2.1 Acute effects of A-bomb radiation

2.1.1 Records of acute effects

The atomic bombings of Hiroshima and Nagasaki on 6 and 9 August 1945 marked the first and only times that atomic bombs were dropped in warfare. Before proceeding I would like to take this opportunity to express my deepest condolences to the families of the 140,000 people in Hiroshima and 70,000 people in Nagasaki who lost their lives. I believe that Japanese experts such as myself have a duty to communicate the human effects of the atomic bombings to the world in an accurate and scientific manner if only to advocate the abolition of all nuclear weapons. I encourage readers to visit the Hiroshima Peace Memorial Museum and the Nagasaki Atomic Bomb Museum to learn more about the sheer devastation and pandemonium that occurred after the bombings. These museums are actively engaged in collecting related documents and artifacts, hosting and recording lectures by survivors and fostering speakers among future generations in order to ensure that the memory of those fateful events does not fade with the passage of time. Visiting “ground zero” is another valuable way to understand what occurred after the explosions.

I would like to begin this chapter by introducing some of the remaining records detailing the human health impact of the atomic bombings. Among the radiation exposure surveys commenced soon after detonation of the A-bomb over Hiroshima were the “Kure Naval District Command Headquarters Hiroshima Air Raid Damage Report” and the “Army Ship Training Division Report on the Aerial Bombardment of Hiroshima”. Several survey teams were dispatched by the Imperial Headquarters to investigate the bombings including the Army Department’s “Hiroshima Disaster Survey Group” and the “Imperial
Headquarters Survey Group Joint Meeting”, and Dr. Yoshio Nishina from the Institute of Physical and Chemical Research (Riken) also performed radiation measurements\(^1\). However, immediately after the end of the war, the General Headquarters (GHQ) of the Supreme Commander for the Allied Powers (SCAP) classified all matters relating to the atomic bombings as a military secret and prohibited any research or studies by the Japanese.

Despite this, the U.S. military actively undertook a number studies, some of which also involved Japanese experts. The findings of the A-bomb survivor studies were eventually compiled into an immense, detailed six-volume report released by the Army Institute of Pathology in 1951 entitled “Medical Effects of Atomic Bombs: The Report of the Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan”\(^2\). This document is a comprehensive scientific record of the acute effects of the atomic bombs, and can be viewed in its entirety at the library of the Radiation Effects Research Foundation (RERF) or the Nagasaki Atomic Bomb Museum. Alternatively volumes 1 to 6 can be downloaded free of charge over the internet\(^2\).

The Japanese were not permitted to release the findings of independent studies conducted during the occupation but did undertake joint studies with the U.S. military. Following the signing of the Treaty of San Francisco in 1951, a summary of the Joint Commission Report was published in Japanese by the Japan Society for the Promotion of Science for the Science Council of Japan\(^3\). The first and second volumes of the report were subsequently published in Japanese in 1953\(^4\). I would like to take this opportunity to pay homage to the remarkable enthusiasm and dedication of the Japanese scientists and researchers who compiled this record of A-bomb effects. A layman’s version of this record was also prepared by the “Editing Committee on Atomic Bomb Damage in Hiroshima and Nagasaki” and released by Iwanami Shoten, Publishers\(^5\).

Other records on the bombs’ acute effects have also been published primarily by the Hiroshima Prefectural Medical Association (HPMA) in Hiroshima Prefecture and Nagasaki University in Nagasaki Prefecture. Nagasaki University in particular is the world’s only such institution to have been exposed to radiation from an atomic bomb—the main campus was located just a few hundred meters (m) from ground zero and all of the professors and students attending lectures at the time of the bombing perished. On the 50\(^{th}\) anniversary of the bombings, a collection of eyewitness accounts was published in which doctors at the University’s School of Medicine recounted their observations of dying colleagues and students despite themselves suffering the effects of the
2.1.2 Mortality from acute radiation effects—Bombing- and radiation-induced fatalities

As I mentioned in the previous chapter, the atomic bombings had claimed the lives of 140,000 people in Hiroshima and 70,000 people in Nagasaki by the end of 1945. In examining the causes of death we must first understand the nature of the bombs themselves. However, Japan’s lack of practical experience in constructing an A-bomb and the taboo previously surrounding discussion of the matter means that we must look to the U.S. to obtain this information.

After the end of World War II, the U.S. conducted an experiment on the effects of the atomic bombings by detonating nuclear devices over mock wooden houses built in the Nevada desert and examining the damage. The results of these tests were ultimately reported in a book by Glasstone and Dolan entitled “The Effects of Nuclear Weapons.” Strong demand for this book saw it republished three times, with the 653-page third edition released in 1977. A brief glance at the book’s table of contents shows that it is divided into the following key topics: air blast; thermal radiation; initial nuclear radiation; residual nuclear radiation; and fallout. This book is mainly concerned with the effects of radiation so I will provide more details later in the chapter but one intriguing statement worth noting here is that only 15% of the explosive energy of a nuclear fission weapon is released as various nuclear radiations whereas 35% is in the form of thermal radiation and 50% is air blast and shock. Thus, it is important to bear in mind that the damage caused by an A-bomb does not derive solely from its nuclear radiation but also from the impact of its air blast and the heat of its thermal radiation. Put simply, one of the most frightening aspects of an A-bomb is the extraordinary scale of its explosion.

Judging from the extent of the calamity that ensued, even an understanding of the energy released by the A-bombs would not allow us to accurately determine how many of the 210,000 victims (30% of the total residents of both cities) were killed by the air blast, how many were killed by the thermal radiation and how many were killed by the nuclear radiation. However, the 1986 Chernobyl disaster victims who died from acute radiation sickness and the two workers who died in the 1999 Tokaimura criticality accident did not perish on the day of the respective incidents despite exposure to lethal doses, suggesting that those who died on the day of the respective Hiroshima and Nagasaki atomic
bombings were killed not by nuclear radiation but by the effects of the massive explosion such as the air blast or thermal radiation. Evidence indicates that the subsequent mortality rate among those who survived for the first few days was just a few percent rather than 30%. Furthermore, around half of the A-bomb victims exposed to a radiation dose of 3 Sv reportedly died.

It should also be noted that in the chaos immediately after the bombings, appalling conditions prevailed not only in terms of the medicine supply but also accommodation and food provisions.

2.1.3 Clinical symptoms of acute effects

The aforementioned Joint Commission Report contains autopsy findings and other detailed records on the clinical symptoms of A-bomb victims with acute effects. The doctors at Nagasaki University also kept meticulous medical records of their colleagues up until death.

Typical symptoms prior to death were nausea and vomiting in the initial stages followed by gastrointestinal symptoms such as diarrhea and melena, and skin symptoms including flushing, blisters and desquamation. In serious cases, victims initially presented with impaired consciousness followed by leukopenia-induced secondary infection resulting from bone marrow disorder, and ultimately death due to organ failure. However, individual clinical symptoms were varied, ranging from fatal cases of severe melena and impaired consciousness to recovery.

2.2 Late effects of A-bomb radiation

2.2.1 Characteristics & survey methods

I will now explain the methods for investigating cancers and other late health effects based on the presumption that the cause of radiation exposure in the above-mentioned individuals is indeterminate. Both epidemiological and statistical methods are employed when studying late effects. For example, if we followed up a large cohort of individuals exposed to A-bomb radiation and a large cohort of unexposed individuals until mortality and discovered a statistically significant incidence of fatal disease in the exposed cohort, we could surmise that the probability of disease had increased due to the effects of radiation. Alternatively, if we estimated the exposure dose of our cohorts
and discovered a higher incidence of fatal disease (or simply morbidity as many cancer cases are non-fatal) in people with a higher dose of radiation, then we could infer that radiation increases the risk of contracting the disease. In practice, the latter of these two methods is generally adopted.

Moreover, even if we were to deduce the increased probability of a certain disease, we could not distinguish which cases were due to radiation and which had developed spontaneously.

Late effects studies hinge on an understanding that no matter how thoroughly we examine a cancer patient, it is impossible to discriminate between cases induced by radiation and those attributable to other causes.

For these reasons, the following conditions are essential when undertaking a scientific study of late health effects: (1) selecting an appropriate cohort; (2) accurately estimating radiation exposure of the subjects; (3) conducting long-term health surveillance (to identify current diseases and cause of death); and (4) using epidemiological and statistical techniques to demonstrate the relationship between exposure dose and disease incidence.

Much time has passed since the Atomic Bomb Casualty Commission (ABCC; currently the RERF) was established during the occupation of Japan but it is still irrefutably the largest repository of scientific study findings on the atomic bombings in Japan and indeed the world. Below I will explain the concept of late effects with reference to the “Brief Description” provided on RERF’s website.

2.2.2 Study populations

In the epidemiological and statistical surveys forming the basis of the RERF late effects studies, the selection of a valid study population proved crucial. In 1950, five years after the atomic bombs were dropped on Japan, a survey of A-bomb survivors was carried out in conjunction with the first post-war national census. The survey identified some 284,000 Japanese A-bomb survivors who admitted to being exposed to radiation. Of these respondents, about 195,000 residents of Hiroshima and Nagasaki were selected as the master sample from which various study cohorts were subsequently drawn to suit ABCC/RERF’s study objectives (Figure 2.1).

Study cohort respondents who lived within 2,500 m of the “hypocenter” (i.e., the location on the ground directly beneath the atomic explosion) and those who lived 2,500 m or more from the hypocenter were estimated to comprise
approximately 50% and 25% of the 284,000 exposed survey respondents respectively. However, distance from the hypocenter was not included in the census so the exact numbers of people residing at these ranges are unclear.

**a. Life Span Study cohort (120,000 people)**
The “Life Span Study” (LSS) cohort constituted the core of master sample. It contained all eligible A-bomb survivors from the master sample who were legally domiciled in Hiroshima or Nagasaki within 2,000 m of the hypocenter (group 1; “proximally-exposed survivors”), all eligible persons domiciled from 2,000 to 2,500 m from the hypocenter (group 2), eligible survivors domiciled from 2,500 to 10,000 m from the hypocenter who were matched with the group 1 subjects in terms of age and sex (group 3; “distally-exposed survivors”) and persons age- and sex-matched to group 1 who were not residing in either city at the time of the bombings. In the 1960s, the LSS cohort was expanded to include all master sample survivors exposed within 2,500 m of the hypocenter regardless of domicile.

**b. Adult Health Study cohort (23,000 people)**
The “Adult Health Study” (AHS) cohort was established to collect information on disease incidence and health status through health exams conducted once every two years. The AHS cohort consisted of approximately 5,000 A-bomb survivors who were within 2,000 m of the hypocenter and who had signs and symptoms of acute radiation syndrome. The cohort also included three city-, age-, and sex-matched groups drawn from the LSS, each similar in size to the core group. The three groups were: (1) survivors without acute radiation
syndrome who were within 2,000 m of the hypocenters; (2) survivors who were at least 3,000 m from the hypocenters; and (3) persons not in either city at the time of the bombings.

c. In Utero Study cohort (3,600 people)
The “In Utero Study” targeted a fixed cohort assembled mainly from records of around 10,000 births occurring in or near Hiroshima and Nagasaki between the time of the bombings and the end of May 1946.

d. Children of Survivors Study cohort (77,000 people)
The “Children of Survivors Study” featured a fixed cohort based on data from a survey of birth defects and premature deaths among approximately 77,000 births up to the mid-1950s. The cohort consisted of all eligible children with at least one parent within 2,000 m of either hypocenter at the time of the bombings (core group) and two sex- and age-matched comparator groups of the same size as the core group. The first comparator group included children with at least one parent exposed between 2,500 and 10,000 m of the hypocenter of either explosion, and the second group included children with neither parent within 10,000 m of either of the hypocenters.

A summary of the above-mentioned study cohorts is provided in Table 2.1. These cohorts were all established with the aim of obtaining study findings that would prove useful in future health effects research.

### Table 2.1 A-bomb survivor study populations

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Life Span Study (1950–)</td>
<td>120,000 people</td>
</tr>
<tr>
<td>2) Adult Health Study (1958–) (biennial health exam)</td>
<td>23,000 people</td>
</tr>
<tr>
<td>3) In Utero Study (1950–)</td>
<td>3,600 people</td>
</tr>
<tr>
<td>4) Children of Survivors Study (1946–)</td>
<td>77,000 people</td>
</tr>
</tbody>
</table>


#### 2.2.3 Estimation of exposure dose

a. Estimation of physical dose\(^{(1)}\)

Study cohorts were initially determined based on distance from the hypocenter but were later selected according to estimates of individual exposure dose. The current dosimetry system adopted in 2002 (“DS02”)\(^{(2)}\) provides individual dose estimates based on information about each survivor’s location and shielding conditions at the time of the bombings. DS02 is based on the physical nature of the bombs dropped on Hiroshima and Nagasaki and theoretical models developed by nuclear physicists on the amount of radiation released, how this
Radiation was transported through the air and how it was affected by passage through man-made structures and human tissue.

Radiation from an A-bomb obviously travels through the air before coming in contact with the human body. This radiation air dose is validated using calculations of the bomb’s physical properties and the prevailing air conditions as well as measurements of samples of exposed materials such as floor and roof tiles and copper. As a very rough estimate, a person located within 800 m of the hypocenter without any shielding would have been exposed to a dose of at least 10 Sv that would have likely proved fatal. At 2,000 m from the hypocenter the exposure dose would be around 0.1 Sv or less, and at 3,000 m away the dose would be about 0.002 Sv. As described below, in the ABCC/RERF studies our focus is on those subjects who were exposed to a radiation dose of at least 0.005 Sv.

Before it reaches the human body, radiation may be affected by man-made structures and other objects with a shielding effect. Information on the shielding conditions of individual subjects in the LSS cohort at the time of exposure is recorded in interviews conducted in the late 1940s and early 1950s. More detailed shielding histories were obtained in the early 1960s for around 85% of

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**Figure 2.2** Estimation of individual exposure dose using DS02 dosimetry system (2002–)

Kerma is an abbreviation for “kinetic energy released in material” and is used to indicate entrance skin exposure dose whereas organ dose is the absorbed dose adjusted for the radiation shielding effect of surrounding body tissue.
proximally-exposed LSS survivors (Figure 2.2).

Individual exposure dose is therefore estimated based on air dose, shielding conditions, and body position at the time of exposure. Under the DS02 dosimetry system, doses were estimated for 86,671 (92%) of the 93,741 people in the LSS cohort who were within 10,000 m of the hypocenter. However, doses could not be estimated for 7,070 proximally-exposed individuals due to the uncertain or complex nature of their shielding history. The DS02 system estimates gamma and neutron doses for each of the 15 major organs based on body position during exposure, and analysis of cancer in each site of the body is then performed using these organ doses.

b. Residual radiation

As I mentioned in subsection 2.1.2, the term “residual radiation” in common usage can be broadly classified into induced radioactivity and radioactive fallout. Induced radioactivity results from the interaction of neutrons (a small component of A-bomb radiation) with materials, causing them to become radioactive. Doses from induced radioactivity increase in direct proportion to proximity to the hypocenter.

Past investigations have suggested that the maximum cumulative dose from residual radiation at the hypocenter since the bombings is 0.8 Sv in Hiroshima and 0.3 to 0.4 Sv in Nagasaki. At 500 and 1,000 m from the hypocenters, the respective estimates fall rapidly to about 10% and 1% of the hypocenter value. The induced radioactivity decayed very quickly over time, with nearly 80% of the above-listed doses released within the first day, about 10% between days 2 and 5, and the remaining 10% from day 6 onwards. Considering the extensive fires near the hypocenters that prevented people from entering the cities until day 2, it seems unlikely that any person received more than 20% of the maximum possible induced doses (0.16 Sv in Hiroshima and 0.06 to 0.08 Sv in Nagasaki).

Radioactive fallout primarily came from radioactive atoms produced by nuclear fission of uranium or plutonium in the bombs. Radioactive material in the bomb fireball ascended and cooled before some of it fell back to earth as “black rain” that contaminated the ground. However, the prevailing wind direction caused much of this rain to fall in northwest Hiroshima, with the highest measured gamma dose rates from fallout occurring in the Koi–Takasu area to the southwest, and in the Nishiyama area of eastern Nagasaki. Assuming that a person remained in the same place for his/her entire life, the maximum estimates of fallout dose from external exposure to gamma rays are
0.01 to 0.03 Sv in the Koi–Takasu area of Hiroshima, and 0.2 to 0.4 Sv in the Nishiyama area of Nagasaki. The corresponding fallout doses at the hypocenters are believed to be only about 10% of these values. Doses due to internal accumulation of long-lived fallout radioisotopes present in the environment (i.e., due to dietary intake) were estimated from a sampling of Nishiyama residents based on whole body counting (WBC) and other measurements to determine individual body content of a key radioisotope cesium-137, and were found to be minimal\(^{15}\).

Now, more than 60 years after the atomic bombings, the radioactive fallout is difficult to detect and is virtually indistinguishable from fallout generated by atmospheric nuclear tests conducted around the world in the 1950s and 1960s.

2.2.4 Solid cancers\(^{16,17}\)

In the A-bomb survivor studies, solid cancers are globally regarded as the most reliable indicator of late radiation effects, and have been used to establish international scientific consensus. Epidemiologically speaking, the main points of contention in study findings are whether there is a threshold for radiation-induced health effects, and whether these effects are seen at doses of up to 100 mSv. Below is a relatively detailed description of late radiation effects constituting the most trusted external exposure findings of the LSS mortality and incidence studies.

Increased incidence of solid cancers is a typical late effect that appears 10 years or more after exposure and that persists among A-bomb survivors even today.

**a. Study methods & materials**

Medical interview results (including exposure profiles) were recorded for all 120,000 people in the LSS cohort. Deaths are routinely identified through Japan’s family registry system and death certificates can be obtained from national vital statistic death records. The integrity of the family registry means that the study follow-up rate is close to 100%. Needless to say, all relevant legal procedures were adhered to from the outset when collecting information on mortalities. I will now provide some concrete figures to demonstrate just how comprehensive the LSS is.

Table 2.2 shows the number of LSS cohort subjects in 1950 and the survival rate in 1998 when the results of the LSS mortality study were compiled, according to age at exposure. It is important to note that the LSS cohort in 1950
comprised all people in Hiroshima or Nagasaki at the time of the bombings for whom individual doses were estimated using the DS02 dosimetry system, and that it was designed to ensure that each age bracket contained roughly the same number of subjects. In 1998, all persons aged 50 years (y) or older at the time of exposure had deceased but more than 90% of those aged 9 y or younger at the time of the bombings were still alive, meaning that approximately half of the original cohort subjects were still living.

Table 2.3 shows the number of deaths in the LSS cohort due to solid cancers. Unlike Table 2.2, subjects are categorized according to exposure dose (Sv) rather than age, and the second column indicates the number of cancer deaths that occurred between 1950 and 1997. The data demonstrates just how accurate the follow-up surveillance has been. In the LSS mortality study, all 3,833 of the 37,458 A-bomb survivors exposed to a radiation dose of less than 0.005 Sv (5 mSv) are believed to have died due to spontaneous (i.e., “naturally-occurring”) cancers. These spontaneous cancer deaths can also be seen in

Table 2.2 LSS (mortality study)

<table>
<thead>
<tr>
<th>Exposure age (y)</th>
<th>1950 subjects(^a)</th>
<th>1998 survivor ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–9</td>
<td>17,824</td>
<td>91%</td>
</tr>
<tr>
<td>10–19</td>
<td>17,558</td>
<td>80%</td>
</tr>
<tr>
<td>20–29</td>
<td>10,883</td>
<td>66%</td>
</tr>
<tr>
<td>30–39</td>
<td>12,266</td>
<td>31%</td>
</tr>
<tr>
<td>40–49</td>
<td>13,491</td>
<td>4%</td>
</tr>
<tr>
<td>50+</td>
<td>14,550</td>
<td>0%</td>
</tr>
<tr>
<td>Total</td>
<td>86,572(^b)</td>
<td>48%</td>
</tr>
</tbody>
</table>

\(^a\) Number of LSS cohort subjects who were in Hiroshima or Nagasaki when the A-bombs were dropped and for whom an individual exposure dose was estimated.

\(^b\) Of these subjects, 37,458 people had an exposure dose not exceeding 5 mSv.

Table 2.3 Solid cancer deaths in the LSS cohort (1950–1997)

<table>
<thead>
<tr>
<th>Dose (mSv)</th>
<th>Subjects</th>
<th>Cancer deaths</th>
<th>Excess cancer deaths</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5</td>
<td>37,458</td>
<td>3,833</td>
<td>0</td>
<td>0.0%</td>
</tr>
<tr>
<td>5–100</td>
<td>31,650</td>
<td>3,277</td>
<td>44</td>
<td>1.3%</td>
</tr>
<tr>
<td>100–200</td>
<td>5,732</td>
<td>688</td>
<td>39</td>
<td>5.7%</td>
</tr>
<tr>
<td>200–500</td>
<td>6,332</td>
<td>763</td>
<td>97</td>
<td>12.7%</td>
</tr>
<tr>
<td>500–1,000</td>
<td>3,299</td>
<td>438</td>
<td>109</td>
<td>24.9%</td>
</tr>
<tr>
<td>1,000–2,000</td>
<td>1,613</td>
<td>274</td>
<td>103</td>
<td>37.6%</td>
</tr>
<tr>
<td>2,000+</td>
<td>488</td>
<td>82</td>
<td>48</td>
<td>58.5%</td>
</tr>
<tr>
<td>Total</td>
<td>86,752</td>
<td>9,355</td>
<td>440</td>
<td>4.7%</td>
</tr>
</tbody>
</table>
all other exposure dose groups so the “excess death” statistic was calculated to show the number of additional cancer-related fatalities compared to the < 5 mSv group. The precise numbers of excess cancer deaths are shown in the fourth column of Table 2.3. Dividing the number of excess deaths by the number of cancer deaths in each group gives the “attributable risk” ratio which, as the table shows, increases in direct proportion to the exposure dose.

Here I would like to emphasize that, after following up 87,000 subjects for a period of 50 years, there were 440 excess cancer deaths. This is noteworthy because such accurate results would be virtually inconceivable in a typical epidemiological study, and because only 440 of the LSS cohort subjects died over a 50-year period (an average of 10 deaths per year) despite the level of concern that generally surrounds the adverse health effects of radiation and particularly radiation-induced cancer deaths. In fact, this was the first time that such results had been obtained from a definitive study on A-bomb radiation exposure.

The above-mentioned LSS mortality study forms the crux of epidemiological A-bomb survivor statistics and its findings have also been adopted by international organizations under the concept of lifetime risk. However, mortality studies also have limitations in that death certificates make no mention of cancer if a person dies from other causes, and that in some cases the cancer is cured by treatment. This underlines the importance of investigating whether subjects have cancer, which is why the LSS mortality study includes surveillance of cancer incidence. Cancer incidence is confirmed not only on the basis of death certificates but also using surgical and autopsy records and especially the tumor registries and the AHS commenced in Hiroshima and Nagasaki in 1958.

Table 2.4 shows the LSS cohort study findings on the excess risk of solid cancer incidence. The table is sourced from a 2007 report by Preston et al.\(^{16}\) on solid cancer incidence in A-bomb survivors but the figures themselves were obtained from the aforementioned RERF publication\(^{10}\). The number of cancer deaths adheres to the LSS mortality study findings but the number of excess cancers was estimated based on the excess relative risk (ERR) model of linear dose response adjusted for sex, age at exposure and attained age. While all figures are categorized by exposure dose, excess cancers were estimated for all subjects (including those in the < 5 mSv group not shown in the table) according to a model assuming a linear relationship between exposure dose and cancer incidence (“linear model”). As the table shows, there were 848 estimated
excess cancer subjects among the 44,635 subjects with an exposure dose of 5 mSv or greater.

b. Publication of study results based on RERF materials

As I mentioned in subsection 2.2.4-a), the RERF documents on A-bomb survivors are the most comprehensive and accurate materials available, and could not have been compiled without the selfless cooperation of the A-bomb survivors. Given the theme of this book, it would be inappropriate to comment on the occasionally-unfair treatment of A-bomb survivors from the time they were exposed until the present. However, I would like to reiterate my appreciation for their cooperation in putting together these valuable and unique documents.

RERF researchers use the information contained in these documents to publish various study findings. During my tenure as Chairman, we adopted a policy of releasing these research papers under the Foundation’s name but invited any one with doubts or queries about their validity or factual accuracy to make their opinions known in order to facilitate constructive debate and discussion. After all, these valuable documents should not be regarded as the property of RERF researchers but rather as a common resource to be shared by researchers all over the world.

c. Relationship between LNT and ERR of solid cancer mortality or incidence

I would like to preface the following remarks by stating that the above-mentioned stance also applied to the study findings outlined below. The documents described in 2.2.4-a) were analyzed using epidemiological and statistical methods before appearing in papers published by RERF researchers.

<table>
<thead>
<tr>
<th>Colon dose (mSv)</th>
<th>LSS subjects</th>
<th>Cancer-affected subjects</th>
<th>Estimated excess&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Attributable risk&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>5–100</td>
<td>27,789</td>
<td>4,406</td>
<td>81</td>
<td>1.8%</td>
</tr>
<tr>
<td>100–200</td>
<td>5,527</td>
<td>968</td>
<td>75</td>
<td>7.6%</td>
</tr>
<tr>
<td>200–500</td>
<td>5,935</td>
<td>1,144</td>
<td>179</td>
<td>15.7%</td>
</tr>
<tr>
<td>500–1,000</td>
<td>3,173</td>
<td>688</td>
<td>206</td>
<td>29.5%</td>
</tr>
<tr>
<td>1,000–2,000</td>
<td>1,647</td>
<td>460</td>
<td>196</td>
<td>44.1%</td>
</tr>
<tr>
<td>2,000–</td>
<td>564</td>
<td>185</td>
<td>111</td>
<td>61.0%</td>
</tr>
<tr>
<td>Total</td>
<td>44,635</td>
<td>7,851</td>
<td>848</td>
<td>10.7%</td>
</tr>
</tbody>
</table>

<sup>a</sup> Estimated using the excess relative risk (ERR) model of linear dose response adjusted for sex, age at exposure and attained age.

<sup>b</sup> Determined by dividing the number of cancer deaths by the number of estimated excess cancers.
Figure 2.3 illustrates the linear model relationship between exposure dose and ERR* (“linear dose response”) for the LSS mortality study findings. The graph depicts the sex-averaged dose–response function (“dose effect”) of solid cancer mortality for attained age 70 after exposure at age 30.

The solid straight line is the estimated linear dose response function and the dose-category specific ERR correspondingly moves up and down as shown by the respective points. The thick dotted line is a smoothed estimate derived from the points, and the two thin dotted lines represent upper and lower one-standard-error bounds on the smoothed estimate. The study results demonstrate the linear correlation between exposure dose and cancer risk.

Let us now consider the implications of these results with respect to low dose effects. The explanation is slightly convoluted but warrants our attention as an important topic of current debate.

The graphs in Figure 2.4 (a) and 2.4 (b) show the estimated linear relationship and dose category-specific points. In Figure 2.4 (a), there is no significant difference between the observed cancer ERR (y-axis) in the range of

* The relative risk (RR) indicates that the exposed group’s risk is “x” number of times greater than that of the sex- and age-matched control group so a relative risk of 1 means that there is no effect. Excess relative risk (ERR) is equivalent to the value of RR minus 1 and is only indicative of excess risk. Both RR and ERR are expressed as a ratio to the control group.