rate, lung cancer mortality, and the leukemia mortality were lower in the high-background population, $p = 0.05$. In the high-background population, infertility was lower, $p<0.05$ neonatal mortality was only 76 percent that of the controls, $p = NS$, and life expectancy of people over 40 years old was longer, $p<0.05$."

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**Natural Background/China - Yalow 1994b**

Nobel Laureate Dr. Rosalyn Yalow (1994b) states that, "In China, of 150,000 Han peasants living near each other to six generations, about half receive about three times the radiation of the other half from radioactivity in the soil. Investigations since 1972 for doses and health effects find no discernible differences in the health of these populations. Similar negative results are found in higher background areas in Brazil and India."

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**Natural Background/China - Jaworowski 1995b**

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that, "The best radioepidemiological study at low doses to date has been carried out in China. Between 1070 and 1986, 74,000 people in Yangjiang county, which has a high level of natural background radiation (5.5 mSv per year), were compared to 77,000 people in two adjacent low-background counties (Enping and Taishan, 2.1 mSv per year). In the high-background county, the inhabitants receive a 70-year lifetime dose of 385 mSv, which is higher than the intervention level for evacuation adopted for Chernobyl, and 5.5 times higher than the dose limit proposed in the EPA." Should the Chinese government evacuate Yangjiang county? The epidemiological data show that....in an age group of 10-79 years the general (nonleukemia) cancer mortality was 14.6% lower in the high-background county than in the low-background counties. The leukemia mortality among men was 15% lower and among women 60% lower in Yangjiang (Wei et al., 1990)."

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**Natural Background/China - Jaworowski 1995a**

Professor Emeritus Dr. Zbigniew Jaworowski states (1995a) that, "The question arises: why governments of various countries do not relocate populations living in areas where lifetime dose of natural radiation is higher than 350 mSv. For example, why are people not evacuated from Norway where all country average lifetime dose is 365 mSv [Henriksen, 1988], or from high background regions in India with a lifetime dose of > 2000 mSv [Sunta, 1990] and in Iran with lifetime dose of > 3000 mSv [Sohrabi, 1990]? Perhaps in Iran, for example, the government considered not to follow the ICRP guidelines then it considered the fact that in a house in the city of Ramsar several generations were receiving average individual lifetime doses of natural radiation of 17,000 mSv (240 times more than the current ICRP limit for exposure of members of the public to natural sources of radiation). Yet these individuals show no increased incidence of any disease, and some of them lived to 110 years of age [Sohrabi, 1990]."

## 6.3 Natural Background/Other Nations - Luckey 1995

Professor Emeritus Dr. Don Luckey (Luckey 1995) reports that "whole populations in Kerala, India and several towns in Brazil live in apparent good health with ten times the United States average background radiation level. These populations have not been well studied."

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**Natural Background/Other Nations - Jaworowski 1995a**

Professor Emeritus Dr. Zbigniew Jaworowski states (1995a) that, "The question arises: why governments of various countries do not relocate populations living in areas where lifetime dose of natural radiation is higher than 350 mSv. For example, why are people not evacuated from Norway where all country average lifetime dose is 365 mSv (Henriksen, 1988), or from high background regions in India with a lifetime dose of > 2000 mSv (Sunta, 1990) and in Iran with lifetime dose of > 3000 mSv (Sohrabi, 1990)? Perhaps in Iran, for example, the government considered not to follow the ICRP guidelines then it considered the fact that in a house in the city of Ramsar several generations were receiving average individual lifetime doses of natural radiation of 17,000 mSv (240 times more than the current ICRP limit for exposure of members of the public to natural sources of radiation). Yet these individuals show no increased incidence of any disease, and some of them lived to 110 years of age [Sohrabi, 1990]."

**6.4 Natural Background/Radon and Lung Cancer - Kondo 1993**

**[Figs are earlier version of Cohen]**

*Radon*

Professor Emeritus Dr. Sohei Kondo finds (Kondo 1993, Section 4.2.1) that "Cohen (1990) reported an epidemiological test of the linear no-threshold hypothesis on an accumulated data set covering 411 counties in all the US states except Hawaii, Mississippi and Nebraska, plus the District of Columbia. The age-adjusted mortality rates for lung cancer in 1950-69 for white females and males in the 411 counties in which (radon measurements) were available. In those days, women were not heavy cigarette smokers, spent a large fraction of their lives at home and seldom migrated from one place to another.

"Figure 1 shows a plot of lung cancer mortality rates for females in the 411 counties against the mean indoor levels of radon in the corresponding counties. ...the trend of the ... relation is the same whether we consider a line through medians ... or the regression line. For males also (Fig. 1A, B), the regression line is very close to a line through the medians for each range of radon levels."

"One of the weak points in this analysis is that ecological studies, on which Figure 1 is based, are susceptible to confounding. To study possible confounding effects on lung cancer rates, Cohen (1990) carried out multiple regression analyses of the data Cigarette smoking is the most important cause of lung cancer. Since data on cigarette sales in the USA are available only on a state-wide level, lung cancer rates were compared with the mean radon levels for each state. When cigarette sales per capita were introduced into the regression analysis, the negative slope for dependence on radon levels was essentially enchanged."

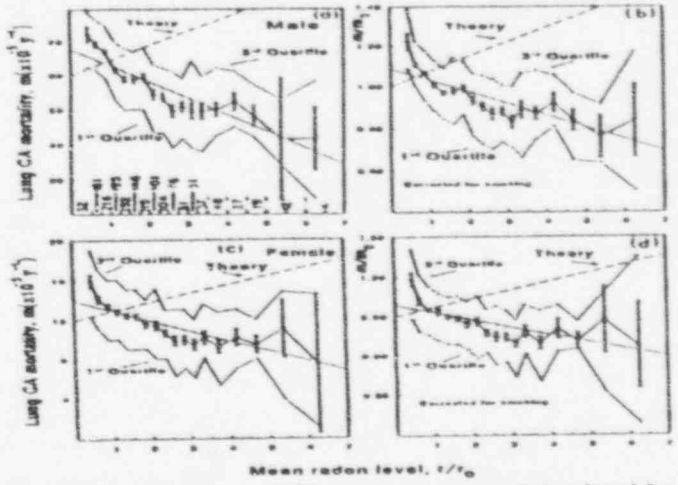


**Fig. 1. Lung cancer mortality rates vs. mean radon level for 1,601 U.S. counties. Data points shown are average of ordinates for all counties within the range of r-values shown on the baseline of Fig. 1a; the number of counties within that range is also shown there. Error bars are standard deviation of the mean, and the first and third quartiles of the distributions are also shown. Fig. 1c, d are lung cancer rates corrected for smoking prevalence ($m/m_0$) vs. radon level [eqns (5) and (6)]. Theory lines are arbitrarily normalized lines increasing at a rate of 7.3%/$r_0$.**

Dr. Kondo reports (Kondo 1993, Section 4.2.3) that, "The negative correlations of home radon levels with lung cancer rates ... are based on ecological studies on groups of people; they can be taken as strong evidence against the validity of non-threshold hypothesis that is adopted for the assessment of radiation risk by the EPA and corresponding agencies in many other countries in the world. The question of whether there is causal relationship between risk of lung cancer and exposure to natural radon cannot, however, be answered by ecological studies because it is conducted on groups of people rather than on individuals. ... (B)ut recently Blot et al. (1990) conducted a case-control study on lung cancer patients in China, measuring radon levels in their dwellings."

problems. The study comprised 308 eligible lung cancer patients, who were female residents of Shenyang in the age range 30-96 years and in whom primary lung cancer had been diagnosed in 1985-87.... (A)ll suspected cases of lung cancer and supporting diagnostic materials were reviewed and classified by an expert panel of pulmonary disease physicians and pathologists. As a control group, 356 healthy female residents of Shenyang were randomly selected to match the age distribution of the cases.

"Radon was measured ... in the houses of patients and controls for one year; two detectors were placed in each house, one in the bedroom and the other in the living room. ...The median levels were 2.8 pCi/L in the houses of patients and 2.9 pCi/L in the houses of control subjects.

"Except for a slight, nonsignificant upward trend for small-cell carcinoma, no evidence of increasing risk with increasing radon level was found. On the contrary, a downward trend in cancer risk with increase in indoor radon levels was seen, which was clearer for adenocarcinoma than for squamous-cell carcinoma. According to Blot et al. (1990), if the no-threshold hypothesis of the BEIR-IV Report (1988) were true, an odds ratio of 1.8 would be found for lung cancer with exposure to a radon level >8 pCi/L in comparison with the level 0.1-1.9 pCi/L; this value is significantly higher than the observed ratio 0.7, with an upper confidence limit of 1.3. The currently adopted no-threshold hypothesis thus overestimates the risk represented by radon."

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**Natural Background/Radon and Lung Cancer - Kondo 1993**

*Professor Emeritus Dr. Sohei Kondo reports that there is a "negative association between lung cancer rates and indoor radon levels in the United Kingdom"*

Professor Emeritus Dr. Sohei Kondo reports (Kondo 1993, Section 4.2.2) that "Haynes (1988) reported, using aggregate data on counties in England and Wales, a negative association between mean radon concentrations in dwellings and standardized mortality ratios for lung cancer, when regional variations in smoking, diet, social class and population density were controlled.

"The highest of mean domestic radon concentrations were recorded in Cornwall (110 Bq/m3) and Devon (74 Bq/m3); the two counties have, respectively, 8,000 and 5,000 dwellings whose indoor concentrations of radon exceed 400 Bq/m3--a level above which building modifications are recommended to reduce radon gas in the UK. In spite of the high radon levels, the number of annual lung cancer deaths, 1980-83, in those counties was within the range to be expected from relationships not involving radon, as observed in the rest of the country. The observed: expected deaths were 204:213 for males 63:69 in Cornwall, and 496:485 for males and 181:160 for females in Devon (Haynes, 1988)."

Blot et al, 1990, reports that "In a significant study conducted jointly by the Liaoning Public Health and Anti-Epidemic Station, Shenyang, China, and the US National Cancer Institute, in an area with a known high variation in radon and with unusually high lung cancer in women, radon detectors were placed for 1 year in the homes of 308 cases of newly-diagnosed lung cancer cases and 356 suitably matched controls. The median time in residences was 24 years. Median radon was 2.3 pCi/L, with 20% >4 pCi/L. The report shows that the lung cancer "levels were not higher in homes of women who developed lung cancer than in homes of controls, nor did lung cancer risk increase with increasing radon level. The data suggest that projections from surveys of miners exposed to high radon levels may have overestimated the overall risks of lung cancer associated with levels typically seen in homes in this Chinese city."

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**Natural Background/Radon and Lung Cancer - Luckey 1994**

Professor Emertis Dr. Don Luckey reports (1994) that, "There is a strong negative correlation between the radon in homes and lung cancer mortality in males, $p < 0.001$ (Figure 11) (Cohen and Shah, 1991; Cohen, 1992). About 90% of the population of the United States resides in the 1730 counties represented. Cohen obtained comparable data with both males and females. His results were comparable with corrections for smoking."

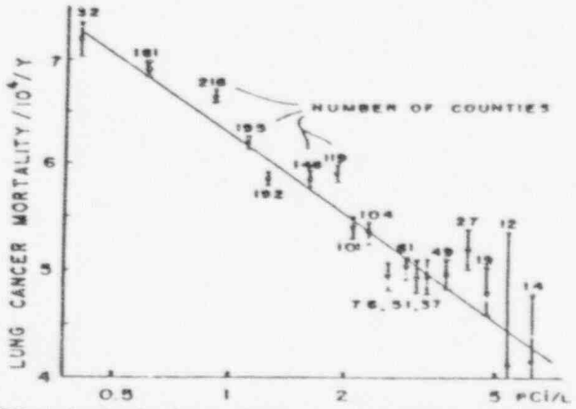


FIGURE 11. Effect of radon levels upon lung cancer mortality in the United States (Cohen and Shah, 1991). About 90% of the population of the United States lives in the 1730 counties surveyed. The number of counties surveyed for each point is indicated near each 5% error bar.

**Natural Background/Radon and Lung Cancer - Luckey 1995**

Professor Emeritus Dr. Don Luckey (Luckey 1995) reports that "radon concentrations in a quarter-million United States homes present another human experience with chronic exposure to different levels of ionizing radiation. The inverse correlation between radon concentrations in United States homes and the incidence of lung cancer mortality has been presented (Cohen 1992) These data were comparable for males and females, with or without corrections for smoking. Since the negative slope of the curve did not change throughout the study, the optimum chronic radon (with progeny) concentration for the reduction of lung cancer mortality appears to be greater than 8 pCi/L.

"As a public health issue, it is pertinent to evaluate the lung cancer deaths predicted by the two different theses. BEIR IV states that there are 350 lung cancer mortalities per million person-WLM (working level month). (BEIR IV 1988). Radiation from one WL is equivalent to 100 pCi radon with progeny. One working month is 170 hours. Half time at home for one month would be twice that long, about 340 hours. Thus, their model suggests home radon would cause 700 lung cancer deaths per million persons at 100 pCi per liter of radon and progeny and 70 per 10 pCi/L. In contrast, the Cohen data indicate that one million people would have 250 fewer lung cancer deaths at 5 pCi/L radon (with progeny) than at 0.5 pCi/L."

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**Natural Background/Radon and Lung Cancer - Pollycove 1994**

Professor Emeritus Myron Pollycove, MD, (1994) reports that, "The BEIR IV report (1988) based upon a linear-no threshold extrapolation or the incidence of lung cancer in uranium mine workers exposed to high radon concentrations, predicts that the lifetime mortality risk of lung cancer is increased linearly by 10.8% per pCiL-1. One pCiL-1 approximates the world average (UNSCEAR 1982) and is equivalent to 0.2 working-level-month (WLM). (NCRP 1984) The American Cancer Society projects for the United States 170,000 new cases of lung cancer in 1993. Accordingly, prior continued home exposure of the population to one additional pCiL-1 of radon would have produced 18,000 additional new cases of lung cancer in 1993. Five-year survival of treated lung cancer is only slightly more than 10%. Relying upon the BEIR IV theoretical prediction, the Environmental Protection Agency (EPA) considers radon in the home to be the nation's leading health hazard.

"However, there is no epidemiologic evidence to support the risks predicted by BEIR IV. To the contrary, epidemiologic studies in the United States (Cohen 1986, 1987, 1989), Sweden (Swedjemark 1984), Finland (Castren et al 1984), and China (Hofman et al 1983) with increased radon concentrations up to 12 pCiL-1, as well as in those areas below the average radon concentration of 1 pCiL-1 (George and Breslin 1980, Nero et al 1983, Wrixon 1984) have all demonstrated a negative correlation of lung cancer with radon concentration. For a variety of reasons, these studies which contradict the linear-no threshold theory have been considered invalid by the National Academy of Sciences Committee on Biological Effects of Ionizing Radiation (BEIR), National Council on Radiation Protection and Measurements (NCRP), and the International Commission on Radiologic Protection (ICRP). Criticisms have included poor statistical power, inadequate controls, and inadequate determination of the degree to which data have been altered by smoking and confounding factors such as numerous socioeconomic variables, geography, altitude, and climate. An extensive University of Pittsburgh National Survey of radon in homes was completed in 1992 that addresses these criticisms with excellent statistical power.

"The University of Pittsburgh nationwide study based upon 272,000 measurements in the homes of 1217 counties was completed in 1992. This study and nine individual state studies were normalized to the EPA National Residential Radon Survey. The combined data set compiled from Pittsburgh, states, and EPA studies includes 1729 counties containing nearly 90% of the U.S. population. After deleting Arizona, California, and Florida, states with high retirement migration, and counties with incomplete data, 1601 counties remain included (Cohen 1994) see Kondo - 1993 p. 6-6. Figure 1 shows plots of mean ageadjusted lung cancer mortality rates (m) for white males (Figure 1a) and females (Figure 1c) vs mean radon levels (r) in homes of all counties within various ranges of r, along with the standard deviation of the mean, first and third quarties, and the best linear fit to' the data for individual counties, m = 1 a(i + br). These mortality rates are corrected for smoking by use of Bureau of Census Population Surveys of smoking prevalence and BEIR IV risk estimates for smokers and nonsmokers, and are shown togetherwith the best linear fit, M = m/m. = A + Br in Figures 1b and 1d. BEIR IV theory lines are normalized lines with slope B increasing mortality at a rate of 7.3%/pCiL-1. After correction for variations in smoking frequency, there is a very strong tendency for lung cancer mortality to decrease with increasing mean radon level in homes, in sharp contrast to the increased mortality expected from the linear-no threshold theory. The discrepancy between theoretical and measured slopes is by 20 standard deviations. An earlier study based upon data for 965 counties furnished additional details'of methodology and somewhat less steep negative slopes of m/m, vs r, with the discrepancy between theoretical and measured slopes by 7 standard deviations.(Cohen and Colditz, 1994)

"Correction for the effects of smoking was made using the separate risk estimates for smokers and nonsmokers given by BEIR IV theory and estimations of the fraction(s) of the adult populations that smoke cigarettes in each county derived from Bureau of Census Surveys, with a correction factor for the fraction of the county population that lives in an urban area. The resultant slopes (B) in units of % per pCiL-1 are -7.3 ± 0.6 SD males and -8.3 ± 0.8 females, discrepant by 20 SD with the slope expected from BEIR IV theory, B = + 7.3. Many other factors in addition to smoking are carefully analyzed to see whether any can explain this discrepancy. Pittsburgh radon measurements are consistent with EPA and state measurements. Potential problems concerning outliers and sampling issues are demonstrated to be absent. Uncertainties in lung cancer mortality rates (m) and smoking prevalence (S) are given elaborate consideration and shown to be unimportant causes of the discrepancy between theoretical and measured slopes.

"A careful investigation was made of the possibility that one or more socioeconomic confounding factors other than smoking could correlate strongly and with opposite signs with both m and r. Those would introduce a strong negative correlation between m and r which would not be due to a direct causal relationship. There are 54 socioeconomic variables (SEV) which are analyzed singly and in combination. The 54 values of B free of confounding by each SEV vary for males from -5.6 to -7.7, mean -6.9 ± 0.5, and for females from -5.4 to -9. 1, mean -7.7 ± 0.8, and are quite close to values for the entire data set -7.3 and -8.3, respectively. Extensive statistical analysis of the possibility that some combination of SEV may act cooperatively to confound the m-r relationship concluded that the actual effect of confounding by combinations of SEV is to reduce the discrepancy between slopes by no more than 10%. Confounding by geography was also analyzed by considering the 34 states with at least 20 counties having known radon levels. The average of B-values is -6.1 for males and -7.2 for females; reductions in the discrepancy by 8.2(Vo and 7.1%, respectively.

"In addition to the 54 SEV and geography, also considered are the possible confounding physical features of altitude, average winter and summer temperatures, inches of precipitation, number of days per year with more than 0.01 inch precipitation, average wind speed, and percent of time with sunshine as compared with the maximum possible. Studies of these physical features concluded that none is an important confounding factor. The strong decrease in lung cancer mortality rates corrected for smoking frequency with increasing radon exposure is found in only the low altitude states or only the high altitude states; in only the warmest or only the coldest; in only the wettest or only the driest; etc. It is also found in only the states selected where the physical features are close to average. The BEIR IV theoretical prediction of lung cancer mortality from radon exposure corrected for smoking, M = m/m. = A + Br, does not take into account two recognized r-S correlations: (1) urban houses have 25% lower radon levels than rural houses and urban people smoke more frequently, and (2) houses of smoke have 10% lower radon levels than houses of nonsmokers. An extensive statistical stud of the effects of these r-S correlations leads to the conclusion that the BEIR IV predictio of B is reduced from + 7.3 to + 6.9, which contributes very little to decreasing th discrepancy with tile large negative values of B, -7.3 and -8.3 obtained from the actu measured and reported data.

"Linear-no threshold theories other than BEIR IV are considered which involv different treatments of smoking. Also considered is the "intensity of smoking." Analyt statistical study of these considerations lead to the conclusion that other theoretic predictions of B could reduce the discrepancy between 30% and 81%. The possibilit that an unrecognized confounding factor could explain the discrepancy is recognize However, tile following properties are required of an unrecognized confounder that cou resolve the discrepancy: (1) It must have a very strong correlation with lung cance comparable to that of cigarette smoking, but still be unrecognized; (2) It must have a ver strong correlation of opposite sign with radon levels; (3) It must tiot be strongly correlate with any of the 54 socioeconomic variables (SEV); (4) It must be applicable in a wid variety of geographic areas and independent of altitude and climate. The first propert alone requires of the unrecognized confounder that it must have increased by orders o magnitude since the beginning of this century, and have been much more important i males in the first half of the century, with effects on females rapidly catching up in recer years. The remaining properties impose additional requirements that are also mos difficult to meet singly, while to satisfy the four simultaneously becomes incredibl These multiple restrictions upon an unknown confounder make it extremely improbabl that one exists that would resolve the discrepancy.

"These tests of the linear-no threshold theoretical prediction of lung cancer mortalit induced by radon exposure, with the slope of the line determined by high dose exposures demonstrate that the theory fails badly by gross overestimation of mortality in low dose low dose rate range of radiation. A likely explanation is that stimulated biologica defense mechanisms more than compensate for the radiation "insult" and are protectiv against cancer in a low dose, low dose rate range."

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**Natural Background/Radon and Lung Cancer - Jaworowski 1995a**

Professor Emeritus Dr. Zbigniew Jaworowski (1995) states that, "Epidemiologica studies of a relation between the radon levels in homes and lung cancer seem to be als in disagreement with the non-threshold principle, and may suggest a hormetic effect. I the United States, in a study covering 89% of population, the people living in houses wit

radon air concentration higher than average level were found to have a lower mortality from lung cancer (Cohen, 1993). In China, in a meticulous study, the radon level was measured during one year in the houses of several hundred women with lung cancers, and in houses of similar number of healthy women. The results demonstrated at 95% statistical confidence level that women who lived in high-radon houses (more than 350 Bq per m3) had a 30% lower lung cancer risk than those living in low radon houses (4-70 Bq per M3). This result is opposite to the no-threshold-principle estimate, according to which the lung cancer risk in the high-radon houses should be 805 higher than the normal risk (Blot, 1990).

"Similarly, in a region of Japan with an a average indoor radon level of 35 Bq per m3 the lung cancer incidence was 51% of that in low-level radon region (11 Bq per m3), and mortality due to all types of cancer was 37% (Mifune, 1992). Similar results or showing a lack of positive correlation between the indoor radon level and lung cancers were reported from Canada, Sweden, Denmark, Finland, France and Great Britain."

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### Natural Background/Radon and Lung Cancer - Jaworowski 1995b

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Zbigniew Jaworowski (1995b) states that, "In China, a meticulous study measured the radon level for 1 year in the houses of several hundred women with lung cancers and in homes of a similar number of healthy women. The results demonstrated at a 95% confidence level that women who lived in high-level radon houses (more than 350 Bq/m$^3$) had a 80% lower lung cancer risk than those living in low-level radon houses (4 to 70 Bq/m$^3$).

"This result is opposite to the no-threshold principle estimate, according to which the lung cancer risk in high-radon houses should be 80% higher than the normal risk." (Blot et al.,1990)

"Similarly, in one region of Japan with an average indoor level of 35 Bq/m$^3$, the lung cancer incidence was 51% of that in a a low-level radon region (11 Bq/m$^3$) and the mortality caused by all types of cancer was 37% lower. (Mifune et al., 1992). Similar results showing a lack of positive correlation between lung cancer and indoor radon levels were reported from Canada, Sweden, Denmark, Finland, France, and Great Britain (see UNSCEAR, 1994, for references).

"Despite the evidence from these studies the U.S. Environmental Protection Agency has recommended remedial action when indoor radon concentrations reach 150 Bq/m$^3$. The EPA considers that remedial action at any level down to 70 Bq/m^3 would be cost-effective, even for the cost of reducing the level from 150 to 70 Bq/m^3 at approximately $2 million per life hypothetically saved. (Schiager, 1992)"

### 6.5 Radon and Radium Spas - Kondo 1993

*Professor Emeritus Dr. Sohei Kondo reports that there is evidence of "health-stimulating effects of natural radon in Japan."*

Professor Emeritus Dr. Sohei Kondo (Kondo 1993, Section 4.2.4) states that "Residents of Misasa, an urban area where there are radon spas, showed significantly lower mortality from cancers at all sites than residents of the suburbs of Misasa, as seen in Tables 4.11 and 4.12."

**Table 4.11A Standardized mortality ratios (SMR) for male inhabitants of Misasa radon spa area and a control area, 1952–85**

| Site of cancer | Misasa | | | Control area | | |
|---|---|---|---|---|---|---|
| | Observed | Expected | SMR | Observed | Expected | SMR |
| All sites | 53 | 98.48 | 0.538** | 228 | 268.23 | 0.850* |
| Buccal mucosa | 0 | 2.83 | 0 | 10 | 7.64 | 1.309 |

**Table 4.12 Relative risks[a] for dying from cancer at various sites for inhabitants of the Misasa radon spa area versus a control area**

| Site of cancer | Relative risk | 95% Confidence Interval |
|---|---|---|
| All sites | 0.67 | 0.53–0.85 |
| Stomach | 0.59 | 0.39–0.88 |
| Lung | 0.55 | 0.25–1.24 |
| Colon, rectum | 0.32 | 0.10–1.06 |

From Mifune *et al.* (1992). Copyright © Japanese Cancer Association. Reproduced with permission

[a]Estimated by Poisson regression analysis in which variables for sex, age and period were also included

* $p < 0.05$; ** $p < 0.01$

**Table 4.11B Standardized mortality ratios (SMR) for female inhabitants of Misasa radon spa area and a control area, 1952–88**

| Site of cancer | Misasa | | | Control area | | |
|---|---|---|---|---|---|---|
| | Observed | Expected | SMR | Observed | Expected | SMR |
| All sites | 37 | 79.88 | 0.463** | 156 | 202.64 | 0.770** |
| Buccal mucosa | 0 | 1.40 | 0.000 | 2 | 3.65 | 0.547 |
| Pharynx | | | | | | |
| Larynx | | | | | | |
| Stomach | 12 | 26.55 | 0.452** | 58 | 68.58 | 0.846 |
| Colon, rectum | 1 | 7.07 | 0.142* | 13 | 18.14 | 0.717 |
| Liver | 2 | 9.14 | 0.219* | 19 | 23.67 | 0.803 |
| Pancreas | 2 | 3.08 | 0.649 | 7 | 7.77 | 0.901 |
| Peritoneum | 2 | 1.16 | 1.721 | 9 | 3.00 | 3.000** |
| Lung | 1 | 5.34 | 0.187 | 5 | 13.57 | 0.369* |
| Breast | 1 | 3.89 | 0.257 | 5 | 8.89 | 0.563 |
| Uterus | 4 | 8.97 | 0.446 | 12 | 22.45 | 0.535* |
| Unknown primary site | 0 | 2.16 | 0.000 | 2 | 5.51 | 0.363 |
| Leukemia | 1 | 1.87 | 0.534 | 5 | 4.26 | 1.174 |
| Others | 11 | 9.24 | 1.190 | 19 | 23.18 | 0.820 |

## Natural Background/Radon and Radium Spas - Hattori 1994

Dr. Sadao Hattori, Vice President of CRIEPI (BELLE 1994) reports that, "Professor Emeritus of Osaka University Dr. Kondo and Dr.Tanooka, former Chairman of Japan Radiation Research Society, conducted statistical comparisons of cancer of the people of Misasa villages (i.e. high radon levels in drinking water), adjacent villages and all Japan. The result was meaningful as shown in Fig. 4."

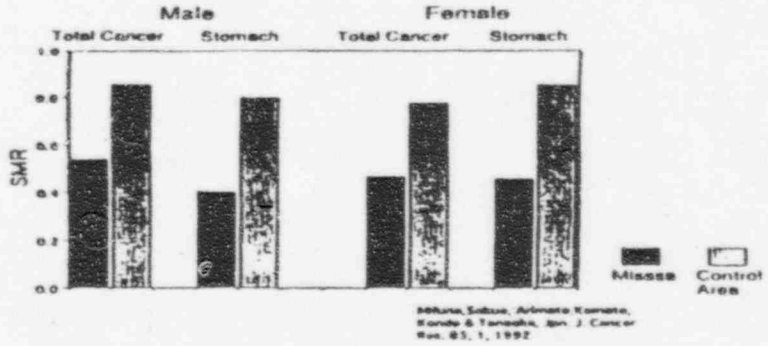


**Figure 4 Comparison of standardized mortality ratio, Misasa/control area.**

## 7.0 Non-Human Biological Data

### 7.1 Non-Human Biological Data/Biological Populations- Lorenz 1954

Dr. Egon Lorenz of the National Cancer Institute reports (1954) on the initial experiments with mice over generations exposed to a wide range of doses and dose rates, "SUMMARY

"Under the conditions of the experiment there seems to be no significant damage to the hematopoietic system as evidenced by counts of the peripheral blood.

"Male C3H mice conceived and living continuously under exposure to 4.4 r/24-hr day up to dotal doses of over 2000 r are comparable with nonirradiated mice as far as weight, coat color, and activity are concerned.

"Mammary-tumor incidence is not significantly changed in mice exposed for 10 to 15 months to doses ranging from 4.4 to 0.04 r per 24-hr day.

"Histologically only the gonads show radiation damage, and that mainly in the mice receiving continuous doses of 4.4 r/24-hr day. In males this damage consists in diminished spermatogenesis and reduction in the number of mature spermatozoa in the epidymis. This damage is reversible. Testes return to normal after removal from the exposure field.

"In contrast to the testes, radiation damage to the ovaries, observed principally in the mice receiving continuous doses of 4.4 r/24-hr day and perhaps also in those receiving 1.1 r/24-hr day, is irreversible and progressive and results in some cases in tubular downgrowths of the germinal epithelium that progress to early tumor formation. Breeding experiments indicate that C3H female mice are permanently sterilized with doses of 465 r applied at the rate of 4.4 r/24-hr day.

"Subsequent generations reared and living under exposure of 1.1 and 0.11 r per 24-hr day show no damage to chromosomes as evidenced by the raising of five to six generations with normal litter size and an apparently normal life span."

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### Non-Human Biological Data/Biological Populations - Luckey 1982

Professor Emeritus Dr. T.D. Luckey (1982) finds that "Radiation hormesis of animal growth rates was observed experimentally by many investigators. When compared with controls, the stimulation of growth rates in irradiated Daphnia (Mar66), flies (Kin55), moths (Kak67), silkworms (Has 12; Mall68; Par68), and blue crab (Ap75) supports reports of radiation hormesis of growth rates in vertebrates in the past 25 yr s. Consistency of results is well illustrated by the repeated confirmation of the report of Lorenz (Lor50) in which other investigators found growth stimulation at about the same daily dose for mice. In our work where special care was taken to handle unexposed mice in the same manner as irradiated mice, growth rates of lightly irradiated mice were statistically greater than that of control ($p<0.01$) using the Student test (Luc80)."

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### Non-Human Biological Data/Biological Populations - Sheppard 1987

Sheppard and Regitnig report (1987) on extensive research on the stimulating hormetic effect of plant growth:

*Irradiation increased the yield and value of some vegetable and field crops*

"Research on plants performed supporting commercial application on the stimulatory effects of radiation on plants. They show that "statistically significant hormesis responses occurred in a number of vegetable and field crops (Figs. 1-3).

"In several cases the response increased the yield and value of the crop, particularly when premium prices for early vegetable production are considered. Lettuce development was advanced to the detriment of late harest crop value (Fig. 2), because the plants from irradiated seed produced flowers, or bolted, earlier than plants fom the unirradiated control seeds.

"There are numerous examples in the literature showing hormesis in the species that were not responsive in Figs 1-3 (see Pahlow 1976) which suggests that hormesis can occur for seed irradiation of any plant species."

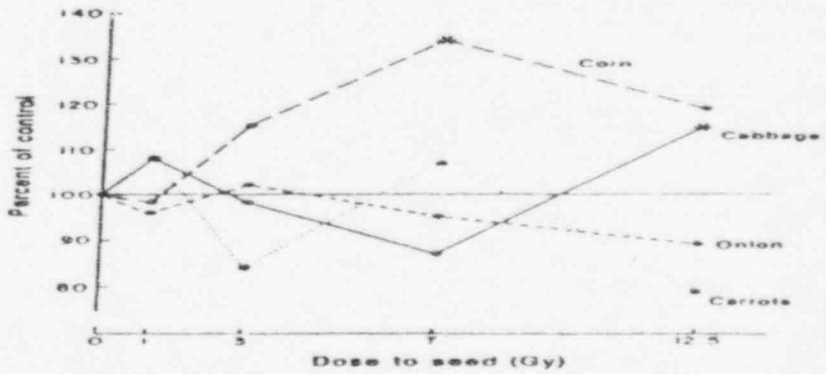


Fig. 1. Dry weight yield of vegetable crops grown from irradiated seed in a field experiment in Saskatchewan in 1972 (Re75). The points indicated by an asterisk were reported as significantly different from the controls ($p < 0.05$).

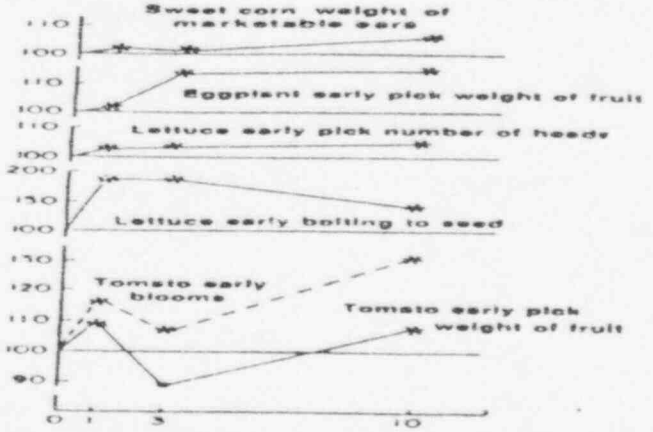


Fig. 2. Response of vegetable crops grown from irradiated seed in field experiments in Ontario in 1969. Points indicated by an asterisk were reported as significantly different from the controls (unpublished data, 1969, of V. W. Nuttall and L. H. Lyall, Ottawa Research Station, Agriculture Canada, Ottawa, Canada).

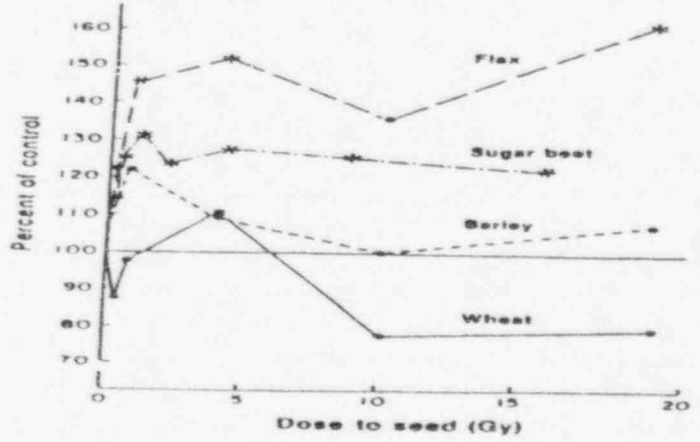


Fig. 3. Early counts of plant emergence for four crop species grown from irradiated seed in growth chamber experiments. Points indicated by an asterisk were significantly different from the controls ($p < 0.05$).

They report on numerous additional examples (eg, Figs 9, 10 and 11).

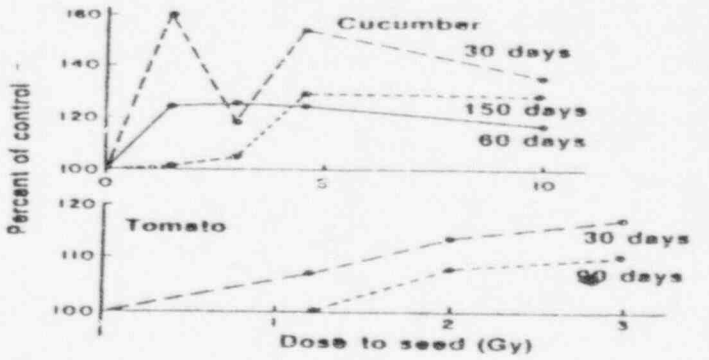


Fig. 9. Fresh weight yield from cucumber and tomato seed irradiated 30 to 150 d before planting in field experiments undertaken in Ontario in 1971 (In71). The relative yields from the first three pickings of cucumber and the first four pickings of tomato are shown. No statistical tests were given but the coefficients of variation were about 14%.

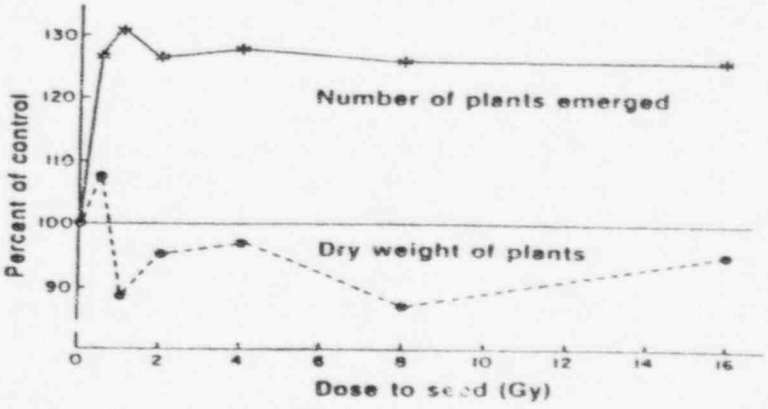


Fig. 10. Number of plants which emerged and single-plant dry weight of sugar beets grown from irradiated seed for 22 d in a growth chamber. Points indicated by an asterisk were significantly different from the controls ($p < 0.05$).

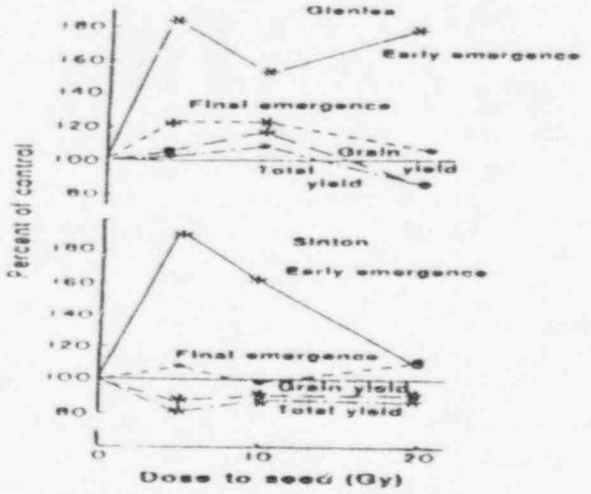


Fig. 11. Response at various stages of two cultivars of spring wheat, Glenlea and Sinton, grown from irradiated seed in a field experiment undertaken in Manitoba in 1984 (Sh86). Points indicated by an asterisk were significantly different from the controls ($p < 0.05$).

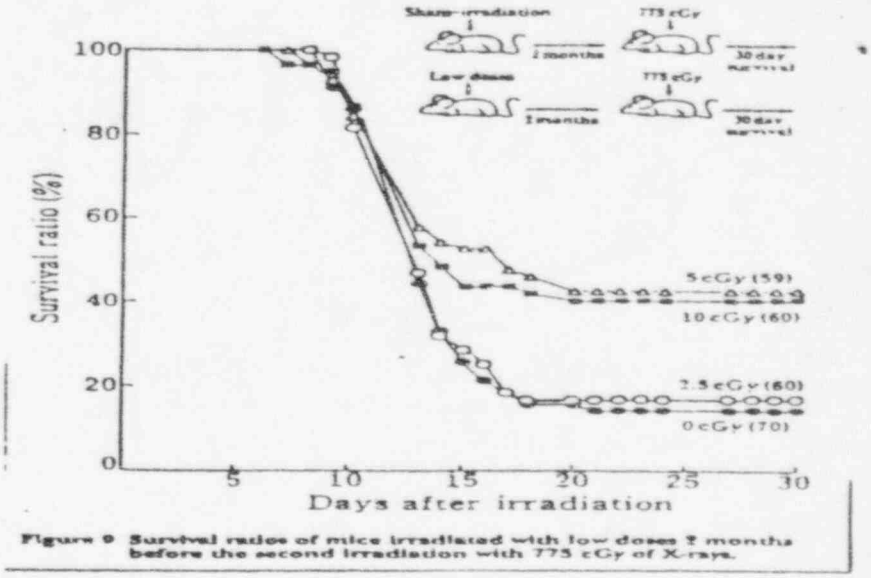


Figure 9 Survival ratios of mice irradiated with low doses 2 months before the second irradiation with 775 cGy of X-rays.

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**Non-Human Biological Data/Biological Populations - Hattori 1994**

Dr. Sadao Hattori, Vice President and Director of Research of CRIEPI reports (BELLE 1994) that, "Misonoo of CRIEPI estimated the optimum irradiation dose for radio-adaptation as shown in Fig. 9."

Dr. Hattori reports that, "Yonezawa of University of Osaka prefecture confirmed two phases of radio-hormetic responses by using a priming dose and survival after a sublethal dose administration. He found that a low (i.e. priming) dose (i.e. hormetic dose) enhanced resistance to sublethal x-radiation given two months but not two weeks later. Opposite results were observed when the primary dose was substantially greater."

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**Non-Human Biological Data/Biological Populations - Luckey 1986**

Professor Emeritus Dr. T.D. Luckey finds (1986) that, "Control Populations increased from 200 to approximately 24 000/ml during the 6 day incubation. The reproduction rate T. pyriformis was statistically lower ($P<0.01$) in subambient radiation than it was in near ambient radiation levels, 0.5 mrad/day (Fig. 3). Cultures irradiated at levels of 7.3 and 45 mrad/day reproduces faster ($P<0.01$) than did those at near ambient levels of radiation."

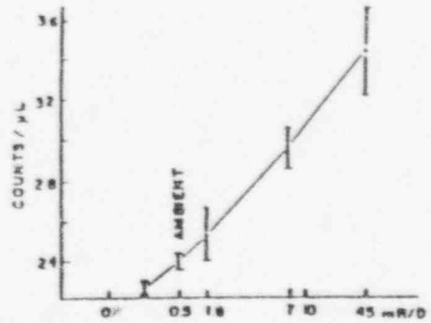


FIG. 3. The 6-day population of *T. pyriformis* per milliliter (ordinate) at different exposures (abscissa). Average values with one standard error of the mean of four replicates are displayed; the control is on the left and the exposed is on the right. Values with no overlap were significantly different from each other ($P < 0.01$).

"When T. Pyriformis was incubated at ambient radiation levels in the surface laboratory, no difference was found in growth rates with different K nuclides in the media. However, in the subambient radiation laboratory cultures with 39KCl consistently grew at slower rates then did control cultures with NKCl. When 39KCl was supplements with 40KCl at three times the level estimated to be NKCl, the growth rate was comparable iwth that of controls and faster than that of cultures containing only 39KCl.

"These data with pure cultures of T. pyriformis in a chemically defined medium confirm the results of Planel et al (Planel 1970, 1979) with bacteria-fed protozoans in natural media. The results with protozoa are remarkable similar to those of Conter et al. (Conter 1982) with an alga ... The cumulative results clearly indicate that ionizing radiation is essential for fast growth rates in these organisms. In most natural microbic habitats, fast growth rate represents survival in the competition for food."

"The results with different K nuclides suggest that radiation from 40K may fulfill at least part of the requirement for ionizing radiation. This supports the suggestion of Moore and Shastry (Moore, 1982) that the gamma rays, particularly Auger electrons, should be considered in radiology. The quantitative contribution of 40K is difficult to determine in these experiments. Had the contribution of the K+ in the medium been equivalent to that of the cells, about 200 mM, rather than 0.5 mM, then the contribution from 40K radiation could be compared with that from the external source."

"...In spite of great differences between microbes and metazoans, the general nature of hormesis with ionizing radiation (Luckey 1980, 1982) and the unity of metabolic processes and nutrition throughout all living organisms (Luckey 1960,1977, Moore 1982) suggest that the answer may be comparable for metazoan organisms despite the obvious variations between kingdom, phyla, and even species. Thus the cumulative evidences suggests that stimulation by ionizing radiation may generally result from increased amounts of an essential agent."

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**Non-Human Biological Data/Biological Populations - Planel 1987**

Planel reports (1987) on research (at the Laboratoire de Biologie Medicale in France) on paramecia that in "cultures placed in two identical chambers, the shielded chamber surrounded by a Pb wall, 5 or 10 cm thick. Cell populations were allowed to grow until the eighth day and then to decrease, due to nutrient exhaustion. As shown in Fig. 2, the cell growth rate of shielded cultures is lower than that of controls: the thicker the Pb shielding device, the more obvious the effect."

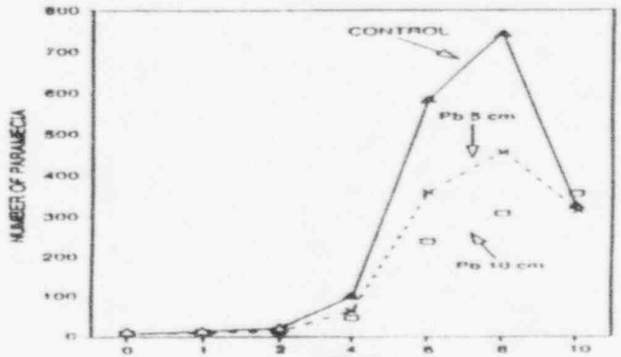


Fig. 3. Effect of shielding on proliferation of *Paramecium tetraurelia*, cultured in identical chambers, of which one was shielded with 5 cm or 10 of lead. The non-shielded control animals were exposed to normal natural gamma radiation rate of 1.75 mGy per year and animals shielded by 10 cm of lead were exposed to 0.3 mGy per year. On the 8th day, the proliferation of paramecia in the chamber shielded with 5 cm and 10 cm of lead was 80% and 40%, respectively, of proliferation of the non-shielded control animals. Adapted from Planel *et al* [14]

"He states that "the effect of radioprotection is not due to the presence of a toxic compound inside the shielded chamber; the same results were obtained when cells were cultivated in sealed glass ampullae. Responses to shielding also cannot be ascribed to radioactive compounds which might be present in Pb walls. Yearly dose rates... were 1.75 mGy in the control chamber and 0.3 mGy in the 10-cm Pb shielded chamber. Furthermore, no peak was detected by gamma spectrometry. On the other hand, this stimulatory effect of background radiation is confirmed by several complementary experiments.

"(1) When cells are cultivated in the control chamber, in a shielded chamber (10-cm Pb), and in an identical shielded chamber - but in the presence of a 232Th source giving a dose rate of 7 mGy/y - Fig. 2 shows that the inhibitory effect of shielding disappears when shielded cells are exposed to a level of radiation close to background.

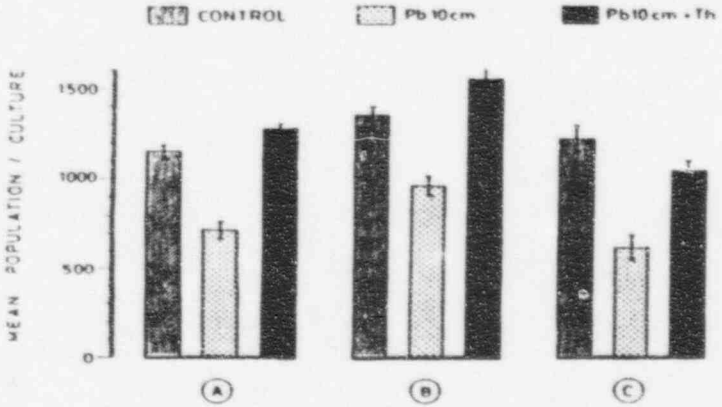


Fig. 2. The growth rate is restored when shielded cultures are exposed to a level of ambient radiation comparable to background.

"(2) The cell growth rate decreases when paramecia are cultivated in a cave, at the underground laboratory of the Centre National de la Recherche Scientifique under 200 m of rocks. A more obvious inhibitory effect is observed when the cultures are shielded against radioactivity of cave walls by 5 cm of Pb (Fig. 3). Yearly dose rates were 1.65 mGy for controls and 0. 1 mGy in the cave, using a Pb shielding. A normal generation time is restored when the cultures are exposed in the cave to 60Co gamma irradiation at a dose rate of 4 mGy/y (Fig. 3).

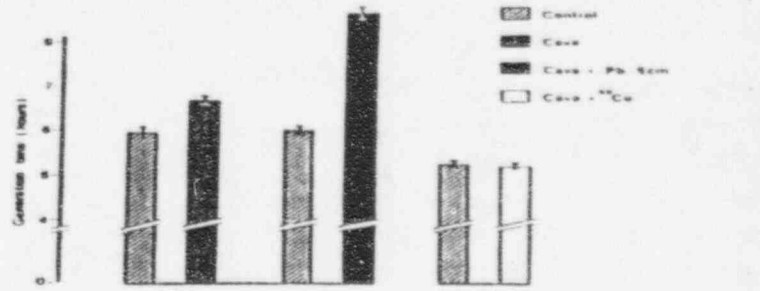


Fig. 3. Generation time of paramecia cultivated in an underground laboratory at different levels of ambient radiation.

"(3) Chronic irradiation by a 60Co source stimulates cell proliferation, as shown in Fig. 4, which expresses the results of three different experiments. Whatever the proliferation capacity, irradiated populations, measured on the third, fouth and fifth days, are larger than controls: total absorbed doses ranged fom 0.02 to 0.07 mGy."

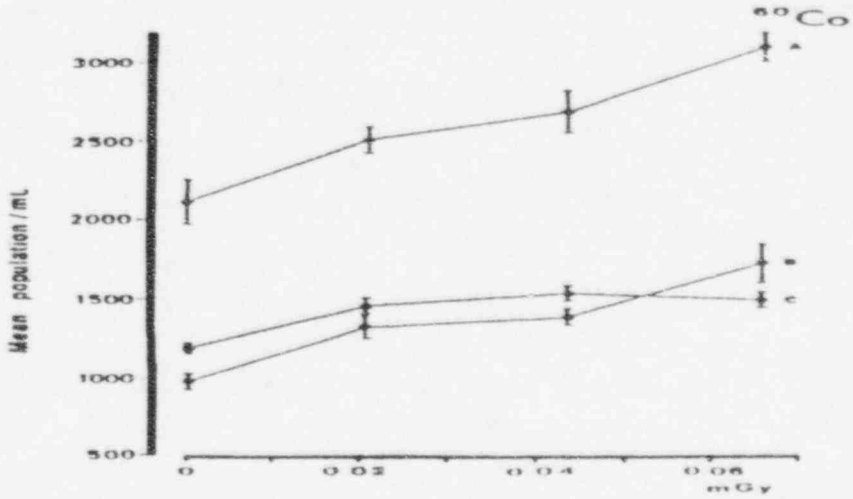


Fig. 4. Effect of chronic γ irradiation. (three experiments).

Planel reports further that similar results are obtained on cyanobacteria, showing that "shielding results in a lower cell growth rate (Fig. 8); this growth inhibition disappears when shielded cultures are simultaneously irradiated. Dose rates per year were 1.49 mGy in the control chambers, 0.27 in the shielded chamber, and 1.59 in the shielded chamber including a thorium nitrate source. Cell proliferation is stimulated when cultures are irradiated at a dose rate of 20.90 mGy/y.

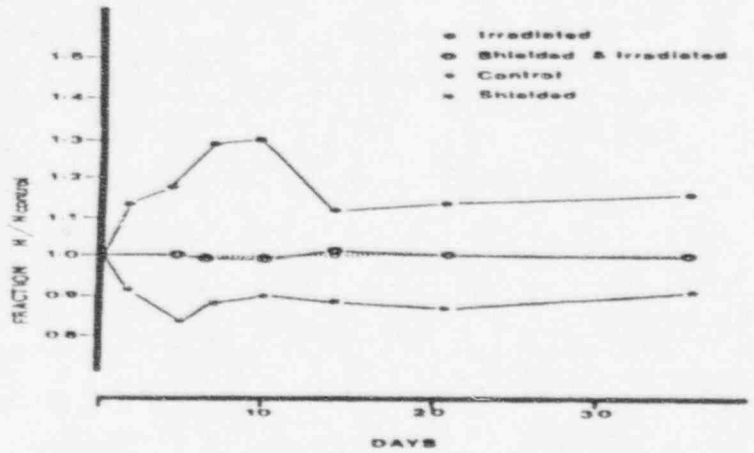


Fig. 8. Effect of shielding, irradiation and shielding combined with irradiation on relative cell concentrations in *Synechococcus lividus*.

In control experiments, cultures placed in the two chambers without a shielding device or a radioactive source exhibit the same growth rate."

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**Non-Human Biological Data/Biological Populations - Boxenbaum 1992**

Dr. Harold Boxenbaum reports (1992) that dependence on high-dose data, "Further support that γ-radiation produces longevity hormesis is supplied in Fig. 1.10. However, in this case, the data deal with chipmunks living in the wild. The animals were live-trapped, irradiated with either a single-dose of 200 or 400 Roentgens γ-radiation, except for controls, and then returned to the wild. It is readily apparent that γ-radiation exposure, within the dose-range utilized, enhanced longevity."

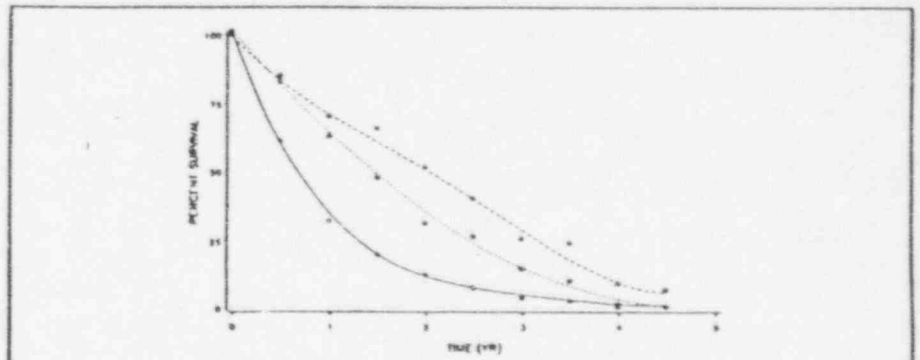


Figure 1.10. Representative survivalship functions for eastern chipmunks (*Tamias striatus*) living under natural conditions in the wild. These curves characterize data for female animals of known birthdate at location site 101 in Crawford County, Pennsylvania. Empiric polynomial functions were fitted to the three sets of population data: ● = controls; ■ = animals receiving a single dose of 200 R ionizing radiation; and ○ = animals receiving a single dose of 400 R. Time on the abscissa began at capture. Note the improved survival of irradiated animals. Thompson et al [47] equated Roentgens with rads; however, a more appropriate conversion factor is 1 R is approximately 0.95 rad with respect to muscle tissue [51]. The original data came from Tryon and Snyder.[52] Reprinted from Thompson et al.,[47] p. 278, by permission of Marcel Dekker, Inc.

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**Non-Human Biological Data/Biological Popluations - Jaworowski 1995b**

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that, "In mammals, radiation hormesis enhances defense reactions against neoplastic and infectious diseases, increases longevity and improves fertility. ..in an experiment with mice the incidence of leukemia, cancers, and sarcomas was lower in animals irradiated with cesium-187 gamma radiation doses of 2.5 to 20 mSy than it was in nonirradiated controls. The number of all malignant neoplasms in animals exposed to a single doses of 10 mSv was more than 30% lower than in nonirradiated controls. In several experiments, small initial radiation doses have been shown to improve the survival of animals subsequently irradiated with large, near lethal doses. In other experiments, an increased life span was found in animals irradiated with doses between 250 and 3000 mSv. ....a group of French studies started in the early 1960's, indicate that protozoa and bacteria exposed to artificially lowered levels of natural radiation demonstrate deficiency symptoms expressed as dramatically decreased proliferations. This indicates that ionizing radiation may be essential for life."

"In 1943, during the early stages of the Manhattan Project, it was found that the animals exposed to inhalation of uranium dust at levels that were expected to be fatal actually lived longer, appeared healthier, and had more offspring than the noncontaminated control animals. For years, these results were treated as an anomaly but later studies produced similar results. (Brucer,1989) The first UNSCEAR report to the General Assembly of the United Nations presented the results of experiments showing longer survival times of mice and guinea pigs exposed to small doses of gamma radiation (UNSCEAR, 1958)."

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**Non-Human Biological Data/Biological Populations - Patterson 1982**

H.Wade Patterson, former Editor of Health Physics Journal, quotes Spalding et al, 1982, from the Abstract:

"C57BL/6J male mice were exposed to 5 external doses from Co60 gamma radiation delivered at 6 different dose rates. Total doses ranged from 20 to 1620 rad at exposure rates ranging from 0.7 to 36,000 R/day. The ages of the mice at exposure were newborn, 2, 6, or 15 months."

From Section III, Results: "Most of irradiated animals lived longer or no differently than did the non-irradiated controls; however, in several cases differences were significant. For newborn mice exposed to 180 rad at 0.07 R/day, the life span was significantly longer than it was for controls. At all dose levels the 2-month age group lived significantly longer than did the median controls. Although there were no differences among the 6-month-old mice, the 15 month group with the 20-rad dose lived significantly longer than did the controls."

From Section IV, Discussion: "Our data obtained over widely ranging dose, exposure-rate, and exposure-age conditions fail to consistently support any mathematical function that may predict radiation-induced life shortening from radiation exposures approaching background levels. In fact, our data suggest beneficial effects from low-dose and low-dose-rate gamma-ray exposure."

**7.2 Non-Human Bilogical Data/Cellular and Molecular Biology and Genetics - Hattori 1994**

Sadao Hattori, Vice President of CRIEPI reports (BELLE Newsletter 1994) on research that demonstrates reversals of aging effects in cells, that "Yamaoka of CRIEPI measured the properties of cell membranes and superoxide disutase activities. (Fig. 7)" (Yamaoka, 1991)

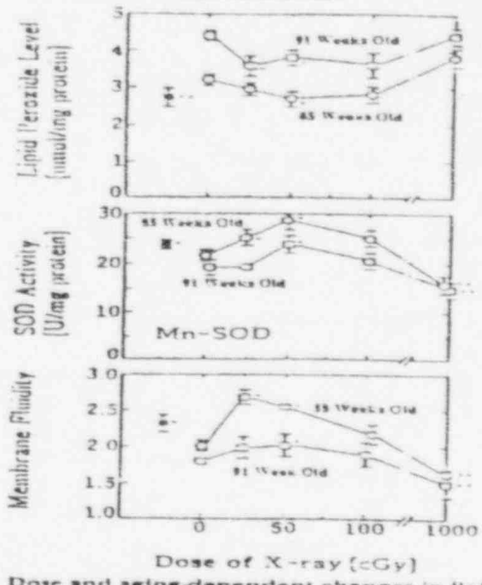


Figure 7 Dose and aging-dependent changes in lipid peroxide (TBARS) level, SOD activity and membrane fluidity (W/S ratio) of rat's brain cortex by X-ray irradiation.
Membrane fluidity was determined by spin-label method using ESR spectrometer. W/S means ratio of weak to strong bands. ■ shows the data from sham-irradiated 7 weeks old control. Each value indicates the means ± S.E.M.
The number of rats per experimental point is 10–15.
*P<0.05 and **P<0.01 vs sham-irradiated 85 or 91 weeks old control (t test).

*Suppression of Lung Cancer*

Dr. Hattori also reports on studies that demonstrate reduced cancer induced in mice by low level radiation doses that,"Ishii of CRIEPI and Hosoi of Tohoku Univ. examined the suppression of metastasis by counting lung colonies of mice, (Fig. 5).

"Ishii also measured the activation of rat splenocytes, as shown in Fig. 6 by low dose radiation exposure."

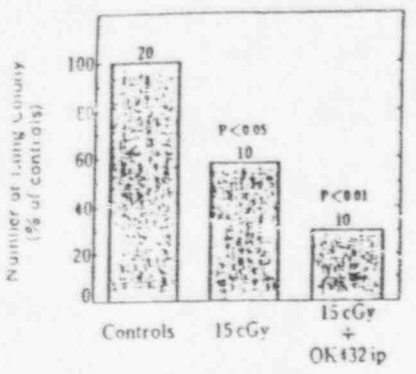


Figure 5 Inhibition of spontaneous metastasis to lung by whole body X-ray irradiation with 15 cGy and combined treatment. 15 cGy was irradiated 20 days after transplantation with murine squamous cell carcinoma.

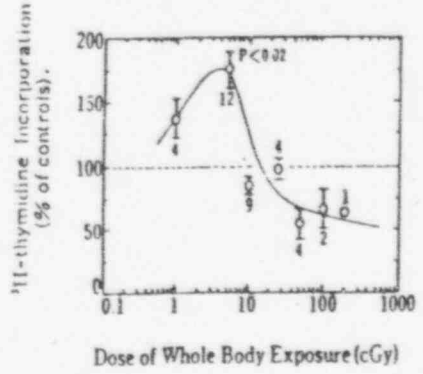


Figure 6 Effect of various doses of whole body X-ray irradiation on Con A-induced proliferative response of rat splenocytes. The splenocytes were obtained from rats at 4 hrs after X-ray irradiation.

*Radiation Adaptation*

Dr. Hattori reports (BELLE 1994) that, "Ikushima of Kyoto Univ. examined the radio-adaptive response as shown in Fig. 8. Chinese hamster V 79 Cells were incubated with 3H-Thymidine for 16 hrs (one cell cycle) and irradiated with a dose of 1 Gy of 60Co gamma-rays (0.4 Gy/min). The cells were fixed and assayed for the formation frequency of the micronucleus 6 hrs after irradiation."

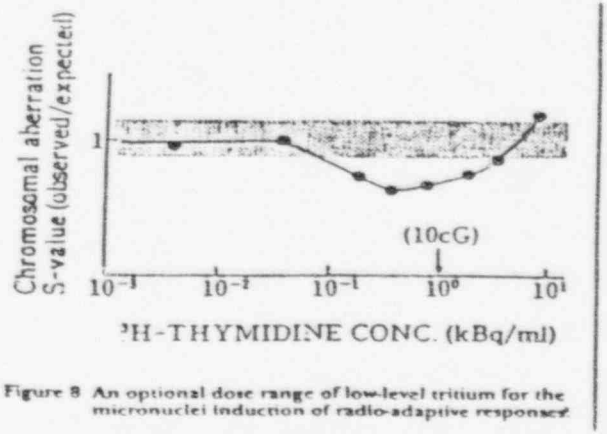


Figure 8 An optional dose range of low-level tritium for the micronuclei induction of radio-adaptive response?

*Vitalization of human cells*

Dr. Hattori reports (BELLE 1994) that,"Watanabe (1992) of Nagasaki Univ. compared the growth rate of human embryonic cells which had been exposed to a high acute dose or to periodic multiple doses. Cells which received 7.5 cGy/week showed an hormetic response. Fig. 11 shows one of his experimental results."

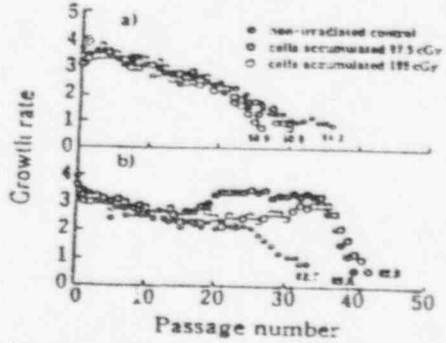


Figure 11 The growth rate at each passage in human embryonic fibroblasts (HE7) irradiated with single dose at passage 0(A) and multiple doses of 7.5 cGy of $Cs^{137}$ gamma-rays (B).

Dr. Hattori also reports on the configuration that low level radiation stimulates the production of the DNA repair protein that, "Professor Ohnishi of Nara Medical College discovered a marked increase of stress protein production by p53 genes. Doses of 10 to 25 cGy were effective. Fig. 13 shows his experimental results."

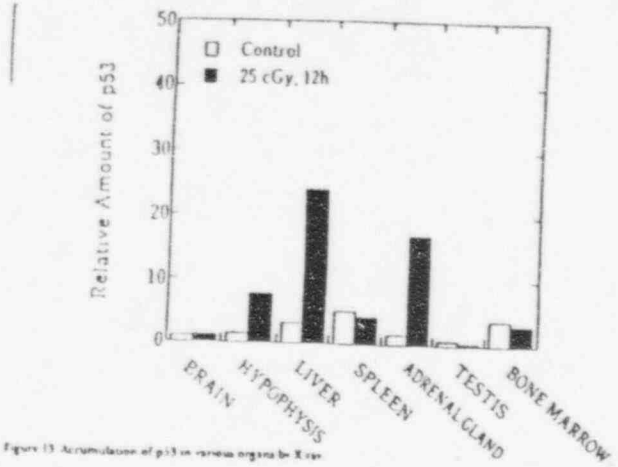


Figure 13. Accumulation of p53 in various organs by X-ray

**Non-Human Biological Data/Biological Populations - von Borstel 1995**

Professor Dr. R.C. von Borstel of the Department of Biological Sciences, U. Alberta, in a review (1995) states that, Kondo (1993) addresses "the possible mechanisms of action by radiation on cells that might lead to the beneficial effects. ... "Radiation-induced damage mostly follows the no-threshold rule; when a threshold is involved, a specific cellular or molecular mechanism unrelated to the action of radiation upon DNA must be sought. Consequently, Kondo argued that death of damaged cells by very low levels of radiation was followed by replacement with undamaged cells, so that a threshold would be the inevitable result. It has been known for most of this century that cells from an early morula can be separated, and consequently give rise to cells that replace the missing ones completely, thus producing a normal embryo. The occurrence of identical twins or triplets is clear evidence that this happens also in mammals. What Kondo posits is that cells killed by radiation can be replaced with normal cells until a threshold dose is reached at which the cell damage can no longer be compensated for. He brings forth a great deal of evidence that this must be the explanation for the radiation threshold necessary for fetal damage...

"We know that cancer initiation results from the same mechanisms as mutation; thresholds do not exist for mutations, other than that induced mutations must push their way through the background noise of spontaneous mutations in order to be detected. The background noise was termed an 'apparent threshold' by Yataro Tazima. Nevertheless, there is a true threshold for certain kinds of radiation-induced cancer, as Kondo demonstrates conclusively in Chapter 3, using data on survivors of the Nagasaki atomic bomb. Because radiation is such a powerful agent for turning on and turning off carcinogenic action, and also because radiation can be more accurately measured than other carcinogens, Kondo develops two all-encompassing hypotheses to explain the threshold for human cancer. ... The no-threshold hypothesis of stem-cell mutation and the wound-healing error hypothesis.

"The no-threshold hypothesis posits that a number (say between 5 and 7) of mutations must occur in the same somatic stem cell before neoplastic growth can take place. Kondo points out that, with known spontaneous mutation rates of individual cells, the no-threshold hypothesis would be excluded unless either (1) the cell was a mutator ... Or (2) certain individual genes in a stem-cell in interphase might have high mutation rates. Kondo made a brilliant case for supporting explanation (2). It is now known that explanantion (1) is certainly true (Fishel, et al, 1993, Cell, 75:1027). Nevertheless, explanation (2) has not been excluded; we sometimes find that both of two alternative hypotheses may be correct....

"Kondo notes that the wound-healing error hypothesis is a problem of cell society; that is, perhaps there is a stimulation of growth after a tissue is wounded by radiation, and that oncogenes may be involved in the process of cellular adaptation to environmental change. Kondo suggests that continued epigenetic changes necessary for cells to become cancerous are induced by over expressing healing activity of endogenous factors recruited for tissue repair'. He marshals a great deal of data to support this idea, and almost all the small paradoxes formerly argued about come together within his paradigm."

**Non-Human Biological Data/Cellular and Molecular Biology and Genetics - Jaworowski 1995b**

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that , "UNSCEAR (1994) reviewed the most important publications on the stimulating effects of radiation... effects were found at biochemical, cellular and organic levels, in cell cultures, bacteria, plants, and animals."

"UNSCEAR 1994 concentrates on the elucidation of mechanism by which radiation hormesis acts at the level of cell control systems such as protein synthesis, gene activation, DNA repair, stress-response protein production, radical detoxification, activation of membrane receptors, proliferation of splenocytes, and stimulation of the immune system."

## 8.0 Costs

Prof Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that in its December 1994 rule making proposal by "the US EPA ... four UNSCEAR documents, from 1977, 1982, 1986, and 1988, are used to support a need for revision of the current radiation standards. The most recent UNSCEAR document from 1994, however, on the adaptive effects of low doses of radiation, is not taken into account. .. in which a new radiation limit for the public of 1 mSv/year (70 mSv in a lifetime) is proposed.. .. (S)uch a low radiation standard, only about 3% of the natural radiation background in many regions of the world, would bring enormous costs for society, and it would be ethically fair only through a large reduction of identifiable health hazards . . .

"The four UNSCEAR documents quoted by the EPA as estimating that the risks 'of cancer have increased roughly threefold and have become more certain' were critically examined by UNSCEAR during the past 8 years, especially the interpretation of the results of epidemiological studies in Hiroshima and Nagasaki . . .

"The most important message of the recent UNSCEAR (1994) document, however, is the recognition of the existence of stimulating and adaptive effects of ionizing radiation. During the past 4 decades these effects were ignored in radiation protection philosophy and practice.

"Each human life hypothetically saved by implementing the U.S. Nuclear Regulatory Commission's regulations costs about $2.5 billion (Cohen, 1992). Such spending is morally questionable. Studies of radiation hormesis suggest that such expenditures may be futile and actually have an adverse effect on the health of the population."

## 9.0 Conclusions

Professor Emeritus Dr. T.D. Luckey (1994) states that, "The consistent, statistically significant results showing radiation hormesis in cancer invalidate the zero thesis and all linear models derived by linear interpolation (often mislabeled "extrapolation") from large doses to controls. There are no comparable data which support the linear models. Results from miners are not convincing because it is difficult to separate radiation carcinogenesis from particulate and fume oncogenesis. Information from human cells in culture have less meaning than well-controlled animal studies. Cells in culture are laboratory artifacts with little intercellular communication and negligible hormonal, neurologic or immune control systems. These are the reasons that the apparent optimum for humans far exceeds the recommended minimums set by various agencies."

"In addition to lowered cancer mortality rates, physiologic functions which appear to be enhanced include growth and development, auditory and visual acuity, learning and memory fecundity, and resistance to infection. These results are noted with both acute or chronic whole-body exposures. The subsequent increased average life span appears to explain the decreased mortality from infections and cancer; this appears to be due to a stimulation of immune competence (Luckey, 1991, 1994; Sugahara et al, 1992)."

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**Conclusions - Pollycove 1994**

Professor Emeritus, Myron Pollycove, MD, reports (Pollycove 1994) that, "Significant positive health effects associated with low level radiation have been demonstrated in a review of five epidemiologic studies: decreased mortality of nuclear shipyard workers, decreased noncancer mortality of atomic bomb survivors in both Hiroshima and Nagasaki and Nagasaki alone, decreased lung cancer mortality associated with increased radon exposure of the U.S. population, and decreased breast cancer mortality of women in Canada after having received multiple fluoroscopic examinations. The tendency to neglect or reject data that contradicts the linear-no threshold theory of radiation carcinogenesis is supported by confidence that chromosome aberration and gene mutation can be produced by a single particle of ionizing radiation and so initiate a malignancy. The number of such interactions with cell nuclei is both logically and demonstrably proportional to the dose. However, no consideration is given to biological defense mechanisms that could be stimulated further by low level increments of radiation above the background level. Such stimulated defense mechanisms could also decrease carcinogenesis by chemical and other non-ionizing agents as well as high level ionizing radiation. Multiple defense mechanisms at molecular, cellular, organ, and systemic levels involving enzymatic, ormonal, immunologic, and stress protein interactions are currently being demonstrated and confirmed by numerous investigators. (Calabrese, Ed. 1992, Luckey, 1991, Sughara et al., 1992) Recently a human radiation repair gene has been cloned and transfected into a mutant Chinese hamster with sensitivity to both ionizing radiation and certain alkylating agents resulting from defective repair of DNA strand breaks. These transfected mutants demonstrate overexpression of the human DNA repair minigene with repair capacity increased above that of the wild-type Chinese hamsters." (Caldecott, 1992)

"Mounting reproducible evidence of the operation of various defense mechanisms and their stimulation by low dose ionizing radiation will provide further details of how biological defense mechanisms, nonoperative at high doses, are stimulated and enhanced by low level radiation damage so as to overcorrect and predominate. These investigations have clarified why the negative health effects observed at high levels of radiation that effectively overwhelm these defense mechanisms cannot be extrapolated to the low levels in which these stimulated defense mechanisms predominate with decreased cancer induction, decreased mortality, and other observed positive health effects."

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**Conclusions - Yalow 1994**

Nobel Laureate Dr. Rosalyn Yalow (1994) states, "Populations have been studied in geographic areas of increased natural radiation, in radiation-exposed workers, in patients medically exposed, and in accidental exposures. No reproducible evidence exists of harmful effects from increases in background radiation three to ten times the usual levels. There is no increase in leukemia or other cancers among American military participants in nuclear testing, no increase in leukemia or thyroid cancer among medical patients receiving I-131 for diagnosis or treatment of hypothyroidism, and no increase in lung cancer among nonsmokers exposed to increased radon in the home.

"The association of radiation with the atomic bomb and with excessive regulatory and health physics ALARA radiation levels practices has created a climate of fear about the dangers of radiation at any level. However, there is no evidence that radiation exposures at the levels equivalent to medical usage are harmful.

"The unjustified excessive concern with radiation at any level, however, precludes beneficial uses of radiation and radioactivity in medicine, science, and industry."

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**Conclusions - Jaworowski 1995b**

Professor Emeritus, and Member of the UN Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), of the Central Laboratory for Radiological Protection, Dr. Zbigniew Jaworowski states (1995b) that "the ICRP assumption on linearity was not very realistic. It was ... accepted, however, because it simplified regulatory work by allowing extrapolation ... The original purpose was to regulate ... a relatively small group of occupationally exposed persons and it did not involve exceedingly high costs to society.

"The dose limit for the public was set at 50 mSv over a lifetime ... less than one third of the global average lifetime dose from background radiation ... and many tens or hundreds of times lower than the lifetime dose in many regions of the world.

"Limiting exposure below the levels of natural radiation at which millions of people have lived since time immemorial is a logical consequence of the ... assumption from 1959: if such dose is detrimental, then one should also attempt decrease the risk of background radiation ... or the risk of man-made radiation even at such trivial levels as 1 mSv/year.

"Yet such reasoning was less than palatable to many scientists ... not only because of the epistemological problem of trespassing beyond the limits of knowledge ... but also because of the absurd practical consequences and the moral aspects.

"As demonstrated by Walinder (1987), on the complementarily principle, the stochastic phenomenon of radiation carcinogenesis cannot be for an open system, such as a human being or a population. It can only be done if the radiation dose is much more powerful than the natural dose, combined with other carcinogenic factors ... A conception that mathematical models adapted for high-dose effects can be limitlessly extrapolated to low doses and still represent a biological reality is epistemologically unacceptable (Walinder 1987). The absurd practical consequences were exposed by the Chernobyl accident.

"Long before that Professor W.V. Mayneord, one of the most notable persons in radiation protection and a former member of the UK delegation to UNSCEAR and of ICRP stated (Mayneord 1964): 'I have always felt that the argument because at higher values of dose an observed effect is proportional to dose, at very low doses there is necessarily some "effect" of dose, however small, is nonsense.

"Dr. Lauriston Taylor, former president of the US NCRP, defined applications of the linear, no-threshold dose-effect relationship to such calculations as 'deeply immoral uses of our scientific heritage' (Taylor 1980).

"The no-threshold arithmetic ... led to a decision by the Supreme Soviet (but against the advice of the leading Soviet scientists (Ilyin 1993) to evacuate about 116,000 inhabitants of Ukraine and Belarus, causing unspeakable suffering and a loss of many billions of dollars, equivalent to about 1.5% of the GNP of the ... Soviet Union (ICP 1991).

"The intervention level for evacuation was a 70-year lifetime radiation dose of 350 mSv, about twice the world average natural background dose (168 mSv). All families with pregnant women and children less than 12 years of age were relocated from areas ... [where] the Cs-137 body burden in children still living in these areas was ... between 40 and 2250 Bq, which is less than the natural burden of radioactive K-40 (4000 Bq) in adults. Body burdens of several thousand Bq are now common in Northern Canada and were as high as 100,000 Bq during weapons tests in the 1960s (Tracy 1994).

"...one might ask why governments ... do not relocate populations in (high natural background) areas ... why isn't everyone evacuated from Norway, where the average lifetime dose is 365 mSv (Henriksen and Saxebol 1988) and in some districts 1500 mSv? Should not regions of India with >2000 mSv (Sunta 1990) be depopulated?

"What about areas of Iran with >3000 mSv? ... (I)n the city of Ramsar several generations in one household have been receiving average individual lifetime doses of natural radiation of 17,000 mSv, 240 times the current ICRP limit. Yet these individuals show no increased incidence of disease, and some of them have lived to be 110 years of age (Sohrabi, 1990)."

"The recognition by UNSCEAR, the most distinguished international scientific body on the matters of ionizing radiation, of the possibility that low doses of radiation may result in changes in cells and organisms which reflect an ability to adapt to the effects of radiation, may inspire the authorities to begin a more realistic approach to problems of estimating and managing the risks of ionizing radiation. The past 4 decades witnessed regulatory activity, stemming from the linearity principle, steadily decreasing radiation standards to an absurd subnatural level of 1 mSv per year. The time is ripe for renunciation of linearity principle in radiation protection of the public and for considering a practical threshold dose as a basis for radiation standards."

"Dr. Jaworowski also reports, "Since the 1960s, (hormetic) effects have been ignored in radiation protection practice, while research on stimulating and adaptive effects of radiation, the radiation hormesis, has continued over several decades. The results of more than 1200 published papers on hormesis were recently reviewed by Luckey (1990) - many of them in an excellent book by Kondo (1988). The studies on hormesis were also presented at four international conferences (Oakland, CA, 1985); Frankfurt, Germany, 1987; Kyoto, Japan, 1992; and Changchun, China, 1993). It is astonishing, however, that even recently the obvious hormetic effects appearing in the epidemiological studies were

often not noticed, not only by the readers, but by the authors themselves (see for example Section 3.4 Pollycove 1994, Figure 4).

"Radiation hormesis goes beyond the notion that radiation has no deleterious effects at small doses; at small doses new stimulatory effects occur that are not observed at high doses and these new effects may be beneficial to the organisms.

"Recognition of the existence of hormesis opens up an important new field of research.

"In mammals, radiation hormesis enhances defense reactions against neoplastic and infectious diseases, increases longevity and improves fertility . . . in an experiment with mice the incidence of leukemia, cancers, and sarcomas was lower in animals irradiated with cesium-137 gamma radiation doses of 2.5 to 20 mSy than it was in nonirradiated controls. The number of all malignant neoplasms in animals exposed to a single dose of 10 mSv was more than 30% lower than in nonirradiated controls. In several experiments, small initial radiation doses have been shown to improve the survival of animals subsequently irradiated with large, near lethal doses. In other experiments, an increased life span was found in animals irradiated with doses between 250 and 3000 mSv. . . . a group of French studies started in the early 1960's, indicate that protozoa and bacteria exposed to artificially lowered levels of natural radiation demonstrate deficiency symptoms expressed as dramatically decreased proliferations. This indicates that ionizing radiation may be essential for life."

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**Conclusions - von Borstel 1995**

Dr. R.C. von Borstel (1995) in a review of Kondo 1993 states, "Linear extrapolation from higher doses to low doses turns out not only to be a conservative way to calculate risks, but also to be errant and even misleading. Although this has been known for at least 30 years, national and international regulatory agencies are institutionalized and authoritative, and thus have left the door open for journalists and even radiation experts to predict damages to human beings from radioactivity fallout. The misjudgement based on linear extrapolation has had its consequences even when there was no radioactive fallout, such as... at Three Mile Island: The townspeople ... were led to believe that they had been the survivors of a nuclear holocaust.

"Now we have before us an eminently logical book by Sohei Kondo. He uses the available data on irradiated human subjects to conclude that individuals subjected to low levels of radiation have longer life spans than those in control populations, and fewer cases of most types of cancer as a bonus."

Dr. von Borstel states, "This book is clearly written by an extremely wise man. Let us hope that regulators of nuclear policy around our planet can use the compiled data and its conclusions in an equally sagacious manner."

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**Conclusions - Thomas 1995**

Dr. Robert G. Thomas reports (1995) that, "The analysis of the radium luminizer epidemiology study presented demonstrates that it is time to evaluate data objectively instead of formatting an extrapolation scheme beforehand and forcing data to fit a preconceived pattern such as linearity through a dose-effect origin. The no-effect dose levels discussed should signal that it is also time to reevaluate (again) the large variations in background radiation levels throughout the world and to cease being concerned with, and regulating against, minuscule doses for which no biomedical effects on humans have ever been satisfactory identified or quantified."

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**Conclusions - Boxenbaum 1992**

"Dr. Harold Boxenbaum reports (1992) that dependence on high-dose data "is a major problem in long-term toxicity studies which typically employ relatively high doses of toxicants (generally about 12.5 to 100% of the maximum tolerated dose per day in an attempt to assess risk at much lower doses. Although both Boxenbaum et al. (1988) and Neafsey (1989-90) have recently addressed the problem of potentially overlooked longevity hormesis, the risk assessment community has failed to give it serious consideration. Previously, Smyth (1967) had taken notice of the fac that low doses of otherwise toxic substances can be beneficial. His reward - the epithet: "Dr. Smyth and his fellow poisoners" (Ottoboni, 1984). Although the scientific community envisages itself as the epitome of institutionalized rationality (Newton-Smith, 1981), many researchers have noted the high degree to which anomalous information is ignored if it disconfirms basic assumptions of established paradigms (Star, 1985). Once a group agrees that a particular kind of reality is desirable, they develop a style that permits them to deal with observations solely on their own terms -- and woe to the individual with different ideas (Becker, 1968) (vide supra - Dr. Smyth). For most individuals, escape from these intellectual-scientific fetters is difficult, for the obduracy of established perspective locks practitioners together in a rigid framework of beliefs that is not readily overcome (Echberg & Hill, 1989; Star, 1985).

Conclusions - Walinder 1996a

Professor Dr. Gunnar Walinder, radiobiology and medicine, U.Stockholm and U. Uppsala, a member of UNSCEAR and ICRP, states (1996b) that, "I have found and adduced arguments for that the current pretentions to knowledge about low-dose transformations of cells into malignant phenotypes are inconsistent with modern oncology as well as entirely futile on purely epistemological grounds. In this respect, modern oncology has clearly shown that the contribution of a small (non-dominant) radiation dose is not a stochastic event but a highly conditional one.

"Furthermore, a malignant cell transformation is not synonymous with cancer. The transformed cell has to divide and, thus, new copies of the genome have to be formed more than a billion number of times before we can speak of a tumor or establish that an organism has contracted cancer. This is what the Nobel prize winner Murray Gell-Mann means when he characterizes cancer as multi-iterative process in a complex, adaptive system. He (and others) has shown that the outcome of such a process is fundamentally unpredictable.

"It is difficult for me to understand how people can believe that such an enormously complex phenomenon as the dose-response of radiogenic cancer can be adequately identified with an equation of the first degree. They do not confine themselves to saying that the dose-response can be approximated to a straight line but it is stated that it is linear. The linerarity is thus considered an inherent characteristic of the dose-response, a "fact" that permits us to extrapolate or interpolate the observed data even outside the dose area within which we have made our observerations.

"I don't hesitate to say that this is one of the great scientific scandals in our century."

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Dr. Mark Johnson presented the following statements with references on behalf of the "Center for Atomic Radiation Studies, Inc." (CARS) to support indicating the existence of evidence of low level radiation health effects. These materials were presented to the ACRP on June 2, 1995. The listed references, except a few on the Japanese survivors, were reviewed. Extracts of the applicable sections of these references that consistently fail to support the proposed conclusions were presented to the ACRP Sep 8, 1995. The extracted materials were provided to the ACRP, and to Dr. Johnson and CARS. Several contacts with Dr. Johnson on his assessment and response to these conclusions, and possible misinterpretations of the evidence. However, no response to these determinations were provided to the ACRP or the author.

Dr. Johnson and CARS were also provided with many of the references from this data compilation that Dr. Johnson identified as not readily available in the research libraries. A number of discussions were held with Dr. Johnson on his review of these sources of credible scientific data and analysis which had been provided in the original presentations and in the March 8, 1995 draft report compiling the slide presentation materials from those sources. No response has been forthcoming as of the scheduled March 29, 1996 ACRP meeting.

## STATEMENTS BY MARK JOHNSON

## REVIEW OF SOURCE REFERENCES

**"A-bomb survivor follow-up studies:"**

As reported in this compilation of data from many independent and credible sources, the lack of Japanese survivor effects at low doses are well established. The specific below referenced reports have not all been reviewed. Certain of the reports are large volumes of tables of results for which finding proposed support for the general conclusions was not possible without more specific references which have not been provided. Several reports were not returned in a literature request. Additional literature reviews may be possible with more specific support/response from Dr. Johnson.

"1.) Both the A-bomb survivor mortality (1950-1985) and incidence data (1950-1987) fail to suggest the existence of a threshold for cancer induction down to very low doses. (Vaeth et al., 1992, Shimuzu et al., 1992, Dohy et al., 1994)"

**Vaeth et al., 1992:** not returned
**Shimuzu et al., 1992:** large tabular presentation
**Dohy et al., 1994:** not returned
The statement that the "fail to suggest the existence of a threshold" does not mean that a threshold does not exist. The formal RERF "models" presume a linear relationship, then state that a non-linear relationship is not found.

"2.) Doses less than 5 cGy and probably as low as 1.6 cGy have been associated with excess cases of leukemia among A-bomb survivors (Carter, 1993, Tomonaga et al., al., 1991)"

**Carter, 1993;** not returned
**Tomonaga et al., al., 1991**

"3.) Doses in the range from less than one to a few cGy have been associated with brain damage in prenatally exposed children of A-bomb survivors (Schull et al., 1991)"

**Schull et al., 1991** reports on the physical conditions of brains from a few brain damaged individuals exposed at high doses. The statement is not supported by the reference.

"4.) Mortality for solid cancers in the 6-19 cGy dose group (mean colon dose 10.9 cGy) (mean colon dose 10.9 cGy) is significantly higher ($p<0.01$) than it is in 0-5 cGy dose group (mean colon dose 0.7 cGy) and there is evidence for a convex dose relation (Shimuzu et al., 1988)"

**Shimuzu et al., 1988;** a large volume of data tables, does not support the proposed 'finding'. The Japanese survivor data, as presented by Prof. Sohei Kondo, explicitly address colon cancer, because it has lowest cancer at low doses compared to the unexposed population, of the various radiogenic cancers. Dr. Kondo correlates that data to the biological mechanisms of colon cancer tumorigenesis and cell repair as indicating the mechanism and biological plausibility of hormesis or cancer-protection beneficial effects, consistent with extensive biological evidence.

## "Government-sponsored nuclear worker studies:

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| **"Finding: Significant association between cancer induction and low dose radiation exposures were found for the following types of cancer:"** | **The posited "finding" is unsupported by the references** |
| "prostate cancer (Inskip et al., 1987, Beral et al., 1985)," | **Inskip et al., 1987:** "Analyses of non-fatal cancers in relation to re-estimated cumulative whole body exposures (table 11) showed no clearly significant dose response relations at the 5% level, although the trends for skin cancer and bladder cancer approached statistical significance ($p = 0.06$ for both cancers." (Table A1) Contrary to the proposed "finding", there is no linear trend in this source for this cancer.<br>Table 5, "Mortality by whole body exposure..." (Table A2), prostatic cancer excess at high doses (4.59 at 50-100 mSv, 2.22 at >100 mSv), lower than expected at <10 mSv (0.78), and lowest at 10-20 mSv (0.30). If the data show anything, it is a stronger hormesis (beneficial) response than a linear response.<br>Table 9: "Mortality by surface exposure..." (Table A3), prostate cancer excess at >100 mSv (2.88), and lowest rates at 10-20 mSv and 20-50 mSv (0.57 and 0.51), the data again show more a hormesis (beneficial at moderate exposure) response than a linear response.<br>Inskip et al use mortality rates in this population for the "expected" values. They do not compare the population to other workers or the general population.<br>Inskip, et al, 1987 does not support the 'finding' posited.<br>**Beral et al., 1985:** Table III, "Cause-specific SMRs..." (Table A4), show insignificant, slight, increase in prostate cancer. Table V, "Relation of mortality to radiation exposure..." (Table A5), prostate cancer excess deaths at >100 mSv, lower than expected at <10 and 10-20 mSv (0.70 and 0.35) with slight, insignificant excess at 10-20 mSv. The data again show more of a hormesis (beneficial) response than a linear response.<br>Beral et al, 1985 does not support the 'finding' posited.<br><br>NOTE ALSO: **Gilbert et al 1993a** Table III (Table A6) shows a negative trend for prostate cancer in the Hanford, ORNL, Rocky Flats workers, and Gilbert et al 1989 notes that "Other cancers found to exhibit correlations with radiation in the two British studies, were cancer of the prostate in UKAEA workers (Beral et al, 1985) ... Neither of these findings were supported by the ...Hanford data."<br><br>**The above NOTE is generally applicable to these sources. The data in referenced papers posited to 'find' cancer increases in a selected cancer, are directly contradicted by the data in other studies that find those same cancers with no increase, or even a decrease. (This is without reporting on the larger literature that refutes the biased methods and conclusions presented here.) Review the data on specific cancers in the Tables that are referred to associated with for specific cancers. (Specific referenced cancers are underscored, other referenced cancers in others studies are identified by arrows.)**<br>**A subsequent draft revision of this report may explicitly identify this contradictory data in these Tables vs. the proposed 'findings' of selected cancer increases while ignoring the reports of other cancers 'found' to be 'increased' in other studies.** |

"multiple myeloma (Gilbert et al., 1993a, Smith and Douglas, 1986, Gilbert et al 1989, Gilbert et al 1993b),"

**Gilbert et al., 1993a:** Table III, "Results of Analyses of Specific Types of Cancer..." (Table A6) shows Multiple myeloma cancer excess at high doses (5/2.1 obs/exp at >50 mSv), as expected at <10 mSv (18/17.9), and lowest at 10-50 mSv (2/4.9). If the data show anything, it is a stronger hormesis (beneficial) response than a linear response.

**Smith and Douglas, 1986:** "Radiation workers had lower death rates from all causes than other workers, but the death rate from cancer in the two groups were similar. Compared with the general population radiation workers had statistically significant deficits of liver and gall bladder cancer, lung cancer and Hodgkin's disease. There were excesses of deaths myeloma (7 obs, 4.2 exposure) and prostatic cancer (19 obs, 15.8 exposure) but these were not significant and there was no evidence of an excess of leukemia (10 obs, 12.2 exposure) or cancer of the pancreas (15 obs, 17.8 exposure). For no type of cancer was the ratio of obs/exp deaths significantly different between radiation and non-radiation workers. For non-neoplastic conditions radiation workers in general lower death rates than other workers, and for none of the causes of death examined was the mortality significantly higher among radiation workers." Table VII, "Observed (O) and Expected (E) deaths form specific cancers among radiation and other workers and SMRs" (Table A7) shows "Multiple myeloma and other cancers". The "linear trend is significant for multiple myeloma only for doses lagged 15 years". Table XII "Deaths from selected causes among radiation workers by radiation dose accumulated 15 or more years previously..." (Table A8) shows that the primary influence for a linear trend is from 1 death occurring where 0.2 are expected at the highest dose, >400 mSv. Note: the Trend test is positive because the "computer simulation one-tailed p test" effectively ignores the lower-than-expected value, **presuming** the linear model in order to demonstrate the linear model results.

**Gilbert et al 1989:** Table 3 "Results of analyses of external exposures in monitored Hanford Site workers (deaths 1955-1981 on a 10-year lag and 1947-1981 on a two-year lag" (Table A9) shows a multiple myeloma trend test statistic of 4.40. The data show 11 Obs/12.7 Exp deaths for <20 mSv, 0/0.9 lower than normal for 20-50 mSv, and 3/0.5 at >50 mSv. If anything, a lower-than-normal hormetic effect more strongly than a linear model."

Table 8: "Results of analyses of exposures of monitored Hanford Site workers..." (Table A10) shows a multiple myeloma trend test statistic 2.48 for exposure lagged for 10 years, but with 14/15.7 obs/exp <20 mSv, 0/1.2 at 20-50 mSv, and 4/1.1 at > 50 mSv. If anything, the data show a lower-than-normal hormetic effect more strongly than a linear model. Ignoring the hormetic curve, and arbitrarily applying a linear model, the report states: "The relative risk for multiple myeloma...was 55% per 10 mSv...may be compared with a risk of 0.51% per 10 mGy from the A-bomb survivor data" and "comparisons are inexact, but (Hanford data) are inconsistent with...A-bomb survivor data". ... "workers at Sellafield identified as statistically significant correlation (Smith and Douglas, 1986 [see above])The correlation was significant when exposures were lagged 15y but was not close to significant with 0 or 2y lags. 2 of the deaths contributing to the correlation had exposures that exceeded 500 mSv. In UK workers (Beral et al, 1985 [see above]) there was no indication of a statistically significant correlation of multiple myeloma with radiation exposure. Studies of workers at Oak Ridge (Checkoway et al, 1985) and Rocky Flats (Wilkinson et al, 1987) reported only one death each from multiple myeloma. These negative findings may result from lack of power and are not necessarily inconsistent."

"Evidence for radiation-induced multiple myeloma was reviewed by Cuzick (1981), who noted an excess of myeloma in most cohorts...studied, including A-bomb survivors exposed to more than 1 Gy [100 rad!]Miller and Beebe provided a brief review and suggested that diagnostic bias may have contributed to some of the observed excesses summariaed by Cuzick. They noted that if the association were real the minimal latent period for multiple myeloma might be as long as 15 y, consistent with Hanford and Sellafield. The NIH Working Group to Develop Radioepidemiological Tables (1985) noted that the size of the reproted excess in various studies was marginal regardless of the size of the dose, and this group did not include multiple myeloma as a cancer for which this group did not include multiple myeloma as a cancer for which risk estimates were developed."

**Gilbert et al 1993b:** Table 5 "Results of analyses of external dose in monitored workers at the Hanford site..." (Table A11) shows multiple myeloma has a low 10y lag Trend test statistic of 1.54, and that at <10 mSv there are 17/17.0 obs/exp, with a lower 2/4.9 at the 10-50 mSv, and 5/2.1 obs/exp at > 50 mSv."

Table 9: "Results of analyses of external dose for selected cancer categories...noted on the death certificate, but not considered to be the underlying cause of death" (Table A12) shows a multiple myeloma trend test 2.50 (again a biased "computer simulation one-tailed p test") with 0-10 mSv dose at 17/17.3 obs/exp, 2/6.1 at 10-50 mSv (lower than expected), and 7/2.6 obs/exp >50 mSv. Additional analyses show identical results, with lower doses in th mid-dose range in Tables 10 and 11 (Tables A13 and A14).

Table 6 "Relative risks by external dose category..." (Table A17) shows a risk of 1.0 at <10 mSv, 0.4 at 10-50 mSv, and 4.2 at 50-100 mSv, 5.9 at 100-200 mSv, and 21 at >200 mSv. Again, this limited data shows that a hormetic effect is more likely than a linear dose-response in this data.

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| "lymphatic hemopoietic neoplasms and bladder cancer (Smith and Douglas, 1986)." | **Smith and Douglas, 1986):** See report at ...uple myeloma" above on the contrary conclusions of this study. Table XII (Table A8) shows that "All lymphatic and haem..atopoietic ..arcinomas" are 13/12.8 obs/exp at <10 mSv, and a total of 11/13.7 at 10-400 mSv, with 3/0.5 at >400 mSv (providing the influence to the calculated (biased) "linear trend" result. |
| "leukemia (Wilkinson and Dreyer, 1991)." | **Wilkinson and Dreyer, 1991:** This study attempts to combine many nuclear worker populations (except the US Nuclear Shipyard Worker Study, which has the most significant population, the best dosimetry, and the fewest confounding factors that limit other early worker studies). Table 3 "Rate Ratios for Leukemia by Dose Category..." (Table A16) shows that the rate/100,000 for the total population is 4.9 at <10 mSv, 10.1 at 10-50 mSv, and 6.7 at >50 mSv. This conflicts with the "linear model". In addition, by reviewing the variations in each dose group in each study population, there is no consistent basis to believe that radiation could be contributing to leukemia in these populations. |
| "lung cancer (Rinsky et al., 1988, Checkoway et al., 1988)." | **Rinsky et al., 1988:** "Analysis of data on radiation exposure, controlling for exposures to asbestos and welding, found reductions in initial estimates of radiation risk at all levels of ..adiation exposure...suggests that radiation workers were more heavily exposured to asbestos and welding fumes than were other workers and that those exposures confounded the observed association between radiation and lung cancer. Analysis of mortality by time since first exposure to radiation revealed no pattern of progressive increase as latency increased... The results of this study do not preclude a possible association between radiation exposure at the Portsmouth Naval Shipyard and excess mortality from lung cancer. However, they provide no evidence in support of such a relation."<br><br>**Checkoway et al., 1988:** "Dose-response trends were detected for lung cancer mortality with respect to cumulative alpha and gamma radiation, with the most pronounced trend occurring for gamma radiation among workers who received >5 rem of alpha radiation. These trends diminished in magnitude when a 10-year latency assumption was applied. Under a zero-year latency assumption, the rate ratio for lung cancer mortality associated with joint exposure of >5 versus <1 rem of both types of radiation is 4.60 (95% confidence limits (CL) 0.91, 23.35), while the corresponding result, assuming a 10-year latency, is 3.05 (95% CL 0.37, 24.83). While these rate ratios, which are based on three and one death, respectively, lack statistical precision, the observed dose-response trends indicate potential carcinogenic effects to the lung of relatively low-dose radiation." This statement is clearly arbitrary and unsupported on its face by the data based on 1 death presented. Clearly a zero-year latency does not apply to lung cancer even if the data were relevant. The data in the body of the report are similarly unsupportive. |

## "Cancer mortality among Hanford workers:"

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| "Gilbert et al., al. (1993a) found positive associations with dose for 12 types of cancer, those for cancer of the esophagus and the larynx as well as for Hodgkins disease were statistically significant. The study corroborates the conclusion by Kneale and Stewart (1993) of a strong increase of sensitivity for radiogenic cancers with age." | **Gilbert et al., al. (1993a):** Table 4, "SMRs and obs deaths (OBS) for specific cancer types..." (Table A17) shows an SMR of 2.84 in all monitored male workers (0.0 in females) for cancer of the esophagus, 0.60 (1.19, 1 case, in females) for cancer of the larynx, and 0.93 (1.26, 3 cases, in females) for Hodgkin's disease. Table 5, "Results of analyses of external dose in monitored workers on a 10y lag" (Table A18) shows that only Hodgkin's disease "computer simulated, one-tailed test" slight positive "trend test statistic" of 1.54, influenced by having 1 case/0.5 expected at 200+ mSv. The paper does not support the posited 'finding' of support to the "linear model". |

"**Age and Cancer:**
"Kneale and Stewart (1993) found that there was much greater sensitivity to cancer induction by radiation after, rather than before, 50 years of age. For all recorded exposures of Hanford workers, the estimated doubling dose was close to 26 rem, for after 62 years, it was less than 1 rem. This challenges BEIR V which argues that dose rate is more important than exposure age and which states that even a single exposure to 10 rem would only increase the normal cancer risk by four percent. Estimated proportions of radiogenic cancers was much higher for the 175 non-fatal cancers (which had other certified causes of death) than for 1732 fatal cases."

**Kneale and Stewart (1993):** This paper does not present substantive data, but is rather an analysis. Inquiries have found that this paper is largely an unfounded rationalization. Specific reviews of this paper have not been obtained. The paper does not present a substantive basis for the "linear model" in the absense of validating data.

"**Cancer among Oak Ridge Workers:**
"Wing et al., (1991) and Wing et al., (1991 and 1992) studied more than 8000 Oak Ridge Workers (1943-1984) with accumulated occupational doses under 50 cSv for all but 0.2% of the workers us an excess of leukemias among the workforce, compared to the general population and and the incremental relative risk for all than the risk estimate in BEIR V for low-dose exposures if their recommended DREF of 2 for low dose exposures is applied. (A-bomb survivor follow-up studies indicate that a dose rate effectiveness factor (DERF) should not be applied at low dose exposures.)"

**Wing et al., (1991) and Wing et al., (1991 and 1992):** The JAMA study by Wing et al is not science. The data do not support the conclusions in the paper. Numerous technical faults exist in the analysis, including the lack of consideration of confounding factors, internal contamination by this specific group, the lack of linear response (the high-dose group has lower effects than the low-dose group). (Dr. Wing is a sociologist.) Further, Wing presents, in his own paper and words, a telling polemic that: "The low dose carcinogenic impact of ionizing radiation is a topic of great public concern due to fears about cancer and about an invisible exposure that emanates, in part, from secretive industries associated with production of nuclear weapons with high destructive potential...low-dose health effects...should be placed in the context in which it occures. Other factors studied,... showed much stronger relationships to mortality than does radiation, and only a few percentage points can be statistically attributed to external penetrating radiation. Conversely, while factors other than radiation clearly predominate the statistical analysis of mortality [illegible] this population, the public health impact of these radiation exposures and the industry that produces them extend far beyond the low-dose occupational exposures themselves, which are estimated to constitute only 0.3% of the population dose of the U.S. The exposure of workers in this setting, and any attending health effects depends on the historical development of an industry linked to a concentration of resources in military spending, which iteself has gross health effects. By providing aa alternative to fossil fuels...the industry encourages ever increasing energy consumption, a factor of potential health effects of global climate change..." Substantial work exists to discount the technical significance of the work, and more importantly the political "conclusions" presented by Wing.

"**Mutational effects among radiotherapy technicians:**
"Messing et al., (1989) investigated whether mutant frequency in peripheral T-lymphocytes of radiotherapy technicians exposed on the average to 0.3 cGy per month of cobalt-60 gamma radiation can be associated with recently absorbed dose. (Controlled experiment) Finding: Mutation frequency is linearly correlated with dose in the range from 0-0.7cGy."

**Messing et al., (1989)** presents cellular data confirming well-established scientific evidence that radiation exposure can be identified by examing T-lymphocytes. This result indicates [illegible] ability to identify exposures in patients, workers and in research that tracks well in selected cases with standard dosimetry. No adverse result is implicated in the identification. The nature of the cellular changes is consistent with many environmental causes, including medicines, and may indicate improved function as well as degraded function. Further, the study confirms that the effect disappears after approximately 6 months in the case of the radiotherapy technicans (who had received 13.4 to 77.8 mSv, 1340 to 7780 mrem exposure). Note that this was in comparison to physiotherapy technicans selected because they would have least likely exposure to other mutagens such ethylene oxide, anticancer drugs or anesthetic gases would wold produce equivalent mutagenic (normal) responses in the exposed population.

**"Cancers among commercial airline pilots:**
"Airline pilots, subject to cosmic radiation, accumulate yearly does up to about 1 cGy, or the equivalent of three to four times natural background for the average U.S. citizen (Barish, 1990). A cancer mortality and incidence study among about 900 Canadian males pilots showed significant excess rates for several cancers including Hodgkins disease and nonmelanoma skin cancer (Band et al., 1990). High altitude exposure and/or aviator status also correlate significantly with cancerous conditions of the skin, testicles, bladder, and thyroid in a study of U.S. pilots (Krain, 1991). A study of chromosome aberrations induced in lymphocytes of pilots and stewardesses also confirms effects of very low-dose exposures in this occupation (Scheid et al., 1993)"

**Barish, 1990,** states that "flight attendants and pilots on ... aircraft can receive annual doses approaching 10 mSv/y [1000 mrem/y]. I argue that flight **crewmembers** should receive specific education..., that a suitable dosimeter ...be employed" and goes to argue that "frequent flyers" be similarly educated, and that special attention be paid to times of solar proton events, especially for pregnant crew members. No indication of any health effect from such doses, which are less then populations in high radiation background areas of the world with no adverse effect, is presented.

**Band et al., 1990,** present general data on mortality and cancer incidence in a group of commercial airline pilots, stating that "Excess deaths were observed for aircraft accidents, brain cancer, and rectal cancer. Excess cancer incidence was noted for non-melanoma skin cancer, brain cancer, and Hodgkin's disease. These findings suggest an excess risk for certain cancers in pilots, are based on small numbers, and need to be confirmed in larger cohort studies." The group were pilots employed for 1 year or more by Canadian Pacific Airlines, finding a small group (891), and arbitrarily comparing the group to the British Columbia population. No test for longevity of service is reported, so no "dose-response" is possible. Numerous potential causes, of which radiation was identified as a potential cause, provides no basis for this study to indicate any radiation relationship.

**Krain, 1991,** states: "High altitude exposure and/or aviator status correlate significantly with cancerous conditions of the skin, testicles, bladder, and thyroid based on a literature review and survey of government sources. Other lesser significantly associated conditions include leukemia, lymphosarcoma, and Hodgkin's disease. Although radiation and sunlight are strongly associated with cancer incidence and risk at high altitudes, other intervening variables are discussed and critically reviewed." The study identifies numerous contibutors and associations and makes no indication of an association with radiation.

**Scheid et al., 1993** identify chromosome aberrations in flight personnel. There is a general discussion of radiation as a contributor. There is no indication of adverse health effects. Much higher indications of chromosome aberrations exist in exposed radiation workers, and other highly exposed groups for which no adverse health effects are identified at moderate doses. Scheid et al provides no indication of an association between radiation exposure and adverse health effects.

**"Hormesis: Some studies have claimed lower cancer mortality rates in geographic locations with higher background exposures:"**

"Weinberger et al., (1987:388) noted, however, that when such studies are adjusted linearly for altitude, the negative correlations between mortality and background radiation all disappear and become positive. They concluded that they could see no support for the claim that ionizing radiation is beneficial at low doses."

**Weinberger et al., (1987:388):** Since cosmic radiation is an altitude effect, eliminating altitude as a confounding variable will necessarily make the analysis eliminate the radiation effect. The "analysis" acknowledges that higher altitude/cosmic radiation are associated with lower cancer, but just that we can't take credit for the radiation as the responsible contributing agent. On the other hand, this applies to the aircrews proposed as arguing for a radiation effect of altitude. The rationale is that lower pressure is responsible for the beneficial health effect of altitude. This is totally contradicted in animal experiments. (See Section 7.1)

"Hatch and Susser (1990) found a significant association between childhood cancer incidence and a variation in annual external background gamma ray dose rate by nearly a factor of two (0.05-0.092 cGy per year) over an area within a radius of approximately 10 miles for the Three Mile Island nuclear plant. The study found a 50% increase in estimated annual background gamma ray dose."

**Hatch and Susser, 1990** state: Our incidence data contain an ambiguity, however. The numbers of leukemias observed in children were just over half those expected from national and regional rates ... we reviewed records at refferral centers as well as local hospitals. Thus the reason for the low incidence is ... unknown. For mortality from childhood leukemia, the rates are not lower than expected and they show no association with exposure to background radiation ..." and "In the data preseted here, radiation exposure has been assigned from address at diagnosis or death which is less than ideal."

This is an anomolous result of scatter in a study that is not medically significant. It could be the basis for a medical analysis and an actual epidemiological study that would examine the health and histories of the identified population. Hatch and Susser, 1990, does not support an association of childhood leukemia with radiation.

"Andersson and Chiangmai (1992) found that there was no adaptive response (hormetic effect) of Chinese hamster ovary cells exposed to very low doses (0.02 GY, X-ray) of ionizing radiation. Pre-treatments of Chinese Hamster ovary cells with a low, conditioning dose of ionizing radiation did not render the cells more resistant to the induction of chromotid aberrations by a subsequent, higher, challenging dose of ionizing radiation."

**Andersson and Chiangmai, 1992** is a study of cells in culture that assesses resistence to induction of chromatid aberations (not health or other physiological effects). The paper does accept, and state that Samson and Cairns (1977) identified " 'adaptive response'... as an inducible response that occurs during growth in the presence of low levels of a mutagenic alkylating agent and enables cells both to survive better and to be less mutated than control cultures during a subsequent challenge with a higher dose of mutagen ... (S)imilar protective effects against exposure to comparatively high concentrations of mutagens have been found ... also mammalian and plant cells. At the molecular level, the adaptation ... has been related to the induction of ... repair enzymes. The fact that radiation is also able to provoke an analogous adaptive response was shown ... (from) low adapting doses of ionizing radiation from incorporated (tritium) or x-rays, thereby making them less susceptible to induction of chromatid aberrations by a ... challenging dose of x-rays. ... These observations have recently been confirmed by other investigators... Though (speculation is) that the adaptive response ... Depends on the induction of enzymes important for DNA double-strand break repair, very little is actually known."

This paper ackowledges and accepts the proof of the existence of adaptive response, ie, hormesis, and reports on a specific study that did not indicate adaptive response in particular cells under particular conditions. This paper is focused on the science of testing hypotheses and explanations for the known adaptive, hormetic response, not whether the response exists.

Andersson and Chiangmai, 1992 does not support the posited 'finding' of the lack of hormesis that it purports to show.

"Final point

"Many processes that result in low level radiation exposure also bear the concurrent potential for a disastrous high level radioactive release ( Marshall, 1990). The risk of such a high level catastrophe play an obvious role in the formulation of public policy concerning the issue of low level ionizing radiation."

**Marshall, 1990,** is a news article that states that **"Half the workers at the Cheliabinsk site in the Ural Mountains east of Moscow were routinely receiving 100 rem/y** in the late 1940s and early 1950s. The consequences of the very large doses to workers in the USSR are not fully revealed in the Nikipelov report. But, it tantalizingly mentions that 8 to 9% of the staff who began work before 1958 and received high radiation doses (more than 100 rem) died of cancer. In addition, the... Report says that nearly a quarter of the workers between 1950 and 1952 were suffering from "chronic radiation disease," which (Ralph) Lapp takes to mean blood disorders. Although Nikipelov does not give the numbers, he mentions that cancer mortality among severely exposed workers (100 rem and above) was 88% higher than among those who received less than 100 rem. Nikipelov explains that...managers realized they had 'underestimated the radiation factor' and appealed for permission to improve conditions. No changes were made until 1952 however, (with) new safety standards, but exceptions were always given for urgent repairs."

It is not credible that the 'risk' indicated by >100 rem/yr exposure of 1940s/50s USSR high-dose radiation conditions is an indication of a "concurrent potential for disastrous high level radioactive release" or that "the risk of such a high-level catastrophe play an obvious role in the formulation of public policy concerning...low level ionizing radiation."

A1

Table 11 *Registrations of selected non-fatal cancers in ex-employees with a radiation record by cumulative whole body radiation exposure, 1971–9 (adjusted for age, sex, social class, calendar period, and authority establishment*). Missing and below threshold exposures estimated (assumption B)*

| Cancer site (ICD code, 8th revision) | Cumulative whole body exposure (mSv) | | | | | | | | | | Test for trend | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | < 10 | | 10– | | 20– | | 50– | | ≥ 100 | | | |
| | O/E† | (O) | O/E | (O) | O/E | (O) | O/E | (O) | O/E | (O) | SND statistic | p Value |
| All malignant neoplasms (140–209) | 0·92 | (51) | 1·13 | (19) | 0·99 | (21) | 1·17 | (10) | 1·13 | (7)‡ | 0·67 | 0·5 |
| Intestinal cancer (152–153) | 0·79 | (4) | 1·67 | (3) | 1·79 | (4) | — | (0) | — | (0) | −1·01 | 0·3 |
| Skin cancer (172–173) | 0·96 | (14) | 0·98 | (5) | 0·53 | (4) | 1·38 | (3) | 2·40 | (4) | 1·41 | 0·06 |
| Breast cancer (174) | 0·82 | (7) | 1·16 | (2) | 1·35 | (2) | 4·18 | (1) | — | (0) | 0·46 | 0·6 |
| Prostatic cancer (185) | 0·67 | (3) | 1·43 | (3) | 0·88 | (2) | 1·51 | (1) | 1·68 | (1) | 0·75 | 0·5 |
| Testicular cancer (186) | 2·80 | (1) | — | (0) | — | (0) | 14·06 | (1) | — | (0) | 1·24 | 0·2 |
| Bladder cancer (188) | 0·91 | (6) | — | (0) | 1·80 | (3) | — | (0) | 3·31 | (3) | 1·89 | 0·06 |
| All lymphatic and haematopoietic neoplasms (200–209) | 1·21 | (3) | 1·48 | (1) | 1·16 | (1) | — | (0) | — | (0) | −0·98 | 0·3 |
| Leukaemia (204–207) | 2·45 | (2) | — | (0) | — | (0) | — | (0) | — | (0) | −0·97 | 0·3 |

*Excludes Dounreay employees.
†O/E = Observed registrations divided by expected registrations. (Observed registrations (O) are shown in parentheses.) Expected registrations are calculated using the registration rates in the total population analysed.
‡The difference between this total and the sum for the specific cancer sites is explained by multiple cancer in the same individual.

A2

Table 5 *Mortality by cumulative whole body exposure for selected causes of death, 1946–79, lagged by 15 years and estimating below threshold and missing values (adjusted for age, sex, social class, calendar period, and authority establishment*)*

| Cause of death (ICD code, 8th revision) | Cumulative whole body exposure (mSv) | | | | | | | | | | Test for trend | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | < 10 | | 10– | | 20– | | 50– | | ≥ 100 | | | |
| | O/E† | (O) | O/E | (O) | O/E | (O) | O/E | (O) | O/E | (O) | SND statistic | p Value |
| All malignant neoplasms (140–209) | 0·99 | (305) | 0·99 | (39) | 1·03 | (45) | 1·42 | (19) | 0·57 | (5) | 0·45 | 0·7 |
| Intestinal cancer (152–153) | 1·04 | (20) | 1·37 | (3) | 0·39 | (1) | — | (0) | 2·98 | (1) | 0·72 | 0·8 |
| Lung cancer (162) | 1·01 | (116) | 0·85 | (12) | 1·17 | (18) | 1·01 | (5) | 0·32 | (1) | 0·95 | 0·3 |
| Prostatic cancer (185) | 0·78 | (12) | 0·30 | (1) | 1·25 | (4) | 6·59 | (5) | 2·33 | (2) | 2·93 | 0·003 |
| All lymphatic and haematopoietic (200–209) | 1·09 | (25) | 0·39 | (1) | 1·00 | (3) | 1·16 | (1) | — | (0) | −0·77 | 0·5 |
| Multiple myeloma (203) | 1·29 | (2) | — | (0) | — | (0) | 7·06 | (1) | — | (0) | 0·24 | 0·8 |
| Leukaemia (204–207) | 1·09 | (11) | 0·99 | (1) | 0·80 | (1) | — | (0) | — | (0) | −0·93 | 0·3 |
| All causes (000–999) | 0·96 | (1033) | 1·14 | (169) | 1·11 | (182) | 1·25 | (59) | 0·79 | (24) | 0·46 | 0·7 |

*Excludes Dounreay employees.
†O/E = Observed deaths divided by expected deaths. (Observed deaths (O) are shown in parentheses.) Expected deaths are calculated using the mortality rates in the total population analysed.

A3

Table 9 *Mortality by cumulative surface exposure for selected causes of death, 1946–79, (adjusted for age, sex, social class, calendar period, and authority establishment*)*

| Cause of death (ICD code, 8th revision) | Cumulative surface exposure (mSv) | | | | | | | | | | Test for trend | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | < 10 | | 10– | | 20– | | 50– | | ≥ 100 | | | |
| | O/E† | (O) | O/E | (O) | O/E | (O) | O/E | (O) | O/E | (O) | SND statistic | p Value |
| All malignant neoplasms (140–209) | 0·92 | (175) | 0·89 | (65) | 1·05 | (109) | 1·09 | (57) | 1·20 | (81) | 2·30 | 0·02 |
| Intestinal cancer (152–153) | 1·06 | (13) | 0·43 | (2) | 1·02 | (3) | 1·21 | (4) | — | (0) | 0·66 | 0·5 |
| Lung cancer (162) | 0·95 | (66) | 0·81 | (22) | 0·97 | (37) | 1·29 | (24) | 1·18 | (29) | 1·44 | 0·1 |
| Breast cancer (174) | 1·16 | (3) | 1·19 | (1) | — | (0) | — | (0) | — | (0) | −0·75 | 0·5 |
| Prostatic cancer (185) | 0·67 | (6) | 0·57 | (2) | 0·51 | (3) | 1·03 | (3) | 2·88 | (11) | 4·16 | <0·001 |
| All lymphatic and haematopoietic neoplasms (200–209) | 0·80 | (13) | 1·56 | (9) | 0·81 | (7) | 0·44 | (2) | 1·72 | (10) | 1·44 | 0·1 |
| Non-Hodgkin's lymphoma (200, 202) | 0·74 | (4) | 1·52 | (3) | 0·36 | (1) | 1·21 | (2) | 1·79 | (4) | 1·40 | 0·2 |
| Multiple myeloma (203) | 0·66 | (1) | 2·00 | (1) | — | (0) | — | (0) | 3·63 | (2) | 1·82 | 0·07 |
| Leukaemia (204–207) | 0·91 | (6) | 1·19 | (3) | 1·46 | (6) | — | (0) | 1·16 | (3) | 0·00 | 1·00 |
| All causes (000–999) | 0·94 | (667) | 0·99 | (280) | 1·10 | (429) | 1·06 | (209) | 0·98 | (254) | 0·37 | 0·8 |

*Includes Dounreay employees.
†O/E = Observed deaths divided by expected deaths. (Observed deaths (O) are shown in parentheses.) Expected deaths are calculated using the mortality rates in the total population analysed.

TABLE II—Cause specific standardised mortality ratios at ages 15-74 years, 1946-79

| Cause of death (ICD code (8th revision)) | Men: Without a radiation record (observed deaths) | Men: With a radiation record (observed deaths) | Women: Without a radiation record (observed deaths) | Women: With a radiation record (observed deaths) | Total (95% confidence interval) |
|---|---|---|---|---|---|
| All malignant neoplasms (140-209) | 84** (—) | 90** (787) | 87* (179) | 101 (28) | 90** (76-45) |
| Stomach cancer (151) | 73 (28) | 76 (46) | 81 (6) | — (0) | 76** (55-88) |
| Intestinal cancer (152-153) | 119 (24) | 81 (26) | 105 (13) | 47 (1) | 96 (76-122) |
| Rectal cancer (154) | 121 (18) | 68 (16) | 18* (1) | 104 (1) | 80 (56-111) |
| Pancreatic cancer (157) | 124 (17) | 81 (18) | 78 (4) | — (0) | 93 (66-127) |
| Lung cancer (162) | 74** (103) | 60** (151) | 99 (18) | 64 (2) | 72** (64-81) |
| Brain cancer (170) | 56 (1) | 73 (2) | 141 (1) | — (0) | 74 (20-190) |
| Connective and soft tissue cancer (171) | 104 (1) | 120 (2) | — (0) | — (0) | 91 (19-266) |
| Breast cancer (174) | 600 (2) | — (0) | 93 (38) | 55 (4) | 89 (65-120) |
| Uterus and ovarian cancer (180-183) | — | — | 68 (19) | 183 (9) | 85 (57-123) |
| Prostatic cancer (185) | 78 (9) | 118 (19) | — | — | 166 (67-146) |
| Testicular cancer (186) | 175 (4) | 142 (6) | — | — | 153 (73-281) |
| Bladder and urinary cancer (188-189) | 88 (15) | 66 (18) | 27 (1) | — (0) | 70* (60-98) |
| Brain and other central nervous system cancer (191-192) | 85 (8) | 30** (5) | 60 (3) | 353 (3) | 60* (36-93) |
| Thyroid cancer (193) | 158 (1) | 193 (2) | — (0) | — (0) | 122 (25-356) |
| All lymphomas and haematopoietic neoplasms (200-202) | 99 (22) | 92 (35) | 136 (14) | 159 (3) | 102 (80-128) |
| Non-Hodgkin's lymphoma (200,202) | 88 (5) | 121 (12) | 119 (3) | — (0) | 107 (66-166) |
| Hodgkin's disease (201) | 69 (3) | 39 (3) | 177 (3) | 295 (1) | 71 (34-131) |
| Multiple myeloma (203) | 134 (4) | 60 (3) | 69 (1) | — (0) | 83 (36-163) |
| Leukaemia (204-207) | 115 (10) | 110 (16) | 160 (7) | 247 (2) | 123 (86-171) |
| All benign and unspecified neoplasms (210-239) | 163 (7) | 43 (3) | 42 (4) | — (0) | 96 (52-161) |
| All diseases of the nervous system (320-389) | 50* (8) | 62 (16) | 42 (4) | — (0) | 53** (35-76) |
| All diseases of the circulatory system (390-458) | 82** (691) | 80** (764) | 70** (116) | 65 (18) | 80** (76-84) |
| All diseases of the respiratory system (460-519) | 46** (69) | 49** (109) | 45** (16) | 82 (5) | 48** (41-55) |
| All diseases of the digestive system (520-577) | 35** (13) | 62** (35) | 68 (9) | — (0) | 52** (40-68) |
| All diseases of the genitourinary system (580-629) | 42** (8) | 61 (23) | 59 (5) | 68 (1) | 55** (46-67) |
| Hyperplasia of prostate (600) | 32 (1) | 131 (5) | — | — | 86 (32-18[illegible]) |
| Accidents, suicide, and violence (800-999) | 88 (80) | 84** (102) | 76 (22) | 92 (5) | 79** (65-96) |

Significance of difference from 100: *p < 0·05; **p < 0·01.

TABLE V—*Relation of mortality from selected causes of death to cumulative radiation exposure (adjusted for age, sex, social class, calendar period, and United Kingdom Atomic Energy Authority establishment). Results expressed as ratio of observed to expected deaths (observed numbers of deaths in parentheses)*[*]

| Cause of death (ICD code (8th revision)) | Cumulative exposure (mSv): <10 | 10– | 20– | 50– | ≥100 | p Value, test for linear trend (direction of trend) |
|---|---|---|---|---|---|---|
| All malignant neoplasms (140-209) | 0·96 (291) | 1·06 (54) | 1·17 (58) | 1·23 (35) | 0·99 (35) | 0·5 (+) |
| Stomach cancer (151) | 1·01 (32) | 1·41 (8) | 0·70 (4) | 0·87 (3) | 0·89 (4) | 0·7 (−) |
| Intestinal cancer (152-153) | 0·95 (19) | 0·90 (3) | 0·85 (3) | 0·93 (2) | 1·99 (5) | 0·1 (+) |
| Pancreatic cancer (157) | 1·19 (17) | 0·47 (1) | 0·75 (2) | 0·76 (1) | 0·63 (1) | 0·6 (−) |
| Lung cancer (162) | 0·95 (103) | 0·98 (19) | 1·33 (25) | 1·26 (13) | 0·77 (10) | 0·8 (−) |
| Bone, soft tissue, and thyroid cancer (170, 171, 193) | 0·78 (2) | 1·52 (1) | 1·03 (1) | 1·23 (1) | 1·01 (1) | 0·9 (+) |
| Breast cancer (174) | 1·12 (4) | — (0) | — (0) | — (0) | — (0) | 0·6 (−) |
| Uterus and ovarian cancer (180-183) | 0·87 (7) | — (0) | 3·31 (2) | — (0) | — (0) | 0·5 (+) |
| Prostatic cancer (185) | 0·79 (11) | 0·35 (1) | 1·1[illegible] (1) | 1·41 (3) | 3·24 (3) | <0·001 (+) |
| Testicular cancer (186) | 0·86 (4) | 2·06 (1) | [illegible] (1) | — (0) | 3·48 (1) | 0·1 (+) |
| Brain and central nervous system cancer (191-192) | 1·03 (7) | 3·03 (2) | [illegible] (2) | — (0) | — (0) | 0·3 (+) |
| All lymphomas and haematopoietic neoplasms (200-209) | 0·8[illegible] (1) | 0·90 (4) | 1·[illegible] (6) | 2·13 (6) | 1·01 (3) | 0·3 (+) |
| Non-Hodgkin's lymphoma (200, 202) | 0·7[illegible] (1) | 0·76 (1) | 1·29 (2) | 2·21 (2) | 1·37 (2) | 0·2 (+) |
| Hodgkin's disease (203) | 0·8[illegible] (2) | — (0) | 1·72 (1) | 3·57 (1) | — (0) | 0·8 (+) |
| Leukaemia (204-207) | 0·91 (10) | 0·86 (2) | 1·20 (3) | 1·46 (2) | 0·84 (1) | 0·9 (−) |
| All (000-999) | 0·99 (1136) | 1·04 (210) | 1·15 (230) | 0·95 (107) | 0·85 (116) | 0·1 (−) |

*Tables includes deaths at all ages to controls with a radiation record, no members entered those in tables II-IV.
†10 subjects 1 rem.

## TABLE III

### Results of Analyses of Specific Types of Cancer in Monitored Workers Employed at Least 6 Months at the Hanford Site, ORNL, or Rocky Flats

| Cause of death (type of cancer) | Trend test statistic* | Observed and expected deaths by exposure category in mSv (obs/exp)† 0– | 10– | 50– | 100– | 200– | 400– |
|---|---|---|---|---|---|---|---|
| Buccal | −1.58 | 29/27.2 | 10/8.9 | 0/1.5 | 1/1.2 | 0/1.0 | 0/0.2 |
| Esophagus | 2.07‡ | 25/24.8 | 7/9.2 | 2/2.0 | 2/1.2 | 0/0.7 | 2/0.2 |
| Stomach | −0.01 | 60/58.5 | 18/18.4 | 2/3.5 | 3/2.4 | 1/1.9 | 1/0.3 |
| Colon | 0.05 | 135/125.4 | 41/38.7 | 4/6.7 | 6/4.2 | 2/3.3 | 1/0.7 |
| Rectum | 0.30 | 28/27.2 | 10/10.1 | 1/2.0 | 2/1.4 | 0/1.1 | 1/0.2 |
| Liver | 1.25 | 16/16.0 | 4/4.2 | 0/0.7 | 0/0.6 | 2/0.4 | 0/0.1 |
| Gallbladder | −1.18 | 16/13.8 | 4/4.8 | 0/0.7 | 0/0.5 | 0/0.2 | 0/0.0 |
| Pancreas | 0.60 | 72/70.8 | 27/27.3 | 3/4.8 | 4/3.6 | 1/2.2 | 2/0.4 |
| Larynx | 3.42‡ | 12/11.9 | 3/4.3 | 2/0.8 | 0/0.6 | 0/0.4 | 1/0.02 |
| Lung | 0.07 | 325/335.1 | 139/138.2 | 33/26.8 | 25/20.0 | 15/14.7 | 1/3.2 |
| Bone | −0.75 | 4/3.1 | 0/0.6 | 0/0.1 | 0/0.1 | 0/0.03 | 0/0.0 |
| Female breast | −0.05 | 45/46.5 | 7/5.3 | 1/0.9 | 1/1.0 | 0/0.3 | 0/0.0 |
| Cervix and uterus | 0.96 | 3/5.0 | 3/0.8 | 0/0.2 | 0/0.06 | 0/0.00 | 0/0.0 |
| Ovary | 0.79 | 12/12.1 | 1/1.5 | 0/0.2 | 1/0.2 | 0/0.04 | 0/0.0 |
| Prostate | −1.32 | 76/78.9 | 43/36.0 | 6/6.9 | 3/4.2 | 2/3.3 | 0/0.7 |
| Bladder | −0.08 | 20/21.6 | 11/9.3 | 1/1.5 | 2/1.3 | 1/1.0 | 0/0.2 |
| Kidney | 0.78 | 35/30.7 | 4/10.4 | 2/2.0 | 4/1.5 | 1/1.1 | 0/0.3 |
| Brain and central nervous system | −1.28 | 43/41.9 | 18/15.8 | 2/3.2 | 2/2.2 | 0/1.5 | 0/0.3 |
| Thyroid | −0.42 | 2/1.7 | 1/1.0 | 0/0.2 | 0/0.1 | 0/0.03 | 0/0.0 |
| Non-Hodgkin's lymphoma | −0.90 | 44/47.3 | 22/16.9 | 3/3.3 | 2/2.3 | 1/1.7 | 0/0.4 |
| Hodgkin's disease | 1.65‡ | 17/18.0 | 3/3.5 | 0/0.6 | 1/0.4 | 2/0.4 | 0/0.1 |
| Multiple myeloma | 1.53‡ | 18/17.9 | 2/4.9 | 2/0.9 | 1/0.6 | 2/0.5 | 0/0.1 |
| Chronic lymphatic leukemia (CLL) | −0.31 | 8/9.1 | 5/3.7 | 2/1.0 | 0/0.5 | 0/0.3 | 0/0.1 |
| Leukemia excluding CLL | −0.47 | 41/41.0 | 20/17.6 | 1/3.5 | 3/2.5 | 1/2.0 | 1/0.5 |

*Note*: Doses lagged for 10 years.

* The trend test statistic was calculated from individual doses, not the six exposure categories. It may be compared with a standard normal distribution to assess statistical significance. However, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote ‡.

† Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, sex, number of years monitored, and general socioeconomic category.

‡ Based on computer simulations, the one-tailed *P* values associated with the trend test were estimated to be 0.015 for cancer of the esophagus, 0.019 for cancer of the larynx, 0.048 for Hodgkin's disease, and 0.100 for multiple myeloma.

TABLE VII—*Observed (O) and expected (E) deaths from specific cancers among radiation and other workers and standardised mortality ratios (S.M.R.)*

| Cancer site (ICD codes (8th revision))† | Radiation workers | | | Other workers | | | All workers | | | All workers (S.M.R. based on Cumberland rates) |
|---|---|---|---|---|---|---|---|---|---|---|
| | O | E | S.M.R. | O | E | S.M.R. | O | E | S.M.R. | |
| Lip (140) | 0 | 0·19 | — | 0 | 0·09 | — | 0 | 0·27 | — | — |
| Tongue (141) | 2 | 1·58 | 145 | 0 | 0·58 | — | 2 | 1·95 | 103 | — |
| Mouth and pharynx (143–149) | 2 | 4·15 | 48 | 3 | 1·73 | 173 | 5 | 5·88 | 85 | — |
| Oesophagus (150) | 16 | 11·79 | 136 | 4 | 4·54 | 88 | 20 | 16·33 | 122 | 124 |
| Stomach (151) | 50 | 43·36 | 115 | 21 | 18·55 | 113 | 71 | 61·92 | 115 | 97‡ |
| Small intestine (152) | 1 | 0·90 | 111 | 0 | 0·38 | — | 1 | 1·28 | 78 | — |
| Colon (153) | 30 | 25·19 | 119 | 12 | 12·65 | 95 | 42 | 37·83 | 111 | 100‡ |
| Rectum (154) | 18 | 18·66 | 97 | 12 | 8·28 | 145 | 30 | 26·92 | 111 | 108 |
| Liver and gall bladder (155–156, 197·7, 197·8) | 2 | 7·25 | 28** | 2 | 3·25 | 62 | 4 | 10·50 | 38** | 35 |
| Pancreas (157) | 15 | 17·82 | 84 | 9 | 7·33 | 123 | 24 | 25·15 | 95 | 92 |
| Larynx (161) | 1 | 3·89 | 26 | 1 | 1·64 | 69 | 2 | 5·33 | 38 | — |
| Lung (162–163) | 147 | 169·22 | 87** | 58 | 58·03 | 100 | 205 | 227·24 | 90 | 104‡ |
| Bone (170) | 1 | 1·80 | 56 | 1 | 0·82 | 122 | 2 | 2·62 | 76 | — |
| Connective tissue (171) | 2 | 1·36 | 147 | 0 | 0·55 | — | 2 | 1·91 | 105 | — |
| Melanoma (172·0–172·4, 172·6–172·9) | 3 | 2·92 | 101 | 1 | 1·25 | 80 | 4 | 4·23 | 95 | — |
| Breast (174) | 4 | 3·88 | 103 | 10 | 15·28 | 65 | 16 | 19·15 | 73 | 82‡ |
| Uterus (180–182) | 0 | 1·06 | — | 5 | 5·14 | 97 | 5 | 6·20 | 81 | 82 |
| Ovary (183) | 1 | 1·03 | 97 | 4 | 4·96 | 81 | 5 | 6·00 | 83 | 88 |
| Other female genital (184) | 0 | 0·07 | — | 0 | 0·40 | — | 0 | 0·47 | — | — |
| Prostate (185) | 19 | 15·85 | 120 | 4 | 6·23 | 64 | 23 | 22·08 | 104 | 123 |
| Testis (186) | 4 | 2·55 | 157 | 1 | 0·62 | 161 | 5 | 3·16 | 158 | — |
| Other male genital (172·5, 187) | 0 | 0·67 | — | 0 | 0·22 | — | 0 | 0·89 | — | — |
| Bladder (188) | 16 | 14·19 | 99 | 3 | 5·61 | 53 | 17 | 19·81 | 86 | 105 |
| Kidney (189) | 6 | 7·68 | 78 | 1 | 2·77 | 36 | 7 | 10·44 | 67 | 67 |
| Brain and central nervous system (191–192) | 10 | 12·52 | 80 | 4 | 4·56 | 88 | 14 | 17·08 | 82 | 83 |
| Thyroid (193) | 2 | 0·83 | 241 | 0 | 0·49 | — | 2 | 1·31 | 153 | — |
| Ill defined and secondary (195–197·6, 197·9–199) | 17 | 13·96 | 122 | 13 | 5·77 | 225** | 30 | 19·74 | 152** | 132 |
| Non-Hodgkin's lymphoma (200, 202) | 9 | 7·89 | [illegible] | 1 | 3·00 | 33 | 10 | 10·89 | 92 | 104 |
| Hodgkin's disease (201) | 1 | 5·04 | 2** | 2 | 1·84 | 109 | 3 | 6·88 | 44 | — |
| Multiple myeloma (203) | 7 | 4·23 | 16 | 2 | 1·74 | 115 | 9 | 5·96 | 151 | 134 |
| Leukaemia (204–209) | 10 | 12·21 | 82 | 1 | 5·16 | 19** | 11 | 17·34 | 63 | 68 |
| Other neoplasms (140–20[illegible]ading above) | 2 | 5·38 | 37 | 1 | 2·33 | 43 | 3 | 7·71 | 39 | — |
| All malignant neoplasms (140–209) | 396 | 418·89 | 95 | 176 | 185·69 | 95 | 572 | 604·38 | 95 | 97‡ |

*p<0·05; **p<0·01; ***p<0·001.
†ICD categories used for deaths occurring when 6th, 7th, or 9th revisions was in operation is available from authors.
‡Age specific Cumberland S.M.Rs estimated (for other causes shown, only overall S.M.Rs for subjects aged 15 or more used to estimate Cumberland rates).

TABLE XI—*Deaths from selected causes among radiation workers by radiation dose accumulated 15 or more years previously (adjusted for age, sex, calendar period, and social class). (Figures in parentheses are expected distribution of deaths assuming no relation between accumulated dose and mortality risk.) [Figures in square brackets are expected numbers of deaths based on death rates in England and Wales]*

| Cause of death (ICD codes (8th revision)) | Radiation dose accumulated (mSv) (lagged by 15 years) | | | | | | | | | | | | Total deaths | z Value for trend† (estimated p value) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | <10 | | 10- | | 20- | | 50- | | 100- | | 200- | | ≥400 | |
| Carcinoma bladder (188) | 3 | (4·8) [6·2] | 2 | (0·9) [1·0] | 1 | (2·3) [2·2] | 1 | (2·0) [1·8] | 3 | (1·9) [1·5] | 2 | (1·4) [1·0] | 2 | (0·7) [0·4] | 14 | (14·0) [14·2] | 2·13 ($p=246/10^4$) |
| Multiple myeloma (203) | 2 | (3·2) [1·8] | 0 | (0·4) [0·3] | 1 | (0·9) [0·5] | 2 | (0·8) [0·5] | 0 | (0·8) [0·5] | 0 | (0·7) [0·3] | 2 | (0·2) [0·1] | 7 | (7·0) [4·2] | 2·66 ($p=115/10^4$) |
| Leukaemia (204–209) | 6 | (6·0) [6·8] | 0 | (0·7) [0·8] | 3 | (1·4) [1·5] | 0 | (1·1) [1·2] | 0 | (0·6) [1·0] | 0 | (0·3) [0·6] | 1 | (0·04) [0·22] | 10 | (10·0) [12·2] | 1·76 ($p=599/10^4$) |
| All lymphatic and haematopoietic carcinomas (200–209) | 13 | (12·8) [16·4] | 1 | (1·9) [1·9] | 5 | (4·0) [3·6] | 2 | (3·4) [2·9] | 2 | (2·7) [2·5] | 1 | (1·7) [1·6] | 3 | (0·5) [0·5] | 27 | (27·0) [29·4] | 2·04 ($p=345/10^4$) |
| All malignant neoplasms (140–209) | 180 | (181·9) [201·2] | 20 | (26·9) [30·1] | 54 | (57·1) [59·8] | 34 | (48·4) [48·6] | 47 | (42·2) [41·8] | 30 | (22·9) [27·7] | 11 | (10·6) [9·6] | 396 | (396·0) [418·9] | 0·72 — |
| All causes | 696 | (707·5) [794·4] | 105 | (104·1) [111·9] | 205 | (218·1) [225·5] | 194 | (186·6) [185·8] | 174 | (157·2) [152·6] | 102 | (103·8) [98·9] | 40 | (38·7) [34·8] | 1516 | (1516·0) [1603·9] | 0·72 — |
| Person years at risk | 154 535 | | 10 001 | | 17 566 | | 12 236 | | 9547 | | 5246 | | 1231 | | 210 362 | | |

†See footnote to table X.
*Conversion: 51 is traditional unit*—Radiation dose: 10 mSv = 1 rem.

Table 3. Results of analyses of external exposures in monitored Hanford Site workers. Includes deaths 1955–1981 for analyses based on a 10-y lag and deaths 1947–1981 for analyses based on a two-year lag.

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| Cause of death (8th revision ICD[a] code) | Trend test statistic[b] Exposure lagged for 10 y | 2 y | Observed and expected deaths by exposure category in mSv (Based on 10-y lag) 0- Obs./Exp.[c] | 20- Obs./Exp. | 50- Obs./Exp. | 150+ Obs./Exp. |
|---|---|---|---|---|---|---|
| All causes | -1.15 | -1.59 | 4234/4216.7 | 319/333.0 | 195/191.7 | 98/104.5 |
| No certificate | -0.33 | -0.08 | 48/45.8 | 0/1.6 | 0/1.1 | 1/0.5 |
| All non-cancers | -0.94 | -1.60 | 3259/3237.0 | 237/254.9 | 147/145.1 | 73/79.0 |
| All cancers (140-209) | -0.65 | -0.40 | 927/933.9 | 82/76.5 | 48/45.6 | 24/25.1 |
| Male | -0.93 | -0.67 | 808/808.4 | 74/72.6 | 42/42.4 | 24/24.6 |
| Female | 1.67[d] | 1.76 | 119/125.5 | 8/3.8 | 6/3.2 | 0/0.5 |
| Buccal (140-9) | -0.77 | -1.14 | 27/24.8 | 1/2.3 | 1/1.2 | 0/0.8 |
| Stomach (151) | -0.17 | -0.07 | 43/42.1 | 2/3.4 | 2/2.2 | 2/1.3 |
| Colon (153) | -0.80 | -1.07 | 88/89.3 | 9/6.1 | 4/3.6 | 0/2.0 |
| Rectum (154) | -0.90 | -0.25 | 23/21.4 | 1/1.9 | 1/1.1 | 0/0.7 |
| Pancreas (157) | 0.27 | 1.24 | 58/58.7 | 5/5.1 | 3/2.7 | 2/1.5 |
| Other digestive (150, 152, 155-6,158-9) | 0.60 | 0.52 | 50/47.3 | 1/3.0 | 0/1.9 | 2/0.9 |
| Lung (162) | 0.11 | 0.36 | 272/282.5 | 32/26.1 | 20/16.0 | 10/9.3 |
| Female breas. (174) | -0.09 | 1.09 | 35/35.2 | 1/1.0 | 1/0.8 | 0/0.07 |
| Female genital (180-3) | 2.19[d] | 1.66 | 9/11.3 | 2/0.3 | 1/0.3 | 0/0.06 |
| Prostate (185) | -1.05 | -1.29 | 69/66.5 | 4/5.4 | 4/3.1 | 0/2.0 |
| Bladder and kidney (188-9) | -0.49 | -0.53 | 40/38.7 | 2/3.3 | 2/1.9 | 1/1.1 |
| Brain (191) | -0.91 | -0.66 | 25/25.8 | 5/2.2 | 0/1.4 | 0/0.6 |
| Other solid tumors (150-., (163,170-3 190,192-9) | -0.49 | -1.16 | 104/103.6 | 10/9.2 | 4/5.4 | 3/2.8 |
| All lymphatic and haematopoietic cancer (200-9) | 0.86 | 1.20 | 82/84.7 | 7/7.2 | 5/4.1 | 4/2.0 |
| Lymphoma (200-2) | 0.60 | 0.65 | 37/38.7 | 4/3.0 | 2/1.6 | 1/0.7 |
| Multiple myeloma (203) | 4.40- | 3.60 | 11/12.7 | 0/0.9 | 2/0.4 | 1/0.1 |
| Chronic lymphatic leukemia (204) | -0.96 | -0.97 | 8/6.. | 0/0.6 | 0/0.5 | 0/0.2 |
| Leukemiae (205-7) | -0.69 | -0.84 | 24/24.0 | 3/2.5 | 1/1.5 | 1/0.9 |
| Leukemiae (Based on 2-y lag) | | | 28/27.4 | 3/3.3 | 2/1.9 | 1/1.4 |
| Person-years (Based on 10-y lag) | | | 361,017 | 28,531 | 16,867 | 6,979 |

[a] ICD, International Classification of Diseases, Eighth Revision

[b] The trend test statistic was calculated from individual doses, not the four exposure categories. It may be compared with a standard normal distribution to assess statistical significance. However, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote e.

[c] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, sex and length of employment.

[d] Based on computer simulations the one-tailed p-values associated with the trend test with a 10-y lag were estimated to be 0.061 for all cancers in females, 0.046 for female genital cancer and 0.002 for multiple myeloma.

[e] Excluding chronic lymphatic leukemia.

Table 8. Results of analyses of exposures of monitored Hanford Site workers including deaths occurring in the State of Washington 1982–1985 in addition to those presented in Table 3. Exposures lagged for 10 and 2 y[a]

A10

| Cause of death (8th revision ICD[b] code) | Trend test statistic[c] Exposure lagged for 10 y | 2 y | Observed and expected deaths by exposure category in mSv (Based on 10-y lag) 0- Obs./Exp.[d] | 20- Obs./Exp. | 50- Obs./Exp. | 150+ Obs./Exp. |
|---|---|---|---|---|---|---|
| All cancers (140-209) | -0.36 | -0.21 | 1048/1059.4 | 115/102.4 | 60/62.5 | 47/45.7 |
| All digestive cancer (150-9) | 0.15 | 0.47 | 285/287.4 | 30/25.9 | 14/15.5 | 11/11.2 |
| Lung cancer (162) | 0.06 | 0.23 | 303/313.9 | 41/33.3 | 23/20.7 | 16/15.. |
| Prostatic cancer (185) | -0.55 | -0.85 | 76/75.4 | 7/7.6 | 5/4.4 | 3/3.2 |
| All lymphatic and haematopoietic (200-9) | 1.08 | 1.37 | 102/103.1 | 9/10.9 | 7/6.8 | 8/5.2 |
| Multiple myeloma (203) | 2.48 | 3.41 | 14/15.7 | 0/1.2 | 2/0.7 | 2/0.4 |
| Leukemiae (205-7) | 0.03 | -0.33 | 26/27.5 | 4/3.3 | 2/2.2 | 2/1.. |
| Leukemiae (2-y lag) | | | 32/31.5 | 5/4.6 | 2/2.6 | 2/2.3 |

[a] Deaths occurring 1982-85 were analyzed using proportional mortality analysis and the results were combined with results of analyses presented in Table 3.

[b] ICD, International Classification of Diseases, Eighth Revision.

[c] The trend test statistics may be compared with a standard normal distribution to assess statistical significance. However, statistical significance may be exaggerated for diseases with a small number of deaths.

[d] Expected deaths for the years 1955-81 were calculated from the experience of all workers in the study population allowing for age, calendar year, sex and length of employment. Expected deaths for the years 1982-85 were calculated from all Washington deaths during this period, allowing for age, calendar year and sex.

[e] Excluding chronic lymphatic leukemia.

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Table 5. Results of analyses of external dose in monitored workers employed at the Hanford Site for at least 6 mo. Except where noted, this is based on a 10-y lag.

| Cause of death | Trend test statistic[a] Exposure lagged for: 10 y | 2 y | Observed and expected deaths by exposure category (mSv) 0– Obs/Exp[b] | 10– Obs/Exp | 50– Obs/Exp | 100– Obs/Exp | 200+ Obs/Exp |
|---|---|---|---|---|---|---|---|
| All causes | −0.90 | −1.91 | 4,361/4,267.7 | 1,333/1,378.9 | 213/227.9 | 158/169.8 | 155/155.7 |
| Cause unavailable | −1.28 | −1.32 | 61/59.0 | 22/19.5 | 0/3.2 | 2/2.1 | 1/2.1 |
| All cancer | −0.29 | −0.50 | 975/982.9 | 346/334.6 | 58/60.1 | 47/44.9 | 40/43.5 |
| Male | −0.29 | −0.54 | 816/818.8 | 316/310.7 | 55/56.4 | 43/41.5 | 40/42.7 |
| Female | 0.02 | 0.28 | 159/164.2 | 30/23.9 | 3/3.7 | 4/3.4 | 0/0.8 |
| Smoking-linked cancers[c] | 0.10 | −0.01 | 380/389.0 | 154/148.3 | 30/26.9 | 22/20.7 | 19/20.1 |
| Residual | −0.49 | −0.67 | 595/593.9 | 192/186.3 | 28/33.2 | 25/24.2 | 21/23.4 |
| Buccal | −1.45 | −1.58 | 24/22.3 | 8/7.3 | 0/1.3 | 1/1.0 | 0/1.1 |
| Esophagus | 0.14 | −0.38 | 20/18.6 | 5/5.9 | 1/1.1 | 0/0.7 | 1/0.7 |
| Stomach | 0.18 | 0.37 | 43/44.1 | 14/12.7 | 2/2.5 | 3/1.9 | 1/1.9 |
| Colon | −0.35 | −0.63 | 103/106.6 | 36/29.1 | 2/4.7 | 4/3.1 | 2/3.4 |
| Rectum | 0.40 | 0.14 | 25/22.9 | 6/7.7 | 1/1.4 | 1/0.9 | 1/1.0 |
| Liver | 1.37 | 1.93* | 13/14.5 | 3/3.9 | 0/0.6 | 0/0.6 | 2/0.4 |
| Gallbladder | −1.27 | −1.30 | 13/10.8 | 1/2.5 | 0/0.3 | 0/0.3 | 0/0.06 |
| Pancreas | 1.59* | 2.36* | 51/52.5 | 19/18.3 | 2/3.1 | 3/2.2 | 3/1.9 |
| Larynx | −0.62 | −0.94 | 9/8.5 | 3/3.0 | 1/0.6 | 0/0.5 | 0/0.3 |
| Lung | 0.16 | 0.01 | 236/265.0 | 107/104.2 | 24/19.2 | 17/15.0 | 14/14.6 |
| Bone | −0.68 | −0.70 | 3/2.3 | 0/0.4 | 0/0.1 | 0/0.1 | 0/0.03 |
| Female breast | −0.05 | 0.58 | 45/46.5 | 7/5.3 | 1/0.9 | 1/1.0 | 0/0.3 |
| Cervix and uterus | 0.96 | 0.29 | 3/5.0 | 3/0.8 | 0/0.2 | 0/0.06 | 0/0.00 |
| Ovary | 0.79 | 0.61 | 12/12.1 | 1/1.5 | 0/0.2 | 1/0.2 | 0/0.04 |
| Prostate | −1.15 | −1.51 | 59/61.7 | 32/27.1 | 5/4.4 | 2/3.4 | 2/3.4 |
| Bladder | −0.34 | −0.66 | 17/18.9 | 11/8.7 | 1/1.3 | 1/1.0 | 1/1.1 |
| Kidney | 0.72 | 0.60 | 27/23.3 | 2/7.9 | 2/1.4 | 3/1.2 | 1/1.2 |
| Brain and central nervous system | −0.90 | −0.66 | 28/30.1 | 16/11.3 | 1/2.4 | 2/1.7 | 0/1.5 |
| Thyroid | −0.42 | −0.46 | 2/1.7 | 1/1.0 | 0/0.2 | 0/0.1 | 0/0.0 |
| Non-Hodgkin's lymphoma | −0.85 | −1.14 | 37/39.1 | 17/13.7 | 3/2.6 | 1/1.8 | 1/1.8 |
| Hodgkin's disease | 1.80* | 2.38* | 14/14.8 | 2/2.8 | 0/0.5 | 1/0.3 | 2/0.5 |
| Multiple myeloma | 1.64* | 2.37* | 17/17.0 | 2/4.9 | 2/0.8 | 1/0.6 | 2/0.6 |
| Chronic lymphatic leukemia (CLL) | −0.34 | −0.45 | 6/5.6 | 1/2.1 | 2/0.6 | 0/0.3 | 0/0.4 |
| Leukemia excluding CLL | −0.81 | | 27/26.2 | 14/11.7 | 1/2.4 | 1/1.7 | 1/2.0 |
| Leukemia excluding CLL (2-y lag) | | −0.85 | 25/23.7 | 14/13.1 | 1/2.7 | 3/2.0 | 1/2.6 |
| All noncancer | −0.70 | −1.78 | 3,305/3,225.7 | 965/1,024.8 | 155/164.5 | 109/122.8 | 114/110.1 |
| Circulatory | −0.48 | −1.28 | 2,193/2,120.6 | 642/699.7 | 102/113.0 | 76/83.6 | 81/77.0 |
| Respiratory excluding pneumonia | 0.10 | −0.16 | 194/208.2 | 96/84.7 | 19/12.8 | 8/10.0 | 9/10.3 |
| Cirrhosis | 0.46 | 0.39 | 86/88.0 | 22/22.7 | 6/3.5 | 2/2.8 | 3/2.0 |
| External | −1.81 | −1.96 | 374/366.1 | 76/75.9 | 11/13.5 | 11/9.9 | 2/6.6 |
| Person-years | | | 499,847 | 96,731 | 17,545 | 11,430 | 7,958 |

[a] The trend test statistic was calculated from individual doses, not the five exposure categories. It may be compared with a standard normal distribution to assess statistical significance. However, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote c.

[b] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, gender, number of years monitored, and general socioeconomic category.

[c] The smoking-linked cancers were respiratory cancer, buccal cancer, and cancer of the esophagus, pancreas, and bladder.

[d] Based on computer simulations, the one-tailed $p$ values associated with the trend test with a 10-y lag were estimated to be 0.065 for cancer of the pancreas, 0.038 for Hodgkin's disease, and 0.10 for multiple myeloma. For the 2-y lag, these $p$ values were estimated to be 0.056 for cancer of the liver, 0.026 for cancer of the pancreas, 0.029 for Hodgkin's disease, and 0.030 for multiple myeloma.

A12

Table 9. Results of analyses of external dose for selected cancer categories in monitored workers employed at the Hanford Site for at least 6 mo including cancers noted on death certificate, but not considered to be the underlying cause of death. Except where noted, this is based on a 10-y lag.

| Cause of death | Trend test statistic[a] Exposure lagged for: 10 y | 2 y | Observed and expected deaths by exposure category (mSv) 0– Obs/Exp[b] | 10– Obs/Exp | 50– Obs/Exp | 100– Obs/Exp | 200+ Obs/Exp |
|---|---|---|---|---|---|---|---|
| All cancer | −0.17 | −0.29 | 1,079/1,082.1 | 381/377.7 | 69/66.9 | 50/49.6 | 46/48.7 |
| Pancreas | 1.45[c] | 2.20[c] | 52/54.0 | 20/19.3 | 3/3.3 | 3/2.4 | 3/2.1 |
| Multiple myeloma | 2.50[c] | 2.95[c] | 17/17.3 | 2/6.1 | 2/1.1 | 2/0.7 | 3/0.8 |
| Leukemia[d] | −0.98 | | 33/31.3 | 15/13.2 | 1/2.5 | 1/1.8 | 1/2.2 |
| Leukemia[d] (2-y lag) | | −1.01 | 31/28.6 | 15/14.6 | 1/2.8 | 3/2.1 | 1/2.8 |

[a] The trend test statistic was calculated from individual doses, not the five exposure categories. It may be compared with a standard normal distribution to assess statistical significance; however, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote c.

[b] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, gender, number of years monitored, and general socioeconomic category.

[c] Based on computer simulations, the one-tailed $p$ values associated with the trend test with a 10-y lag were estimated to be 0.080 for cancer of the pancreas and 0.023 for multiple myeloma. For the 2-y lag, these $p$ values were estimated to be 0.032 for cancer of the pancreas and 0.007 for multiple myeloma.

[d] Excluding chronic lymphatic leukemia.

Table 10. Results of analyses of external dose for selected cancer categories in monitored workers employed at the Hanford Site for at least 6 mo, including cancers occurring in the state of Washington from 1987–1989. Except where noted, this is based on a 10-y lag.

A13

| Cause of death | Trend test statistic[a] Exposure lagged for: 10 y | 2 y | Observed and expected deaths by exposure category (mSv) 0– Obs/Exp[b] | 10– Obs/Exp | 50– Obs/Exp | 100– Obs/Exp | 200+ Obs/Exp |
|---|---|---|---|---|---|---|---|
| All cancer | –0.20 | –0.38 | 1,023/1,031.5 | 392/381.4 | 70/71.1 | 56/53.2 | 52/55 |
| Pancreas | 1.91[c] | 2.57[c] | 54/55.4 | 19/19.4 | 2/3.6 | 4/2.4 | 4/2.2 |
| Multiple myeloma | 1.9[c] | 2.90[c] | 18/18.2 | 2/6.3 | 3/1.3 | 2/1.0 | 3/1.2 |
| Leukemia[d] | –0.78 | | 29/27.8 | 15/13.2 | 1/2.7 | 2/1.9 | 1/2.3 |
| Leukemia[d] (2-y lag) | | –0.84 | 27/25.2 | 15/14.7 | 1/3.0 | 4/2.2 | 1/2.9 |

[a] The trend test statistic was calculated from individual doses, not the five exposure categories. It may be compared with a standard normal distribution to assess statistical significance; however, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote c.
[b] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, gender, number of years monitored, and general socioeconomic category.
[c] Based on computer simulations, the one-tailed $p$ values associated with the trend test with a 10-y lag were estimated to be 0.033 for cancer of the pancreas and 0.040 for multiple myeloma. For the 2-y lag, these $p$ values were estimated to be 0.019 for cancer of the pancreas and 0.011 for multiple myeloma.
[d] Excluding chronic lymphatic leukemia.

Table 11. Results of analyses of external dose for selected cancer categories in monitored workers employed at the Hanford Site for at least 6 mo including cancers occurring in the state of Washington from 1987–1989, and including cancers noted on the death certificate, but not considered to be the underlying cause of death. Except where noted, this is based on a 10-y lag.

A14

| Cause of death | Trend test statistic[a] Exposure lagged for: 10 y | 2 y | Observed and expected deaths by exposure category (mSv) 0– Obs/Exp[b] | 10– Obs/Exp | 50– Obs/Exp | 100– Obs/Exp | 200+ Obs/Exp |
|---|---|---|---|---|---|---|---|
| All cancer | 0.05 | –0.04 | 1,131/1,135.3 | 434/431.7 | 82/79.3 | 61/59.1 | 60/62.5 |
| Pancreas | 1.61[c] | 2.28[c] | 56/57.1 | 20/20.9 | 3/3.9 | 4/2.6 | 4/2.5 |
| Multiple myeloma | 2.67[c] | 3.45[c] | 18/18.6 | 2/7.5 | 3/1.5 | 3/1.1 | 4/1.4 |
| Leukemia[d] | –0.94 | | 33/32.8 | 16/14.8 | 1/2.9 | 2/2.0 | 1/2.3 |
| Leukemia[d] (2-y lag) | | –0.99 | 33/30.2 | 16/16.2 | 1/3.2 | 4/2.3 | 1/3.1 |

[a] The trend statistic was calculated from individual doses, not the five exposure categories. It may be compared with a standard normal distribution to assess statistical significance; however, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote c.
[b] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, gender, number of years monitored, and general socioeconomic category.
[c] Based on computer simulations, the one-tailed $p$ values associated with the trend test with a 10-y lag were estimated to be 0.062 for cancer of the pancreas and 0.011 for multiple myeloma. For the 2-y lag, these $p$ values were estimated to be 0.028 for cancer of the pancreas and 0.003 for multiple myeloma.
[d] Excluding chronic lymphatic leukemia.



Table 6. Relative risks[a] (with 90% confidence limits) by external dose category for monitored Hanford Site workers employed at least 6 mo (based on a 2-y lag for leukemia and a 10-y lag for other categories).

A15

| Dose category (mSv) | All cancer | All noncancer | Leukemia[b] | Multiple myeloma |
|---|---|---|---|---|
| 0– | 1.00 | 1.00 | 1.0 | 1.0 |
| 10– | 1.04 (0.9, 1.2) | 0.89 (0.8, 1.0) | 0.8 (0.4, 1.6) | 0.4 (0.1, 1.3) |
| 50– | 1.01 (0.8, 1.3) | 0.85 (0.7, 1.0) | 0.3 (0.03, 1.6) | 4.2 (0.7, 19) |
| 100– | 1.17 (0.9, 1.5) | 0.83 (0.7, 1.0) | 1.5 (0.4, 4.8) | 5.9 (0.5, 41) |
| 200– | 0.93 (0.7, 1.3) | 0.96 (0.8, 1.2) | 0.3 (0.02, 1.3) | 21 (2.1, 270) |

[a] The relative risks are the ratio of the risk for the indicated category relative to that of the lowest dose category (0–9 mSv).
[b] Excluding chronic lymphatic leukemia.

TABLE 3. Rate Ratios for Leukemia by Dose Category and Study Population

| Study and Reference Number | Less Than 10 mSv | 10–50 mSv | More Than 50 mSv | Total |
|---|---|---|---|---|
| Atomic Energy Authority[2,13]† | | | | |
| Observed | 10 | 5 | 3 | 18 |
| PYRs | 219,102 | 71,516 | 37,817 | 328,435 |
| Rate/100,000 | 4.6 | 7.0 | 7.9 | 5.5 |
| RR | 1.0 | 1.5 | 1.7 | |
| 90% CL | | 0.6,3.8 | 0.6,5.1 | |
| Atomic Weapons Establishment[13] | | | | |
| Observed | 4 | 0 | 0 | 4 |
| PYRs | 136,366 | 7,694 | 1,655 | 145,715 |
| Rate/100,000 | 2.9 | 0.0 | 0.0 | 2.8 |
| RR | 1.0 | 0.0 | 0.0 | |
| 90% CL | | 0,14 | 0,64 | |
| Hanford[11]‡§ | | | | |
| Observed | 24 | 3 | 2 | 29 |
| PYRs | 361,017 | 28,531 | 23,846 | 413,394 |
| Rate/100,000 | 6.6 | 10.5 | 8.4 | 7.0 |
| RR | 1.0 | 1.6 | 1.3 | |
| 90% CL | | 0.6,4.3 | 0.4,4.2 | |
| Oak Ridge National Laboratory[10]‡‖ | | | | |
| Observed | 5 | 6 | 0 | 11 |
| PYRs[3] | 112,080 | 31,380 | 12,100 | 155,560 |
| Rate/100,000 | 4.5 | 19.1 | 0.0 | 7.1 |
| RR | 1.0 | 4.3 | 0.0 | |
| 90% CL | | 1.7,10.7 | 0,5,4 | |
| Portsmouth[4]† | | | | |
| Observed | 3 | 3 | 1 | 7 |
| PYRs | 65,326 | 21,769 | 11,128 | 98,223 |
| Rate/100,000 | 4.6 | 13.8 | 9.0 | 7.1 |
| RR | 1.0 | 3.0 | 2.0 | |
| 90% CL | | 0.8,10.8 | 0.3,12.6 | |
| Rocky Flats[12]‡ | | | | |
| Observed | 3 | 0 | 1 | 4 |
| PYRs | 64,609 | 8,645 | 4,528 | 77,782 |
| Rate/100,000 | 4.6 | 0.0 | 22.1 | 5.1 |
| RR | 1.0 | 0.0 | 4.8 | |
| 90% CL | | 0.8,6 | 0.9,26.6 | |
| Sellafield[11]¶ | | | | |
| Observed | 6 | 3 | 1 | 10 |
| PYRs | 154,535 | 27,567 | 28,260 | 210,362 |
| Rate/100,000 | 3.9 | 10.9 | 3.5 | 4.8 |
| RR | 1.0 | 2.8 | 0.9 | |
| 90% CL | | 0.9,8.5 | 0.2,5.4 | |
| Total | | | | |
| Observed | 55 | 20 | 8 | 83 |
| PYRs | 1,113,035 | 197,102 | 119,334 | 1,429,471 |
| Rate/100,000 | 4.9 | 10.1 | 6.7 | 5.8 |
| RR | 1.0 | 2.1 | 1.4 | |
| 90% CL | | 1.4,3.1 | 0.7,2.5 | |
| $RR_{adj}$ | | 2.1 | 1.4 | |
| 90% CI | | 1.4,3.3 | 0.8,2.6 | |
| $RR_{[illegible]}$ | | 1.8 | 1.2 | |

†No lag period.
‡Exposure lagged 10 years.
§Dose categories are: 0–20 mSv, 20–50 mSv, 50+ mSv.
‖Person-years were estimated by multiplying the number of workers in each dose category by the average length of follow-up.
¶Exposure lagged 15 years.

Table 4. Standardized mortality ratios[a] (SMRs) and observed deaths (OBS) for specific cancer types in monitored and unmonitored white male and female Hanford Site workers from 1945–1986.

A17

| Type of cancer (eighth revision ICD[b] code) | Males | | | | Females | | | | All workers | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Monitored | | Unmonitored | | Monitored | | Unmonitored | | | |
| | SMR | OBS | SMR | OBS | SMR | OBS | SMR | OBS | SMR | OBS |
| Buccal cavity and pharynx (140–149) | 0.81 | 42 | 0.66 | 6 | 0.00 | 0 | 1.08 | 3 | 0.76 | 51 |
| Digestive organs and peritoneum (150–159) | 0.87 | 414 | 0.80 | 72 | 0.74 | 46 | 0.75 | 37 | 0.84 | 569 |
| Esophagus (150) | 0.84 | 35 | 0.56 | 4 | 0.00 | 0 | 1.75 | 3 | 0.80 | 42 |
| Stomach (151) | 0.84 | 70 | 0.46 | 8 | 0.82 | 6 | 0.66 | 4 | 0.77 | 88 |
| Colon (153) | 0.88 | 141 | 0.71 | 21 | 0.70 | 19 | 0.84 | 18 | 0.84 | 199 |
| Rectum (154) | 0.72 | 35 | 0.41 | 4 | 0.52 | 3 | 0.85 | 4 | 0.67 | 46 |
| Liver and gall bladder (155–156) | 1.01 | 37 | 0.86 | 6 | 0.85 | 5 | 0.64 | 3 | 0.94 | 51 |
| Pancreas (157) | 0.91 | 84 | 1.57[c] | 26 | 0.85 | 10 | 0.56 | 5 | 0.96 | 125 |
| Respiratory system (160–163) | 0.80 | 491 | 0.93 | 93 | 1.02 | 41 | 0.97 | 28 | 0.83 | 653 |
| Larynx (161) | 0.60 | 15 | 1.36 | 6 | 1.19 | 1 | 1.61 | 1 | 0.74 | 23 |
| Lung (162) | 0.80 | 470 | 0.92 | 87 | 1.01 | 39 | 0.97 | 27 | 0.84 | 623 |
| Bone (170) | 0.44 | 3 | 0.74 | 1 | 1.13 | 1 | 0.00 | 0 | 0.51 | 5 |
| Skin (172–173) | 0.76 | 25 | 0.97 | 5 | 0.94 | 4 | 0.66 | 2 | 0.80 | 36 |
| Female breast (174) | | | | | 0.95 | 60 | 0.89 | 42 | 0.92 | 102 |
| All uterus (180–182) | | | | | 0.33 | 7 | 0.99 | 17 | 0.63 | 24 |
| Other female genital organs (183–184) | | | | | 0.78 | 16 | 1.02 | 16 | 0.89 | 32 |
| Prostate (185) | 0.95 | 116 | 1.00 | 27 | | | | | 0.96 | 143 |
| Testes and other male genital organs (186–187) | 0.71 | 6 | 1.44 | 2 | | | | | 0.82 | 8 |
| Bladder (188) | 0.68 | 34 | 0.67 | 7 | 0.36 | 1 | 1.34 | 3 | 0.69 | 45 |
| Kidney (189) | 0.94 | 41 | 1.22 | 9 | 0.72 | 3 | 0.63 | 2 | 0.95 | 55 |
| Eye (190) | 1.43 | 2 | 0.00 | 0 | 0.00 | 0 | 0.00 | 0 | 0.97 | 2 |
| Brain and other central nervous system (191–192) | 0.96 | 48 | 0.52 | 4 | 0.78 | 6 | 0.53 | 3 | 0.86 | 61 |
| Thyroid (193) | 0.61 | 2 | 1.74 | 1 | 1.19 | 1 | 0.00 | 0 | 0.75 | 4 |
| All other solid tumors (171, 194–199) | 1.02 | 126 | 1.47[c] | 31 | 0.81 | 16 | 0.80 | 12 | 1.03 | 185 |
| All lymphatic and haematopoietic cancer (200–209) | 0.96 | 158 | 0.76 | 22 | 0.92 | 22 | 1.01 | 18 | 0.93 | 220 |
| Lymphosarcoma and reticulosarcoma (200) | 1.04 | 32 | 0.37 | 2 | 0.92 | 4 | 1.48 | 5 | 0.98 | 43 |
| Hodgkin's disease (201) | 0.93 | 16 | 1.03 | 3 | 1.24 | 3 | 0.56 | 1 | 0.94 | 23 |
| Multiple myeloma (203) | 0.93 | 21 | 1.29 | 5 | 0.83 | 4 | 0.55 | 2 | 0.91 | 32 |
| Leukemia and aleukemia (204–207) | 0.84 | 56 | 0.74 | 9 | 0.76 | 7 | 1.16 | 8 | 0.84 | 80 |
| Other lymphatic tissue (202–203, 208–209) | 0.99 | 50 | 0.98 | 8 | 1.02 | 8 | 0.70 | 4 | 0.97 | 70 |

[a] The SMR is the ratio of observed and expected deaths, where expected deaths were calculated from age-specific and calendar year-specific mortality rates for U.S. caucasian males or females. The SMRs were corrected for those deaths with no certificates on the assumption that the distribution of causes was similar for those with and without certificates.

[b] ICD = International Classification of Diseases, Eighth Revision.

[c] These SMRs are based on the years 1950–1986 since U.S. mortality rates for these cancer types were not available prior to 1950.

[d] Significantly elevated at the 0.05 level based on a one-tailed test.

Table 5. Results of analyses of external dose in monitored workers employed at the Hanford Site for at least 6 mo. Except where noted, this is based on a 10-y lag.

A18

| Cause of death | Trend test statistic[a] Exposure lagged for: 10 y | 2 y | Observed and expected deaths by exposure category (mSv) 0– Obs/Exp[b] | 10– Obs/Exp | 50– Obs/Exp | 100– Obs/Exp | 200+ Obs/Exp |
|---|---|---|---|---|---|---|---|
| All causes | −0.90 | −1.91 | 4,341/4,267.7 | 1,333/1,378.9 | 213/227.9 | 158/169.8 | 155/155.7 |
| Cause unavailable | −1.28 | −1.32 | 61/59.0 | 22/19.5 | 0/3.2 | 2/2.1 | 1/2.1 |
| All cancer | −0.29 | −0.50 | 975/982.9 | 346/334.6 | 58/60.1 | 47/44.9 | 40/43.5 |
| Male | −0.29 | −0.54 | 816/818.8 | 316/310.7 | 55/56.4 | 43/41.5 | 40/42.7 |
| Female | 0.02 | 0.28 | 159/164.2 | 30/23.9 | 3/3.7 | 4/3.4 | 0/0.8 |
| Smoking-linked cancers[c] | 0.10 | −0.01 | 380/389.0 | 154/148.3 | 30/26.9 | 22/20.7 | 19/20.1 |
| Residual | −0.49 | −0.67 | 595/593.9 | 192/186.3 | 28/33.2 | 25/24.2 | 21/23.4 |
| Buccal | −1.45 | −1.58 | 24/22.3 | 8/7.3 | 0/1.3 | 1/1.0 | 0/1.1 |
| Esophagus | 0.14 | −0.38 | 20/18.6 | 5/5.9 | 1/1.1 | 0/0.7 | 1/0.7 |
| Stomach | 0.18 | 0.37 | 43/44.1 | 14/12.7 | 2/2.5 | 3/1.9 | 1/1.9 |
| Colon | −0.35 | −0.63 | 103/106.6 | 36/29.1 | 2/4.7 | 4/3.1 | 2/3.4 |
| Rectum | 0.40 | 0.14 | 25/22.9 | 6/7.7 | 1/1.4 | 1/0.9 | 1/1.0 |
| Liver | 1.37 | 1.93[d] | 15/14.5 | 3/3.9 | 0/0.6 | 0/0.6 | 2/0.4 |
| Gallbladder | −1.27 | −1.30 | 13/10.8 | 1/2.5 | 0/0.3 | 0/0.3 | 0/0.06 |
| Pancreas | 1.59[d] | 2.36[d] | 51/52.5 | 19/18.3 | 2/3.1 | 3/2.2 | 3/1.9 |
| Larynx | −0.62 | −0.94 | 9/8.5 | 3/3.0 | 1/0.6 | 0/0.5 | 0/0.3 |
| Lung | 0.16 | 0.01 | 256/265.0 | 107/104.2 | 24/19.2 | 17/15.0 | 14/14.6 |
| Bone | −0.68 | −0.70 | 3/2.3 | 0/0.4 | 0/0.1 | 0/0.1 | 0/0.03 |
| Female breast | −0.05 | 0.58 | 45/46.5 | 7/5.3 | 1/0.9 | 1/1.0 | 0/0.3 |
| Cervix and uterus | 0.96 | 0.29 | 3/5.0 | 3/0.8 | 0/0.2 | 0/0.06 | 0/0.00 |
| Ovary | 0.79 | 0.61 | 12/12.1 | 1/1.5 | 0/0.2 | 1/0.2 | 0/0.04 |
| Prostate | −1.15 | −1.51 | 59/61.7 | 32/27.1 | 5/4.4 | 2/3.4 | 2/3.4 |
| Bladder | −0.34 | −0.66 | 17/18.9 | 11/8.7 | 1/1.3 | 1/1.0 | 1/1.1 |
| Kidney | 0.72 | 0.60 | 27/23.3 | 2/7.9 | 2/1.4 | 3/1.2 | 1/1.2 |
| Brain and central nervous system | −0.90 | −0.66 | 28/30.1 | 16/11.3 | 1/2.4 | 2/1.7 | 0/1.5 |
| Thyroid | −0.42 | −0.46 | 2/1.7 | 1/1.0 | 0/0.2 | 0/0.1 | 0/0.0 |
| Non-Hodgkin's lymphoma | −0.85 | −1.14 | 37/39.1 | 17/13.7 | 3/2.6 | 1/1.8 | 1/1.8 |
| Hodgkin's disease | 1.80[d] | 2.38[d] | 14/14.8 | 2/2.8 | 0/0.5 | 1/0.3 | 2/0.5 |
| Multiple myeloma | 1.54[d] | 2.23[d] | 17/17.0 | 2/4.9 | 2/0.9 | 1/0.6 | 2/0.6 |
| Chronic lymphatic leukemia (CLL) | −0.34 | −0.45 | 6/5.6 | 1/2.1 | 2/0.6 | 0/0.3 | 0/0.4 |
| Leukemia excluding CLL | −0.81 | | 27/26.2 | 14/11.7 | 1/2.4 | 1/1.7 | 1/2.0 |
| Leukemia excluding CLL (2-y lag) | | −0.85 | 25/23.7 | 14/13.1 | 1/2.7 | 3/2.0 | 1/2.6 |
| All noncancer | −0.70 | −1.78 | 3,305/3,225.7 | 965/1,024.8 | 155/164.6 | 109/122.8 | 114/110.1 |
| Circulatory | −0.48 | −1.28 | 2,193/2,120.6 | 642/699.7 | 102/113.0 | 76/83.6 | 81/77.0 |
| Respiratory excluding pneumonia | 0.10 | −0.16 | 194/208.2 | 96/84.7 | 19/12.8 | 8/10.0 | 9/10.3 |
| Cirrhosis | 0.46 | 0.39 | 86/88.0 | 22/22.7 | 6/3.5 | 2/2.8 | 3/2.0 |
| External | −1.81 | −1.96 | 374/366.1 | 74/75.9 | 11/13.5 | 11/9.9 | 2/6.6 |
| Person-years | | | 499,847 | 96,731 | 17,545 | 11,430 | 7,958 |

[a] The trend test statistic was calculated from individual doses, not the five exposure categories. It may be compared with a standard normal distribution to assess statistical significance. However, statistical significance may be exaggerated for diseases with a small number of deaths. See footnote d.

[b] Expected deaths were calculated from the experience of all workers in the study population, allowing for age, calendar year, gender, number of years monitored, and general socioeconomic category.

[c] The smoking-linked cancers were respiratory cancer, buccal cancer, and cancer of the esophagus, pancreas, and bladder.

[d] Based on computer simulations, the one-tailed $p$ values associated with the trend test with a 10-y lag were estimated to be 0.065 for cancer of the pancreas, 0.038 for Hodgkin's disease, and 0.10 for multiple myeloma. For the 2-y lag, these $p$ values were estimated to be 0.056 for cancer of the liver, 0.026 for cancer of the pancreas, 0.029 for Hodgkin's disease, and 0.030 for multiple myeloma.

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