Nuclear law stands on thin ice

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Abstract: Revealing the questionable actions of many radiobiologists exposes the Achilles heel of nuclear law. Documentation of systematic deception is reason enough to change nuclear law. Much evidence comes from mis-statements by seven committees for the Biologic Effects of Ionizing Radiation (BEIR). These fraudulent interpretations led ignorant physicians, the media and government officials to accept the 'linear no threshold' (LNT) dogma. These misrepresentations are fully rebutted by rational interpretation of data. The best data comes from exposed nuclear workers. Eight independent epidemiological studies, involving almost 12 million person-years, consistently showed that increased exposure to ionising radiation was associated with decreased cancer mortality rates. These questionable actions kept ionising radiation from its role in abundant health.

Keywords: atomic bomb victims; BEIR reports; cancer; lifespan; misinformation; nuclear workers; radiation effects.

Biographical notes: Sir Samurai T. D. Luckey, PhD, is Emeritus Professor of the University of Missouri, MO, USA and Honorary Professor of the Free University of Herborn, Germany. He has a BS (1941) in chemistry from Colorado State University and both an MS (1944) and PhD (1946) in biochemistry/nutrition from the University of Wisconsin. His thesis included the first use of dietary antibiotics for animals. His group at Notre Dame University achieved the first reproduction in germfree animals. From 1954 to 1968, he was Chairman of the Department of Biochemistry, University of Missouri, Columbia, MO. He was guest lecturer for Group 6 Astronauts and was nutrition consultant to NASA for Apollos 11–17. In 1984, he was knighted, *Ritter von Greifenstein*, for his world leadership in intestinal microecology. In 2003, he was made Honorary Samurai for giving Japan the concept of radiation hormesis for health.

1 Introduction

> "The exact contrary of what is generally believed is often the truth." Jean de le Bruyere (1645–1696)

Hormesis is the biphasic effect of any agent upon physiological processes. Small doses are biopositive; large doses are bionegative. The inflection point is the threshold.

This presentation refers to whole body exposures. Data from cells in culture, tissues and micro-organisms are less reliable for interpretation about human populations. In order to present a wide range of doses, exposures are generally expressed on a logarithmic basis. This accounts for the straight line relationship between dose and physiological response. Most authors use arithmetic dose increments which produce a variety of curves. The straight line allows more certain predictability than any curve and provides the power to increase statistical significance.

Essential agents which exhibit hormesis include hormones, vitamins, essential minerals, oxygen, light, gravity and ionising radiation. The term *radiation hormesis* is used to signify that high and low doses of ionising radiation evoke opposite effects. There are over 3000 scientific papers (with some overlap) which show that low dose irradiation is biopositive as reviewed by Luckey (1980, 1991) and Muckerheide (2002). Radiation activated immune competence is largely responsible for the benefits of low dose radiation in cancer prevention, infectious diseases and average lifespan (see Liu, 2002 and Chapter 5 in Luckey, 1991). In contrast, no significant papers in English show harm from low dose irradiation in normal mammals.

Radiophobia began in the early1940s when the media extrapolated mutations in heavily irradiated fruit flies into genetic monsters in people exposed to a tiny amount of ionising radiation. Brucer (1990, p.275) noted: "The two-headed baby [illustrated in the magazine, *Time*, February 1983] is still the hallmark of radiation genetics" and "Few (health physicists and radiation geneticists) made any attempt to stem merging radiation hysteria." Eisenbud and Gesell (1997, p.18) stated: "Until the early 1960s the genetic consequences were thought to be the most important delayed effect of radiation exposure. A major publication prepared for the AEC in 1958 on the biological effects of radiation placed great emphasis on the genetic effects and included hardly any information on cancer." However, when over 50,000 children of Japanese bomb victims revealed no genetic monsters (Schull et al., 1981), cancer became the focus of radiation hysteria. "Cancer induction is the only source of somatic risk that needs to be taken into account in setting radiation protection standards for the general population." (BEIR I, 1972, p.91). "Cancers arising in a variety of organs and tissues are the principle late somatic effects of radiation exposure." (BEIR III, 1980, p.2).

With no regard for scientific evidence to the contrary, many well established radiobiologists, physicians and the media accepted the proposition that *all* radiation is harmful. This dogma was then accepted by national and international organisations. One example comes from the opening paper of the symposium on Biological Effects of Low-Level Radiation, sponsored by IAEA and WHO. Dr Pochin commented on the risks of radiation at low dose rates: "They may, however, be reliably inferred from the observed rates of exposure to rather larger doses delivered at higher dose rates. . ." (Pochin, 1983, p.3). Dr Pochin gave no hint that low dose irradiation might be beneficial. As will be documented, the linear no threshold (LNT) paradigm is based upon a one-tailed statistic which does not allow any biopositive effect to be observed. The use of two-tailed statistics allows the biopositive effects of low doses of ionising radiation to become evident. Two-tailed statistics were generally ignored by those who accepted the LNT dogma. Reliance upon this misinformation focused on risk and created fear.

Evidence of the questionable writings of radiobiologists is documented by comparing the observations, summaries and conclusions drawn by authors and officials with information from published scientific data. Rarely are the two compatible. Authors'

written evaluations were promulgated by quasi official groups (such as the biological effects of ionising radiation (BEIR) committees), the media, the medical professions and the US government, with no rigorous examination of the abundant scientific data. This misrepresentation was also accepted by national and international agencies and other governments. After a half century of misdirection, the concept that all radiation is harmful and the LNT dogma became the law of the land. Numerous specific examples are provided in order to reveal the bias of unscrupulous proceedings and to release present restrictions. Although the focus is on the BEIR reports, a comparable indictment could be made using publications from other established national and international agencies. As shown by the extensive study of the French Academy of Sciences and the National Academy of Medicine (Aurengo et al., 2005), the evidence and arguments presented here represent only the tip of an iceberg of scientific loss of integrity on a world wide basis.

Scientific evidence is presented in order to contradict the direct quotations giving misinformation and misdirection by many radiobiologists. Much of the information comes from Japanese atomic bomb victims, exposed nuclear workers and medical applications of low dose irradiation. The abundance of data makes cancer an important factor in understanding the role of the BEIR committees in mismanaging the data. This deception produced health conditions which appear to be responsible for hundreds of thousands of preventable cancer deaths each year. It is estimated that 42% of people in the United States will have cancer (BEIR VII Committee, 2005, p.15). The abundance of information makes cancer the focus for these discussions (Luckey, 1997a).

2 Seven BEIR reports

The framework to understand how our laws contradict valid evidence is based upon seven committees appointed to advise the government on the biological effects of ionising radiation (BEIR). Without consideration of the data within scientific reports, the BEIR committees accepted the recommendations of the preceding biological effect of atomic radiation (BEAR) committees and written comments and conclusions of radiobiologists who used only a one-tailed statistic. This provided a false basis for recommendations and for laws about harm from low dose radiation.

Some BEIR committees state they do not advise regulators: "the Committee has no responsibility to recommend regulatory limits" (BEIR III, 1980, p.1). However, numerous statements show this is blatantly false. Examples include:

- "Cancer induction is the only source of somatic risk that needs to be taken into account in setting radiation protection standards for the general population." (BEIR I, 1972, p.91)

- "The first report of the Committee on the Biological Effects of Ionising Radiation (BEIR I) has profoundly influenced governmental regulations and the public attitude towards radiation. It is to be expected that the impact of the current report (BEIR III) will be equally significant." (BEIR III, 1980, p.254)

- "We hope that the information contained herein will serve . . . as a scientific basis for the development of suitable radiation protection standards." (BEIR III, 1980, p.ix)

More statements are provided in the Appendix.

2.1   BEIR I

In agreement with the previous BEAR committees, the BEIR I committee was committed to the paradigm that all radiation is harmful: ". . .every effort should be made to encourage the maintenance of radiation doses as far below this guide as possible." (BEIR I, 1972, p.8). Their summary states: "we can calculate that five rem (cSv) per generation would eventually lead to an increase of 5% in the ill-health of the population." (BEIR I, 1972, p.2). This is half the exposure recommended by the BEAR committee (BEIR I, 1972, p.1). Also consider the statement: "Such calculations based on these data from irradiated humans lead to the prediction that additional exposure of the US population of five rem (cSv) per 30 years could cause from roughly 3000 to 15,000 cancer deaths annually." (BEIR I, 1972, p.2). As shown below, abundant scientific data deny these statements.

The data from the BEIR I committee (BEIR I, 1972, p.102) showed that low dose irradiation reduced leukemia in Nagasaki atomic bomb victims (Figure 1). In 2527 persons (37,600 person-years) there was no leukaemia; this included groups exposed to an average of 31 and 69 cGy. The threshold, defined by the incidence of leukaemia in more heavily exposed people (not on the graph), was about 100 cGy. The BEIR I committee (1972, p.103) concluded: ". . . excess leukaemia cases in Nagasaki amount to about one per 10^6/year/rad." As shown by the zero leukaemia in many Japanese atomic bomb victims, this is misinformation.

Figure 1   Leukaemia in atomic bomb victims of Nagasaki. The P-Y unit of the ordinate is 100,000 person-years. Taken from BEIR I (p.102, 1972)
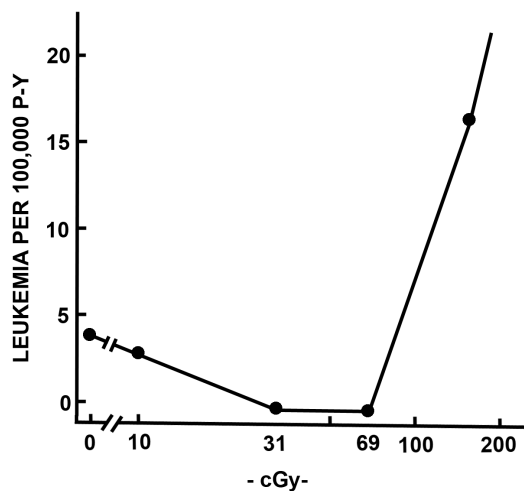
2.2   BEIR II

The BEIR II committee adhered strictly to the LNT dogma: "The position that there is no safe or threshold level for human exposure to radiation has been adopted by the NRC (National Research Council) and EPA (Environmental Protection Agency). Therefore, regulation cannot be promised on achieving and maintaining a level of radiation in the

ambient environment, above natural background, that is safe for human health." (BEIR II, 1977, p.90). Obviously, the BEIR II committee had no conceptual or actual information about the beneficial effects of low dose irradiation. This is confirmed by statements such as: "Where a population is irradiated coincidentally with achieving another purpose, as in the generation of nuclear power, only the negative effects of the radiation need be evaluated." (BEIR II, 1977, p.39) and (p.127): "The excess radiation with which we are concerned as a cost is that released outside the reactor and from which there are no benefits."

Although the title and much discussion include the "health benefit-cost analysis", the BEIR II committee adhered to the LNT paradigm: "The reduction of radiation risk is considered as a means to achieve improvement in the benefit-cost ratio." (BEIR II, 1977, p.9). The BEIR II committee never considered health benefits from low dose irradiation as a positive factor. The committee ignored over 50 reviews written before 1977 which presented research showing biopositive effects following low dose irradiation (Luckey, 1980, p.66).
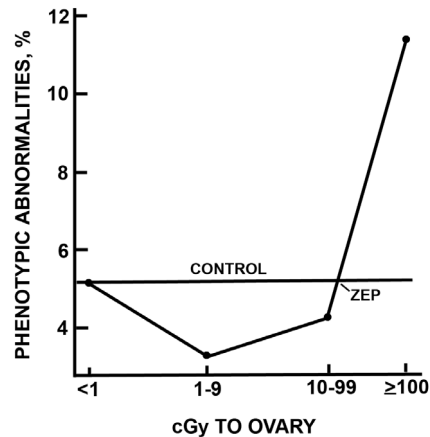
2.3 BEIR III

> "The BEIR committee endeavoured to ensure that no sources of relevant knowledge or expertise were overlooked in this study." (p.ix, BEIR III, 1980).

This is false. The committee ignored one thousand publications which showed that low dose irradiation was stimulatory and/or beneficial (Luckey, 1980).

The BEIR III committee also failed to consider the possibility that low dose irradiation could be beneficial. "The committee was in general agreement that, for most radiation-induced solid cancers, the dose-response relationship for low to intermediate doses of low-LET radiation is best described by a linear-quadratic function of dose with nonnegative curvature" (BEIR III, 1980, p.142). This is refuted by many examples. It also negates any possibility to observe the threshold between biopositive and bionegative effects, the hallmark of radiation hormesis. It is noteworthy that the BEIR III chapter on cancer has no discussion of increased immune competence, repair mechanisms or decreased cancer mortality rates from low doses of ionizing radiation.

When considering the risks of genetic effects from ionising radiation, the BEIR III committee stated: "Such an exposure of 1 rem received in each generation is estimated to result, at genetic equilibrium, in an increase of 60–1100 serious genetic disorders per million liveborn offspring" (BEIR III, 1980, p.5). The BEIR III committee undoubtedly knew the soon-to-be published results of Schull et al. (1981) from the Radiation Effects Research Foundation (RERF) which showed that newborn Japanese whose mothers received 1–9 cSv (and fathers received < 1cSv) had fewer mutations than the controls (Figure 2). The threshold (the zero equivalent point (ZEP) in the figure) was about 100 cGy. Excess mutations were noted only in children whose mothers received more than 100 Gy. When either one or both parents received up to 50 cSv, there was no increase in child mortality rates. Hall's mis-statement is: ". . .other than cancer, developmental effects on the unborn child are of the greatest concern" (Hall, 1987, p.446).

Figure 2   Mutations in newborn from Japanese atomic bomb victims. The ordinate shows the percentage of babies with phenotypic abnormalities. The fathers received less than 1 cSv. The control was persons in the city more than 3 km from the bomb epicenter (Schull et al., 1981)

The BEIR III committee stated: "Little is known about the effects of protracted low-dose exposure to the gonads" (BEIR III, 1980, p.478). They ignored the work of Kaplan (1949, 1959) who treated infertile women with 1 Gy of X radiation (approximate ovary dose) over a three week period. Of the 644 women treated, 351 produced 688 babies. There was no apparent genetic harm in the children or grandchildren. Also, ovarian disfunction is routinely treated with radon therapy in Russia (Bogoljubov, 1988). Finally, increased fertility, reduced spontaneous abortions and lower infant mortality were found in Chinese peasants who had three times more natural radiation than the controls (HBRRG, 1981).

The BEIR conclusion is: "As the embryo implants in the uterus and enters the period of major organogenesis, it becomes abruptly sensitive to the radiation induction of major malformations. Mortality induced by exposures during that period is no longer of the very early prenatal type but occurs mainly at birth or during infancy" (BEIR III, 1980, p.479). There is abundant evidence to refute such claims in the very extensive research on the benefits of low dose irradiation in reproduction (Brown et al., 1964; see also Chapter 4 in Luckey, 1991).

The BEIR III committee noted that in the absence of good human data: "Experimental data from laboratory organisms must be used" (BEIR III, 1980, p.94). However, their summary of animal research gives a false impression. Their statement is: "At the lower doses, impaired fertility and fecundity were manifested as high litter mortality, decreased litter size and diminished litter frequency" (BEIR III, 1980, p.496). Luckey (1991) cites 24 publications, published before 1980, in which embryo exposures to low dose irradiation improved reproductive performance. The BEIR III committee concluded: "Irradiation of the mouse and rat ovary results in early and progressive decline in the numbers of oocytes and ovarian follicles" (BEIR III, 1980, p.496). Harm from low dose irradiation during reproduction is contradicted by the extensive research (Brown et al., 1964 and Spalding and Brooks, 1972). They used many different dose rates for many generations of rats. In one experiment 12 generations of rats were exposed to 2 cGy/d of gamma radiation. This cohort showed no evidence of increased genetic or physiologic abnormalities. Lightly exposed animals showed superior fertility; one example is cited. A comparison of results

in 30 control rats (one group of control rats was discarded due to infections) and 47 irradiated rats is, respectively: dams with litters, 80% and 100%; average litter size, 6.1 and 10.0; average litter weight, 36.0 and 63.0 g; average number weaned, 6.0 and 7.3; total weight of weaned rats, 336 and 384 g.

In their summary of leukaemia the BEIR III committee stated: "Induction of leukaemia by radiation stands out because of the natural rarity of the disease, the relative ease of its induction by radiation and its short latent period (2–4 yr)" (BEIR III, 1980, p.2) and "Because of the low natural occurrence of leukaemia, the high radiosensitivity of stem cells and the short minimal latent period (2–3 yr), leukaemia was recognised early as a potential consequence of high-level radiation exposure in man" (BEIR III, 1980, p.353).

In his extensive examination of Nagasaki atomic bomb survivors, Land (1980) recorded the zero incidence of leukaemia in both a table and a graph. No mention was made of this phenomenon in his abstract, text or summary and it was not included in the numerous curves presented. He concluded: "There seems to be no way to evade extrapolation from high-dose estimates of the risk." As can be seen in Figure 3, none of the Land curves accommodates the zero incidence of leukaemia at 39 cGy for 25,643 person-years from Nagasaki atomic bomb victims. Land's next figure (not shown), which explored very low doses of ionising radiation, conveniently stopped before providing the data at 39 cGy; thus, no hormesis was shown by this graph. He insisted upon using high dose incidence to interpret low dose data using the LNT dogma. When the Nagasaki data were examined with computer (Figure 4), low dose irradiation was found to decrease leukaemia mortality. The data indicated radiation hormesis in leukaemia. The BEIR III committee also ignored these data: "The increased incidence of leukaemia in irradiated human populations is a well documented effect" (BEIR III, 1980, p.354). The BEIR VII committee also referred to the data from Japanese bomb victims: "The arguments for thresholds or beneficial health effects are not supported by these data" (BEIR VII, 2005, p.19).

Figure 3   Four dose-response curves for leukaemia in Nagasaki atomic bomb victims (Land, 1980). The ordinate indicates the number of leukaemia cases per 100,000 person-years (PY). All four age adjusted curves ignore the zero incidence of leukaemia at 39 cGy
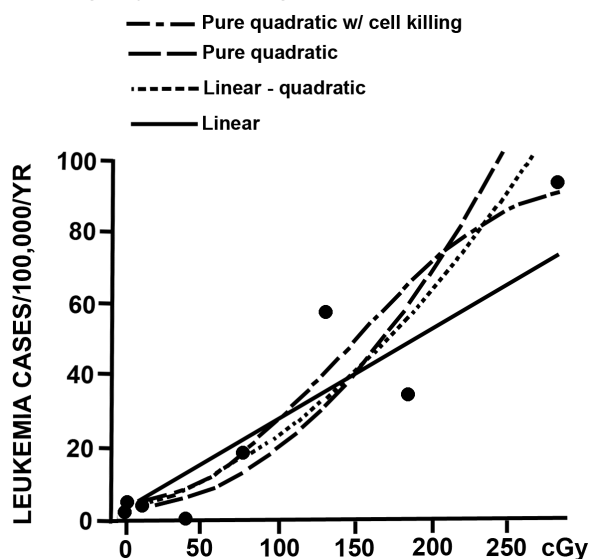
Figure 4   Detail of the data in Figure 3. The ordinate indicates the number of leukaemia cases per 100,000 person-years. One standard deviation is shown. This computer generated curve shows radiation hormesis for leukaemia in Nagasaki atomic bomb victims
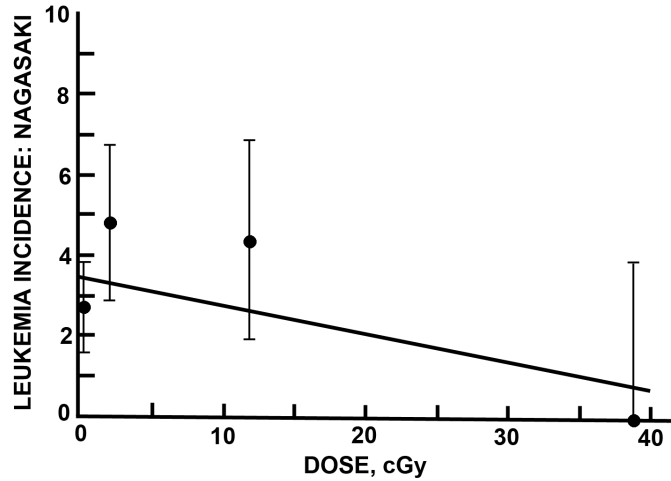
The BEIR III committee stated: "Experimental studies from laboratory organisms must be used" (BEIR III, 1980, p.96). They summarised the epic studies of Spalding et al. (1964) who exposed 4000 male mice; each was exposed to 200 cGy of gamma ray irradiation. Some were exposed for 45 generations. The committee stated: "There were no significant differences between the irradiated and control strains in growth or in mortality; the lifetime survival curves are almost identical in the two groups" (BEIR III, 1980, p.116). In great contrast to the BEIR statement, Spalding et al. (1964) found that, when compared with the 50% survival of unexposed mice, the survival rate was over 70% for mice exposed to 0.7, 2.1, 6.3, 18.9 and 56.7 cGy/d. Data from the group of 25 mice exposed to 18.9 cGy/d is illustrated (Figure 5). Note the threshold was about 17 Gy. The BEIR III committee ignored a large inventory of data which were typical of the results from Spalding et al. Prior to 1980, dozens of similar experiments confirmed the increased average lifespan in animals exposed to different types of low dose irradiation (Luckey, 1991).

Figure 5   Increased lifespan in mice (25 mice per group) exposed at six months to 19 cGy/d of gamma rays. The ordinate indicates the percent survival of exposed mice when 50% of the control mice had died (Spalding et al., 1964)
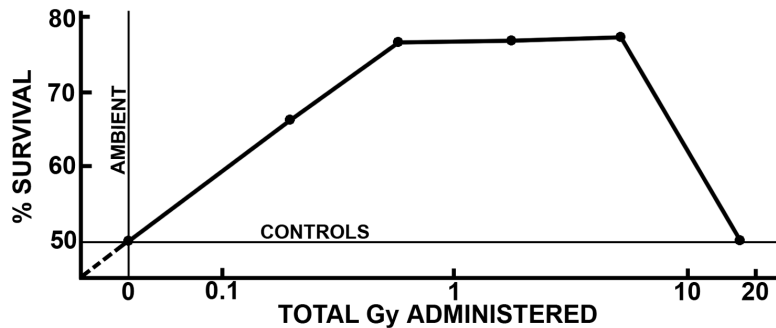
The BEIR III committee stated: "On the basis of animal experiments, the hypothesis has been advanced that radiation exposure induces premature aging, one consequence of which is dose dependent life shortening" (BEIR III, 1980, p.501). The data of Lorenz et al. (1955) is a striking refutation of this statement. The 236 unexposed mice had an average survival time which was shorter then that of 231 mice exposed to gamma rays, 1.1 mGy/d. In spite of this, the authors maintained: "All radiation produces deleterious effects . . ." They discarded the first experiment due to infection in the control colony. Luckey (1991) presents 36 scientific studies (published before 1980) in which the average lifespan of animals exposed to low dose irradiation was statistically longer than that of controls.

The BEIR III committee stated: "General growth retardation can result and may be temporary or permanent" (BEIR III, 1980, p.480). Land (1980, p.1197) expressed the concept for those who used a one-tailed statistic: "a sample of 100,000 may be needed for (to determine the effect of) a 100 rad exposure and about 10 million for 1 rad." Two of many experiments which were performed with 25 animals per group (Figure 6) gave statistically valid evidence for increased growth to disprove this hypothesis (Luckey, 1991). The solid line shows the results of Lorenz (1950). The dashed line is taken from data of medical students when they repeated the experiments of Lorenz. Many other examples are recorded in Chapter 3 of *Radiation Hormesis* (Luckey, 1991).

Figure 6 Growth of mice exposed to low doses of X rays. The experiments of Morris et al. (1963) were performed to replicate those of Lorenz (1950) from the previous decade. Those marked "a" had statistically significant ($p < 0.01$) increased growth rate when compared with the growth of controls
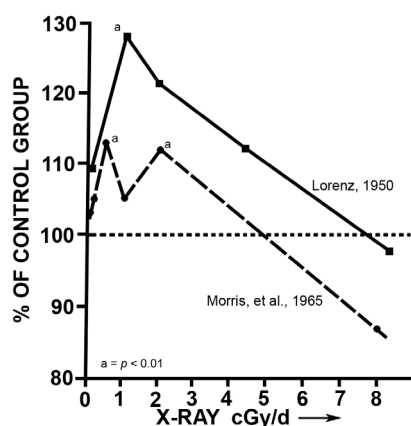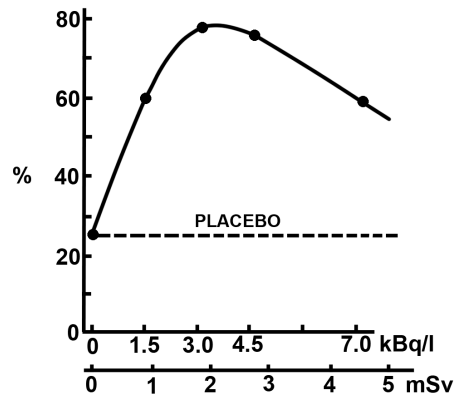

2.4 BEIR IV

The BEIR IV committee was also committed to the LNT paradigm: "The frequency of such effects (somatic and genetic) increases with low-level radiation as a linear, nonthreshold function of the dose" (BEIR IV, 1988, p.4) and: "Underground miners, exposed to radon daughters in a mine's air, have an increased risk of lung cancer that has been demonstrated in numerous populations" (BEIR IV, 1988, p.77). As is shown repeatedly, this is not true for low doses of radon. Health spas throughout the world have evidence of benefits from radon (Becker, 2003). Two large hospitals in Russia treated thousands of patients daily with radon (Bogoljubov, 1988). Their clinically documented

successes for a variety of diseases required multiple exposures (Figure 7). The optimum concentration for radon was about 2 mSv per treatment.

Figure 7   Radon therapy in Russia (Bogoljubov, 1988). The ordinate indicates the percent of clinically evaluated success of radon therapy in the treatment of Russian patients having a variety of syndromes. The placebo was nitrogen gas. Multiple treatments were administered weekly


When considering harm from radium, the BEIR IV committee considered all radiation to be harmful: ". . . as a working hypothesis, radiation is assumed to be carcinogenic even at the lowest dose levels, although there is no unequivocal evidence to support this hypothesis" (BEIR IV, 1988, p.176). The well known evidence of Evans (1974) contradicted this statement. He found no bone cancer in US female radium dial painters who had been exposed to low dose irradiation. He suggested the practical threshold for skeletal radium was 10 Gy. These data were refined (Figure 8) by Rowland et al. (1983). The great majority of radium dial painters had no cancers. The threshold for over one thousand US female dial painters employed before 1950 was about 0.1 mCi for total body content of radium. Those exposed to less than the threshold lived normal, healthy lives. As shown by Baverstock et al. (1986), had a two-tailed statistic been used instead of a one-tailed statistic, the data would have revealed radiation hormesis (see BEIR V).

The BEIR IV committee stated: "In the absence of sufficient human surveys to calculate risk estimates for cancer induction, the animal data, together with data of radium-224 and radium-226 in humans, provide a basis for cancer risk estimation" (BEIR IV, 1988, p.16). The committee certainly knew that Schoeters and Vanderborght (1986) had found mice injected with low dose of ^{226}Ra had a statistically longer average lifespan than control mice.

Although cancer from exposure to plutonium is rare, the committee estimated: "the analysis yields, for plutonium deposition in human bone, a lifetime risk estimate of 3×10^{-3} per person-Gy (300 excess bone-cancer deaths per million person-rad) to bone" (BEIR IV, 1988, p.17). This concept was not evident in the data from plutonium workers in the Manhattan Project (Voelz et al., 1979) and Wilkinson et al., 1987).

The concept was certainly denied by the lack of cancer deaths in persons injected with 46 to 6400 cSv Pu (Luckey, 1998; Moss and Eckhardt, 1995); none of these people died with cancer 1.2–44 years following their injection.

Figure 8 Dose-response curve for bone sarcomas in 1468 US female radium dial painters employed before 1950 (Rowland et al., 1983). The ordinate indicates the number of bone cancers per person-year of exposure. The abscissa indicates the measured intake of radium isotopes
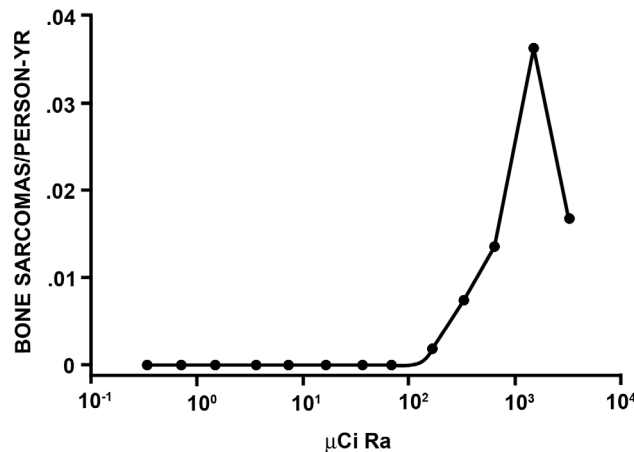

2.5 BEIR V

The biologic effect of low doses of many radionuclides is the subject of BEIR V (1990). Adherence to the dogma, all radiation is harmful, is evident throughout the BEIR V report. The BEIR V Committee stated: "Of the various types of biomedical effects that may result from irradiation at low doses and low dose rates, alterations of genes and chromosomes remain the best documented"(BEIR V, 1990, p.4). This is a misconception; it is certainly not true at the physiological level.

Studies consistently showed fewer genetic alterations in children of Japanese bomb victims than in controls (see Figure 2). The evidence did not deter the BEIR V committee from its central dogma: ". . .the frequency of such effects (somatic and genetic damage) increases with low-level radiation as a linear, non-threshold function of the dose" (BEIR V, 1990, p.4). More specifically, for the variety of radionuclides addressed in this report, the committee stated: "The carcinogenic and mutagenic effectiveness per Gy of neutrons and other high-LET radiation remains constant or may even increase with decreasing dose and dose rate" (BEIR V, 1990, p.7). These conclusions are not in agreement with the results from 77,000 children of Japanese atom bomb survivors using the most sensitive changes in electrophoretic patterns of serum proteins (Neel et al., 1988; Schull et al., 1981).

Low doses of ionising radiation decreased mortality rates for both leukaemia and solid cancer in 32,000 military observers of atomic bombs Robnette et al. (1985). The data (Figure 9), which show decreased cancer and leukaemia with increased exposures, defy the statement in the BEIR V overview: "The studies have provided no evidence to date that risk estimates for leukaemias and other types of cancer combined are in error, based on extrapolation from high-dose studies" (BEIR V, 1990, p.4). The committee preferentially ignored the main data base and used the statement of Caldwell et al. (1983) regarding the much publicised statement for a small segment of the Pacific tests: "Statistically significant increased frequency of occurrence and mortality was found only for leukaemia." This statement was true for only one (SMOKY) of many Pacific atomic bomb tests.

Figure 9   Cumulative leukaemia (the ordinate at the right) and solid cancer (left ordinate) mortality rates in the US military observers of atomic bombs (Robinette et al., 1985). The total doses were determined from data on film badges
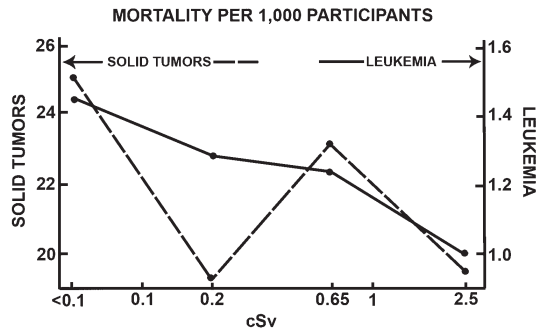

The risk of leukaemia was considered to be a linear function by the BEIR V committee: "The risks of acute leukaemia and of chronic myeloid leukaemia are increased by irradiation of the haemopoietic cells, the magnitude of the increase depending on the dose of radiation" (BEIR V, 1990, p.252). This concept is found to be untrue by the following evidence. The data (Figure 10) of Shimizu et al. (1987, 1990) showed low dose irradiation of Japanese atomic bomb survivors gave less leukaemia mortality. Ignoring the facts, the BEIR V committee (1990) had two comments. On page 354 the BEIR V committee "estimated the risk per unit absorbed dose to be about 200–250 excess cancer deaths/10,000 person-Gy in the first ten years of life, with one half of these malignancies being leukaemia and one quarter tumours of the nervous system" and on page 383: "The studies have provided no evidence to date that risk estimates for leukaemia and other types of cancer combined are in error, based on extrapolation from high-dose studies." The BEIR V committee adhered to the LNT dogma and failed to note that those who received less than the threshold, about 14 cGy, had less leukaemia mortality than the control population.

Figure 10   Leukaemia mortality ratios for all Japanese atomic bomb victims (Shimizu et al., 1987, 1990)
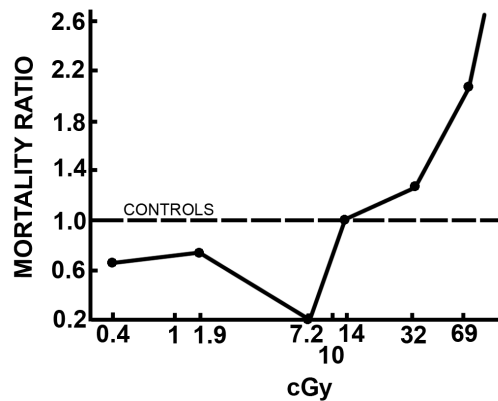
The BEIR V committee also ignored the data of Baverstock et al. (1986) who recorded deaths from radium: ". . .the lifetime risk of bone cancer from internally deposited ^{224}Ra has been estimated to be about 2×10^{-2}/person Gy" (BEIR V, 1990, p.310). When radium dial painters stopped licking the points of their brushes, about 1925, bone cancer deaths in painters hired subsequently were dramatically diminished. When compared with the control population, the standard mortality rate (SMR) for cancer deaths in 1203 British female dial painters who had less than 40 person-years of work had cancer death rates below that of the control population (Figure 11). Rowland (1997) commented: "Risk, not health, is the only issue considered. BEIR and Mays ignore hundreds of dial painters exposed to below 10 Gy with no bone sarcomas." Except for persons with bone sarcomas, dial painters lived as long as and in as good health as the rest of the population.

Figure 11   Dose-response curve for bone sarcomas in British female radium dial painters
(Baverstock et al., 1986). The ordinate indicates the standard mortality rate. The
abscissa indicates person-years at work
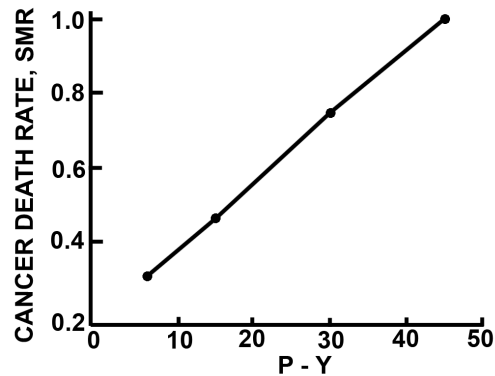
The BEIR V committee inadvertently acknowledged the role of radiation in activation of the immune system (BEIR V, 1990, p.364): "In each experiment, however, the survival of the non-irradiated controls was compromised by mortality from undercurrent infection." Radiation activated immunity is thoroughly discussed with specific examples in Chapter 5 of Luckey (1991).

2.6   BEIR VI

BEIR VI begins with a biased view of the history of lung cancer in miners: "For a century, it has been known that some underground miners suffered from higher rates of lung cancer than the general population." Lorenz (1944) cited several 19th century investigators who found no lung cancer in over 700 active miners in the Schneeberg mountains (east of Dresden). Beckmann (1989) showed that the original study was faulted: "No cancer was found in 323 active Joachimsthal miners. Active miners of the 19th century died from accidents, emphysema and suffocation, not lung cancer. Early publications compared the lung cancer death rate of old, retired miners with that of the general population." Since cancer is a disease of the aged, this is unacceptable epidemiology.

The BEIR VI Committee (1999) presented a summary graph (p.89) of lung cancer death rates from 11 studies of underground miners which involved 68,000 men and 2799

lung cancer deaths (Figure 12). The data show there is no significant change in relative risk of cancer deaths up to 11 pCi/l of radon (this is the maximum found in homes in the US; the average is 1.25 pCi/l). Mis-information from the BEIR VI committee (1999) promulgate the LNT dogma. Compare their own data (Figure 12) with their statement: "The committee agreed with several earlier groups of experts that the risk of developing lung cancer increases linearly as the exposure increases; for example, doubling the exposure doubles the risk and halving the exposure halves the risk. Furthermore, the existing biologic evidence suggests that any exposure, even very low, to radon might pose some risk" (BEIR VI, 1999, p.2). Their executive summary states (p.4): "Radon . . . has been conclusively shown in epidemiologic studies of underground miners to cause lung cancer" and on p.18, they inform us: "The carcinogenicity of radon is convincingly documented through epidemiologic studies of underground miners, all showing a markedly increased risk of lung cancer." In order to explain any increase in lung cancer in miners, other factors must be considered:

- smoking
- dust and metal particulates
- diesel fumes
- arsenic and selenium
- moulds and mycotoxins
- poor ventilation
- other radionuclides
- interactions of the above.

Radon has been shown to cause lung cancer in rats only when exposed to 10,000 pCi/l; 100 pCi/l and even 1000 pCi/l showed no effect (Cross et al., 1986).

Figure 12   Summary of the relative risk of lung cancer mortality from radon in 11 studies of underground miners. The abscissa indicates the concentration of radon. The 95% confidence limits are displayed. (Modified from p.89, BEIR VI, 1999)
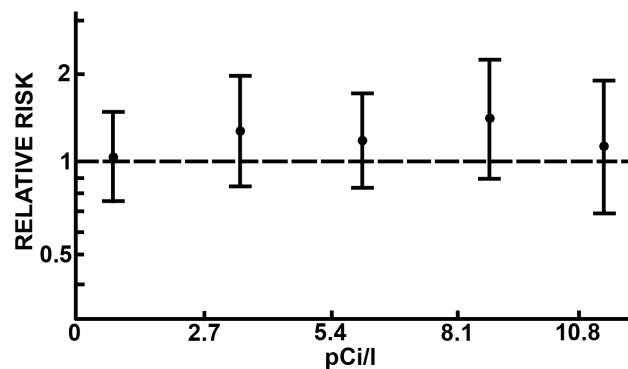
Although the BEIR VI committee had reservations about case-control studies for radon caused lung cancer, it expressed confidence in meta analysis of such data; a large section of the report was devoted to this topic. The case-control method involves direct, long term measurements of radon in homes. Data from these case-control studies indicate there is no significant increase in lung cancer mortality rates due to radon in homes (Figure 13). Since it involves considerable time and effort, the data are sparse. Cancer cases for each location are: Finland I, 238; Finland II, 1055; Sweden, 1281; Stockholm, 201; Shenyang, 308; Winnipeg, 738; Missouri, 538 and New Jersey, 480. However, on page 2 the committee estimated radon causes 15,400 to 21,800 lung cancers per year in the US. This statement is refuted by their own data. Finally, the BEIR VI committee (1999) stated: "Nevertheless, this indicates a public health problem and makes radon the second leading cause of lung cancer after cigarette-smoking." The committee does not concede that the epic study of Cohen (1995) proves that radon in homes prevents, not causes, lung cancer.

Figure 13   Relative risks of lung cancer from radon in homes from eight case-control studies. The abscissa indicates the concentration of radon. Modified from Figure G-1, p.177, BEIR VI, 1999
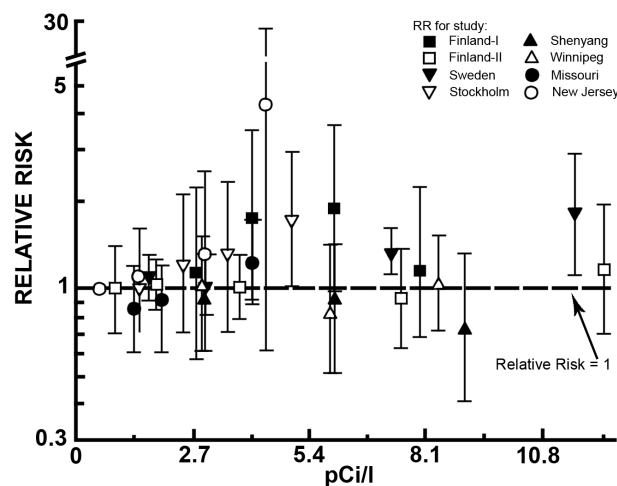
In spite of the above statement, the BEIR VI committee (1999, p.361) acknowledged the "strong negative correlation between estimated county-average radon exposure and lung cancer mortality" of Cohen (1995). In about 700,000 radon tests from 1600 counties of the US, increased radon concentrations were strongly associated with decreased lung cancer deaths (Figure 14). This could not be explained by 54 possible confounding factors. The BEIR VI committee's evaluation of Cohen's massive work was: "The finding was considered to be an inappropriate basis for concluding that indoor radon is not a potential cause of lung cancer" (BEIR VI, 1999, p.379). They adhered to the LNT dogma.

Figure 14   Lung cancer deaths decrease in direct proportion with the logarithm of the radon
concentration in US homes (Cohen, 1995). The numbers of US counties for each
point are noted
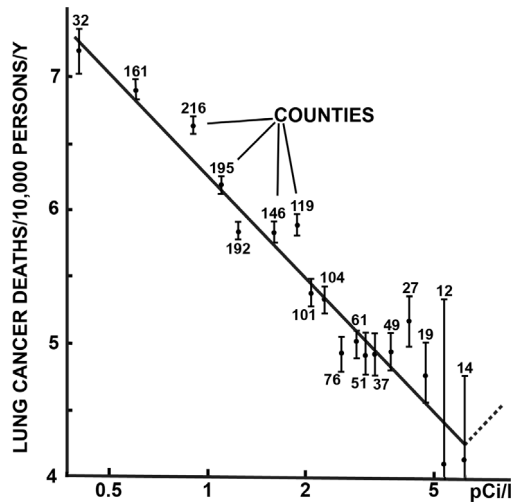
2.7   BEIR VII

The BEIR VII Committee (2005) devoted about 600 pages to the LNT dogma with little
new information. "The committee and staff ensured that BEIR VII's conclusions were
informed by a thorough review of published, peer reviewed materials relevant to the
committee's formal Statement of Task" (BEIR VIII, 2005, p.18). The summary for BEIR
VII states: "The main studies establishing the health effects of ionising radiation are those
analysing survivors of the Hiroshima and Nagasaki atomic bombings in 1945" (BEIR VII,
2005, p.19).

The data show unexpected benefits from acute exposure to low dose irradiation. Mine
et al., (1996) found that those Japanese survivors of atomic bombs who received less than
14 cSv had a longer average lifespan (p < 0.01) than the control population (Figure 15).
The relative risk was 1.3 at 325 cGy.

Figure 15   Relative risk for total mortality in Japanese atomic bomb survivors from 1950–1985
(Mine et al., 1996)
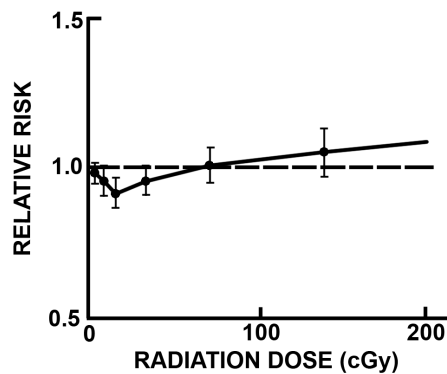
When non-cancer death rates of the Japanese atomic bomb survivors were considered (Figure 16), the threshold was 155 cSv (Shimizu et al., 1982, 1992). Their data showed no life shortening in 20,777 Japanese atomic bomb victims. The BEIR VII committee states: "Instead, the committee concludes that the preponderance of information indicates that there will be some risk, even at low doses" (BEIR VII, 2005, p.19). This statement is not justified by the data.

Figure 16 Relative risk of non-cancer deaths in Japanese survivors from atomic bombs (Shimizu et al., 1992). The abscissa indicates total body dose as estimated from position and protection at the time of the explosion
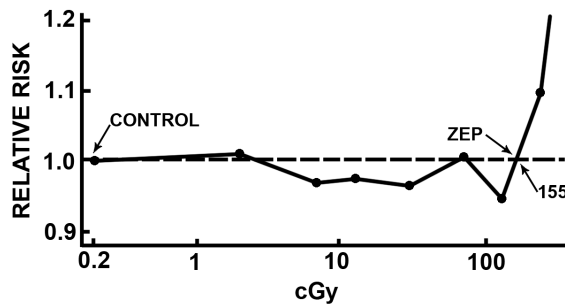
The committee failed to recognise the significant decrease ($p < 0.01$) in cumulative, total cancer mortality rates in Japanese victims of atomic bombs (Figure 17). The cumulative total cancer death rate of the 16,665 persons exposed to less than the threshold value (about 7 cSv) was less than that of the controls. The 23,104 persons who received less than 10 cSv had cancer death rates which were not statistically greater than that of the control group. Mis-representation by Shimizu et al. (1992) was: "In general, the dose response . . . failed to suggest the existence of radiation hormesis."

Figure 17 Cumulative total cancer mortality rates in 86,000 Japanese survivors of atomic bombs. The dose is presented on a logarithmic scale. Compiled from the table, p.72 (Shimizu et al., 1992). Note, two data points provided a weighted average for one point on this graph
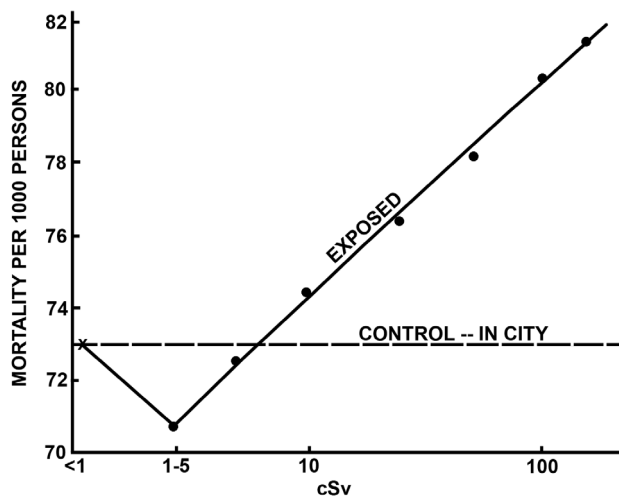
Representatives of the Radiation Effects Research Foundation (RERF), Pierce and Preston (2001), continue to mislead the public; compare the data in Figure 1 with their summary statement: "There is every reason to conclude from the RERF data that the usual linear cancer risk (i.e., LNT) estimation is appropriate for low doses." They ignore the fact that the total cancer death rate in 7430 persons exposed to 1–1.9 cSv was significantly less ($p < 0.01$ with the chi square statistic) than that of the 45,148 persons in the control population.

Much of the decreased cancer mortality of Japanese victims of atomic bombs (Figure 17) may be attributed to the decreased cumulative leukaemia deaths in this cohort (Figure 18). The fallacious response of Shimizu et al. (1990) was: "The excess in leukaemia mortality has continued to decline with time but remains slightly and significantly elevated in 1981–1985 in Hiroshima."

Figure 18   Cumulative leukaemia death rates of Japanese survivors of atomic bombs (Shimizu et al., 1987, 1990). The cohort size is indicated within the graph
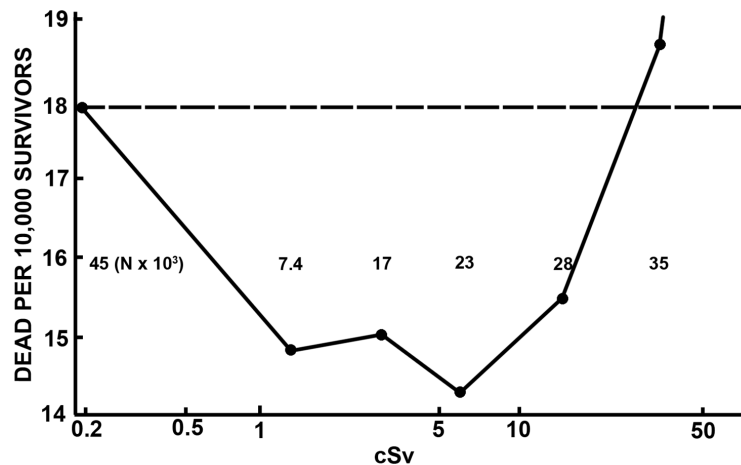
Many times the BEIR VII committee agreed with radiobiology authors' misrepresentations and consistently ignored the data presented: "The committee concludes that the current scientific evidence is consistent with the hypothesis that there is a linear, no-threshold dose-response relationship between exposure to ionising radiation and the development of cancer in humans" (BEIR VII, 2005, p.30). One example which disproves that statement comes from 31,700 Canadian women who were monitored with multiple fluoroscopic examinations during treatment for tuberculosis (Miller et al., 1989). The data show hormesis (Figure 19). Low dose irradiation was beneficial ($p < 0.01$) and there was a threshold at about 49 cGy. The authors make no mention of this effect in their abstract, text or summary. The BEIR committee's conclusions misrepresent these data: "The data were most consistent with a linear dose-response relation" and ". . .the most appropriate form of dose response relation is a simple linear one" (BEIR VII, 2005, p.321)

Figure 19 Mortality rates from breast cancer in women examined for tuberculosis by repeated fluoroscopic examinations to monitor lung collapse therapy (Miller et al., 1989). The abscissa indicates total exposures from x rays. One standard deviation is expressed
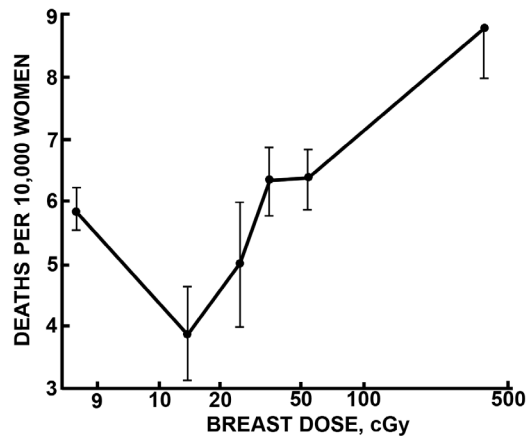
3 Exposed nuclear workers

The well controlled epidemiological studies of exposed nuclear workers represent the best large database for low radiation dose-response effects in humans. Most were white males. The healthy worker effect was negated by careful selection of a control worker to correspond with each exposed worker. Each control was chosen from the same working environment and had the same sex, age and socio-economic background as the exposed worker. They had similar medical examinations and care. Exposures were caused by an array of accidents. Lifetime exposures were calculated from film badge data.

When considering mortality rates, data from exposed nuclear workers are more reliable than those from Japanese atomic bomb victims. As the BEIR III committee pointed out: "Because the atomic bomb survivors had high mortality rates from infectious disease for several years after the bombings, atomic bomb victims with early radiogenic cancers may have succumbed to fatal infections to such an extent that estimates of carcinogenic risks based on the atomic bomb experience would not be generally applicable to populations for which radiation protection guidelines are written" (BEIR III, 1980, p.156). In addition, the nuclear workers did not

- suffer the trauma of an atomic bomb explosion

- live in a devastated environment

- eat or drink undetermined radionuclides in food and drinks

- have a hint of radiation hormesis in controls taken only 3 km from the zero point

- have guestimated exposures which depended upon where each person thought they were at the time of the bomb and where they went thereafter.

4   Lifespan

In Chapter 3 of *Radiation Hormesis,* Luckey (1991) reviewed the increased average lifespan of lightly irradiated laboratory animals and humans. The average lifetime of exposed nuclear workers was consistently found to be somewhat greater than that of the carefully unexposed workers. The lower mortality rate of exposed workers was due to fewer infections, lung diseases, cancers and aging. Since accidental deaths were similar in the two groups, the difference may be attributed to activation of the immune system in lightly irradiated persons (see Chapter 5 in Luckey, 1991). Specific examples follow.

- From a survey of 95,000 workers in atomic plants in Britain, Kendall et al. (1992b) found the total mortality rate of exposed workers was 17% less than that of the general population. Although this difference was statistically significant ($p < 0.001$), the total mortality rate of nuclear workers was about 10% less than that of all industrial workers.

- In a study of 8000 nuclear workers at the Oak Ridge National Laboratory, Wing et al. (1991) found the total death rate of all exposed workers was only 37% that of unexposed controls. The authors' statement from large doses of radiation is deceiving: ". . .all cause mortality increased 2.68% per 10 mSv."

- From a pool of 700,000 nuclear shipyard workers, Matanoski (1991) carefully selected 32,500 non-nuclear workers (NNW) to match 38,000 exposed workers. The results showed that increased radiation decreased total mortality rates (Figure 20). This was not evident from her text or publicity releases from government agencies. Although the results of this decades-long evaluation were known to the BEIR V committee (1990) (Dr A. Upton was chair of the BEIR V committee and chaired the technical advisory committee that advised Dr Matanoski at John Hopkins University), the BEIR V committee conclusion states: "The bulk of the epidemiological data appear to be consistent with the data from laboratory animals" and "In laboratory mammals exposed to whole body radiation life expectancy decreases with increasing dose." (BEIR V, 1990, pp.363–364). These statements do not agree with the 24% reduction in mortality rates in the results of Matanoski. As shown in Figures 5 (Spalding et al., 1964) and 6 (Lorenz et al. 1955), the BEIR V committee's summary of these data from laboratory animals was obviously wrong

- Wiggs et al. (1994) recorded 3196 deaths among 15,727 workers at the Los Alamos National Laboratory which produced the first atomic bomb. The mortality rate of workers exposed to > 1 cSv was only 91% that of workers exposed to < 1 cSv.

- Cardis et al. (1995) noted that the data from 84,000 workers showed no change in total mortality rates for workers who received between1 and 50 cSv. Since they used only a one-tailed statistic, their reasoning allowed no possibility for the data to exhibit radiation hormesis.

- Frome et al. (1997) examined the total mortality rates for 4786 exposed nuclear workers (602,000 worker-years) from the Oak Ridge National Laboratory (Figure 21). The threshold was about 20 cSv. As indicated earlier, careful epidemiology eliminated any healthy worker effect. Although they noted that the standard mortality rate (SMR) was 0.80, they stated: "For external radiation dose with a 10-year lag, the excess relative risk was 0.31 per Sv for all causes." They ignored the benefit from low dose irradiation and provided mis-information for their own reasons.

Figure 20   Total mortality rates in exposed nuclear shipyard workers compared with that of carefully selected non-nuclear workers (NNW) (Matanoski, 1991). The abscissa indicates exposures from lifetime film badges. The numbers of workers in each group are indicated
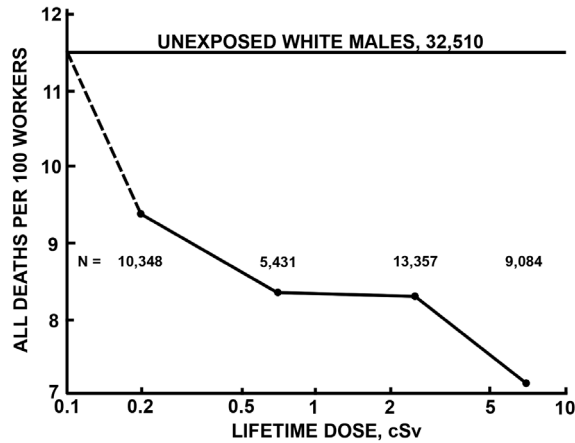
Figure 21   Dose-response data for all mortality in 4786 deaths of exposed nuclear workers at the Oak Ridge National Laboratories (Frome et al., 1997). The relative risk for all deaths is plotted against the total exposure as determined by film badges
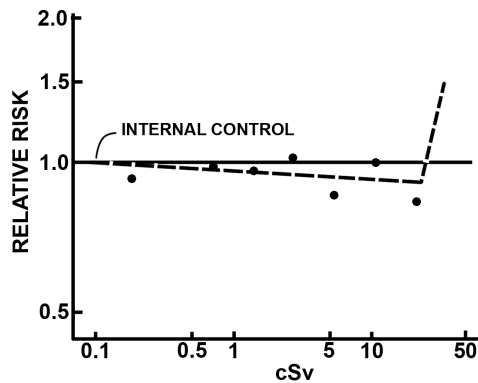
5   Cancer mortality

The committee considers cancer induction to be the most important somatic effect of low-dose ionising radiation" (BEIR III,1980, p.136). This misstatement ignores the considerable evidence which indicates:

- People live in environments with over twice the world average of ionising radiation (Luckey, 2007).

- Radiation prevents cancer (Luckey, 1999, 2008). Although the BEIR committees had the literature processed by experts, they refused to give serious consideration to radiation hormesis.

Cancer death rates in exposed nuclear workers illustrate the benefits of low dose irradiation. Most epidemiological studies had a ten year lag period for cancer and a two

year lag period for leukaemia before mortality data are registered. These well executed epidemiological studies negate any "healthy worker effect" by careful selection of a comparable unexposed control for each exposed worker. Exposures were determined by film badges. Routinely, the authors omitted reference to hormesis or any beneficial effect.

There were eight studies (Figure 22) which showed the cancer mortality rates of exposed nuclear workers decreased in direct proportion to increased exposure to ionising radiation (Luckey, 1997a). With some overlap, the data involve 149,000 (7 million person-years) nuclear workers as controls with 152,000 (4.7 million person-years) exposed workers (Luckey, 1999). Specific comments follow the numbering system used in Figure 22.

Figure 22   Cancer mortality rates from eight studies of exposed nuclear workers.
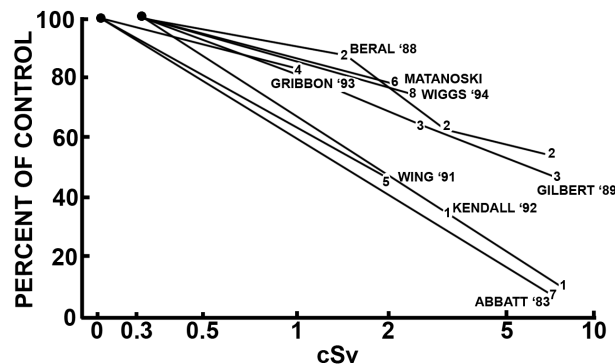
Notes: The sites, numbers of exposed workers and references are, respectively:
  1) British weapons, 36,000 (Kendall et al.,1992a,b); 2) British weapons, 9,000 (Beral et al., 1988); 3) Hanford Site/Rocky Flats, 15,000 (Gilbert et al., 1989); 4) Canada Energy, 4000 (Gribbin et al., 1993); 5) Oak Ridge National Laboratory, 6000 (Wing et al., 1991); 6) nuclear shipyards, 41,000 (Matanoski et al., 1984); 7) Canada Energy, 4000 (Abbatt et al., 1983); and 8) Los Alamos, 8000 (Wiggs et al., 1994). Individual exposures were determined by film badges

1   Kendall et al. (1992a,b) reported on 95,217 nuclear workers in the National Registry for Radiation Workers from 1952–1988. This complex study includes the decreased starting age of the workforce with time, the increased cancer incidence with age and the reduction of average annual radiation within the plants from 8–2 mSv/y. The age corrected total dose-response curve for all cancer (Figure 23) shows a striking protection for those who received the most radiation. The standard cancer mortality rate for all workers was 85 % that of the British-Wales population. The authors' conclusions do not fit their data: "Although the standard mortality ratios for these disease groupings ('All malignant neoplasms') were below 100, there was some evidence for an increase in mortality with radiation dose. . .." They stated: "The positive trend with dose for all cancers, from which the risk estimate was derived, was not significant" and "With excess relative risks derived from internal analysis of the registry the central estimate of the total risk of radiation induced cancer for a British worker population is 10%/Sv." They concluded: "There is evidence for an association between radiation exposure and mortality from cancer. . .." They apparently ignored the three important variables noted above.

2 Beral et al. (1988) examined the records of 23,700 workers in the British atomic weapons establishment for an average of 19 years. Using data from their Table VII, the results show radiation hormesis (Figure 24). Authors' comments ignore the data; for example on p.757: "Mortality from malignant neoplasms as a whole showed a weak and non-significant increasing trend with increasing level of cumulative whole body exposure to external radiation." "When the exposures were lagged by ten years, the trend became stronger and significant, the estimated increase in relative risk per 10 mSv being 7.6%" and on p. 769: "With a lag of ten years, however, mortality from all malignant neoplasms increased significantly with increasing level of cumulative whole body exposure (Table VII). . .." Also on p.769: "With a ten year lag the relative risk was estimated, by using a multiplicative model, to increase by 7.6% per 10 mSv increment in exposure." Apparently, the authors believed repetition would hide their data.

3 Gilbert et al. (1989) found the cancer mortality rate of 23,755 exposed male nuclear workers at the Hanford Site decreased dramatically with increasing exposure (Figure 25). From 1944–1981 the cancer death rate of workers exposed to 6 cSv was 50% that of carefully selected control workers. The authors' statement was misleading: "The absolute excess risk estimate for all cancer except leukaemia obtained from the Hanford data was 13 deaths per million person-years per 10 mSv." With no thought of evaluating any biopositive effect, the BEIR committee simply accepted the authors' evaluation. These data prove the fallacious nature of the BEIR V committee's (1990, p.383) response: ". . .the frequency of such effects (somatic and genetic damage) increases with low-level radiation as a linear non-threshold function of the dose" and "the absolute excess risk estimate for all cancer except leukaemia obtained from the Hanford data was 13 deaths per million person years per 10 mSv." Both the authors and the BEIR V committee considered only risk with no concept of radiation hormesis.

4 Gribbin et al. (1993) evaluated the cancer mortality rates of 8977 males in the Atomic Energy of Canada, Ltd.; 4260 were exposed to low LET (linear energy transfer) radiation: ". . .the values reported are one-sided since the hypothesis of interest is that radiation exposure increases risk." None of the values reported with this small sample were statistically significant. Neither this nor the other Canadian study, Abbatt et al. (1983), used selected internal controls as had all the other studies in Figure 22.

5 Wing et al. (1991) examined data from 1524 deaths in 8318 nuclear workers at Oak Ridge National Laboratory (ORNL) from 1943–1984. Their data (Figure 26) show the average cancer death rate in all exposed workers was only 50% that of unexposed controls. The authors misleading conclusion (1991, p.1400): ". . . all cancer mortality increased 4.94% per 10 mSv." On the same page, these authors also stated: "The radiation-cancer dose response is ten times higher than estimated from the follow-up of survivors of the bombings of Hiroshima and Nagasaki." The obvious concern of the authors was risk, not health.

6   Matanoski et al. (1984, 1991a,b) chose 33,000 non-nuclear workers (NNW), from
    about 700,000 shipyard workers, to match 39,000 exposed nuclear workers in order
    to examine the effect of low dose irradiation on cancer induction (Figure 27). Most
    of the exposure to shipyard workers was gamma rays from cobalt-60. Although
    Matanoski et al. provide evidence of radiation hormesis, they discuss only the harm
    from ionising radiation.

7   Abbatt et al. (1983) examined the cancer mortality rates of 40,000 energy workers in
    three Canada corporations of the nuclear fuel cycle. Uniformity of personnel was
    provided by all employees having the same screening and medical care. The results
    (Figure 28) showed that the cancer death rate of workers in the nuclear industry was
    40% less that that of the other (gas and coal) energy industries. The mortality rates
    of thermal workers were comparable with the mortality rate of males in Ontario from
    1971 to 1976.

8   Wiggs et al. (1994) examined records of 15,727 male workers at the Los Alamos
    National Laboratory. The 36 years of records includes some men who were exposed
    to plutonium. Using a ten year lag, the total cancer deaths of those exposed to 1–5
    cSv was 86% that of the control group who were exposed to < 1 cSv. The authors'
    comments (p.581) included: "Mortality from all causes and all cancers did not
    demonstrate a trend with radiation dose." Their data indicate radiation hormesis.

Figure 23   Age adjusted cancer mortality rates in 95,000 British workers in weapons plants
           (Kendall et al., 1988a,b). The numbers of workers in each group are noted
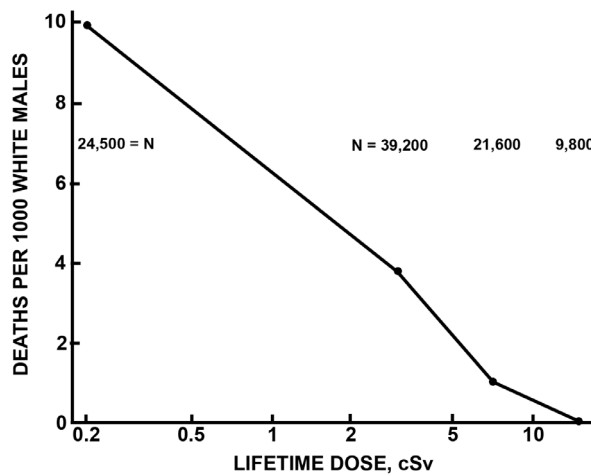
Figure 24 Cancer mortality of workers at British atomic industries (Beral et al., 1988). There were 13,163 unexposed workers and 9899 exposed workers (three exposed to > 100 mSv are not shown here)
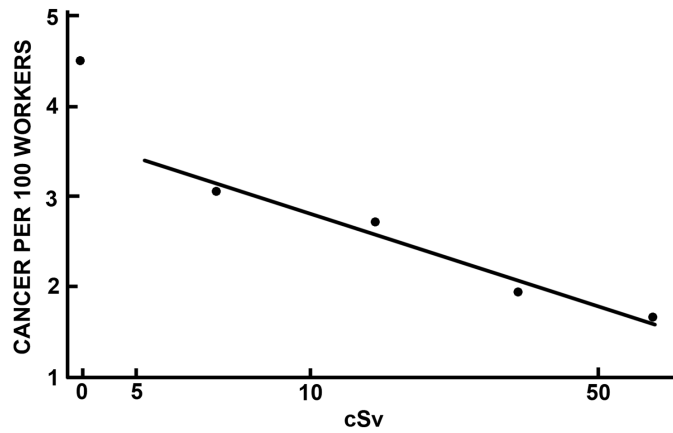

Figure 25 The cancer mortality rates for about 24,000 nuclear workers at the Hanford Plant decreased with increased exposure (Gilbert et al., 1989). The number of workers in each group is indicated within the graph
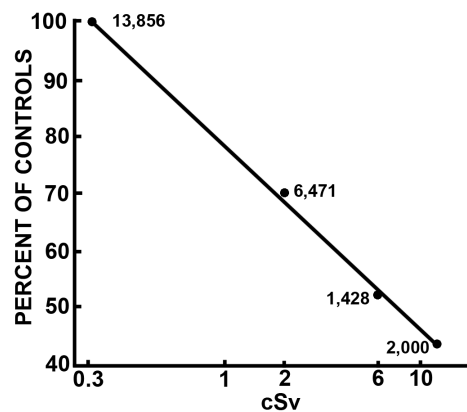

In a study which overlapped that in Figure 22, Cardis et al. (1995) noted: "Combining data from seven cohorts in three countries has provided the opportunity to obtain the most comprehensive and precise direct estimates to date of the carcinogenic effect of low LET radiation at low doses and low dose rates." The data showed radiation hormesis (Figure 29). The 32,000 exposed nuclear workers had cancer mortality rates which were less than the rate for the 45,825 control nuclear workers. The authors' misstatements (p.117) include: "There was no evidence of an association between radiation dose and mortality from all causes or from all cancers" and "As there is no reason to suspect that exposure to radiation would be associated with a decrease in any specific type of cancer, one sided tests are presented throughout." Had they graphed their data, radiation hormesis would have been indicated.

Figure 26   The cancer mortality rate of 6189 exposed nuclear workers was 50% that of 2129 control workers at Oak Ridge National Laboratory (Wing et al., 1991)
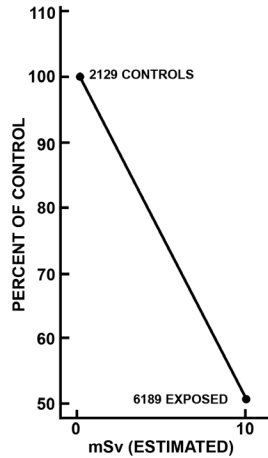
Figure 27   Cancer mortality rates of nuclear shipyard workers (Matanoski, 1991). The mortality rate for 39,000 exposed workers was less than that of 33,000 carefully chosen unexposed nuclear workers (NNW)
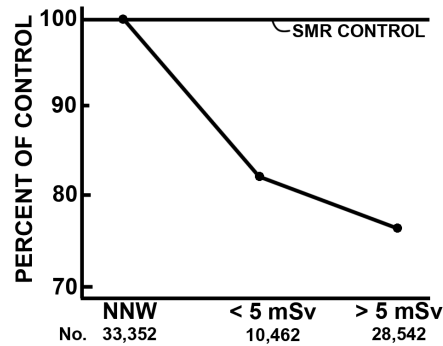
Figure 28   The standard mortality rate (SMR) for cancer mortality in the general population of Ontario and that of workers in thermal energy plants is compared with 40,000 workers and pensioners (mining, reactor and research) of the nuclear fuel cycle (Abbatt et al., 1983)
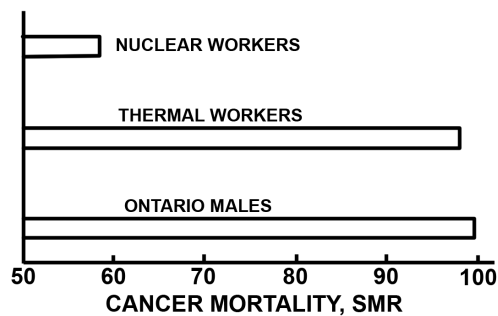
Figure 29 Total cancer mortality rates in nuclear workers (Cardis et al., 1995). The numbers of workers in each group are indicated within the graph
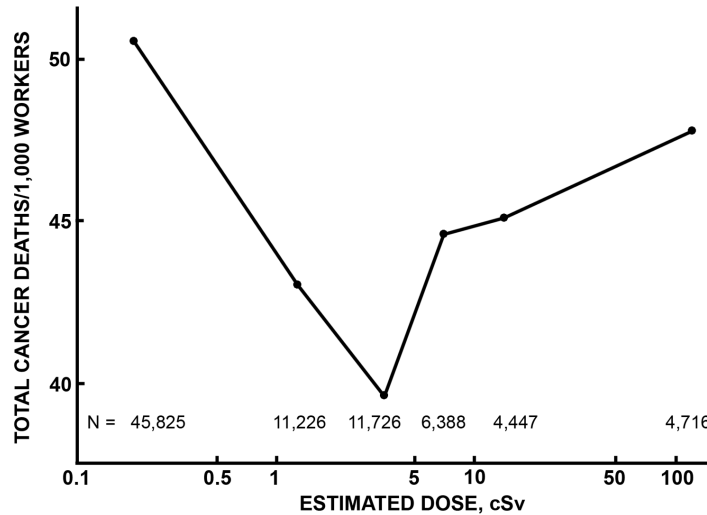
Another example of radiation hormesis comes from the plutonium exposures of 21,000 Russian nuclear workers at the Mayak Facility (Tokarskaya et al., 1997). From 1948–1972, about 11,000 workers received an average of 1.2 Gy (BEIR VII, 2005, pp.357–358). The lung cancer mortality rate was lower in those groups which received low dose irradiation; in those who received 1.2 cSv, lung cancer mortality was only 61% that of the control group (Figure 30). The committee did not mention the possibility of any beneficial effect which was significant ($p = 0.05$) for the relative risk of cancer deaths in this group. Since they did not use a two-tailed statistic (which would reveal a biopositive effect), the BEIR VII committee could report: "The arguments for thresholds or beneficial effects are not supported by these data" (BEIR VII, 2005, p.19). This statement fits the LNT dogma, not the data.

Figure 30 Relative rates (RR) of lung cancer deaths among plutonium workers at the Mayak Facility in Russia (Tokarskaya, 1997)
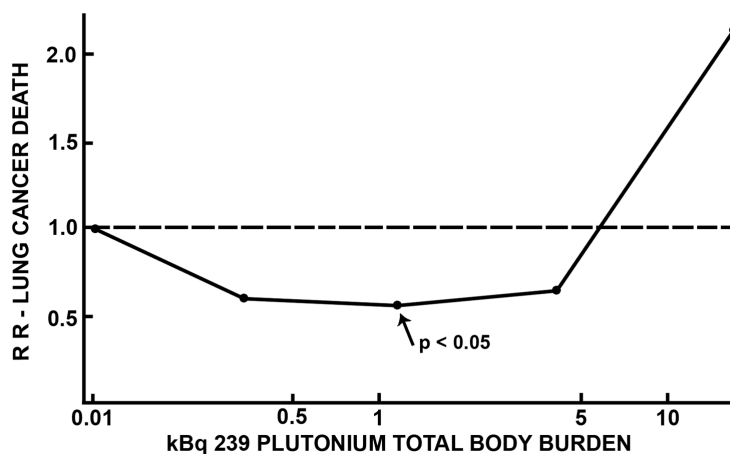
6   Discussion

The evidence is abundant and clear. Many specific studies have shown that low dose irradiation decreases cancer mortality rates. There are no scientifically valid studies which show harm from low dose irradiation to normal humans or laboratory mammals. Misinterpretation of the data by radiobiologists and, particularly, by the BEIR committees, has rejected health. A comparable dissection and indictment could be made for many national and international committees and agencies (Aurengo et al. 2005). The LNT and "all radiation is harmful" dogmas have no scientific basis in mammalian physiology.

The deceptions of the BEIR committees were not performed in a vacuum. Many radiobiologists, our government, national and international societies, physicians, the media and most people believe the LNT dogma; i.e., all radiation is harmful. Publications from other agencies in this country (NCRP, ICRP, EPA and even the *Journal of Health Physics*) and in other countries reveal comparable discrepancies. In 1975, both the National Research Council (NRC) and the Environmental Protection Agency (EPA) adopted the LNT paradigm: "There is no safe level and no threshold for human exposure to ionising radiation" (BEIR II, 1977, p.90). For more than 60 years the LNT paradigm has dictated the flow of research monies and the acceptance of scientific papers.

Responsibility for making the LNT paradigm the law of the land includes the highest authorities in science. The NRC, which repeatedly accepted the BEIR reports and continued to appoint biased committees, was the working arm of the National Academy of Science (NAS). Both NRC and NAS included honoured scientists who routinely accepted the deceptions of the BEIR committees.

Due to the abundance of information, radiation induced cancer was used here as the focal point for health. The evidence indicates that, prior to aging, about 90% of cancers could be prevented. This would directly affect 500,000 cancer traumatised families and deaths in the US (Anon, 2006) and several million in the world every year. This should be condemned as serial murder on a massive scale. It is subliminal terrorism! Ignorance should not be an excuse for physicians and health physicists to allow millions of preventable deaths. Is it ethical for physicians to "do no harm" while patients die from a preventable radiation deficiency?

Health is a many-faceted entity. New concepts for health include:

- ionising irradiation is an essential agent (Luckey, 1991, 1997a)
- we live in a partial deficiency of ionising radiation (Luckey, 1997a), the optimum exposure is about 60 mSv/y (Luckey, 2007)
- optimum radiation levels would markedly reduce death rates from cancer and infections (due to an activated immune system (Luckey, 1991, Liu, 2002)) prior to aging (Luckey, 1999, 2007)
- greater exposure to ionising radiation would perceptibly increase the average lifespan (Luckey, 1991)
- radiobiologists, physicians and health physicists should provide adequate exposure to ionising radiation for every person
- abrogate all old laws
- new laws should be based upon health, not risk and fear
- the maximum allowable radiation should be 1000 times the present legal maximum; the threshold is 8 Sv/y (Luckey, 2007).

Freedom from unnatural restrictions will allow us to live in harmony with ionising radiation. Freedom from the dogma of "all radiation is harmful" will lead not only to more abundant health; it will release industry, particularly nuclear power, from needless shackles (Muckerheide and Rockwell, 1992). As Dr S. Hattori (1994) stated: "If radiation hormesis exists, our daily activities in radiation management have been extremely erroneous."

7 Conclusions

Many radiobiologists ignored radiation hormesis and joined the NLT mafia. The seven committees of the BEIR reports adhered to the "all radiation is harmful" dogma. They have guided the US government into ruinous health and energy policies. This treatise on health, with emphasis on cancer mortality, reveals our failure to fully use ionising radiation as a valuable agent in our environment.

Acknowledgements

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Appendix

The BEIR I Committee (p.6, 1972) noted: "On 2 December, 1970 the activities and functions of the FRC (Federal Radiation Council) were transferred to the Radiation Office of the EPA. Because the FRC had ceased to exist as a specific body, the NAS-NRC Advisory Committee requested a change in title. The President of the NAS renamed the Committee, the Advisory Committee on the Biological Effects of Ionising Radiation (BEIR): functions, activities, membership and staffing were not changed." More on p.6: "Independently and not as an agent of the government, the contractor shall furnish to the Department of Health, Education and Welfare, for the Federal Radiation Council, consultation and advisory services on the evaluation and interpretation of scientific problems pertaining to the biological effects of ionising radiation", on p.8 "The present standards used by the Federal Government are based on the recommendations of the Federal Radiation Council (FRC)" and finally, (p.58, BEIR I, 1972): "We remind all who may use our estimates as a basis for policy decisions that . . .". A major adherent continues to be the Environmental Protection Agency (EPA).

The Foreword of the report (BEIR I Committee, 1972) stated: "We hope that the information contained herein will serve . . . as a scientific basis for the development of suitable radiation protection standards" and the preface (p.iii, BEIR I Committee, 1972) began: "This report of the National Academy of Sciences – National Research Council Advisory Committee on the Biological Effects of Ionising Radiation (BEIR Committee) deals with the scientific basis for the establishment of radiation protection standards. . .".

There are other examples. The BEIR II Committee (p.7, 1977) stated: "The goals of this report are to . . . provide a basis for more informed governmental decision-making and public participation in the issues" and on p.9: "It is hoped that this report may assist regulatory agencies in carrying out missions in the environmental protection field" and the task of BEIR II (p.16, 1977) was: "Evaluate associated factors of benefits and risks in ways that could be used in the establishment of reasonable protection guides." Here is the summary statement for chapter V (p.75, BEIR II, 1977): "The final sections of the chapter include discussions of basic legal and institutional considerations arising from uses of benefit-cost analysis in regulation and conclusions applicable to the formulation of national policy and federal programs for toxic and carcinogenic pollutants, including but not limited to, ionising radiation." The BEIR II conclusion begins (p.112, 1977): "This review of benefit-cost has been limited to its use in regulatory context . . . "

Finally, on pp.9 and 10 of BEIR III (1980) is this statement. In 1970, the president of the National Academy of Sciences named the first committee: the Advisory Committee on the Biological Effects of Ionising Radiation; this is what the Committee called itself.