Appendix F Radiation Effects at Low Doses

For the Hiroshima and Nagasaki bombs, the exposure of the population was primarily from gamma rays and neutrons emitted almost simultaneously with the bomb explosion. The observational evidence for radiation-induced cancer in humans comes largely from these exposures and others in which large doses were received over short periods of time. However, for the setting of environmental standards and for gauging the consequences of exposures routinely received by the general public, the most important doses are relatively small doses received over long periods of time.

Conventional Assumption for Low Doses: the Linearity Hypothesis

In the absence of directly applicable observational evidence, the rate of cancer induction at low doses and dose rates is estimated by extrapolation from observations at high doses. A particularly simple extrapolation estimate is provided by the widely-adopted *linearity hypothesis*, according to which the increased risk is proportional to the excess radiation dose.

This hypothesis has been adopted by the major advisory bodies in their recent publications. There are some differences in details and several of these groups (ICRP, NCRP and UNSCEAR, but not BEIR V) have included a dose and dose rate effectiveness factor (variously, DDREF or DREF) of about 2, which halves the risk per unit dose at low doses or low dose rates (or both) compared to the risk given by a linear extrapolation from the high dose region. [See end note at the end of Chapter 15 for identification of these groups.]

Taking the DDREF into account, as well as other minor differences in the estimates, an overall consensus estimate for low doses and low dose rates is:

risk of eventual fatal cancer: 0.05 per Sv (0.0005 per rem).

This risk factor can be taken to apply to an "average person" but in its most precise form applies to a general population. Consider a population of 100,000, with a representative distribution by age and sex. Then, for example, if each person receives a 20 mSv dose, the collective exposure is 2000 person-Sv and the calculated number of excess eventual cancer deaths is 100.

Despite being widely accepted as a guideline in setting standards for protecting public health, the linearity hypothesis is not firmly established as an expression of scientific knowledge. Thus, the BEIR V report expresses the following major reservation:

> ... departure from linearity cannot be excluded at low doses below the range of observation. Such departures could be in the direction of either an increased or decreased risk. Moreover, epidemiological data cannot rigorously exclude the existence of a threshold in the millisievert dose range. Thus the possibility that there may be no risks from exposures comparable to natural background radiation cannot be ruled out. At such

low doses and dose rates, it must be acknowledged that the lower limit of the range of uncertainty in the risk estimates extends to zero.

Reflecting the uncertainties, many alternative forms have been proposed for the shape of the curve relating cancer risk and radiation dose (see Figure F-1). These include:

1. The linearity assumption [curve B].
2. Greater risk at low doses than implied by linearity ("supra-linearity") [curve A].
3. A linear-quadratic curve in which the low-dose risk is depressed [curve C].
4. A negative region at very low doses, corresponding to a beneficial effect (this is termed hormesis) [curve D].
5. A threshold, below which there is no appreciable cancer induction [not shown].
6. A DDREF which reduces the risk below that calculated for linearity [not shown].

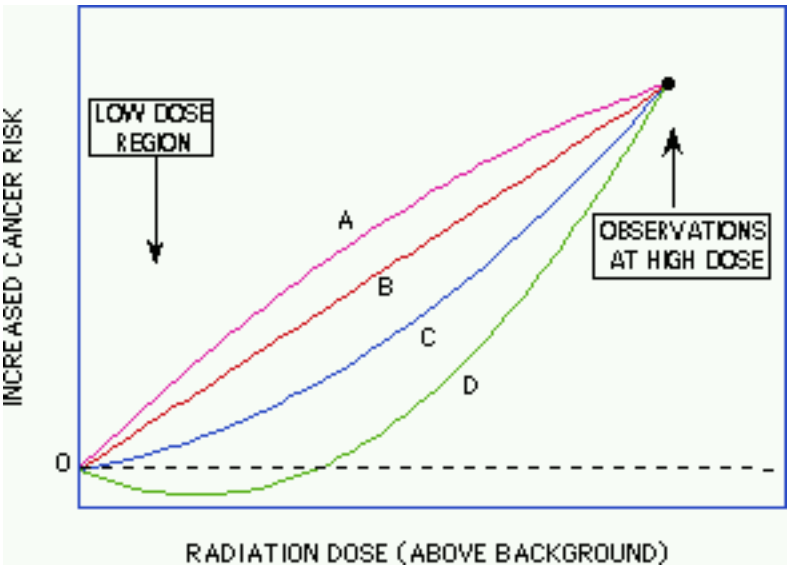

Figure F-1**.** Alternative assumptions for the extrapolation of the cancer risk vs. radiation dose to low-dose levels, given a known risk at a high dose: supra-linearity (A), linear (B), linear-quadratic (C) and hormesis (D).

The conventional wisdom, as reflected by the chief advisory bodies, is to accept linearity (1), usually with inclusion of a DDREF (6). Alternatives (2) and (4) are outside the mainstream of standard assessments, and can be considered to be maverick opinions, although of late more serious attention has been given to hormesis (4) and to the possibility of adaptive mechanisms that might explain it.[1]

The most substantial dissent from the conventional wisdom is the contention that at low doses the effects are much lower than implied by linearity. This view is reflected in a position statement issued in early 1996 by the *Health Physics Society*, a leading US professional organization. According to this statement, for doses below 100 mSv (10 rem) "risks of health effects are either too small to be observed or are non-existent." This statement reflects the very controversial status of the assessment of the radiation risks at low doses.

Evidence on Radiation Effects at Low Doses and Low Dose Rates

In principle, the uncertainties surrounding low-dose/low-dose-rate effects could be settled by the study of populations that have been exposed to slightly above-average radiation doses. Such populations exist in many countries, including China, India, Brazil and the United States.[2] However, due to statistical uncertainties, the difficulties of establishing appropriate comparison groups, and lack of consistency among studies, these studies have not provided convincing evidence either to support or refute the linearity hypothesis.

A second possibility is to look at the experience of workers in the nuclear industry. The results of individual studies have been inconclusive, and to investigate the matter further a combined analysis has been carried out of seven studies—three for sites in the United States (Hanford, Oak Ridge, and Rocky Flats), three for sites in the United Kingdom, and one for Canada. A total of 95,673 workers was included, of whom 60% received effective doses above 10 mSv (1 rem). In the entire population, there were 15,825 deaths, of which 3976 were from cancer. The comprehensive results for all cancers taken together showed a very slight decrease in cancer rate with increasing dose. However, this result had no statistical significance. Of possible greater statistical significance is a slight increase with radiation dose for some types of leukemia. Overall, the statistical uncertainties were large enough that the analysis did not rule out linearity or any of the other alternative dose-response curves indicated in Figure 15-1—although it does set an upper limit on the possible magnitude of a hypothesized supra-linearity effect.

There is one group of nuclear industry workers for which there has been well-established harm, namely uranium miners who received large doses from radon and clearly have elevated lung cancer rates. The radon in mines originates from the decay of radium and the seepage of the resulting radon into the mine. There is similar seepage of radon into houses, causing a buildup of indoor radon, although usually at levels far below those experienced by the early uranium miners. The EPA estimates that indoor radon now leads to 7000 to 30,000 lung cancer fatalities per year in the United States.

Efforts to confirm directly the effects of indoor radon have led to mixed and highly controversial conclusions. One class of studies, termed ecological studies, looks for correlations between the average radon level in a region and the lung cancer fatality rate. In the largest and best known of these studies, covering 1,729 counties in the United States, Bernard Cohen finds the county-by-county lung cancer rates to be inversely correlated with average radon levels. Although many readers have interpreted this study as suggesting hormesis, Cohen limits his conclusions to saying that the results refute the linearity hypothesis. This study covered most of the US population, and therefore the statistical uncertainties are small.

However, these conclusions are hotly disputed by those who contend that ecological studies are inherently flawed. They call instead for reliance on epidemiological studies in which comparisons are made between groups of individuals, where the radon exposure and health history is determined for each person. In *case-control* studies, a group

of lung cancer victims is matched against a non-diseased control group and the history of past radon exposure is compared. In an analysis published in early 1997, Jay Lubin and John Boice carried out a combined analysis of the eight largest case-control studies. From this combined data, they find a positive correlation between risk and dose, consistent with a linear extrapolation from the data on miners. The contradiction between this result and that of Cohen will probably not be resolved without additional studies.

In summary, none of these approaches has provided unambiguous evidence of cancer induction at low dose levels, and the issue remains highly controversial. In a 1990 report, the ICRP concluded that: "Overall, studies at low dose, while potentially relevant to the radiation protection problem, have contributed little to quantitative estimates of risk." Progress since 1990 does not appear to have decisively changed the situation. It is not obvious that epidemiological or ecological studies of any sort will be able to resolve the question of the effects of low-level radiation, although it should be possible to set upper limits on the magnitude of any effects.

In the end, the answers may have to come from a better understanding of damage and repair mechanisms at the cellular or molecular level. Here as well, however, the fundamental issues are still unsettled. For example, in a 1994 UNSCEAR report on adaptive responses to radiation, the state of knowledge was summarized in the following cautious manner:

> It is to be hoped that better understanding of mechanisms of radiation effects obtained in molecular studies might provide a basis upon which to judge the role of adaptive response in the organism. In the meantime, it would be premature to conclude that cellular adaptive responses could convey possible beneficial effects to the organism that would outweigh the detrimental effects of exposure to low doses...

It is these uncertainties, on biological as well as epidemiological questions, that keep the controversies alive.

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[1]The term *adaptive* refers to processes that, in the words of the 1994 UNSCEAR Report, "... may condition cells so as to induce processes that reduce either the natural incidence of cancer in its various forms or the likelihood of excess cancers being caused by further ionizing radiation."

[2]For example, it has been pointed out that cancer rates are lower in Colorado than in Louisiana, although the doses from terrestrial radiation and cosmic rays are roughly 1 mSv/yr greater in Colorado due to the mineral content of the ground and the higher altitude. But this comparison carries little significance without extensive comparisons of other factors that might influence the cancer rates in the two populations.