

Debunking the myth: an examination of intergenerational radiation effects and low-dose radiation effects in A-bomb survivors
By Shari Yasin, McMaster University

In a society where war veterans are honored and tribute is paid to survivors of all kinds, most individuals would be surprised to discover that for many years this was not the case in Japan. Far from being treated with dignity and respect, atomic bomb (A-bomb) survivors were highly stigmatized in Japanese society and so many concealed their identities as survivors for years. For these *hibakusha* as they were often called, the psychological suffering after the bomb far outweighed any physical suffering endured. They were often prevented from marrying into families for fear that their children would have grotesque malformations or would die prematurely. In fact, some Japanese parents went as far as hiring private investigators to determine if their future in-laws were survivors (Gup 1995). Survivors were also discriminated against occupationally since companies were reluctant to hire or promote individuals who had been injured or who they believed were in poor health and at risk for diseases as a result of radiation exposure (Gup 1995). These strongly held misperceptions caused great pain and sadness as exemplified in the comments of one survivor Yoshiki Yamauchi, “I wanted to get married when I was 25 or 30...[but] what woman would have me?” (Gup 1995). Similarly, in a recent talk at the Hiroshima Youth Hostel, another survivor Hiroto Kuboura, recounted with sadness holding the same low level position at a railway power station for twenty years without ever being promoted.

Over sixty years later the psychological effects of the bomb are still present as many survivors head into their disease prone years. The prospect of death is forefront on the minds of those who believe that their lives have been compromised by radiation

exposure. As a result, many survivors spend their days worrying over whether they could be next to die. This latter belief has been fueled partly by concerns that even small amounts of radiation may be harmful—the basic tenet of the widely accepted “linear no-threshold model.” At the end of the war, a number of studies were initiated by the Atomic Bomb Casualty Commission and Radiation Effects Research Foundation to determine if radiation had in fact had a detrimental impact on the longevity of survivors and the health of their children. The results of these and other independent studies have important implications for public beliefs about radiation effects as well as implications for radiation protection standards throughout the world.

This essay is divided into four sections. The first section will briefly discuss ionizing radiation, the atomic bomb events and known effects of ionizing radiation. The second section will provide background information on the Atomic Bomb Casualty Commission and Radiation Effects Research Foundation. The third section will discuss in detail some of the studies that have been done on intergenerational radiation effects and mortality among A-bomb survivors. In the final section, the implications of these study findings will be explored, followed by some final thoughts.

Ionizing radiation consists of electromagnetic waves (x-rays) and subatomic particles which have sufficient energy to displace electrons or protons from atoms in the material it collides with (http://www.ccohs.ca/oshanswers/phys_agents/ionizing.html). Electrons or protons that are lost and gained result in charged (“ionized”) atoms. There are several different types of subatomic radiation particles including gamma rays, beta particles, alpha particles and neutrons. Gamma rays and neutrons are most relevant to a discussion of atomic bomb radiation. Gamma rays are highly penetrating particles that

indirectly cause damage to DNA via ionization of other molecules (i.e. water), which then produce free radicals that disrupt DNA and other cellular processes. Neutrons are also highly penetrating, but instead of displacing electrons they interact with atomic nuclei, ejecting protons or alpha particles (helium nuclei). Neutrons may directly or indirectly affect DNA. Unless repaired, damage caused to DNA by these ionization events will result in mutation, altered cell function or cell death.

On August 6, 1945 at 8:16 a.m. the first atomic bomb used on a human population was dropped over Hiroshima (Hiroshima Peace Memorial Museum 2004). Initial and residual radiation released accounted for 5 percent and 10 percent respectively of the bomb's total energy (the blast and heat rays accounted for the rest of the energy) (Hiroshima Peace Memorial Museum 2004). All four radiation particles previously mentioned were emitted, but gamma rays and neutrons were thought to have caused most of the damage since alpha and beta particles would have likely been absorbed in the air. Little radiation appeared to have reached beyond 2.5 km from the hypocenter. In general, the closer a person was to the hypocenter the greater the radiation dose received, although individual doses varied according to differences in shielding from buildings and hills (See Table I):

Table I. Radiation dose according to distance from hypocenter in Hiroshima (Hiroshima Peace Memorial Museum Booklet 2004)

Initial Radiation Dose (unit: gray (Gy))

Distance from Hypocenter	100m	500m	1000m	1500m	2000m	2500m
Gamma rays (r)	117.0	35.0	3.93	0.487	0.071	0.012
Neutron rays	33.1	6.04	0.227	0.008	0.000	0.000

It is estimated that the radiation dose decreased about one-half for every 200m from the hypocenter and about half of the radiation dose that would have normally been received,

was absorbed by houses that some survivors were in at the time of the bombing (Radiation Effects Research Foundation 2005). The majority of residual radiation came from radioactive fallout (radioactive soot and dust), which fell primarily northwest of the hypocenter. Maximum doses received from this fallout are estimated at 0.006-0.02 Sv (Radiation Effects Research Foundation 2005). A much smaller proportion of residual radiation came from neutron activation of soil and building materials. By 1945, the number of deaths caused by the bombing was estimated at 140 000, with 20 percent of these deaths having been attributed to radiation (Hiroshima Peace Memorial Museum 2004).

On August, 9, 1945 at 11:02 a.m. a second atomic bomb devastated the city of Nagasaki. The radiation energy released traveled a maximum distance of 3 km from the hypocenter. As in Hiroshima, the severity of dose was dependent on one's distance from the hypocenter (See Figure I). It is estimated that doses received from radioactive fallout

Figure 4. Distance from the hypocenter and air dose (without shielding) according to DS02 (Dosimetry System 2002*)

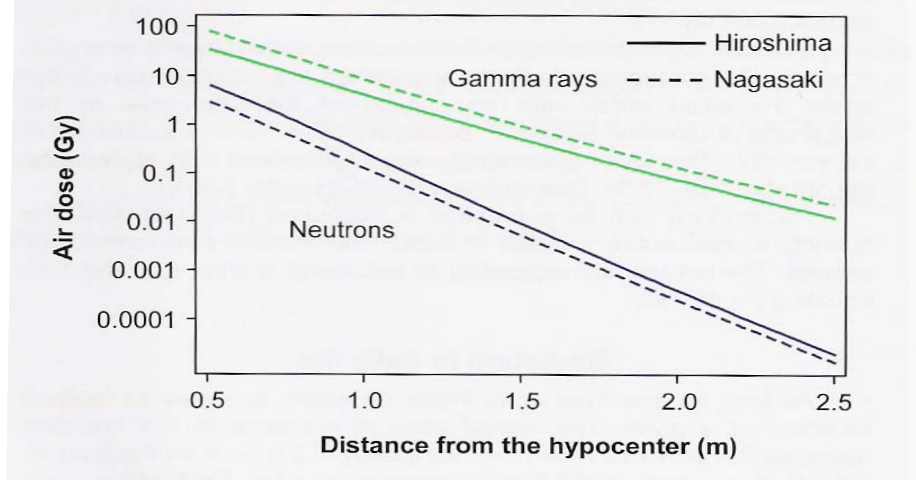


Figure I. Air doses received according to distance from the hypocenter (Introduction to the RERF Booklet 2005)

were 0.12-0.24 Sv. The estimated number of deaths from radiation-related effects and physical injury was 73 884 with almost 80 000 additionally wounded (<http://www1.city.nagasaki.nagasaki.jp/na-bomb/museum/m2-1e.html>).

Some of the known effects of ionizing radiation on the human body are summarized in the table below:

Table II. Effect of Radiation on the Human Body (Hiroshima Peace Memorial Museum Booklet 2004)

Effect of Radiation on Human Body (unit: gray (Gy))	
100	: Unconsciousness or coma. Death within several hours.
10	: Destruction of bone marrow, severe radiation sickness and reduced white blood cells and platelets. Death within 30 days.
1	: Nausea and vomiting. Reduced cell formation in bone marrow, temporary reduction of white blood cells.
0.1	: Changes appear in lymphocytes produced by bone marrow.
0.01	: No apparent symptoms.

As a basis for comparison, annual exposure from natural sources of radiation is typically 2 mSv (equivalent to 0.002 Gy for gamma rays and 0.02-0.04 Gy for neutrons) (Radiation Effects Research Foundation 2005). Acute effects of radiation exposure are generally seen above 0.1 Gy. Changes in blood cells (especially lymphocytes) are seen in dose ranges of 0.1 to 1 Gy. Above 1 Gy symptoms such as nausea, vomiting and decreased numbers of blood cells are experienced. Above 10 Gy individuals experience severe radiation sickness, destruction of blood cells and death occurs within 30 days. At 100 Gy or more, death occurs in a matter of hours. The effects of much lower doses of radiation are less well characterized and have attracted a great deal of attention from the scientific community in recent years. Although no clear picture of low-dose effects exists as yet, many studies are currently in progress to help elucidate such effects. Many of these studies have provided important insights, some of which challenge common assumptions about low-dose radiation exposure.

Two organizations, the Atomic Bomb Casualty Commission (ABCC) and Radiation Effects Research Foundation (RERF) have been systematically collecting data and conducting studies on A-bomb survivors and their children since the late 1940s. The ABCC, the predecessor organization to the RERF, was established in Hiroshima in 1947 and in Nagasaki a year later following the recommendation of a U.S. army-navy task force that a long-range study of the medical and biological effects of the atomic bomb be done (“Genetic Effects” 1991). In 1948, a cooperative research effort between the ABCC and Japanese National Institute of Health was initiated and this partnership continued until 1975 when the RERF was established (Hiroshima Peace Memorial Museum 2004). The RERF, a nonprofit Japanese foundation, is funded and managed equally by the Japanese and U.S. governments. The foundation aims not only to understand how radiation exposure affected the long-term health of A-bomb survivors, but also to use this knowledge in guiding health care programs for survivors and protecting other individuals (the world relies heavily on data from the RERF to set radiation safety standards). The RERF studies differ from other studies of populations exposed to radiation (i.e. Chernobyl accident survivors, World War I radium dial workers), in that only the RERF studies have followed a large population of over 100 000 survivors for more than fifty years. Therefore, these studies provide the most comprehensive information to date on the late effects of radiation in humans. A few of the major research projects undertaken by the ABCC/RERF are the Life Span Study (≈120 000 A-bomb survivors followed), Adult Health Study (≈20 000 survivors followed), Offspring/F1 studies (≈77 000 children followed) and in-utero exposure studies (≈3600 children followed) (Kodama 2005). A

number of independent investigations have also been done by other researchers, but most of these rely on data collected by the ABCC and RERF.

The first major study of children born to survivors (F1 generation), began in 1947 and was a study of the relationship between parental radiation dose and “untoward pregnancy outcome,” defined as a child who had a major congenital defect, who was stillborn or who died in the first fourteen days of life (Otake *et al.* 1991). Over 76 000 pregnancies were followed until 1954 using a registry system where pregnant women recorded their pregnancies with the ABCC at the same time they registered with the government for extra food rations (Schull 2003). Pregnancy outcomes were reported to the ABCC by midwives and physicians. The most common birth defects recorded following the bombings were anencephaly (brain and skull abnormality), harelip, cleft palate, club foot, polydactyly (abnormal number of digits) and syndactyly (fusion of digits) (Schull 2003). Although a positive association between joint parental dose and pregnancy outcome was demonstrated, no *significant* increase in the frequency of untoward pregnancy outcomes was found. Both the original study and a 1991 reanalysis of the data failed to find a significant radiation effect, even when each endpoint (malformation, still birth and neonatal death) was analyzed separately with parental radiation dose. Furthermore, in the 1991 reanalysis, several dose-response models were applied to the data to determine if there was a stronger suggestion of a radiation effect, but none was found (See Table III, next page).

Biochemical and developmental studies on the children of survivors have further validated the above findings. 1 256 000 biochemical tests have been done, scoring for enzyme mutations and lowered enzyme activity in the blood cells of children born to

Table III. Relationship of joint parental exposure to untoward pregnancy outcomes (Otake 1991)

TABLE X
Increments or Decrements of Change in the Individual Frequencies of Congenital Malformation, Stillbirths, and Neonatal Deaths in the Original Cohort of Births per Sievert of Joint Parental Gonadal Dose Equivalent Based Upon an Assumed Neutron RBE of 20, Extended (DS86 + ad hoc) Cohort

Variable	Regression coefficient	Standard error
Regression model: $P_i = \text{Constant} + \sum_{j=1}^6 b_j x_{ij} (\text{Background}) + b_D \text{Dose}_i$		
Malformation		
Joint parental exposure	0.00101	0.00154
Birth order of child	0.00064*	0.00036
Year of birth	0.00131**	0.00028
Stillbirths		
Joint parental exposure	0.00092	0.00163
Birth order of child	-0.00059	0.00038
Year of birth	-0.00028	0.00032
Neonatal deaths		
Joint parental exposure	0.00128	0.00185
Birth order of child	0.00026	0.00043
Year of birth	0.00068*	0.00037
Regression model: $P_i = 1 - \exp - (\text{Constant} + \sum_{j=1}^6 b_j x_{ij} (\text{Background}) + b_D \text{Dose}_i)$		
Malformation		
Joint parental exposure	0.00120	0.00194
Birth order of child	0.00064	0.00047
Year of birth	0.00130**	0.00038
Stillbirths		
Joint parental exposure	0.00091	0.00200
Birth order of child	-0.00075	0.00048
Year of birth	-0.00029	0.00040
Neonatal deaths		
Joint parental exposure	0.00128	0.00218
Birth order of child	0.00047	0.00052
Year of birth	0.00074*	0.00044

Significance levels: *($P < 0.10$), **($P < 0.01$).

survivors (Schull 2003). Of these tests, only seven mutants have been identified in the F1 generation (Schull 2003). Assessments of the growth and development of the F1 generation were also made using measurements of height, weight and chest circumference taken shortly after birth and then annually in the public school system

(Schull 2003). To date, no evidence of radiation-related delays on the growth and development of these children has surfaced.

A number of studies by the ABCC and RERF have also looked at the frequency of chromosomal abnormalities in the offspring of survivors. Initial studies between 1962 and 1966 examined the frequency of autosomal aneuploidy (Down Syndrome) and sex chromosomal aneuploidy (Turner Syndrome and Klinefelter Syndrome) in this generation. The premise here was that increasing doses of ionizing radiation would have increased the frequency of nondisjunction (failure of the chromosomes to separate during meiosis), resulting in gametes with more or less than the normal number of chromosomes. None of these studies found evidence of a relationship between parental radiation and these disorders. A more systematic study was initiated in 1967 which examined over 16 000 children born to radiation exposed and unexposed parents for sex chromosomal abnormalities and autosomal structural rearrangements (Schull 2003). Despite some limitations in obtaining data, the investigators determined that children of exposed parents did not exhibit significant increases in the frequency of these chromosomal abnormalities when compared to the control group (i.e. children of unexposed parents).

Even more convincing evidence for a nonsignificant effect of parental radiation has come from DNA studies. Several of these studies have compared the mutation rates at several DNA loci in children born to survivors with rates in children born to unexposed parents. These DNA loci represent highly mutable regions of tandem repeat sequences, termed “minisatellites” (Kodaira *et al.* 2004). In a recent RERF study by Kodaira *et al.* (2004), mutations were determined by a Southern blot analysis that compared offspring

DNA bands to parental DNA bands for changes in size. Consistent with the results of the initial 1995 and 1996 RERF studies, no significant genetic effect of parental radiation was found in the F1 generation (See Table IV). However, there have been mixed findings with studies that have examined the children of Chernobyl cleanup workers; therefore, further studies are needed to confirm the present results.

Table IV. Comparison of mutations at eight loci in offspring of exposed and unexposed parents (Kodaira, 2004)

TABLE 2
Mutations at Eight Hypermutable Loci

Locus	Exposed group			Control group			Difference (95% CI)
	No. of mutations	No. of alleles tested	Mutation rate (%)	No. of mutations	No. of alleles tested	Mutation rate (%)	
Mutation in paternal alleles							
<i>CEB15</i>	0	30	0	5	89	5.6	
<i>CEB25</i>	1	30	3.3	4	89	4.5	
<i>CEB36</i>	0	30	0	1	89	1.1	
<i>MS31</i>	0	30	0	0	89	0	
<i>MS32</i>	2	30	6.7	1	87	1.2	
<i>B6.7</i>	3	30	10.0	4	89	4.5	
<i>CEB1^a</i>	4	30	13.3	10	88	11.4	
<i>MS1^a</i>	1	30	3.3	8	89	9	
Total	11	240	4.6	33	709	4.7	-0.07% (-2.89%, 3.36%)
Mutation in maternal alleles							
<i>CEB15</i>	0	32	0	1	87	1.1	
<i>CEB25</i>	0	32	0	1	87	1.1	
<i>CEB36</i>	1	32	3.1	1	87	1.1	
<i>MS31</i>	0	32	0	0	87	0	
<i>MS32</i>	1	32	3.1	0	87	0	
<i>B6.7</i>	0	32	0	0	85	0	
<i>CEB1^a</i>	0	32	0	0	87	0	
<i>MS1^a</i>	0	32	0	3	87	3.4	
Total	2	256	0.8	6	694	0.9	-0.08% (-1.36%, 1.62%)

^a Results from our previous study (ref. 8).

Finally, a number of mortality studies have examined the lifespan of the F1 generation (beyond the neonatal period). The first study followed a group of children born to survivors between 1946 and 1958 for nine years (Neel *et al.* 1974). There were three cohorts of children grouped according to the following parental radiation exposures a) one or both parents within 2000m of the hypocenter at the time of bombing b) one or both parents 2500m or more from the hypocenter c) neither parent in the city during the bombing (Neel *et al.* 1974). This study was extended for an additional eight years after publication of the initial results. Together, the original cohorts and extension constitute a study population of 76 817 children, and among these individuals, parental doses could

be estimated directly for 67 586 cases (Schull 2003). When only these latter cases are considered, the frequency of non-cancer deaths does increase slightly with joint parental dose, but not significantly so (Schull 2003). Regarding those deaths attributed to cancer, “no clear trend in the occurrence of either leukemia or other cancers before the age of 20, or after, with increasing parental dose exists as yet” (Schull 2003).

A number of mortality studies have also been done on A-bomb survivors. Of increasing interest is the effect of low doses of radiation (≈ 0.1 -150mSv) on the longevity of these individuals. Results of such studies examining low-dose effects have been mixed, but of importance here is the finding of several investigators that deaths among survivor cohorts from non-cancerous diseases have not differed significantly from matched cohorts of unexposed individuals. For example, several studies conducted between 1950 and 1978 by Jablon *et al.* (1965), Beebe *et al.* (1978) and Kato *et al.* (1982) all concluded that there had been no harmful effects of radiation exposure other than cancers in this population (In: Mine *et al.* 1990). In fact, the death rates of survivors in these studies appeared to be lower than those of unexposed control groups, although the authors did not comment on these findings (Mine *et al.* 1990). Complicating matters further, a 1994 UNSCEAR report announced that no increase in total *cancer* deaths had been found in epidemiological studies of survivors who received doses less than 200 mSv. Similar analyses of A-bomb survivor data by Heidenreich *et al.* (1997), Shimizu *et al.* (1990) and Land *et al.* (1993), have found no evidence of significant cancer risk for radiation exposures below 200 mSv (In: Mossman 1998). Additionally, Ron *et al.* (1995) identified a threshold dose of 100mSv (i.e. lowest associated dose) for childhood thyroid cancer based on the pooled results of several studies (In: Mossman 1998).

In other studies, a *beneficial* effect of low dose radiation (termed “hormesis”) has actually been observed, with some survivors outliving their unexposed counterparts. For example, Okumura and Mine (1997), followed over 3000 A-bomb survivors who received doses ranging from 1 to 599 cGy and a comparative control group who received no dose. When death rates between exposed and unexposed cohorts were compared, the death rate of A-bomb survivors was found to be lower than that of the unexposed group, although it is unclear whether these results were significant. Furthermore, a statistically significant reduction in the relative risk of death from all causes was observed for males at doses of 21-40 cGy and no significant increase in mortality risk was observed for either sex at any dose. In a 1984 study by Stewart and Kneale, the observed number of non-cancer deaths among survivors who received low to mid doses of radiation was significantly lower than the expected number of deaths (this effect was pronounced for survivors born prior to 1963). These results suggested a U-shaped rather than linear dose-response curve for non-cancer deaths; that is, survivors who received low to mid doses of radiation appeared to live significantly longer than unexposed individuals or those who received to higher doses (See Figure II). It should be noted that these

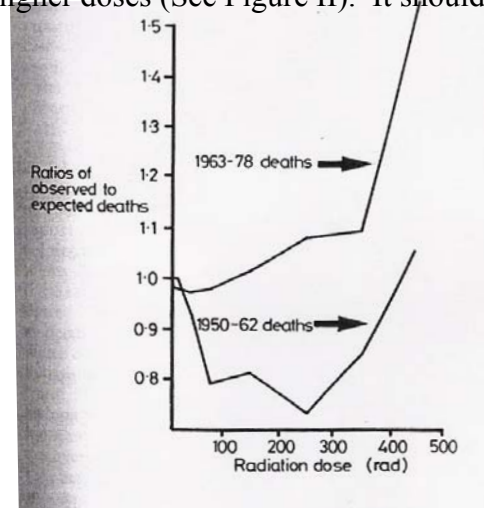


Figure II. U-shaped dose-response curve: Non-cancer deaths of A-bomb survivors excluding cardiovascular death and trauma (Stewart & Kneale 1984)

findings are highly controversial, with other investigators having shown an opposite, negative effect of low-dose radiation on survivor longevity.

The above studies demonstrate that at the phenotypic, molecular and genetic levels, there was no significant intergenerational radiation effect and further, that the lifespan of the F1 generation does not appear to be shortened. Importantly, these findings help to dispel some of the erroneous beliefs about radiation effects that were held by many Japanese people and that are even held by some individuals today. Contrary to popular belief, there was no epidemic of misshapen babies born to survivors; in fact, no significant effects, even at a cellular level, were found when comparisons were made with children whose parents had received negligible doses. Therefore, the stigmatization of A-bomb survivors was unwarranted and can be partly attributed to the lack of available information on radiation effects at the time. It should be noted that because no significant effect of parental dose was demonstrated in these studies does not mean that there wasn't one. It may simply be that such effects have not yet manifested or are undetectable with current research methods, so further studies are needed to confirm these results. Nonetheless, the accumulating evidence continues to show no effect of parental exposure in the F1 generation.

The mortality studies call into question whether low-level radiation could be less harmful than commonly believed. This has important implications not only for many A-bomb survivors whose concerns over premature death are a continual source of anxiety, but also for radiation protection policies throughout the world. Government standards of radiological protection are based on the recommendations of organizations like the International Commission of Radiological Protection (ICRP), United Nations Scientific

Committee on the Effects of Atomic Radiation (UNSCEAR), International Atomic Energy Agency (IAEA) and National Council on Radiation Protection (NCRP) among others. Underlying the recommendations of all of these organizations is the acceptance of a powerful theorem, the “linear no threshold (LNT) model”. This model assumes that there is no “safe” dose of radiation and that there is a linear dose-response relationship even at very small doses with the frequency of stochastic effects at low doses being proportionate to those at higher doses (<http://www.epa.gov/radiation/terms/termjklm.htm#l>). However, a growing body of evidence suggesting both null effects and beneficial effects of low-dose radiation is incompatible with this model. Again, such findings do not mean that low-dose radiation did not have a negative health impact (a number of investigators have indeed found an increased frequency of cancer deaths for all doses); nonetheless, it is clear that the LNT model is not the whole story on low-dose effects. Forty percent of survivors are still living today and until all mortality data has been collected on these individuals, the findings of previously mentioned studies should not be blindly ignored.

Both the F1 generation studies and survivor mortality studies underscore a need for reassessment of the LNT model and its appropriateness as a guide to policy-making. As scientists, we have been taught to look at what the data tells us. For stochastic effects such as radiation-induced mutations in offspring, the data is clear: no significant genetic effects have been observed in the children of survivors. For other stochastic effects such as cancer induction and radiation-related deaths (i.e. cancer and non-cancer deaths), the findings are mixed with both negative and null dose-response relationships having been demonstrated at low-doses. In light of what the data says, it is troubling that regulatory

organizations like the ICRP continue to cling to the LNT hypothesis. The ICRP has conceded that some of the assumptions of the LNT model may be incorrect, but it is reluctant to do away with the model since a better alternative that will not lead to an underestimation of risk has not presented itself (Mossman 1998). In the meantime, the Health Physics Society has criticized the ICRP for its overly conservative regulations which are based largely on “empirically unproven assumptions” (Mossman 1998). Billions of dollars are spent in North America each year enforcing stringent radiation standards. A U.S. study by Hahn and Hird (In: Mossman 1998), found that regulations designed to minimize occupational and health risks totaled \$9 billion in 1988 with only negligible benefits (See Table V). According to author Joby Warrick (1997), “if the

Table V. Cost/benefit analysis of dollars spent on radiation protection measures (Mossman 1988)

TABLE I. Costs and benefits of regulation.^a

Regulations	Costs (1988 dollars)	Benefits (1988 dollars)
Environment	\$55.4–77.6 billion	\$16.5–135.8 billion
Occupational safety and health	\$8.5–9.0 billion	negligible
Nuclear power	\$5.3–7.6 billion	not available

^aSource: Hahn and Hird (1991), Ref. 8.

government relaxed radiation exposure standards, by even a small degree, it could result in enormous savings for utilities, hospitals and other businesses that use radioactive materials. Taxpayers could save billions of dollars if cleanup standards were eased for the dozens of lightly contaminated sites across around the country.”

Based on the emerging data from epidemiological populations of survivors and their children, it is clear that an alternative to the increasingly outdated LNT model is needed. As previously discussed, the assumptions inherent in this model have had many

negative consequences including wasted money and resources, fostering public anxiety and fear (which does more harm than good in an age of nuclear power) and most critically, imposing unnecessary mental anguish on persons exposed to radiation. Until a suitable alternative can be found, it is up to radiation policy makers to act responsibly; specifically, to consider all of the available data when setting radiation standards so that these negative consequences may be avoided in the future.

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