NUREG/CR–4214
Rev. 2, Part I
ITRI–141

Health Effects Models for Nuclear Power Plant Accident Consequence Analysis

Part I: Introduction, Integration, and Summary

Prepared by:
J. S. Evans, S. Abrahamson, M. A. Bender, B. B. Boecker,
E. S. Gilbert, B. R. Scott

Inhalation Toxicology Research Institute
Lovelace Biomedical and Environmental Research Institute

Prepared for
U.S. Nuclear Regulatory Commission

AVAILABILITY NOTICE

Availability of Reference Materials Cited in NRC Publications

Most documents cited in NRC publications will be available from one of the following sources:

1. The NRC Public Document Room, 2120 L Street, NW, Lower Level, Washington, DC 20555-0001

2. The Superintendent of Documents, U.S. Government Printing Office, Mail Stop SSOP, Washington, DC 20402-9328

3. The National Technical Information Service, Springfield, VA 22161

Although the listing that follows represents the majority of documents cited in NRC publications, it is not intended to be exhaustive.

Referenced documents available for inspection and copying for a fee from the NRC Public Document Room include NRC correspondence and internal NRC memoranda; NRC Office of Inspection and Enforcement bulletins, circulars, information notices, inspection and investigation notices; Licensee Event Reports; vendor reports and correspondence; Commission papers; and applicant and licensee documents and correspondence.

The following documents in the NUREG series are available for purchase from the GPO Sales Program: formal NRC staff and contractor reports, NRC-sponsored conference proceedings, and NRC booklets and brochures. Also available are Regulatory Guides, NRC regulations in the *Code of Federal Regulations*, and *Nuclear Regulatory Commission Issuances*.

Documents available from the National Technical Information Service include NUREG series reports and technical reports prepared by other federal agencies and reports prepared by the Atomic Energy Commission, forerunner agency to the Nuclear Regulatory Commission.

Documents available from public and special technical libraries include all open literature items, such as books, journal and periodical articles, and transactions. *Federal Register* notices, federal and state legislation, and congressional reports can usually be obtained from these libraries.

Documents such as theses, dissertations, foreign reports and translations, and non-NRC conference proceedings are available for purchase from the organization sponsoring the publication cited.

Single copies of NRC draft reports are available free, to the extent of supply, upon written request to the Office of Information Resources Management, Distribution Section, U.S. Nuclear Regulatory Commission, Washington, DC 20555-0001.

Copies of industry codes and standards used in a substantive manner in the NRC regulatory process are maintained at the NRC Library, 7920 Norfolk Avenue, Bethesda, Maryland, and are available there for reference use by the public. Codes and standards are usually copyrighted and may be purchased from the originating organization or, if they are American National Standards, from the American National Standards Institute, 1430 Broadway, New York, NY 10018.

NUREG/CR-4214
Rev. 2, Part I
ITRI-141
CF,C4,CO,RE,RH,9E,9H,9U

Health Effects Models for Nuclear Power Plant Accident Consequence Analysis

Part I: Introduction, Integration, and Summary

Manuscript Completed: September 1993
Date Published: October 1993

Prepared by
J. S. Evans[1], S. Abrahamson[2], M. A. Bender[3], B. B.Boecker[4],
E. S. Gilbert[5], B. R. Scott[4]

S. S. Yaniv, NRC Project Manager

Inhalation Toxicology Research Institute
Lovelace Biomedical and Environmental Research Institute
P. O. Box 5890
Albuquerque, NM 87185

Prepared for
Division of Regulatory Applications
Office of Nuclear Regulatory Research
U.S. Nuclear Regulatory Commission
Washington, DC 20555-0001
NRC FIN L1264

---

[1]Harvard School of Public Health, Boston, MA 02115
[2]University of Wisconsin, Madison, WI 53706
[3]Brookhaven National Laboratory, Upton, NY 11973
[4]Inhalation Toxicology Research Institute, Albuquerque, NM 87185
[5]Battelle Pacific Northwest Laboratories, Richland, WA 99352

UNITED STATES
NUCLEAR REGULATORY COMMISSION
WASHINGTON, D.C. 20555-0001

September 30, 1993

FOREWORD

The Nuclear Regulatory Commission (NRC) has sponsored several studies to identify and quantify, through the use of models, the potential health effects of accidental releases of radionuclides from nuclear power plants. The Reactor Safety Study (WASH-1400, Appendix VI) provided the basis for most of the earlier estimates related to these health effects. Subsequent efforts by NRC-supported groups resulted in improved health effects models that were published in report entitled "Health Effects Models for Nuclear Power Plant Consequence Analysis", NUREG/CR-4214, 1985 and revised further in the 1989. Part II of NUREG/CR-4214, Rev. 1, was published in May 1989 and Part I of NUREG/CR-4214, Rev. 1 " Introduction, Integration, and Summary" was published in January 1990. The health effects models presented in 1989 NUREG/CR-4214 report were developed for exposure to low-linear energy transfer (LET) (beta and gamma) radiation based on the best scientific information available at that time. Since the 1989 report was published, two addenda to that report have been prepared to 1) incorporate other scientific information related to low-LET health effects models and 2) extend the models to consider the possible health consequences of the addition of alpha-emitting radionuclides to the exposure source term.

The first addendum report, entitled "Health Effects Models for Nuclear Power Plant Accident Consequence Analysis, Modifications of Models Resulting from Recent Reports on Health Effects of Ionizing Radiation, Low LET Radiation, Part II: Scientific Bases for Health Effects Models," was published in 1991 as NUREG/CR-4214, Rev. 1, Part II, Addendum 1.

The second addendum report, entitled "Health Effects Models for Nuclear Power Plant Accident Consequence Analysis, Modification of Models Resulting from Addition of Effects of Exposure to Alpha-Emitting Radionuclides," was published in 1993 as NUREG/CR-4214, Rev. 1, Part II, Addendum 2.

This report, which is revision of NUREG/CR-4214, Rev. 1, Part I is directed specifically to incorporating the new information presented in these two addenda as they may impact on the models and the recommended parameters associated with the health effects models. Those portions of the earlier

Part I report that were not impacted by the two addenda are included without revision in this version of Part I for completeness.

NUREG/CR-4214, Rev. 2 Part 1 is not a substitute for NRC regulations, and compliance is not required. The approaches and/or methods described in this NUREG are provided for information only. Publication of this report does not necessarily constitute NRC approval or agreement with the information contained herein.

Donald A. Cool, Chief
Radiation Protection and Health
Effects Branch
Division of Regulatory Applications
Office of Nuclear Regulatory Research

ABSTRACT

This report is a revision of NUREG/CR-4214, Rev. 1, Part I (1990), *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis*. This revision has been made to incorporate changes to the Health Effects Models recommended in two addenda to the NUREG/CR-4214, Rev. 1, Part II, 1989 report. The first of these addenda provided recommended changes to the health effects models for low-LET radiations based on recent reports from UNSCEAR, ICRP and NAS/NRC (BEIR V). The second addendum presented changes needed to incorporate alpha-emitting radionuclides into the accident exposure source term. Particular attention was directed to the inhalation route of exposure and alpha irradiation of the lung, liver, bone, and bone marrow. As in the earlier version of this report, models are provided for early and continuing effects, cancers and thyroid nodules, and genetic effects.

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, IV and V as well as other current reports.

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.

CONTENTS

CONTENTS

FIGURES

TABLES

TABLES

PREFACE

In the early 1980's, the U.S. Nuclear Regulatory Commission recognized the need to review and revise the health effects models that had been used in the Reactor Safety Study. At that time, a group of us at Harvard were asked to identify experts who could contribute to the revision of health effects models and to coordinate the development of a complete suite of revised health effects models. Two issues were of particular interest to the NRC. First, that an open and scrutable process be used to develop the new models—i.e., identification of experts, selection of members of our advisory committee, model formulation and review. Second, that the uncertainty in the health effects models was to be quantitatively characterized.

Considerable efforts were made in the initial work, 1981-85, to ensure that these goals were achieved. Experts in radiation health were identified on the basis of a systematic review of the published literature using publication counts and peer-group nominations as indices of expertise. Twenty individuals so-identified agreed to serve as members of our advisory committee. This advisory committee played an active role in model development and review during the early phases of the work. Uncertainty was addressed in this initial work by providing central, upper and lower estimates of radiation health risks for each effect of interest.

Since the middle 1980's the original report has been revised several times to reflect advances in knowledge about the effects of radiation. The current revision incorporates new information about the health effects of alpha particles and modifications of cancer risk assessment models necessitated by the ongoing followup of the survivors of the atomic bombs at Hiroshima and Nagasaki.

Although the models have been repeatedly revised, they have not been subjected to the degree of peer review that characterized the initial model development. In particular, the approach taken for characterizing uncertainty is a bit outdated and deserves reconsideration. In the areas of air pollution risk assessment, chemical carcinogenesis, and engineering risk assessment, there have been great advances in formal approaches for incorporating expert scientific judgment in risk analysis.

Should it become necessary for these models to be further revised, it would be desirable to incorporate these advances throughout the model development process, using recent approaches for characterizing the degree of uncertainty and disagreement among experts about the health risks posed by ionizing radiation.

John S. Evans, Sc.D.
Harvard School of Public Health

ACKNOWLEDGEMENTS
TO NUREG/CR-4214 Rev. 1, Part I (1990a)

This report reflects the efforts of many individuals. Primary among these are the Working Group Leaders: Dr. Bobby Scott, Inhalation Toxicology Research Institute; Dr. Ethel Gilbert, Battelle Pacific Northwest Laboratories; Dr. Seymour Abrahamson, University of Wisconsin; and Dr. Harry Maxon, University of Cincinnati, who were responsible for reviewing the literature, making recommendations for dose-response models, and preparing reports summarizing their findings. Drs. Douglas Cooper and Dade Moeller of Harvard University, who initiated the project, coordinated the selection of the Working and Advisory Groups, and actively participated in the early phases of model development, also deserve special mention.

The project has benefited immensely from the efforts of the members of our Advisory Committee:

Seymour Abrahamson, Ph.D.	University of Wisconsin
William J. Bair, Ph.D.	Battelle Pacific Northwest Laboratories
Michael A Bender, Ph.D.	Brookhaven National Laboratory
Bruce B. Boecker, Ph.D.	Inhalation Toxicology Research Institute
Victor P. Bond, M.D., Ph.D.	Brookhaven National Laboratory
Richard G. Cuddihy, Ph.D.	Inhalation Toxicology Research Institute
Keith F. Eckerman, Ph.D.	Oak Ridge National Laboratory
Jacob I. Fabrikant, M.D., Ph.D.	University of California
Marvin Goldman, Ph.D.	University of California
George B. Hutchison, M.D., M.P.H.	Harvard School of Public Health
Dade W. Moeller, Ph.D.	Harvard School of Public Health
Edward P. Radford, M.D.	Radiation Effects Research Foundation
Eugene L. Saenger, M.D.	University of Cincinnati College of Medicine
Warren K. Sinclair, Ph.D.	National Council on Radiation Protection and Measurements
Niel Wald, M.D.	University of Pittsburgh
Edward W. Webster, Ph.D.	Massachusetts General Hospital
Shlomo S. Yaniv, Sc.D.	U.S. Nuclear Regulatory Commission

The members of the Advisory Committee critically reviewed both the original report and this version of that report. Every effort has been made to respond to their comments. Nevertheless, membership on the Advisory Committee should not be taken to imply endorsement of the health effects models.

Many other scientists have made significant contributions to the development of the models presented in this report. Drs. Niel Wald, Albert Spritzer, and Joseph Watson of the University of Pittsburgh reviewed data on human radiation injury that was used in the development of the early health effect models. Drs. Roy Shore and Nan Laird provided technical information that supported development of the skin and thyroid cancer models. Dr. Syed Mansur performed many of the calculations needed to develop population-based cancer and genetic risk models. Chapter reviews were provided by the following experts: Drs. Gilbert Beebe and Charles Land, National Cancer Institute; Dr. Thomas Cochran, National Resources Defense Council; Dr. James Crow, University of Wisconsin; Drs. Troyce Jones and Clarence Lushbaugh, Oak Ridge Associated Universities; and Dr. Robert Young, Defense Nuclear Agency.

ADDITIONAL ACKNOWLEDGEMENTS FOR NUREG/CR-4214, Rev. 2, Part I (1993)

The present report is a revision of the NUREG/CR-4214, Rev. 1, Part I report (NRC, 1990a). This revision was made to incorporate changes to the Health Effects Models recommended in two addenda to the NUREG/CR-4214, Rev. 1, Part II report (NRC, 1989). The authors of these addenda and this revision were the principal authors of the earlier reports: Drs. Seymour Abrahamson, Michael Bender, Ethel Gilbert and Bobby Scott, and an additional author, Dr. Bruce Boecker, who also coordinated this effort. These authors provided an important continuity of concepts, approaches and knowledge. Ms. Margie Mueller performed many of the life-table analyses needed for the tables in Appendix B. Ms. Mary G. Campos was once again a key ingredient in the drafting and production of this report through her tireless efforts and careful attention to details. Both of the two addenda reflected in this report were reviewed critically by as many of the members of the Advisory Committee listed above as possible to provide additional continuity to this effort. The input from all these participants is gratefully acknowledged.

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. 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 (Ritchie, 1983).

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 Cooper at Harvard University, concluded that several components of the Reactor Safety Study health effects models required revision.

In 1982, the NRC initiated an effort to prepare improved health effects models to replace those used in the Reactor Safety Study. The focus of this initial effort was to review the models for low-LET radiations. An Advisory Committee, consisting of 17 experts, was assembled. Nominations for appointment to the Advisory Committee were solicited from over 300 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 reviewing the literature, recommending health-effects models, and preparing reports giving the scientific basis for each model recommended. The entire project was managed by scientists at Harvard University, initially by Dr. Douglas W. Cooper and later by Dr. John S. Evans.

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

NUREG/CR-4214 (NRC, 1985), which dealt with low-LET radiation, was published in July 1985. The NRC circulated the document widely; more than 1000 copies of the report were distributed for public review and comment. The new models were formally presented in Washington, DC, in October 1985, and in Luxembourg in April 1985.

Since their publication in 1985, the NUREG/CR-4214 health-effects models have been revised twice. A primary goal of the first revision was to ensure that the models for early effects of low-LET radiation were consistent with the data on humans who had been accidentally or therapeutically exposed to radiation. Scientists at the University of Pittsburgh, led by Dr. Niel Wald, were retained to review the

available human data; to assist in the interpretation of these revisions; 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 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 parameters for pulmonary syndrome mortality be critically reviewed.

In addition to achieving these two goals, the NRC sought to update the models for late somatic effects to reflect data from 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 peri-implantation embryo losses (spontaneous abortions) induced by radiation. The authors of the late somatic effects and genetic effects chapters of the original report, Drs. Ethel Gilbert and Seymour Abrahamson, were asked to review their chapters in response to these concerns. Reports reflecting these first revisions were published in 1989 and 1990: *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis, Low-LET Radiation. Part I: Introduction, Integration and Summary* (NRC, 1990a) and *Part II: Scientific Bases for Health Effects Models* (NRC, 1989).

The second revision, which began in 1989, had two basic goals: (i) to compare the NUREG models for cancers and genetic effects with models presented in UNSCEAR (1988), BEIR V (NAS/NRC, 1990), and ICRP Publication 60 (ICRP, 1991) and to make modifications where necessary, and (ii) to recommend approaches to estimate risks from exposure to high-LET, alpha-emitting radionuclides. This project was managed by a group at the Inhalation Toxicology Research Institute led by Dr. Bruce Boecker. These revisions were made by several authors of the original report, including Dr. Scott - ITRI, Dr. Gilbert - PNL, Dr. Abrahamson - University of Wisconsin, and Dr. Mike Bender - Brookhaven National Laboratory. As a result of these efforts, two addenda to NUREG/CR-4214, Rev. 1, Part II (NRC, 1989) have been published. Addendum 1 is entitled *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis, Modifications of Models Resulting from Recent Reports on Health Effects of Ionizing Radiation, Low-LET Radiation, Part II: Scientific Bases for Health Effects Models* (NRC, 1991) and Addendum 2 is entitled *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis, Modifications of Models Resulting from Addition of Effects of Exposure to Alpha-Emitting Radionuclides, Part II: Scientific Bases for Health Effects Models* (NRC, 1993).

Addendum 1 (NRC, 1991) presented reviews of new reports that could impact the health effects models for low-LET radiations given in the NUREG/CR-4214 report (NRC, 1989), especially the reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1988), the National Academy of Sciences/National Research Council BEIR V Committee (NAS/NRC, 1990), and revised recommendations of the International Commission on Radiological Protection Publication 60 (ICRP, 1991). Most of the recommended changes to the NUREG/CR-4214 health effects models were related to the late somatic effects. The most important of these changes fell in three areas. First, the recommended dose and dose rate reduction factors (DDREF) for calculating central and lower bound estimates for low dose and low dose rate exposure to low-LET radiations were modified. The previous factor of 3.3 for the central estimate was changed to 2 and the previous factor of 10 for the lower bound

was changed to 4. Second, it was recommended that central estimates for most cancer types be based on age-specific coefficients rather than the non-age-specific treatment used earlier. Finally, many of the risk coefficients were modified to account for recent data and analyses, particularly analyses of the Japanese A-bomb survivors based on revised dosimetry. For early occurring and continuing effects, the model presented in NUREG/CR-4214 for severe mental retardation associated with *in utero* exposure was modified to allow for uncertainty associated with threshold dose. For genetic effects, the treatment of irregularly inherited diseases was changed to include the new natural incidence estimates of irregularly inherited diseases and their corresponding estimates of radiation-induced risks.

All of the NUREG/CR-4214 health effects models presented up through the Addendum 1 report were directed to brief or protracted exposures to low-LET radiations. Because nuclear power plants also have alpha-emitting radionuclides in their fuel inventories, it was necessary to also incorporate the health risks from possible exposures to the high-LET radiations from these radionuclides. Chronic internal radiation from alpha particles is more effective in producing biological effects than is low-LET radiation. The Addendum 2 report (NRC, 1993) presented the changes needed to incorporate alpha-emitting radionuclides into the accident exposure source term. Particular attention was directed to the inhalation route of exposure and irradiation of the lung, liver, bone and bone marrow. Possible genetic effects were also discussed.

This report, which is a revision of NUREG/CR-4214, Rev. 1, Part I (NRC, 1990a), is directed specifically to incorporating the new information presented in these two addenda (NRC 1991, 1993) as they may impact on the models and the recommended parameters associated with these models. Those portions of the earlier NUREG/CR-4214 report that were not impacted by the two addenda are included without revision in this version of Part I for completeness. This report 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 analyzing the consequences of nuclear power plant accidents. 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 the PRA Procedures Guide (NRC, 1983) and several volumes of a more recent report (NRC 1990b) for discussions of these matters.

The purpose of this report is simply to document the dose-response models recommended for estimating the health effects of nuclear power plant accidents. 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.

1.1 Treatment of Uncertainty

The health risks caused by radiation cannot be predicted precisely. The initial statement of work leading to this report 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 uncertainty and model uncertainty. Parameter uncertainty arises in the process of drawing inferences about processes that 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 estimate 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 Beagle dogs and estimates of genetic risks are based on studies in mice. The accuracy of such estimates depends on the adequacy of the analogies. Similarly, most estimates of radiation-induced cancer risk for low-LET radiation 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. Furthermore, there is uncertainty about how to transport cancer risks from the Japanese to the U.S. population. 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 collective 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 25 effects that may be induced by ionizing radiation. The report also includes some information about the severity of each effect. For most early effects, the nature and duration of symptoms are briefly described.

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 are provided that illustrate the temporal aspects of these risks.

1.3 Organization of Part I

The remainder of this volume is organized in two chapters and two appendices. 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, and the revisions recommended in Addendum 1 (NRC, 1991) and Addendum 2 (NRC, 1993). 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, Computational Aspects, has several purposes, primarily 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 analyses computer codes. In addition, this chapter briefly considers other topics of computational interest, e.g., the risks of early effects, based on different models (Weibull, probit and logistic) are compared.

Appendix A includes baseline demographic and mortality data used in the calculations. Appendix B presents a set of tables useful in estimating risks for a population exposed to the plume.

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, 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 geographic cell surrounding a nuclear power plant. This set of organ-specific absorbed doses for both low-LET and alpha radiations is one input required by our model. Information on dose rate is also required.

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, the organ doses that are required as inputs to evaluate these models, and the types of radiation, i.e., α, β, γ, for which models have been developed.

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 pregnant women 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 is derived largely from four sources: (i) studies of radiation-related side effects among humans exposed therapeutically, (ii) analyses of the experience of the survivors of the atomic bombings of Hiroshima and Nagasaki, (iii) examination of the health effects observed among the relatively small number of individuals who received large radiation doses in various accidents, and (iv) investigations of the effects observed in animals experimentally exposed to radiation.

Models for early and continuing effects of low-LET radiation were developed by Dr. Scott and Dr. Hahn of the Inhalation Toxicology Research Institute. The models are based in part on data concerning human radiation injury reviewed by Dr. Wald, Dr. Joseph Watson and Dr. Albert Spritzer of the University of Pittsburgh. Information on thyroid effects was provided by Dr. Harry Maxon and several of his colleagues. The models for early and continuing effects due to irradiation of the lungs and bone marrow were subsequently modified by Dr. Scott to reflect the impact of dose from high-LET alpha particles.

Table 2.1

Early effects included in these health effects models

Effect	Model developed		Types of radiation	Target organ
	Mortality	Morbidity		
Hematopoietic syndrome	✓	-	α, β, γ	Bone marrow
Pulmonary syndrome	✓	✓	α, β, γ	Lung
Gastrointestinal syndrome	✓	-	β, γ	Small intestine[a] - colon
Prodromal symptoms				
Vomiting	-	✓	β, γ	Abdomen[b]
Diarrhea	-	✓	β, γ	Abdomen[b]
Pneumonitis	-	✓	β, γ	Lung
Thyroid effects				
Thyroiditis	-	✓	β, γ	Thyroid
Hypothyroidism	-	✓	β, γ	Thyroid
Skin effects				
Erythema	-	✓	β, γ	Epidermis[c]
Transepidermal injury	-	✓	β, γ	Epidermis[c]
Cataracts	-	✓	β, γ	Lens of eye
Embryo/Fetus				
Microencephaly	-	✓	β, γ	Embryo/Fetus
Severe mental retardation	-	✓	β, γ	Embryo/Fetus
Death of embryo/fetus	✓	-	β, γ	Embryo/Fetus

[a] The dose to the 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 these health effects models

Effect	Model developed		Types of radiation	Target organ
	Mortality	Morbidity		
Somatic effects				
Leukemia	✓	-	β, γ	Red bone marrow
in utero	✓	-	β, γ	Fetus
Bone cancer	✓	-	α, β, γ	Bone
Breast cancer	✓	✓	$\beta. \gamma$	Breast
Lung cancer	✓	✓	α, β, γ	Lung
Gastrointestinal cancer[a]	✓	✓	α, β, γ	Lower large intestine[b]
Thyroid cancer	✓	✓	β, γ	Thyroid
Skin cancer	-	✓	β, γ	Epidermis[c]
Other cancer	✓	✓	β, γ	Other[d]
in utero	✓	-	β, γ	Fetus
Benign thyroid nodules	-	✓	β, γ	Thyroid
Genetic effects				
Single gene				
Dominant	-	✓	α, β, γ	Gonads
X-linked	-	✓	α, β, γ	Gonads
Chromosome aberrations				
Numerical	-	✓	α, β, γ	Gonads
Structural	-	✓	α, β, γ	Gonads
Multifactorial	-	✓	α, β, γ	Gonads
Pregnancy loss[e]	-	✓	α, β, γ	Gonads

[a] And liver cancer risks from high-LET radiation.

[b] A weighted combination of the doses to the esophagus, stomach, colon and liver is recommended for use in evaluation of risks from low-LET radiation. However, to evaluate liver cancer risks from high-LET radiation only the dose to the liver should be used.

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

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

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

Scientific understanding of the biological nature of early effects indicates that most 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 deterministic (non-stochastic) effects are simply reflections of the distributions of individual thresholds, or tolerances, among the population.

The risks of early and continuing effects of low-LET irradiation 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 in the absence of competing risks, exhibit the effect of interest, and H, the cumulative hazard, is a function of both the dose received by the person and the dose rate. For exposure at a fixed dose rate, 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:

$$H = 0.693\,[D/D_{50}]^{V} \text{ for } D > T$$

where H is the cumulative hazard, D is the (mean 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 D_{50} depends on dose rate.

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 particularly 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 % of the median lethal doses rather than at the values selected by the early effects working group.

The choice of this particular form of dose-response 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 in Section 3.1.6, Form of Dose-Response Model.

For most early effects of low-LET radiation, 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 dose used in the hazard function. The simplest adjustment is one in 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 early effects of low-LET radiation when there is insufficient information for developing a fully dose-rate-dependent model, 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)[a]. The term involving brief dose is necessary only when the external gamma dose rate exceeds 0.06 Gy/hour. 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.

In the evaluation of protracted dose, a fixed RBE may be used to account for the increased effectiveness of the contributions of alpha-emitting radionuclides, i.e.,

$$D_p = D_{p,\beta,\gamma} + RBE * D_{p,\alpha}$$

where D_p represents the adjusted total dose from alpha, beta and gamma-emitting radionuclides, $D_{p,\beta,\gamma}$ is the protracted dose from beta- and gamma-emitting radionuclides, $D_{p,\alpha}$ is the protracted dose from alpha-emitting radionuclides, and RBE is the relative biological effectiveness for alpha radiation for inducing the early effects of interest. The RBEs are endpoint-specific.

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 adjusted dose rate (low-LET dose rate plus RBE times alpha dose rate) at time t, $D_{50}(\dot{D})$ is the median lethal dose applicable to dose received at the adjusted 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 the adjusted 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) for low-LET radiation, $\dot{D}$ is the

---

[a] A problem arises in applying this approach when the shape parameters (V) appropriate for brief and protracted exposures are substantially different. An example is pulmonary syndrome mortality, where the shape parameter for brief exposure to gamma radiation is 12 (central estimate), and the shape parameter for protracted exposure to alpha or beta irradiation is 5 (central estimate). A solution recommended by Scott *et al.* (NRC, 1993) is to replace the brief dose term, $D_b/D_{50,b}$, in the equation with $(D_b/D_{50,b})^{(V_b/V_p)}$, where V_b is the shape parameter appropriate for brief dose, and V_p is the parameter appropriate for protracted dose.

instantaneous adjusted dose rate (Gy/hr), and Θ_1 is a parameter reflecting the sensitivity of the median lethal adjusted dose to the adjusted dose rate (Gy^2/hr). As noted above, the RBEs used in deriving the adjusted dose rates are end-point specific.

To account for any differences in the shape parameters for various radiations, it is also necessary to replace V with V_{mix}, the shape parameter appropriate for simultaneous exposure to alpha, beta and gamma irradiation. V_{mix} is evaluated using:

$$\frac{1}{V_{mix}} = \frac{g_\alpha}{V_\alpha} + \frac{g_\beta}{V_\beta} + \frac{g_\gamma}{V_\gamma}$$

where g_α, g_β, and g_γ represent the fractions of the total normalized dose due to alpha, beta, and gamma irradiation, respectively, and V_α, V_β, and V_γ are the shape parameters appropriate for these irradiations. The fraction g_α is computed as:

$$g_\alpha = \frac{\int_0^t \frac{RBE_\alpha * \dot{D}_\alpha}{D_{50}(\dot{D})}\, dt}{\int_0^t \frac{\dot{D}}{D_{50}(\dot{D})}\, dt}$$

where all terms retain their previous definitions. Similar equations are used to compute g_β and g_γ, with $RBE_\beta = RBE_\gamma = 1$.

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 causes of early 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 to external gamma rays. 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 white blood cells or platelets 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 LD_{50} 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% 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 medical 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 13 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 facilities and personnel available for treatment of radiation accident victims. The limited data available are not convincing (Anderson, 1982). Until such data become available, we recommend that no allowance be made for the lives that might be saved by intensive treatment efforts such as bone marrow transplantation.

Those who survive the effects of the brief initial exposure to cloudshine 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 introductory section on early effects.

Some individuals may accumulate rather large protracted doses. Fortunately, protracted doses received at low dose rate are not as effective in producing early effects 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 mice, rats, dogs, swine, goats, and sheep. In mice and rats the LD_{50} for low-LET radiation increases by a factor of between 1.5 and 2 as the dose rate is reduced from 10^{0} to 10^{-1} Gy per hour. In larger mammals, dogs, swine, 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 of low-LET radiation received at low dose rates also suggests that these doses may 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 irradiation 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 anyone 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.

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 of low-LET radiation. After reviewing these models, the Working Group recommended that the LD_{50} (Gy) for hematopoietic syndrome mortality be evaluated using the equations given in Table 2.3.

Scott *et al.* (1988) noted that high-LET radiation from α-emitters is not expected to contribute significantly to the risk of hematopoietic syndrome mortality because these contributions to marrow dose are expected to be small in most nuclear power plant accident scenarios. Nonetheless, central, lower, and upper estimates of RBE_{α} (for bone marrow syndrome mortality) of 2, 1, and 3, respectively, were recommended (NRC, 1993). In addition, Scott *et al.* recommended that modified values of the shape parameter be used to account for additional uncertainty about the combined effects of low-LET and alpha radiation. For circumstances involving relatively large exposures to alpha radiations, the lower and upper estimates of the shape parameter given by Scott *et al.* were 3 and 9, respectively.

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 (Ricthie) and MACCS (NRC, 1990b) for estimating the risks of hematopoietic syndrome mortality.

2.1.1.2 Pulmonary Syndrome

The lungs may be irradiated both from external sources, 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.

Table 2.3

Equations for computing the LD_{50} for mortality from the hematopoietic syndrome as a function of dose rate to the bone marrow

	Medical treatment[a]	
Estimate	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/$\dot{D}$

[a] $\dot{D}$ is the adjusted instantaneous dose rate to the bone marrow (low-LET dose rate plus RBE times the alpha dose rate) (Gy/hr).

Because large doses are 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 successful, pulmonary effects may become a concern. More generally, however, these effects will be expected to occur primarily as a result of inhaling 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) 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 did not report the typical dose rates involved, but Van Dyk *et al.* noted that all 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 to low-LET radiation, 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 *et al.*'s 1978 study suggested a threshold of about 6 Gy. Van Dyk *et al.*'s reanalysis of Fryer's data indicated that if patients with pre-existing lung disease, e.g., chronic bronchitis, emphysema, were excluded from consideration, the clinical threshold was more nearly 7.5 Gy. Keane *et al.* (1981) reported 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 to low-LET radiation.

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

Doses of low-LET radiation delivered at low dose rate are much less effective for inducing radiation pneumonitis than doses delivered at high dose rate. The clinical studies that provided the basis for the Working Group's estimate of a 10-Gy LD_{50} for low-LET radiations 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.

Studies of Beagle dogs exposed to various beta-emitting radionuclides at the Inhalation Toxicology Research Institute (ITRI) have provided striking evidence of the importance of dose rate (McClellan *et al.* 1982). The LD_{50}s observed in these experiments ranged from 94 Gy for dogs exposed to ^{90}Y (effective half-life 2.6 days) to 540 Gy for dogs exposed to ^{144}Ce (effective half life 200 days). Although these studies did not include a component in which dogs were exposed to brief external irradiation, an LD_{50} in such a scenario would be expected to be similar to those seen in other mammals studied, i.e., between 10 and 20 Gy (Scott *et al.*, 1989). These studies by McClellan *et al.* suggest that protracted internal exposures are between $1/10^{th}$ and $1/50^{th}$ as effective as brief external exposures in producing early occurring effects.

Using chronic radiation data from dogs and rats and data from brief exposure of humans, Scott, Filipy, and Hahn estimated the parameters of a model relating the median lethal dose for pulmonary syndrome to the dose rate (Scott *et al.*, 1989). The Early Effects Working Group endorses this approach and recommends that the risk be evaluated using:

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

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

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

where LD_{50} is the median lethal dose (Gy), and $\dot{D}$ is the adjusted instantaneous dose rate (Gy/hr) to the lung, which reflects the contributions of both low-LET and high-LET radiation.

The contribution of alpha-emitting radionuclides to the adjusted dose rate may be evaluated using an RBE_{α} of 7 (central estimate), with lower and upper estimates of 5 and 10, respectively. These values of RBE_{α} are based on a review of animal studies reported in ICRP Publication 58 (ICRP, 1991). In these studies, which involved both chronic alpha and chronic neutron irradiation, the RBE ranged from 5 to 10 with beta or gamma radiation as the reference. The reference for neutrons was gamma rays, and the reference for alpha radiation was beta radiation. The RBEs for chronic high-LET irradiation did not appear to vary with dose.

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. The lower and upper estimates of the shape parameter for brief external exposure are 9 and 14, and for protracted internal exposure they are 3 and 6. When mixed exposures are anticipated, the Working Group recommends that a shape parameter of 7 be used, with lower and upper estimates of 3 and 12, respectively.

As noted previously, current consequence codes for nuclear power plant accidents 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 mortality from the pulmonary syndrome.

The effects of age at exposure and pre-existing lung disease are less well understood. In studies of Beagles dogs exposed to ^{144}Ce, a strong effect of age at exposure has been demonstrated. Old dogs were found to be 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 (Rubin and Casarett, 1968) tended to discount the importance of age at exposure. However, recent studies of patients treated with whole-body radiation indicate that the incidence of interstitial pneumonitis increases with age and is about twice as large in middle-aged patients (40-60 years) as in younger patients (1 to 20 years) (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) note that mammals tend to respond similarly following gastrointestinal irradiation and suggest that data from animal studies may reasonably indicate the risks in humans. Sullivan *et al.* (1959) found that a brief external X-ray dose of about 15 Gy was required to kill about half of the rats exposed in their experiments. Data on rats exposed to high levels of beta-emitting radionuclides (Cross et al., 1978) have been interpreted as suggestive of an LD_{50} of about 35 Gy for humans following protracted 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 to low-LET irradiation 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 to low-LET irradiation are 25 and 50 Gy, respectively. The critical organ for assessing the effects of protracted exposure is the colon.

The Early Effects Working Group concluded that the gastrointestinal syndrome was unlikely to be induced by exposure to alpha particles, because the range of alpha particles is not sufficient to irradiate critical stem cells (NRC, 1993). Therefore, no modifications of parameters or models are needed to account for high-LET irradiation.

2.1.1.4 Summary - Early Mortality

To assess the overall risk of early mortality from dose to the bone marrow, lungs, and gastrointestinal tract, one simply 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 to low-LET radiation at high dose rates are summarized in Table 2.4. The effects of protracted exposures (to either low-LET or alpha radiation or combinations of the two) should be evaluated using the dose-rate-dependent models described in Section 2.1 with the parameter values for hematopoietic syndrome and pulmonary syndrome given in Sections 2.1.1.1 and 2.1.1.2, respectively. 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 non-lethal 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 2.4

Models of early mortality from brief exposure to low-LET radiation[a,b]

Effect	Risk estimate								
	Central			Lower[c]			Upper[c]		
	LD_{50}	T	V	LD_{50}	T	V	LD_{50}	T[d]	V
Hematopoietic syndrome[e]									
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 syndrome[e,f]	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. 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 doses 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] If the exposure involves both low-LET and alpha radiation, an adjusted dose equal to the low-LET dose plus RBE times the alpha dose, should be used in these calculations for the pulmonary and hematopoietic syndromes. Effect-specific RBEs are given in the body of the text.

[f] 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. For these endpoints, it was assumed that only low-LET radiation was important. The Early Effects Working Group's central estimates of the median adjusted low-LET radiation doses of 2 Gy for vomiting and 3 Gy for diarrhea are based largely on retrospective analyses of the experiences of 2000 patients treated therapeutically with whole-body radiation (Lushbaugh and Ricks, 1972).

2.1.2.2 Pulmonary Morbidity

Irradiation of the lungs may lead to pneumonitis and other forms of injury, which may result in reduced lung volume, increased stiffness of the parenchymal region, non-uniform gas distribution, and reduced alveolar-capillary gas exchange efficiency.

Our understanding of the radiation pneumonitis in humans comes primarily from studies of patients treated with low-LET radiation either as an element of cancer therapy or in conjunction with bone marrow transplants. In these settings, those who develop radiation pneumonitis nearly always die as a result of the pneumonitis.

Much of our knowledge about less severe forms of pulmonary injury comes from studies of animals experimentally exposed to radiation. In rats whose lungs were exposed to alpha- or low-energy beta-emitting radionuclides, impairment of lung function was seen at doses ¼ as large as those required to induce lethal pneumonitis. However, when rats were exposed to high energy beta-emitting radionuclides, there was little difference between the doses that produced impairment of lung function and those that led to pneumonitis.

Although the data base for developing estimates of (non-fatal) pulmonary morbidity is weak, Scott *et al.* (1989) suggested reducing the low-LET LD_{50} for pulmonary mortality by a factor of 2 to estimate pulmonary morbidity. This would lead to a central estimate of the ED_{50} for pulmonary morbidity following brief external exposure of 5 Gy, with lower and upper estimates of 4 Gy and 6 Gy, respectively. Scott *et al.* also recommended using the shape parameters developed for pulmonary mortality to estimate pulmonary morbidity risks.

Because the lung may receive rather large doses from chronic alpha emitters, procedures are necessary to estimate the risk of pulmonary morbidity in these circumstances. To account for alpha radiation dose to the lung, the adjusted dose must be computed. In such calculations, an RBE_{α} of 7 should be used to calculate central estimates of risk. For lower estimates of risk, an RBE_{α} of 5 is recommended, and for upper estimates an RBE_{α} of 10 is appropriate.

To estimate risk from prolonged exposure at low dose rates, a dose-rate-dependent ED_{50} is recommended. Scott *et al.* suggest that risks be estimated using:

$$ED_{50,\ central} = 5 + 15/\dot{D}$$

$$ED_{50,\ upper} = 6 + 22.5/\dot{D}$$

$$ED_{50,\ lower} = 4 + 7.5/\dot{D}$$

where ED_{50} is the median effective dose (Gy), and $\dot{D}$ is the adjusted instantaneous dose rate (Gy/hr) to the lung, which reflects the contributions of both low-LET and alpha radiation.

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 most 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 noted that hypothyroidism is almost certainly a threshold effect and recommended that a 10 Gy threshold be used in projections of risks of hypothyroidism following ^{131}I exposure.

Animal studies suggest that brief exposure to external low-LET radiation is about five 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) noted that none of the clinical studies involved ^{131}I doses less than 10 Gy; that 8 to 12% prevalence of hypothyroidism was 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 concluded that there is no experimental basis for the existence

of a 10 Gy threshold.[a] Therefore, we recommend that upper estimates of hypothyroidism risks be computed using thresholds well below the Working Group's recommended values of 2 Gy for external radiation and 10 Gy for ^{131}I. Further, all estimates of hypothyroidism risk generated using our models should be regarded as indicative of the early onset of hypothyroidism.

Radiation thyroiditis is an acute condition occurring within 2 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 Beirwalters and Johnson (1956) in about 5% 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% 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 a nuclear power plant 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 low-LET 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 doses large enough to produce skin burns would be almost certain to die from the hematopoietic syndrome.

---

[a] Watson also pointed out that in all ^{131}I treatment groups, the prevalence of hypothyroidism increases with time since treatment. For example, in the lowest dose group studied by Sridama *et al.* (1984) the observed prevalence was 12% at 1 year post-treatment, 33% at 6 years, 47% at 9 years, and 73% at 11 years. According to Watson, the risk functions developed by the Thyroid Effects Working Group probably reflect the prevalence that would be expected about 1 year after an accident.

However, skin burns might also occur in individuals who receive relatively large doses to the skin from beta emitters. 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. Alpha particles are of little concern, because they do not have sufficient range in tissue to produce skin burns.

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 review (1987).

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 low-LET radiation ED_{50}s of 6 Gy for erythema and 20 Gy for transepidermal injury are derived from Lushbaugh *et al.*'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 30 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% of the time. In contrast, when ^{91}Y (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, Moritz 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) hypothesized 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:

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

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% of all children are borne to mothers younger than 40, 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. Doses greater than 8 Gy can produce permanent suppression in women under 40.

The Working Group's central estimate of the population threshold dose of low-LET radiation 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 ED_{50} for permanent ovulation suppression is 3.5 Gy with lower and upper estimates of 2.5 and 4.5 Gy.

The testes are also 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 nor 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 the 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 ED_{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 two-year suppression of sperm count following brief external exposure to low-LET radiation.

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/fetus following irradiation of the pregnant mother is limited. However, in rats and mice lethality has been observed following low-LET radiation 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 low-LET LD_{50} of 1 Gy during preimplantation (0 - 18 days postconception), 1.5 Gy during the period of growth and development (18 - 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. Section 3.1.4 provides an approach for estimating the risk of fetal death, accounting for the fraction of fetuses/embryos in each developmental stage.

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, if he was completely unmanageable, or had been institutionalized. Most of the children so classified had never been enrolled in school. The few who had entered school had IQs 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.

On the basis of studies of Japanese A-bomb survivors irradiated *in utero*, it was concluded, as reported in BEIR V, that the prevalence of radiation-induced mental retardation was highest in persons irradiated during the 8-15 week period after conception, was less in those irradiated between 16-25 weeks after conception, and was negligible or absent in those irradiated either before 8 weeks or later than 25 weeks after conception. For those irradiated during the 8-15 week, post-conception period, the prevalence of mental retardation appeared to increase with dose in a manner consistent with a linear, nonthreshold response. A linear exponential model was also consistent with the data. The risk at 1 Gy was estimated to be about 43% under the DS86 dosimetry with the linear model and 48% with the linear-exponential model. However, the data do not exclude a threshold in the 0.1 - 0.2 Gy region (Otake *et al.*, 1989). Evidence for a threshold is stronger for the 16-25-week, post-conception period than for the 8-15-week period. The lower estimate of the threshold is 0.21 Gy for the 16-25 week period.

The BEIR V publication also included a discussion of some of the uncertainties associated with these estimates, including the number of cases; the appropriateness of the comparison groups; errors in the estimates of the absorbed dose and calculated prenatal ages at exposure; variation in the severity of mental retardation, and other confounding factors, including malnutrition and diseases.

Brent (1986) and other embryologists have questioned the use of linear models and advocated the use of thresholds. Neumeister and Wasser (1985) recommended continuation of pregnancy following doses as large as 0.1 Gy.

Based on consideration of information provided in BEIR V (NAS/NRC, 1990), ICRP 60 (ICRP, 1991) and UNSCEAR 88 (UNSCEAR, 1988), it was recommended in Addendum 1 (NRC, 1991) that the NUREG/CR-4214 model (NRC, 1990) for mental retardation be modified to allow for uncertainty about threshold dose. In Addendum 1, the Early Effects Working Group recommended that linear models be used for central, upper and lower estimates of risk. They also recommended that the central estimates of the risk of mental retardation incorporate thresholds—specifically 0.1 Gy for those in the 8 to 15 week group and 0.2 Gy for those in the 16 to 25 week group. Lower estimates of risk should also incorporate thresholds of 0.2 and 0.5 Gy for these two age groups. Upper estimates should be evaluated without thresholds.

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 2.5 through 2.7. The values given in Tables 2.5 (general morbidity) and 2.6 (*in utero* effects) apply to brief external exposures to low-LET radiation. Those given in Table 2.7 are appropriate for estimating the risks from protracted exposures to low-LET radiation. The Early Effects Working Group recommends that exposures at dose rates of 0.06 Gy per hour or less be considered protracted exposures. For pulmonary morbidity, if alpha-emitting radionuclides are present in addition to the protracted low-LET radiation, an adjusted dose equal to low-LET dose plus RBE times the alpha dose should be used. 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 from low-LET radiations 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-irradiation 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.

Table 2.5

Models of early morbidity from brief exposures to low-LET radiation[a]

Effect	Risk estimate								
	Central			Lower[b]			Upper[b]		
	ED_{50}	T	V	ED_{50}	T	V	ED_{50}	T	V
Pulmonary morbidity	5	2.5	12	6	3	14	4	2	9
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, except for the prodromal syndrome. For the prodromal syndrome, the dose to the mid-line, mid-plane upper abdomen should be used. The units are gray (Gy). The parameters, ED_{50}, T, and V given in this table are defined in the text of this report. 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 ED_{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, upper estimates of risk should be computed with a threshold much smaller than 2 Gy.

Table 2.6

Models of early morbidity or lethality from brief exposures *in utero* to low-LET radiation[a]

Effect	Risk estimate								
	Central			Lower[b]			Upper[b]		
	D_{50}	T	V	D_{50}	T	V	D_{50}	T	V
Microencephaly	0.7	0.05	0.4	0.8	0.1	1	0.5	0.05	0.2
0-17 weeks									
Severe mental retardation									
8-15 weeks	1.5	0.1	1	3	0.2	1	1	0	1
16-25 weeks	7	0.2	1	10	0.5	1	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[c]	-	-	-	-	-	-	-	-	-

[a] Brief exposure parameters are appropriate for doses 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. 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 D_{50}, T, and V results in the lower estimates of risk, and vice versa.

[c] 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 2.7

Models of early morbidity from protracted exposures to low-LET radiation[a]

Effect	Risk estimate								
	Central			Lower[b]			Upper[b]		
	ED_{50}	T	V	ED_{50}	T	V	ED_{50}	T	V
Pulmonary morbidity[c]	5	2.5	12	6	3	14	4	2	9
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[d]	300	10	-	300	10	-	300	-[e]	-
Erythema	20	6	5	30	8	6	10	4	4
Transepidermal injury	80	40	5	100	50	6	60	30	4
Reproductive[f] effects	-	-	-	-	-	-	-	-	-
Cataracts[g]	-	-	-	-	-	-	-	-	-

[a] Protracted exposure parameters are appropriate for doses received at low dose rate ($\leq$0.06 Gy/hr). The doses referred to in this table are organ-specific absorbed doses. The units are gray (Gy). 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 D_{50}, T, and V results in the lower estimates of risk, and vice versa.

[c] As noted in the text, the Working Group recommends using the dose-rate-dependent model to obtain model parameters. Parameter values for ED_{50} and T should be one-half of the values used for lethality from pulmonary injury. The shape parameter V for morbidity is assigned the same value as for mortality. If alpha-emitting radionuclides are also present, use an adjusted dose = low-LET dose plus RBE times the alpha-dose. To evaluate the contributions of α-emitting radionuclides, a fixed RBE of 7, is recommended with upper and lower estimates of 5 and 10; respectively.

[d] 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.

[e] 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.

[f] Parameters for protracted exposure were not developed.

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

Most 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.

Even fewer human populations are available for study to estimate cancer risks for internally deposited alpha-emitting radionuclides. The major populations that are available are persons that ingested ^{226}Ra in the course of their work, were injected with ^{224}Ra for therapeutic reasons, were injected with Thorotrast for medical diagnostic purposes, or inhaled radon and its progeny while mining uranium. Because there are many alpha-emitting radionuclides whose metabolism and dosimetry are different from these naturally occurring radionuclides, it is necessary to supplement these human data bases with results from life-span studies in laboratory animals. The use of data from laboratory animals requires the additional complexity of extrapolation to human health risks.

To derive risk estimates for most cancers for low-LET radiations, the Late Somatic Effects Working Group recommends the use of proportional models of the form:

$$R = \left(\frac{1}{DDREF}\right) cD$$

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, and DDREF is the dose and dose-rate effectiveness factor.

To derive central risk estimates for most cancers, the Late Somatic Working Group recommends that the DDREF for low-LET radiation be chosen so that doses received at low dose and dose rate are only one-half as effective as equivalent doses received at high rates, i.e., DDREF=2. This central estimate of the DDREF was chosen from a range of values—2 to 10 as discussed in Addendum 1 (NRC, 1991). Many European accident consequence calculation codes rely on low dose rates being 45% as effective as high rates. To reflect the uncertainty in this choice, the lower risk estimates for most cancers are based on a DDREF of 4 and their upper estimates assume that doses received at low dose rates are as effective as doses received at high rates, i.e., DDREF=1. A DDREF greater than 1 should be used for all doses received at rates less than 0.1 Gy per hour and for all total doses less than 0.2 Gy regardless of dose rate.

Two exceptions to this general approach are the models for breast and thyroid cancer. For thyroid cancer, no DDREF is used. For breast cancer, no DDREF is used for the upper or central risk estimate, but a DDREF of 4 is included in the lower bound risk estimate.

The upper and lower estimates for low-LET radiation reflect uncertainty from several sources including a factor of two for the choice of DDREF. The uncertainty from sources other than the choice of DDREF applies to alpha as well as low-LET radiation. In Addendum 2, this factor of two was also included, but it was attributed to uncertainty regarding the shape of the dose-response functions and to uncertainty regarding the dependence of risks on factors such as the chemical and physical form of the radionuclide and uncertainty in RBE.

The two approaches most commonly used for projecting the impact of incomplete followup are absolute risk projection and relative risk projection. Both of these approaches allow for a latency period—during which there is no radiation-induced cancer risk—and an expression period—during which the effects of exposure to radiation are expressed. The expression 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. With an absolute risk projection model, the excess risk is assumed to be constant for a specified range of ages. With a relative risk projection model, the excess risk is assumed to be a constant fraction of the baseline age-specific risk. Because background rates for most cancers increase strongly with age, relative risk models tend to yield projections of risk that are higher than those derived using absolute risk models.

An important issue related to the evaluation of relative risk 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, the Working Group's central and upper estimates of the risks for the solid tumors are based on the assumption that relative risks depend on age at exposure. For these cancers, the relative risks for those under 20 at the time of exposure are typically two to four times as great as for those over 20.

Many nuclear power plant accident scenarios involve the potential for exposures to alpha-emitting radionuclides, e.g., plutonium, americium, and curium. Scott *et al.* (NRC, 1993) suggest that inhalation of actinide radionuclides would be the primary mode of exposure to alpha emitters, and that the critical targets would be the lung, liver, and skeleton. To account for the effects of combined exposure to low-LET beta and gamma and high-LET alpha radiations, the Working Group recommends that the risks from low-LET and alpha radiation be evaluated separately and added. Using this approach, the risk is given by an equation of the form

$$\text{Risk}_{\alpha,\beta,\gamma} = \text{Risk}_{\beta,\gamma} + \text{Risk}_{\alpha} = K_{hi}D_{hi} + K_{lo}D_{lo} + K_{\alpha}D_{\alpha}$$

where $\text{Risk}_{\alpha,\beta,\gamma}$ is the overall risk; $\text{Risk}_{\beta,\gamma}$ and Risk_{α} are the low- and high-LET risks, respectively; D_{hi} is the low-LET dose received at high dose rates; D_{lo} is the low-LET dose received at low dose rates and any dose less than 0.2 Gy regardless of dose rate; and D_{α} is the alpha radiation dose. K_{hi} and K_{lo} are the risk coefficients associated with D_{hi} and D_{lo} of low-LET radiation, respectively, and K_{α} is the risk coefficient for alpha radiation. From the equation on p. 30, $K_{hi} = c$, $K_{lo} = c/\text{DDREF}$ and $K_{\alpha} = (c/\text{DDREF})(\text{RBE})$. To facilitate evaluation of risk and its associated uncertainties, the above equation can be rewritten as

$$\text{Risk}_{\alpha,\beta,\gamma} = K_{hi}\,[D_{hi} + U(D_{lo} + \text{RBE}\,D_{\alpha})]$$

where U is a factor based on collective judgement that reflects uncertainty due to the effectiveness of low-LET radiation delivered at low dose rates and low doses delivered at any dose rate compared to that delivered at high doses and dose rates and uncertainty from other sources for alpha radiation. U will take

on different values for central, upper, and lower bound estimates. For some models, K_{hi} also takes on different values for the three estimates, reflecting uncertainty in projection over time, treatment of age at exposure, and transportation. An RBE of 20 is used for alpha radiation relative to low LET exposure received at low dose rates (ICRP, 1991).

For low-LET radiation, 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, ovary, and lymphomas), and for both leukemias and other cancers associated with *in utero* exposure. For alpha radiation, models are provided for cancers of the lung, liver and skeleton as these are the only organs likely to be exposed. This site-specific approach was taken because of the non-uniformity of the organ doses that may occur in nuclear power plant accidents.

Both incidence and mortality risks have been estimated 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% 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.

One situation that deserves special attention is analysis of risk associated with radionuclides inhaled from an airborne plume. Several 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 years after they are inhaled and deposited in the body. Their dose will be delivered gradually. As time proceeds, the population exposed to the plume will age and dwindle in size. Direct application of our basic risk models will lead to an overestimation of the radiation-induced cancer risk faced by this population. Tables are given in Appendix B that account for the changing size and age-structure of this population.

2.2.1 Leukemia

The estimates of leukemia risk are based on absolute risk projection with a latency period of 2 years and an expression period of 25 years. In fact, more of the risk will occur in the early part of the expression period than in the later part, and some risk will occur more than 27 years after exposure. However, these two effects tend to offset each other.

The recommended risk coefficient, 4.5×10^{-4} deaths per person-yr-Gy, was derived by doubling the coefficient from the BEIR III analysis of the early data (1950-1971) from the Japanese atomic bomb survivors. The doubling is necessary to account for the impacts of revisions in the atomic bomb dosimetry (a factor of 1.7) and additional follow-up of the survivors (a factor of 1.2).

A proportional dose-response model is recommended. The only difference in the central, upper, and lower estimates is in the treatment of dose received at low dose and dose rate. For upper estimates, it is recommended that the dose received at low dose and dose rate is assumed to be as effective as the dose received at high dose rate. For central and lower estimates, the use of DDREF of 2 and 4, respectively, is recommended.

The resulting models of leukemia risk are:

$$R_{upper} = 9.7\ (D_{hi} + D_{lo})$$

$$R_{central} = 9.7\ (D_{hi} + 0.5\ D_{lo})$$

$$R_{lower} = 9.7\ (D_{hi} + 0.25\ D_{lo})$$

where R is the lifetime population risk (deaths/1000 persons), D_{hi} is the dose (Gy) to the red bone marrow received at high dose rate, and D_{lo} is the dose to this same tissue received at low dose rate and any dose less than 0.2 Gy regardless of dose rate.

The loss of life expectancy associated with a leukemia death is estimated to be 40 years.

2.2.2 Bone Cancer

The Working Group's estimates of bone cancer risks uses absolute risk projection with a latency period of 2 years and an expression period of 25 years. The original low-LET risk coefficient (1.0×10^{-5} deaths per person-yr-Gy), based on the BEIR III estimate of 1×10^{-4} deaths per person-yr-Gy (alpha) observed among patients given ^{224}Ra injections and on data described in UNSCEAR 77, was increased in the Addendum 1 report (NRC, 1991) by a factor of 2 to make it more consistent with the value recommended in ICRP Publication 60 (ICRP, 1991).

Subsequent to the publication of Addendum 1, Puskin *et al.* (1992) noted problems with the derivation of bone-cancer risk from the radium-224 data as done in ICRP 60. The value in ICRP 60 was based on the average dose to the skeletal mass, not the dose to endosteal bone surfaces. Thus, to obtain a corresponding mortality risk estimate for low-dose-rate, low-LET irradiation, this risk coefficient should first be expressed as a function of the dose to bone surfaces, rather than average dose to the skeleton, because ^{224}Ra mainly irradiates the surfaces, and because critical target cells are presumed to reside at these surfaces. One first divides by 7.5 (Puskin *et al.*, 1992) to obtain 12.4 alpha-radiation-induced, bone sarcoma deaths per 10^4 person-Gy, based on dose to the surface of bone. The corresponding low-LET

estimate is 0.6 bone sarcoma deaths per 10^4 person-Gy, based on an RBE of 20 for alpha radiation and on dose to the surface of bone. This discrepancy was corrected in the Addendum 2 report (NRC, 1993).

A proportional dose-response model is recommended. The only difference in the central, upper, and lower estimates for low-LET radiation is in the treatment of the dose received at low dose and low dose rates. For upper estimates, it is recommended that the dose received at low dose rate is assumed to be as effective as the dose received at the high dose rate. For central and lower estimates, DDREFs of 2 and 4, respectively, are recommended.

The resulting models of bone cancer risk are:

$$R_{upper} = 0.12\ (D_{hi} + D_{lo} + 20\ D_{\alpha}),$$

$$R_{central} = 0.12\ [D_{hi} + 0.5\ (D_{lo} + 20\ D_{\alpha})],$$

$$R_{lower} = 0.12\ [D_{hi} + 0.25\ (D_{lo} + 20\ D_{\alpha})],$$

where R is the lifetime population risk (deaths/1000 persons) and D is the dose (Gy) to the bone. The loss of life expectancy associated with a bone cancer is estimated to be 40 years.

2.2.3 Breast Cancer

The central and upper estimates of breast cancer risk are based on relative risk projection with a (minimal) latency period of 10 years, a minimum age at induction of 30 years, and a lifetime expression period. Both central and upper estimates reflect a dependence of risk on age at exposure.

The excess relative risk coefficients recommended for central estimates are—70% per Gy for women under 20 at the time of exposure, 30% per Gy for women between 20 and 40, and 10% per Gy for those over 40. The strong influence of age at exposure is consistent with the BEIR V model and with the study by Miller *et al.* (1989) of Canadian women who were treated with fluoroscopy for tuberculosis. For upper estimates, the recommended coefficients are 100% per Gy for women under 20, and 40% per Gy for those older than 20. These were derived from BEIR III and are 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 lower estimate of breast cancer risk is based on absolute risk projection with a latency period of 10 years and a lifetime expression period. Absolute risk coefficients of 7.4×10^{-4} cases per woman-yr-Gy and 2.6×10^{-4} deaths per woman-yr-Gy are recommended for incidence and mortality, respectively. The incidence estimate was derived by pooling the age-specific absolute risk coefficients from BEIR III, i.e., 10.4×10^{-4} cases per woman-yr-Gy for those between 10 and 19 at the time of exposure, and 6.6×10^{-4} cases per woman-yr-Gy for those over 20, weighting by the inverse variances of the estimates. The

mortality coefficient was obtained by multiplying this estimate by the ratio of background mortality to background incidence.

A proportional dose-response model is recommended. Because, for breast cancer, there is little evidence of decreased effectiveness of dose received at a low dose rate, only in the lower estimates is there any adjustment for dose rate. For both upper and central estimates, it is recommended that the dose received at low doses and dose rates is assumed to be as effective as dose received at high dose rates. For lower estimates, a DDREF of 4 is recommended.

The resulting estimates of breast cancer risk are:

$$R_{I,upper} = 25\ (D_{hi} + D_{lo})$$

$$R_{I,central} = 16\ (D_{hi} + D_{lo})$$

$$R_{I,lower} = 12\ (D_{hi} + 0.25\ D_{lo})$$

$$R_{M,upper} = 8.4\ (D_{hi} + D_{lo})$$

$$R_{M,central} = 5.4\ (D_{hi} + D_{lo})$$

$$R_{M,lower} = 4.3\ (D_{hi} + 0.25\ D_{lo})$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons), D_{hi} is the low-LET dose (Gy) to the breasts received at high dose rate, and D_{lo} is the low-LET dose to this same tissue received at the low dose rate and any dose less than 0.2 Gy regardless of dose rate. Note that these estimates apply to the entire population. Risks to women would be twice this large.

The loss of life expectancy associated with a radiation-induced breast cancer is estimated to be 17 years under the assumptions used in the upper or central 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 central and upper estimates of lung cancer risk are based on relative risk projection with a (minimal) latency period of 10 years, a minimum age at induction of 40 years, and a lifetime expression period. Both central and upper estimates reflect a dependence of risk on age at exposure.

The excess relative risk coefficients recommended for upper estimates are, 150% per Gy for people under 20 at the time of exposure and 50% per Gy for those over 20. The estimate of 50% per Gy was obtained by averaging the BEIR V relative risk coefficients for males (42% per Gy) and females (64% per Gy).[a] The use of relative risk coefficients three times as large for those under 20 as for those over 20 is consistent with Preston and Pierce (1987). For central estimates, the recommended coefficients are 60% per Gy for people under 20, and 30% per Gy for those older than 20. The 30% per Gy value was derived by reducing the relative risk coefficient obtained directly from the Japanese data by a factor of two. This choice reflects the difference between the additive and multiplicative transport models.

The lower estimate of lung cancer risk is based on absolute risk projection with a latency period of 10 years and a lifetime expression period. Absolute risk coefficients of 2.7×10^{-4} cases per person-yr-Gy and 2.5×10^{-4} deaths per person-yr-Gy are recommended for incidence and mortality, respectively. The mortality coefficient is the value derived from analysis of the Japanese Life-Span Study. It has been adjusted to reflect the impact of the revised A-bomb dosimetry. The incidence coefficient was obtained by scaling this value by the ratio of background incidence to background mortality.

A proportional dose-response model is recommended. The central, upper, and lower estimates for low-LET radiation differ in the treatment of the doses received as low total doses or at low dose rates. For upper estimates, it is recommended that the dose received at the low dose rate is assumed to be as effective as the dose received at the high dose rate. For central and lower estimates, DREFs of 2 and 4, respectively, are recommended.

The resulting estimates of lung cancer risk are:

$$R_{I,upper} = 37\,(D_{hi} + D_{lo} + 20\,D_{\alpha})$$

$$R_{I,central} = 17\,[D_{hi} + 0.5\,(D_{lo} + 20\,D_{\alpha})]$$

$$R_{I,lower} = 7.2\,[D_{hi} + 0.25\,(D_{lo} + 20\,D_{\alpha})]$$

$$R_{M,upper} = 33\,(D_{hi} + D_{lo} + 20\,D_{\alpha})$$

$$R_{M,central} = 16\,[D_{hi} + 0.5\,(D_{lo} + 20\,D_{\alpha})]$$

$$R_{M,lower} = 6.7\,[D_{hi} + 0.25\,(D_{lo} + 20\,D_{\alpha})]$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons), D_{hi} is the low-LET dose (Gy) to the lungs received at high dose rate and D_{lo} is

[a] Although the preferred BEIR V model includes a decrease in risk with time since exposure, the values reported were taken from an alternative BEIR V model without such a decrease.

the low-LET dose to the lung received at low dose rate and any low-LET dose less than 0.2 Gy regardless of dose rate. D_α is the alpha dose to the same tissue. Note that the ratio of the coefficient of D_α to that for D_{lo} yields an RBE of 20.

The loss of life expectancy associated with a radiation-induced lung cancer is estimated to be about 15 years under the assumptions used in the central and upper models, and 18 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.

As is true in any of the age-dependent models used in this report, these models for lung cancer risk apply only to a population with a specified composition related to age at exposure and gender. These estimates would be different for populations that were either much younger or much older than those assumed here.

2.2.5 Gastrointestinal Cancer

The central and upper estimates of gastrointestinal cancer risk are based on relative risk projection with a latency period of 10 years and a lifetime expression period. Both central and upper estimates reflect a dependence of risk on age at exposure.

The excess relative risk coefficients recommended for central and upper estimates are 120% per Gy for those younger than 20 at the time of exposure, and 40% per Gy for those older than 20. The coefficient, 120% per Gy, is quite similar to the average of the BEIR V relative risk coefficients for males and females who were under 25 at the time of exposure. These relative risks are assumed to apply to both incidence and mortality.

The lower estimate of gastrointestinal cancer risks is based on absolute risk projection with a 10-year latency period and a lifetime expression period. Risk coefficients of 6.8×10^{-4} cases and 4.0×10^{-4} deaths per person-yr-Gy are recommended for incidence and mortality, respectively. The mortality coefficient is based on the absolute risk coefficient, 3.4×10^{-4} deaths per person-yr-Gy (shielded kerma), given by Shimizu *et al.* (1990). It has been adjusted to permit analysis on the basis of dose to the gastrointestinal tract, rather than shielded kerma. The incidence coefficient was obtained by scaling the mortality coefficient by the background ratio of incidence to mortality.

A proportional dose-response model is recommended. The central, upper, and lower estimates for low-LET radiation differ in the treatment of the dose received at low dose and low dose rates. For upper estimates it is recommended that the dose received at the low dose rate is assumed to be as effective as the dose received at the high dose rate. For central and lower estimates, DREFs of 2 and 4, respectively, are recommended.

The effect of α-emitters on gastrointestinal cancer is computed by assuming that 10% of GI cancers are liver cancers, that only these are affected by alpha radiation, and that the RBE of 20 used for the lung and bone cancers also applies here.

The resulting estimates of gastrointestinal cancer risk are:

$$R_{I,upper} = 58\ (D_{hi} + D_{lo} + 2\ D_{\alpha})$$

$$R_{I,central} = 58\ [D_{hi} + 0.5\ (D_{lo} + 2\ D_{\alpha})]$$

$$R_{I,lower} = 23\ [D_{hi} + 0.25\ (D_{lo} + 2\ D_{\alpha})]$$

$$R_{M,upper} = 34\ (D_{hi} + D_{lo} + 2\ D_{\alpha})$$

$$R_{M,central} = 34\ [D_{hi} + 0.5\ (D_{lo} + 2\ D_{\alpha})]$$

$$R_{M,lower} = 14\ [D_{hi} + 0.25\ (D_{lo} + 2\ D_{\alpha})]$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons), D_{hi} is the low-LET dose (Gy) received at the high dose rate, D_{lo} is the low-LET dose to this same tissue received at a low dose rate, and any low-LET dose less than 0.2 Gy regardless of dose rate. D_{α} is the alpha dose received by the liver.

To calculate gastrointestinal cancer risk, it is recommended that a composite of the low-LET doses to the esophagus, stomach, colon, and liver be used. The recommended weighted low LET dose is:

$$D_{gi\ tract} = 0.05\ D_{esophagus} + 0.30\ D_{stomach} + 0.55\ D_{colon} + 0.10\ D_{liver}$$

where the D_{organs} are the doses (Gy) to each relevant organ. When alpha-emitting radionuclides are included, D_{α} to the liver must also be included, adjusted by the relative effectiveness of alpha radiation. A relative effectiveness factor of 20 is used here.

The loss of life expectancy associated with a radiation-induced gastrointestinal cancer 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.

To compute the risk of liver cancer separately, the following relationships are used.

$$R_{I,upper} = 5.8\ [D_{hi} + D_{lo} + 20\ D_{\alpha}]$$

$$R_{I,central} = 5.8\ [D_{hi} + 0.5\ (D_{lo} + 20\ D_{\alpha})]$$

$$R'_{I,lower} = 2.3\ [D_{hi} + 0.25\ (D_{lo} + 20\ D_{\alpha})]$$

$$R_{M,upper} = 3.4\,[D_{hi} + D_{lo} + 20\,D_{\alpha}]$$

$$R_{M,central} = 3.4\,[D_{hi} + 0.5\,(D_{lo} + 20\,D_{\alpha})]$$

$$R_{M,lower} = 1.4\,[D_{hi} + 0.25\,(D_{lo} + 20\,D_{\alpha})]$$

where R_I is the lifetime incidence risk (causes/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons), D_{hi} is the low-LET dose (Gy) to the liver received at high dose rate, D_{lo} is the low-LET dose to the liver received at low dose rate, and D_{α} is the high-LET dose to this same tissue.

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% of all radiation-induced thyroid cancers would be fatal. The resulting estimates of population risk are:

$$R_I = 7.2\,D \text{ and } R_M = 0.7\,D$$

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 low-LET dose (Gy) to the thyroid gland from external irradiation.

Studies of thyroid cancer following exposure to ^{131}I have produced largely negative results, 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 9.3×10^{-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 gamma irradiation. Doses from internal low-LET sources, such as ^{131}I, are thought to be only 1/5th as effective as doses from brief exposure to gamma radiation 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. Rather than developing site-specific risk estimation models for each of these cancers, the Working Group developed a lumped model for "other cancers."

The central and upper estimates of the risk of "other cancers" are based on relative risk projection with a latency period of 10 years and a lifetime expression period. Both central and upper estimates reflect a dependence of risk on age at exposure.

The excess relative risk coefficients recommended for central and upper estimates are, 110% per Gy for those younger than 20 at the time of exposure and 25% per Gy for those older than 20. The coefficient, 110% per Gy, is the average of the BEIR V relative risk coefficients for males and females who were between 5 and 15 at the time of exposure. The strong influence of age at exposure is consistent with the BEIR V analysis of other cancers. These relative risks are assumed to apply to both incidence and mortality.

The lower estimate of other cancer risks is based on absolute risk projection with a 10-year latency period and a lifetime expression period. Risk coefficients of 6.8×10^{-4} cases and 3.5×10^{-4} deaths per person-yr-Gy are recommended for incidence and mortality, respectively. Shimizu *et al.* (1990) indicate that the sum of the absolute risk coefficients for cancers other than leukemia, breast, lung, and gastrointestinal is 2.6×10^{-4} deaths per person-yr-Gy (kerma). If this is adjusted to reflect organ dose rather than shielded karma, the result is approximately 3.5×10^{-4} deaths per person-yr-Gy. Our incidence coefficient was obtained by scaling this mortality coefficient by the background ratio of incidence to mortality.

A proportional dose-response model is recommended. The central, upper, and lower estimates differ in the treatment of low-LET dose received at low dose rate. For upper estimates it is recommended that the dose received at the low dose rate is assumed to be as effective as the dose received at the high dose rate. For central and lower estimates, DREFs of 2 and 4, respectively, are recommended.

The resulting estimates of the risk of other cancers are:

$$R_{I,upper} = 55\,(D_{hi} + D_{lo})$$

$$R_{I,central} = 55\,(D_{hi} + 0.5\,D_{lo})$$

$$R_{I,lower} = 23\,(D_{hi} + 0.25\,D_{lo})$$

$$R_{M,upper} = 28\ (D_{hi} + D_{lo})$$

$$R_{M,central} = 28\ (D_{hi} + 0.5\ D_{lo})$$

$$R_{M,lower} = 12\ (D_{hi} + 0.25\ D_{lo}),$$

where R_I is the lifetime incidence risk (cases/1000 persons), R_M is the lifetime mortality risk (deaths/1000 persons), D_{hi} is the low-LET dose (Gy) received at the high dose rate, and D_{lo} is the low-LET dose received at the low dose rate and any total low-LET dose less than 0.2 Gy regardless of dose rate.

Selection of an appropriate measure of dose to use for calculating the risks of "other cancers" 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. It is recommended that a composite of the low-LET doses to the bone marrow, kidney, urinary bladder, brain, and ovary be used. Weights proportional to the background incidence rates of cancers associated with each of these organs could be used to construct the composite dose. Based on the 1980 background cancer rates, the weighted dose computed using this approach would be:

$$D_{other} = 0.06\ D_{bone} + 0.11\ D_{kidney} + 0.26\ D_{bladder} + 0.09\ D_{brain} + 0.48\ D_{ovary}$$

where the D_{organs} are the doses (Gy) to each of the relevant organs.

The loss of life expectancy associated with other cancers induced by radiation 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:

$$R_{leukemia} = 0.3\ D$$

$$R_{other\ childhood\ cancer} = 0.3\ D$$

where D is the low-LET dose (Gy) to the fetus, and R is the risk (childhood cancers/1000 exposed

persons). 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.

Our central (and lower) estimates of childhood cancers from *in utero* exposures are based on the UNSCEAR (1972) estimate of 2.3×10^{-2} total childhood cancers per embryo per Gy. This estimate, which includes both leukemia and other childhood cancers, was not modified in the subsequent UNSCEAR reports (UNSCEAR 1977; 1986). It is about 40% 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 and 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 non-uniform. To account for this, Dr. Keith Eckerman of Oak Ridge National Laboratory (personal communication) recommended that the following low-LET dose estimates be used:

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

$$D_{\text{fetus, other organ}} = 0.03\, D_{\text{mother's bone marrow, strontium}} + 0.5\, D_{\text{mother's uterus, cesium}} + 0.05\, D_{\text{mother's thyroid, iodine}} + 0.0\, D_{\text{mother's maximum organ dose, other radioisotopes}} + 1.0\, D_{\text{mother's uterus, external sources}}$$

2.2.9 Skin Cancer

Most skin cancers are not lethal and are 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 increased incidence of skin cancer.

The risk of skin cancer following a nuclear power plant accident is quite difficult to estimate. Most studies of radiation-induced skin cancer have involved exposure to X rays. The importance of the differences in penetrating power of beta emitters 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 can be successfully treated with only minor inconvenience to the patient, they are not reported reliably in tumor registries. Available epidemiological results vary considerably and include a number of studies with largely negative results. Existing data are not adequate to determine the shape of the dose-response function, the latency, or the effect of age-at-exposure.

The central and upper estimates of the skin cancer risk are based on relative risk projection with a latency period of 10 years and a lifetime expression period. The excess relative risk coefficient of 50% per Gy is based on an analysis by Shore (1990) that combines risk estimates from several studies—considering the area of the body irradiated, and providing separate coefficients for those parts of the body exposed to ultraviolet irradiation (face, neck and dorsal aspect of the hands and arms) and those parts not exposed (remainder of the body). Shore's coefficient of 58% per Gy was reduced (by 90%) to account for the fact that about 90% of all skin cancers occur on parts of the body exposed to ultraviolet irradiation.

The lower estimate of skin cancer risks is based on absolute risk projection with a 10-year latency period and a lifetime expression period. The recommended risk coefficient of 6.7×10^{-4} per person-yr-Gy is the absolute risk value given in ICRP Publication 60 for UVR-exposed skin (ICRP, 1991).

A proportional dose-response model is recommended. The central, upper, and lower estimates differ in the treatment of dose received at low dose rate. For upper estimates, it is recommended that the dose received at the low dose rate is assumed to be as effective as the dose received at the high dose rate. For central and lower estimates, DREFs of 2 and 4, respectively, are recommended. Although Publication 60 did not reduce risks from protracted exposures, it indicated that such a reduction was likely (ICRP, 1991).

The resulting estimates of the skin cancer risk are:

$$R_{I,upper} = 89\ (D_{hi} + D_{lo})$$

$$R_{I,central} = 89\ (D_{hi} + 0.5\ D_{lo})$$

$$R_{I,lower} = 22\ (D_{hi} + 0.25\ D_{lo})$$

where R_I is the lifetime population risk (persons with skin cancer/1000 persons), D_{hi} is the low-LET dose (Gy) to the UVR-exposed skin received at the high dose rate, and D_{lo} is the low-LET dose to this same tissue received at the low dose rate and any total dose of low-LET radiation less than 0.2 Gy regardless of dose rate. Note that this model predicts the number of people with skin cancer, rather than the total number of skin cancers.

The Late Somatic Effects Working Group recommends that the risk calculated be on the basis of dose to the face because about 85% of basal cell carcinomas (the predominant type resulting from ionizing radiation exposure) 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 emitters 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 2.8 (morbidity) and Table 2.9 (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 manifest itself 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, great-grandchildren, and subsequent generations. In addition, there would be small increases in the rates of spontaneous abortions, 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 extrapolation from animal models. The limited human data relevant for genetic risk assessment come from studies of the children of survivors of the atomic bombings of Hiroshima and Nagasaki. Although they have not revealed any excess incidence of genetic defects, these studies are not powerful enough to reject current theories of genetic risk.

Table 2.8

Models of cancer morbidity[a]

Effect	Lifetime risk (cases/1000)		
	Upper	Central	Lower
Breast cancer[b]	25 (D_{hi} + D_{lo})	16 (D_{hi} + D_{lo})	12 (D_{hi} + 0.25 D_{lo})
Lung cancer	37 (D_{hi} + D_{lo} + 20 D_{α})	17 [D_{hi} + 0.5 (D_{lo} + 20 D_{α})]	7.2 [D_{hi} + 0.25 (D_{lo} + 20 D_{α})]
GI cancer[c]	58 (D_{hi} + D_{lo} + 2 D_{α})	58 [D_{hi} + 0.5 (D_{lo} + 2 D_{α})]	23 [D_{hi} + 0.25 (D_{lo} + 2 D_{α})]
Liver cancer	5.8 (D_{hi} + D_{lo} + 20 D_{α})	5.8 [D_{hi} + 0.5 (D_{lo} + 20 D_{α})]	2.3 (D_{hi} + 0.25 (D_{lo} + 20 D_{α})]
Thyroid cancer[d]	7.2 D	7.2 D	7.2 D
Benign thyroid nodules[e]	27 D	27 D	27 D
Skin cancer	89 (D_{hi} + D_{lo})	89 (D_{hi} + 0.5 D_{lo})	22 (D_{hi} + 0.25 D_{lo})
Other cancer[f]	55 (D_{hi} + D_{lo})	55 (D_{hi} + 0.5 D_{lo})	23 (D_{hi} + 0.25 D_{lo})

[a] The doses, D, referred to in this table are organ-specific absorbed doses. The units of dose are gray (Gy). The subscripts "lo" and "hi" are used to distinguish low doses of low-LET radiation, i.e. <0.2 Gy, and doses received at low dose rates, i.e., <0.1 Gy/hr, from the dose received at the high dose rate. D_{α} refers to the dose from α-emitters, regardless of dose rate. 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] The alpha radiation component of this combined risk originates in the liver. The risk for liver alone is given on the next line.

[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] 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.

[f] 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 2.9

Models of cancer mortality[a]

Effect	Lifetime risk (deaths/1000)		
	Upper	Central	Lower
Leukemia	9.7 (D_{hi} + D_{lo})	9.7 (D_{hi} + 0.5 D_{lo})	9.7 (D_{hi} + 0.25 D_{lo})
in utero[b]	0.3 D	0.1 D	0.1 D
Bone cancer	0.12 (D_{hi} + D_{lo} + 20 D_{α})	0.12 [D_{hi} + 0.5 (D_{lo} + 20 D_{α})]	0.12 [D_{hi} + 0.25 (D_{lo} + 20 D_{α})]
Breast cancer[c]	8.4 (D_{hi} + D_{lo})	5.4 (D_{hi} + D_{lo})	4.3 (D_{hi} + 0.25 D_{lo})
Lung cancer	33 (D_{hi} + D_{lo} + 20 D_{α})	16 [D_{hi} + 0.5 (D_{lo} + 20 D_{α})]	6.7 [D_{hi} + 0.25 (D_{lo} + 20 D_{α})]
GI cancer[d]	34 (D_{hi} + D_{lo} + 2 D_{α})	34 [D_{hi} + 0.5 (D_{lo} + 2 D_{α})]	14 [D_{hi} + 0.25 (D_{lo} + 2 D_{α})]
Liver cancer	3.4 (D_{hi} + D_{lo} + 20 D_{α})	3.4 [D_{hi} + 0.5 (D_{lo} + 20 D_{α})	1.4 [D_{hi} + 0.25 (D_{lo} + 20 D_{α})]
Thyroid cancer[e]	0.7 D	0.7 D	0.7 D
Other cancer[f]	28 (D_{hi} + D_{lo})	28 (D_{hi} + 0.5 D_{lo})	12 (D_{hi} + 0.25 D_{lo})
in utero[b]	0.3 D	0.1 D	0.1 D

[a] The doses, D, referred to in this table are organ-specific absorbed doses. The units of dose are gray (Gy). The subscripts "lo" and "hi" are used to distinguish low doses of low-LET radiation, i.e. <0.2 Gy and doses received at the low dose rate, i.e., <0.1 Gy/hr, from the dose received at the high dose rate. Refer to the text for explanation of the organ dose appropriate for estimating the risk of each specific cancer. D_{α} refers to the dose from α-emitters, regardless of dose rate.

[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] The alpha radiation component of this combined risk originates in the liver. The risk for liver alone is given on the next line.

[e] 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.

[f] 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.

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 that 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.

The Genetic Effects Working Group relied heavily on analyses provided in BEIR I, III, IV, and V reports of the National Academy of Sciences (NAS/NRC, 1972, 1980, 1988, 1990), as well as those described in recent reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1977, 1982, 1986, 1988) and the International Commission on Radiation Protection (ICRP, 1991). 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 a dose is received at a 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., Drosophila oogonia mutations and Tradescantia mutations, indicates that when the dose is received at a 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., most 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., $r=(a + bD_{\beta,\gamma})\, cD_{\beta,\gamma}$, 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 (represented by uppercase R) of most genetic effects using models of the form:

$$R = (1 + D_{\beta,\gamma})\, bcD_{\beta,\gamma}$$

where $D_{\beta,\gamma}$ is the gonadal low-LET dose (Gy), the product bc is the risk coefficient observed at low dose rates, and the term $1 + D_{\beta,\gamma}$ modifies the risk to account for the effects of high dose rates. When an accident scenario involves only chronic exposure at low dose rates, the modifying term is dropped, and the risk is computed as:

$$R = bcD_{\beta,\gamma}$$

This simplification may also be used when the dose is received at a 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% 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.

In the evaluation of protracted dose, a fixed RBE may be used to account for the increased effectiveness of the contribution of alpha-emitting radionuclides, i.e.,

$$D_p = D_{p,\beta,\gamma} + RBE * D_{p,\alpha}$$

where $D_{p,\beta,\gamma}$ is the protracted dose from beta- and gamma-emitting radionuclides, $D_{p,\alpha}$ is the protracted dose from alpha-emitting radionuclides, and RBE is the relative biological effectiveness of these two classes of dose for inducing genetic effects. The RBEs are endpoint-specific. In the following analyses, different values of the RBE for alpha radiation are used for different classes of biological effects. An RBE of 2.5 is used for all mutations and 15 is the RBE used for unbalanced translocations (NRC, 1993). Obviously, one can also obtain the same results by evaluating the high-LET and low-LET risks separately and adding the risks. The Genetic Effects Working Group recommended that the latter approach be used.

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 birth rate 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% 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. The experimentally observed single locus mutation rates (37/2646 at a dose of 6 Gy) were adjusted 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 (1/6), dose rate (1/3) and fractionation involved (1/1.9) in the experiments (BEIR III, NAS/NRC, 1980). Upper and lower estimates of 4.5×10^{-3} per gamete (ovum or mature sperm) and 0.5×10^{-3} (sperm only) are obtained using the upper and lower estimates of the number of dominant disorders and the fraction of these thought to yield serious defects. The central estimate of the induction rate of dominant disorders in humans of 1.5×10^{-3} per gamete (ovum or mature sperm) per Gy may be obtained using the geometric mean of the range of values given for the number of dominant disorders and for the fraction of these thought to be serious. Upper and central estimates of population risk assume equal sensitivity of males and females. Lower estimates of population risk assume that only males are sensitive. The Working Group estimated that approximately 80% of dominant disorders are transmitted from one generation to the next.

Using the computational scheme outlined in Section 3.3, the final additive models of integrated risk were derived:

$$R_{\text{dominant, upper}} = 22\, D_{\beta,\gamma} + 22\, D^2_{\beta,\gamma} + 55\, D_{\alpha}$$

$$R_{\text{dominant, central}} = 7.5\, D_{\beta,\gamma} + 7.5\, D^2_{\beta,\gamma} + 19\, D_{\alpha}$$

$$R_{\text{dominant, lower}} = 1.2\, D_{\beta,\gamma} + 1.2\, D^2_{\beta,\gamma} + 3.0\, D_{\alpha}$$

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 $D_{\beta,\gamma}$ is the gonadal dose (Gy) from beta- and gamma emitting radionuclides, and D_{α} is the dose from alpha emitting radionuclides received by a representative individual in the exposed population. An RBE of 2.5 is implicit in the ratio of the β, γ and α risk coefficients.

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% of the risk will be expressed in the first generation, 16% in the second, and so forth. Under the central model an acute dose of 1 Gy of low-LET radiation 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 estimate would be three times this large, and the lower estimate would be $1/6^{th}$ 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% 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 of low-LET radiation was adjusted to reflect the total number of x-linked diseases. McKusick's 1983 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 uncertainty about 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. Upper and central estimates of population risk assume equal sensitivity of the ovum and sperm. Lower estimates assume that the mutation rate is zero in the female.

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

$$R_{\text{x-linked, upper}} = 9\, D_{\beta,\gamma} + 9\, D^2_{\beta,\gamma} + 23\, D_{\alpha}$$

$$R_{\text{x-linked, central}} = 2.2\, D_{\beta,\gamma} + 2.2\, D^2_{\beta,\gamma} + 5.5\, D_{\alpha}$$

$$R_{\text{x-linked, lower}} = 0.45\, D_{\beta,\gamma} + 0.45\, D^2_{\beta,\gamma} + 1.1\, D_{\alpha}$$

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, $D_{\beta,\gamma}$ is the gonadal dose (Gy) from beta- and gamma-emitting radionuclides, and D_{α} is the dose from alpha-emitting radionuclides received by a representative individual in the exposed population. An RBE of 2.5 is implicit in the ratio of the β, γ and α risk coefficients.

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% of the risk will be expressed in the first generation, 16% in the second, and so forth. Under the central model an acute dose of 1 Gy of low-LET radiation would yield a first-generation, radiation-induced risk of x-linked disorders of approximately two defects per 1000 births or one 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 $1/5^{th}$ 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% 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% of all cases it will result in a spontaneous loss of pregnancy. In the remaining 10% 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% of live births. Conditions such as Down's syndrome and both Klinefelter and Turner anomalies are the result of aneuploidy. The spontaneous prevalence of aneuploids among live births is about 0.5%. These defects are relatively severe—both in terms of life expectancy (about 45 years) and level of disability (about 50%). 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 did not develop a risk estimate for radiation-induced aneuploidy (NAS/NRC, 1980). Although our Genetic Effects Working Group acknowledges that zero is a reasonable lower estimate, we recommend that one case per 1000 births per Gy be used as a central estimate and believe that an upper estimate of three cases per 1000 births per Gy is plausible. The risk of aneuploidy is assumed to be proportional to the dose received.

Unbalanced translocations, which result in extremely severe physical and mental disabilities, are naturally present in about 0.1% of all children. Infants born 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 of low-LET radiations, the Working Group obtained:

$$r_{\text{translocation induction}} = 15\ D_{\beta,\gamma} + 15\ D^2_{\beta,\gamma}$$

where $D_{\beta,\gamma}$ is the low-LET dose (Gy) to the gonads, and r is the rate (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 one-fourth this large and the fraction carrying unbalanced translocations is one-half this large. Similarly, only one-sixteenth of induced translocations will result in balanced translocations in mature oocytes, and six-sixteenths will result in unbalanced translocations. Thus, the rates of unbalanced translocations among the mature sperm and ova for low-LET radiations are:

$$r_{\text{sperm, unbalanced translocation}} = 7.5\ D_{\beta,\gamma} + 7.5\ D^2_{\beta,\gamma}$$

$$r_{\text{ovum, unbalanced translocation}} = 5.6\ D_{\beta,\gamma} + 5.6\ D^2_{\beta,\gamma}$$

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% 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:

$$R_{\text{child, unbalanced translocation}} = 1.2\ D_{\beta,\gamma} + 1.2\ D^2_{\beta,\gamma} + 18\ D_{\alpha}$$

where R is risk (affected children/1000 livebirths), and $D_{\beta,\gamma}$ is the dose (Gy) to the gonads from beta- or gamma-emitting radionuclides, and D_{α} is the dose from alpha-emitting radionuclides received by the child's parents.

The dynamics of inheritance of unbalanced translocations are such that the risk in the second generation is one-fourth of that in the first and that in each succeeding generation the risk decreases by 50%.

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:

$$R = \{1.2\ D_{\beta,\gamma} + 1.2\ D^2_{\beta,\gamma} + 18\ D_{\alpha}\}\ \{1 + 1/4 + 1/8 + 1/16 + \ldots\}\ \{500/1000\}$$

$$R = 0.9\ D_{\beta,\gamma} + 0.9\ D^2_{\beta,\gamma} + 14\ D_{\alpha}$$

where R is the cumulative risk (number of affected children/1000 exposed people), $D_{\beta,\gamma}$ is the gonadal

dose (Gy) from beta and alpha emitters, and D_α is the dose from alpha emitters received by the population. An RBE of 15 is implicit in the ratio of the β, γ and α risk coefficients.

Upper and lower estimates are derived using this same approach, but using 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, the Working Group recommends using a male gametic induction rate only one-fifth as large as that used in derivation of the central estimate 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 one-eighth 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 to manifest an effect. This largest class of genetic disease includes congenital malformations (e.g., spina bifida, cleft palate), constitutional diseases, and degenerative diseases.

Estimates of the fraction of the population with genetically related multifactorial disease have increased substantially. In 1980, the BEIR III committee estimated that only 9% of the population would be affected by such diseases (NAS/NRC, 1980). Ten years later, the BEIR V committee suggested that all members of the population would, on average, suffer from an average of somewhat more than one multifactorial disorder during their lifetimes (NAS/NRC, 1990). This enormous change reflects the view that the bulk of cardiovascular and neoplastic disease has an inherited component (though its magnitude is currently unknown).

The Genetic Effects Working Group developed separate estimates of the risks of congenital anomalies and three specific categories of irregularly inherited disease: cancers, cardiovascular disease, and "selected other" diseases. Their estimates of the risk of congenital anomalies are based on the BEIR V estimate that exposure of each generation of parents to an additional dose of 1 rem (equivalent to 0.01 Gy of low-LET radiation) would eventually lead to an equilibrium risk of between 10 and 100 additional congenital anomalies per million live births[a]. As their central estimate of risk, the Working Group simply took the geometric mean of the two values given by BEIR V, i.e. $(10 \cdot 100)^{0.5}$ or 32 congenital anomalies per million live births. In a population of 1 million, with a birthrate of 480,000 children per 30-year generation, this estimate corresponds to 15 additional congenital malformations due to this single

[a] It can be demonstrated mathematically that the equilibrium risk in a population exposed chronically to 1 rem per generation is numerically identical to the cumulative risk resulting from exposure of a single generation of parents to 1 rem.

0.01 Gy exposure of one generation of parents. The Working Group adopted the BEIR V range as their lower and upper estimates. The resulting models are:

$$R_{congenital,\ upper} = 4.8\ D_{\beta,\gamma} + 4.8\ D^2_{\beta,\gamma} + 12\ D_{\alpha}$$

$$R_{congenital,\ central} = 1.5\ D_{\beta,\gamma} + 1.5\ D^2_{\beta,\gamma} + 4.0\ D_{\alpha}$$

$$R_{congenital,\ lower} = 0.5\ D_{\beta,\gamma} + 0.5\ D^2_{\beta,\gamma} + 1.25\ D_{\alpha}$$

where R is the cumulative risk (number of children born with congenital malformations per 1000 exposed people), $D_{\beta,\gamma}$ is the gonadal (Gy) dose from beta- and gamma-emitting radionuclides, and D_{α} is the dose from alpha-emitting radionuclides received by the population. An RBE of 2.5 is implicit in the ratio of β, γ, and α risk coefficients.

The number of congenital malformations expected in the first generation was not estimated separately. The Working Group noted that these effects were (implicitly) included in their estimate of first-generation dominant effects.

The typical impact of a congenital malformation was assessed on the basis of information provided in UNSCEAR which suggests that such defects involve an 8-year reduction in life expectancy and approximately 25% impairment. In view of this, the Working Group recommended using 24 (effective) years as an estimate of the loss of life associated with a congenital malformation.

The Genetic Effects Working Group's central estimate of the irregularly inherited cancer risk is based on the assumption that there are between 50 and 100 tumor suppressor genes, each of which (on average) responds to radiation with the same sensitivity that the Selbys (1977) observed in studies involving specific-locus mutations in male mice, i.e., its probability of being mutated by low-LET radiation is $10^{-5}D + 10^{-5}D^2$, and that the majority of individuals who inherit a mutant tumor suppressor gene will develop cancer as a result of the inherited mutation. The resulting model is:

$$R_{cancer} = 19\ D_{\beta,\gamma} + 19\ D^2_{\beta,\gamma} + 48\ D_{\alpha}$$

where R is the cumulative risk (number of cancers/1000 exposed people), $D_{\beta,\gamma}$ is the gonadal dose (Gy) from beta and gamma emitters, and D_{α} is the dose from alpha emitters received by the population. An RBE of 2.5 is implicit in the ratio of the β, γ and α risk coefficients.

The Working Group's central estimate of the fraction of this risk that would occur in the first generation is 5%, based on an assumption that 20 generations will be required to reach genetic equilibrium. Upper and lower bound estimates of 10 and 50 generations, respectively, can be used with this central estimate of 20 generations.

The Working Group notes that the estimate derived in this way is consistent with values derived using a doubling-dose approach. In their alternative calculations, they assume that the background (lifetime) cancer risk faced by an individual is 30%; that 5-10% of such cancers have a hereditary component; that the mutational component of this class of hereditary cancers is high (85% or more), that the dose required to double the incidence of such heritable cancers is 1 Gy, and that the time to equilibrium is between 10 and 50 generations.

Even less is known about the genetic mechanisms underlying cardiovascular disease and other diseases. The Genetic Effects Working Group based their central estimates of cardiovascular risks on a doubling dose approach—assuming that the background (lifetime) cardiovascular disorder rate is 60%; that 13% of cardiovascular diseases have a genetic component; and that a dose of 1 Gy delivered acutely would double this risk. For chronic exposure, the doubling dose would be 0.6 Gy. Estimates of the risks of "selected other" diseases of complex etiology were derived similarly; however, a background rate of 30% was used. The resulting models are:

$$R_{\text{cardiovascular}} = 38\, D_{\beta,\gamma} + 38\, D^2_{\beta,\gamma} + 95\, D_{\alpha}$$

$$R_{\text{selected other}} = 19\, D_{\beta,\gamma} + 19\, D^2_{\beta,\gamma} + 48\, D_{\alpha}$$

where R is the cumulative risk (number of cardiovascular disorders/1000 exposed people), $D_{\beta,\gamma}$ is the gonadal dose (Gy) from beta and gamma emitters, and D_{α} is the dose from alpha emitters received by the population. To obtain estimates of first generation risks, the Working Group assumed 20 generations would be required to reach genetic equilibrium.

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 four 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] 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 two cases per 1000 births, is:

$$R_{\text{recessive}} = 2\, D_{\beta,\gamma} + 2\, D^2_{\beta,\gamma}$$

[a] 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."

where R is the equilibrium risk (number of affected children/1000 births), and $D_{\beta,\gamma}$ is the low-LET gonadal dose (Gy) received.

It should be noted that recessive risks are expressed very slowly, their mean persistence 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 2.10 and 2.11 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.

Table 2.10

Models of genetic risks[a,b]

Effect	Integrated risk (cases/1000)		
	Upper	Central	Lower
Single gene			
Dominant	$22\ D_{\beta,\gamma} + 22\ D^2_{\beta,\gamma} + 55\ D_\alpha$	$7.5\ D_{\beta,\gamma} + 7.5\ D^2_{\beta,\gamma} + 19\ D_\alpha$	$1.2\ D_{\beta,\gamma} + 1.2\ D^2_{\beta,\gamma} + 3.0\ D_\alpha$
X-linked	$9\ D_{\beta,\gamma} + 9\ D^2_{\beta,\gamma} + 23\ D_\alpha$	$2.2\ D_{\beta,\gamma} + 2.2\ D^2_{\beta,\gamma} + 5.5\ D_\alpha$	$0.45\ D_{\beta,\gamma} + 0.45\ D^2_{\beta,\gamma} + 1.1\ D_\alpha$
Chromosome aberrations[c]			
Numerical	$1.5\ D_{\beta,\gamma} + 3.8\ D_\alpha$	$0.5\ D_{\beta,\gamma} + 1.3\ D_\alpha$	0
Structural	$6.3\ D_{\beta,\gamma} + 6.3\ D^2_{\beta,\gamma} + 100\ D_\alpha$	$0.9\ D_{\beta,\gamma} + 0.9\ D^2_{\beta,\gamma} + 14\ D_\alpha$	$0.1\ D_{\beta,\gamma} + 0.1\ D^2_{\beta,\gamma} + 1.5\ D_\alpha$
Multifactorial diseases			
Congenital anomalies	$4.8\ D_{\beta,\gamma} + 4.8\ D^2_{\beta,\gamma} + 12\ D_\alpha$	$1.5\ D_{\beta,\gamma} + 1.5\ D^2_{\beta,\gamma} + 4.0\ D_\alpha$	$0.5\ D_{\beta,\gamma} + 0.5\ D^2_{\beta,\gamma} + 1.3\ D_\alpha$
Cardiovascular[d]	$380\ D_{\beta,\gamma} + 380\ D^2_{\beta,\gamma} + 950\ D_\alpha$	$38\ D_{\beta,\gamma} + 38\ D^2_{\beta,\gamma} + 95\ D_\alpha$	$3.8\ D_{\beta,\gamma} + 3.8\ D^2_{\beta,\gamma} + 9.5\ D_\alpha$
Cancer[d]	$190\ D_{\beta,\gamma} + 190\ D^2_{\beta,\gamma} + 480\ D_\alpha$	$19\ D_{\beta,\gamma} + 19\ D^2_{\beta,\gamma} + 48\ D_\alpha$	$1.9\ D_{\beta,\gamma} + 1.9\ D^2_{\beta,\gamma} + 4.8\ D_\alpha$
Selected other[d]	$190\ D_{\beta,\gamma} + 190\ D^2_{\beta,\gamma} + 480\ D_\alpha$	$19\ D_{\beta,\gamma} + 19\ D^2_{\beta,\gamma} + 48\ D_\alpha$	$1.9\ D_{\beta,\gamma} + 1.9\ D^2_{\beta,\gamma} + 4.8\ D_\alpha$
Losses of pregnancy[c]			
Numerical	$14\ D_{\beta,\gamma} + 870\ D_\alpha$	$4.5\ D_{\beta,\gamma} + 11\ D_\alpha$	0
Structural	$58\ D_{\beta,\gamma} + 58\ D^2_{\beta,\gamma} + 870\ D_\alpha$	$8.1\ D_{\beta,\gamma} + 8.1\ D^2_{\beta,\gamma} + 120\ D_\alpha$	$0.9\ D_{\beta,\gamma} + 0.9\ D^2_{\beta,\gamma} + 14\ D_\alpha$

[a] The doses, $D_{\beta,\gamma}$ and D_α, referred to in this table are the low-LET and alpha 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.

[b] No formal model of risk of recessive disease was developed, but the Working Group provided some information suggesting the possible magnitude of these risks (see Section 2.3.4).

[c] Chromosomal defects may lead to early foetal losses, early miscarriages or to children born with severe physical and mental defects. Most early foetal losses occur as a result of failure of the fertilized egg to implant in the uterine wall.

[d] Recognizing that our current knowledge on the inherited component of multifactorial diseases and the impact of radiation exposure on this component is extremely limited, these estimates of possible upper and lower bounds are also extremely tenuous at this time. Factors of 10 were used for roughly estimating the upper and lower bounds of these poorly defined risks.

Table 2.11

Time distribution of genetic risks[a]

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[b]	unknown					
Miscarriages						
Numerical	100	-	-	-	-	-
Structural	67	17	8	4	2	2

[a] Entries in the body of the table give the percentage of the cumulative genetic risk (see Table 2.10) expected in each time interval.

[b] The timing of congenital anomalies is uncertain. Using a central estimate of 20 generations to equilibrium, about 5% of the total impact is expected in each of the first several generations. However, the equilibrium time could be as low as 10 generations or as high as 50 generations.

3.0 COMPUTATIONAL ASPECTS

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

3.1 Early and Continuing Effects

The structure of the nuclear power plant accident consequence code MACCS (NRC, 1990b) is based on the health effects models recommended in the first edition of NUREG/CR-4214 (NRC, 1985). 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, the size of arrays and matrices used to store results are restricted, which limits the number of effects that can be considered.

Below, some approaches are outlined for implementing the 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, dose rate, and 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, 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 (NRC, 1975). Dr. Niel Wald (personal communication, 1989), one of the authors of the Reactor Safety Study (also called WASH-1400), 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..."

Dr. 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 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 the medical treatment is randomly distributed.

Ideally one would use a model that 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%.

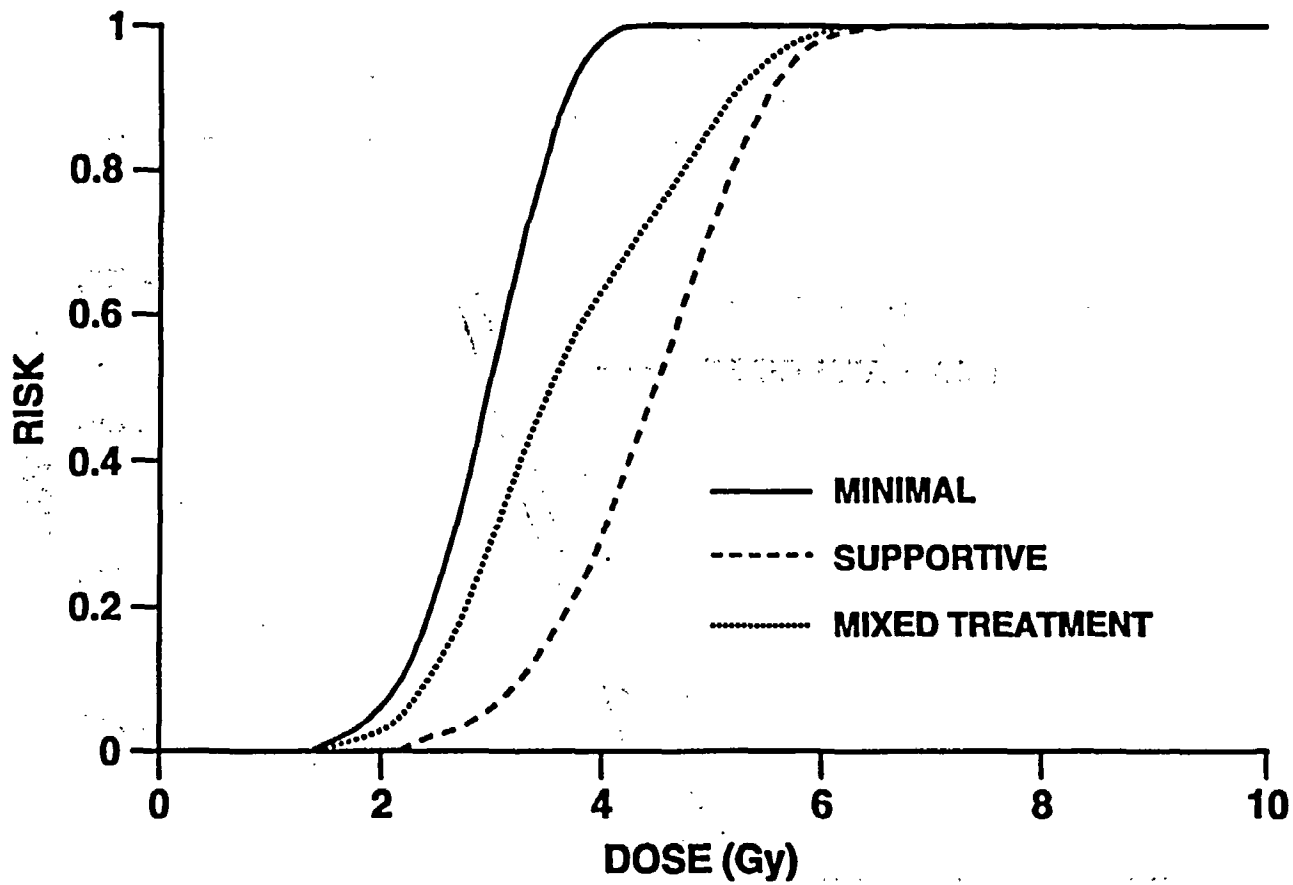


Figure 3.1 Risks of mortality from the hematopoietic syndrome for minimal, supportive, and mixed treatments: central estimates for exposure at a high dose rate.

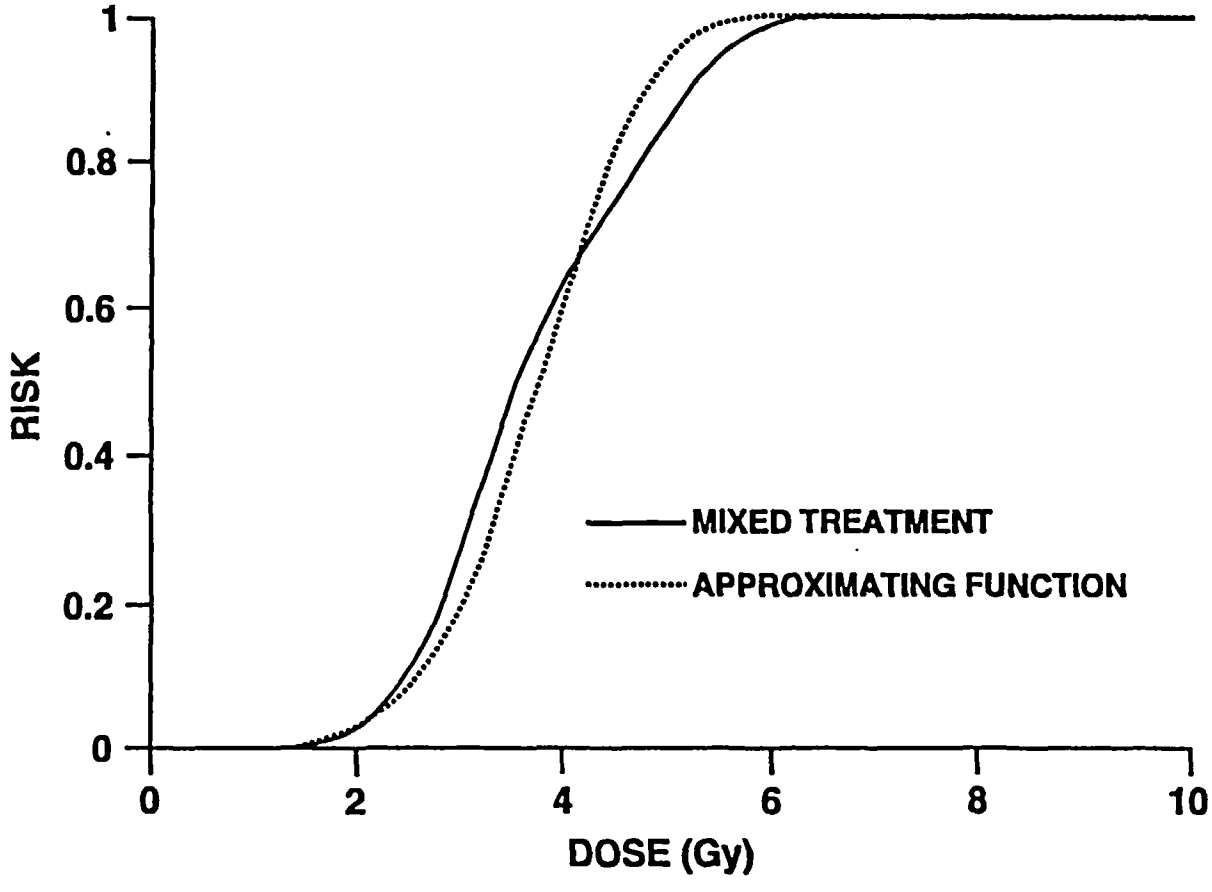


Figure 3.2 Comparison of the mixed-treatment model and an approximating function for estimating mortality risks from the hematopoietic syndrome: central estimates for exposure at high dose rate.

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 (Ritchie, 1983), 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:

$$D = D_{external} + D_{internal,\ day\ 0\text{-}1} + 1/2\ D_{internal,\ day\ 2\text{-}14} + 1/4\ D_{internal,\ day\ 15\text{-}30}$$

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 (NRC, 1990b).

The dose-rate-dependent models developed by Scott *et al.* (1988, 1989) and endorsed by the Early Effects Working Group allow one to express the median lethal dose, LD_{50} (Gy), as a function of adjusted dose rate, $\dot{D}$ (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_∞ (Gy)	θ_1 (Gy^2/hr)
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% of its limiting value.

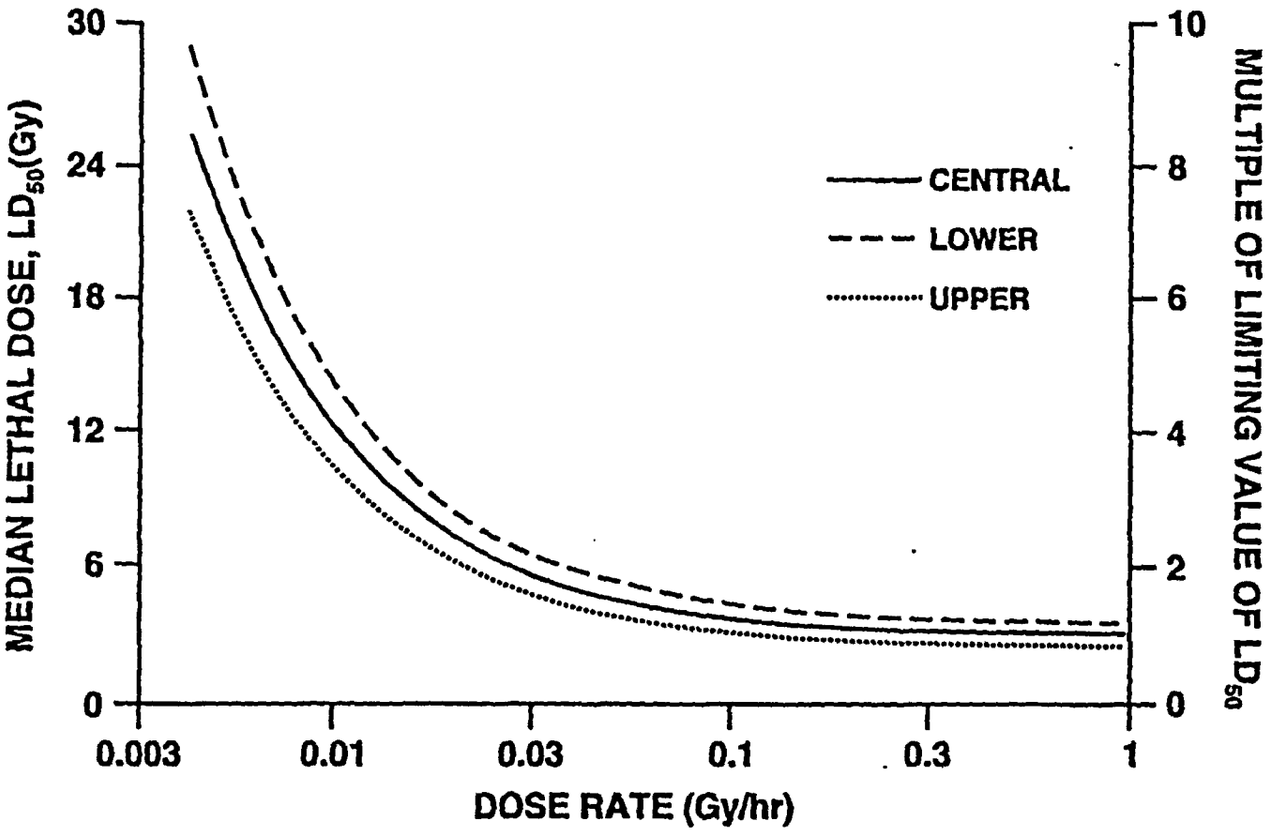


Figure 3.3 Dependence of median lethal dose on adjusted dose rate: mortality from the hematopoietic syndrome.

Both CRAC and MACCS use a fixed-time interval approach for computing the risks of pulmonary syndrome. An adjusted lung dose, D, is derived using dose-rate effectiveness factors:

$$D = D_{external} + D_{internal,\ 0\text{-}1\ day} + 1/16\ D_{internal,\ 2\text{-}14\ days}$$

$$+ 1/37\ D_{internal,\ 15\text{-}200\ days} + 1/92\ D_{internal,\ 201\text{-}365\ days}$$

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 *et al.*, 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 healthy young adults are:

Estimate	θ_∞ (Gy)	θ_1 (Gy^2/hr)
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 (internal and external) pulmonary 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% and 99%, 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:

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

$$H = 0.693 \left[\int_0^\infty \frac{\dot{D}}{\theta_\infty + \theta_1 / \dot{D}}\, dt \right]^V$$

$$\dot{D} = \dot{D}_0\, e^{-0.693 (t/t_{1/2})}$$

for several levels of initial dose rate, $\dot{D}_0$, 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

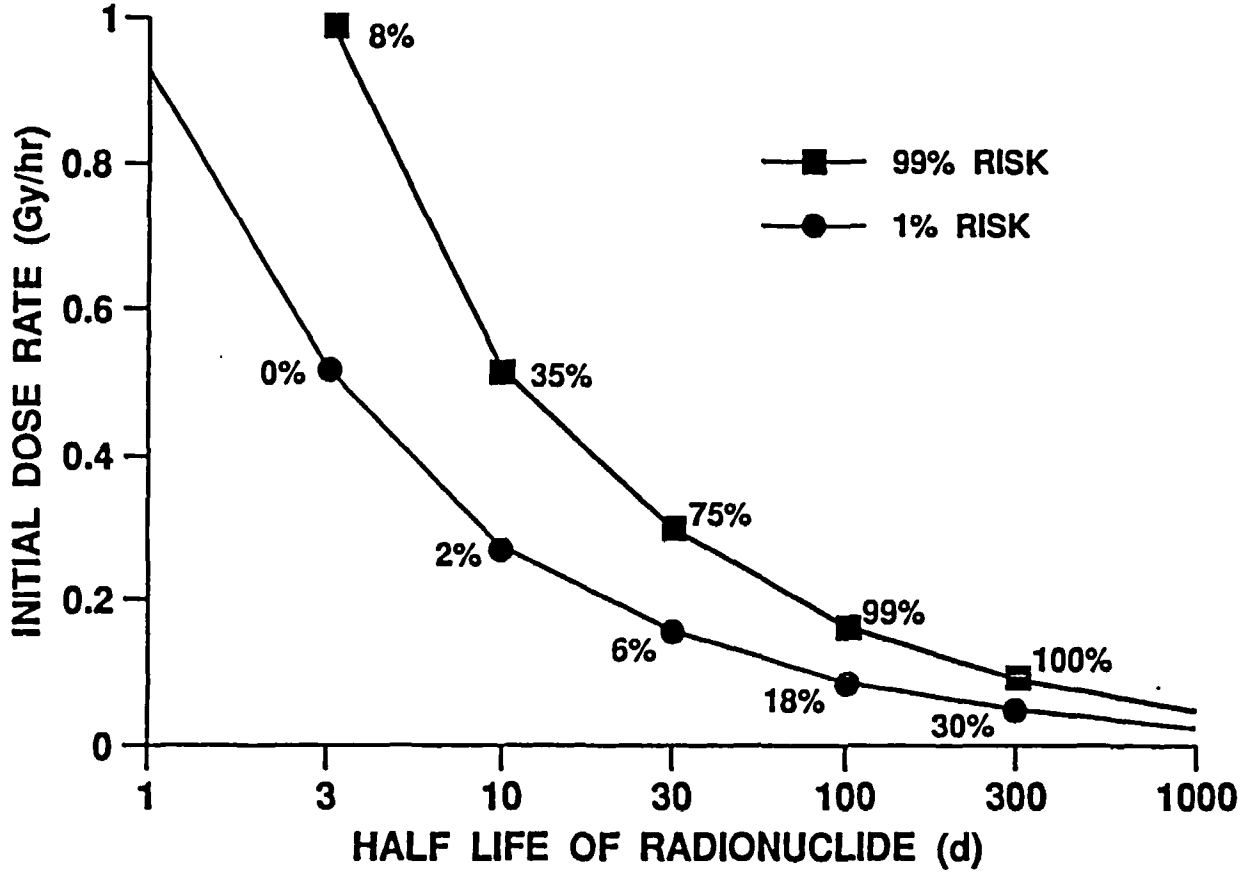


Figure 3.4 Comparison of mortality risk estimates for the pulmonary syndrome: dose-rate dependent model (central estimates) vs. fixed time interval model. Values for fixed-time-interval method are given as numbers next to the curves generated from the dose-rate dependent model.

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, and 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 ^{106}Ru, a relatively insoluble radionuclide (clearance class Y), with a radiological half-life of 366 days.[a] 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; 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.

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.[b] 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.

[a] The calculations upon which Figure 3.5 is based assume that 40% of the ^{106}Ru is cleared from the lung with an effective half-life on the order of 1 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 (NRC, 1990b).

[b] The calculations upon which Figure 3.6 is based assume that 99.5% of the ^{131}I is cleared from the lung with an effective half-life on the order of 1 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 (NRC, 1990b).

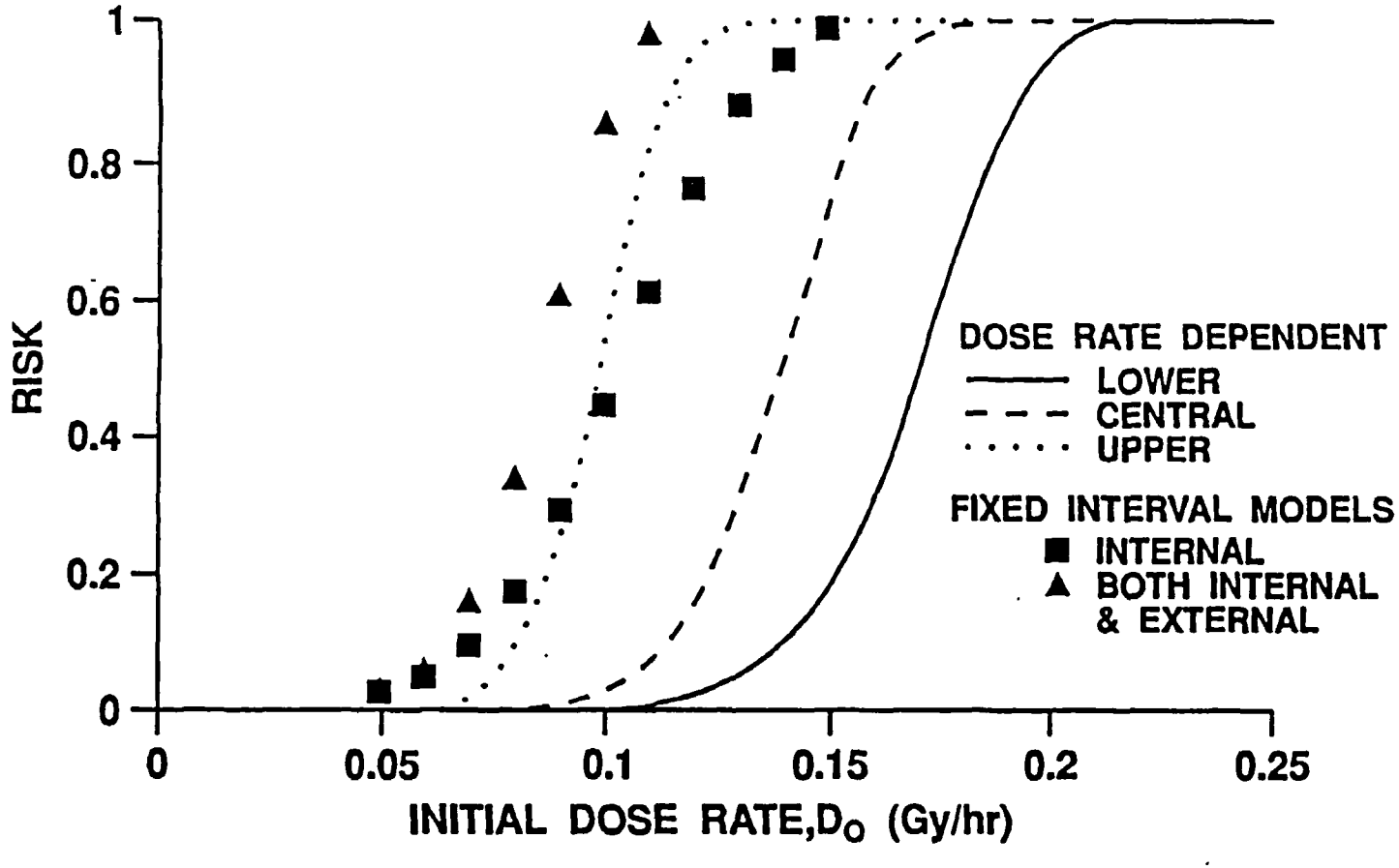


Figure 3.5 Example of the risk of mortality from the pulmonary syndrome after an acute inhalation exposure to ^{106}Ru.

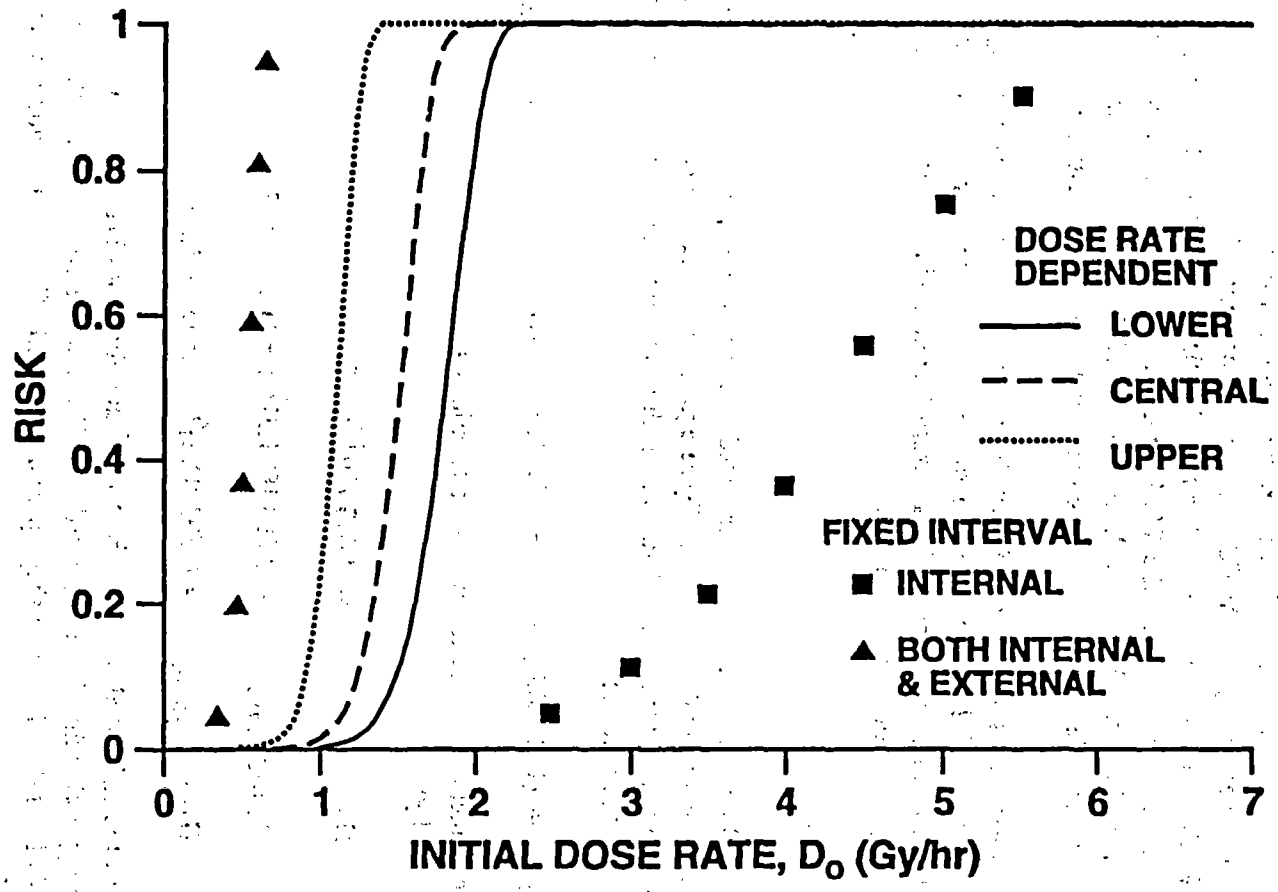


Figure 3.6 Example of the risk of mortality from the pulmonary syndrome after an acute inhalation exposure to ^{131}I.

3.1.3 Hypothyroidism

The Thyroid Effects Working Group recommended that the risk of hypothyroidism, following internal exposure to ^{131}I, be estimated using a linear-threshold model with a threshold of 10 Gy and a slope of 17×10^{-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%.

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 85×10^{-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

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:

$$R_{\text{typical fetus}} = (18/280)R_{\text{0-18 days}} + (132/280)R_{\text{18-150 days}} + (130/280)R_{\text{150 days-term}},$$

where the hazard functions associated with risk functions $R_{\text{0-18 days}}$, $R_{\text{18-150 days}}$, and $R_{\text{150-term}}$ are given, respectively, by:

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

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

$$H_{\text{150 days-term}} = 0.693\,[D/3]^6 \quad \text{for } D > 1.5 \text{ Gy}.$$

Figure 3.8 shows the risk faced by a representative fetus for doses between 0 to 5 Gy. Also shown in the figure is an approximating function, based on a Weibull hazard 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.

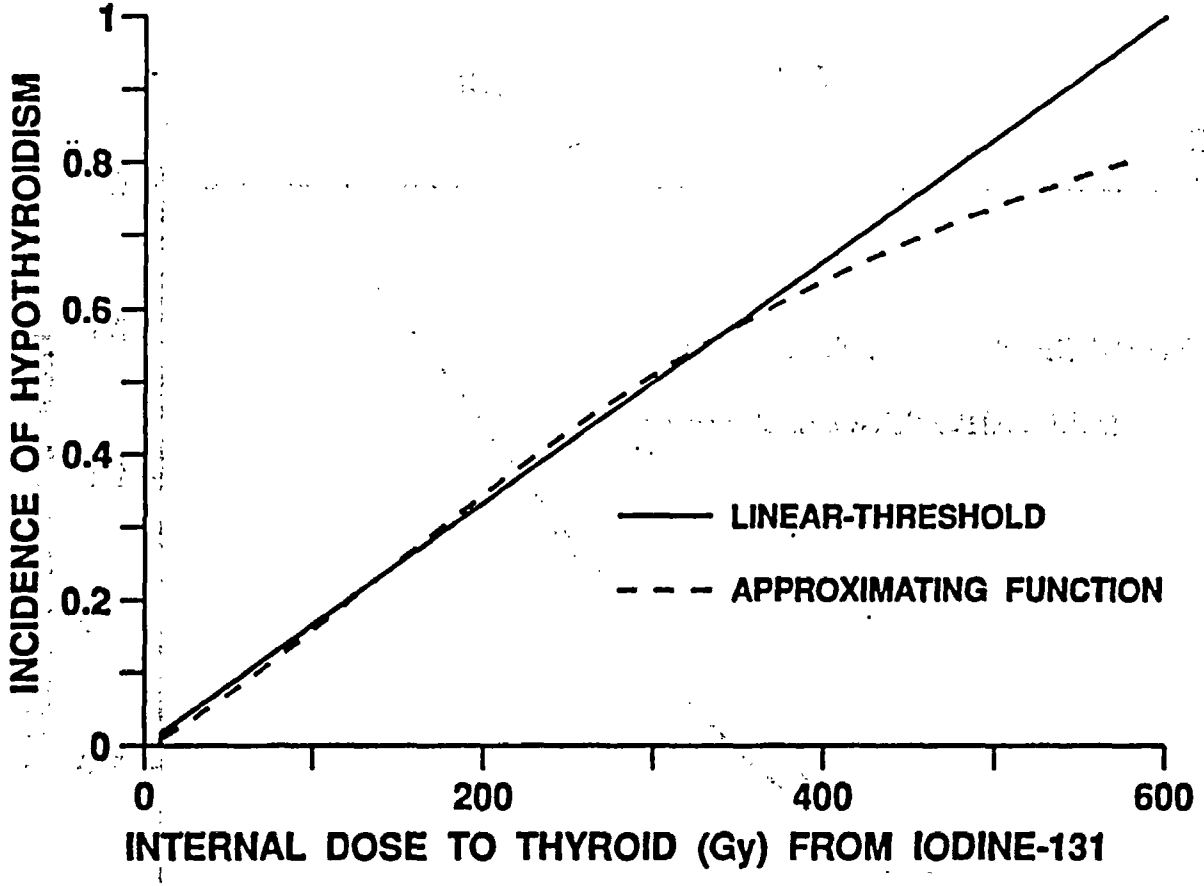


Figure 3.7 Approximation of linear threshold model with hazard function for dose-response analysis after an acute exposure to ^{131}I.

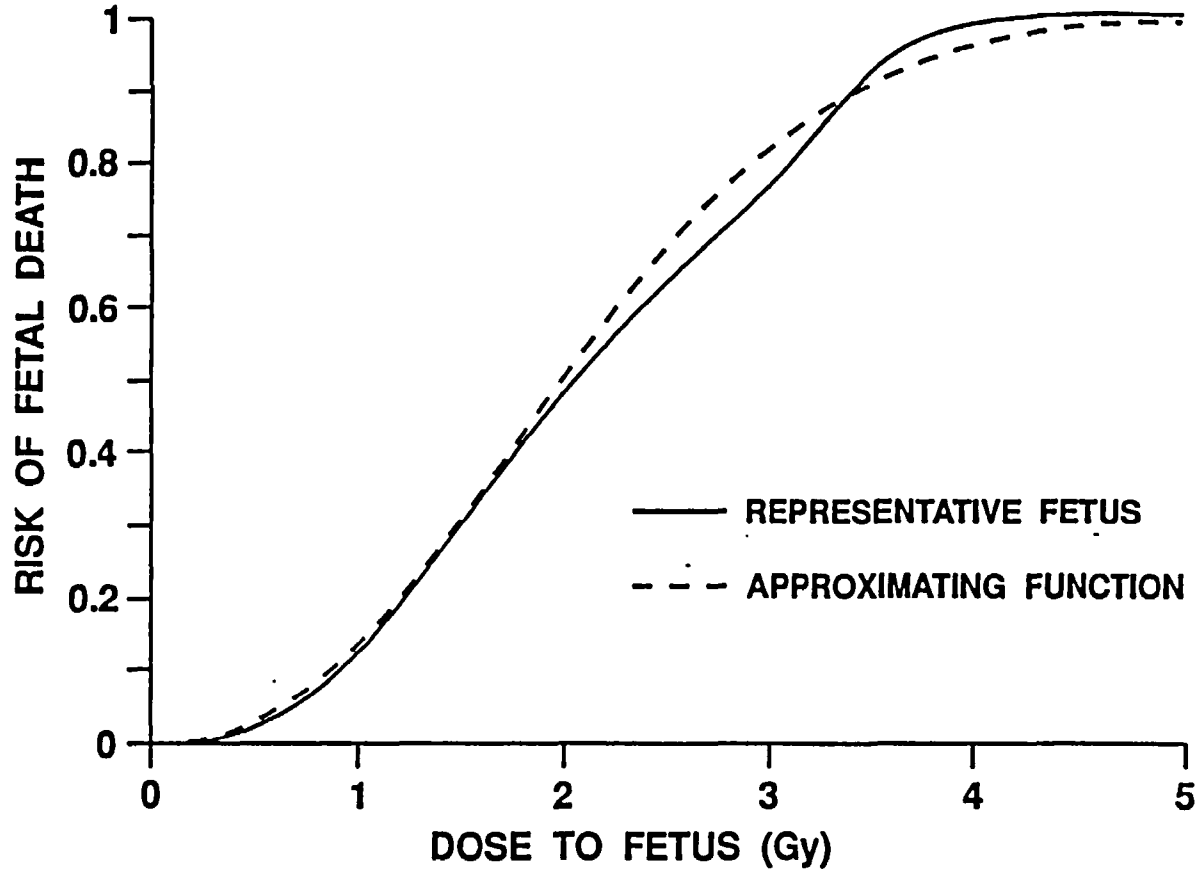


Figure 3.8 Comparison of the mixed developmental age model and an approximating hazard function for estimating the risk of death for a representative fetus after exposure to ionizing radiation.

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 Group 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:

$$H_{8\text{-}15\ \text{weeks}} = 0.693[D/1.5] \text{ for } D \geq 0.1$$

$$H_{16\text{-}25\ \text{weeks}} = 0.693[D/7.0] \text{ for } D \geq 0.2$$

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:

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

where $R_{8\text{-}15\ \text{weeks}}$ and $R_{16\text{-}25\ \text{weeks}}$ 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 fetus is well approximated by the expression:

$$R_{\text{typical fetus}} = 0.4[1\text{-}\exp[-0.693(D/2.5)]] \text{ for } D > 0.1 \text{ Gy},$$

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.

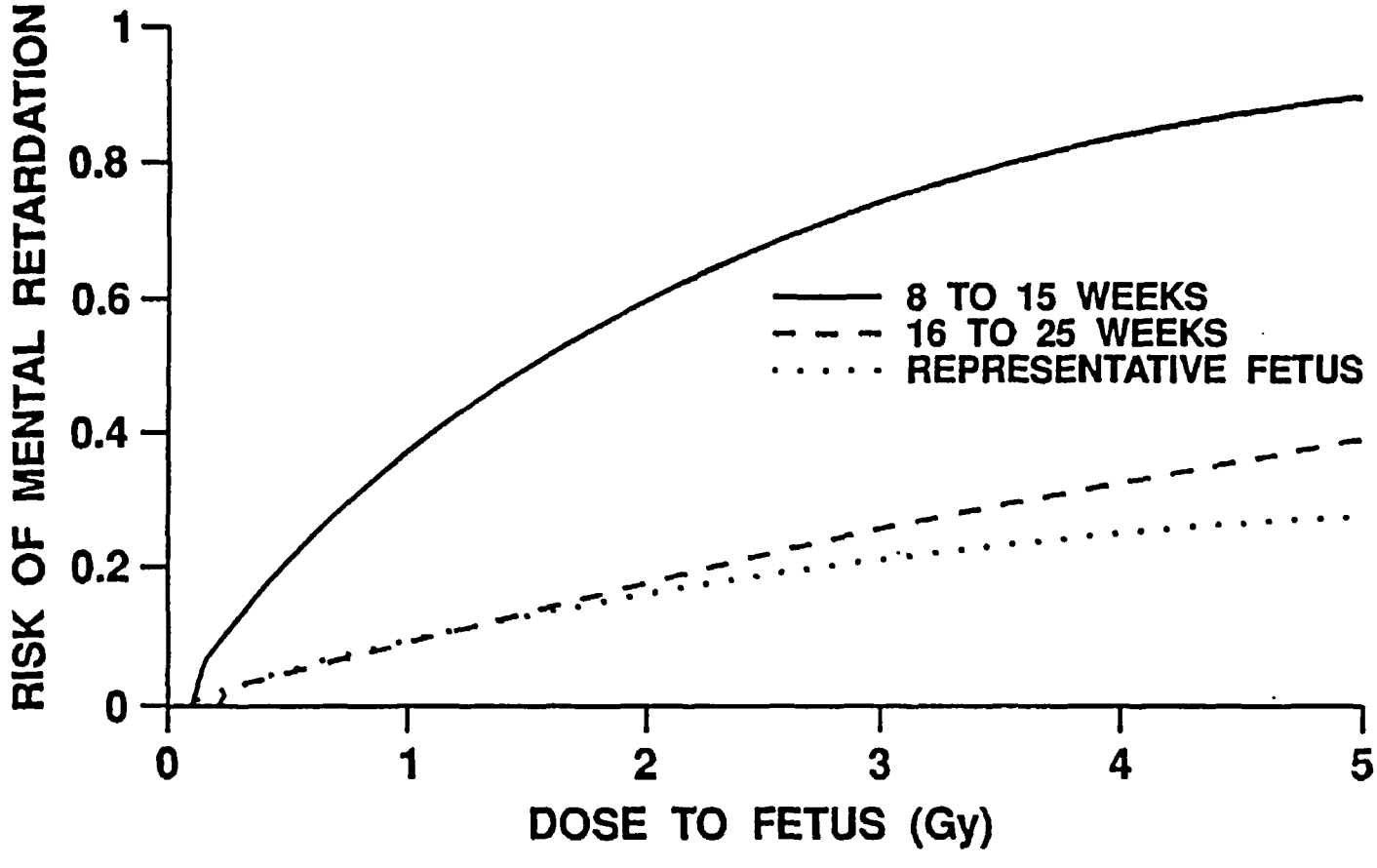


Figure 3.9 Risk of mental retardation after an exposure to ionizing radiation at different gestational ages.

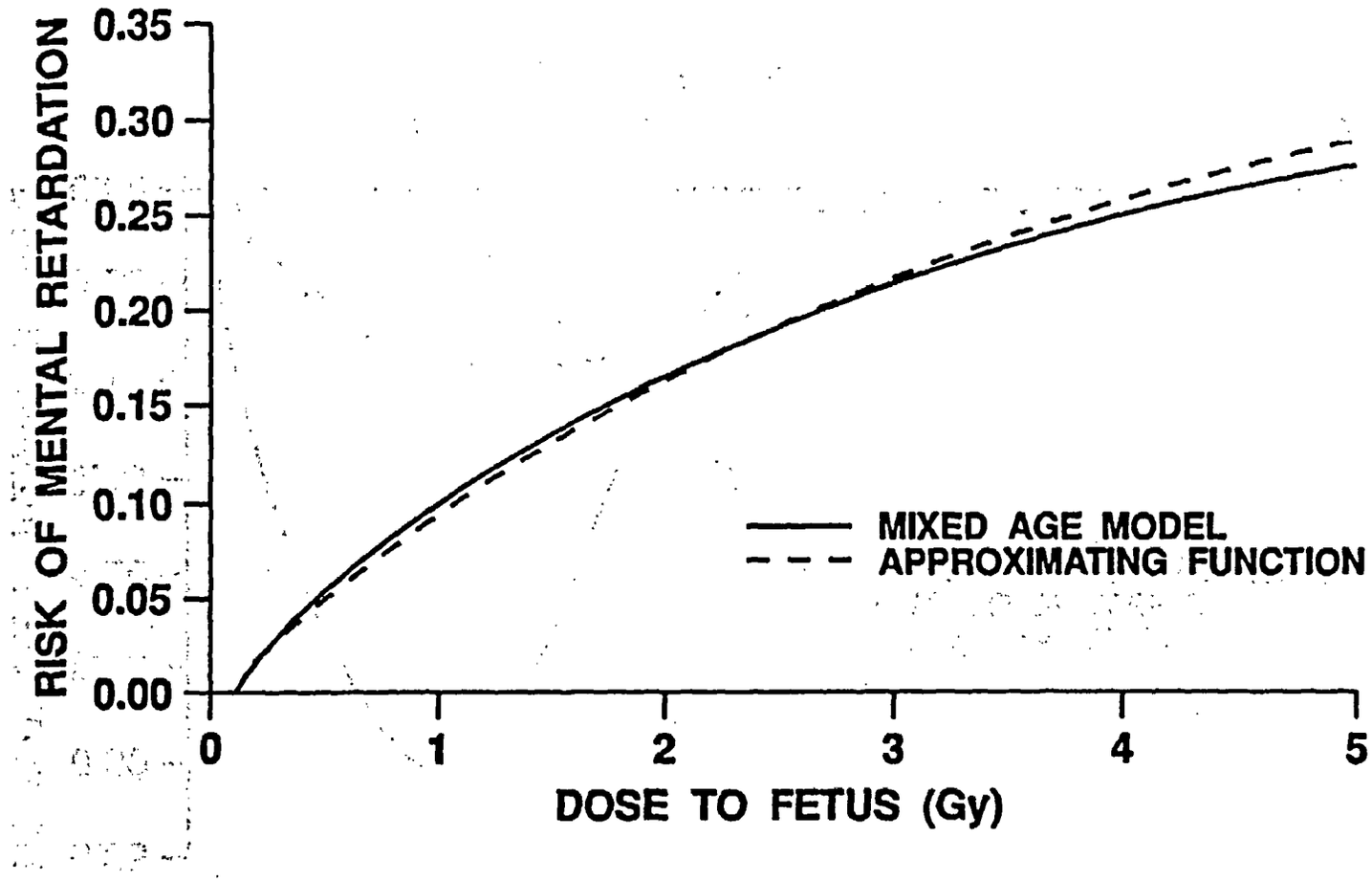


Figure 3.10 Comparison of the mixed gestational-age model and an approximating function for describing the risk of mental retardation after exposure of the fetus to ionizing radiation.

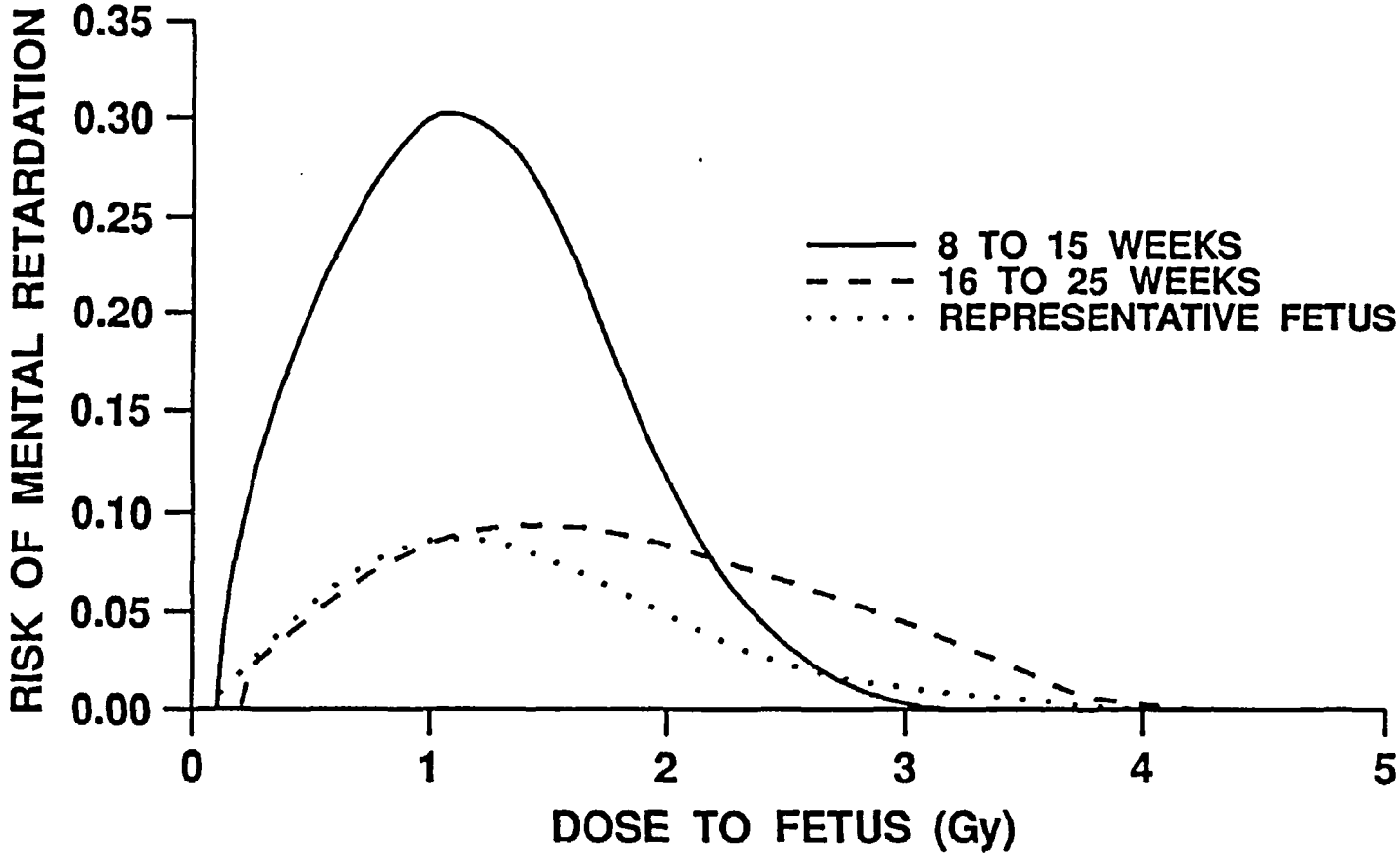


Figure 3.11 Risk of mental retardation as a function of dose and gestational period in which dose to fetus is received after accounting for fetal deaths.

3.1.6 Form of Dose-Response Model

The risk 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, but the probit or logistic also would have been satisfactory. The probit model is:

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

where μ is the dose at which 50% incidence is expected, i.e., the D_{50}, 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:

$$R = \frac{1}{1 + e^{-\eta-\theta D}}$$

where η is a location parameter, related to background incidence, and Θ is a shape parameter. Large values of Θ indicate homogeneity of response and steep dose-response functions. In the logistic model the median lethal (or effective) dose is $-\eta/\Theta$.

With appropriate parameters, all three models yield essentially identical estimates of risk in the region of experimental data, i.e., $0.1 < R < 0.9$. Outside of this region, they may diverge considerably. This point is illustrated in Figures 3.12 and 3.13[a]. Figure 3.12 shows the Early Effects Working Group's central estimate of hematopoietic syndrome mortality risk, for 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% while the probit and logistic models give estimates of 0.7% and 1.4%, respectively. In principle, these differences, which are small in absolute terms—could lead to significant differences in estimated risks for accident scenarios that 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 risks of early effects.

3.2 Late Somatic Effects

For nuclear power plant consequence analysis, it is necessary to estimate 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 estimate 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

[a] In both figures, risks are shown below the threshold doses recommended by the Early Effects Working Group.

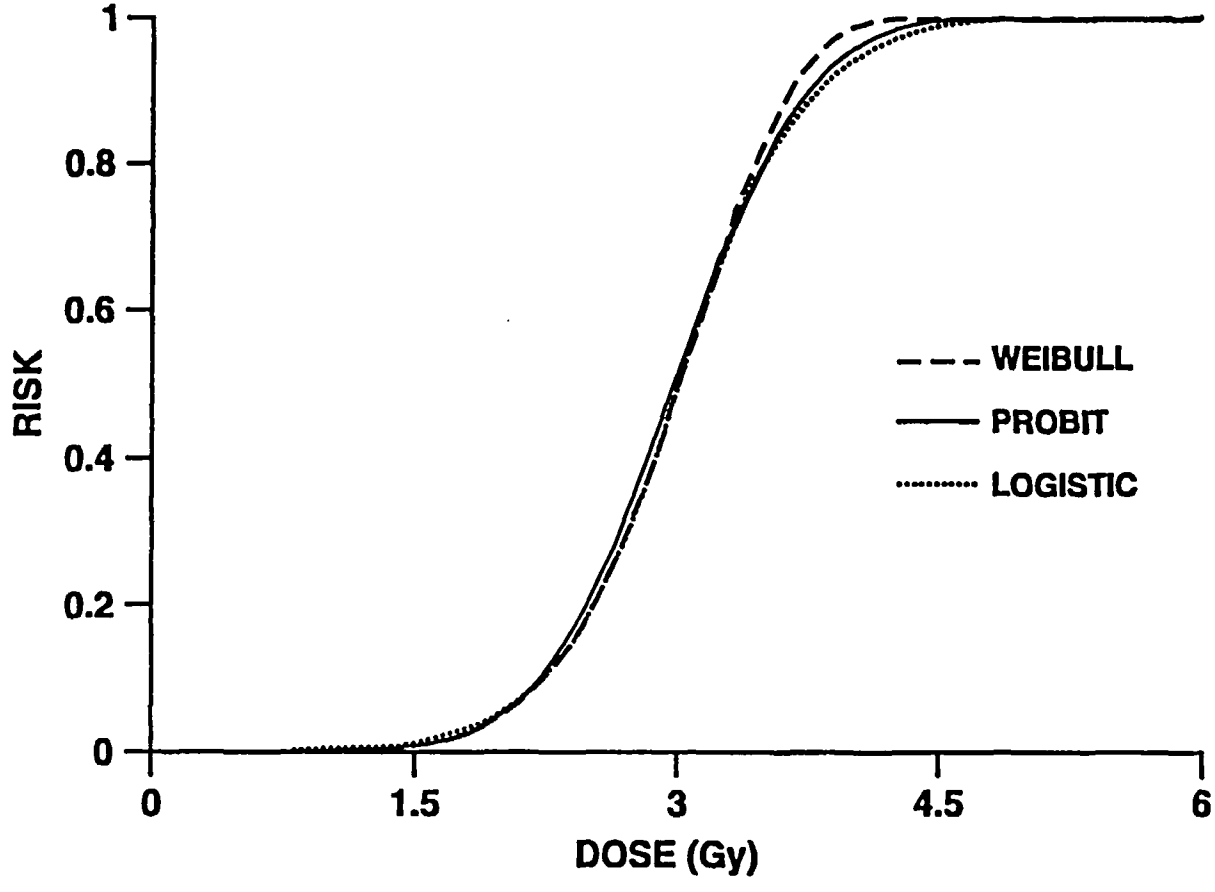


Figure 3.12 Comparison of Weibull, probit, and logistic models for estimating hematopoietic syndrome mortality in individuals receiving minimal medical treatment.

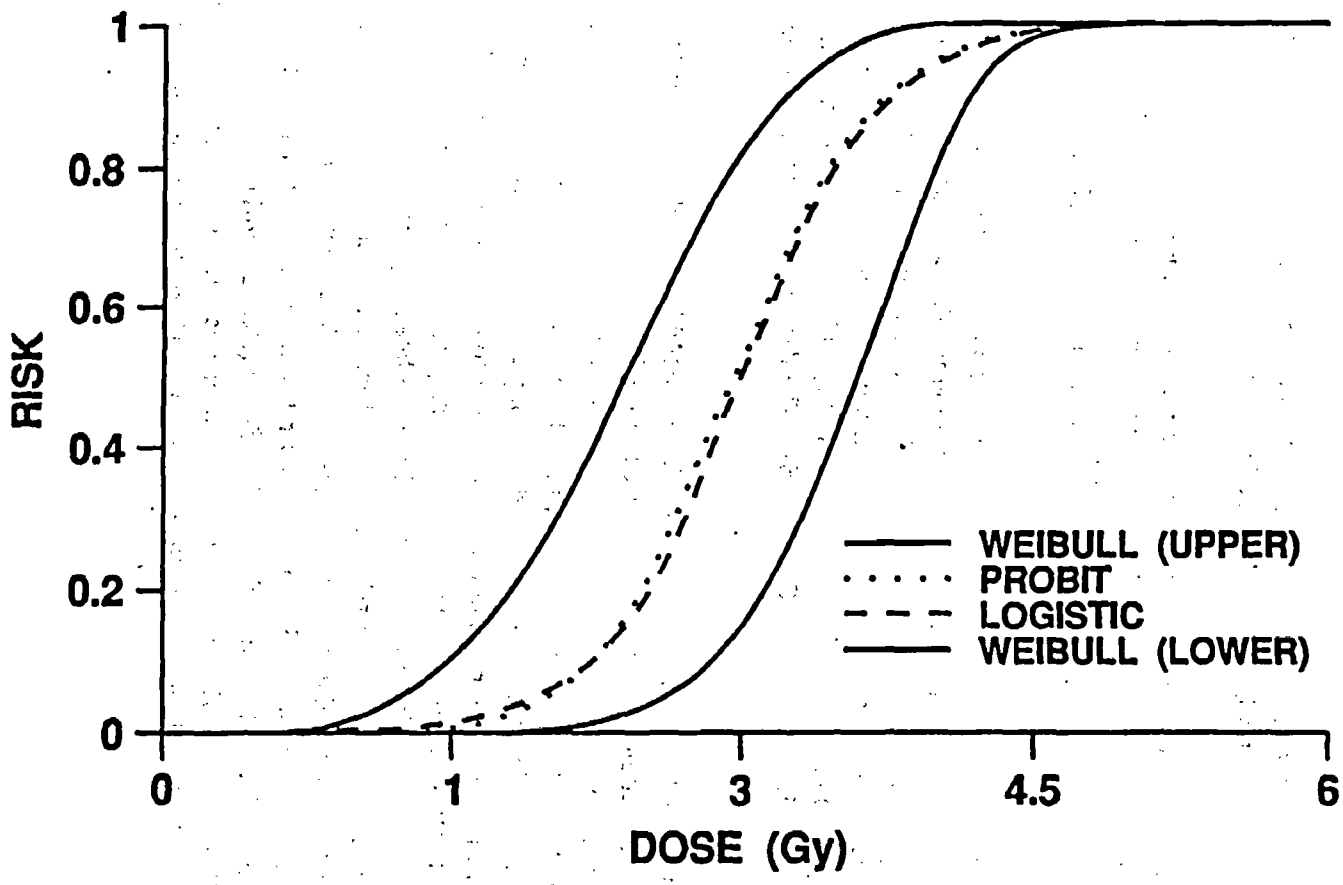


Figure 3.13 Model uncertainty in perspective—probit and logistic central estimates contrasted with upper and lower Weibull models for mortality from the hematopoietic syndrome in individuals receiving minimal medical treatment.

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 τ years after receiving a dose D is:

$$R(\tau,D) = \sum_k f_k \, s_k(\tau) \, r_k(\tau,D)$$

where k is an index of age at exposure, f_k is the fraction of the population in the k^{th} age at exposure group, $s_k(\tau)$ is the fraction of the k^{th} age at exposure group that will survive all other causes of death for τ years, and $r_k(\tau,d)$ is the risk that will be experienced by individuals in the k^{th} age at exposure group τ years after receiving a dose D.[a] 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 $s_k(\tau)$ 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 $r_k(\tau,D)$ 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 or fraction of the spontaneous age-specific risk, expected during each year of the expression period following a 1 Gy dose. The background rates of cancer mortality 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) except for the values for skin cancer which came from Scotto *et al.* (1974, 1983) and Fears and Scotto (1982). These baseline cancer rates are provided in Appendix A.

---

[a] Theoretically this approach—which does not adjust the survival probabilities, $s_k(\tau)$, to reflect radiation-induced deaths—could lead to overestimation of risk at high dose. However, in most accident scenarios the most 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.

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:

$$R(D) = \sum_{\tau} R(\tau,D)$$

This approach is the one used to obtain the estimates 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 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 with 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% of the exposed population would survive 20 years; 65% would survive 40 years; 40% would survive 60 years; and about 15% 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 $R(\tau,D)$ 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.

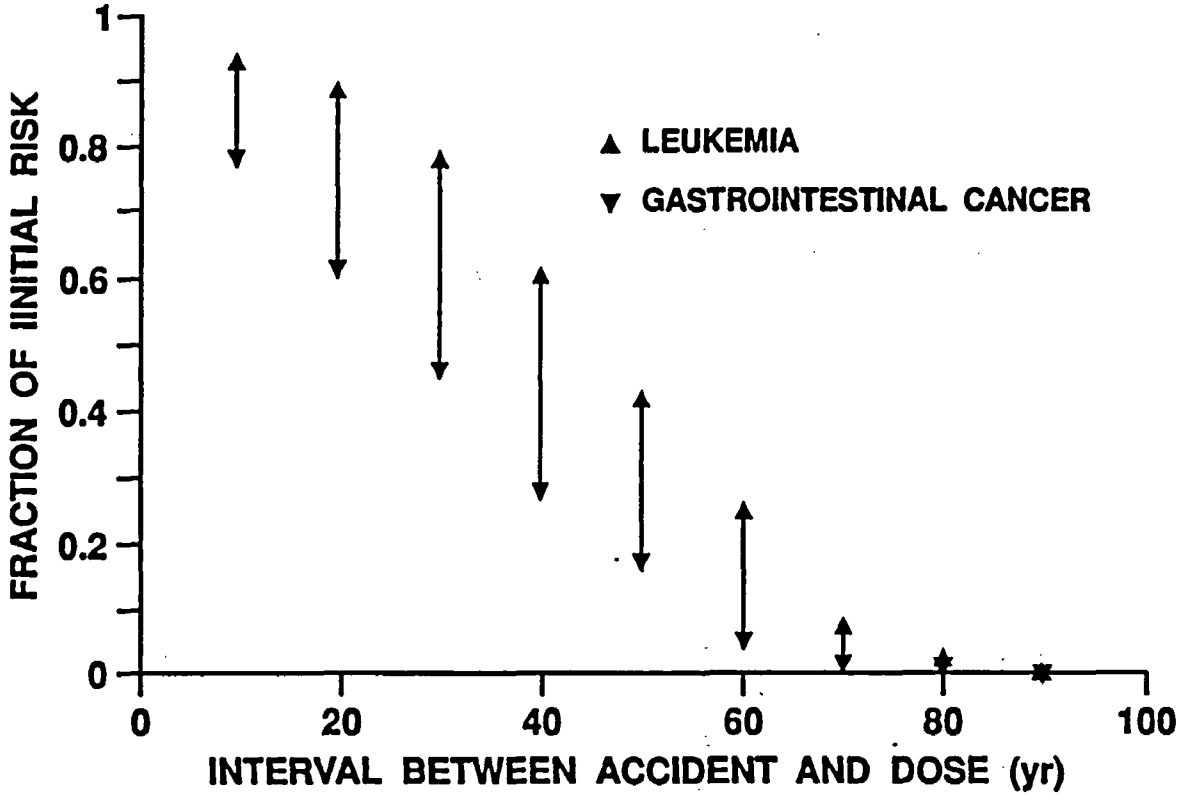


Figure 3.14 Risk among those inhaling radionuclides as a function of time at which dose is received.

The degree of overestimation that would occur if risk was calculated 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 key factor 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 (BOC, 1983). Figure 3.15 shows the distribution of births by age of the mother.

To estimate the number of children born with genetic defects in the first generation after the accident, the total number of births expected is multiplied by the risk that a child will suffer from genetic disease. In a 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 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 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[a]. In a stable population the number of children born with radiation-induced genetic defects in the second, third, or k^{th} generation would be:

$$N_2 = N_1\ T$$

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

$$\vdots$$

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

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:

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

[a] The 0.5 in this equation represents a rounding of the 480,000 births per million population per 30 year in the generational interval.

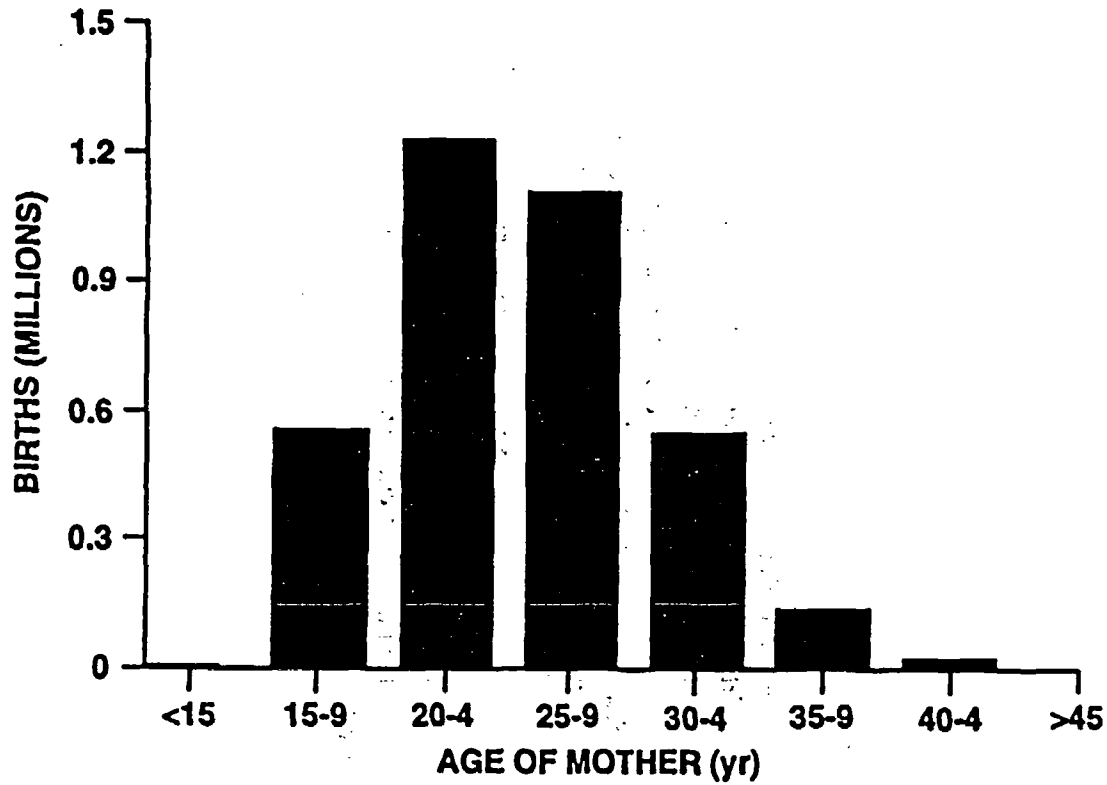


Figure 3.15 Distribution of live births in the United States in 1980 as a function of the age of the mother.

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 one-fourth of the risk faced by the first generation, but in each succeeding generations, the risk diminishes by 50%.

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% per year, the growth rate since 1900 has been more moderate, i.e., about 1.4% 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 was due to immigration. Currently, the immigration component of population growth is only about 0.2% per year.

To apply this model of genetic impact, it is necessary to derive the 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 found 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, and $r_p(D_t)$ is the paternal gametic induction function based on the dose to the testis. 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 an accident at a nuclear power plant, there could be a wide distribution of gonadal doses among the pool of prospective parents. If all of the individual doses were received at low dose rate, 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.

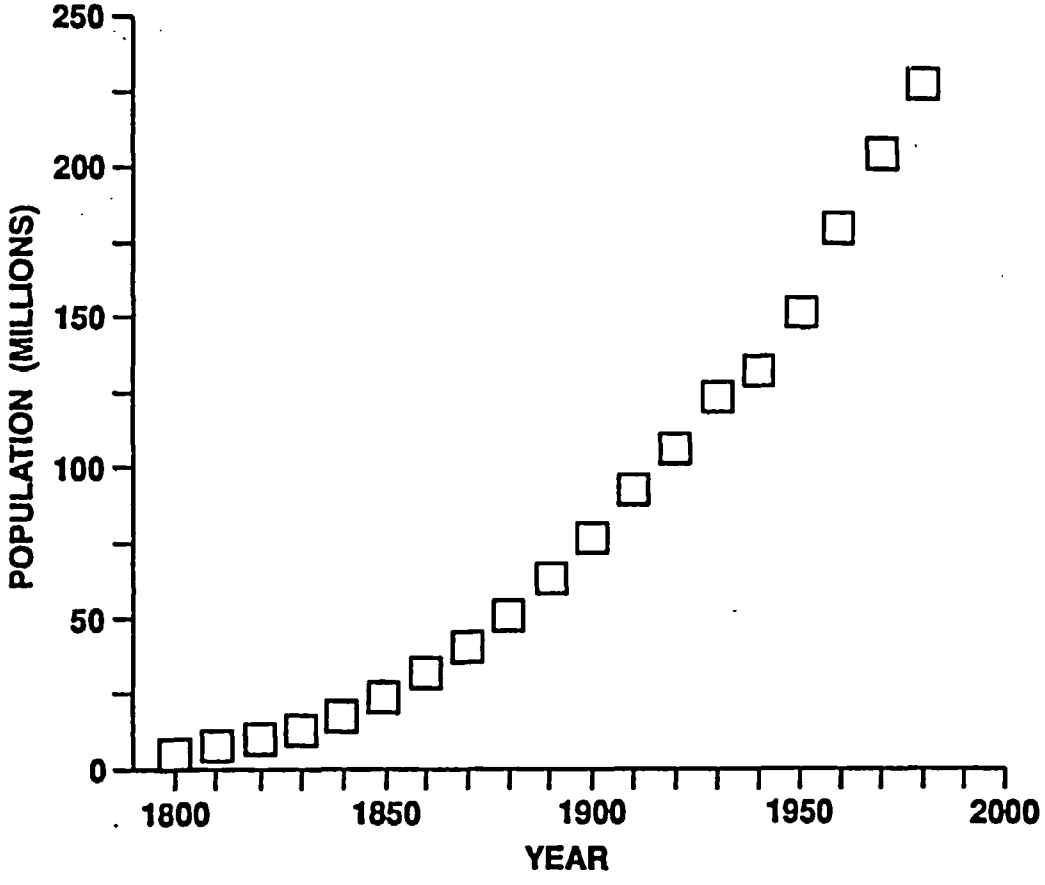


Figure 3.16 Population of the United States, 1800-2000.

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 children with defects per 1000 children born in the k^{th} generation. Alternatively, an estimate of prevalence may be combined 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[a]

Age	Both	Male	Female	Age	Both	Male	Female
0	3534	1806	1727	25	4116	2053	2064
1	3270	1674	1595	6	3978	1979	1999
2	3224	1648	1576	7	3932	1952	1980
3	3179	1626	1554	8	3709	1840	1869
4	3142	1608	1534	9	3787	1881	1905
5	3163	1618	1544	30	3727	1847	1880
6	3109	1589	1520	1	3608	1781	1826
7	3273	1673	1600	2	3712	1833	1879
8	3395	1736	1659	3	3654	1805	1849
9	3760	1923	1837	4	2861	1411	1449
10	3717	1902	1815	35	2902	1430	1472
1	3581	1829	1752	6	2929	1439	1490
2	3519	1796	1723	7	2983	1465	1518
3	3643	1857	1787	8	2599	1273	1326
4	3783	1933	1850	9	2553	1254	1298
15	4060	2070	1990	40	2468	1209	1259
6	4181	2135	2046	1	2376	1164	1212
7	4224	2160	2064	2	2326	1139	1186
8	4252	2153	2098	3	2237	1092	1145
9	4452	2237	2215	4	2263	1104	1159
20	4387	2200	2187	45	2242	1094	1148
1	4286	2145	2141	6	2139	1040	1099
2	4284	2145	2139	7	2223	1077	1146
3	4200	2097	2103	8	2164	1052	1112
4	4162	2077	2085	9	2321	1125	1196

(concluded on next page)

Table A.1 (Concluded)

Population of the U.S. (1000's) - by single years of age[a]

Age	Both	Male	Female	Age	Both	Male	Female
50	2347	1134	1213	75	1111	443	668
1	2295	1106	1189	6	1029	406	623
2	2363	1137	1226	7	952	367	585
3	2337	1119	1218	8	829	315	514
4	2368	1125	1243	9	873	318	555
55	2390	1130	1260	80	723	261	462
6	2330	1102	1227	1	640	224	416
7	2313	1092	1221	2	567	197	370
8	2330	1100	1231	3	528	179	349
9	2252	1058	1194	4	477	158	319
60	2161	1010	1151	85	413	135	278
1	2074	964	1110	6	351	112	239
2	2008	931	1077	7	307	96	211
3	1931	889	1042	8	236	72	164
4	1913	876	1037	9	214	63	151
65	1905	863	1042	90-4	557	159	398
6	1814	814	1000				
7	1764	784	979	95-9	131	35	96
8	1679	740	939				
9	1621	702	920	>100	32	10	22
70	1517	653	863				
1	1440	612	828				
2	1371	577	794				
3	1262	521	741				
4	1208	490	718				

[a] 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[a,b]

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

(continued on next page)

Table A.2 (Continued)

Life table[a,b]

Age	Both sexes		Males		Females	
	Number alive	Life expectancy	Number alive	Life expectancy	Number alive	Life expectancy
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
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

(continued on next page)

Table A.2 (Continued)

Life table[a,b]

Age	Both sexes		Males		Females	
	Number alive	Life expectancy	Number alive	Life expectancy	Number alive	Life expectancy
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
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

(concluded on next page)

Table A.2 (Concluded)

Life table[a,b]

Age	Both sexes		Males		Females	
	Number alive	Life expectancy	Number alive	Life expectancy	Number alive	Life expectancy
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
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

[a] 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.

[b] 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	3.2	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.

Table A.5

Skin cancer rates per 1000 person-years[a]

Ages	Males	Females
0-4	0.2	0.2
5-9	0.3	0.3
10-14	0.5	0.5
15-19	0.9	2.1
20-24	7.1	6.5
25-29	20.9	22.5
30-34	41.9	40.0
35-39	92.3	70.2
40-44	177.5	122.1
45-49	286.0	194.5
50-54	421.8	258.3
55-59	600.2	334.9
60-64	786.7	402.0
65-69	1079.7	492.0
70-74	1286.1	634.5
75-79	1636.5	812.0
80-84	1889.7	907.2
85+	1773.2	955.6

[a] Based on Scotto *et al.* (1974,1983) and Fears and Scotto (1982).

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. Each interval involves an exposure to low-LET radiation at a constant dose rate of 0.1 Gy/yr for 10 years. 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. The inclusion of some alpha-emitting radionuclides in the source term would change the risks for some organs or tissues as described in the text.

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[a]

Time to dose (yr)[b]	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	4.9×10^{-3}	15	40	37	8	-	-	-	-	-	-
10-19	4.6×10^{-3}	+	15	40	37	8	-	-	-	-	-
20-29	4.3×10^{-3}	+	+	15	41	37	7	-	-	-	-
30-39	3.8×10^{-3}	+	+	+	16	42	35	7	-	-	-
40-49	3.0×10^{-3}	+	+	+	+	17	43	34	6	-	-
50-59	2.1×10^{-3}	+	+	+	+	+	19	45	32	4	-
60-69	1.2×10^{-3}	+	+	+	+	+	+	23	51	24	2
70-79	3.7×10^{-4}	+	+	+	+	+	+	+	36	55	9
80-89	4.2×10^{-5}	+	+	+	+	+	+	+	+	63	37
90-99	$< 10^{-6}$	+	+	+	+	+	+	+	+	+	100

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Multiply central estimate of the lifetime risk by 2 to obtain upper estimate; divide central estimate by 2 to obtain lower estimate.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.2

Bone cancer mortality - central estimate[a]

Time to dose (yr)[b]	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	6.0×10^{-5}	15	40	37	8	-	-	-	-	-	-
10-19	5.4×10^{-5}	+	15	40	37	8	-	-	-	-	-
20-29	5.2×10^{-5}	+	+	15	41	37	7	-	-	-	-
30-39	4.5×10^{-5}	+	+	+	16	42	35	7	-	-	-
40-49	3.7×10^{-5}	+	+	+	+	17	43	34	6	-	-
50-59	2.6×10^{-5}	+	+	+	+	+	19	45	32	4	-
60-69	1.4×10^{-5}	+	+	+	+	+	+	23	51	24	2
70-79	4.5×10^{-6}	+	+	+	+	+	+	+	36	55	9
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	63	37
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Multiply central estimate of the lifetime risk by 2 to obtain upper estimate; divide central estimate by 2 to obtain lower estimate.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.3

Breast cancer mortality - central estimate[a]

Time to dose (yr)[b]	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	5.4×10^{-3}	-	6	13	16	19	18	16	9	3	-
10-19	4.7×10^{-3}	+	-	8	19	20	21	18	11	3	-
20-29	3.9×10^{-3}	+	+	-	12	24	26	21	13	4	-
30-39	2.9×10^{-3}	+	+	+	-	17	33	28	17	5	-
40-49	1.9×10^{-3}	+	+	+	+	-	25	41	27	7	-
50-59	9.4×10^{-4}	+	+	+	+	+	-	38	48	14	-
60-69	3.0×10^{-4}	+	+	+	+	+	+	-	62	37	1
70-79	3.3×10^{-5}	+	+	+	+	+	+	+	-	92	8
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risk estimates apply to the entire population. Risks for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.4

Lung cancer mortality - central estimate[a]

Time to dose (yr)[b]	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	7.8×10^{-3}	-	5	10	15	18	22	18	10	2	-
10-19	7.0×10^{-3}	+	-	6	16	21	23	21	11	2	-
20-29	6.0×10^{-3}	+	+	-	10	24	27	23	13	3	-
30-39	4.6×10^{-3}	+	+	+	-	16	34	30	17	3	-
40-49	3.0×10^{-3}	+	+	+	+	-	26	44	25	5	-
50-59	1.5×10^{-3}	+	+	+	+	+	-	43	48	9	-
60-69	3.9×10^{-4}	+	+	+	+	+	+	-	70	29	1
70-79	3.1×10^{-5}	+	+	+	+	+	+	+	-	93	7
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.5

Gastrointestinal cancer mortality - central estimate[a]

Time to dose (yr)[b]	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	1.7×10^{-2}	-	4	9	11	16	20	20	15	5	-
10-19	1.6×10^{-2}	+	-	5	12	18	21	23	16	5	-
20-29	1.4×10^{-2}	+	+	-	8	19	24	25	18	6	-
30-39	1.1×10^{-2}	+	+	+	-	12	28	31	22	7	-
40-49	7.9×10^{-3}	+	+	+	+	-	20	40	31	9	-
50-59	4.4×10^{-3}	+	+	+	+	+	-	34	50	16	-
60-69	1.5×10^{-3}	+	+	+	+	+	+	-	61	38	1
70-79	1.8×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	1.3×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.6

Thyroid cancer mortality - central estimate[a]

Time to dose (yr)[b]	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	7.0×10^{-4}	2	13	15	15	15	14	13	8	4	1
10-19	5.9×10^{-4}	+	3	15	18	17	17	14	11	4	1
20-29	4.9×10^{-4}	+	+	3	19	21	20	18	12	6	1
30-39	3.7×10^{-4}	+	+	+	4	24	25	22	16	8	1
40-49	2.6×10^{-4}	+	+	+	+	5	31	30	22	10	2
50-59	1.6×10^{-4}	+	+	+	+	+	7	41	34	16	2
60-69	7.3×10^{-5}	+	+	+	+	+	+	23	51	24	2
70-79	2.0×10^{-5}	+	+	+	+	+	+	+	20	68	12
80-89	1.6×10^{-6}	+	+	+	+	+	+	+	+	43	57
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Upper and lower estimates of lifetime risk differ only in the treatment of internal sources such as ^{131}I. See Section 2.2.6.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.7

Other cancer mortality - central estimate[a,b]

Time to dose (yr)[c]	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	1.4×10^{-2}	-	5	9	14	17	19	19	13	4	-
10-19	1.3×10^{-2}	+	-	6	14	19	21	22	14	4	-
20-29	1.1×10^{-2}	+	+	-	9	21	25	24	16	5	-
30-39	8.7×10^{-3}	+	+	+	-	14	30	30	20	6	-
40-49	6.0×10^{-3}	+	+	+	+	-	22	41	29	8	-
50-59	3.2×10^{-3}	+	+	+	+	+	-	36	50	14	-
60-69	1.1×10^{-3}	+	+	+	+	+	+	-	62	37	1
70-79	1.2×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	+	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] 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.

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.8

Leukemia *in utero* mortality - central estimate[a]

Time to dose (yr)[b]	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	1.2×10^{-4}	46	53	1	-	-	-	-	-	-	-
10-19	1.2×10^{-4}	+	46	53	1	-	-	-	-	-	-
20-29	1.0×10^{-4}	+	+	46	53	1	-	-	-	-	-
30-39	2.4×10^{-5}	+	+	+	46	53	1	-	-	-	-
40-49	$< 10^{-6}$	+	+	+	+	46	53	1	-	-	-
50-59	-	+	+	+	+	+	-	-	-	-	-
60-69	-	+	+	+	+	+	+	-	-	-	-
70-79	-	+	+	+	+	+	+	+	-	-	-
80-89	-	+	+	+	+	+	+	+	+	-	-
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Multiply central estimates of the lifetime risk by 10/4 to obtain upper estimates. Lower estimates are identical to central estimates. These risks apply to the entire population. Risks to the children exposed *in utero* would be 100 times larger.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.9

Other cancer *in utero* mortality - central estimate[a]

Time to dose (yr)[b]	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	1.2×10^{-4}	46	53	1	-	-	-	-	-	-	-
10-19	1.2×10^{-4}	+	46	53	1	-	-	-	-	-	-
20-29	1.0×10^{-4}	+	+	46	53	1	-	-	-	-	-
30-39	2.4×10^{-5}	+	+	+	46	53	1	-	-	-	-
40-49	$< 10^{-6}$	+	+	+	+	46	53	1	-	-	-
50-59	-	+	+	+	+	+	-	-	-	-	-
60-69	-	+	+	+	+	+	+	-	-	-	-
70-79	-	+	+	+	+	+	+	+	-	-	-
80-89	-	+	+	+	+	+	+	+	+	-	-
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Multiply central estimates of the lifetime risk by 10/4 to obtain upper estimates. Lower estimates are identical to central estimates. These risks apply to the entire population. Risks to the children exposed *in utero* would be 100 times larger.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.10

Breast cancer mortality - lower estimate[a]

Time to dose (yr)[b]	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	1.1×10^{-3}	-	8	17	19	17	14	12	9	3	1
10-19	9.2×10^{-4}	+	-	11	22	20	18	14	10	4	1
20-29	7.2×10^{-4}	+	+	-	15	25	23	18	12	6	1
30-39	5.1×10^{-4}	+	+	+	-	18	31	25	17	8	1
40-49	3.3×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	1.8×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	7.1×10^{-5}	+	+	+	+	+	+	-	48	45	7
70-79	1.4×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risk estimates apply to the entire population. Risks for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.11

Lung cancer mortality - lower estimate[a]

Time to dose (yr)[b]	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	1.7×10^{-3}	-	6	14	18	18	17	13	9	4	1
10-19	1.5×10^{-3}	+	-	9	20	21	19	15	11	4	1
20-29	1.2×10^{-3}	+	+	-	13	26	23	19	12	6	1
30-39	8.8×10^{-4}	+	+	+	-	18	31	25	17	8	1
40-49	5.7×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	3.1×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	1.2×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	2.4×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.12

Gastrointestinal cancer mortality - lower estimate[a]

Time to dose (yr)[b]	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	3.4×10^{-3}	-	10	19	17	17	14	11	8	3	1
10-19	2.7×10^{-3}	+	-	12	22	20	17	14	10	4	1
20-29	2.1×10^{-3}	+	+	-	15	25	23	18	12	6	1
30-39	1.5×10^{-3}	+	+	+	-	18	31	25	17	8	1
40-49	9.7×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	5.3×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	2.1×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	4.2×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.13

Other cancer mortality - lower estimate[a,b]

Time to dose (yr)[c]	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	3.0×10^{-3}	-	10	19	17	17	14	11	8	3	1
10-19	2.4×10^{-3}	+	-	12	22	20	17	14	10	4	1
20-29	1.9×10^{-3}	+	+	-	15	25	23	18	12	6	1
30-39	1.3×10^{-3}	+	+	+	-	18	31	25	17	8	1
40-49	8.6×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	4.6×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	1.8×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	3.7×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	1.5×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

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

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.14

Breast cancer mortality - upper estimate[a]

Time to dose (yr)[b]	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	8.4×10^{-3}	-	5	13	15	19	19	16	10	3	-
10-19	7.4×10^{-3}	+	-	8	17	21	22	18	11	3	-
20-29	6.2×10^{-3}	+	+	-	11	25	25	22	13	4	-
30-39	4.7×10^{-3}	+	+	+	-	17	33	28	17	5	-
40-49	3.0×10^{-3}	+	+	+	+	-	25	41	27	7	-
50-59	1.5×10^{-3}	+	+	+	+	+	-	38	48	14	-
60-69	4.8×10^{-4}	+	+	+	+	+	+	-	62	37	1
70-79	5.3×10^{-5}	+	+	+	+	+	+	+	-	92	8
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risks apply to the entire population. Risks for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.15

Lung cancer mortality - upper estimate[a]

Time to dose (yr)[b]	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	3.3×10^{-2}	-	5	10	15	18	22	18	10	2	-
10-19	3.0×10^{-2}	+	-	6	16	21	23	21	11	2	-
20-29	2.5×10^{-2}	+	+	-	10	24	27	23	13	3	-
30-39	2.0×10^{-2}	+	+	+	-	16	34	30	17	3	-
40-49	1.3×10^{-2}	+	+	+	+	-	26	44	25	5	-
50-59	6.2×10^{-3}	+	+	+	+	+	-	43	48	9	-
60-69	1.7×10^{-3}	+	+	+	+	+	+	-	70	29	1
70-79	1.3×10^{-5}	+	+	+	+	+	+	+	-	93	7
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.16

Gastrointestinal cancer mortality - upper estimate[a]

Time to dose (yr)[b]	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	3.4×10^{-2}	-	4	9	11	16	20	20	15	5	-
10-19	3.1×10^{-2}	+	-	5	12	18	21	23	16	5	-
20-29	2.7×10^{-2}	+	+	-	8	19	24	25	18	6	-
30-39	2.2×10^{-2}	+	+	+	-	12	28	31	22	7	-
40-49	1.6×10^{-2}	+	+	+	+	-	20	40	31	9	-
50-59	8.8×10^{-3}	+	+	+	+	+	-	34	50	16	-
60-69	3.0×10^{-3}	+	+	+	+	+	+	-	61	38	1
70-79	3.5×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	2.6×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-I.17

Other cancer mortality - upper estimate[a,b]

Time to dose (yr)[c]	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	2.8×10^{-2}	-	5	9	14	17	19	19	13	4	-
10-19	2.5×10^{-2}	+	-	6	14	19	21	22	14	4	-
20-29	2.1×10^{-2}	+	+	-	9	21	25	24	16	5	-
30-39	1.7×10^{-2}	+	+	+	-	14	30	30	20	6	-
40-49	1.2×10^{-2}	+	+	+	+	-	22	41	29	8	-
50-59	6.4×10^{-3}	+	+	+	+	+	-	36	50	14	-
60-69	2.1×10^{-3}	+	+	+	+	+	+	-	62	37	1
70-79	2.3×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	1.7×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

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

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

APPENDIX B - Part II

Cancer Morbidity Models for Those Exposed to the Plume

Table B-II.1

Breast cancer morbidity - central estimate[a]

Time to dose (yr)[b]	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	1.6×10^{-2}	-	7	15	18	18	17	15	8	2	-
10-19	1.4×10^{-2}	+	-	10	20	21	20	17	9	3	-
20-29	1.1×10^{-2}	+	+	-	14	25	25	21	12	3	-
30-39	7.9×10^{-3}	+	+	+	-	19	33	27	17	4	-
40-49	5.0×10^{-3}	+	+	+	+	-	27	41	25	7	-
50-59	2.4×10^{-3}	+	+	+	+	+	-	40	47	13	-
60-69	7.3×10^{-4}	+	+	+	+	+	+	-	64	35	1
70-79	7.8×10^{-5}	+	+	+	+	+	+	+	-	92	8
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risk estimates apply to the entire population. Risk for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.2

Lung cancer morbidity - central estimate[a]

Time to dose (yr)[b]	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	8.7×10^{-3}	-	5	10	15	20	21	18	9	2	-
10-19	7.8×10^{-3}	+	-	7	16	21	23	20	11	2	-
20-29	6.6×10^{-3}	+	+	-	11	24	27	23	13	2	-
30-39	5.1×10^{-3}	+	+	+	-	17	34	30	16	3	-
40-49	3.3×10^{-3}	+	+	+	+	-	27	45	23	5	-
50-59	1.5×10^{-3}	+	+	+	+	+	-	44	47	9	-
60-69	4.1×10^{-4}	+	+	+	+	+	+	-	70	29	1
70-79	3.4×10^{-5}	+	+	+	+	+	+	+	-	93	7
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.3

Gastrointestinal cancer morbidity - central estimate[a]

Time to dose (yr)[b]	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	2.9×10^{-2}	-	4	9	12	17	20	20	14	4	-
10-19	2.6×10^{-2}	+	-	5	13	18	22	22	16	4	-
20-29	2.3×10^{-2}	+	+	-	8	20	25	25	17	5	-
30-39	1.8×10^{-2}	+	+	+	-	13	29	31	21	6	-
40-49	1.3×10^{-2}	+	+	+	+	-	21	41	30	8	-
50-59	7.0×10^{-3}	+	+	+	+	+	-	36	49	15	-
60-69	2.3×10^{-3}	+	+	+	+	+	+	-	62	37	1
70-79	2.6×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	1.9×10^{-6}	+	+	+	+	+	+	+	+	+	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.4

Thyroid cancer morbidity - central estimate[a]

Time to dose (yr)[b]	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	7.2×10^{-3}	2	13	15	15	15	14	13	8	4	1
10-19	5.9×10^{-3}	+	3	15	18	17	17	14	11	4	1
20-29	4.9×10^{-3}	+	+	3	19	21	20	18	12	6	1
30-39	3.7×10^{-3}	+	+	+	4	24	25	22	16	8	1
40-49	2.6×10^{-3}	+	+	+	+	5	31	30	22	10	2
50-59	1.6×10^{-3}	+	+	+	+	+	7	41	34	16	2
60-69	7.3×10^{-4}	+	+	+	+	+	+	23	51	24	2
70-79	2.0×10^{-4}	+	+	+	+	+	+	+	20	68	12
80-89	1.6×10^{-5}	+	+	+	+	+	+	+	+	43	57
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Upper and lower estimates of lifetime risk differ only in treatment of internal sources such as ^{131}I. See Section 2.2.6.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.5

Skin cancer morbidity - central estimate[a]

Time to dose (yr)[b]	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	4.4×10^{-2}	-	5	10	14	18	19	19	12	3	-
10-19	4.0×10^{-2}	+	-	6	16	19	22	20	13	4	-
20-29	3.4×10^{-2}	+	+	-	10	22	25	23	16	4	-
30-39	2.7×10^{-2}	+	+	+	-	15	31	29	20	5	-
40-49	1.8×10^{-2}	+	+	+	+	-	23	41	28	8	-
50-59	9.4×10^{-3}	+	+	+	+	+	-	37	49	14	-
60-69	3.0×10^{-3}	+	+	+	+	+	+	-	63	36	1
70-79	3.2×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	2.3×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Multiplication of central estimates of lifetime risk by 2 gives upper estimate; division by 2 gives lower estimates.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.6

Other cancer morbidity - central estimate[a,b]

Time to dose (yr)[c]	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	2.8×10^{-2}	-	5	12	15	18	19	18	10	3	-
10-19	2.4×10^{-2}	+	-	7	16	21	22	19	12	3	-
20-29	2.1×10^{-2}	+	+	-	11	23	26	22	14	4	-
30-39	1.6×10^{-2}	+	+	+	-	16	32	29	18	5	-
40-49	1.0×10^{-2}	+	+	+	+	-	25	42	26	7	-
50-59	5.3×10^{-3}	+	+	+	+	+	-	39	48	13	-
60-69	1.6×10^{-3}	+	+	+	+	+	+	-	64	35	1
70-79	1.7×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	1.3×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] 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.

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.7

Benign thyroid nodule morbidity - central estimate[a]

Time to dose (yr)[b]	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	2.7×10^{-2}	-	9	16	16	16	16	13	9	4	1
10-19	2.2×10^{-2}	+	-	11	19	19	18	16	11	5	1
20-29	1.7×10^{-2}	+	+	-	13	24	22	20	14	6	1
30-39	1.3×10^{-2}	+	+	+	-	17	29	26	18	9	1
40-49	8.9×10^{-3}	+	+	+	+	-	23	36	27	12	2
50-59	4.9×10^{-3}	+	+	+	+	+	-	32	44	21	3
60-69	2.0×10^{-3}	+	+	+	+	+	+	-	48	45	7
70-79	3.9×10^{-4}	+	+	+	+	+	+	+	-	74	26
80-89	1.6×10^{-5}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. Upper and lower estimates of lifetime risk differ only in the treatment of internal sources such as ^{131}I. See Section 2.2.6.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.8

Breast cancer morbidity - lower estimate[a]

Time to dose (yr)[b]	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	3.1×10^{-3}	-	8	17	19	17	14	12	9	3	1
10-19	2.6×10^{-3}	+	-	11	22	20	18	14	10	4	1
20-29	2.0×10^{-3}	+	+	-	15	25	23	18	12	6	1
30-39	1.4×10^{-3}	+	+	+	-	18	31	25	17	8	1
40-49	9.2×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	5.0×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	2.0×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	4.0×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risk estimates apply to the entire population. Risks for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.9

Lung cancer morbidity - lower estimate[a]

Time to dose (yr)[b]	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	1.8×10^{-3}	-	6	14	18	18	17	13	9	4	1
10-19	1.6×10^{-3}	+	-	9	20	21	19	15	11	4	1
20-29	1.3×10^{-3}	+	+	-	13	26	23	19	12	6	1
30-39	9.3×10^{-4}	+	+	+	-	18	31	25	17	8	1
40-49	6.0×10^{-4}	+	+	+	+	-	24	36	26	12	2
50-59	3.3×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	1.3×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	2.6×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.10

Gastrointestinal cancer morbidity - lower estimate[a]

Time to dose (yr)[b]	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	5.7×10^{-3}	-	10	19	17	17	14	11	8	3	1
10-19	4.6×10^{-3}	+	-	12	22	20	17	14	10	4	1
20-29	3.5×10^{-3}	+	+	-	15	25	23	18	12	6	1
30-39	2.5×10^{-3}	+	+	+	-	18	31	25	17	8	1
40-49	1.6×10^{-3}	+	+	+	+	-	24	36	26	12	2
50-59	8.9×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	3.5×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	7.1×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	2.8×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.11

Other cancer morbidity - lower estimate[a,b]

Time to dose (yr)[c]	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	5.7×10^{-3}	-	10	19	17	17	14	11	8	3	1
10-19	4.6×10^{-3}	+	-	12	22	20	17	14	10	4	1
20-29	3.6×10^{-3}	+	+	-	15	25	23	18	12	6	1
30-39	2.5×10^{-3}	+	+	+	-	18	31	25	17	8	1
40-49	1.6×10^{-3}	+	+	+	+	-	24	36	26	12	2
50-59	8.9×10^{-4}	+	+	+	+	+	-	32	44	21	3
60-69	3.5×10^{-4}	+	+	+	+	+	+	-	48	45	7
70-79	7.1×10^{-5}	+	+	+	+	+	+	+	-	74	26
80-89	$< 10^{-6}$	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] 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.

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.12

Breast cancer morbidity - upper estimate[a]

Time to dose (yr)[b]	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	2.5×10^{-2}	-	6	14	17	19	18	15	9	2	-
10-19	2.1×10^{-2}	+	-	9	19	22	20	17	10	3	-
20-29	1.7×10^{-2}	+	+	-	13	26	25	21	12	3	-
30-39	1.3×10^{-2}	+	+	+	-	19	33	27	17	4	-
40-49	8.0×10^{-3}	+	+	+	+	-	27	41	25	7	-
50-59	3.9×10^{-3}	+	+	+	+	+	-	40	47	13	-
60-69	1.2×10^{-3}	+	+	+	+	+	+	-	64	35	1
70-79	1.3×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	9.4×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy. These risk estimates apply to the entire population. Risks for women would be twice as large.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.13

Lung cancer morbidity - upper estimate[a]

Time to dose (yr)[b]	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	3.7×10^{-2}	-	5	10	15	20	21	18	9	2	-
10-19	3.3×10^{-2}	+	-	7	16	21	23	20	11	2	-
20-29	2.8×10^{-2}	+	+	-	11	24	27	23	13	2	-
30-39	2.2×10^{-2}	+	+	+	-	17	34	30	16	3	-
40-49	1.4×10^{-2}	+	+	+	+	-	27	45	23	5	-
50-59	6.6×10^{-3}	+	+	+	+	+	-	44	47	9	-
60-69	1.7×10^{-3}	+	+	+	+	+	+	-	70	29	1
70-79	1.5×10^{-4}	+	+	+	+	+	+	+	-	93	7
80-89	1.0×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.14

Gastrointestinal cancer morbidity - upper estimate[a]

Time to dose (yr)[b]	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	5.8×10^{-2}	-	4	9	12	17	20	20	14	4	-
10-19	5.2×10^{-2}	+	-	5	13	18	22	22	16	4	-
20-29	4.6×10^{-2}	+	+	-	8	20	25	25	17	5	-
30-39	3.6×10^{-2}	+	+	+	-	13	29	31	21	6	-
40-49	2.6×10^{-2}	+	+	+	+	-	21	41	30	8	-
50-59	1.4×10^{-2}	+	+	+	+	+	-	36	49	15	-
60-69	4.6×10^{-3}	+	+	+	+	+	+	-	62	37	1
70-79	5.2×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	3.8×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[b] Years after accident that exposure occurs (in 10-yr intervals).

Table B-II.15

Other cancer morbidity - upper estimate[a,b]

Time to dose (yr)[c]	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	5.5×10^{-2}	-	5	12	15	18	19	18	10	3	-
10-19	4.8×10^{-2}	+	-	7	16	21	22	19	12	3	-
20-29	4.2×10^{-2}	+	+	-	11	23	26	22	14	4	-
30-39	3.2×10^{-2}	+	+	+	-	16	32	29	18	5	-
40-49	2.0×10^{-2}	+	+	+	+	-	25	42	26	7	-
50-59	1.1×10^{-2}	+	+	+	+	+	-	39	48	13	-
60-69	3.2×10^{-3}	+	+	+	+	+	+	-	64	35	1
70-79	3.4×10^{-4}	+	+	+	+	+	+	+	-	92	8
80-89	2.6×10^{-6}	+	+	+	+	+	+	+	+	-	100
90-99	-	+	+	+	+	+	+	+	+	+	-

[a] 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.

[b] Risk estimates apply to exposure at a constant dose rate of 0.1 Gy/yr for 10 years. Total dose to an individual over the 10-yr exposure interval is therefore 1 Gy.

[c] Years after accident that exposure occurs (in 10-yr intervals).

REFERENCES

Anderson, C. G. (1982) *Emergency Medical Response Capability Analysis*, Associated Research Analysis Corp., Fairfax, VA.

Archambeau, J. O. (1987) Relative Radiation Sensitivity of the Integumentary System: Dose Response of the Epidermal, Microvascular, and Dermal Populations. In: *Advances in Radiation Biology*, Vol. 12, Relative Radiation Sensitivities of Human Organ Systems, J. T. Lett, and K. I. Altman, eds., pp. 147-203. Academic Press, New York.

Becker, D. V., McConahey, W. M., Dobyns, B. M., Tompkins, E., Sheline, G. E., and Workman, J. B. (1971) The Results of Radioiodine Treatment of Hyperthyroidism, A Preliminary Report of the Thyrotoxicosis Therapy Follow-up Study. In: *Further Advances in Thyroid Research*, Vol. 1, K. Fellinger, and R. Hofer, eds., pp. 603-609. Verlag der Wiener Medizinischen Akademie, Vienna.

Beierwalters, W. H. and Johnson, P. C. (1956) Hyperthyroidism Treated with Radioiodine: Seven-Year Experience, *Arch. Intern. Med. 97*:393-398.

Bond, V. P., Fliedner, T. M., and Archambeau, J. O. (1965) *Mammalian Radiation Lethality: A Distribution in Cellular Kinetics*, Academic Press, New York.

Brent, R. L. (1986) The Effects of Embryonic and Fetal Exposure to X-Ray, Microwaves, and Ultrasound, *Clin. Perinatol. 13*:615-648.

Brent, R. L., Beckman, D. A., and Jersh, R. P. (1987) Relative Radiosensitivity of Fetal Tissue. In: *Advances in Radiation Biology*, Vol. 12, Relative Radiation Sensitivities of Human Organ Systems, pp. 238-256, Academic Press, New York.

BOC (1983) Bureau of the Census, *General Population Characteristics, United States Summary, 1980 Census of Population*, U.S. Department of Commerce, Washington, DC.

Coggle, J. E., Hansen, L. S., Wells, J., and Charles, M. W. (1984) Non-Stochastic Effects of Different Energy β-Emitters on Mouse Skin, *Radiat. Res. 99*:336-345.

Cohen, L. (1966) Radiation Response and Recovery: Radiobiological Principles and Their Relationship to Clinical Practice. In: *The Biological Basis of Radiation Therapy*, E. E. Schwartz, ed., Lippincott, Philadelphia.

Cross, F. T., Endres, G. W., and Sullivan, M. F. (1978) Dose to the GI Tract from Ingested Insoluble Beta Emitters, *Radiat. Res. 73*:37-50.

Damewood, M. D., and Grochow, L. B. (1986) Prospects for Fertility after Chemotherapy or Radiation for Neoplastic Disease, *Fertil. Steril. 45*:443-459.

Fears, T. and Scotto, J. (1982) Changes in Skin Morbidity Between 1971-72 and 1977-78, *J. Natl. Cancer Inst. 69*:365-370.

Fryer, C. J. H., Fitzpatrick, P. J., Rider, W. D., and Poon, P. (1978) Radiation Pneumonitis: Experience Following a Large Single Dose of Radiation, *Int. J. Radiat. Oncol. Biol. Phys. 4*:931-936.

Gus'kova, A. S. (1987) *Early Acute Effects of the Chernobyl Accident*, ICRP/87/C:G-01, translation provided by R. W. Young, Defense Nuclear Agency, Washington, DC.

ICRP (1991) International Commission on Radiological Protection, *1990 Recommendations of the International Commission on Radiological Protection*, ICRP Publication 60, Pergamon Press, Oxford, UK.

Jablon, S. and Kato, H. (1970) Childhood Cancer in Relation to Prenatal Exposure to Atomic-Bomb Radiation, *Lancet 2*:1000-1003.

Kato, H. and Schull, W. J. (1982) Studies of the Mortality of A-Bomb Survivors, 7. Mortality, 1950-1978: Part I. Cancer Mortality, *Radiat. Res. 90*:395-432.

Keane, T. J., Van Dyk, J., and Rider, W. D. (1981) Idiopathic Interstitial Pneumonia Following Bone Marrow Transplantation: The Relationship with Total-Body Irradiation, *Int. J. Radiat. Oncol. Biol. Phys. 7*:1365-1370.

Laird, N. M. (1987) Thyroid Cancer Risk from Exposure to Ionizing Radiation: A Case Study in the Comparative Potency Model, *Risk Analysis 7*:299-309.

Langham, W. H. (1967) *Radiobiological Factors in Manned Space Flight*, NAS-NRC 1487, Washington, DC.

Lushbaugh, C. C. and Ricks, R. C. (1972) Some Cytokinetic and Histopathologic Considerations of Irradiated Male and Female Gonadal Tissues. In: *Frontiers of Radiation Therapy and Oncology*, Vol. 6, J. M. Veath, and S. Karger, eds., pp. 228-248. Basel.

Lushbaugh, C. C., Fry, S. A., Ricks, R. C., Hubner, K. F., and Burr, W. W. (1986) Historical Update of Past and Recent Skin Damage Radiation Accidents, *Brit. J. Radiol. Suppl. No. 19*:7-12.

MacVittie, T. J., Monroy, R. L., Patchen, M. L., and Darden, J. H. (1984) Acute Lethality and Radiosensitivity of the Canine Hematopoietic System to Cobalt-60 Gamma and Mixed Neutron-Gamma Irradiation. In: *Response of Different Species to Total Body Irradiation*, J. J. Broerse and T. J. MacVittie, eds., pp. 113-129. Martinus Nijhoff Publishers, Boston.

Maxon, H. R., Thomas, S. R., Saenger, E. L. Buncher, C. R., Kareiakes, J. G. (1977) Ionizing Irradiation and the Induction of Clinically Significant Disease in the Human Thyroid Gland, *Am. J. Med. 63*:967-978.

McClellan, R. O., Boecker, B. B., Cuddihy, R. G., Griffith, W. C., Hahn, F. F., Muggenburg, B. A., Scott, B. R., and Seiler, F. A. (1982) Health Effects from Internally Deposited Radionuclides Released in Nuclear Disasters. In: *The Control of Exposure of the Public to Ionizing Radiation in the Event of Accident or Attack*, pp. 28-39. Proceedings of a Symposium Sponsored by the National Council on Radiation Protection and Measurements, Reston, VA, April 27-29, 1981.

McKusick, V. A. (1983) *Mendelian Inheritance in Man: Catalogs of Autosomal Dominant, Autosomal Recessive, and X-Linked Phenotypes*, 6th Ed., The Johns Hopkins University Press, 1378, Baltimore, MD.

Miller, A. B., Howe, G. R., Sherman, G. J., Yaffe, M. J., Dinner, P., Risch, H. A., and Preston, D. C. (1989) Breast Cancer Mortality Following Irradiation in a Cohort of Canadian Tuberculosis Patients, *N. Engl. J. Med. 321*:1285-1289.

Moritz, A. R. and Henriques, F. W. Jr. (1952) Effect of Beta Rays on the Skin as a Function of the Energy Intensity and Duration of Radiation: II, Animal Experiments, *Lab Invest. 1*:167-185.

Morris, M. D. and Jones, T. D. (1988) A Comparison of Dose-Response Models for Death from Hematological Depression in Different Species, *Int. J. Radiat. Biol. 53*:439-456.

Morris, M. D. and Jones, T. D. (1989) Hematopoietic Death of Unprotected Man from Photon Irradiations: Statistical Modeling from Animal Experiments, *Int. J. Radiat. Biol. 55*:445-461.

NAS/NRC (1972) National Academy of Sciences/National Research Council, *The Effects of Populations of Exposures to Low Levels of Ionizing Radiation, BEIR I*, Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, DC.

NAS/NRC (1980) National Academy of Sciences/National Research Council, *The Effects on Populations of Exposures to Low Levels of Ionizing Radiation, BEIR III*, Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, DC.

NAS/NRC (1988) National Academy of Sciences/National Research Council, *Health Risks of Radon and Other Internally Deposited Alpha-Emitters, BEIR IV,* Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, DC.

NAS/NRC (1990) National Academy of Sciences/National Research Council, *Health Effects of Exposure to Low Levels of Ionizing Radiation, BEIR V,* Committee on the Biological Effects of Ionizing Radiations, National Academy Press, Washington, DC.

NCHS (1981) National Center for Health Statistics, *1978 Vital Statistics of the United States, Volume II,* Public Health Service, U.S. Department of Health and Human Services, DHHS Publication No. (PHS) 81-1104, Hyattsville, MD.

NCHS (1985) National Center for Health Statistics, *U.S. Decennial Life Tables for 1979-81, Volume I, Number 1, United States Life Tables,* Public Health Service, U.S. Department of Health and Human Services, DHHS Publication No. (PHS) 85-1150, Washington, DC.

NCI (1981) National Cancer Institute, *Cancer Incidence and Mortality in the United States, 1973-1977,* NCI Monograph No. 57, Surveillance, Epidemiology, and End Results (SEER) Program, National Institutes of Health, Bethesda, MD.

NCRP (1980) National Council on Radiation Protection and Measurements, *Influence of Dose and Its Distribution in Time on Dose-Response Relationships for Low-LET Radiations.* Report 64, NCRP, Washington, DC.

Neumeister, K. and Wasser, S. (1985) Clinical Data for Radiation Embryology: Investigation Programme 1967, Report 1984, *Radiat. Environ. Biophys. 24*:227-237.

NIH (1985) National Institute of Health, *Report of the National Institute of Health Ad Hoc Working Group to Develop Radioepidemiological Tables,* NIH Publication No. 85-2748, U.S. Department of Health and Human Services, Washington, DC.

NRC (1975) *Reactor Safety Study: An Assessment of Accident Risks in the U.S. Commercial Nuclear Power Plants, Appendix VI, Calculation of Reactor Accident Consequences,* WASH-1400, NUREG-75/014, United States Nuclear Regulatory Commission, Washington, DC.

NRC (1977) *Overview of the Reactor Safety Study Consequence Model,* NUREG-0340, Probabilistic Analysis Branch, Office of Nuclear Regulatory Research, United States Nuclear Regulatory Commission, Washington, DC.

NRC (1983) *PRA Procedures Guide—A Guide to the Performance of Probabilistic Risk Assessments for Nuclear Power Plants,* NUREG/CR-2300, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1985) *Health Effects Model for Nuclear Power Plant Accident Consequence Analysis*, NUREG/CR-4214, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1989) *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Low LET Radiation. Part II: Scientific Bases for Health Effects Models*, Report NUREG/CR-4214 Rev. 1, Part II, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1990a) *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Low LET Radiation. Part I: Introduction, Integration and Summary*, Report NUREG/CR-4214 Rev. 1, Part I, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1990b) *MELCORE Accident Consequence Code System (MACCS)*, Report NUREG/CR-4691, SAND86-1562, Vols. 1-3, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1991) *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Modifications of Models Restulting from Recent Reports on Health Effects of Ionizing Radiation. Low LET Radiation. Part II: Scientific Bases for Health Effects Models*, Report NUREG/CR-4214 Rev. 1, Part II, Addendum 1, U.S. Nuclear Regulatory Commission, Washington, DC.

NRC (1993) *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Modifications of Models Restulting from Addition of Effects of Exposure to Alpha-Emitting Radionuclides. Part II: Scientific Bases for Health Effects Models*, Report NUREG/CR-4214 Rev. 1, Part II, Addendum 2, U.S. Nuclear Regulatory Commission, Washington, DC.

Otake, M., Yoshimaru, H., and Schull, W. J. (1987) *Effects of Ionizing Radiation on the Developing Human Brain*, pp. 203-217 in Proceedings of the NCRP 23rd Annual Meeting, April 8, 1987, Proceedings No. 9, NCRP, Washington, DC.

Otake, M., Yoshimaru, H. and Schull, J. (1989) Prenatal Exposure to Atomic Radiation and Brain Damage, *Cong. Anom. 29*:309-320.

Peel, D. M. and Hopewell, J. W. (1984) Nonstochastic Effects of Different Energy Beta Emitters on Pig Skin, *Radiat. Res. 99*:372-382.

Perman, V., Cronkite, E. P., Bond, V. P., and Sorensen, D. K. (1962) The Regenerative Ability of Hematopoietic Tissue Following Lethal X-Irradiation in Dogs, *Blood 19*:724-737.

Phillips, T. L. and Margolis, L. (1972) Radiation Pathology and the Clinical Response of Lung and Esophagus, *Front. Radiat. Ther. Oncol. 6*:254-273.

Preston, D. L. and Pierce, D. A. (1987) *The Effect of Changes in Dosimetry on Cancer Mortality Risk Estimates in the Atomic Bomb Survivors*. RERF Technical Report 9-87 (Hiroshima City 730, Japan: Radiation Effects Research Foundation).

Ritchie, L. T. (1983) *Calculations of Reactor Accident Consequence Version 2 CRAC2: Computer Code, User's Guide*, Report NUREG/CR-2326, U.S. Nuclear Regulatory Commission, Washington, DC.

Rubin, P. and Casarett, G. W. (1968) *Clinical Radiation Pathology*, W. B. Saunders Co., Philadelphia.

Scott, B. R., Hahn, F. F., McClellan, R. O., and Seiler, F. A. (1988) Risk Estimators for Radiation-Induced Bone Marrow Syndrome Lethality in Humans, *Risk Anal. 8*:393-402.

Scott, B. R., Filipy, R. E., and Hahn, F. F. (1989) *Models for Pulmonary Lethality and Morbidity After Irradiation from Internal and External Sources*, Report NUREG/CR-5351, U.S. Nuclear Regulatory Commission, Washington, DC.

Scott, B. R. (1989) Probabilistic Models for Early and Continuing Radiobiological Effects of Nuclear Power Plant Accidents. In: *Proceedings for PSA '89 International Topical Meeting, Probability, Reliability, and Safety Assessment*, pp. 732-737. Sponsored by American Nuclear Society, April 2-7, 1989, Pittsburgh.

Scotto, J., Kopf, A. and Urbach, F. (1974) Non-Melanoma Skin Cancer Among Caucasians in Four Areas of the United States, *Cancer 34*: 1333-1338.

Scotto, J., Fears, T. and Fraumeni, J. (1983) Incidence of Nonmelanoma Skin Cancer in the United States, U.S. Department of Health and Human Services, NIH Publication No. 83-2433.

Selby, P. B. and Selby, P. R. (1977) Gamma-Ray-Induced Dominant Mutations That Cause Skeletal Abnormalities in Mice, *Mutation Res. 43*:357-375.

Shimizu, Y., Kato, H., and Schull, W. J. (1990) Studies of Mortality of A-Bomb Survivors. 9. Mortality, 1950-1985: Part 2. Cancer Based on the Recently Revised Doses (DS 86), *Radiat. Res. 121*:120-141.

Shore, R. E., Albert, R. E., Reed, M. Harley, N., and Pasternack, B. S. (1984) Skin Cancer Incidence Among Children Irradiated for Ringworm of the Scalp, *Radiat. Res. 100*:192-204.

Shore, R. E. (1990) Overview of Radiation-Induced Skin Cancer in Humans, *Int. J. Radiat. Biol. 57*:809-827.

Sridama, V., McCormick, M., Kaplan, E. L., Fauchet, R., and DeGroat, L. J. (1984) Long-Term Follow-Up Study of Compensated Low-Dose ^{131}I Therapy for Graves Disease, *N. Engl. J. Med. 311*:426-432.

Stewart, A. and Kneale, G. W. (1968) Changes in the Cancer Risk Associated with Obstetric Radiography, *Lancet 1*:104-107.

Sullivan, M. F., Marks, S., Hackett, P. L., and Thompson, R. C. (1959) X-Irradiation of the Exteriorized or *In Situ* Intestine of the Rat, *Radiat. Res. 11*:653-666.

Thomas, E. D., Storb, R., and Clift, R. A. (1975) Bone Marrow Transplantation (First of Two Parts), *New. Engl. J. Med. 292*:832-843.

UNSCEAR (1972) *Ionizing Radiation: Levels and Effects*, United Nations Scientific Committee on the Effects of Atomic Radiation Report to the General Assembly, with Annexes, United Nations, New York.

UNSCEAR (1977) *Sources and Effects of Ionizing Radiation*, United Nations Scientific Committee on the Effects of Atomic Radiation Report to the General Assembly, with Annexes, United Nations, New York.

UNSCEAR (1982) *Ionizing Radiation: Sources and Biological Effects*, United Nations Scientific Committee on the Effects of Atomic Radiation Report to the General Assembly, with Annexes, United Nations, New York.

UNSCEAR (1986) *Genetic and Somatic Effects of Ionizing Radiation*, United Nations Scientific Committee on the Effects of Atomic Radiation, United Nations, New York.

UNSCEAR (1988) *Sources, Effects and Risks of Ionizing Radiation*, United Nations Scientific Committee on the Effects of Atomic Radiation Report to the General Assembly, with Annexes, United Nations, New York.

Van Dyk, J., Keane, T. J., Kan, S., Rider, W. D. and Fryer, C. J. H. (1981) Radiation Pneumonitis Following Large Single Dose Irradiation: A Reevaluation Based on Absolute Dose to Lung, *Int. J. Radiat. Oncol. Biol. Phys. 7*:461-467.

Von Essen, C. F. (1969) Radiation Tolerance of the Skin, *Acta Radiol. Ther. Phys. Biol. 8*:311-330.

Vriesendorp, H. M., and van Bekkum, D. W. (1984) Susceptibility to Total Body Irradiation. In: *Response of Different Species to Total-Body Irradiation*, J. J. Broerse, and J. T. MacVittee, eds., Martinus Nijhoff Publishers, Boston.

Wakabayashi, T., Kato, H., Ikeda, T. and Schull, W. J. (1983) Studies of the Mortality of A-Bomb Survivors, Report 7, Part III. Incidence of Cancer in 1959-1978, Based on the tumor Registry, Nagasaki, *Radiat. Res. 93*:112-146.

Weiner, R. S., Bortin, M. M., Gale, R. P., Gluckman, E., Kay, H. E., Kolb, H. J., Hartz, A. J., and Rimm, A. A. (1986) Interstitial Pneumonitis After Bone Marrow Transplantation, *Ann. Int. Med. 140*:168-175.

Young, R. W. (1986) Mechanisms and Treatment of Radiation-Induced Nausea and Vomiting. In: *Nausea and Vomiting: Mechanisms and Treatment*, C. H. Davis, G. V. Lake-Bakaar, and D. G. Grahame-Smith, eds., pp. 94-109. Springer-Verlag, Berlin.

NRC FORM 335 (2 89) NRCM 1102, 3201, 3202

U.S. NUCLEAR REGULATORY COMMISSION

BIBLIOGRAPHIC DATA SHEET

(See instructions on the reverse)

1. REPORT NUMBER (Assigned by NRC, Add Vol., Supp., Rev., and Addendum Numbers, If any.)
NUREG/CR-4214
Rev. 2, Part I
ITRI-141

2. TITLE AND SUBTITLE
Health Effects Models for Nuclear Power Plant Accident Consequence Analysis

Part I: Introduction, Integration, and Summary

3. DATE REPORT PUBLISHED
MONTH: October | YEAR: 1993

4. FIN OR GRANT NUMBER
FIN L1264

5. AUTHOR(S)
J. S. Evans[1], S. Abrahamson[2], M. A. Bender[3], B. B.Boecker[4], E. S. Gilbert[5], B. R. Scott[4]

6. TYPE OF REPORT
Technical

7. PERIOD COVERED *(Inclusive Dates)*

8. PERFORMING ORGANIZATION – NAME AND ADDRESS *(If NRC, provide Division, Office or Region, U.S. Nuclear Regulatory Commission, and mailing address; if contractor, provide name and mailing address.)*
Inhalation Toxicology Research Institute
Lovelace Biomedical and Environmental Research Institute
P.O. Box 5890, Albuquerque, NM 87185

[1]Harvard School of Public Health, Boston, MA 02115
[2]University of Wisconsin, Madison, WI 53706
[3]Brookhaven National Laboratory, Upton, NY 11973
[4]Inhalation Toxicology Research Institute, Albuquerque, NM 87185
[5]Battelle Pacific Northwest Laboratories, Richland, WA 99352

9. SPONSORING ORGANIZATION – NAME AND ADDRESS *(If NRC, type "Same as above"; if contractor, provide NRC Division, Office or Region, U.S. Nuclear Regulatory Commission, and mailing address.)*
Division of Regulatory Applications
Office of Nuclear Regulatory Research
U.S. Nuclear Regulatory Commission
Washington, DC 20555 -0001

10. SUPPLEMENTARY NOTES

11. ABSTRACT *(200 words or less)*

This report is a revision of NUREG/CR-4214, Rev. 1, Part I (1990), *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis*. This revision has been made to incorporate changes to the Health Effects Models recommended in two addenda to the NUREG/CR-4214, Rev. 1, Part II, 1989 report. The first of these addenda provided recommended changes to the health effects models for low-LET radiations based on recent reports from UNSCEAR, ICRP and NAS/NRC (BEIR V). The second addendum presented changes needed to incorporate alpha-emitting radionuclides into the accident exposure source term. As in the earlier version of this report, models are provided for early and continuing effects, cancers and thyroid nodules, and genetic effects. 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. Linear and linear-quadratic models are recommended for estimating the risks of seven types of cancer in adults—leukemia, bone, lung, breast, gastrointestinal, thyroid, and "other." For most cancers, both incidence and mortality are addressed. Five classes of genetic disease—dominant, x-linked, aneuploidy, unbalanced translocations, and multifactorial diseases—are also considered. Data are provided that should enable analysts to consider the timing and severity of each type of health risk.

12. KEY WORDS/DESCRIPTORS *(List words or phrases that will assist researchers in locating the report.)*
nuclear power plant accidents
health effects of radiation
early effects of radiation
late somatic effects of radiation
genetic effects of radiation
alpha-emitting radionuclides

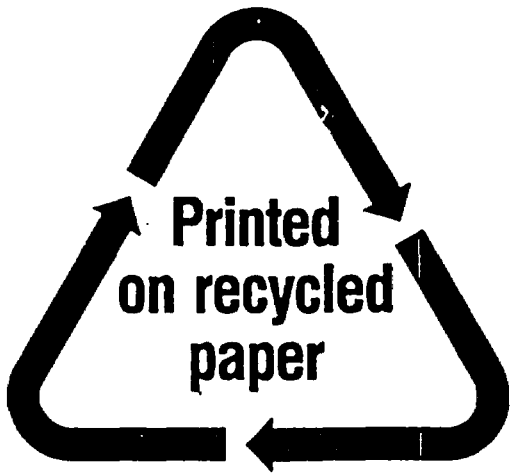
13. AVAILABILITY STATEMENT
unlimited

14. SECURITY CLASSIFICATION
(This Page) unclassified
(This Report) unclassified

15. NUMBER OF PAGES

16. PRICE

NRC FORM 335 (2-89)



Federal Recycling Program

UNITED STATES
NUCLEAR REGULATORY COMMISSION
WASHINGTON, D.C. 20555-0001

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, $300