Health Effects Models for Nuclear Power Plant Accident Consequence Analysis

Low LET Radiation

Part I: Introduction,
Integration, and Summary

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Part I: Introduction,
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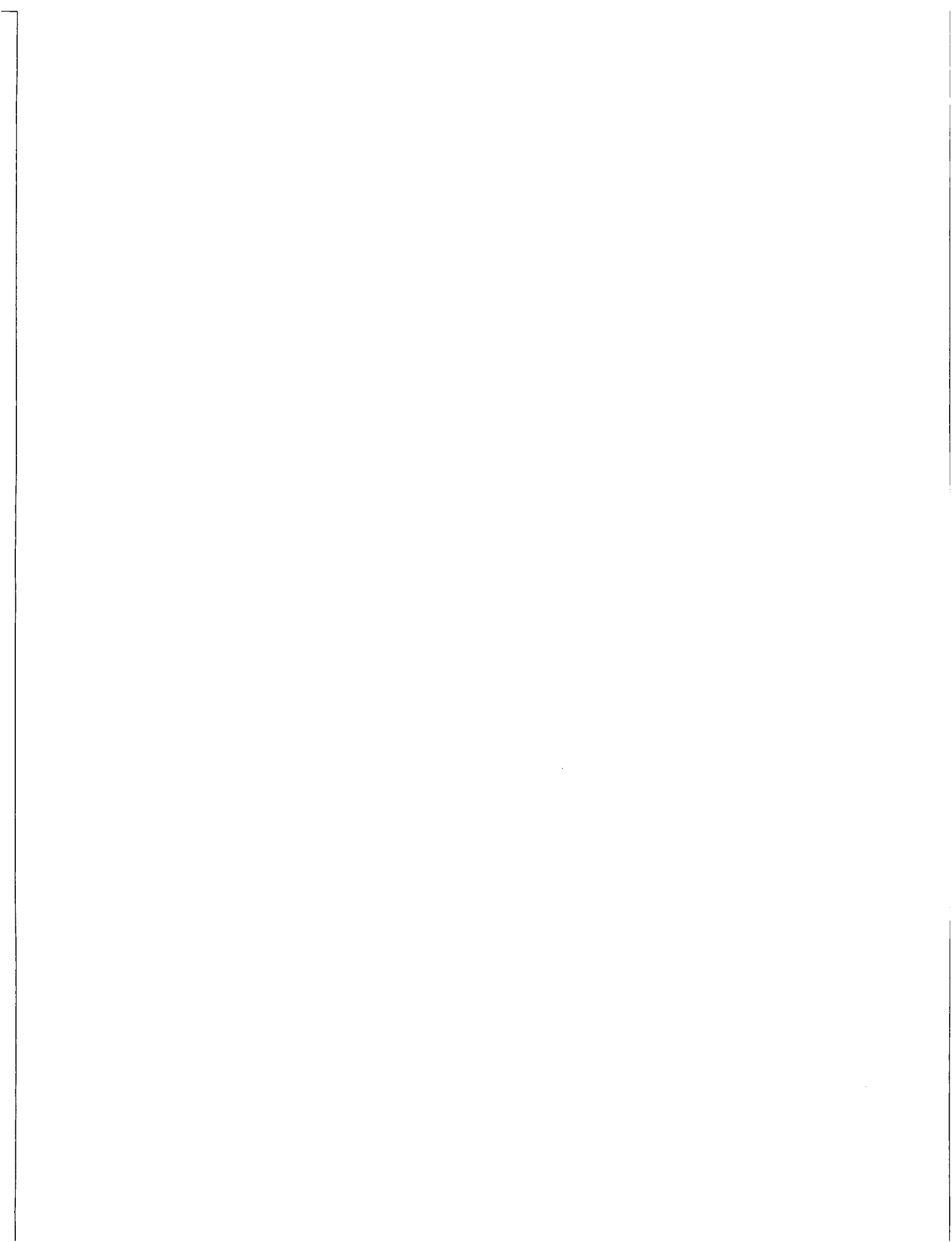DISTRIBUTION OF THIS DOCUMENT IS UNLIMITED

ABSTRACT

This report describes dose-response models intended to be used in estimating the radiological health effects of nuclear power plant accidents. Models of early and continuing effects, cancers and thyroid nodules, and genetic effects are provided.

Weibull dose-response functions are recommended for evaluating the risks of early and continuing health effects. Three potentially lethal early effects--the hematopoietic, pulmonary, and gastrointestinal syndromes--are considered. In addition, models are included for assessing the risks of several nonlethal early and continuing effects—including prodromal vomiting and diarrhea, hypothyroidism and radiation thyroiditis, skin burns, reproductive effects, and pregnancy losses.

Linear and linear-quadratic models are recommended for estimating cancer risks. Parameters are given for analyzing the risks of seven types of cancer in adults--leukemia, bone, lung, breast, gastrointestinal, thyroid, and "other." The category, "other" cancers, is intended to reflect the combined risks of multiple myeloma, lymphoma, and cancers of the bladder, kidney, brain, ovary, uterus and cervix. Models of childhood cancers due to in utero exposure are also developed. For most cancers, both incidence and mortality are addressed. The models of cancer risk are derived largely from information summarized in BEIR III—with some adjustment to reflect more recent studies. The effect of the revised dosimetry in Hiroshima and Nagasaki has not been considered in the analysis of cancer risks.

Linear and linear-quadratic models are also recommended for assessing genetic risks. Five classes of genetic disease—dominant, x-linked, aneuploidy, unbalanced translocations, and multifactorial diseases—are considered. In addition, the impact of radiation-induced genetic damage on the incidence of peri-implantation embryo losses is discussed.

The uncertainty in modeling radiological health risks is addressed by including central, upper, and lower estimates of all model parameters. Data are provided that should enable analysts to consider the timing and severity of each type of health risk.

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ACKNOWLEDGMENTS

1.0 INTRODUCTION

For several decades there has been interest in predicting the health effects of accidental releases of radionuclides from nuclear power plants. In 1975 the U.S. Nuclear Regulatory Commission (NRC) issued the Reactor Safety Study, which gave quantitative estimates of the health and economic consequences of such accidents [NRC, 1975]. The health effects models developed for the Reactor Safety Study have provided the basis for most of the official estimates of the health consequences of nuclear power plant accidents. They are used in several health consequence computer codes, e.g., CRAC.

In 1981 the NRC, through a contract with Sandia National Laboratories, began a critical review of the Reactor Safety Study health effects models. The review, which was directed by Dr. Douglas W. Cooper at Harvard University, concluded that several components of the Reactor Safety Study health effects models required revision.

In the fall of 1982 the NRC initiated an effort to prepare improved health effects models to replace those used in the Reactor Safety Study. An Advisory Committee, consisting of seventeen experts, was assembled. Nominations for appointment to the Advisory Committee were solicited from over three hundred scientists. The Advisory Committee was responsible for oversight and review of the model development process and for assisting in the selection of Working Groups.

The Working Groups were responsible for conducting literature reviews, making recommendations for health effects models, and preparing reports giving the scientific basis for each model recommended. The entire project was managed by a group of scientists at Harvard University, led initially by Dr. Douglas W. Cooper and later by Dr. John S. Evans.

The first draft of NUREG/CR-4214 was completed in the summer of 1983. It was reviewed at a meeting of the Working Group Chairpersons on 29 August 1983 and, after minor revisions, at a joint meeting of the Advisory and Working Groups on 26-27 January 1984. A second draft of the report was completed in the summer of 1984. It was reviewed by the Advisory Group, the Working Groups, Sandia National Laboratories, the NRC, and a small group of external reviewers who had not been involved in the model development process.

NUREG/CR-4214 [NRC, 1985] was published in July of 1985. The NRC circulated the document widely. More than 1000 copies of the report were distributed for public review and comment. Formal public presentations of the new models were made in Washington, DC, on 10 October 1985, and in Luxembourg on 19 April 1985.

In the spring of 1987, the NRC initiated a project to further revise the models. One of the primary goals of the revision was to ensure that the models for early effects were consistent with data on humans who had been accidentally or therapeutically exposed to radiation. A group of scientists at the University of Pittsburgh, led by Dr. Niel Wald, was retained to review the available human data; to assist in the interpretation of that data; and to recommend values of population injury thresholds based on the human data. A second goal was to develop upper and lower estimates of parameters for all early effects to reflect the uncertainties inherent in the models. Drs. Bobby Scott and Fletcher Hahn of the Lovelace Inhalation Toxicology Research Institute, the developers of the early effects models presented in the original report, were retained to revise those models. The NRC was particularly concerned that the original parameters for pulmonary syndrome mortality be critically reviewed.

In addition to achieving these two primary goals, the NRC sought to update the models for late somatic effects to reflect the continuing follow-up of the survivors of the atomic bombings at Hiroshima and Nagasaki and to expand the definition of genetic effects to include consideration of the peri-implantation embryo losses induced by radiation. The authors of the late somatic effects and genetic effects chapters of the original report, Dr. Ethyl Gilbert and Dr. Seymour Abrahamson, were asked to review their chapters in response to these concerns.

The revisions to the late somatic effects and genetic effects chapters were relatively minor and were completed by the end of the summer of 1987. The revisions of the early effects models were much more extensive and were completed in two phases. The first phase reviewed the human data on early effects and developed lower, central, and upper estimates of parameter values for all of the early effect models. The revised parameter values were selected in a series of meetings of the early effects working group. In the course of these meetings, the working group determined that models which explicitly accounted for the dependence of risk on dose rate would be desirable. The second phase developed such models for bone marrow and pulmonary syndrome mortality. The revised early effects models including the new dose rate models were completed in the fall of 1988.

This report, which has been published in two volumes, represents an effort to summarize the revised models, to describe the sources of recommended model parameters, and to discuss the bases for key assumptions. Part I: Introduction, Integration and Summary, which was prepared by the group at Harvard charged with oversight of the project, is an overview, based largely on material developed in Part II. It assumes only rudimentary familiarity with mathematics and little prior knowledge of biology or health physics, and is intended to make the models available to the widest possible audience. Part II: Scientific Bases for Health Effects Models, which was prepared by the scientists in the various working groups, is intended to provide epidemiologists,

radiobiologists and other health scientists with detailed information on the origins of the models.

The models presented in this report are intended for use in nuclear power plant accident consequence analysis. They represent one element of a much larger effort to improve the computer codes used by the NRC to estimate the health and economic consequences of various potential accident scenarios. Other components of the accident consequence codes consider the probabilities of initiating events, the likelihood and magnitude of the releases, the environmental fate and transport of radionuclides, and the organ-specific doses expected. Although important, these topics are not addressed in this report. Interested readers should consult, for example, the PRA Procedures Guide [NRC, 1983] for discussions of these matters.

The report is not intended as a guide for physicians or others involved in the handling of radiation emergencies. It is also not intended to represent a compendium of information on radiobiology. Its purpose is simply to document the dose-response models recommended for estimating the health effects of nuclear power plant accidents.

1.1   Treatment of Uncertainty

The health risks caused by radiation cannot be predicted precisely. The statement of work that initiated this program reflected an awareness of this and sought:

> ". . . a realistic assessment of the health effects and risks due to the radiation dose levels and types expected from nuclear reactor accidents. The uncertainties associated with each health effect relationship shall be described and, to the extent possible, quantified. For those cases where the uncertainty can't be fully quantified, upper and lower bounds should be estimated."

The uncertainties in modeling health risks are of two types—parameter uncertainties and model uncertainties. Parameter uncertainty arises in the process of drawing inferences about processes which are to some extent random (or are observed with error) from small samples. If this were the only source of uncertainty, it would be relatively simple to provide complete descriptions of the uncertainty in each of our estimates of health risk. Unfortunately, the other source of uncertainty—model uncertainty—is not amenable to simple analysis. Model uncertainty arises from the need to rely on analogy. For example, estimates of the risks of pulmonary syndrome mortality are based in part on evidence from studies of beagles. The accuracy of such estimates depends on the adequacy of the analogy. Similarly, most estimates of radiation-induced cancer risk are based on studies of the survivors of the bombings at Hiroshima and Nagasaki. Again, the accuracy of the extrapolation from the high doses and high dose rates received by the Japanese survivors to

the low doses and dose rates frequently of interest depends on the validity of the analogy. Estimation of the extent of the uncertainty in these analogies is unavoidably subjective.

We have taken a first step toward addressing uncertainty by providing three estimates of each effect: a central estimate, a lower estimate and an upper estimate. The central estimates are intended to be realistic estimates, reflecting the judgment of the scientists involved in model development. The upper and lower estimates are intended to reflect alternative assumptions that are reasonably consistent with available evidence and that may be preferred by some scientists.

The uncertainties in estimating the health effects induced by exposure to radiation are considerable. In view of this, it is important that accident consequence analyses consider the spectrum of possible consequence estimates rather than focusing attention on the central estimates.

1.2 Measures of Accident Consequences

Any complete description of risk involves both probability and severity. This report provides models for estimating the probabilities of more than twenty-five effects that may be induced by ionizing radiation. The report also includes some information about the severity of each effect. For most early effects, brief descriptions of the nature and duration of symptoms are given.

For each type of cancer, in addition to the models of morbidity and mortality risk, the report gives two measures of severity: (1) the average interval (years/case) between diagnosis and death (an index of the length of illness), and (2) the average loss of life (years/death) among those who die from the disease.

For each class of genetic disease, the report provides estimates of the typical interval (years/case) between the onset of symptoms and death and of the average loss of life expectancy (years/case). In addition, examples of the types of genetic diseases (defects) included within each class are described.

Some analysts may be concerned about the distribution of radiation-induced cancers and genetic effects over time. Tables in the body of the report illustrate the temporal aspects of these risks.

1.3 Organization of Part I

The remainder of this volume is organized in two chapters. Chapter 2, Model Descriptions, gives the mathematical forms of the models and summarizes the parameter values recommended for central, lower, and upper estimates of health risks. In most cases the parameter values recommended are those presented in Part II as developed by the Working Groups.

In the few cases where alternative values have been chosen, the reasons for departing from the recommendations of the Working Groups are given. Chapter 3, <u>Computational Aspects</u>, has several purposes. Its primary purposes are to describe the mathematical procedures used to obtain the population-based models of health risks needed for accident consequence analysis and to discuss approaches for implementing the models in accident consequence analysis computer codes. In addition, this chapter briefly considers other topics of computational interest, e.g., the relationship between the Weibull, probit, and logistic models.

2.0 MODEL DESCRIPTIONS

The health effects model represents one of many components within the family of nuclear power plant accident consequence models. Other models are used to describe the release and transport of contaminants, to analyze the need for and effectiveness of emergency countermeasures such as evacuation, sheltering, and respiratory protection, and to calculate the doses received as a result of an accident. The Overview of the Reactor Safety Study Consequence Models [NRC, 1977] provides a clear introduction to consequence modeling. The output from the release, transport, and dosimetry models is a set of estimates of organ-specific doses expected to be received by the population in each of several geographic cells surrounding a nuclear power plant. This set of organ-specific absorbed doses is the input required by our model.

The health effects model is a collection of models. The collection includes three broad classes of effects: early and continuing effects, late somatic effects and genetic effects. Tables 2.1 and 2.2 list the effects for which models have been developed and the target organ for each model.

The models are intended to permit estimation of risks associated with exposure to low-LET radiation.

2.1 Early and Continuing Effects

In the event of a severe nuclear power plant accident, those living nearby may receive doses large enough to suffer from the "early and continuing" effects of radiation. The early effects, which include the potentially lethal hematopoietic, pulmonary and gastrointestinal syndromes and several less severe effects such as vomiting, diarrhea and skin burns, typically occur within the first few days or weeks after exposure. Continuing effects, such as hypothyroidism, pneumonitis, diminution of sperm count/suppression of ovulation, and cataracts, may require somewhat longer to develop or may involve symptoms that persist for several years after exposure. Irradiation of women pregnant at the time of the accident may also lead to increased risks of embryo loss, fetal death or mental retardation among those babies that survive.

Knowledge of the risks of these effects derives largely from four sources: studies of radiation-related side effects among humans exposed therapeutically, analyses of the experience of the survivors of the atomic bombings of Hiroshima and Nagasaki, examination of the health effects observed among the relatively small number of individuals who have received large radiation doses in various accidents, and investigations of the effects observed in animals experimentally exposed to radiation.

Our models for early and continuing effects were developed by Drs. Bobby Scott and Fletcher Hahn of the Lovelace Inhalation Toxicology Research

Table 2.1

Early Effects Included in Health Effects Models

	Model Developed		
Effect	Mortality	Morbidity	Target Organ
Hematopoietic Syndrome	✓	-	Bone Marrow
Pulmonary Syndrome	✓	-	Lung
Gastrointestinal Syndrome	✓	-	Small Intestine[a] —Colon
Prodromal Symptoms			
Vomiting	-	✓	Abdomen[b]
Diarrhea	-	✓	Abdomen[b]
Pneumonitis	-	✓	Lung
Thyroid Effects			
Thyroiditis	-	✓	Thyroid
Hypothyroidism	-	✓	Thyroid
Skin Effects			
Erythema	-	✓	Epidermis[c]
Transepidermal Injury	-	✓	Epidermis[c]
Cataracts	-	✓	Lens of Eye
Embryo/Fetus			
Microencephaly	-	✓	Embryo/Fetus
Severe Mental Retardation	-	✓	Embryo/Fetus
Death of Embryo/Fetus	✓	-	Embryo/Fetus

[a] The dose to small intestine is used to estimate the risk from brief external exposure. The dose to the colon is used to estimate the risk from protracted internal exposure.

[b] Midline, midplane upper abdominal dose.

[c] Dose to the basal cells (about 0.1 mm depth) of an area of 50 to 100 cm^2.

Table 2.2

Late Effects Included in Health Effects Models

Effect	Model Developed		Target Organ
	Mortality	Morbidity	
Somatic Effects			
Leukemia	√	-	Red Bone Marrow
in utero	√	-	Fetus
Bone Cancer	√	-	Bone
Breast Cancer	√	√	Breast
Lung Cancer	√	√	Lung
Gastrointestinal Cancer	√	√	Lower Large Intestine
Thyroid Cancer	√	√	Thyroid
Skin Cancer	-	√	Epidermis[a]
Other Cancer	√	√	Other[b]
in utero	√	-	Fetus
Benign Thyroid Nodules	-	√	Thyroid
Genetic Effects			
Single Gene			
Dominant	-	√	Gonads
X-linked	-	√	Gonads
Chromosome Aberrations			
Numerical	-	√	Gonads
Structural	-	√	Gonads
Multifactorial	-	√	Gonads
Pregnancy Loss[c]	-	√	Gonads

[a] Dose to the basal cells (about 0.1 mm depth) of an area of 50 to 100 cm^2.

[b] A weighted combination of the doses to the bone marrow, brain, kidney, bladder, ovary and uterus is recommended.

[c] Most of these losses will occur within the first few days of the pregnancy before the fertilized egg is implanted in the uterine wall.

Institute. The models are based in part on data concerning human radiation injury reviewed by Drs. Niel Wald, Joseph Watson and Albert Spritzer of the University of Pittsburgh. Information on thyroid effects was provided by Dr. Harry Maxon of the University of Cincinnati and several of his colleagues.

Scientific understanding of the biological nature of early effects indicates that they are threshold effects; i.e., in any individual the effect will not be experienced unless a threshold dose is exceeded. Population dose-response functions for these nonstochastic effects are simply reflections of the distributions of individual thresholds—or tolerances—among the population.*

The risks of early and continuing effects have been modeled using hazard functions. The relationship between risk and hazard is given by:

$$r = 1 - e^{-H}$$

where r is the probability that a person will exhibit the effect of interest and H is the cumulative hazard function. The relationship between dose and risk is implicit in the relationship between dose and hazard. The cumulative hazard functions used to predict early effects are two-parameter Weibull functions of the form:

$$H = 0.693 \left[d/D_{50}\right]^v \quad \text{for } d > T$$

where d is the (average absorbed) dose to the relevant organ, D_{50} is the dose at which half of the population experiences the effect, v is the shape parameter, and T is the population threshold dose.

The choice of the two-parameter Weibull hazard function is somewhat arbitrary, as almost any sigmoidal function would fit the data in the experimental region. The alternatives to and implications of this choice are discussed briefly in Section 3.1.6, Form of Dose-Response Model.

For most early effects, dose received at low dose rate is much less effective than dose received at high dose rate. This phenomenon can be accounted for by adjusting the value of the median lethal (or effective) dose used in the hazard function. The simplest adjustment is one in

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* There is consensus that early effects are threshold effects. However, for many effects the available data are too weak to permit precise identification of population thresholds. This is particulary true for effects such as the pulmonary syndrome, where some individuals (those with preexisting lung disease) may be especially sensitive to radiation. One scientist who reviewed the early effects models recommended setting population thresholds at 10 to 20 percent of the median lethal doses rather than at the values selected by the early effects working groups.

which two values of D_{50} are used: one appropriate for dose received at high dose rate, and another for dose received at low dose rate. With this approach, which has been recommended by the early effects working group for computing the risks of most early effects, the cumulative hazard is:

$$H = 0.693 \left[\frac{d_b}{D_{50,b}} + \frac{d_p}{D_{50,p}} \right]^v$$

where d_b is the brief dose (received at high dose rate) and d_p is the protracted dose (received at low dose rate). Although simple, this approach may yield relatively imprecise estimates of risk, especially when the median lethal dose is a strong function of the dose rate.

Better estimates of risk are obtained by increasing the number of terms in the model. In the limit, a continuous form of the model is reached:

$$H = 0.693 \left[\int \frac{\dot{D}}{D_{50}(\dot{D})} \, dt \right]^v$$

where $\dot{D}$ is the instantaneous dose rate at time t, $D_{50}(\dot{D})$ is the median lethal dose applicable to dose received at dose rate $\dot{D}$ and H is the cumulative hazard function. This is the approach recommended by the early effects working group for computing the risks of death from the hematopoietic and pulmonary syndromes. For these effects, the relationship between dose rate and median lethal dose is modeled using:

$$D_{50}(\dot{D}) = \Theta_\infty + \Theta_1 / \dot{D}$$

where Θ_∞ is the limiting value of the median lethal dose (Gy), $\dot{D}$ is the instantaneous dose rate (Gy/hr), and Θ_1 is a parameter reflecting the sensitivity of the median lethal dose to the dose rate (Gy^2/hr).

The parameter values recommended for evaluating the risks of early effects were selected in a series of meetings attended by Drs. Hahn, Scott, Spritzer, Wald, Watson, Yaniv, and Young. In some cases, where reliable data were unavailable or different studies led to conflicting results, the parameter values are judgmental estimates reflecting the views of these scientists. The next several sections of this report describe the early effects that were considered and review the data used in parameter selection.

2.1.1 Early Mortality

The three early causes of death considered in the health effects models are the hematopoietic syndrome, the pulmonary syndrome, and the

gastrointestinal syndrome. The hematopoietic syndrome will be the dominant cause of early fatalities following brief whole body exposures. The typical loss of life expectancy associated with death from the hematopoietic, pulmonary, or gastrointestinal syndrome is about 45 years.

2.1.1.1 Hematopoietic Syndrome

The effects observed after irradiation of the bone marrow result from killing blood cell precursors (stem cells) in the marrow. If the ensuing depression in peripheral blood cells is severe, the individual may die from infection or hemorrhage. However, for this to happen the number of surviving stem cells must be depressed below a critical level. Otherwise, the numbers of peripheral blood cells will return to normal levels and the individual will survive.

The median lethal dose for humans is not precisely known. Several estimates have been published, ranging from 2.4 to 5.1 Gy to the bone marrow. Some of the higher estimates involve cases where significant medical treatment was administered. When these studies are excluded, the range of estimates narrows considerably. The judgment of our early effects working group was that a central estimate of the LD_{50} appropriate for individuals exposed to external irradiation at high dose rate might be 3 Gy and that reasonable lower and upper estimates would be 2.5 and 3.5 Gy.

Because the risk of hematopoietic syndrome mortality depends upon the level of medical treatment received, two sets of parameters are provided —one appropriate for those receiving "minimal" medical treatment and one appropriate for those receiving "supportive" medical treatment. Minimal medical treatment involves basic first aid. Supportive treatment includes hospitalization with routine reverse isolation procedures, antibiotic therapy, blood transfusions, electrolyte replacement, administration of blood products, and parenteral feeding.

A substantial benefit of supportive medical treatment has been demonstrated in dogs exposed to whole-body irradiation. Perman et al. [1962] found a 50 percent increase in the median lethal dose of dogs given supportive treatment (antibiotics, blood transfusions, parenteral fluids and forced feeding) compared to those not treated. Similar results have been reported by Vriesendorp and van Bekkum [1984] and MacVittie et al. [1984].

A third level of treatment, "intensive" medical treatment involving bone marrow transplantation, may increase the chances of survival of some of those suffering from the bone marrow syndrome. It is common for leukemia patients, who often receive doses greater than 10 Gy in conjunction with bone marrow transplants, to survive the effects of radiation. Bone marrow transplants were given to thirteen victims of the accident at Chernobyl. The doses received by these accident victims were estimated to range from about 5 to 15 Gy. Although the results were not encouraging—only two of the 13 survived [Gus'kova, 1987]—the efficacy of this therapy is still unclear. There were many complicating factors at Chernobyl; e.g., the firefighters who received the transplants

suffered from extensive thermal and radiation burns and the timing of the transplants may have been inappropriate.

It is thought that there are over 100 medical centers in the U.S. capable of providing such treatment. Unfortunately there has never been a credible national survey of the number of beds typically available in these facilities, the capability of these centers to handle radioactively contaminated patients, or the willingness of the administrators of these centers to make their facilities and personnel available for treatment of radiation accident victims. The limited data that exist are not convincing [Anderson, 1982]. Until such data become available, we recommend that no allowance be made for the lives that may be saved by intensive treatment efforts such as bone marrow transplantation.

Those who survive the effects of the brief initial exposure to cloud-shine and groundshine may later die due to the combined effects of this initial exposure and any subsequent exposure from materials that were inhaled or ingested. The risk from the combination of brief external exposure (at high dose rate) and protracted internal exposure (at lower dose rate) may be assessed using the approach described in the introductary section on early effects.

Some individuals may accumulate rather large protracted doses. Fortunately, protracted doses received at low dose rate are not as damaging as similar doses received at high rates. Both Scott et al. [1988] and Morris and Jones [1989] have demonstrated the importance of dose rate in studies of early radiation effects in rats, mice, swine, dogs, goats and sheep. In rats and mice the LD_{50} increases by a factor of between 1.5 and 2 as the dose rate is reduced from 10^3 to 10^{-1} Gy per hour. In larger mammals—swine, dogs, goats and sheep—the LD_{50} increases by a factor between 2 and 4 as the dose rate is reduced from 10 to 10^{-2} Gy per hour.

The limited human evidence on the effects of doses received at low dose rates also suggests that these doses must be less effective than the same doses received at high dose rates. Of 23 Japanese fishermen exposed to fallout, seven were estimated to have received doses greater· than 4 Gy. All of them survived. Other anecdotal evidence is found in the experience of a Mexican family accidentally exposed to radiation from a cobalt source. It has been estimated that all five members of the family received doses greater than 8 Gy. One of them survived. If these doses had been received at high dose rates, it is unlikely that any of these individuals would have survived. Although these observations are weakly consistent with the animal data, they should not be overinterpreted. The doses involved are not known accurately and the number of individuals involved is relatively small.

Recently both Scott et al. [1988] and Morris and Jones [1989] have proposed mathematical models that quantitatively express the dependence of the median lethal dose on the dose rate. After reviewing these, the

working group recommended that the LD_{50} (Gy) for hematopoietic syndrome mortality be evaluated using the equations given in the following table.

Estimate	Medical Treatment	
	Minimal	Supportive
Central	$3.0 + 0.07/\dot{D}$	$4.5 + 0.10/\dot{D}$
Lower	$2.5 + 0.06/\dot{D}$	$3.7 + 0.08/\dot{D}$
Upper	$3.5 + 0.08/\dot{D}$	$5.3 + 0.12/\dot{D}$

Current nuclear power plant accident consequence codes cannot take full advantage of these models because the codes do not provide estimates of the rates at which doses are received by various segments of the exposed population. Section 3.1.2 briefly describes the methods used in CRAC and MACCS for estimating the risks of hematopoietic syndrome mortality.

2.1.1.2 Pulmonary Syndrome

The lungs may be irradiated both by external exposures, e.g., cloudshine and groundshine, and by radionuclides that are inhaled. Acute radiation pneumonitis may occur following such exposures. Symptoms of pneumonitis include shortness of breath, fever, nonproductive cough, and hypoxia.

Because of the large doses required to induce this effect, early fatalities from pulmonary injury are not expected to occur as a result of uniform external whole-body irradiation. Where supportive or intensive medical treatment of the hematopoietic syndrome is provided and is successful, pulmonary effects may become a concern. More generally, however, these effects will be expected to occur primarily as a result of the inhalation of radionuclides.

Most human data on the pulmonary effects of irradiation come from studies of patients treated with radiation for breast, lung and other cancers, or given large field irradiation in conjunction with bone marrow transplants for treatment of leukemia and aplastic anemia. Based on radiation therapy data, Phillips and Margolis [1972] have estimated the D_{50} for pulmonary pneumonitis to be 10.4 Gy. Van Dyk et al. [1981] estimated the D_{50} for radiation pneumonitis in humans given single radiation treatments to be 9.3 Gy. Phillips and Margolis do not report the typical dose rates involved, but Van Dyk et al. note that all of the patients in their study received doses at rates between 0.5 and 5 Gy per minute. Because cytotoxic and immunosuppressive drugs—also known to cause lung damage— are frequently administered in conjunction with radiation therapy, it is

difficult to clearly interpret these studies. The early effects working group selected 10 Gy as their central estimate of the LD_{50} for pulmonary syndrome mortality following brief external exposure, and chose lower and upper estimates of 8 Gy and 12 Gy.

Several estimates of the threshold dose have emerged from these clinical studies. Fryer's 1978 study suggests a threshold of about 6 Gy. Van Dyk's reanalysis of Fryer's data indicates that if patients with pre-existing lung disease, e.g., chronic bronchitis, emphysema, are excluded from consideration, the clinical threshold is more nearly 7.5 Gy. Keane [1981] reports that 1 of 11 patients receiving 4 Gy and 3 of 27 patients receiving between 4 and 6 Gy developed radiation pneumonitis. The early effects working group selected 5 Gy as a central estimate of the population threshold for pulmonary syndrome following brief external exposure.

Many factors moderate the risk associated with a specific dose. Three significant factors are dose rate, age-at-exposure, and presence of pre-existing lung disease.

Doses delivered at low dose rate are much less effective for inducing radiation pneumonitis than doses delivered at high dose rate. The clinical studies which provided the basis for the working group's estimate of a 10 Gy LD_{50} involved dose rates in the range of 0.5 to 5 Gy per minute. In the event of a nuclear power plant accident, much of the dose from inhaled radionuclides will be delivered at rates several orders of magnitude lower than this.

Inhalation Toxicology Research Institute (ITRI) studies of beagles exposed to various beta-emitting radionuclides provide striking evidence of the importance of dose rate [McClellan et al., 1982]. The LD_{50}'s observed in these experiments varied from 94 Gy for beagles exposed to yttrium-90 (effective half-life 2.6 days) to 540 Gy for beagles exposed to cerium-144 (effective half-life 200 days). Although these studies did not include experiments in which beagles were exposed to brief external irradiation, their LD_{50} in such a setting would be expected to be similar to those seen in other mammals studied, i.e., between 10 and 20 Gy [Scott et al., 1989]. McClellan's study suggests that protracted internal exposures are between 1/10th and 1/50th as effective as brief external exposures.

Using data from beagles and rats, Scott, Filipy and Hahn have estimated the parameters of a model relating the median lethal dose for pulmonary syndrome to the dose rate [Scott, 1989]. The early effects working group endorses this approach and recommends that the risk be evaluated using:

$$LD_{50,central} = 10 + 30/\dot{D}$$

$$LD_{50,upper} = 12 + 45/\dot{D}$$

$$LD_{50,lower} = 8 + 15/\dot{D}$$

where the LD_{50} is the median lethal dose (Gy) and $\dot{D}$ is the instantaneous dose rate (Gy/hr).

For pulmonary syndrome, the shape parameter also depends on the nature of the exposure. The working group's central estimates of appropriate shape parameters are 12 for brief external exposure and 5 for protracted internal exposure. When mixed exposures are anticipated, they recommend that a shape parameter of 7 be used.

As noted previously, current nuclear power plant accident consequence codes cannot take full advantage of these models because the codes do not evaluate dose rate patterns in any detail. Section 3.1.2 briefly describes the method used in CRAC and MACCS for estimating the risks of pulmonary syndrome mortality.

The effects of age-at-exposure and pre-existing lung disease are less well understood. In studies of beagles exposed to cerium-144, a strong effect of age-at-exposure has been demonstrated. Old beagles were found to be about twice as sensitive to radiation-induced pneumonitis as young adult dogs [McClellan et al., 1982]. The pattern of age sensitivity in humans is less clear. Early reports, e.g., Rubin and Casarett [1968], tended to discount the importance of age-at-exposure. However, recent studies of patients treated with whole-body radiation suggest that the incidence of interstitial pneumonitis increases with age and is about twice as large in middle-aged patients (40 to 60 y.o.) as in younger patients (1 to 20 y.o.) [Weiner et al., 1986].

2.1.1.3 Gastrointestinal Syndrome

Irradiation of the abdomen may lead to the gastrointestinal syndrome. The symptoms experienced, which may include cramps, abdominal pain, diarrhea, shock and death, depend on the dose received. In animal experiments, the gamma or x-ray doses required to cause death from the gastrointestinal syndrome have been in the range of 10 to 50 Gy. These are much higher than the doses necessary to cause death due to bone marrow syndrome.

Very few human data are available on the gastrointestinal syndrome. It is known, however, that cancer patients given whole body doses of 10 Gy or more in conjunction with bone marrow transplantation have survived the effects of the gastrointestinal syndrome [Thomas et al., 1975]. Bond et al. [1965] notes that mammals tend to respond similarly following gastrointestinal irradiation and suggests that data from animal studies may give a reasonable indication of the risks in humans. Sullivan et al. [1959] found that a brief external dose of about 15 Gy was required to kill about half of the rats exposed in his experiments. Data [Cross et al., 1978] on rats exposed to beta emitting radionuclides have been interpreted as suggestive of an LD_{50} of about 35 Gy for humans following protracted internal exposure.

The early effects working group recommends using these values with rather large uncertainty estimates. Their lower and upper estimates of the LD_{50} for humans following brief external exposure are 10 and 20 Gy respectively. The critical organ for assessing risks following brief exposures is the small intestine. The working group's lower and upper estimates of the LD_{50} for humans following protracted exposure are 25 and 50 Gy respectively. The critical organ for assessing the effects of protracted internal exposure is the colon.

2.1.1.4 Summary—Early Mortality

To assess the overall risk of early mortality from dose to the bone marrow, the lungs, and the gastrointestinal tract, one sums the cumulative hazard functions:

$$r = 1 - e^{-(H_b + H_p + H_g)}$$

where H_b is the cumulative hematopoietic (bone marrow) hazard, H_p is the cumulative pulmonary hazard, and H_g is the cumulative gastrointestinal hazard.

The parameters recommended for estimating risks following brief exposure at high dose rates are summarized in Table 2.3. The effects of protracted exposure at lower dose rates should be evaluated using the dose-rate-dependent models described in Sections 2.1.1.1 and 2.1.1.2. The relationship between these dose-rate-dependent models and the fixed time interval models used in most accident consequence analysis codes is discussed in Section 3, Computational Aspects.

2.1.2 Early Morbidity

The nonlethal effects of exposure to radiation include the prodromal syndrome (nausea, fatigue, vomiting and diarrhea), pneumonitis, hypothyroidism and radiation thyroiditis, erythema and transepidermal injury. In addition, exposure of the fetus/embryo may lead to a variety of effects (microcephaly, severe mental retardation and fetal death) depending upon the dose, dose rate and stage of development. Reproductive effects (e.g., permanent suppression of ovulation in females and temporary suppression of spermatogenesis in males) are also possible.

2.1.2.1 Prodromal Syndrome

The prodromal syndrome is a group of symptoms and signs of acute gastrointestinal and neurovascular effects that begin to occur soon (minutes to hours) after brief irradiation at high dose rate. The gastrointestinal symptoms include anorexia, nausea, vomiting, diarrhea, intestinal cramps, salivation and dehydration [Young, 1986]. The neurovascular symptoms include fatigue, listlessness, apathy, sweating and headache.

Table 2.3

Models of Early Mortality from Brief Exposure[a,b]

Effect	Central			Lower[c]			Upper[c]		
	LD_{50}	T	v	LD_{50}	T	v	LD_{50}	T^d	v
Hematopoietic Syndrome									
Minimal Treatment	3.0	1.5	6	3.5	2	8	2.5	1	4
Supportive Treatment	4.5	2	6	5	3	8	4	1.5	4
Pulmonary[e] Syndrome	10	5	12	12	6	14	8	4	9
Gastrointestinal Syndrome	15	8	10	20	8	10	10	8	10

[a] The doses referred to in this table are organ-specific absorbed doses. The units are Gray (Gy). The parameters, LD_{50}, T and v given in this table are defined in the text of this report (pp. I-9). In some cases, the values recommended by the working group have been rounded to avoid conveying a false sense of precision.

[b] Brief exposure parameters are appropriate for dose received at high dose rate. The values shown for hematopoietic syndrome apply to doses received at rates ≥10 Gy/hr. Those for pulmonary syndrome apply to dose rates ≥100 Gy/hr.

[c] For early effects, use of larger values for LD_{50}, T, and v results in the lower estimates of risk, and vice versa.

[d] As explained in the text, available human data are too weak to support clear choice of population thresholds. Analysts may wish to explore the sensitivity of their results to the threshold values used.

[e] The parameters given are thought to be appropriate for young adults. Older people and those with respiratory disease, e.g., chronic bronchitis or emphysema, may be twice as sensitive.

At the median lethal dose, the principal symptoms of the prodromal reaction are anorexia, nausea, vomiting and fatigue. Diarrhea, fever, and hypotension occur primarily in victims who have received supra-lethal doses [Langham, 1967].

Our models focus on two symptoms of the prodromal syndrome: vomiting and diarrhea. The early effects working group's central estimates of the median effective doses at high dose rates of 2 Gy for vomiting and 3 Gy for diarrhea are based largely on Lushbaugh and Rick's retrospective analysis of the experiences of 2000 patients treated therapeutically with whole-body radiation [Lushbaugh and Ricks, 1972].

2.1.2.2 Pulmonary Morbidity

Virtually all human data on radiation pneumonitis come from studies of patients treated with radiation in conjunction with bone marrow trans-platation or for the control of cancer. Most of the cases of pneumonitis seen in these clinical studies result in death.

Other forms of lung impairment, e.g., reduced lung volumes, increased stiffness, reduced gas exchange efficiency, and nonuniformity in gas distribution, could develop as a result of radiation exposure. In rats whose lungs were exposed to low energy beta or alpha emitting radio-nuclides, doses only $1/4^{th}$ as large as those required to cause death lead to impairment in lung function [Scott et al., 1988]. In contrast, in rats exposed to high energy beta emitting radionuclides doses nearly as large as those required to cause death are necessary to lead to signi-ficant functional impairment.

Lacking reliable human data, the working group decided not to develop a dose-response curve for pneumonitis morbidity or for other forms of non-lethal lung damage. However, in a recent publication, Scott, Filipy, and Hahn [1989] suggest reducing LD_{50} values for pulmonary syndrome mortality by a factor of about 2 to estimate pulmonary morbidity.

2.1.2.3 Hypothyroidism and Radiation Thyroiditis

The thyroid gland is of special concern because of its ability to concentrate iodine. Some nuclear power plant accidents may release relatively large quantities of various radioisotopes of iodine. Thus, the potential for large doses to the thyroid exists. Effects of interest include hypothyroidism, thyroiditis, thyroid cancers, and benign thyroid nodules.

Hypothyroidism is a metabolic state resulting from insufficient amounts of thyroid hormone for normal physiologic function. Hypothyroidism may result in fatigue, decreased tolerance to cold, mental sluggishness, fluid retention, muscle cramps, and a generalized decrease in bodily functions. The symptoms are readily treated with oral doses of thyroid hormone.

Based on a comparison of the incidence of hypothyroidism observed among Graves' disease patients treated with ^{131}I [Maxon et al., 1977] and those treated surgically [Becker et al., 1971], the thyroid effects working group estimated the lifetime risk of clinical hypothyroidism following ^{131}I exposure to be 17×10^{-4} per Gy. The thyroid effects working group notes that hypothyroidism is almost certainly a threshold effect and recommends that a 10 Gy threshold be used in projections of risks of hypothyroidism following ^{131}I exposure.

Animal studies suggest that external radiation is about 5 times as effective as ^{131}I for induction of hypothyroidism. This ratio was used to derive an estimate of hypothyroidism risk due to external irradiation, 85×10^{-4} per Gy and a threshold of 2 Gy.

Concerning the threshold, Watson [Personal Communication, 1987] notes that none of the clinical studies involve ^{131}I doses less than 10 Gy; that 8 to 12 percent prevalence of hypothyroidism is typically observed in the lowest dose groups in these studies; and that the lowest doses in such treatments are commonly 30 to 50 Gy. From these observations he concludes that there is no experimental basis for the existence of a 10 Gy threshold. Therefore, we recommend that upper estimates of hypothyroidism risks be computed using thresholds well below the working group's recommendations of 2 Gy for external radiation and 10 Gy for ^{131}I.

Radiation thyroiditis is an acute condition occurring within two weeks of exposure to radiation and characterized by inflammation and eventual necrosis of some or all of the cells in the thyroid gland. The symptoms are usually mild and involve local pain and tenderness.

Mild radiation thyroiditis was noted by Beierwalters and Johnson [1956] in about 5 percent of patients treated with ^{131}I for thyrotoxicosis. Symptoms were rare in patients who received doses less than about 200 Gy. Acute radiation thyroiditis was observed by Maxon and his colleagues [1977] in nearly 90 percent of patients given large doses of ^{131}I to ablate any remaining thyroid tissue following thyroidectomies. Doses in such procedures commonly exceed 2000 Gy.

On the basis of these observations the thyroid effects working group recommended that the risk of thyroiditis following internal exposure to ^{131}I be estimated using a linear-threshold model with a threshold of 200 Gy and a slope of 5×10^{-4} cases per person-Gy.

In the event of an accident, it is unlikely that an individual would receive an external dose sufficient to cause acute thyroiditis without receiving lethal doses to the bone marrow, gastrointestinal tract, lungs or central nervous system. Therefore no model was developed for acute radiation thyroiditis following external exposure.

2.1.2.4 Skin Burns

Exposure to ionizing radiation may produce skin burns. Three levels of severity are commonly recognized. Erythema, a reddening of the skin, is equivalent to a first degree thermal burn or sunburn. Transepidermal injury involves blistering and is equivalent to a second degree burn. Although with medical care these blisters normally heal, the new skin is usually pigmented, thin and easily injured. Dermal necrosis is a severe injury involving sloughing of the skin and widespread cell destruction. The lesions resemble those caused by severe scalding and are accompanied by intense pain. Medical attention is necessary.

The doses required to produce these effects are quite large. Individuals receiving whole-body gamma doses large enough to produce skin burns would be almost certain to die from the hematopoietic syndrome. However, skin burns might also occur in individuals who receive relatively large doses to the skin from beta emitting radionuclides. Because of their limited power to penetrate tissue, beta particles can yield large doses to the skin without correspondingly large doses to critical organs such as the bone marrow, lungs, or intestines.

Widespread lesions of the skin were observed among the firemen involved in emergency response at Chernobyl [Gus'kova, 1987]. These burns—which were caused by a combination of intense heat and radiation exposure—were accompanied by large radiation doses to the marrow. Despite intensive medical attention, most of the victims died as a result of the hematopoietic syndrome. Little new information about the human dose-response for radiation-induced burns resulted from this tragedy.

Our models focus on two symptoms—erythema and transepidermal injury—and are based largely on information from studies described in Archambeau's recent [1987] review.

Analysis of the risk of skin burns is complex. In addition to the dose received, the beta energy involved and the area irradiated both strongly influence the likelihood and severity of burns. The parameters recommended by the early effects working group are based on the dose to the basal cells of the skin, i.e., about 0.1 mm below the surface, and are appropriate for estimating the risk of skin burns when areas of about 50 to 100 cm^2 (about the size of the face) have been exposed. The central estimates of the D_{50}'s of 6 Gy for erythema and 20 Gy for transepidermal injury are derived from Lushbaugh's 1986 analysis of the experiences of victims of 250 major radiation accidents—most involving exposure to sealed radioactive sources.

The influence of beta energy was demonstrated over thirty years ago by Moritz and Henriques [1952]. When pig skin, selected for study because of its similarity to human skin, was irradiated by sulfur-35 (maximum energy 0.2 MeV), a surface dose of about 200 Gy was required to induce transepidermal injury 50 percent of the time. In contrast, when

yttrium-91 (maximum energy of 1.5 MeV) was used as a radiation source, a surface dose of only 15 Gy was required to produce the same effect. On the basis of these, and other similar, experimental findings Mortiz and Henriques demonstrated that the dose about 0.1 mm below the surface is a much better index of skin damage—as it accounts for differences in the penetrating ability of various beta sources. There is a biological basis for this result—the basal cells are located approximately 0.1 mm below the skin surface and it is likely that skin damage is caused by injury of the basal cells.

Coggle et al. [1984] and Peel and Hopewell [1984] hypothesize that the dependence of the likelihood and severity of skin damage on the area irradiated is related to the nature of repair processes in the skin, in which repair of injured skin proceeds from the periphery of the irradiated area toward its center. Cohen [1966] and von Essen [1969] demonstrated that the D_{50} for skin effects is inversely proportional to the sixth root of the area irradiated. Following this approach, the D_{50} (Gy) for transepidermal injury would be related to the area irradiated (cm^2) by:

$$D_{50} \approx 40/(area)^{1/6}$$

According to this model, if the entire skin surface—about 2 m^2—were irradiated only about 8 Gy would be required to induce transepidermal injury among half of the exposed population. The basis for this result is quite tenuous—the $-1/6$th power dependence has been demonstrated only for small circular fields (≤ 400 cm^2) irradiated by a specific range of photon energies—but it does suggest that those individuals with large areas of skin exposed may experience skin burns at relatively low doses.

2.1.2.5 Reproductive Effects

The ovary, a relatively radiosensitive organ, contains germ cells. If these cells are severely damaged by radiation, they cannot by replaced. Because the most tangible effects of loss of ovarian function would be felt by those women intending to bear children, and because over 99 percent of all children are born to mothers younger than 40 years of age, our models focus on the effects of radiation on women in this age group.

Our analysis of the effects of radiation on ovarian function is based largely on Damewood and Grochow's 1986 review of ovarian function in patients who had received radiation therapy. No deleterious effects on reproductive function were observed in women who received doses less than 0.6 Gy. Temporary suppression of ovulation was observed in women with doses between 1.5 and 5 Gy. However, doses greater than 8 Gy were required to produce permanent suppression in women under 40.

The working group's central estimate of the population threshold dose required to cause permanent suppression of ovulation is 0.6 Gy. Their upper and lower estimates of the threshold are 1 Gy and 0.2 Gy, respectively. The working group's central estimate of the D_{50} for ovulation suppression is 3.5 Gy with lower and upper estimates of 2.5 and 4.5 Gy.

The testes are quite sensitive to radiation. Doses as small as 0.1 Gy have caused temporary diminution of sperm count. Doses of at least 2 Gy are required to permanently suppress sperm count.

Recovery time is dose dependent and, after large doses, full recovery may not occur for several years. Japanese fishermen who accumulated doses between 1.7 and 6.9 Gy from radioactive fallout over a 2-week period exhibited severe depression of sperm count. However within two years of exposure their sperm counts began to recover and eventually most of them fathered healthy children.

Based largely on studies reviewed by Damewood and Grochow [1986] of patients therapeutically treated with radiation, the early effects working group recommends that central estimates of risks of suppression of sperm count be modeled using a D_{50} of 0.7 Gy, a population threshold of 0.3 Gy, and a shape factor of 10. These parameter values are appropriate for predicting 2-year suppression of sperm count following brief external exposure.

The testes are unusual in that fractionated exposures may lead to greater damage and slower recovery than a single exposure involving the same dose [Lushbaugh and Ricks, 1972].

2.1.2.6 Effects on the Embryo and Fetus

Human evidence for death of the embryo or fetus following irradiation of the pregnant mother is limited. However, in rats and mice lethality has been observed following doses as low as 0.1 Gy given on the first day of gestation. In experimental studies with animals, sensitivity to the effects of radiation is clearly related to the developmental stage of the embryo.

Our models of embryo lethality are based on data reported by Brent et al. [1987]. The early effects working group selected central estimates of the LD_{50} of 1 Gy during preimplantation (0 to 18 days postconception), 1.5 Gy during the period of growth and development (18 to 150 days), and 3 Gy (equal to the mother's LD_{50}) for the remainder of the pregnancy. The central estimates of thresholds for these same periods are 0.1 Gy, 0.4 Gy, and 1.5 Gy, respectively.

Irradiation of the fetus in utero may increase the risk of mental retardation. The children who were irradiated in utero during the bombing of Hiroshima and Nagasaki have been followed carefully. Otake et al. [1987] provide evidence of a dose-related increase in the prevalence

of mental retardation among these children. In Otake's study a child was considered mentally retarded if he was unable to perform simple calculations, to care for himself, or if he was completely unmanageable or had to be institutionalized. Most of the children so classified had never been enrolled in school. The few who had entered school had IQ's below 70. It should be noted that, using these criteria, only 30 cases of mental retardation were found among the approximately 1600 children included in the study.

A key controversy in the interpretation of these data involves the shape of the dose-response function. It is widely believed that the analyses of Otake et al. offer strong support for a linear model with no threshold. However, as Schull [personal communication, 1989] notes,

- "We fitted linear, linear-quadratic, and quadratic [models], with and without thresholds . . . [and] have repeatedly stated that the data on mental retardation . . . are not extensive enough to permit the exclusion of either the linear or linear-quadratic model through a statistical appraisal of fit alone."

- ". . . Otake, Yoshimaru and I have looked carefully at the issue of a threshold both in the 8 to 15 and 16 to 25 week groups. We have argued that if one does exist within the 8 to 15 week group, the lower confidence bound lies between about 0.1 and 0.2 Gy. We have always contended that within 16 to 25 week period, the data . . . are consistent with a threshold somewhere in the neighborhood of 0.5 Gy."

Brent [1986] and other embryologists question the use of linear models and advocate the use of thresholds. Neumeister and Wasser [1985] recommend continuation of pregnancy following doses as large as 0.1 Gy.

Figures 2.1 and 2.2 show the Japanese data on the prevalence of mental retardation in both age groups and contrast the linear and quadratic dose-response models. When models, which are linear at low dose, are fit to the Japanese data—using the DS86 data estimates—one obtains:

$$R_{8\text{-}15 \text{ weeks}} = 1 - \exp[\ -0.0076 - 0.46\ d]$$

$$R_{16\text{-}25 \text{ weeks}} = 1 - \exp[\ -0.0062 - 0.10\ d]$$

where d is the dose (Gy) received by the embryo and R is the prevalence of mental retardation. As would be expected, these data reflect a strong dependence of risk on developmental age—those 8 to 15 weeks old are nearly five times as sensitive as those 16 to 25 weeks old.

For both developmental age groups, quadratic models fit the prevalence data at least as well as the linear models:

$$R_{8\text{-}15 \text{ weeks}} = 1 - \exp[\ -0.0076 - 0.65\ d^2]$$
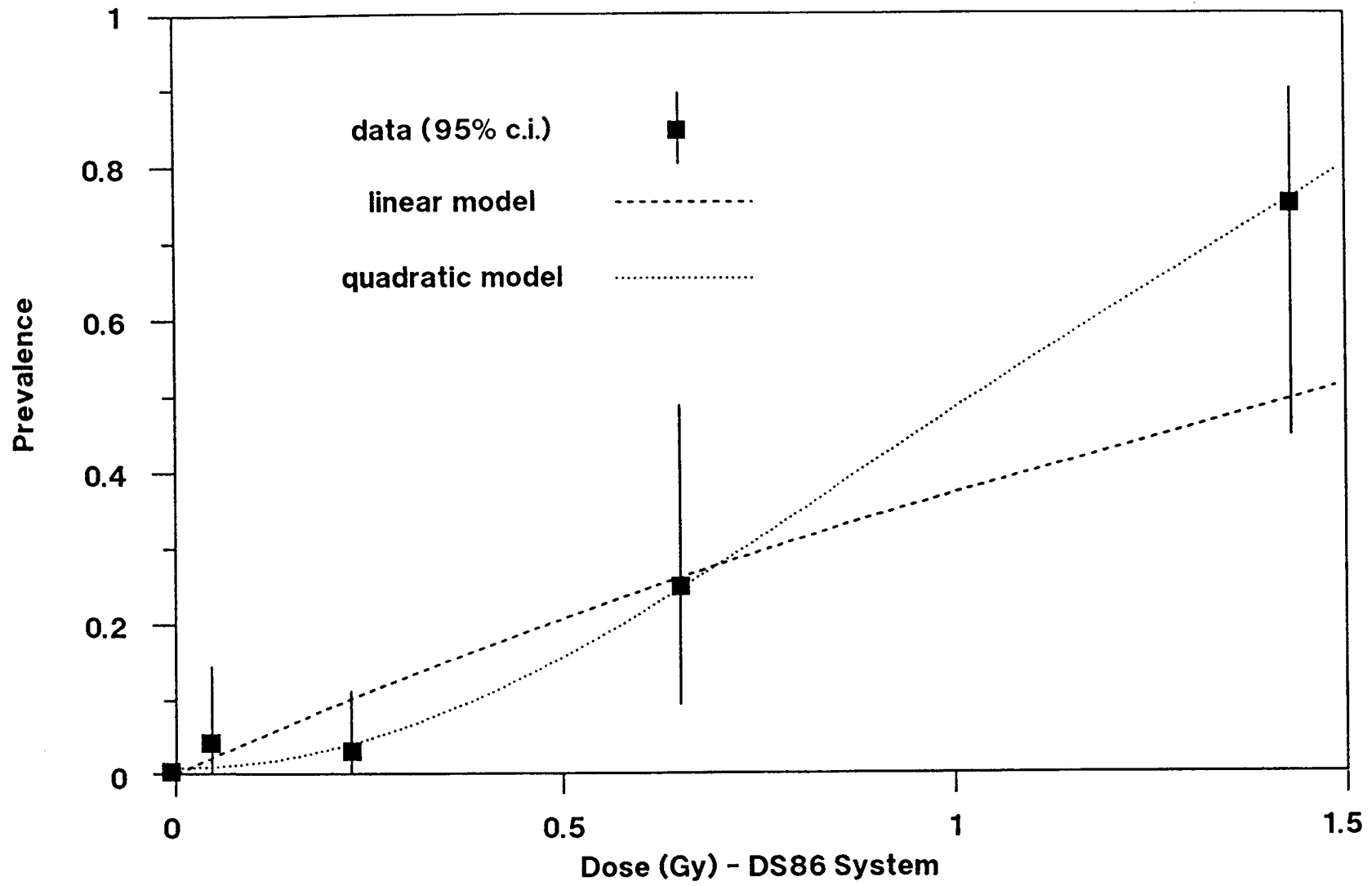
Figure 2.1 Risk of Mental Retardation Among Children
of Atomic Bomb Survivors – 8 to 15 Week Group
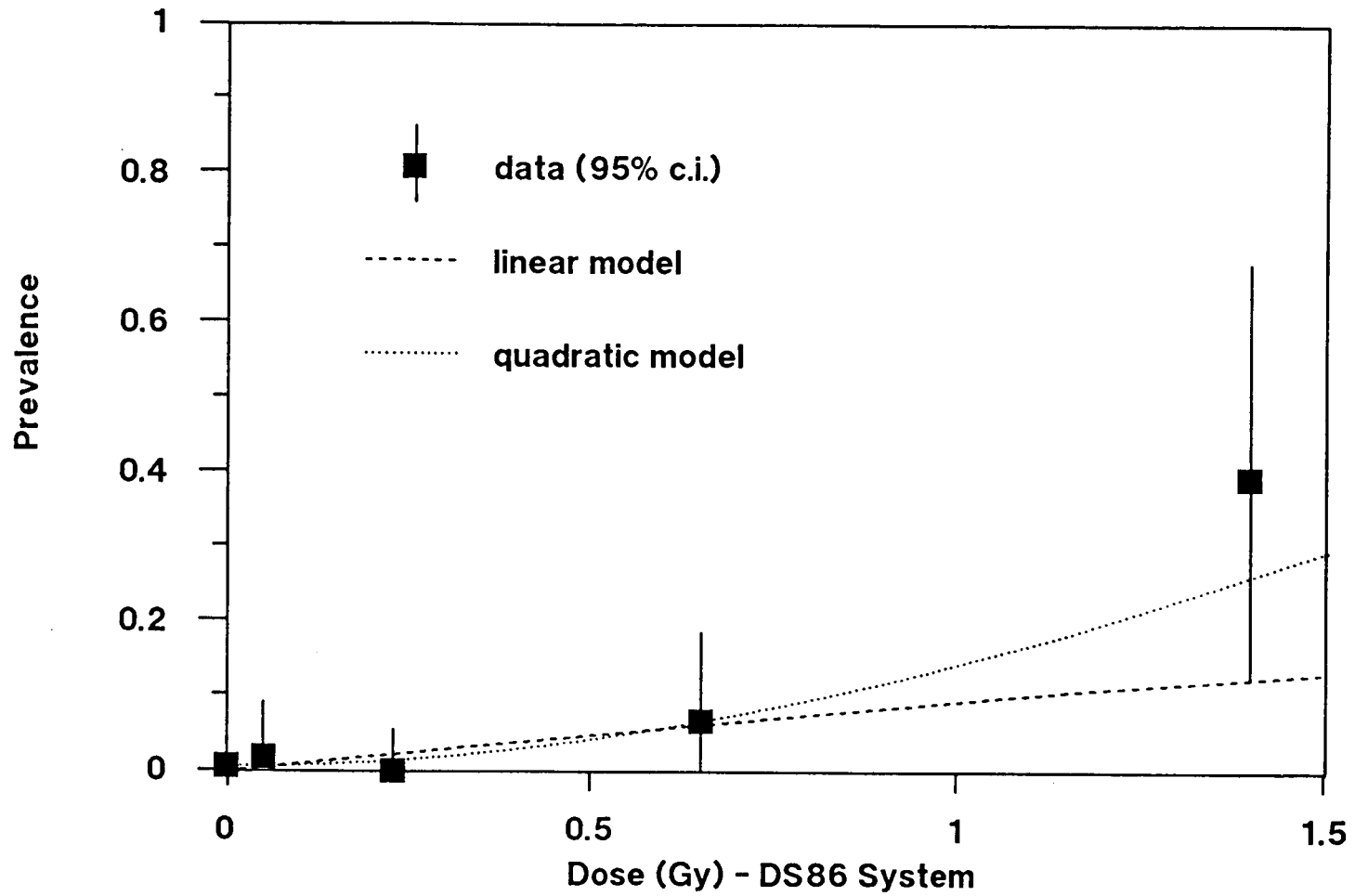
**Figure 2.2   Risk of Mental Retardation Among Children
of Atomic Bomb Survivors – 16 to 25 Week Group**

$$R_{16\text{-}25 \text{ weeks}} = 1 - \exp[\, -0.0062 - 0.15 \; d^2]$$

The early effects working group recommended using linear models (without thresholds) for central, upper and lower estimates of risk. In view of Brent's concern and the ambiguity of the data, we recommend that the lower estimates of the risk of mental retardation incorporate thresholds —specifically 0.1 Gy for those in the 8 to 15 week group and 0.5 Gy for those in the 16 to 25 week group—and that they be based on quadratic models.

Analysts using this approach must be aware that even when no increment in the prevalence of mental retardation is predicted there may still be radiation-induced reductions in the mean IQ of the exposed populations.

2.1.2.7 Summary—Early Morbidity

The parameters recommended for predicting the risks of early morbidity are summarized in Tables 2.4 through 2.6. The values given in Tables 2.4 (general morbidity) and 2.5 (in utero effects) apply to brief external exposures. Those given Table 2.6 reflect the reduced effectiveness of protracted exposures. The early effects working group recommends that exposures at dose rates of 0.05 Gy per hour or less be considered protracted exposures.* The risk from exposures at rates higher than this should be evaluated using parameters appropriate for brief external exposure at high dose rate.

2.2 Late Somatic Effects

Estimates of cancer risks are based primarily on the findings of studies of human populations exposed to ionizing radiation. Examples of such populations include the survivors of the atomic bombings of Hiroshima and Nagasaki, women treated with x-rays for acute postpartum mastitis, children treated by x-ray for ringworm of the scalp, patients treated for ankylosing spondylitis, women given fluoroscopic examinations of the chest, persons treated with ^{131}I for Graves' disease and other thyroid conditions, and children born to women who received x-ray pelvimetry during pregnancy.

Many of these populations were exposed to relatively high doses at high dose rates. Few of the studies are complete; i.e., many of those exposed are still alive. Thus, two key issues in interpretation of these studies are how to extrapolate the results for use in situations involving much lower doses (and dose rates) and how to estimate the impact of incomplete follow-up.

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* The value—0.06 Gy/hr (1 rad/min)—recommended in Part II of this report has been rounded to 0.05 Gy/hr to avoid conveying a false sense of precision.

Table 2.4

Models of Early Morbidity from Brief Exposures[a]

Effect	Central ED$_{50}$	Central T	Central v	Lower[b] ED$_{50}$	Lower[b] T	Lower[b] v	Upper[b] ED$_{50}$	Upper[b] T	Upper[b] v
Prodromal Syndrome									
Vomiting	2	0.5	3	2.5	0.5	3	1.5	0.5	3
Diarrhea	3	1	2.5	4	1	2.5	2.5	1	2.5
Thyroiditis[c]	-	-	-	-	-	-	-	-	-
Hypothyroidism[d]	60	2	-	60	2	-	60	-[e]	-
Erythema	6	3	5	7	4	6	5	2	4
Transepidermal Injury	20	10	5	25	12	6	15	8	4
Reproductive Effects									
Ovulation Suppression	3.5	0.6	3	4.5	1	4	2.5	0.2	2
Suppression of Sperm Count	0.7	0.3	10	0.8	0.4	11	0.6	0.2	9
Cataracts	3	1	2	7	1.5	3	2	0.5	1

[a] Brief exposure parameters are appropriate for dose received at high dose rate. The doses referred to in this table are organ-specific absorbed doses. The units are Gray (Gy). The parameters, D$_{50}$, T and v given in this table are defined in the text of this report (pp. I-9). In some cases, the values recommended by the working group have been rounded to avoid conveying a false sense of precision.

[b] For early effects, use of larger values for LD$_{50}$, T, and v results in the lower estimates of risk, and vice versa.

[c] There is no evidence suggesting that radiation thyroiditis can be induced by brief external exposures.

[d] According to the thyroid working group these parameter values are appropriate for all exposures except internal exposure to ^{131}I. The risk is modeled using a proportional dose response curve, with a slope of 80 cases per 10,000 persons per Gy of brief external dose. See Section 3.1.3 for value of shape factor v.

[e] As explained in the text (pp. I-19), upper estimates of risk should be computed with a threshold much smaller than 2 Gy.

Table 2.5

Models of Early Morbidity from Brief Exposures in utero[a]

Effect	Central			Lower[b]			Upper[b]		
	D_{50}	T	v	D_{50}	T	v	D_{50}	T	v
Microencephaly									
0-17 Weeks	0.7	0.05	0.4	0.8	0.1	1	0.5	0.05	0.2
Severe Mental Retardation									
8-15 Weeks	1.5	0	1	1[c]	0.1[c]	2	1	0	1
16-25 Weeks	7	0	1	2[c]	0.5[c]	2	3	0	1
Death of Embryo or Fetus									
0-18 Days	1	0.1	2	1.5	0.5	2.5	0.5	0	1.5
18-150 Days	1.5	0.4	3	2	0.5	4	1	0.2	2
150-Term[d]	-	-	-	-	-	-	-	-	-

Above columns grouped under heading: **Risk Estimate**

[a] Brief exposure parameters are appropriate for dose received at high dose rate. The doses referred to in this table are doses absorbed by the embryo or fetus. The units are Gray (Gy). The parameters, D_{50}, T and v given in this table are defined in the text of this report (pp. I-9). In some cases, the values recommended by the working group have been rounded to avoid conveying a false sense of precision.

[b] For early effects, use of larger values for LD_{50}, T, and v results in the lower estimates of risk, and vice versa.

[c] A direct comparison of the D_{50} value for the lower risk estimate with the values for the central and upper estimates may lead to an incorrect inference about risk because the lower estimate is based on a quadratic model and the central and upper estimates on linear models.

[d] In this period the fetus and the mother are assumed to have the same radiosensitivity. Parameter values should be selected from Table 2.1 or derived from the dose rate dependent models described in Section 2.1.1.1.

Table 2.6

Models of Early Morbidity from Protracted Exposures[a]

Effect	Central			Lower[b]			Upper[b]		
	ED_{50}	T	v	ED_{50}	T	v	ED_{50}	T	v
Prodromal Syndrome									
Vomiting	5	1.5	3	6	1.5	3	4	1.5	3
Diarrhea	6	2.5	2.5	7.5	2.5	2.5	5	2.5	2.5
Thyroiditis	1200	200	2	1200	200	2	1200	200	2
Hypothyroidism[c]	300	10	-	300	10	-	300	-[d]	-
Erythema	20	6	5	30	8	6	10	4	4
Transepidermal Injury	80	40	5	100	50	6	60	30	4
Reproductive[e] Effects	-	-	-	-	-	-	-	-	-
Cataracts[f]	-	-	-	-	-	-	-	-	-

(Risk Estimate spans the Central, Lower[b], and Upper[b] column groups.)

[a] Protracted exposure parameters are appropriate for dose received at low dose rate (≤0.05 Gy/hr). The doses referred to in this table are organ-specific absorbed doses. The units are Gray (Gy). The parameters, D_{50}, T and v given in this table are defined in the text of this report (pp. I-9). In some cases, the values recommended by the working group have been rounded to avoid conveying a false sense of precision.

[b] For early effects, use of larger values for LD_{50}, T, and v results in the <u>lower</u> estimates of risk, and vice versa.

[c] According to the thyroid working group, these parameter values are appropriate only for internal exposure to [131]I. The risk is modeled using a proportional dose response curve, with a slope of 17 cases per 10,000 persons per Gy of ^{131}I dose.

[d] As explained in the text, upper estimates of risk should be computed with a threshold much smaller than 10 Gy. See Section 3.1.3 for value of shape factor v.

[e] Parameters for protracted exposure were not developed.

[f] Limited evidence suggests that the ED_{50} and threshold values would be five to ten times higher for protracted dose than for brief dose.

To derive central risk estimates for most cancers, the late somatic working group recommends use of a linear-quadratic model of dose-response in which the parameters are chosen so that doses received at low dose rates are 30 percent as effective as equivalent doses received at high rates. This central estimate of the low dose rate effectiveness factor was chosen from a range of values—10 percent to 50 percent—given in NCRP Report 64 [NCRP, 1980]. Many European accident consequence calculation codes rely on a low dose rate effectiveness factor of 45 percent. To reflect the uncertainty in this choice, the working group's lower risk estimates for most cancers are based on an effectiveness factor of 10 percent and their upper estimates assume that doses received at low dose rates are as effective as doses received at high rates.

Computationally, the effects of dose and dose rate are accounted for by using dose-response models of the form:

$$r = (a + bd)\ cd$$

where d is the dose (Gy), c is the unit risk coefficient (cases of cancer or cancer deaths per 1000 persons per Gy) derived from epidemiological studies at high dose and dose rate, a is the low dose rate effectiveness factor (0.3, 0.1 or 1) described above, and b is a parameter selected to ensure that the modifying term, a + bd, equals 1 at a dose of 1.5 Gy.

For doses greater than 1.5 Gy, the modifying term is not applied and the risk is computed using:

$$r = cd$$

Because this model does not incorporate a cell killing term, risks at extremely high doses could be overestimated. This problem is mitigated to some extent in accident consequence analysis codes by computing cancer risks only for those who survive the early effects of radiation.

When doses are received at low dose rates, i.e., less than 0.05 Gy per year, the bd component of the modifying term is dropped and the model becomes:

$$r = acd$$

The two models most commonly used for projecting the impact of incomplete followup are the absolute risk projection model and the relative risk projection model. Both models allow for a latency period—during which there is no radiation-induced cancer risk—and a plateau (or expression) period—during which the effects of exposure to radiation are expressed. The plateau period may be of fixed length, e.g., 25 years, or the risk may be assumed to persist for the remainder of the exposed individual's life. The key difference between the absolute and relative risk projection models is the assumption made with respect to the pattern of radiation-induced risk during the expression period. Under the absolute

risk projection model the risk is assumed to be constant. Under the relative risk projection model the risk is assumed to be a constant fraction of the baseline age-specific risk. Because background rates for most cancers strongly increase with age, the relative risk model tends to yield projections of risk that are higher than those derived using the absolute risk model.

A secondary issue related to the application of the relative risk model is whether the fractional increase in cancer risk associated with a specific dose depends on the age at which the dose is received. Although existing data do not clearly resolve this point, several recent analyses suggest that relative risks decrease with increased age-at-exposure. Therefore while the working group's central estimates of the risks of most of the solid tumors are based on the assumption that relative risks do not depend on age at exposure, their upper estimates for these same cancers assume that relative risks for those under 20 at the time of exposure are about three times as great as for those over 20.

Our models for late somatic effects were developed largely by Dr. Ethel Gilbert of Battelle Pacific Northwest Laboratories with input from Dr. Roy Shore, New York University; Dr. George Hutchison, Harvard School of Public Health; Drs. Gilbert Beebe and Charles Land, National Cancer Institute; Dr. Edward Webster, Massachusetts General Hospital; Dr. Jacob Fabrikant, University of California and Lawrence Berkeley Laboratory; Dr. William Bair, Battelle Pacific Northwest Laboratories; Dr. Marvin Goldman, University of California (Davis); Dr. Warren Sinclair, National Council on Radiation Protection and Measurements; Dr. Edward Radford, Radiation Effects Research Foundation; and Dr. Nan Laird, Harvard School of Public Health. The thyroid effects working group directed by Dr. Harry Maxon of the University of Cincinnati developed the models for thyroid cancers and benign thyroid nodules.

Separate models are provided for estimating the risks of leukemia, bone cancer, breast cancer, lung cancer, gastrointestinal cancers (including cancers of the esophagus, stomach, colon, rectum, pancreas and liver), thyroid cancer and benign thyroid nodules, a residual category of "other" cancers (which is intended to reflect cancers of the bladder, kidney, brain, female reproductive organs and lymphomas), and for both leukemias and other cancers associated with in utero exposure. This site-specific approach was taken because of the non-uniformity of the organ doses that may occur in nuclear power plant accidents.

The working group has provided estimates of both incidence and mortality risks for most cancers. Estimates of the risk of lung, gastrointestinal, and "other" cancers were derived primarily from mortality studies. The estimates of breast and thyroid cancer risk were based largely on incidence data. For lung, breast, gastrointestinal, and other cancers, it was assumed that the relative risks of mortality and incidence were equal; i.e., the same relative risk coefficient (percent increase per Gy) was used to compute incidence and mortality. For thyroid cancer,

mortality risks were taken to be 10 percent of incidence. Risk estimates for leukemia and for cancers resulting from in utero exposure were derived from data collected at a time when these cancers were virtually always fatal. In view of the recent increases in 5-year survival rates for leukemia and other childhood cancers, the estimates of mortality risks for these cancers may be somewhat high.

The models given in the first edition of NUREG/CR-4214 were based primarily on information discussed in BEIR III [1980] and data from the subsequent followup of the survivors of Hiroshima and Nagasaki [Kato and Schull, 1982; Wakabayashi et al., 1983]. Since the first edition of NUREG/CR-4214 was published, the Report of the National Institutes of Health ad hoc Working Group to Develop Radioepidemiological Tables [NIH, 1985] has been issued and new data have become available, including an additional 7 years of follow-up on the Japanese atomic bomb survivors. The models presented below reflect some of the key findings of these more recent analyses; i.e., increased support for the relative risk model and evidence that risks among those who were young at the time of exposure are greater than for those exposed later in life. However, users of these models should be aware that no attempt has been made to speculate about the effects of the reassessment of the doses received by the Japanese survivors. Thus, our models will require reevaluation once the results of the National Academy of Science BEIR V analysis are published.

2.2.1 Leukemia

The working group's central estimate of leukemia risks is based on absolute risk projection with a latency period of two years and an expression period of 25 years. The risk coefficient of 2.2×10^{-4} deaths per person-yr-Gy is taken from the BEIR III analysis of Leukemia Registry data for the Japanese atomic bomb survivors. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 30 percent, is recommended. The resulting estimate of population risk is:

$$R = 1.4\ d + 2.3\ d^2$$

where R is the lifetime population risk (deaths/1000 persons) and d is the dose (Gy) to the red bone marrow.

The upper estimate of leukemia risks is based on similar assumptions, except that a proportional dose response model, without any adjustment for lower effectiveness of doses received at low dose rates, is used. The resulting estimate of population risk is:

$$R = 4.8\ d$$

The lower estimate of leukemia risks relies on the same approach used to derive the central estimate; however, a low dose rate effectiveness

factor of 10 percent is recommended. The resulting estimate of population risk is:

$$R = 0.5\ d + 2.9\ d^2$$

The loss of life expectancy associated with a leukemia death is estimated to be 35 years.

2.2.2 Bone Cancer

The working group's central estimate of bone cancer risks uses absolute risk projection with a latency period of two years and an expression period of 25 years. The low-LET risk coefficient of 1×10^{-5} deaths per person-yr-Gy is based largely on the BEIR III estimate of 1×10^{-4} deaths per person-yr-Gy (alpha) observed among patients given radium 224 injections and on data described in UNSCEAR 77. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 30 percent, is recommended. The resulting estimate of population risk is:

$$R = 0.06\ d + 0.09\ d^2$$

where R is the lifetime population risk (deaths/1000 persons) and d is the dose (Gy) to the bone.

The upper estimate of bone cancer risks is based on similar assumptions, except that a proportional dose response model, without any adjustment for lower effectiveness of doses received at low dose rates, is used. The resulting estimate of population risk is:

$$R = 0.2\ d$$

The lower estimate of bone cancer risks relies on the same approach used to derive the central estimate, however a low dose rate effectiveness factor of 10 percent is recommended. The resulting estimate of population risk is:

$$R = 0.02\ d + 0.12\ d^2$$

The loss of life expectancy associated with a bone cancer death is estimated to be 35 years.

2.2.3 Breast Cancer

The working group's central estimate of breast cancer risks is based on relative risk projection with a latency period of 10 years, a minimum age at induction of 30 years and a lifetime expression period. The risk coefficient was derived from BEIR III [1980] and is based on incidence data from a New York study of women treated with x-rays for acute postpartum mastitis and from a Massachusetts study of women given fluoroscopic examinations of the chest. The age-specific estimates given in BEIR III were pooled, using their inverse variances as weights, to obtain a non-age-specific coefficient of 45 percent per Gy. Because

there is little evidence of decreased effectiveness of dose delivered at low dose rate, a linear dose-response model is recommended. The resulting estimates of population risks are:

$$R_I = 17 \text{ d} \quad \text{and} \quad R_M = 6.0 \text{ d}$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons) and d is the dose (Gy) to the breasts. Note that this estimate, and the upper and lower estimates given below, applies to the entire population. Risks to women are twice this large.

The upper estimate of breast cancer risks is based on similar assumptions, except that the risk coefficient is assumed to depend upon age at exposure. The BEIR III risk coefficients of 103 percent per Gy, for women less than 20 at exposure, and 42 percent per Gy, for those older than 20, are used. The resulting estimates of population risk are:

$$R_I = 25 \text{ d} \quad \text{and} \quad R_M = 8.7 \text{ d}$$

The lower estimate of breast cancer risks is based on absolute risk projection with a latency of 10 years and a lifetime expression period. Risk coefficients of 7.4×10^{-4} cases per woman-yr-Gy and 2.6×10^{-4} deaths per woman-yr-Gy are recommended. The incidence estimate is the value derived from the pooled analysis previously described. The mortality coefficient was obtained by multiplying this estimate of incidence by the ratio of background mortality to background incidence. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 10 percent, is used. The resulting estimates of population risk are:

$$R_I = 1.2 \text{ d} + 7.4 \text{ d}^2$$

$$R_M = 0.4 \text{ d} + 2.4 \text{ d}^2$$

The loss of life expectancy associated with a breast cancer death is estimated to be 17 years under the assumptions used in the central and upper models and 23 years under the assumptions used in the lower model. The average interval between diagnosis of a case and death is estimated to be between 12 and 15 years depending upon which risk model is used.

2.2.4 Lung Cancer

The working group's central estimate of lung cancer risks is based on a relative risk projection with a latency period of 10 years, a minimum age at induction of 40 years and a lifetime expression period. The risk coefficient of 18 percent per Gy was obtained by expressing an estimate of the absolute risk observed in the Japanese Life Span Study, 2.0×10^{-4}

deaths per person-yr-Gy, as a percentage of the background lung cancer risk that would have been expected to occur in a U.S. population followed for a similar period. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 30 percent, is recommended. The resulting estimates of population risks are:

$$R_I = 2.2 \ d + 3.5 \ d^2$$

$$R_M = 2.0 \ d + 3.1 \ d^2$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons) and d is the dose (Gy) to the lungs.

The upper estimate of lung cancer risks is based on similar assumptions, except that a larger risk coefficient is used and that relative risk is assumed to depend upon age at exposure. For persons less than 20 years of age at the time of exposure, a risk coefficient of 111 percent per Gy is used. For those older than 20, a risk coefficient of 37 percent per Gy is used. The estimate of 37 percent per Gy was obtained by direct analysis of the relative risk observed in the Japanese Life Span Study. This direct relative risk estimate is approximately twice as large as the estimate derived above because the background rates of lung cancer in Japan are only about half those observed in the U.S. The use of a rela- tive risk coefficient three times as large for those under 20 as for those over 20 is consistent with the results of Preston and Pierce [1987]. The resulting estimates of population risk are:

$$R_I = 27 \ d$$

$$R_M = 25 \ d$$

The lower estimate of lung cancer risks is based on absolute risk pro- jection with a latency of 10 years and a lifetime expression period. Risk coefficients of 2.2×10^{-4} cases and 2.0×10^{-4} deaths per person-yr- Gy are recommended. The mortality coefficient is the absolute risk value derived from analysis of the Japanese Life Span Study. The incidence coefficient was obtained by scaling this value by the ratio of background incidence to background mortality. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 10 percent, is recommended. The resulting estimates of population risk are:

$$R_I = 0.6 \ d + 3.5 \ d^2$$

$$R_M = 0.5 \ d + 3.0 \ d^2$$

The loss of life expectancy associated with a lung cancer death is estimated to be 14 to 15 years under the assumptions used in the central and upper models and 19 years under the assumptions used in the lower model. The average interval between diagnosis of a case and death is estimated to be about 2 years regardless of which risk model is used.

2.2.5 Gastrointestinal Cancer

The working group's central estimate of gastrointestinal cancer risks is based on a relative risk projection with a latency period of 10 years and a lifetime expression period. The risk coefficient of 39 percent per Gy was obtained by expressing an estimate of the absolute risk observed among the Japanese, 2.7×10^{-4} deaths per person-yr-Gy, as a percentage of the background gastrointestinal cancer risk that would have been expected to occur in a U.S. population followed for a similar period.

The absolute risk coefficient used in this computation is not identical to that obtained directly from the Japanese Life Span Study, 1.8×10^{-4}. It reflects a synthesis of direct estimates of the risks of cancer of the esophagus, stomach, colon and "other and unspecified sites" from the Life Span Study with estimates of the risks of rectal and pancreatic cancer derived from other studies. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 30 percent, is recommended. The resulting estimates of population risks are:

$$R_I = 9.7 \, d + 15 \, d^2$$

$$R_M = 5.7 \, d + 8.9 \, d^2$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons) and d is the dose (Gy) to the lower large intestine.

The upper estimate of gastrointestinal cancer risks is based on similar assumptions, except that the risk coefficient is assumed to depend upon age at exposure. For persons less that 20 at exposure, a risk coefficient of 117 percent per Gy is used. For those older than 20, a risk coefficient of 39 percent per Gy is used. Furthermore, a proportional dose response function, without any adjustment for lower effectiveness of dose received at low dose rate, is used. The resulting estimates of population risk are:

$$R_I = 56 \, d \quad \text{and} \quad R_M = 33 \, d$$

The lower estimate of gastrointestinal cancer risks is based on absolute risk projection with a latency of 10 years and a lifetime expression period. Risk coefficients of 4.6×10^{-4} cases and 2.7×10^{-4} deaths per person-yr-Gy are used. The mortality coefficient is the synthetic absolute risk estimate previously discussed. The incidence coefficient was obtained by scaling this value by the ratio of background incidence

to background mortality. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 10 percent, is recommended. The resulting estimates of population risk are:

$$R_I = 1.6 \ d + 9.4 \ d^2$$

$$R_M = 0.9 \ d + 5.4 \ d^2$$

The loss of life expectancy associated with a gastrointestinal cancer death is estimated to be 12 years under the assumptions used in the central and upper models and 24 years under the assumptions used in the lower model. The average interval between diagnosis of a case and death is estimated to be between 5 and 10 years depending upon which model of risk is used.

2.2.6 Thyroid Cancers and Benign Thyroid Nodules

Our estimates of thyroid cancer risks are based on absolute risk projection with a latency period of 5 years and a lifetime expression period. Age and sex specific risk coefficients are used. The bases of these coefficients are: the attributable risk of 2.5×10^{-4} cases per person-yr-Gy observed in persons exposed during childhood to external irradiation, the evidence that females are about twice as sensitive as males, and the observation that adult exposure carries less risk (no more than half) than childhood exposure. A linear dose response model is recommended. Our estimates of mortality risks associated with thyroid cancer assume that 10 percent of all radiation-induced thyroid cancers would be fatal. The resulting estimates of population risk are:

$$R_I = 7.2 \ d \qquad \text{and} \qquad R_M = 0.7 \ d$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons) and d is the dose (Gy) to the thyroid gland from external irradiation.

Studies of thyroid cancer following exposure to ^{131}I have been largely negative, but have not had sufficient statistical power to conclusively demonstrate inconsistency with the results from studies of external exposure [Laird, 1987]. In reflection of this, our upper estimates assume that the risk from ^{131}I is equal to the risk from external irradiation. Our central estimates assume that dose from ^{131}I is 1/3rd as potent as dose from external irradiation, and our lower estimates assume that it is 1/10th as potent.

Our estimate of the risk of benign thyroid nodules is based on similar assumptions. Absolute risk projection is used with a latency period of 10 years and a lifetime expression interval. Age and sex specific absolute risk coefficients are recommended. These reflect increased sensitivity (2x) of women, increased sensitivity of those young at

exposure (2x), and are ultimately based on the attributable risk of 9.3 x 10^{-4} benign thyroid nodules per person-yr-Gy observed among persons exposed in childhood to external irradiation. A proportional dose response model is used. The resulting estimate of population risk is:

$$R_I = 27\ d$$

where R_I is the lifetime incidence risk (cases/1000 persons) and d is the dose (Gy) to the thyroid gland from external irradiation. Doses from internal sources, such as ^{131}I, are thought to be only 1/5th as effective as doses from external sources.

2.2.7 Other Cancers

There is reasonably good evidence that multiple myeloma and cancers of the bladder, kidney and brain may be induced by radiation. The evidence is somewhat weaker for lymphoma and cancers of the ovary, uterus and cervix uteri. Rather than developing site-specific risk estimates for each of these cancers, the working group developed a lumped model for "other cancers."

BEIR III [1980] gave site-specific estimates of 0.6 x 10^{-4} deaths per person-yr-Gy for urinary cancers, 0.3 x 10^{-4} deaths per person-yr-Gy for lymphoma and 1 x 10^{-4} deaths per person-yr-Gy as a residual value reflecting other cancers. More recent analysis by Kato and Schull [1982] suggests that these estimates may be too high. We have used an estimate of 1.5 x 10^{-4} deaths per person-yr-Gy to reflect all other radiation-induced cancers.

The working group's central estimate of the risk of other cancers is based on relative risk projection with a latency period of 10 years and a lifetime expression period. The risk coefficient of 20 percent per Gy was obtained by expressing the estimate of absolute risk, 1.5 x 10^{-4} deaths per person-yr-Gy, as a percentage of the background cancer risk that would have been expected in a U.S. population followed for a similar period. The background rate used in this calculation was obtained by subtracting the rates of cancers for which site-specific models had been developed—leukemia, bone, breast, lung, gastrointestinal—and cancers not thought to be radiation-induced—e.g., prostate—from the spontaneous rates of all cancers. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 30 percent, is recommended. The resulting estimates of population risks are:

$$R_I = 5.6\ d + 8.8\ d^2$$

$$R_M = 2.9\ d + 4.5\ d^2$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons) and d is the dose (Gy).

Selection of an appropriate measure of dose to use in the calculation of other cancer risks is difficult because the composition of the group of cancers included is not known exactly and the relative sensitivities of the organs nominally included are not known. The working group suggests that a composite of the doses to the bone marrow, kidney, urinary bladder, brain, uterus and ovary be used. Weights, proportional to the background incidence of the cancers associated with each of these organs, could be used in constructing the composite dose. The resulting dose estimate, based on the 1980 background cancer rates, would be:

$$D_{other} = 0.06 \ D_{bone} + 0.11 \ D_{kidney} + 0.26 \ D_{bladder}$$

$$+ \ 0.09 \ D_{brain} + 0.48 \ D_{ovary}$$

The upper estimate of other cancer risks is based on similar assumptions, except that the risk coefficient is assumed to depend upon age at exposure. For persons less that 20 at exposure a risk coefficient of 60 percent per Gy is used. For those older than 20 a risk coefficient of 20 percent per Gy is used. Furthermore, a proportional dose response function, without any adjustment for lower effectiveness of dose received at low dose rate, is used. The resulting estimates of population risk are:

$$R_I = 34 \ d \quad \text{and} \quad R_M = 17 \ d$$

The lower estimate of other cancer risks is based on absolute risk projection with a latency of 10 years and a lifetime expression period. Risk coefficients of 2.9×10^{-4} cases and 1.5×10^{-4} deaths per person-yr-Gy are used. The origin of the mortality coefficient has already been explained. The incidence coefficient was obtained by scaling this value by the ratio of background incidence to background mortality. A linear-quadratic dose response model, with a low dose rate effectiveness factor of 10 percent, is recommended. The resulting estimates of population risk are:

$$R_I = 1.0 \ d + 5.9 \ d^2$$

$$R_M = 0.5 \ d + 3.0 \ d^2$$

The loss of life expectancy associated with a death from other cancers is estimated to be 13 to 14 years under the assumptions used in the central and upper models and 25 years under the assumptions used in the lower model. The average interval between diagnosis of a case and death is estimated to be between 8 and 12 years depending upon which model of risk is used.

2.2.8 Childhood Cancers from In Utero Exposures

The working group's upper estimates of childhood cancers from in utero exposures are based on the results of the Oxford Survey of Childhood Cancer [Stewart and Kneale, 1968]. The Oxford Survey, which examined the rates of childhood cancers among children of women who had received x-ray pelvimetry during pregnancy, found approximately 3×10^{-2} leukemias and 3×10^{-2} other childhood cancers per embryo per Gy. If, as is now true in the U.S., it is assumed that there is approximately 1 viable embryo for each 100 persons in the population, then the resulting estimates of population risks are:

$$R_{leukemia} = 0.3 \ d \qquad\qquad R_{other \ childhood \ cancer} = 0.3 \ d$$

where d is the dose (Gy) to the fetus and R is the risk (childhood cancers/1000 exposed persons). Note that these expressions apply to the entire exposed population rather than to the number of pregnant women in the population.

It should be noted that no excess cancer deaths have been observed among those exposed in utero during the bombings of Hiroshima and Nagasaki and that this finding is inconsistent with the risks found in the Oxford survey [Jablon and Kato, 1970]. Furthermore, a number of biases may have increased the risk attributed to radiation in the Oxford Survey.

The central (and lower) estimates of childhood cancers from in utero exposures are based on the UNSCEAR72 estimate of 2.3×10^{-2} total childhood cancers per embryo per Gy. This estimate, which includes both leukemias and other childhood cancers, was not modified in the subsequent UNSCEAR reports, UNSCEAR77 and UNSCEAR86. It is about 40 percent as large as the value derived directly from the Oxford Survey.

The studies upon which the risk coefficients are based have involved external irradiation of the pregnant mother and therefore essentially uniform dose to the fetus. In the event of a nuclear power plant accident some of the dose to the fetus would come from external irradiation of the mother but some would come from radionuclides inhaled or ingested by the mother. The doses to the various fetal organs from these internal sources could be quite nonuniform. To account for this, Dr. Keith Eckerman of Oak Ridge National Laboratory recommends that the following dose estimates be used:

$$
\begin{aligned}
d_{fetal \ bone \ marrow} = \ &0.3 \ d_{mother's \ bone \ marrow, \ strontium} \\
&+ 0.5 \ d_{mother's \ uterus, \ cesium} \\
&+ 0.05 \ d_{mother's \ thyroid, \ iodine} \\
&+ 0.5 \ d_{mother's \ maximum \ organ \ dose, \ other \ radioisotopes} \\
&+ 1.0 \ d_{mother's \ uterus, \ external \ sources}
\end{aligned}
$$

$$d_{fetus, other\ organs} = 0.03\ d_{mother's\ bone\ marrow,\ strontium}$$

$$+\ 0.5\ d_{mother's\ uterus,\ cesium}$$

$$+\ 0.05\ d_{mother's\ thyroid,\ iodine}$$

$$+\ 0.5\ d_{mother's\ maximum\ organ\ dose,\ other\ radioisotopes}$$

$$+\ 1.0\ d_{mother's\ uterus,\ external\ sources}$$

2.2.9 Skin Cancer

Most skin cancers are not lethal and therefore skin cancer is not expected to be a major contributor to the mortality resulting from nuclear power plant accidents. However, beta emitting radionuclides deposited on the skin can yield extremely high local doses and can lead to an increased incidence of skin cancer.

Estimation of the risk of skin cancer following a nuclear power plant accident is quite difficult. Most studies of radiation-induced skin cancer have involved exposures to x-rays. The importance of the differences in penetrating power of beta emitting radionuclides and x-rays is uncertain. Exposure to ultraviolet radiation seems to potentiate the effect and therefore various areas of the body may have quite different apparent sensitivities to the effects of ionizing radiation. There are also racial differences in sensitivity. Because most skin cancers are not lethal, they are not reliably reported in tumor registries. Available epidemiological results are quite variable and include a number of largely negative studies. Available data are not adequate to determine the shape of the dose-response, the latency, or the effect of age-at-exposure.

The working group's upper estimate of skin cancer risks is based on absolute risk projection with a latency period of ten years and a lifetime expression period. The risk coefficient of 2.0×10^{-4} cases per person-yr-Gy is consistent with data from a study of persons treated as children with x-rays for ringworm of the scalp [Shore et al., 1984]. Risk is assumed to be proportional to dose. The resulting estimate of population risk is:

$$R_I = 6.7\ d$$

where R_I is the lifetime population risk (persons with skin cancer/1000 persons) and d is the dose (Gy) to the skin. Note that this model predicts the number of people with skin cancer, not the total number of skin cancers.

Central and lower estimates are based on similar assumptions, but use low dose rate effectiveness factors of 30 and 10 percent, respectively.

The late somatic effects working group recommends that risk calculations be made on the basis of dose to the face because about 85 percent of basal cell carcinomas (the predominant type resulting from ionizing radiation) occur on the head and neck and because in the event of a nuclear power plant accident the areas of the body with the highest exposure from beta emitting radionuclides would be those least protected by clothing (such as the face). The risk of skin cancers on other parts of the body would presumably be lower than the risk calculated in this manner.

2.2.10 Summary—Late Somatic Effects

The models recommended for predicting the risks of cancer as a result of doses received in a nuclear power plant accident are summarized in Table 2.7 (morbidity) and Table 2.8 (mortality).

2.3 Genetic Effects

A slight increase in the incidence of genetic disease would be expected to occur after a nuclear power plant accident. The genetic risk would be expressed both directly—i.e., as an increased incidence of birth defects among the children of the exposed population—and indirectly—i.e., through latent mutations that will be expressed in their grandchildren, greatgrandchildren and subsequent generations. In addition, there would be small increases in the rates of pregnancy loss, primarily occurring within the first few days of pregnancy before the fertilized ovum is implanted in the wall of the uterus.

Estimates of genetic risks are based on extrapolations from animal models. The limited human data relevant for genetic risk assessment comes from studies of the children of survivors of the atomic bombings of Hiroshima and Nagasaki. Although these studies have not revealed any excess incidence of genetic defects they are not powerful enough to reject current theories of genetic risk.

The responses observed in the spermatogonial cells of the mouse serve as an indicator of the effects that would be expected to occur in spermatogonial cells of men. Unfortunately, there appears to be no adequate mammalian model of the effects expected in the human female. The working group's central and upper estimates of risk are based on the assumption that damage to oocytes and spermatogonia is equivalent. Their lower estimates are derived on the assumption—used in many previous models—that only spermatogonia are damaged by ionizing radiation.

The possible effects are too numerous to be considered individually. Models of major classes of genetic disease have been developed which reflect the key differences in radiation-induction, significance and transmission of these conditions. The three major classes of genetic disease considered in this report are single-gene disorders, chromosome anomalies and multifactorial diseases. In addition, the risk of recessive genetic disease is discussed.

Table 2.7

Models of Cancer Morbidity[a]

Effect	Lifetime Risk (cases/1000)		
	Central	Lower	Upper
Breast Cancer[b]	17 d	$1.2\ d + 7.4\ d^2$	25 d
Lung Cancer	$2.2\ d + 3.5\ d^2$	$0.6\ d + 3.5\ d^2$	27 d
GI Cancer	$9.7\ d + 15\ d^2$	$1.6\ d + 9.4\ d^2$	56 d
Thyroid Cancer[c]	7.2 d	7.2 d	7.2 d
Benign Thyroid Nodules[d]	27 d	27 d	27 d
Skin Cancer	$2.0\ d + 3.1\ d^2$	$0.7\ d + 4.0\ d^2$	6.7 d
Other Cancer[e]	$5.6\ d + 8.8\ d^2$	$1.0\ d + 5.9\ d^2$	34 d

[a] The doses, d, referred to in this table are organ-specific absorbed doses. The units of dose are Gray (Gy). Refer to the text for explanation of the organ dose appropriate for estimating the risk of each specific cancer.

[b] These risks apply to the entire population. Risks for women would be twice this large.

[c] Uncertainty in the thyroid cancer model is reflected in the dose used. For the central estimate ^{131}I is assumed to be one third as effective as external dose. For the lower estimate ^{131}I is assumed to be one tenth as effective as external dose. For the upper estimate ^{131}I is assumed to be as effective as external dose.

[d] In all three estimates of the risk of benign thyroid nodules, ^{131}I is assumed to be only one-fifth as effective as external dose.

[e] Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

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Table 2.8

Models of Cancer Mortality[a]

Effect	Lifetime Risk (deaths/1000)		
	Central	Lower	Upper
Leukemia	$1.4\ d + 2.3\ d^2$	$0.5\ d + 2.9\ d^2$	$4.8\ d$
in utero[b]	$0.12\ d$	$0.12\ d$	$0.3\ d$
Bone Cancer	$0.06\ d + 0.09\ d^2$	$0.02\ d + 0.12\ d^2$	$0.2\ d$
Breast Cancer[c]	$6.0\ d$	$0.4\ d + 2.4\ d^2$	$8.7\ d$
Lung Cancer	$2.0\ d + 3.1\ d^2$	$0.5\ d + 3.0\ d^2$	$25\ d$
GI Cancer	$5.7\ d + 8.9\ d^2$	$0.9\ d + 5.4\ d^2$	$33\ d$
Thyroid Cancer[d]	$0.7\ d$	$0.7\ d$	$0.7\ d$
Other Cancer[e]	$2.9\ d + 4.5\ d^2$	$0.5\ d + 3.0\ d^2$	$17\ d$
in utero[b]	$0.12\ d$	$0.12\ d$	$0.3\ d$

[a] The doses, d, referred to in this table are organ-specific absorbed doses. The units of dose are Gray (Gy). Refer to the text for explanation of the organ dose appropriate for estimating the risk of each specific cancer.

[b] These risks apply to the entire population. Risks to the children exposed in utero would be 100 times this large.

[c] These risks apply to the entire population. Risks for women would be twice this large.

[d] Uncertainty in the thyroid cancer model is reflected in the dose used. For the central estimate ^{131}I is assumed to be one third as effective as external dose. For the lower estimate ^{131}I is assumed to be one tenth as effective as external dose. For the upper estimate ^{131}I is assumed to be as effective as external dose.

[e] Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

Our models for genetic effects were developed by a working group led by Dr. Seymour Abrahamson of the University of Wisconsin and including Dr. Michael Bender, Brookhaven National Laboratory; Dr. William J. Schull, University of Texas; and Dr. Carter Denniston, University of Wisconsin.

The genetic effects working group relied heavily on analyses provided in the BEIR I and BEIR III, reports by the National Academy of Sciences, as well as those described in recent reports of the United Nations Scientific Committee on the Effects of Atomic Radiation [UNSCEAR 1977, 1982]. These basic approaches have been modified in several important respects to reflect new scientific information and improvements in analytic methodologies for modeling genetic risks.

When dose is received at low dose rate, the risk of genetic damage is thought to be proportional to the dose received. However evidence from many different experimental studies—i.e., Drosophilia oogonia mutations, Tradescantia mutations—indicates that when dose is received at high dose rate the yield of mutations expected at a specific dose is better described by a linear-quadratic relationship. Such a result is consistent with radiobiological understanding of the mechanism of damage—i.e., that the majority of radiation-induced mutations in higher organisms are tiny submicroscopic deletions, inversions or insertions encompassing parts of one or more genes; single nucleotide changes appear to be extremely rare.

When a linear-quadratic relationship—e.g., $(a + bd)$ cd—is fit to the data on specific locus recessive mutations in the spermatogonia of mice—with dose expressed in Gy—the coefficients a and b are found to be virtually identical. The working group has used this result as the basis for estimating the risk of most genetic effects using models of the form:

$$r = (1 + d) \; acd$$

where d is the gonadal dose (Gy), the product ac is the risk coefficient observed at low dose rates, and the term $1 + d$ modifies the risk to account for the effects of high dose rates. When an accident scenario involves only chronic exposures at low dose rates, the modifying term is dropped and the risk is computed as:

$$r = acd$$

This simplification may also be used when dose is received at high dose rate as long as the total dose involved is reasonably small. For example, at a dose of 0.5 Gy the risk would be underestimated by only 50 percent using this simplification. For lower doses, the bias in the estimate is even smaller and is negligible in comparison with the uncertainty in current estimates of the fundamental risk coefficients. It should be noted that the equations given above and those given later in this section assume that the doses received by the mother and father are equal. The modifications necessary to allow for differences in the maternal and paternal doses are discussed in Section 3.3.

The models developed here permit one to estimate the fraction of children born in the first (or any subsequent) generation following an accident that will be affected by each class of genetic disease. In addition, they provide estimates of the total number of children in all future generations that will suffer from genetic disease as a result of radiation exposure from an accident.

The estimates of cumulative genetic risks developed by the working group assume population stability, an intergenerational interval of 30 years and a crude birthrate of about 16 births per 1000 persons per year (500 births per 1000 persons per generation). Were the population to increase (decrease) the absolute impact, i.e., number of effects, would increase (decrease) accordingly.

2.3.1 Single Gene Disorders

Single gene disorders are present in about 1 percent of all children. This class of diseases includes both dominant traits—e.g., Huntington's chorea, hypercholesterolemia and achondroplastic dwarfism—and x-linked traits—e.g., muscular dystrophy, hemophilia, and agammaglobulinemia. Some of these disorders are apparent at birth, but others do not appear until later in life.

Genetic information is encoded within the nucleus of the cell in the form of sequences of deoxyribose nucleic acid called genes. Each of the several thousand human genes is composed of thousands of subunits called nucleotides. The alteration of any nucleotide may result in altered function of a gene and to an observable mutation when contributed by the germ cell of a parent. This single gene mutation is called dominant when it exerts an effect in the presence of a normal gene contributed by the other parent. If an altered gene is present on the X chromosome, it will invariably produce an effect in boys—who have only one X chromosome—but will behave as if recessive in girls—who have two X chromosomes. Single gene disorders related to damage of the X chromosome are referred to as x-linked effects.

The genetic effects working group derived their estimates of the risks of dominant disorders from the Selbys' [1977] studies of the rates of specific locus recessive mutations in male mice. Adjustments to the experimentally observed single locus mutation rate (37/2646 at a dose of 6 Gy) were made to account for the total number of dominant disorders (5 to 15), the fraction of these thought to produce serious diseases (1/4 to 3/4), and to adjust for the dose, dose rate and fractionation involved in the experiments. The resulting central estimate of the induction rate of dominant disorders in humans was 1.5×10^{-3} per gamete (ovum or mature sperm) per Gy. Upper and lower estimates of 4.5×10^{-3} per gamete (ovum or mature sperm) and 0.5×10^{-3} (sperm only) reflect uncertainties in the number of dominant disorders, their seriousness, and the relative sensitivities of male and female gametes. The working group also estimated that approximately 80 percent of dominant disorders are transmitted from one generation to the next.

Based on these considerations, the final models of integrated risk were derived:

$$R_{\text{dominant, central}} = 7.5\ d + 7.5\ d^2$$

$$R_{\text{dominant, upper}} = 22\ d + 22\ d^2$$

$$R_{\text{dominant, lower}} = 1.2\ d + 1.2\ d^2$$

where R is the cumulative risk (dominant disorders/1000 exposed persons) —i.e., the risk that a child with a radiation-induced dominant disorder will be born in this or any future generation—and d is the gonadal dose (Gy) received by a representative individual in the exposed population.

The fraction of cumulative risk that will be expressed in each generation is $0.2 * 0.8^{k-1}$ where k is the generation number. Thus 20 percent of the risk will be expressed in the first generation, 16 percent in the second, and so forth. Under the central model an acute dose of 1 Gy would yield a first generation radiation-induced risk of dominant disorders of approximately 6 defects per 1000 births or 3 defects per thousand exposed persons. The upper estimates would be three times this large and the lower estimates would be 1/6th this large.

The working group estimated that dominant disorders involve, on average, a 15-year reduction in longevity and 25 years of life with approximately 33 percent impairment. Thus the total effective loss of life associated with such a defect is equivalent to about 20 years.

The genetic effects working group derived their estimates of the risks of x-linked disorders from estimates of the rates of specific locus mutations in male mice. The specific locus induction rate of 7.2×10^{-6} per Gy was adjusted to reflect the total number of x-linked diseases. McKusick's compendium lists 115 x-linked diseases and an almost equivalent number of genetic diseases of less certain origin. In view of this, the genetic effects working group multiplied the specific locus mutation rate by 250. The resulting central estimate of the induction rate of x-linked disorders in humans was 1.8×10^{-3} per gamete (ovum or mature sperm) per Gy. Upper and lower estimates of 7.2×10^{-3} per gamete (ovum or mature sperm) and 0.7×10^{-3} (sperm only) reflect uncertainties both in the relative susceptibility of spermatogonia and oocytes and in the number of susceptible genes on the X chromosome. The upper estimate assumes that there are 1000 such loci; the lower assumes that there are only 100.

Based on these considerations, the final models of integrated risk were derived:

$$R_{\text{x-linked, central}} = 2.2\ d + 2.2\ d^2$$

$$R_{\text{x-linked, upper}} = 9.0\ d + 9.0\ d^2$$

$$R_{\text{x-linked, lower}} = 0.45\ d + 0.45\ d^2$$

where R is the cumulative risk (x-linked disorders/1000 exposed persons) —i.e., the risk that a boy with an x-linked disorder will be born in this or any future generation—and d is the gonadal dose (Gy) received by a representative individual in the exposed population.

The fraction of cumulative risk that will be expressed in each generation is $0.2 * 0.8^{k-1}$ where k is the generation number. Thus 20 percent of the risk will be expressed in the first generation, 16 percent in the second, and so forth. Under the central model an acute dose of 1 Gy would yield a first generation radiation-induced risk of x-linked disorders of approximately 2 defects per 1000 births or 1 defect per thousand exposed persons. The upper estimate would be four times this large. In deriving the lower estimate it is assumed that there is no damage to the oocytes. Because boys inherit their X chromosome from their mother, the lower estimate of first generation risk is zero. In subsequent generations boys can inherit a damaged X chromosome from their grandfathers. Thus the lower estimate of the cumulative risk of x-linked effects is not zero; it is 1/5th of the central estimate.

The working group estimated that x-linked disorders involve, on average, a 30-year reduction in longevity and 40 years of life with approximately 40 percent impairment. Thus the total effective loss of life associated with such a defect is equivalent to about 45 years.

2.3.2 Chromosomal Aberrations

A specific alignment of genes, usually several hundred or more, exists on a structure known as a chromosome. Most somatic cells in humans contain 23 pairs of chromosomes—with one member of each pair contributed by the sperm and the other contributed by the egg.

When the process of sperm or egg cell production goes awry it can produce germ cells with the wrong number of chromosomes—e.g., 22 or 24 rather than the normal 23. In this case, the fertilized egg will contain 45 or 47 chromosomes. Such a problem—referred to as aneuploidy—is so severe that in about 90 percent of all cases it will result in a spontaneous loss of pregnancy. In the remaining 10 percent of cases a severely affected child will be born.

Chromosomes are also susceptible to breakage and subsequent structural rearrangement. When rearrangements occur in germ cells they can be transmitted to the offspring of those exposed. These structural rearrangements—referred to as translocations—normally yield chromosomes with either too little or too much genetic information. If a child is born with a balanced translocation, he or she normally will not be affected by it but may transmit it to future generations. However, those children born with unbalanced translocations generally suffer from severe physical and mental disabilities.

The normal incidence of chromosomal aberrations—including both aneuploidy and unbalanced translocations—is approximately 0.6 percent. Conditions such as Down syndrome and both Klinefelter and Turner anomalies are the result of aneuploidy. The spontaneous prevalence of aneuploidy is about 0.5 percent. These defects are relatively severe—both in terms of life expectancy (about 25 years) and level of disability (about 50 percent). Aneuploids normally do not have children. Thus, these defects tend to be completely expressed in one generation.

Because human studies have been equivocal and mammalian (mice) studies have been negative, the BEIR III committee refrained from developing a risk estimate for radiation-induced aneuploidy. Although our genetic effects working group acknowledges that zero is a reasonable lower estimate, they recommend that 1 case per 1000 births per Gy be used as a central estimate and believe that an upper estimate of 3 cases per 1000 births per Gy is plausible. The risk of aneuploidy is assumed to be proportional to dose.

Unbalanced translocations, which result in extremely severe physical and mental disabilities, are naturally present in about 0.1 percent of all children. Children with such defects have extremely short life expectancies—typically less than a year.

It is possible to estimate the rate of induction of translocations in primary human spermatocytes directly from experimental data. No such data exist on the rates of induction in oocytes. The upper and central estimates developed by the genetic effects working group assume that the induction rates in males and females are the same. The lower estimates assume that translocations may only be induced in spermatocytes. Using a linear-quadratic dose response relationship—in which the linear and quadratic contributions are equal at a dose of 1 Gy—they obtain:

$$r_{\text{translocation induction}} = 15\ d + 15\ d^2$$

where d is the dose (Gy) to the gonads and r is the risk (translocations/ 1000 spermatocytes or oocytes) of inducing a translocation.

Not all induced translocations are transmitted. As a result of meiotic segregation, the fraction of mature sperm carrying balanced translocations is 1/4 this large and the fraction carrying unbalanced translocations is 1/2 this large. Similarly, only 1/16 of induced translocations

will result in balanced translocations in mature oocytes and 6/16 will result in unbalanced translocations. Thus the rates of unbalanced translocations among the mature sperm and ova are:

$$r_{sperm, unbalanced\ translocation} = 7.5\ d + 7.5\ d^2$$

$$r_{ovum, unbalanced\ translocation} = 5.6\ d + 5.6\ d^2$$

The risk that an unbalanced translocation will be present in a fertilized ovum is simply the sum of the risks given above. Ninety percent of these fertilized ova would be inviable and would result in pregnancy losses, primarily during the peri-implantation period, but occasionally later in the pregnancy. The remaining 10 percent would be viable. Thus, the risk of bearing a child with a defect caused by an unbalanced translocation in the first generation after an accident may be estimated using:

$$r_{child, unbalanced\ translocation} = 1.2\ d + 1.2\ d^2$$

where r is risk (affected children/1000 livebirths) and d is the dose (Gy) to the gonads received by the child's parents.

The dynamics of inheritance of unbalanced translocations are such that the risk in the second generation is 1/4th of that in the first and that in each succeeding generation the risk decreases by 50 percent.

The cumulative risk—i.e., the risk that a child with an unbalanced translocation will be born in this or any future generation—is found by summing the risks over all generations. Using the demographic assumptions recommended by the genetic effects working group—i.e., 500 births per generation (30 years) per thousand population—one would obtain central estimates of:

$$R = [1.2\ d + 1.2\ d^2][1 + 1/4 + 1/8 + ...][500/1000]$$

$$R = 0.9\ d + 0.9\ d^2$$

where R is the cumulative risk (number of affected children/1000 exposed people) and d is the gonadal dose (Gy) received by the population.

Upper and lower estimates are derived using this same approach but applying different estimates of the rates of gametic damage. For upper estimates, the working group recommends using gametic induction rates five times larger for males and ten times larger for females. For lower estimates, they recommend using a male gametic induction rate only one fifth as large and assuming that the female gamete is insensitive to radiation-induced damage. Using these assumptions, the upper estimates are seven times larger than the central estimates and the lower estimates

are about 1/8th as large. The differences in the gametic induction rates used in the central, upper and lower estimates reflect differences in the gamma-ray RBE and low dose rate effectiveness factors used to interpret the experimental data.

2.3.3 Multifactorial Diseases

Multifactorial diseases involve complex patterns of inheritance. A specific combination of mutant genes must be present for an effect to be manifest. This largest class of genetic disease affects about 9 percent of the population and includes congenital malformations (e.g., spina bifida, cleft palate), constitutional diseases and degenerative diseases.

The genetic effects working group developed an estimate of the equilibrium risk of multifactorial diseases using a doubling dose approach. For their calculations, they assumed that between 5 percent and 50 percent of multifactorial disease is due to mutations and that the dose required to double this mutation-related component is between 0.5 and 2.5 Gy. Based on these assumptions, they derived a lower estimate of the integrated risk over all future generations of 0.9 cases per 1000 births per Gy, an upper estimate of 90 cases per 1000 births per Gy, and a central estimate of 14 cases per 1000 births per Gy. The central and upper estimates assume that male and female germ cells are equally sensitive to radiation damage. The lower estimate assumes that only male germ cells are damaged. Although no analysis of the distribution of these risks over time was provided, the genetic effects working group stated that these defects have a mean persistence of approximately ten generations.

2.3.4 Recessive Diseases

Recessive diseases include cystic fibrosis, phenylketonuria and some forms of congenital blindness and deafness. The current prevalence of such diseases is about 4 cases per 1000 births. The working group notes that many recessive mutations are thought to be partially dominant—i.e., they are likely to be eliminated from the population before becoming homozygous—and indicates that these effects have been considered in their analysis of dominant effects. Although the genetic effects working group did not provide a complete analysis of the risk of recessive effects, they did suggest doubling doses of about 0.5 Gy for acute exposure and 1 Gy for chronic exposure.* A linear-quadratic model consistent with these values and with the working group's estimate of the prevalence of recessive disease not accounted for in their dominant effects model—i.e., about 2 cases per 1000 births—is:

$$R_{recessive} = 2\ d + 2\ d^2$$

where R is the equilibrium risk (number of affected children/1000 births) and d is the gonadal dose (Gy) received.

It should be noted that recessive risks are expressed very slowly—their mean persistance is 100 times as long as dominant effects of equal severity. Thus, the vast majority of recessive effects are expected to occur long after the other genetic effects described in this report. These effects would not contribute appreciably to the genetic risk experienced within the first five generations after an accident.

2.3.5 Summary—Genetic Effects

Tables 2.9 and 2.10 summarize the models recommended for estimating the genetic effects resulting from population exposures to ionizing radiation following a major accident in a nuclear power plant.

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* It should be noted that one member of the genetic effects working group pointed out that these choices were "consciously conservative, and are lower than the estimates derived directly from the experiences of the offspring of survivors of the atomic bombing of Hiroshima and Nagasaki."

Table 2.9

Models of Genetic Risks[a,b]

Effect	Integrated Risk (cases/1000)		
	Central	Lower	Upper
Single Gene			
Dominant	$7.5 \; d + 7.5 \; d^2$	$1.2 \; d + 1.2 \; d^2$	$22 \; d + 22 \; d^2$
X-linked	$2.2 \; d + 2.2 \; d^2$	$0.5 \; d + 0.5 \; d^2$	$9 \; d + 9 \; d^2$
Chromosome[c] Aberrations			
Numerical	$0.5 \; d$	0	$1.5 \; d$
Structural	$0.9 \; d + 0.9 \; d^2$	$0.1 \; d + 0.1 \; d^2$	$6.5 \; d + 6.5 \; d^2$
Multifactorial	$7.2 \; d + 7.2 \; d^2$	$0.45 \; d + 0.45 \; d^2$	$45 \; d + 45 \; d^2$
Losses of Pregnancy[c]			
Numerical	$4.5 \; d$	0	$13.5 \; d$
Structural	$8.1 \; d + 8.1 \; d^2$	$0.9 \; d + 0.9 \; d^2$	$58 \; d + 58 \; d^2$

[a] The doses, d, referred to in this table are the doses to the gonads expressed in Gray (Gy). The integrated risk is the risk summed over all future generations—expressed in cases per 1000 persons exposed to dose, d.

[b] No formal model of the risk of recessive disease was developed, but the working group provided some information suggesting the possible magnitude of these risks (see pp. I-51).

[c] Chromosomal defects may lead to early losses of pregnancy or to children born with severe physical and mental defects. The vast majority of such pregnancy losses occur as a result of failure of the fertilized egg to implant in the uterine wall.

Table 2.10

Time Distribution of Genetic Risks*

Effect	Time Since Accident (yr)					
	0-29	30-59	60-89	90-119	120-149	>150
Single Gene						
Dominant	20	16	13	10	8	33
X-linked	20	16	13	10	8	33
Chromosome Aberrations						
Numerical	100	-	-	-	-	-
Structural	67	17	8	4	2	2
Multifactorial	———————————— unknown ————————————					
Losses of Pregnancy						
Numerical	100	-	-	-	-	-
Structural	67	17	8	4	2	2

* Entries in the body of the table give the percentage of the cumulative genetic risk—see Table 2.9—expected in each time interval.

3.0 COMPUTATIONAL ASPECTS

This section of the report covers issues related to the computer implementation and mathematical derivation of certain health effects models.

3.1 Early and Continuing Effects

The structure of the nuclear power plant accident consequence code MACCS [Chanin, 1989] is based on the health effects models recommended in the first edition of NUREG/CR-4214. The risks of all early and continuing effects are computed indirectly using two-parameter Weibull hazard functions. The effect of dose rate on risk is accommodated using different values of the median lethal or effective dose to compute the risk from dose received in different time intervals following the accident— e.g., 0 to 1 day, 2 to 7 days, etc. In addition, there are restrictions on the size of arrays and matrices used to store results which limit the number of effects that can be considered.

Below some approaches are outlined for implementing the new health effects models in MACCS. These should be considered interim solutions. Eventually the code should be rewritten to allow direct implementation of the new models.

3.1.1 Hematopoietic Syndrome Risk in a Population Receiving Mixed Medical Treatment

The risk of death from the hematopoietic syndrome depends on the dose, the dose rate, and the level of medical treatment received. The models of hematopoietic syndrome mortality described earlier in this report give the risks for two levels of medical treatment—minimal treatment and supportive treatment. To compute accident consequences using these models, one must estimate the risks in each medical treatment group separately and then combine these estimates in a manner that reflects the anticipated availability of each class of treatment.

The Reactor Safety Study risk estimates were based on the assumption that 2500 to 5000 beds in hospitals across the U.S. could be made available for supportive treatment of accident victims. Wald [personal communication, 1989], one of the authors of the Reactor Safety Study, explains:

- "... in the absence of appropriate data, we assumed that appropriate supportive therapy could be given at any U.S. hospital that is approved for residency training by the American Board of Internal Medicine, that 10% of the beds could be made available within a few days, and that there would be time and resources enough to transport individuals to these beds during the latent period of the Acute Radiation Syndrome before clinical problems emerged."

- "Unfortunately, the current data base for this information has not improved perceptibly. Although there are more hospitals approved today, the form in which the data are maintained makes it more difficult to determine the real number of beds actually available. I would, therefore, suggest that the same [availability of beds] be used that was used in WASH-1400..."

Wald's comments suggest that central estimates of hematopoietic risk should be calculated assuming that supportive treatment could be provided to as many as several thousand exposed individuals and that for larger accidents some people would receive supportive and others minimal treatment. After a large accident, many people will need medical screening. Only some of these will need supportive treatment. Logistic problems in the screening process may lead to misallocation of treatment. Until such problems have been studied more thoroughly, upper estimates of hematopoietic risk should probably be calculated assuming that all exposed individuals receive minimal treatment, and lower estimates that all receive supportive treatment.

To facilitate evaluation of central estimates of risk when a mix of minimal and supportive treatments is assumed, one can assume that the medical treatment received is independent of the dose received. The risk expected at any dose is then a simple average of the risks in the two treatment groups at that dose. For a population in which half received minimal treatment and half received supportive treatment, the risk would be:

$$R_{mixed} = 0.5 \ R_{minimal} + 0.5 \ R_{supportive}$$

where $R_{minimal}$ and $R_{supportive}$ are the risk functions appropriate for minimal and supportive treatment.

Figure 3.1 shows the risks that would be expected in such a population— and within each treatment group—following exposure at high dose rate. The central estimates of hematopoietic syndrome mortality model parameters were used to develop this example. The resulting population dose-response curve—i.e., for mixed medical treatment—is "lumpy" and cannot be described exactly by a unimodal two-parameter Weibull function. However it can be approximated by a Weibull function with a median lethal dose of 3.8 Gy, a shape parameter of 5, and a threshold dose of 1.5 Gy.

Figure 3.2 compares the estimates of risk given by the mixed treatment model, R_{mixed}, with those given by the approximating hazard function. The systematic errors in the approximation—i.e., underestimation of risk at low dose and overestimation of risk at high dose—are small in comparison with the uncertainties in the underlying model parameters and with the errors introduced by assuming that medical treatment is randomly distributed.

Ideally one would use a model which reflected a more nearly optimal allocation of medical treatment. The error introduced by the assumption of random allocation of treatment is highly variable and depends on the distribution of doses received by the exposed population. In some cases the number of lives that could be saved by more efficient allocation of treatment may be underestimated by as much as 50 percent.
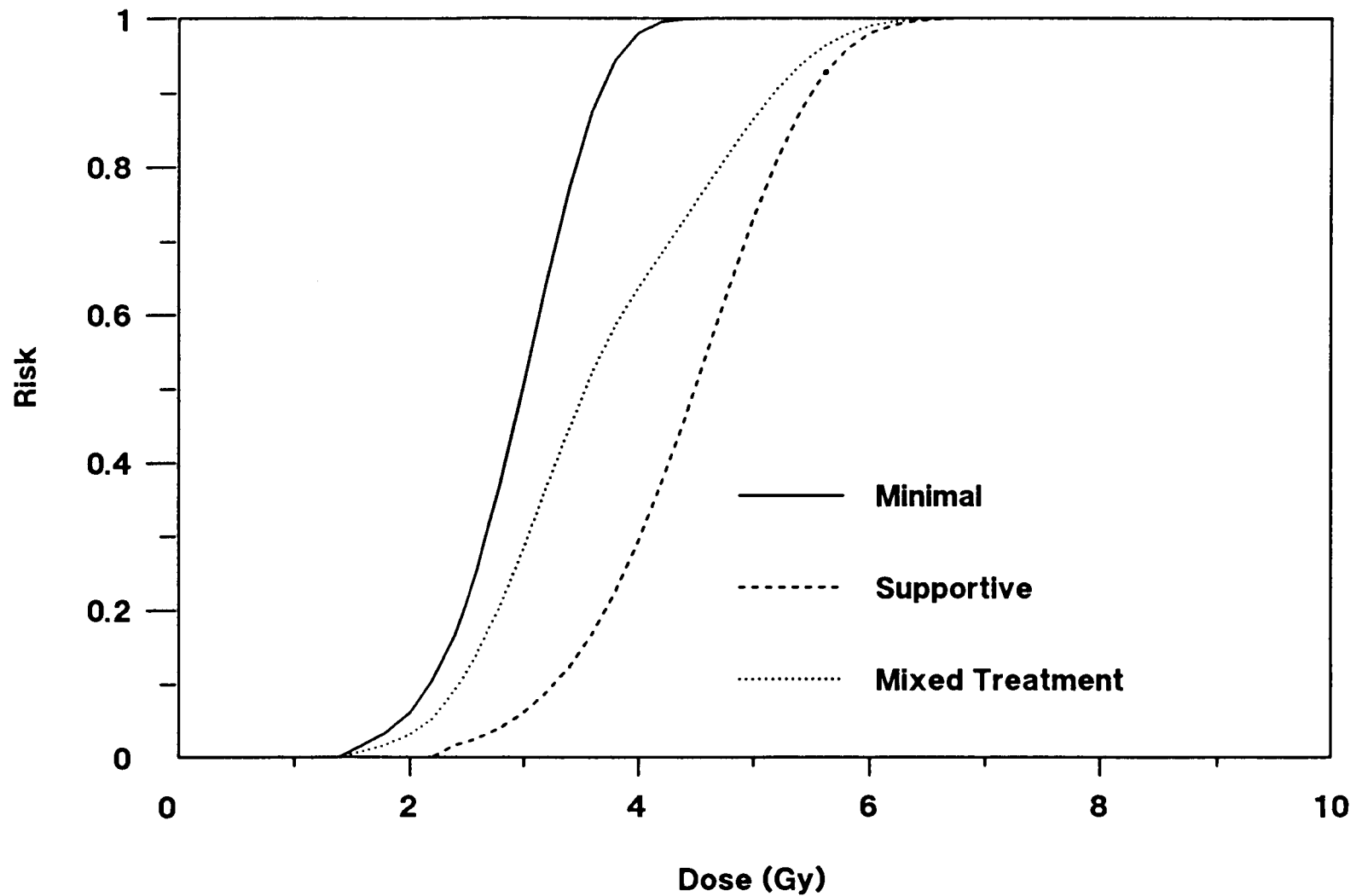
Figure 3.1 Hematopoietic Syndrome Mortality Risks for Minimal Treatment Supportive Treatment and Mixed Treatment -- Central Estimates for Exposure at High Dose Rate.
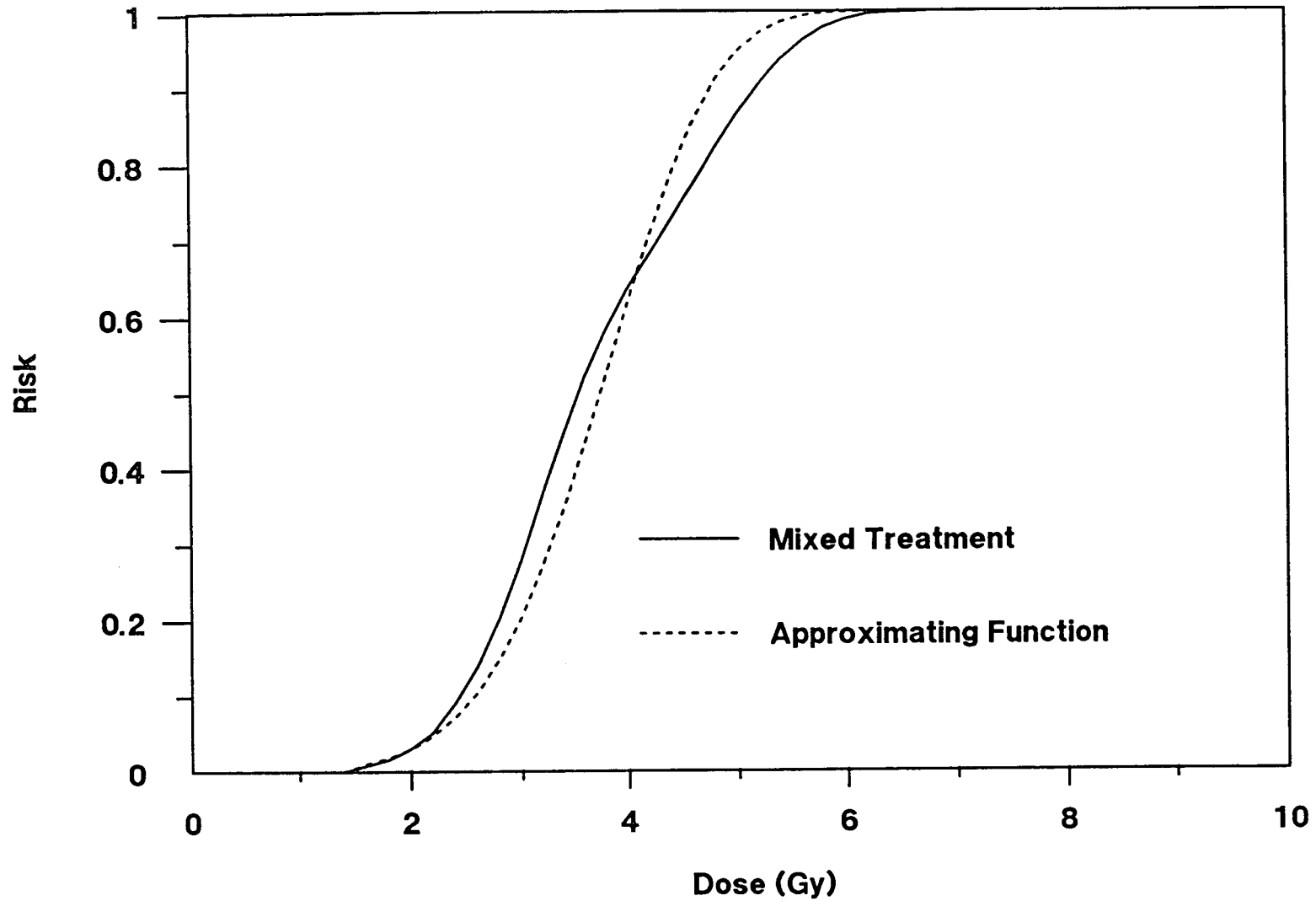
Figure 3.2 Comparison of Mixed Treatment Model and Approximating Hazard Function for Estimating Hematopoietic Syndrome Mortality Risks -- Central Estimates for Exposure at High Dose Rate.

Those responsible for developing improved accident consequence calculation codes should consider developing more sophisticated and flexible approaches for addressing this problem—approaches which permit the analyst to use site-specific data on the availability of medical treatment and to explore the sensitivity of results to the efficiency of allocation of medical treatment. In conjunction with this effort, the NRC should support efforts to develop better estimates of the availability of medical treatment.

3.1.2 Accounting for the Influence of Dose Rate in Accident Consequence Calculations

The risk of early mortality is influenced both by the total dose received and by the rate at which the dose is received. Dose received at low dose rate is less effective than dose received at high rate.

CRAC [NRC, 1975], the accident consequence calculation code developed in support of the Reactor Safety Study, accounted for the influence of dose rate by the use of dose rate effectiveness factors. For example, in the calculation of hematopoietic syndrome mortality risks a synthetic dose estimate was used:

$$d_{effective} = d_{external} + d_{internal, day\ 0-1}$$

$$+ \frac{1}{2} d_{internal, day\ 2-14} + \frac{1}{4} d_{internal, day\ 15-30}$$

where $d_{external}$ is the dose from cloudshine and groundshine, and the $d_{internal}$ terms account for the dose received in each of three time periods from radionuclides that were inhaled and retained within the body. MACCS, the computer code developed to replace CRAC, uses this same approach.

The dose-rate-dependent models developed by Scott and endorsed by the early effects working group allow one to express the median lethal dose, LD_{50}(Gy), as a function of dose rate, D (Gy/hr):

$$LD_{50} = \theta_\infty + \theta_1/\dot{D}$$

where θ_∞ is the limiting value of the median lethal dose at high dose rate, and θ_1 is a parameter reflecting the sensitivity of the median lethal dose to the dose rate. The values of θ_∞ and θ_1 recommended by the working group for estimating hematopoietic syndrome mortality risks in populations receiving minimal medical treatment are:

Estimate	θ_∞	θ_1
Central	3.0	0.07
Lower	2.5	0.06
Upper	3.5	0.08

Figure 3.3 shows the relationships between dose rate and median lethal dose that are obtained using these values for θ_∞ and θ_1 and a shape parameter value of 6. Note that the median lethal dose reaches twice its limiting value at dose rates of about 0.03 Gy/hr and four times its limiting value at dose rates just below 0.01 Gy/hr. For dose received at rates above 1 Gy/hr the exact dose rate is less important, because in this range the median lethal dose is within 20 percent of its limiting value.

Both CRAC and MACCS use a fixed time interval approach for computing the risks of pulmonary syndrome. A synthetic estimate of the effective lung dose is derived using dose rate effectiveness factors:

$$d_{effective} = d_{external} + d_{internal,0-1\ day} + \frac{1}{16} d_{internal,2-14\ days}$$

$$+ \frac{1}{37} d_{internal,15-200\ days} + \frac{1}{92} d_{internal,201-365}$$

where $d_{external}$ and the four $d_{internal}$ terms have the same general interpretation as in the models for hematopoietic syndrome. These particular dose rate effectiveness factors are based on a preliminary reanalysis of the original NUREG/CR-4214 pulmonary syndrome models [Scott, 1989]. They are different from the values given in the original report and from those used in early versions of MACCS.

The dose-rate-dependent models for pulmonary syndrome mortality, described in Section 2.1.1.2 of this report, have the same form as the models for hematopoietic syndrome mortality. The values of θ_∞ and θ_1 recommended by the working group for estimating pulmonary syndrome mortality risks in populations of heathly young adults are:

Estimate	θ_∞	θ_1
Central	10	30
Lower	8	15
Upper	12	45

Shape parameter values of 5, 12, and 7 are recommended by the working group for calculations of internal, external, and mixed pulminary exposures.

Figure 3.4 shows the relationship between pulmonary syndrome risk, effective half-life, and initial dose rate for inhaled radionuclides. Two isoquants of risk—1 percent and 99 percent—are shown for radionuclides with half-lives between 1 and 1000 days and for amounts inhaled that would result in initial dose rates between 0.1 and 1 Gy/hr. The isoquants were identified by evaluating:
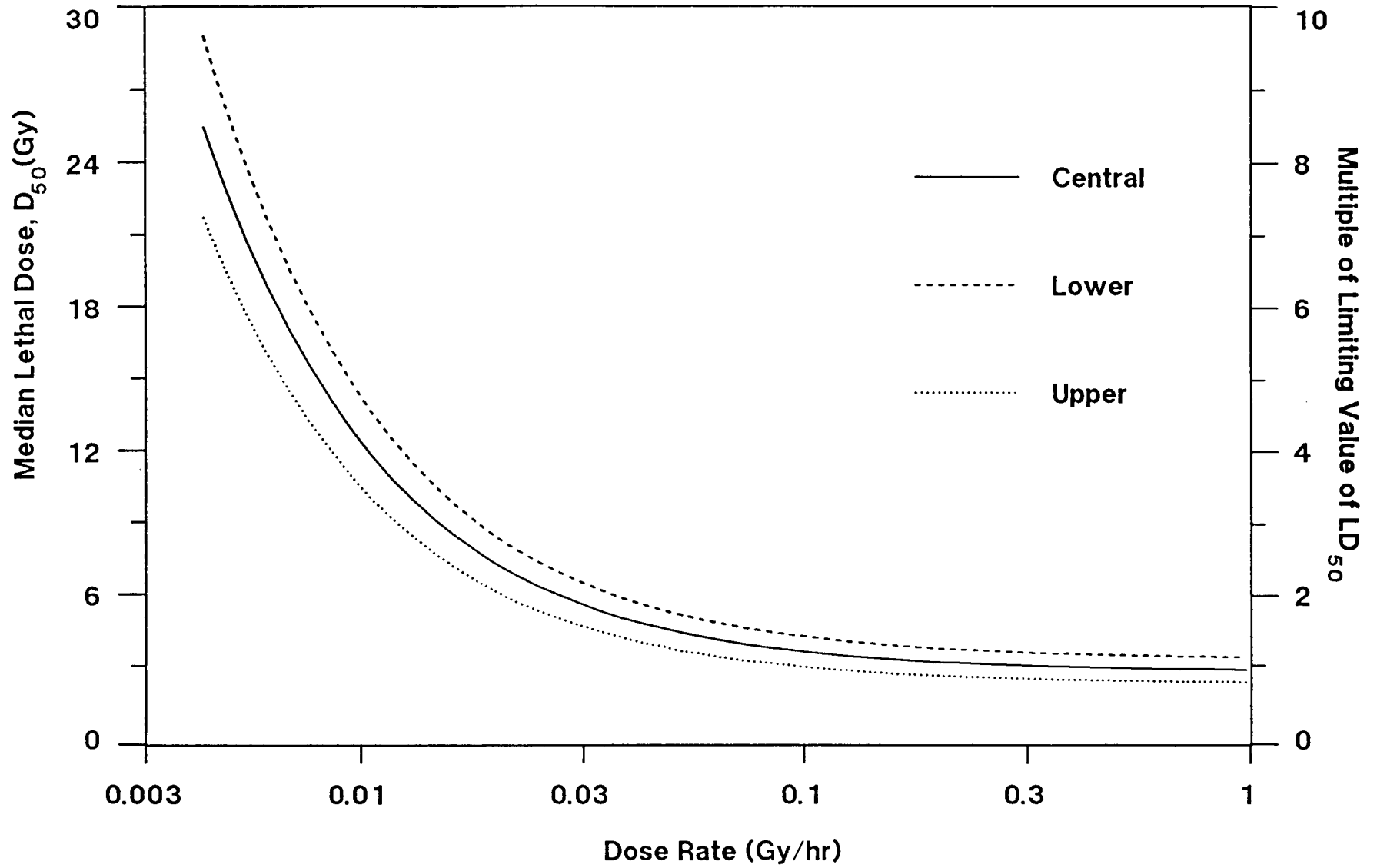
Figure 3.3 Dependence of Median Lethal Dose on Dose Rate
-- Hematopoietic Syndrome Mortality
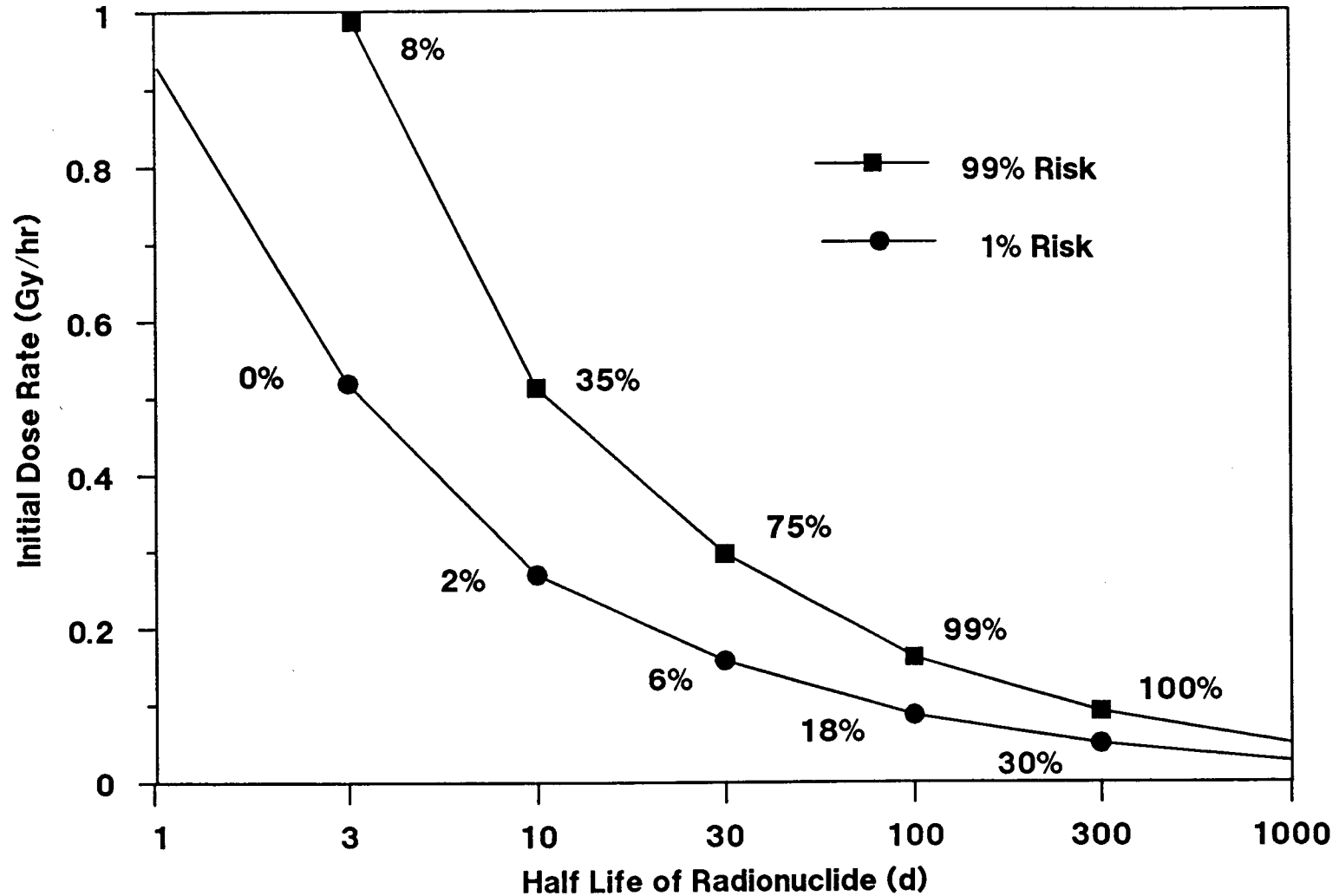
**Figure 3.4   Comparison of Pulmonary Syndrome Mortality Risks Estimates
-- Dose Rate Dependent Model (Central Estimates) vs.
Fixed Time Interval Model**

$$R = 1 - e^{-H}$$

$$H = 0.693 \left[\int_{o}^{\infty} \frac{\dot{D}}{\theta_{\infty} + \theta_1/\dot{D}} \, dt \right]^{v}$$

$$\dot{D} = \dot{D}_o \, e^{-0.693(t/t_{1/2})}$$

for several levels of initial dose rate, D_o, and effective half-life, $t_{1/2}$. The working group's central estimates of θ_{∞} and θ_1 were used. The numbers shown along the curves are the estimates of risk obtained for these same patterns of dose rate with the fixed time interval approach. LD_{50} values of 160 Gy (0 to 14 days), 370 Gy (15 to 200 days), and 920 Gy (201 to 365 days) and a shape parameter value of 5 were used in the calculations.

For radionuclides with half-lives between 10 and 100 days the agreement between the two approaches is reasonable. Outside of this region some bias is evident. The fixed time interval approach appears to underestimate risks for radionuclides with half-lives shorter than 10 days and to overestimate risk for radionuclide with half-lives longer than 100 days. Factors other than radiological decay influence the actual patterns of dose to the lung from inhaled radionuclides. Biological clearance mechanisms—e.g., absorption, mucocilliary transport, clearance by pulmonary macrophages—are also potentially significant. More accurate comparisons of the dose-rate-dependent and fixed time interval models would account for these factors. The two examples that follow consider both biological clearance and radiological decay.

Figure 3.5 shows several estimates of pulmonary syndrome mortality risk from inhaled ruthenium 106—a relatively insoluble radionuclide (clearance class Y), with a radiological half-life of 366 days.* Three of the values shown are the central, lower and upper estimates of risk derived using the dose-rate-dependent model. The other two are based on fixed time interval models. The first fixed interval model, labeled "internal" in Figure 3.5, uses a shape parameter value of 5 and a first-day LD_{50} of 160 Gy. The second fixed interval model, labeled "internal and external" in Figure 3.5, uses a shape parameter value of 7, and on the assumption that external sources such as cloudshine and groundshine will lead to high dose rates on the first day, it uses a first-day LD_{50} of 10 Gy. For [106]Ru, both fixed time interval models overestimate risk.

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* The calculations upon which Figure 3.5 is based assume that 40 percent of the [106]Ru is cleared from the lung with an effective half-life on the order of one day and that the remainder is cleared with an effective half-life on the order of 250 days. These values were derived from an analysis of the dose conversion factors used in the MACCS code [Chanin, 1989].
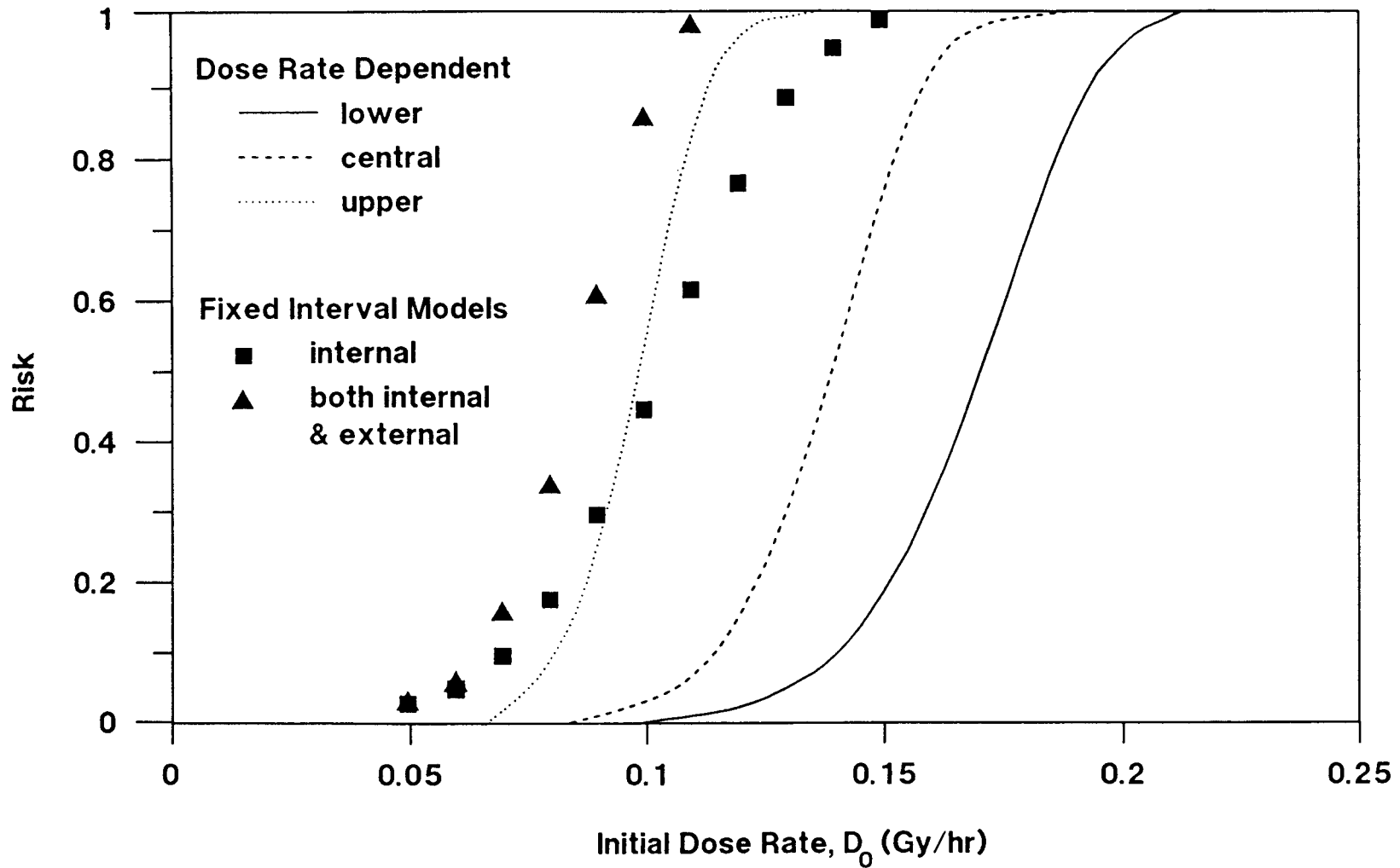
Figure 3.5 Pulmonary Syndrome Mortality Risk – Ruthenium 106

Figure 3.6 presents the results of a similar analysis of pulmonary syndrome mortality risks from inhaled ^{131}I—a relatively soluble radionuclide (clearance class D), with a radiological half-life of 8 days.* For ^{131}I, the two alternative fixed dose rate approaches give quite different results. The calculations which assume high dose rates on the first day significantly overestimate risks, while those which consider only inhaled radionuclides significantly underestimate risk.

3.1.3 Hypothyroidism

The thyroid effects working group recommended that the risk of hypo-thyroidism—following internal exposure to ^{131}I—be estimated using a linear-threshold model with a threshold of 10 Gy and a slope of 17 x 10^{-4} cases per person-Gy. MACCS is set up to compute the risks of all early effects using two-parameter Weibull hazard functions. Weibull function parameters that approximate the linear dose-response model recommended by the working group are a median effective dose of 300 Gy, a threshold of 10 Gy and a shape factor of 1.3. As Figure 3.7 illustrates, the approximation is quite good at low dose and is acceptable at high dose. The bias due to the approximation is always less than 20 percent.

Following all other exposures, the thyroid effects working group recommended using a linear-threshold model with a threshold of 2 Gy and a slope of 85 x 10^{-4} cases per person-Gy. Similarly, this dose-response model can be approximated by a hazard function with a median effective dose of 60 Gy, a threshold of 2 Gy and a shape factor of 1.3.

3.1.4 Fetal Deaths

The risk that an embryo or fetus will die as a result of exposure to ionizing radiation depends on both the dose received and the developmental age of the embryo/fetus at the time of exposure. The early effects working group provided dose-response functions appropriate for three developmental age groups—0 to 18 days, 18 to 150 days, and 150 days to term. Their central estimates of the hazard functions are:

$$H_{0-18days} = 0.693[d/1]^2 \qquad \text{for } d > 0.1 \text{ Gy}$$

$$H_{18-150days} = 0.693[d/1.5]^3 \qquad \text{for } d > 0.4 \text{ Gy}$$

$$H_{150days-term} = 0.693[d/3]^6 \qquad \text{for } d > 1.5 \text{ Gy}$$

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* The calculations upon which Figure 3.5 is based assume that 99.5 percent of the ^{131}I is cleared from the lung with an effective half-life on the order of one day and that the remainder has an effective half-life in the lung that approaches its radioactive half-life (8 days). These values were derived from an analysis of the dose conversion factors used in the MACCS code [Chanin, 1989].
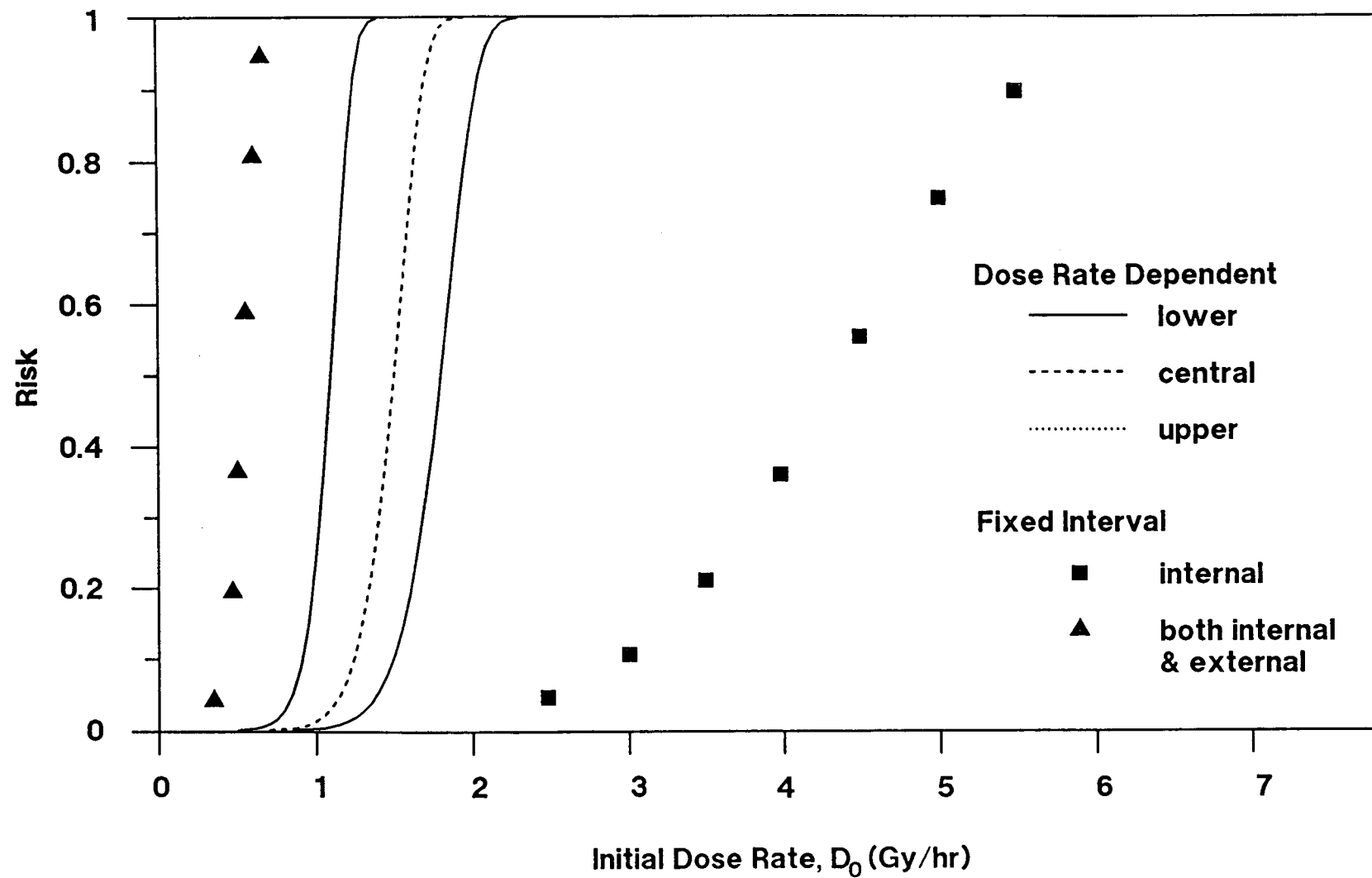
Figure 3.6   Pulmonary Syndrome Mortality Risk – Iodine 131
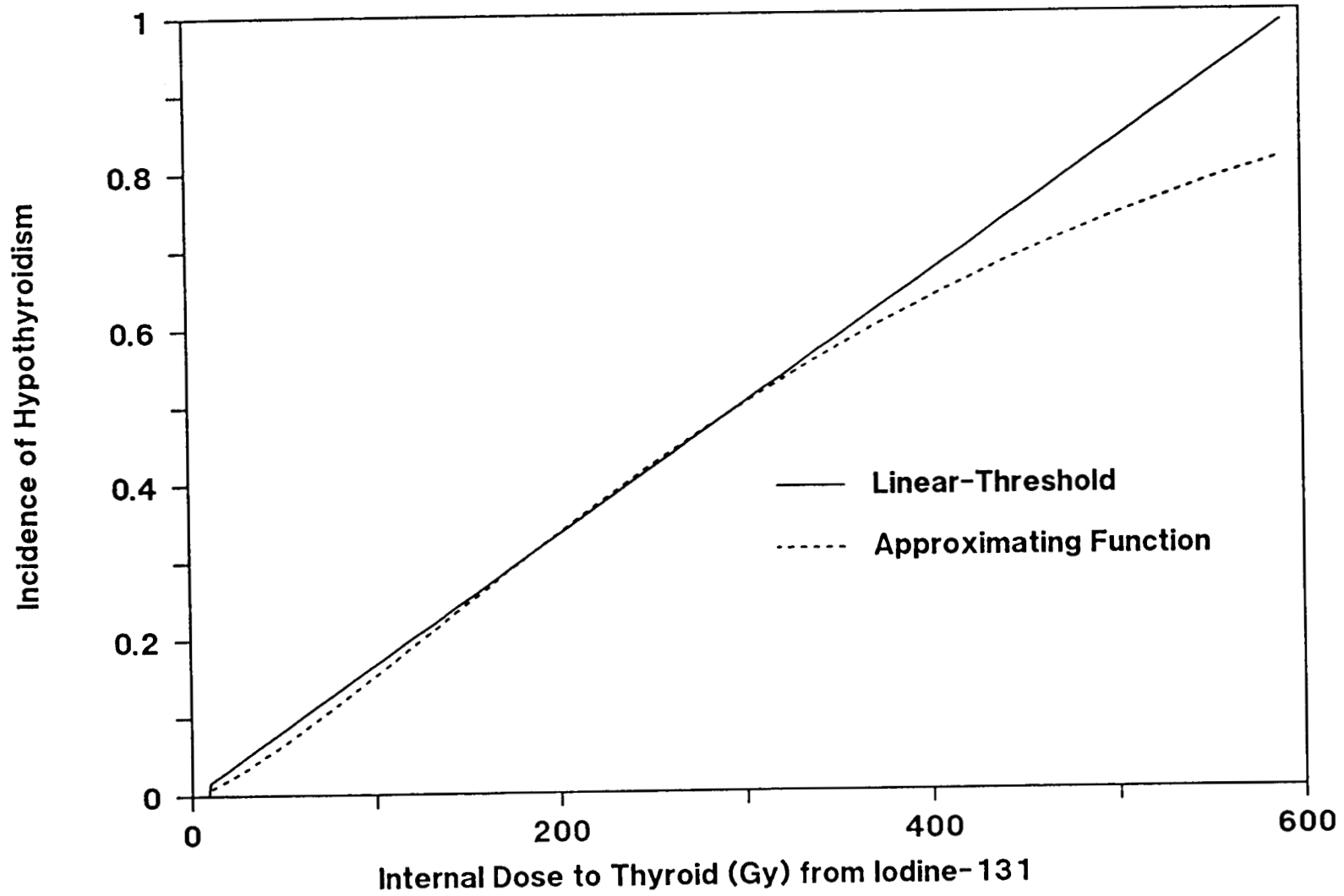
Figure 3.7 Approximation of Linear Threshold Model with Hazard Function

To compute the total number of fetal deaths expected after an accident, it is necessary to account for the risks in all three developmental age groups. These age-specific risk estimates may be combined—using weights corresponding to the fraction of fetuses in each developmental group—to derive a dose response model for a representative fetus:

$$R_{\text{typical fetus}} = (18/280)R_{\text{0-18 days}} + (132/280)R_{\text{18-150 days}}$$

$$+ (130/280)R_{\text{150 days-term}}$$

Figure 3.8 shows the risk faced by a representative fetus for doses between 0 and 5 Gy. Also shown in the figure is an approximating function—a Weibull function with a median lethal dose of 2 Gy, a threshold of 0.1 Gy and a shape parameter of 2.3. The approximation is simple and appears to be quite good.

It should be noted that the weights used above to derive the risk to a representative fetus are proportional to the lengths of the three developmental periods. It would be preferable to use weights based on the actual distribution of developmental ages in the population.

3.1.5 Mental Retardation

The risk of mental retardation among those exposed in utero is a strong function of the gestational age at the time of exposure. The early effects working groups provided dose-response functions for two gestational age groups—8 to 15 weeks, and 16 to 25 weeks. Their central estimates of the hazard functions are:

$$H_{\text{8-15 weeks}} = 0.693[d/1.5] \qquad H_{\text{16-25 weeks}} = 0.693[d/7.0]$$

where d is the fetal dose (Gy). There is no evidence that those exposed within 7 weeks of conception or at gestational ages greater than 25 weeks are at increased risk of mental retardation.

To estimate the number of children expected to be born mentally retarded as a result of radiation exposure following a nuclear power plant accident it is necessary to account for the differences in risk among the gestational age groups. The age-specific risk estimates may be combined—using weights corresponding to the fraction of fetuses in each developmental group—to derive a dose response model for a representative fetus:

$$R_{\text{typical fetus}} = (56/280)R_{\text{8-15 weeks}} + (70/280)R_{\text{16-25 weeks}}$$

where $R_{\text{8-15 wks}}$ and $R_{\text{16-25 wks}}$ are the age-specific risk estimates.

Figure 3.9 illustrates the risk of mental retardation within these two developmental age groups and indicates the risk that would be faced by a representative fetus. Because less than half of all fetuses are at risk, the risk to a representative fetus never reaches 1.0. For many values of dose, it is less than the risk within either developmental age group. As shown in Figure 3.10, the dose-response function for a representative
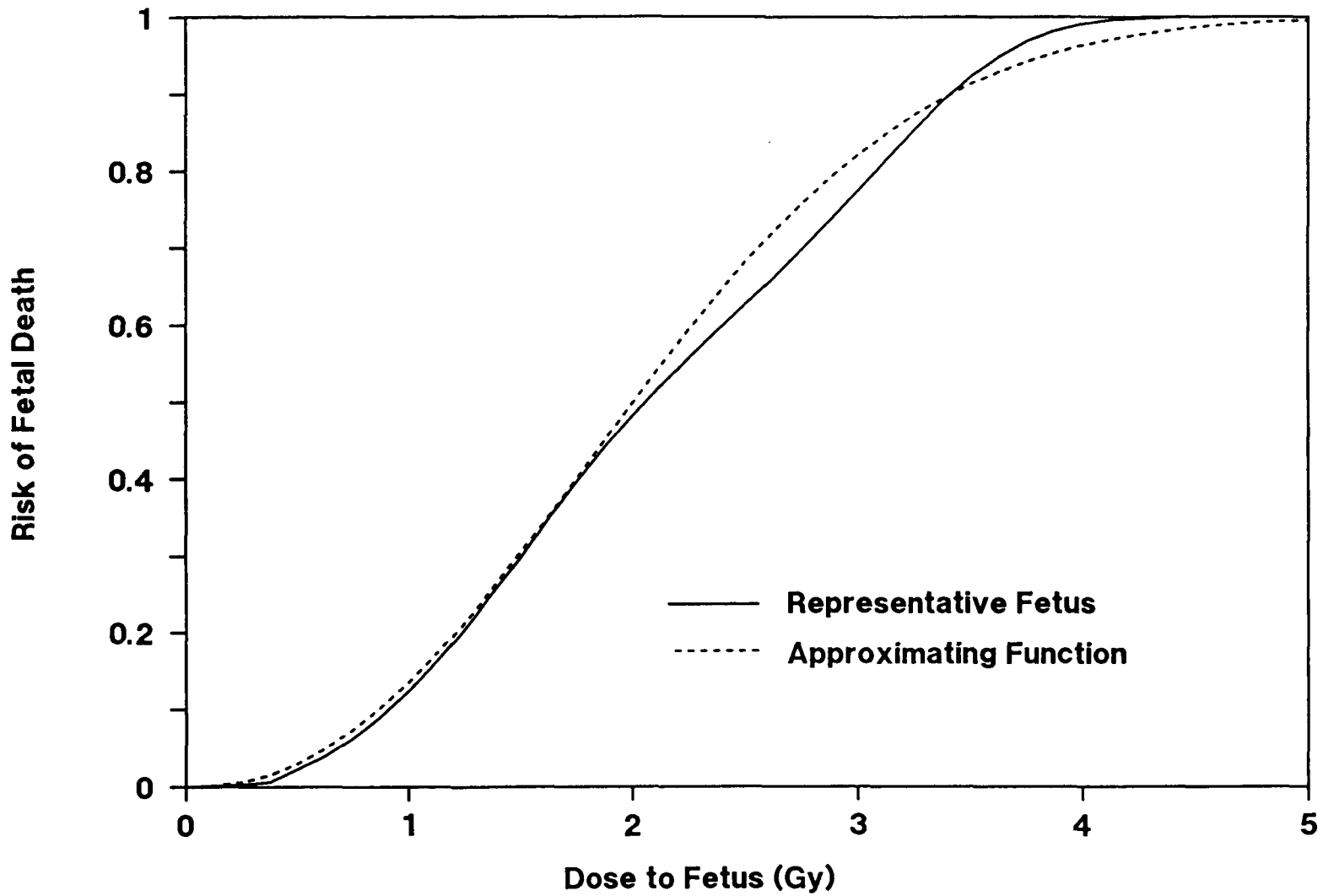
Figure 3.8 Comparison of Mixed Developmental Age Model and Approximating Hazard Function for Estimating the Risk of Death for a Representative Fetus.
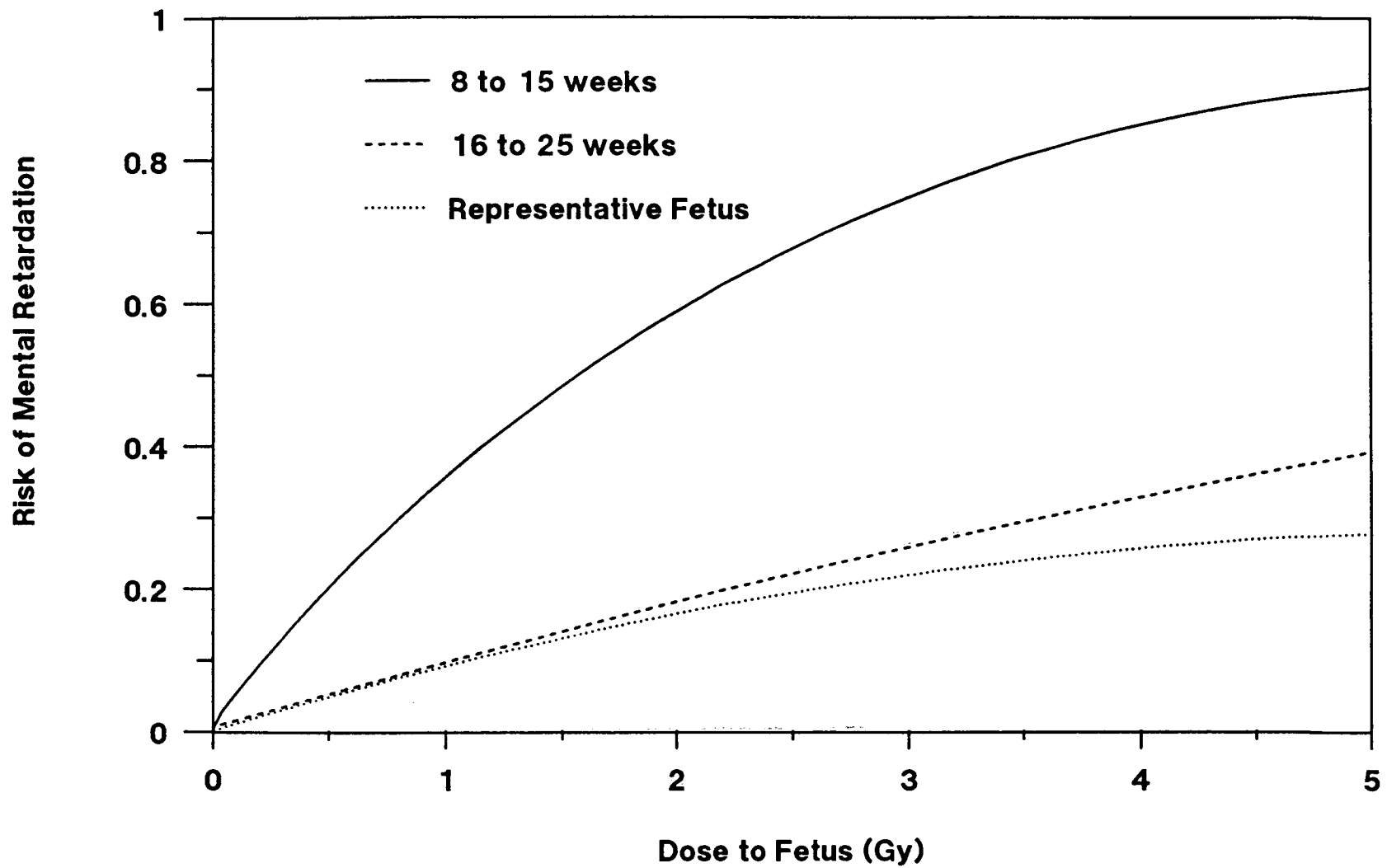
Figure 3.9 Risk of Mental Retardation Within Two Gestational Age Groups and for a Representative Fetus.
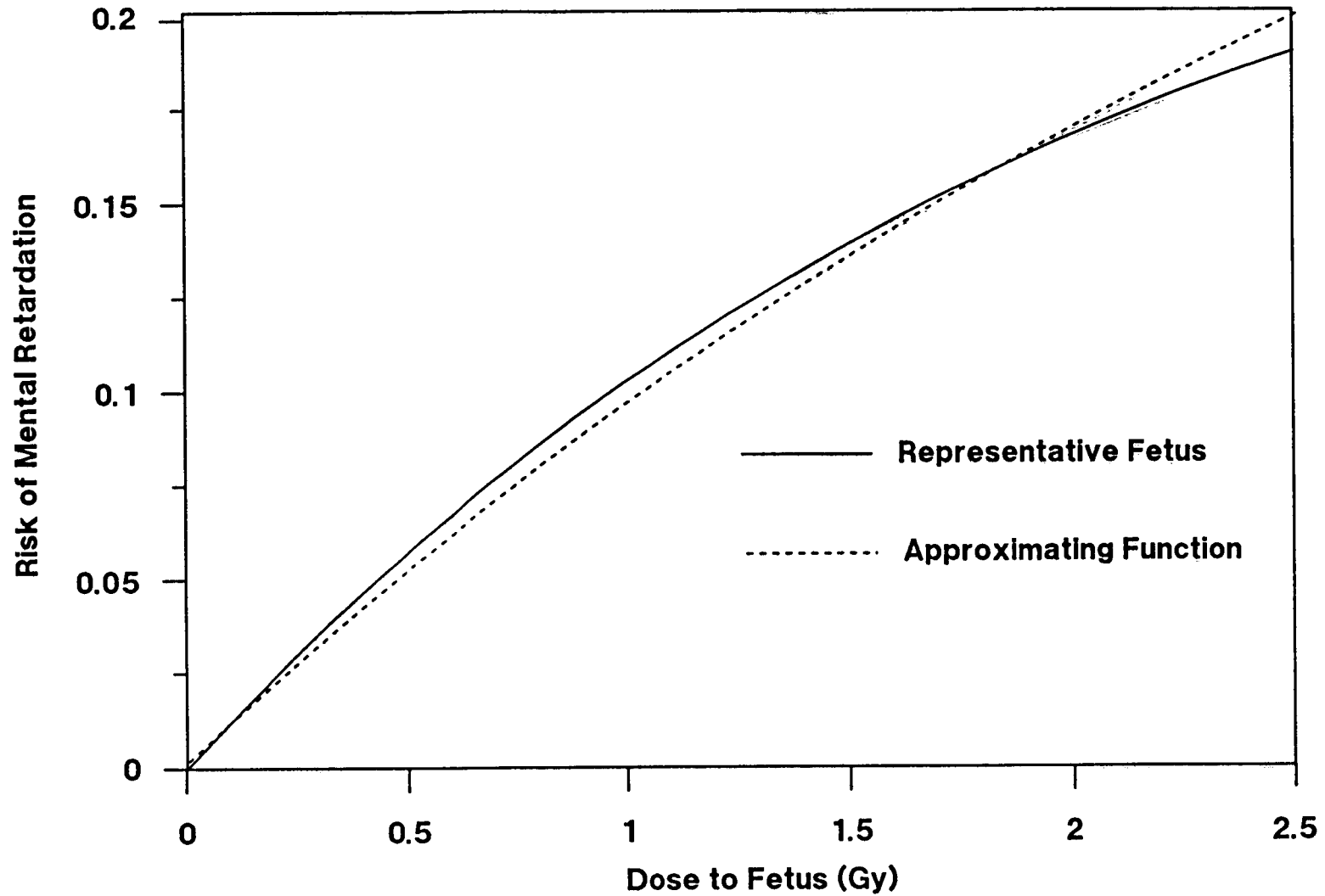
Figure 3.10 Comparison of Mixed Gestational Age Model and
Approximating Hazard Function for Estimating the
Risk of Mental Retardation for a Representative Fetus.

fetus is well approximated by the expression:

$$R_{\text{typical fetus}} = 0.4[1 - \exp[-0.693(d/2.5)]]$$

where d is the dose to the fetus (Gy).

The models given above estimate the risk of mental retardation among those who survive the effects of in utero exposure to radiation. The risk that a fetus will survive the effects of in utero exposure and be born with mental retardation is the product of two terms—the probability of survival and the risk of mental retardation among survivors. As Figure 3.11 illustrates, the risk is maximized for doses between 1 and 2.5 Gy depending on the gestational age of the fetus at the time of exposure. For a representative fetus, the risk is maximized at about 1 Gy. Accident consequence calculations which do not account for fetal death are likely to overestimate the risk of mental retardation.

3.1.6   Form of Dose-Response Model

The risks of early effects could be modeled using almost any sigmoidal function—e.g., the Weibull, the probit, or the logistic function. Our early effects working group selected the two-parameter Weibull function, which has been described extensively in previous sections of the report. The probit model is:

$$r = \left(1/\sqrt{2\pi}\,\sigma\right) \int_{-\infty}^{d} \exp\left\{-1/2\,[(x - \mu)/\sigma]^2\right\}\,dx$$

where μ is the dose at which 50 percent incidence is expected, σ is a measure of the shape of the dose-response function, d is the dose of interest, and x is a dummy argument. Small values of σ reflect low degrees of heterogeneity among the population and therefore steep dose-response functions. The logistic model is:

$$r = \frac{1}{1 + e^{-\alpha - \beta d}}$$

where α is a location parameter—related to background incidence—and β is a shape parameter. Large values of β indicate homogeneity of response and steep dose-response functions. In the logistic model the median lethal (or effective) dose is $-\alpha/\beta$.

With appropriate parameters, all three models will yield essentially identical estimates of risk in the region of experimental data. Outside of this region they may diverge considerably. This point is illustrated in Figures 3.12 and 3.13.* Figure 3.12 shows the early effects working group's central estimate of hematopoietic syndrome mortality risk—for

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* In both figures, risks are shown below the threshold doses recommended by the early effects working group.
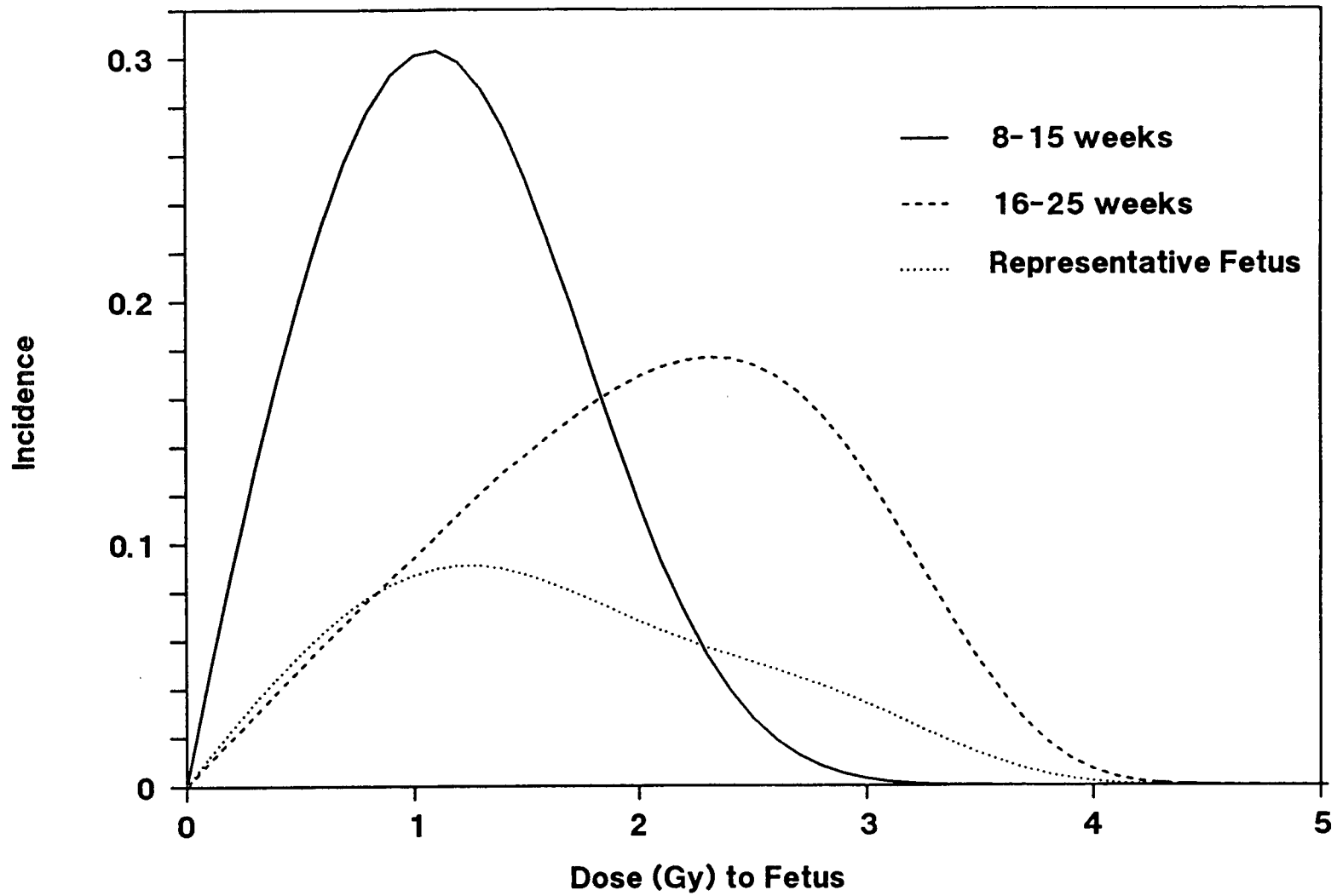
**Figure 3.11 Central Estimates of the Risk of Mental Retardation Among
Those Exposed In Utero -- Accounting for Fetal Deaths**
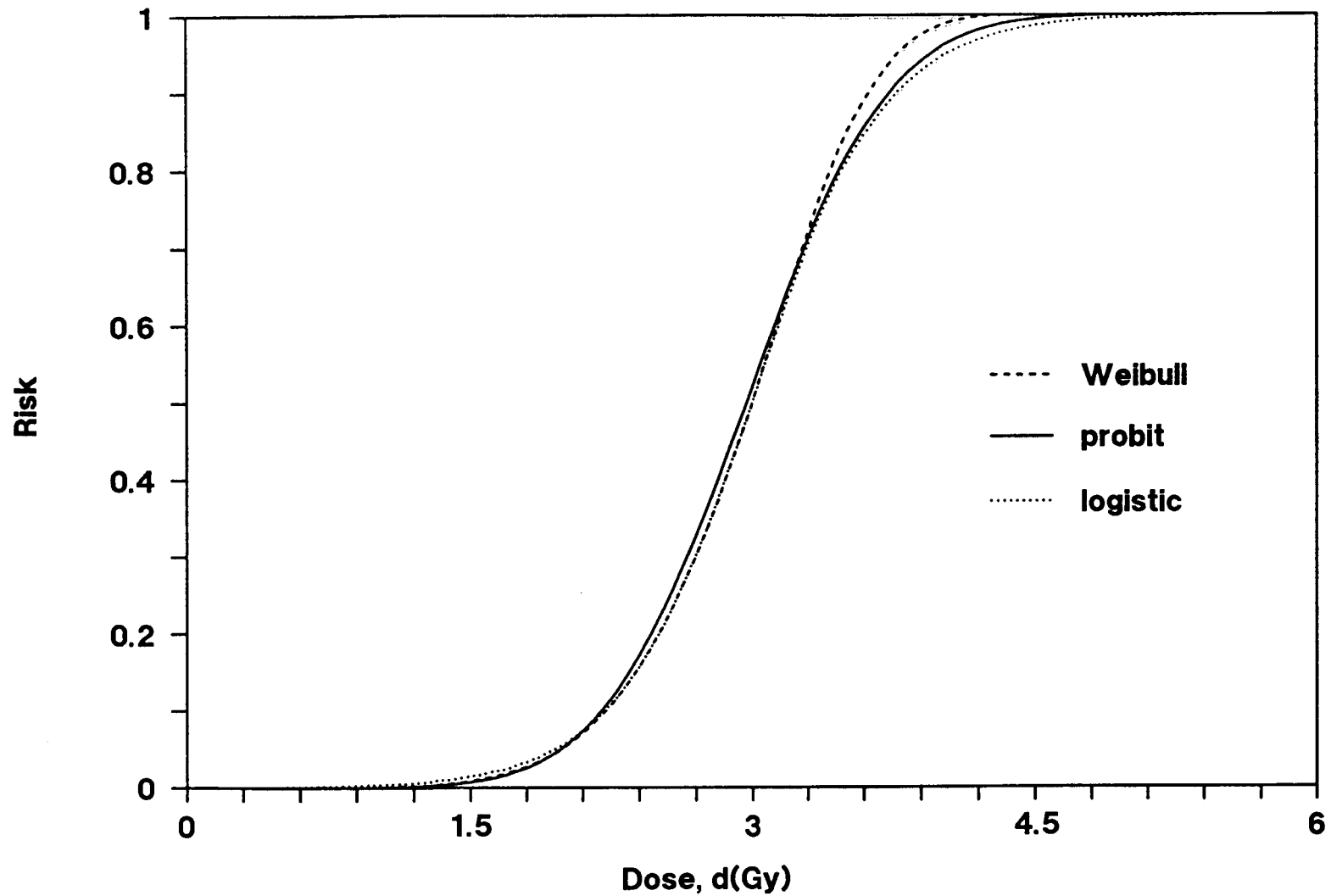
Figure 3.12 A Comparison of Weibull, Probit and Logistic Models
— For Estimating Hematopoietic Syndrome Mortality in
Individuals Receiving Minimal Medical Treatment
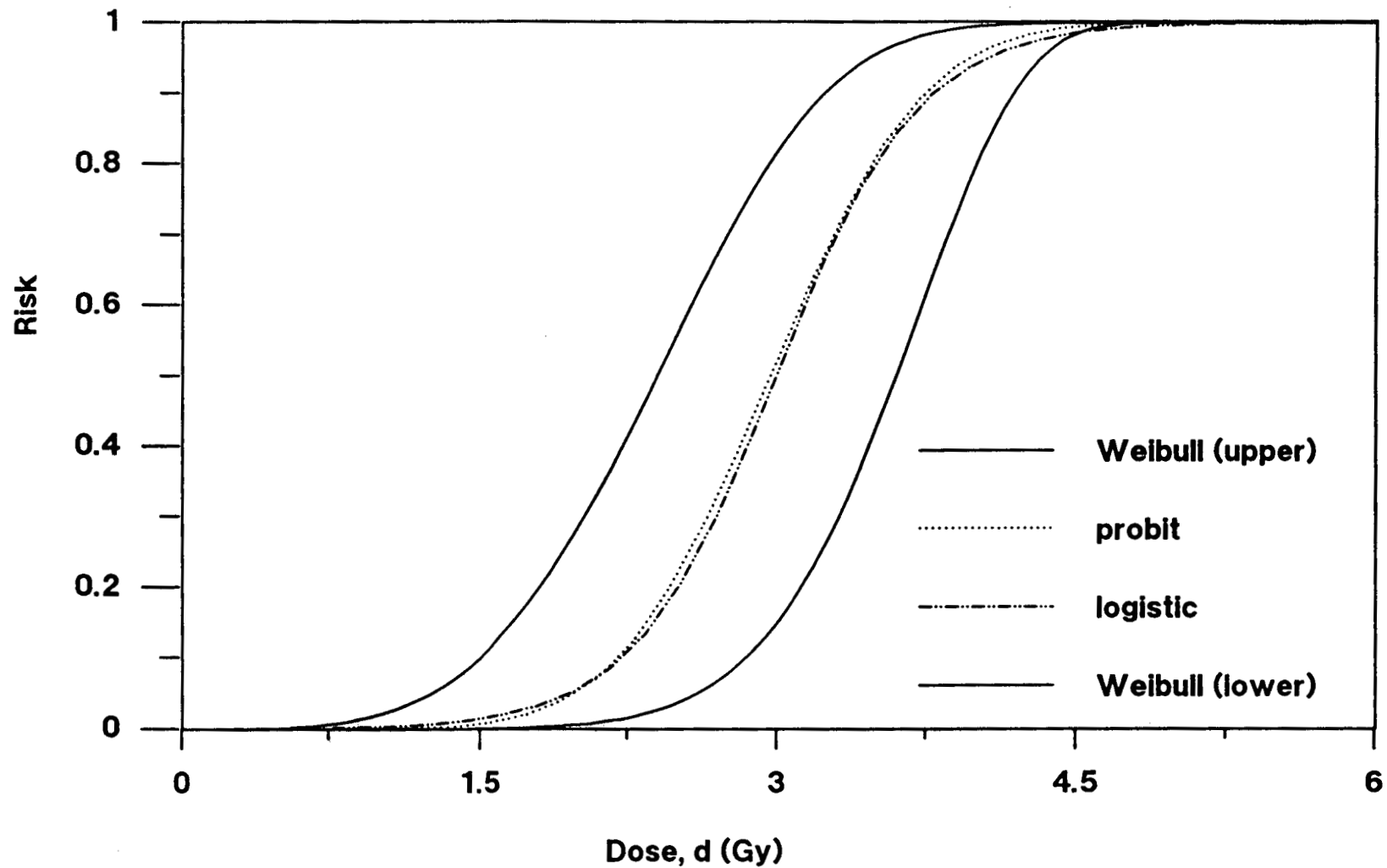
Figure 3.13 Model Uncertainty in Perspective -- Probit and Logistic Central Estimates Contrasted with Upper and Lower Weibull Models for Hematopoietic Syndrome Mortality in Individuals Receiving Minimal Medical Treatment.

individuals receiving minimal medical treatment—and corresponding probit and logistic models. In general, the agreement between the three models is quite good.

At low doses there is some divergence. For example, at the recommended population threshold dose of 1.5 Gy, the Weibull predicts a risk of 0.9 percent while the probit and logistic models give estimates of 0.7 percent and 1.4 percent, respectively. In principle, these differences—which are small in absolute terms—could lead to significant differences in estimated risks for accident scenarios which expose large numbers of people to relatively low doses. However, as Figure 3.13 illustrates, these differences are inconsequential in comparison with the fundamental uncertainties in estimating the risks of early effects.

3.2 Late Somatic Effects

For nuclear power plant consequence analysis it is necessary to predict the fraction of an exposed population that would be expected to develop (or die from) cancer as a result of a specific set of doses. The absolute and relative risk models permit one to predict the risk, as a function of time since exposure, for an individual (i.e., a representative member of an age-sex cohort). Characteristics of the individual, such as gender, race and age at exposure, influence the predicted risk. To obtain estimates of population risk, one must use demographic data and models in conjunction with models of individual risk.

The two most important demographic factors for the prediction of cancer risks are the age structure and age-specific mortality rates in the population of interest.

The risk in a population is found by averaging the risks faced by the various age groups. The fraction of a population that would be expected to die γ years after receiving a dose d is:

$$R\ (\gamma,d) = \sum_k f_k\ s_k(\gamma)\ r_k(\gamma,d)$$

where k is an index of age at exposure, f_k is the fraction of the population in the kth age at exposure group, $s_k(\gamma)$ is the fraction of the kth age at exposure group that will survive all other causes of death for γ years, and $r_k(\gamma,d)$ is the risk that will be experienced by individuals in

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* Theoretically this approach—which does not adjust the survival probabilities, $s_K(\gamma)$, to reflect radiation-induced deaths—could lead to overestimation of risk at high dose. However, in most accident scenarios the overwhelming majority of cancer deaths are predicted to result from the exposure of large populations to relatively low doses. Therefore, as a practical matter, the bias introduced by this simplification is expected to be negligible.

the kth age at exposure group γ years after receiving a dose d.* In our analysis, the values of f_k have been taken from the 1980 U.S. Census of Population [BoC, 1983] and the values of $s_k(\gamma)$ have been taken from the 1979-81 Decennial Life Tables of the United States [NCHS, 1985]. The data used in our calculations are reproduced in Appendix A.

The functions $r_k(\gamma,d)$ are derived from the models of individual risk described in the body of this report. Absolute risk projection models have been used to predict risks of several cancers—including leukemia, bone cancer and thyroid cancer. The parameters of an absolute risk projection model are the latency period, l, the plateau (or expression) period, p, and the absolute risk coefficient, r_a. The risk coefficient indicates the absolute increase in risk expected in each year during the expression period following a 1 Gy dose. Relative risk projection models have been used to derive several of our risk estimates—including the central estimates of breast cancer, lung cancer, gastrointestinal cancer and other cancers. The parameters of a relative risk projection model are the latency period, l, the plateau period, p, and the relative risk coefficient, r_r. The relative risk coefficient indicates the increase in risk—expressed as a percentage of the spontaneous age-specific risk— expected during each year of the expression period following a 1 Gy dose. The background cancer mortality rates used in our calculations are taken from the 1978 Vital Statistics of the United States [NCHS, 1981]. The background incidence rates are from NCI Monograph 57 [NCI, 1981]. These baseline cancer rates are provided in Appendix A.

To estimate the fraction of an exposed population that will eventually develop (or die from) radiation-induced cancer following a dose, d, it is necessary to evaluate:

$$R(d) = \sum_{\gamma} R(\gamma,d)$$

This approach is the one used to develop the models of cancer risk described in the section on late somatic effects.

One situation that deserves special attention is analysis of risk associated with radionuclides inhaled from an airborne plume. Several of the radionuclides that could be released in the event of a nuclear power plant accident have relatively long half-lives. Rather than delivering their dose immediately, these materials will continue to decay for several years after they are inhaled and will deliver dose gradually. As time proceeds, the population of individuals who were alive at the time of the accident will age. Gradually the size of the exposed population will dwindle. Direct application of the basic risk models, which assume a stable age structure, would lead to overestimation of the risks faced by this population.

The modifications necessary to account for these factors are relatively simple. The fraction of the population exposed to the plume expected to

survive all other causes of death for t years after the accident is:

$$F(t) = \sum_k f_{k-t} \; s_{k-t}(t)$$

where f_{k-t} is the fraction of the population in the k-t^{th} age group at the time of the accident, $s_{k-t}(t)$ is the fraction of the k-t^{th} age group expected to be alive t years after the accident, and $f_{k-t} = 0$ when $k < t$. Based on 1980 U.S. vital statistics and census data, it appears that approximately 85 percent of the exposed population would survive 20 years; 65 percent would survive 40 years; 40 percent would survive 60 years; and about 15 percent would survive 80 years.

The changing age structure of the surviving population may be evaluated using:

$$f_k(t) = f_{k-t} \; s_{k-t}(t)/F(t)$$

The risks among the survivors are then computed by substitution of $f_k(t)$ for f_k in the equations given above for R (γ,d) and R (d). The results of these calculations are presented in tabular form in Appendix B.

Figure 3.14 illustrates the results. Two sets of values are plotted—one for leukemia and another for gastrointestinal cancer (lower estimate). These bound the results for all other cancers. The impact of time is somewhat greater for gastrointestinal cancer (absolute risk projection—lifetime expression period) than for leukemia (absolute risk projection—25-year expression period). However, the most striking feature of the graph is the similarity in the time dependence of risk for most cancers.

The degree of overestimation that would occur if risk calculations were made without these modifications would depend on the half-life of the radionuclide of interest. The bias would be greatest for radionuclides with long half-lives. It is worth noting, however, that in the limiting case, i.e., infinite half-life, the maximum possible bias would be a factor of 3. For many radionuclides and cancer types the effect would be smaller than this.

3.3 Genetic Effects

One of the key factors influencing the number of genetic defects observed is the birth rate. In 1980 in the United States there were some 3.6 million births in a population of approximately 226 million. A second important demographic factor is the characteristic intergenerational interval. In 1980 in the United States the mean age of a mother was about 26 years. Figure 3.15 shows the distribution of births by age of mother.

To estimate the number of children born with genetic defects in the first generation after the accident, one multiplies the total number of births expected by the risk that a child will suffer from genetic disease. In a
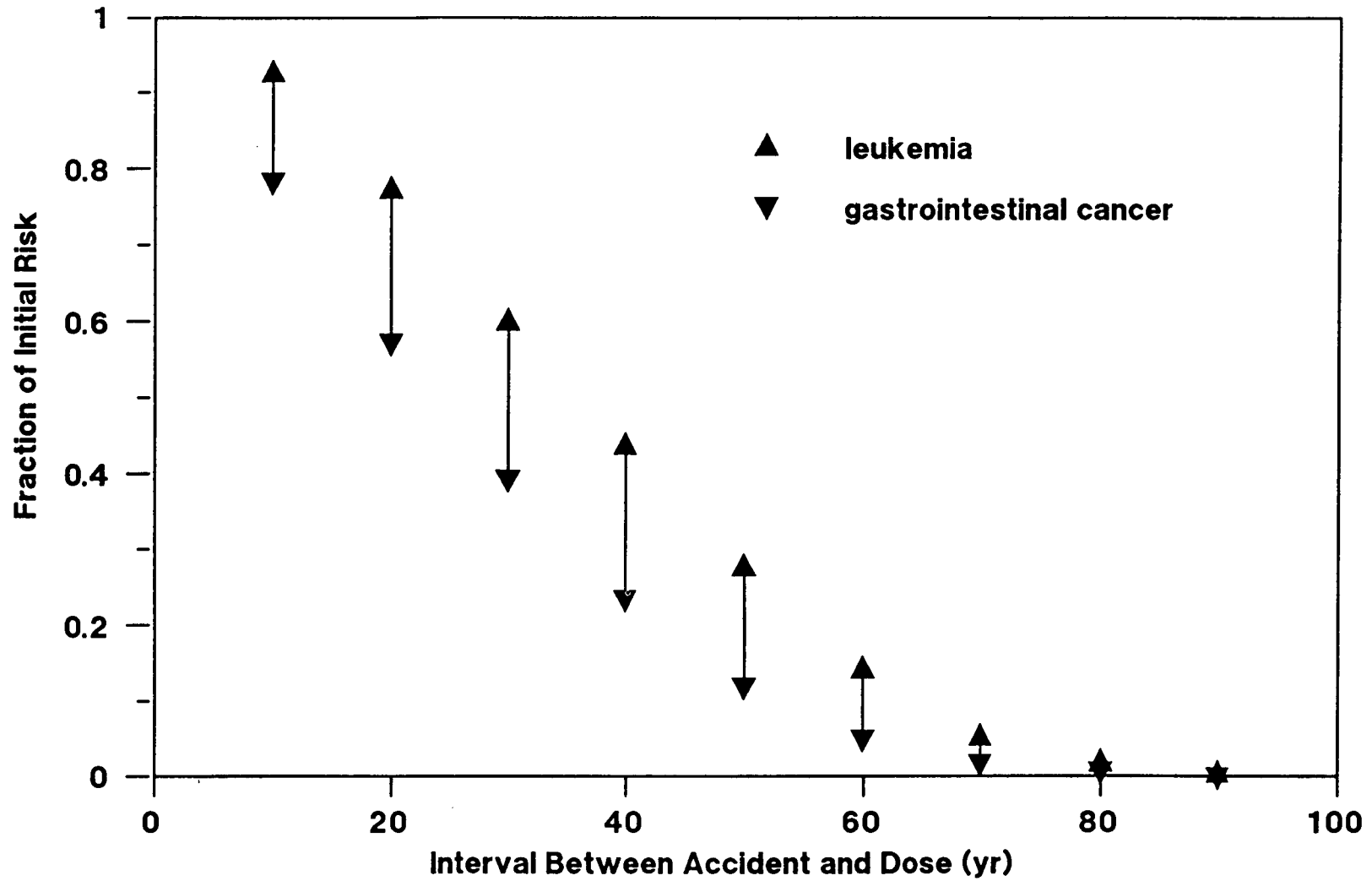
Figure 3.14 Risk Among Those Inhaling Radionuclides as a Function of Time Dose is Received
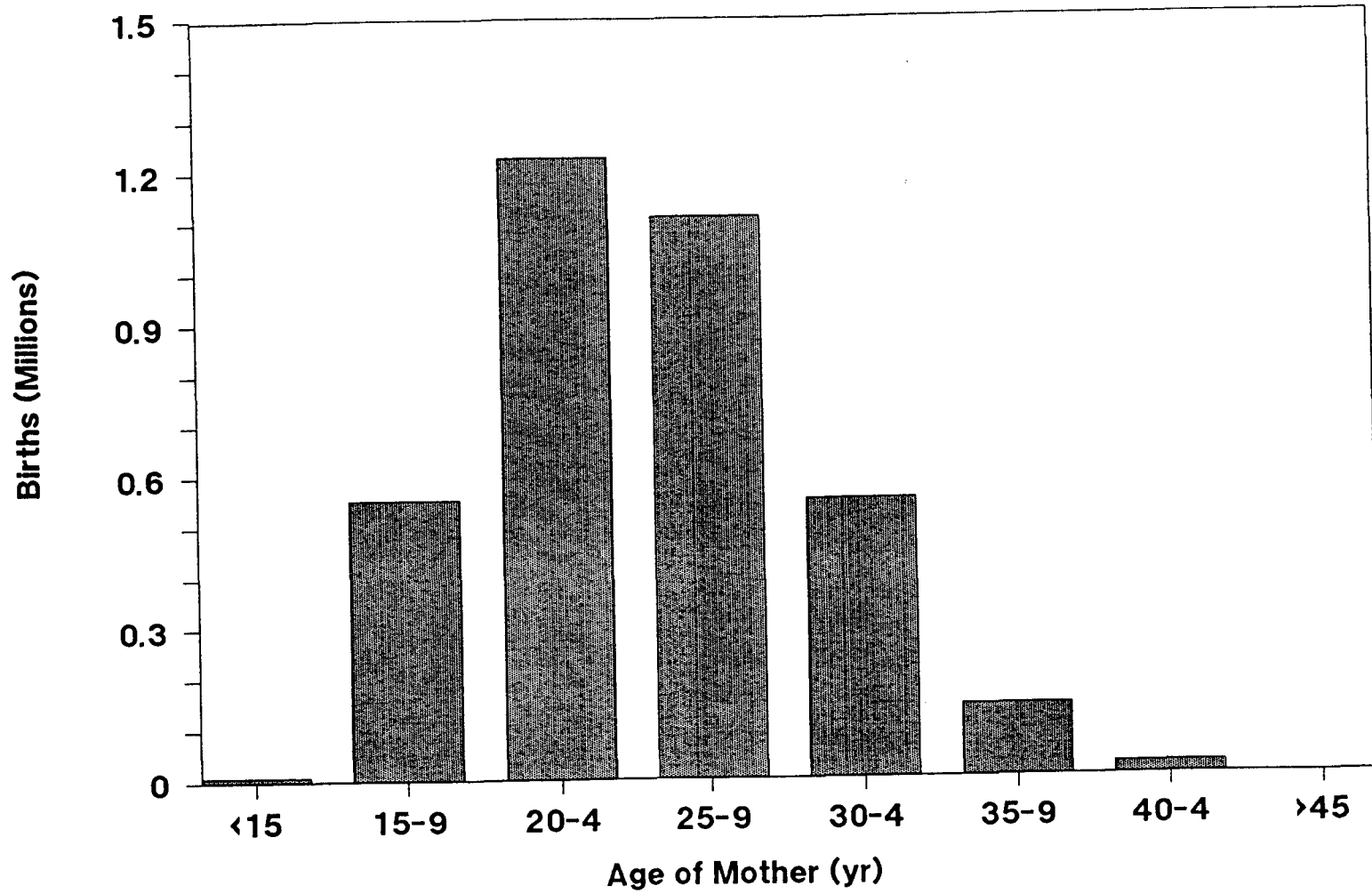
Figure 3.15 Live Births – US – 1980

stable population the number of children born each generation is approximately the product of the birthrate, the intergenerational interval and the size of the population. In the first generation, the risk that a child will suffer from radiation-induced genetic disease is a function of the doses received by his parents. Using the 1980 U.S. birthrate and an intergenerational interval of 30 years, the number of radiation-induced genetic defects in the first generation would be:

$$N_1 = 0.5 \ P \ r(d)$$

where P is the population size and r(d) is the function relating the child's risk to his parents' doses. In a stable population the number of children born with radiation-induced genetic defects in the second, third, or k^{th} generation would be:

$$N_2 = N_1 \ T$$

$$N_3 = N_2 \ T = N_1 \ T^2$$

$$\cdot$$
$$\cdot$$
$$\cdot$$

$$N_k = N_{k-1} \ T = N_{k-2} \ T^2 = \ . \ . \ . \ = N_1 \ T^{k-1}$$

where T is the intergenerational transmission rate—i.e., the fraction of genetic damage transmitted from one generation to the next. Under these assumptions, the integrated risk—i.e., the number of children born in the first and all subsequent generations with genetic defects—is given by:

$$N = \Sigma_k \ N_k = N_1 \ \Sigma_k \ T^{k-1} = [1/(1 - T)] \ N_1$$

This relatively simple approach is directly applicable for estimating the cumulative risk of genetic effects with simple patterns of transmission in stable populations. Some modifications are necessary to allow for more complex patterns of inheritance or for change in the population size.

For example, in the analysis of x-linked effects it is necessary to divide the birthrate by two to account for the fact that such effects occur only in boys. Similarly, when computing the cumulative impact of unbalanced translocations, one must allow for the dynamics of transmission and expression of these defects; i.e., the second generation experiences only $1/4^{th}$ of the risk faced by the first generation, but in each succeeding generation the risk diminishes by 50 percent.

If the population is expected to grow or dwindle, additional modifications are necessary. With a constant growth rate, G (fractional change per generation), the cumulative impact of genetic disease may be estimated using:

$$N = N_1 \Sigma_k (GT)^{k-1} = [1/(1 - GT)] N_1$$

Note that as long as the product, GT, is less than 1 the series will converge.

Figure 3.16 shows the growth of the population of the United States since 1800. Although the average rate of growth over the 200-year period has been 2.8 percent per year, since 1900 the growth rate has been more moderate, i.e., about 1.4 percent per year. Most of the increase has been due to a natural increase of births over deaths. Only 50 million of the over 200 million increase in population since 1800 is due to immigration. Currently the immigration component of population growth is only about 0.2 percent per year.

To apply this model of genetic impact, it is necessary to derive the risk function $r(d_o, d_t)$ from the gametic induction rates given by the genetic effects working group. For most effects, the fraction of children in the first generation who will be affected is estimated using:

$$r(d_o, d_t) = r_m(d_o) + r_p(d_t)$$

where $r_m(d_o)$ is the maternal gametic induction function computed on the basis of the dose to the ovary, d_o, and $r_p(d_t)$ is the paternal gametic induction function based on the dose to the testis, d_t. There are some exceptions. For example, in the analysis of first generation x-linked effects the paternal gametic damage is irrelevant because the boys who are at risk inherit their X chromosome from their mothers.

In the event of a nuclear power plant accident, there could be a wide distribution of gonadal doses among the pool of prospective parents within the exposed population. If all of the individual doses were received at low dose rates, or if they were all below 0.5 Gy, then it would be appropriate to compute the genetic risk on the basis of the average maternal and paternal doses. Otherwise, it would be necessary to evaluate the general linear-quadratic gametic damage functions separately for each dose group and to combine these using weights based on the fraction of the population in each dose group.

Genetic risks are commonly expressed in one of three ways. Sometimes the risk is expressed in terms of its impact on the prevalence of genetic effects among the children born in a specific generation after an accident, i.e., number of chidren with defects per 1000 children born in the kth generation. Alternatively an estimate of prevalence may be combined
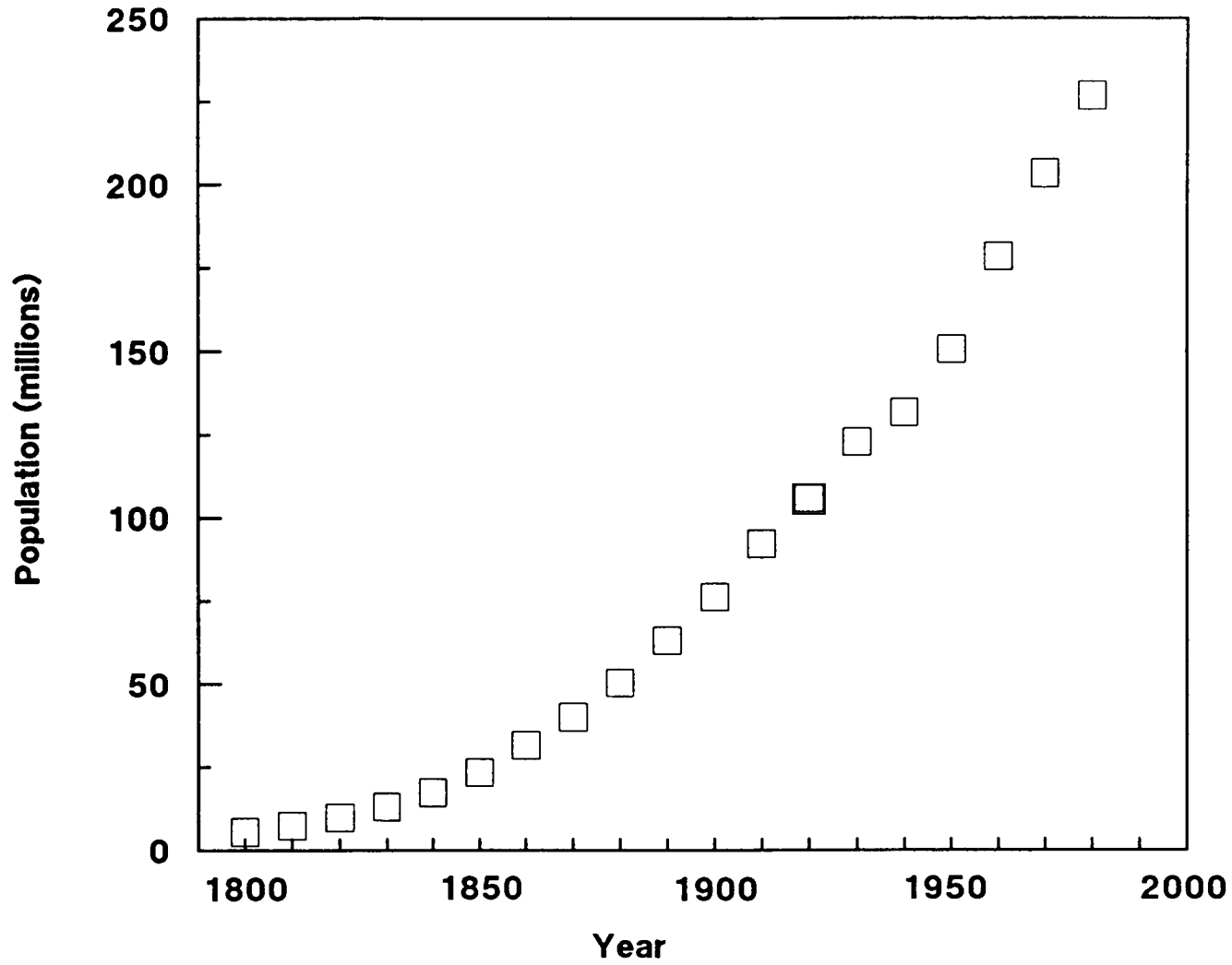
Figure 3.16 Population of the United States, 1800-2000

with an estimate of the birthrate to derive an estimate of the number of children born with genetic defects in a population of a certain size during a specific time interval, e.g., number of children born with genetic defects per year (or per generation) per million persons exposed. Finally, it is possible to express the risk in terms of its cumulative impact, i.e., the number of children that will be born with genetic defects in all future generations as a result of the exposure caused by the accident. Typically cumulative risk estimates are expressed in terms of the number of genetic effects per million persons exposed.

APPENDIX A

Base-Line Demographic and Mortality Data

Table A.1

Population of the U.S. (1000's) - By Single Years of Age*

Age	Both	Male	Female	Age	Both	Male	Female
		Sex				Sex	
0	3534	1806	1727	35	2902	1430	1472
1	3270	1674	1595	6	2929	1439	1490
2	3224	1648	1576	7	2983	1465	1518
3	3179	1626	1554	8	2599	1273	1326
4	3142	1608	1534	9	2553	1254	1298
5	3163	1618	1544	40	2468	1209	1259
6	3109	1589	1520	1	2376	1164	1212
7	3273	1673	1600	2	2326	1139	1186
8	3395	1736	1659	3	2237	1092	1145
9	3760	1923	1837	4	2263	1104	1159
10	3717	1902	1815	45	2242	1094	1148
1	3581	1829	1752	6	2139	1040	1099
2	3519	1796	1723	7	2223	1077	1146
3	3643	1857	1787	8	2164	1052	1112
4	3783	1933	1850	9	2321	1125	1196
15	4060	2070	1990	50	2347	1134	1213
6	4181	2135	2046	1	2295	1106	1189
7	4224	2160	2064	2	2363	1137	1226
8	4252	2153	2098	3	2337	1119	1218
9	4452	2237	2215	4	2368	1125	1243
20	4387	2200	2187	55	2390	1130	1260
1	4286	2145	2141	6	2330	1102	1227
2	4284	2145	2139	7	2313	1092	1221
3	4200	2097	2103	8	2330	1100	1231
4	4162	2077	2085	9	2252	1058	1194
25	4116	2053	2064	60	2161	1010	1151
6	3978	1979	1999	1	2074	964	1110
7	3932	1952	1980	2	2008	931	1077
8	3709	1840	1869	3	1931	889	1042
9	3787	1881	1905	4	1913	876	1037
30	3727	1847	1880	65	1905	863	1042
1	3608	1781	1826	6	1814	814	1000
2	3712	1833	1879	7	1764	784	979
3	3654	1805	1849	8	1679	740	939
4	2861	1411	1449	9	1621	702	920

Table A.1

Population of the U.S. (1000's) - By Single Years of Age*
(Concluded)

Age	Sex			Age	Sex		
	Both	Male	Female		Both	Male	Female
70	1517	653	863	85	413	135	278
1	1440	612	828	6	351	112	239
2	1371	577	794	7	307	96	211
3	1262	521	741	8	236	72	164
4	1208	490	718	9	214	63	151
75	1111	443	668	90-4	557	159	398
6	1029	406	623				
7	952	367	585	95-9	131	35	96
8	829	315	514				
9	873	318	555	>100	32	10	22
80	723	261	462				
1	640	224	416				
2	567	197	370				
3	528	179	349				
4	477	158	319				

*Source - Bureau of the Census (1983): General Population Characteristics, United States Summary, 1980 Census of Population. Data are from Table 41.

Table A.2

Life Table*,**

Age	Both Sexes		Males		Females	
	Number Alive	Life Expectancy	Number Alive	Life Expectancy	Number Alive	Life Expectancy
0	100,000	73.9	100,000	70.1	100,000	77.6
1	98,740	73.8	98,607	70.1	98,880	77.5
2	98,648	72.9	98,508	69.2	98,796	76.6
3	98,584	71.9	98,436	68.2	98,740	75.6
4	98,535	71.0	98,379	67.3	98,699	74.6
5	98,495	70.0	98,333	66.3	98,666	73.7
6	98,459	69.0	98,291	65.3	98,636	72.7
7	98,426	68.1	98,252	64.4	98,609	71.7
8	98,396	67.1	98,217	63.4	98,585	70.7
9	98,370	66.1	98,186	62.4	98,563	69.7
10	98,347	65.1	98,160	61.4	98,544	68.8
1	98,328	64.1	98,139	60.4	98,527	67.8
2	98,309	63.1	98,119	59.4	98,509	66.8
3	98,285	62.1	98,090	58.5	98,489	65.8
4	98,248	61.2	98,043	57.5	98,464	64.8
15	98,196	60.2	97,972	56.5	98,432	63.8
6	98,129	59.2	97,878	55.6	98,392	62.9
7	98,047	58.3	97,762	54.6	98,346	61.9
8	97,953	57.3	97,628	53.7	98,294	60.9
9	97,851	56.4	97,479	52.8	98,240	60.0
20	97,741	55.5	97,316	51.9	98,184	59.0
1	97,623	54.5	97,141	51.0	98,127	58.0
2	97,499	53.6	96,952	50.1	98,068	57.1
3	97,370	52.7	96,756	49.2	98,007	56.1
4	97,240	51.7	96,557	48.3	97,946	55.1
25	97,110	50.8	96,361	47.4	97,883	54.2
6	96,982	49.9	96,169	46.5	97,820	53.2
7	96,856	48.9	95,980	45.6	97,755	52.2
8	96,730	48.0	95,795	44.6	97,689	51.3
9	96,604	47.1	95,612	43.7	97,621	50.3
30	96,477	46.1	95,430	42.8	97,551	49.3
1	96,350	45.2	95,247	41.9	97,477	48.4
2	96,220	44.2	95,066	41.0	97,400	47.4
3	96,088	43.3	94,882	40.1	97,319	46.5
4	95,951	42.4	94,695	39.1	97,233	45.5

Table A.2

Life Table*,**
(Continued)

	Both Sexes		Males		Females	
Age	Number Alive	Life Expectancy	Number Alive	Life Expectancy	Number Alive	Life Expectancy
35	95,808	41.4	94,501	38.2	97,140	44.5
6	95,655	40.5	94,297	37.3	97,039	43.6
7	95,492	39.6	94,081	36.4	96,928	42.6
8	95,317	38.6	93,852	35.5	96,807	41.7
9	95,129	37.7	93,607	34.6	96,675	40.7
40	94,926	36.8	93,345	33.6	96,531	39.8
1	94,706	35.9	93,062	32.7	96,374	38.9
2	94,465	35.0	92,754	31.9	96,200	37.9
3	94,201	34.1	92,417	31.0	96,009	37.0
4	93,913	33.2	92,049	30.1	95,799	36.1
45	93,599	32.3	91,649	29.2	95,570	35.2
6	93,256	31.4	91,213	28.4	95,230	34.3
7	92,882	30.5	90,737	27.5	95,047	33.4
8	92,472	29.7	90,214	26.7	94,748	32.5
9	92,021	26.8	89,639	25.8	94,419	31.6
50	91,526	27.9	89,007	25.0	94,060	30.7
1	90,986	27.1	88,317	24.2	93,669	29.8
2	90,402	26.3	87,570	23.4	93,245	29.0
3	89,771	25.5	86,761	22.6	92,788	28.1
4	89,087	24.7	85,885	21.8	92,294	27.2
55	88,348	23.9	84,936	21.1	91,760	26.4
6	87,551	23.1	83,912	20.3	91,185	25.6
7	86,695	22.3	82,813	19.6	90,567	24.7
8	85,776	21.5	81,634	18.8	89,903	23.9
9	84,789	20.8	80,370	18.2	89,187	23.1
60	83,726	20.0	79,012	17.5	88,414	22.3
1	82,581	19.3	77,553	16.8	87,577	21.5
2	81,348	18.6	75,990	16.1	86,670	20.7
3	80,024	17.9	74,317	15.5	85,691	20.0
4	78,609	17.2	72,535	14.8	84,641	19.2

Table A.2

Life Table*,**
(Continued)

	Both Sexes		Males		Females	
Age	Number Alive	Life Expectancy	Number Alive	Life Expectancy	Number Alive	Life Expectancy
65	77,107	16.5	70,646	14.2	83,520	18.4
6	75,520	15.9	68,656	13.6	82,328	17.7
7	73,846	15.2	66,566	13.0	81,061	17.0
8	72,082	14.6	64,377	12.5	79,712	16.3
9	70,218	13.9	62,083	11.9	78,269	15.5
70	68,248	13.3	59,681	11.4	76,720	14.8
1	66,165	12.7	57,171	10.8	75,055	14.2
2	63,972	12.1	54,557	10.3	73,273	13.5
3	61,673	11.6	51,856	9.8	71,368	12.8
4	59,279	11.0	49,088	9.4	69,340	12.2
75	56,799	10.5	46,272	8.9	67,186	11.6
6	54,239	10.0	43,419	8.5	64,910	11.0
7	51,599	9.4	40,533	8.0	62,506	10.4
8	48,878	8.9	37,626	7.6	59,960	9.8
9	46,071	8.5	34,714	7.2	57,253	9.2
80	43,180	8.0	31,810	6.8	54,372	8.7
1	40,208	7.5	28,925	6.4	51,315	8.2
2	37,172	7.1	26,074	6.1	48,098	7.7
3	34,095	6.7	23,282	5.8	44,744	7.2
4	31,012	6.3	20,586	5.4	41,289	6.8
85	27,960	6.0	18,020	5.1	37,772	6.4
6	24,961	5.6	15,602	4.9	34,218	6.0
7	22,038	5.3	13,343	4.6	30,657	5.6
8	19,235	5.0	11,268	4.3	27,156	5.3
9	16,598	4.7	9,395	4.1	23,782	5.0
90	14,154	4.4	7,732	3.9	20,578	4.7
1	11,908	4.2	6,275	3.7	17,561	4.4
2	9,863	3.9	5,012	3.5	14,747	4.1
3	8,032	3.7	3,932	3.3	12,172	3.9
4	6,424	3.5	3,025	3.1	9,871	3.7

Table A.2

Life Table*,**
(Concluded)

	Both Sexes		Males		Females	
Age	Number Alive	Life Expectancy	Number Alive	Life Expectancy	Number Alive	Life Expectancy
95	5,043	3.3	2,279	3.0	7,862	3.5
6	3,884	3.2	1,683	2.9	6,147	3.3
7	2,939	3.1	1,222	2.8	4,719	3.2
8	2,185	2.9	871	2.7	3,560	3.0
9	1,598	2.8	612	2.6	2,641	2.9

*Source - National Center for Health Statistics (1985): U.S. Decennial
Life Tables for 1979-1981, Volume I, Number 1, United States
Life Tables. Data are from Tables 1, 2, and 3.

**The entries in the body of the table are the number of survivors
expected in a hypothetical cohort of 100,000 and the remaining life
expectancy (yr) at each single year of age.

Table A.3

Cancer Mortality Rates (Deaths/100,000 Per Year)

Age	Mortality Rate[a]				
	Breast[b] Cancer	Lung Cancer	Gastrointestinal Cancer	All[c] Cancers	Other[d] Cancers
0-4	--	--	0.2	3.1	2.9
5-9	--	--	0.1	2.2	2.1
10-14	--	--	0.1	1.8	1.7
15-19	--	--	0.2	2.9	2.7
20-24	0.2	0.1	0.4	4.5	3.9
25-29	1.2	0.3	1.0	7.8	5.9
30-34	5.6	1.3	2.4	14.7	8.2
35-39	11.7	4.8	5.2	28.3	12.3
40-44	22.9	15.1	11.8	62.3	23.6
45-49	41.4	36.2	25.0	124.1	41.6
50-54	60.1	70.6	48.1	219.5	69.5
55-59	75.9	110.2	79.1	333.1	103.9
60-64	91.4	166.4	133.1	505.6	157.1
65-69	89.9	201.3	184.8	633.4	196.8
70-74	110.7	238.2	266.8	829.6	260.0
75-79	128.4	245.0	376.3	1041.1	340.8
80-84	139.9	218.3	467.4	1171.4	394.4
85-89	157.2	147.1	513.3	1178.5	408.6

[a]Source - 1978 Vital Statistics of the United States, (NCHS, 1981).

[b]These are the rates among women.

[c]Excluding leukemia and cancers of the bone, skin, thyroid and prostate.

[d]All cancers minus cancers of the breast, lung and gastrointestinal tract.

Table A.4

Cancer Incidence Rates (New Cases/100,000 Per Year)

Age	Incidence Rate[a]				
	Breast[b] Cancer	Lung Cancer	Gastrointestinal Cancer	All[c] Cancers	Other[d] Cancers
0-4	--	--	0.7	10.2	9.5
5-9	--	--	0.2	5.8	5.6
10-14	--	0.1	0.3	6.5	6.1
15-19	0.2	0.2	0.5	11.5	10.7
20-24	1.1	0.2	1.3	20.4	18.3
25-29	8.3	0.7	2.4	33.2	25.9
30-34	26.7	2.3	5.5	55.4	34.1
35-39	57.2	7.1	11.9	93.5	45.3
40-44	106.2	20.4	24.9	170.4	70.6
45-49	173.8	47.7	50.2	300.6	113.7
50-54	195.9	79.8	89.4	457.3	187.2
55-59	228.9	130.2	155.5	682.1	277.6
60-64	251.2	185.6	240.5	910.5	351.8
65-69	282.9	235.5	351.2	1163.4	420.1
70-74	302.0	258.5	475.2	1399.4	489.6
75-79	338.0	255.9	617.9	1646.9	564.4
80-84	350.0	211.4	708.9	1733.3	586.2
85-89	376.3	166.0	795.6	1831.0	611.3

[a]Source - Cancer Incidence and Mortality in the U.S., 1973-7 (NCI, 1981).

[b]These are the rates among women.

[c]Excluding leukemia and cancers of the bone, skin, thyroid and prostate.

[d]All cancers minus cancers of the breast, lung and gastrointestinal tract.

APPENDIX B - Part I

Cancer Mortality Models for Those Exposed to the Plume

The tables that follow give estimates of risk for the population exposed
to radionuclides inhaled from an airborne plume. The need for these
tables and the methods used to develop the numbers in them are described
in Section 3.2. The two columns at the left of each table indicate the
risk associated with a 1 Gy dose received in each of ten 10-year time
intervals after the accident. Because these doses are assumed to be
delivered at low dose rates, the risk values reflect only the linear
terms of the dose response models described in the text and summarized in
Table 2.7 and 2.8. The numbers in the body of the table indicate the
percentage of this risk expressed in each time interval. Minus (-)
indicates <1 percent. Plus (+) indicates a time period prior to receipt
of dose and therefore contains no risk.

For some cancers, the same assumptions about latency, plateau and risk
projection are used in the central, lower and upper models. For these
cancers, i.e., leukemia, bone, thyroid, skin, and all cancers due to in
utero exposure, the dynamics of population risk do not depend on which
model is used. Therefore, the only tables provided for these cancers are
those for the central estimates of risk.

Table B-I.1

Leukemia Mortality - Central Estimate*

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.4e-3	35	40	25	-	-	-	-	-	-	-
10-19	1.3e-3	+	36	40	24	-	-	-	-	-	-
20-29	1.1e-3	+	+	37	40	23	-	-	-	-	-
30-39	8.6e-4	+	+	+	39	40	21	-	-	-	-
40-49	6.3e-4	+	+	+	+	43	39	18	-	-	-
50-59	4.0e-4	+	+	+	+	+	48	38	14	-	-
60-69	2.1e-4	+	+	+	+	+	+	56	35	9	-
70-79	7.9e-5	+	+	+	+	+	+	+	69	28	3
80-89	1.8e-5	+	+	+	+	+	+	+	+	86	14
90-99	1.7e-6	+	+	+	+	+	+	+	+	+	100

*Multiplication of the central estimates of lifetime risk by 10/3 gives upper estimates; division by 3 gives lower estimates.

Table B-I.2

Bone Cancer Mortality - Central Estimate*

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	6.0e-5	35	40	25	-	-	-	-	-	-	-
10-19	5.6e-5	+	36	40	24	-	-	-	-	-	-
20-29	4.8e-5	+	+	37	40	23	-	-	-	-	-
30-39	3.8e-5	+	+	+	39	40	21	-	-	-	-
40-49	2.8e-5	+	+	+	+	43	39	18	-	-	-
50-59	1.8e-5	+	+	+	+	+	48	38	14	-	-
60-69	9.3e-6	+	+	+	+	+	+	56	35	9	-
70-79	3.5e-6	+	+	+	+	+	+	+	69	28	3
80-89	8.1e-7	+	+	+	+	+	+	+	+	86	14
90-99	1.2e-7	+	+	+	+	+	+	+	+	+	100

*Multiplication of the central estimates of lifetime risk by 10/3 gives upper estimates; division by 3 gives lower estimates.

Table B-I.3

Breast Cancer Mortality - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	6.0e-3	-	12	14	17	18	16	13	7	3	-
10-19	5.3e-3	+	-	17	19	20	19	14	8	3	-
20-29	4.4e-3	+	+	-	23	24	22	17	10	4	-
30-39	3.4e-3	+	+	+	-	31	29	22	13	4	1
40-49	2.4e-3	+	+	+	+	-	42	32	19	6	1
50-59	1.4e-3	+	+	+	+	+	-	55	32	11	2
60-69	6.1e-4	+	+	+	+	+	+	-	72	25	3
70-79	1.7e-4	+	+	+	+	+	+	+	-	90	10
80-89	1.8e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.4

Lung Cancer Mortality - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.0e-3	-	12	14	17	19	18	13	6	1	-
10-19	1.8e-3	+	-	16	19	21	20	15	7	2	-
20-29	1.5e-3	+	+	-	22	25	24	17	9	3	-
30-39	1.2e-3	+	+	+	-	33	31	22	11	3	-
40-49	7.7e-4	+	+	+	+	-	46	33	16	5	-
50-59	4.2e-4	+	+	+	+	+	-	62	30	7	1
60-69	1.6e-4	+	+	+	+	+	+	-	79	19	2
70-79	3.4e-5	+	+	+	+	+	+	+	-	91	9
80-89	3.0e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.5

Gastrointestinal Cancer Mortality - Central Estimate

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr) 0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.7e-3	-	11	13	14	17	17	15	9	4	-
10-19	4.9e-3	+	-	14	16	19	20	17	10	4	-
20-29	4.2e-3	+	+	-	19	22	23	20	12	4	-
30-39	3.4e-3	+	+	+	-	27	28	24	15	6	-
40-49	2.5e-3	+	+	+	+	-	38	33	21	7	1
50-59	1.5e-3	+	+	+	+	+	-	53	33	12	2
60-69	7.2e-4	+	+	+	+	+	+	-	71	26	3
70-79	2.1e-4	+	+	+	+	+	+	+	-	89	11
80-89	2.3e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.6

Thyroid Cancer Mortality - Central Estimate*

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr) 0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	7.0e-4	11	20	18	16	14	10	7	3	1	-
10-19	5.5e-4	+	12	23	20	17	13	9	5	1	-
20-29	4.2e-4	+	+	15	26	22	17	12	6	2	-
30-39	3.0e-4	+	+	+	18	31	24	16	8	3	-
40-49	2.0e-4	+	+	+	+	23	37	24	12	4	-
50-59	1.2e-4	+	+	+	+	+	29	42	21	7	1
60-69	5.6e-5	+	+	+	+	+	+	39	45	14	2
70-79	1.9e-5	+	+	+	+	+	+	+	53	42	5
80-89	3.4e-6	+	+	+	+	+	+	+	+	72	28
90-99	2.0e-7	+	+	+	+	+	+	+	+	+	100

*Upper and lower estimates of lifetime risk differ only in the treatment of internal sources such as [131]I. See Section 2.2.6.

Table B-I.7

Other* Cancer Mortality - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.9e-3	-	12	14	15	17	17	14	8	3	-
10-19	2.5e-3	+	-	16	18	19	19	16	9	3	-
20-29	2.1e-3	+	+	-	21	23	23	18	11	4	-
30-39	1.7e-3	+	+	+	-	29	29	23	14	5	-
40-49	1.2e-3	+	+	+	+	-	41	33	19	6	1
50-59	7.0e-4	+	+	+	+	+	-	55	33	11	1
60-69	3.2e-4	+	+	+	+	+	+	-	72	25	3
70-79	8.7e-5	+	+	+	+	+	+	+	-	89	11
80-89	9.3e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

Table B-I.8

Leukemia* In Utero Mortality - Central Estimate**

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.2e-4	83	17	-	-	-	-	-	-	-	-
10-19	1.2e-4	+	83	17	-	-	-	-	-	-	-
20-29	1.0e-4	+	+	83	17	-	-	-	-	-	-
30-39	2.4e-5	+	+	+	83	17	-	-	-	-	-
40-49	8.1e-7	+	+	+	+	83	17	-	-	-	-
50-59	-	+	+	+	+	+	-	-	-	-	-
60-69	-	+	+	+	+	+	+	-	-	-	-
70-79	-	+	+	+	+	+	+	+	-	-	-
80-89	-	+	+	+	+	+	+	+	+	-	-
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risks apply to the entire population. Risks to the children exposed in utero would be 100 times this large.

**Multiplication of the central estimates of lifetime risk by 10/4 gives upper estimates. Lower estimates are identical to central estimates.

Table B-I.9

Other Cancer* In Utero Mortality - Central Estimate**

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.2e-4	91	9	-	-	-	-	-	-	-	-
10-19	1.2e-4	+	91	9	-	-	-	-	-	-	-
20-29	1.0e-4	+	+	91	9	-	-	-	-	-	-
30-39	2.4e-5	+	+	+	91	9	-	-	-	-	-
40-49	8.1e-7	+	+	+	+	91	9	-	-	-	-
50-59	-	+	+	+	+	+	-	-	-	-	-
60-69	-	+	+	+	+	+	+	-	-	-	-
70-79	-	+	+	+	+	+	+	+	-	-	-
80-89	-	+	+	+	+	+	+	+	+	-	-
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risks apply to the entire population. Risks to the children exposed in utero would be 100 times this large.

**Multiplication of the central estimates of lifetime risk by 10/4 gives upper estimates. Lower estimates are identical to central estimates.

Table B-I.10

Breast* Cancer Mortality - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	4.3e-4	-	16	19	20	17	13	8	5	2	-
10-19	3.7e-4	+	-	23	24	20	16	10	5	2	-
20-29	2.8e-4	+	+	-	31	27	20	13	7	2	-
30-39	1.9e-4	+	+	+	-	39	29	19	10	3	-
40-49	1.2e-4	+	+	+	+	-	48	31	16	5	-
50-59	6.2e-5	+	+	+	+	+	-	60	30	10	-
60-69	2.5e-5	+	+	+	+	+	+	-	75	23	2
70-79	6.4e-6	+	+	+	+	+	+	+	-	90	10
80-89	6.4e-7	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risk estimates apply to the entire population. Risks for women would be twice this large.

Table B-I.11

Lung Cancer Mortality - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.3e-4	-	16	19	20	18	13	8	4	2	-
10-19	4.7e-4	+	-	22	24	22	16	10	5	1	-
20-29	3.6e-4	+	+	-	31	28	20	13	6	2	-
30-39	2.5e-4	+	+	+	-	41	30	18	9	2	-
40-49	1.5e-4	+	+	+	+	-	50	31	15	4	-
50-59	7.4e-5	+	+	+	+	+	-	61	29	9	1
60-69	2.9e-5	+	+	+	+	+	+	-	75	23	2
70-79	7.2e-6	+	+	+	+	+	+	+	-	90	10
80-89	7.6e-7	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.12

Gastrointestinal Cancer Mortality - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	9.1e-4	-	24	22	18	15	11	6	3	1	-
10-19	7.1e-4	+	-	29	24	19	14	9	4	1	-
20-29	5.1e-4	+	+	-	34	27	20	12	6	1	-
30-39	3.4e-4	+	+	+	-	41	30	18	9	2	-
40-49	2.0e-4	+	+	+	+	-	50	31	15	4	-
50-59	1.0e-4	+	+	+	+	+	-	61	29	9	1
60-69	3.9e-5	+	+	+	+	+	+	-	75	23	2
70-79	9.3e-6	+	+	+	+	+	+	+	-	90	10
80-89	1.0e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.13

Other* Cancer Mortality - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.0e-4	-	24	22	18	15	11	6	3	1	-
10-19	4.0e-4	+	-	29	24	19	14	9	4	1	-
20-29	2.8e-4	+	+	-	34	27	20	12	6	1	-
30-39	1.9e-4	+	+	+	-	41	30	18	9	2	-
40-49	1.1e-4	+	+	+	+	-	50	31	15	4	-
50-59	5.6e-5	+	+	+	+	+	-	61	29	9	1
60-69	2.2e-5	+	+	+	+	+	+	-	75	23	2
70-79	5.4e-6	+	+	+	+	+	+	+	-	90	10
80-89	5.7e-7	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

Table B-I.14

Breast* Cancer Mortality - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	8.7e-3	-	8	10	14	18	19	16	11	4	-
10-19	7.9e-3	+	-	11	15	19	20	18	12	4	1
20-29	7.0e-3	+	+	-	17	22	23	20	13	4	1
30-39	5.9e-3	+	+	+	-	26	27	24	16	6	1
40-49	4.4e-3	+	+	+	+	-	37	33	22	7	1
50-59	2.7e-3	+	+	+	+	+	-	52	34	12	2
60-69	1.3e-3	+	+	+	+	+	+	-	71	26	3
70-79	3.8e-4	+	+	+	+	+	+	+	-	89	11
80-89	4.0e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risk estimates apply to the entire population. Risks for women would be twice this large.

Table B-I.15

Lung Cancer Mortality - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.4e-2	-	8	9	13	19	22	18	9	2	-
10-19	1.8e-2	+	-	12	15	19	21	19	11	3	-
20-29	1.2e-2	+	+	-	24	27	24	16	7	2	-
30-39	1.1e-2	+	+	+	-	35	32	22	9	2	-
40-49	8.7e-3	+	+	+	+	-	49	33	15	3	-
50-59	5.9e-3	+	+	+	+	+	-	64	29	6	1
60-69	3.2e-3	+	+	+	+	+	+	-	80	18	2
70-79	1.2e-3	+	+	+	+	+	+	+	-	91	9
80-89	3.0e-4	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.16

Gastrointestinal Cancer Mortality - Upper Estimate

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	3.3e-2	-	7	8	11	15	19	21	14	5	-
10-19	2.5e-2	+	-	11	13	16	19	19	15	6	1
20-29	1.7e-2	+	+	-	20	23	23	19	11	4	-
30-39	1.6e-2	+	+	+	-	28	29	24	14	4	1
40-49	1.4e-2	+	+	+	+	-	40	33	20	6	1
50-59	1.1e-2	+	+	+	+	+	-	55	33	11	1
60-69	7.5e-3	+	+	+	+	+	+	-	72	25	3
70-79	3.9e-3	+	+	+	+	+	+	+	-	90	10
80-89	1.1e-3	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-I.17

Other* Cancer Mortality - Upper Estimate

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.7e-2	-	8	9	12	16	20	19	12	4	-
10-19	1.3e-2	+	-	12	14	17	19	18	13	6	1
20-29	8.6e-3	+	+	-	22	24	23	18	10	3	-
30-39	7.8e-3	+	+	+	-	31	30	23	12	4	-
40-49	6.6e-3	+	+	+	+	-	43	33	18	5	1
50-59	5.0e-3	+	+	+	+	+	-	57	32	10	1
60-69	3.3e-3	+	+	+	+	+	+	-	74	23	3
70-79	1.6e-3	+	+	+	+	+	+	+	-	90	10
80-89	4.4e-4	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

APPENDIX B - Part II

Cancer Morbidity Models for Those Exposed to the Plume

Table B-II.1

Breast* Cancer Morbidity - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.7e-2	-	13	16	18	18	16	11	6	2	-
10-19	1.5e-2	+	-	18	20	21	18	13	7	3	-
20-29	1.3e-2	+	+	-	25	25	22	16	9	3	-
30-39	9.5e-3	+	+	+	-	33	29	21	12	4	1
40-49	6.3e-3	+	+	+	+	-	44	32	18	5	1
50-59	3.5e-3	+	+	+	+	+	-	57	32	10	1
60-69	1.5e-3	+	+	+	+	+	+	-	73	24	3
70-79	4.1e-4	+	+	+	+	+	+	+	-	90	10
80-89	4.2e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risk estimates apply to the entire population. Risks for women would be twice this large.

Table B-II.2

Lung Cancer Morbidity - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.2e-3	-	13	14	17	19	18	12	6	1	-
10-19	2.0e-3	+	-	16	19	22	20	14	7	2	-
20-29	1.6e-3	+	+	-	23	26	24	17	8	2	-
30-39	1.3e-3	+	+	+	-	33	31	22	11	3	-
40-49	8.4e-4	+	+	+	+	-	47	33	16	4	-
50-59	4.4e-4	+	+	+	+	+	-	63	29	7	1
60-69	1.7e-4	+	+	+	+	+	+	-	79	19	2
70-79	3.5e-5	+	+	+	+	+	+	+	-	90	10
80-89	3.4e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.3

Gastrointestinal Cancer Morbidity - Central Estimate

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	9.7e-3	-	11	13	15	17	17	15	9	3	-
10-19	8.3e-3	+	-	15	17	19	20	16	10	3	-
20-29	7.1e-3	+	+	-	20	22	23	19	11	4	1
30-39	5.7e-3	+	+	+	-	28	28	24	14	5	1
40-49	4.1e-3	+	+	+	+	-	39	33	20	7	1
50-59	2.5e-3	+	+	+	+	+	-	54	33	12	1
60-69	1.1e-3	+	+	+	+	+	+	-	72	25	3
70-79	3.2e-4	+	+	+	+	+	+	+	-	89	11
80-89	3.5e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.4

Thyroid Cancer Morbidity - Central Estimate*

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	7.0e-3	11	20	18	16	14	10	7	3	1	-
10-19	5.5e-3	+	12	23	20	17	13	9	5	1	-
20-29	4.2e-3	+	+	15	26	22	17	12	6	2	-
30-39	3.0e-3	+	+	+	18	31	24	16	8	3	-
40-49	2.0e-3	+	+	+	+	23	37	24	12	4	-
50-59	1.2e-3	+	+	+	+	+	29	42	21	7	1
60-69	5.6e-4	+	+	+	+	+	+	39	45	14	2
70-79	1.9e-4	+	+	+	+	+	+	+	53	42	5
80-89	3.4e-5	+	+	+	+	+	+	+	+	72	28
90-99	2.0e-6	+	+	+	+	+	+	+	+	+	100

*Upper and lower estimates of lifetime risk differ only in the treatment of internal sources such as [131]I. See Section 2.2.6.

Table B-II.5

Skin Cancer Morbidity - Central Estimate*

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.0e-3	-	24	22	18	15	11	6	3	1	-
10-19	1.6e-3	+	-	28	24	19	14	9	4	2	-
20-29	1.1e-3	+	+	-	34	27	20	12	5	2	-
30-39	7.5e-4	+	+	+	-	41	29	18	9	3	-
40-49	4.4e-4	+	+	+	+	-	50	31	14	4	1
50-59	2.2e-4	+	+	+	+	+	-	61	29	9	1
60-69	8.6e-5	+	+	+	+	+	+	-	75	22	3
70-79	2.2e-5	+	+	+	+	+	+	+	-	89	11
80-89	2.3e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Multiplication of the central estimates of lifetime risk by 10/3 gives upper estimates; division by 3 gives lower estimates.

Table B-II.6

Other* Cancer Morbidity - Central Estimate

Time To Dose	Lifetime Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.6e-3	-	13	15	17	18	17	12	6	2	-
10-19	4.8e-3	+	-	17	19	20	19	14	8	3	-
20-29	4.0e-3	+	+	-	23	25	23	17	9	3	-
30-39	3.1e-3	+	+	+	-	32	30	22	12	4	-
40-49	2.1e-3	+	+	+	+	-	44	32	17	6	1
50-59	1.2e-3	+	+	+	+	+	-	58	31	10	1
60-69	4.9e-4	+	+	+	+	+	+	-	74	23	3
70-79	1.3e-4	+	+	+	+	+	+	+	-	89	11
80-89	1.4e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

Table B-II.7

Benign Thyroid Nodule Morbidity - Central Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.4e-2	-	22	20	18	15	12	8	4	1	-
10-19	1.8e-2	+	-	26	23	19	15	10	5	2	-
20-29	1.3e-2	+	+	-	31	26	20	14	7	2	-
30-39	9.3e-3	+	+	+	-	37	29	20	10	3	1
40-49	5.8e-3	+	+	+	+	-	47	31	16	5	1
50-59	3.1e-3	+	+	+	+	+	-	59	31	9	1
60-69	1.3e-3	+	+	+	+	+	+	-	74	23	2
70-79	3.3e-4	+	+	+	+	+	+	+	-	89	11
80-89	3.5e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Upper and lower estimates of lifetime risk differ only in the treatment of internal sources such as [131]I. See Section 2.2.6.

Table B-II.8

Breast* Cancer Morbidity - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.2e-3	-	16	19	20	17	13	9	5	1	-
10-19	1.0e-3	+	-	23	24	20	16	10	5	2	-
20-29	8.0e-4	+	+	-	31	27	20	13	7	2	-
30-39	5.5e-4	+	+	+	-	39	29	19	10	3	-
40-49	3.4e-4	+	+	+	+	-	48	31	16	5	-
50-59	1.8e-4	+	+	+	+	+	-	60	30	9	1
60-69	7.1e-5	+	+	+	+	+	+	-	75	23	2
70-79	1.8e-5	+	+	+	+	+	+	+	-	90	10
80-89	1.8e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risk estimates apply to the entire population. Risks for women would be twice this large.

Table B-II.9

Lung Cancer Morbidity - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.8e-4	-	16	19	20	18	13	8	4	2	-
10-19	5.2e-4	+	-	23	24	21	15	10	5	2	-
20-29	4.0e-4	+	+	-	32	28	20	12	6	2	-
30-39	2.7e-4	+	+	+	-	41	30	18	8	3	-
40-49	1.6e-4	+	+	+	+	-	50	31	14	4	1
50-59	8.2e-5	+	+	+	+	+	-	61	29	9	1
60-69	3.2e-5	+	+	+	+	+	+	-	75	23	2
70-79	7.9e-6	+	+	+	+	+	+	+	-	90	10
80-89	8.4e-7	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.10

Gastrointestinal Cancer Morbidity - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	1.6e-3	-	24	22	18	15	11	6	3	1	-
10-19	1.2e-3	+	-	28	24	19	14	9	5	1	-
20-29	8.7e-4	+	+	-	34	27	19	12	6	2	-
30-39	5.7e-4	+	+	+	-	41	29	18	9	3	-
40-49	3.4e-4	+	+	+	+	-	50	31	14	4	1
50-59	1.7e-4	+	+	+	+	+	-	61	29	9	1
60-69	6.6e-5	+	+	+	+	+	+	-	75	22	3
70-79	1.7e-5	+	+	+	+	+	+	+	-	90	10
80-89	1.8e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.11

Other* Cancer Morbidity - Lower Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	9.8e-4	-	24	22	18	15	11	6	3	1	-
10-19	7.6e-4	+	-	28	24	19	14	9	5	1	-
20-29	5.5e-4	+	+	-	34	27	19	12	6	2	-
30-39	3.2e-4	+	+	+	-	41	29	18	9	3	-
40-49	2.1e-4	+	+	+	+	-	50	31	14	4	1
50-59	1.1e-4	+	+	+	+	+	-	61	29	9	1
60-69	4.2e-5	+	+	+	+	+	+	-	75	22	3
70-79	1.1e-5	+	+	+	+	+	+	+	-	90	10
80-89	1.1e-6	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

Table B-II.12

Breast* Cancer Morbidity - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.5e-2	-	9	11	15	19	18	15	10	3	-
10-19	2.3e-2	+	-	12	17	20	20	17	10	4	-
20-29	2.0e-2	+	+	-	19	23	23	19	12	4	-
30-39	1.7e-2	+	+	+	-	28	28	23	15	5	1
40-49	1.2e-2	+	+	+	+	-	39	33	20	7	1
50-59	7.2e-3	+	+	+	+	+	-	54	34	11	1
60-69	3.3e-3	+	+	+	+	+	+	-	72	25	3
70-79	9.2e-4	+	+	+	+	+	+	+	-	90	10
80-89	9.6e-5	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*These risk estimates apply to the entire population. Risks for women would be twice this large.

Table B-II.13

Lung Cancer Morbidity - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	2.7e-2	-	8	9	13	19	23	18	8	2	-
10-19	2.0e-2	+	-	13	15	19	21	19	10	3	-
20-29	1.4e-2	+	+	-	24	27	24	16	7	2	-
30-39	1.2e-2	+	+	+	-	36	32	21	9	2	-
40-49	9.4e-3	+	+	+	+	-	49	33	14	4	-
50-59	6.3e-3	+	+	+	+	+	-	65	28	6	1
60-69	3.4e-3	+	+	+	+	+	+	-	80	18	2
70-79	1.3e-3	+	+	+	+	+	+	+	-	90	10
80-89	3.3e-4	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.14

Gastrointestinal Cancer Morbidity - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	5.5e-2	-	7	9	11	15	20	20	13	4	1
10-19	4.2e-2	+	-	12	13	16	19	19	14	6	1
20-29	2.9e-2	+	+	-	21	23	23	18	11	4	1
30-39	2.7e-2	+	+	+	-	29	29	24	13	4	1
40-49	2.3e-2	+	+	+	+	-	41	33	19	6	1
50-59	1.8e-2	+	+	+	+	+	-	57	32	10	1
60-69	1.2e-2	+	+	+	+	+	+	-	73	24	3
70-79	6.0e-3	+	+	+	+	+	+	+	-	90	10
80-89	1.7e-3	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

Table B-II.15

Other* Cancer Morbidity - Upper Estimate

Time To Dose	Life-time Risk @ 1Gy	Time Since Accident (yr)									
		0-9	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80-89	90-99
0-9	3.3e-2	-	9	10	14	18	20	17	9	3	-
10-19	2.5e-2	+	-	14	16	18	20	17	11	4	-
20-29	1.6e-2	+	+	-	25	26	23	16	8	2	-
30-39	1.4e-2	+	+	+	-	34	30	22	11	3	-
40-49	1.2e-2	+	+	+	+	-	46	32	16	5	1
50-59	8.3e-3	+	+	+	+	+	-	60	30	9	1
60-69	5.1e-3	+	+	+	+	+	+	-	75	22	3
70-79	2.4e-3	+	+	+	+	+	+	+	-	90	10
80-89	6.6e-4	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

*Includes lymphoma, multiple myeloma, and cancers of the brain, kidney, bladder, and uterus. Excludes skin and prostate cancer and all cancers for which separate risk models have been developed.

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10. SUPPLEMENTARY NOTES

11. ABSTRACT *(200 words or less)*

This report provides dose-response models intended to be used in estimating the radiological health effects of nuclear power plant accidents. Models of early and continuing effects, cancers and thyroid nodules, and genetic effects are provided.

Two-parameter Weibull hazard functions are recommended for estimating the risks of early and continuing health effects. Three potentially lethal early effects—the hematopoietic, pulmonary and gastrointestinal syndromes—are considered. In addition, models are provided for assessing the risks of several non-lethal early and continuing effects—including prodromal vomiting and diarrhea, hypothyroidism and radiation thyroiditis, skin burns, reproductive effects, and spontaneous abortions.

Linear and linear-quadratic models are recommended for estimating cancer risks. Parameters are given for analyzing the risks of seven types of cancer in adults—leukemia, bone, lung, breast, gastrointestinal, thyroid and "other". The category, "other" cancers, is intended to reflect the combined risks of multiple myeloma, lymphoma, and cancers of the bladder, kidney, brain, ovary, uterus and cervix. Models of childhood cancers due to in utero exposure are also provided. For most cancers, both incidence and mortality are addressed. The models of cancer risk are derived largely from information summarized in BEIR III—with some adjustment to reflect more recent studies. The effect of the revised dosimetry in Hiroshima and Nagasaki has not been considered.

Linear and linear-quadratic models are also recommended for assessing genetic risks. Five classes of genetic disease—dominant, x-linked, aneuploidy, unbalanced translocations and multifactorial diseases—are considered. In addition, the impact of radiation-induced genetic damage on the incidence of peri-implantation embryo losses is discussed.

The uncertainty in modeling radiological health risks is addressed by providing central, upper, and lower estimates of all model parameters. Data are provided which should enable analysts to consider the timing and severity of each type of health risk.

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