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The Economic Costs of Radiation-Induced Health Effects: Estimation and Simulation

Prepared by L.A. Nieves, J.J. Tawil

Pacific Northwest Laboratory
Operated by
Battelle Memorial Institute

Prepared for
U.S. Nuclear Regulatory
Commission

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PREFACE

Work on this model and documentation was largely completed before the accident at the Chernobyl nuclear plant (U.S.S.R.) in April 1986. Ongoing research related to population exposures and health effects due to the Chernobyl disaster may provide information relevant to HECOM input data and model structure. Revised input data may be easily substituted for the default data.

SUMMARY

The research effort covered by this report was performed by Pacific Northwest Laboratory (PNL) for the Office of Nuclear Reactor Regulation of the Nuclear Regulatory Commission. The purpose of this effort is to improve the quantitative information available for use in evaluating actions that alter health risks due to population exposure to ionizing radiation. To project the potential future costs of changes in health effects risks, PNL constructed a probabilistic computer model, Health Effects Costs Model (HECOM), which utilizes the health effect incidence estimates from accident consequences models to calculate the discounted sum of the economic costs associated with population exposure to ionizing radiation. Application of HECOM to value-impact and environmental impact analyses should greatly increase the quality of the information available for regulatory decision making.

Three major types of health effects present risks for any population sustaining a significant radiation exposure: acute radiation injuries (and fatalities), latent cancers, and impairments due to genetic effects. The literature pertaining to both incidence and treatment of these health effects was reviewed by PNL and provided the basis for developing economic cost estimates.

The economic costs of health effects estimated by HECOM represent both the value of resources consumed in diagnosing, treating, and caring for the patient and the value of goods not produced because of illness or premature death due to the health effect. Additional costs to society, such as pain and suffering, are not included in the PNL economic cost measures since they do not divert resources from other uses, are difficult to quantify, and do not have a value observable in the marketplace.

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First, we would like to thank Guy McWethy for his long-suffering effort in programming the code. Because of the multidisciplinary nature of the effort involved in developing estimates of health effects costs, the authors have depended on the guidance and criticism of individuals from several fields. We wish to thank each of them for their contribution to our efforts. Guidance in regard to the nature and incidence of genetic defects was provided by an epidemiologist, Lowell Sever, and text was reviewed by a biologist, Roy Thompson, Jr. Ethel Gilbert, a statistician, assisted by reviewing the report sections related to cancers. Radiation injury related sections were reviewed by a physician, Bryce Breitenstein, President of Hanford Environmental Health Foundation, and a health physicist, Joseph Soldat. William Bair also provided an overall review from his perspectives in health effects and environmental studies. We also wish to thank Don Cleary at NRC who provided support for this effort.

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1.0 INTRODUCTION

This report was prepared by the Pacific Northwest Laboratory (PNL) for the Office of Nuclear Reactor Regulation (NRR) of the Nuclear Regulatory Commission (NRC). The purpose of this effort is to improve the quantitative information used in evaluating actions that alter health risks associated with exposure to radionuclides. To fulfill this purpose, PNL 1) evaluated the conceptual and informational basis for measuring the total cost to society of radiation-induced health effects, 2) estimated the economic cost component for the major categories of potential radiation-induced health effects, and 3) developed a flexible computer code [Health Effects Cost Model (HECOM)] for calculating costs that could result over time due to a single incident of population exposure to radionuclides. As a result of this effort, quantitative estimates of the economic costs associated with health effects risks are available for inclusion in environmental impact statements for nuclear facility siting and for evaluation of safety-related measures. The HECOM model has been designed for compatibility with health effect output of the NRC's CRAC and MACCS models.

The introductory section of this report discusses the need for health effect cost estimates, the nature of the radiation-induced health effects, the composition of resulting costs, and the scope and capabilities of the PNL cost simulation model. An outline of the report structure is also provided.

1.1 THE NEED FOR HEALTH EFFECTS COST ESTIMATES

Estimates of the health effects that may result from radiation exposure are used by the NRC in many types of analyses. Unlike other types of potential accident consequences, such as offsite property damage, a dollar value has not generally been ascribed to potential health effects. This is due in part to the relative lack of economic models and data for the costing of health effects. A number of recent efforts have substantially improved the economic data in this area and this present work offers a comprehensive economic model of health effect costs.

The lack of economic treatment of health effects has also been due to the argument that it is inappropriate to place an explicit value on human life and health. This study does not attempt to estimate the total value of human life or health; it estimates the economic losses to society that could occur due to radiation-induced illness and injury. While the argument may be made that property damages and human health effects are qualitatively different, the measurable economic costs of health effects are better included in risk-related decision making than excluded. Although the cost information developed in this study is incomplete, having it is preferable to having no information as to the relative magnitude of health effects costs.

The cost estimates resulting from this study have applications in several types of analyses carried out by the NRC. They may be used in developing health effects impacts assessments for the nuclear fuel cycle, in total or in part. They are needed to evaluate safety goals, especially the benefits of

avoiding health risks. In addition, there are applications in nuclear facility licensing procedures and National Environmental Policy Act (NEPA) related assessments. The cost estimates are also broadly applicable to risk analyses involving either acute radiation exposure, or other hazardous substance exposures resulting in induction of cancers or genetic effects.

1.2 OVERVIEW OF SOCIETAL (a) COSTS OF RADIATION-INDUCED HEALTH EFFECTS

The value of avoiding radiation exposure, of the general population or of workers, is determined by the total cost to society that is likely to result from the effects of exposure. All health effects result in costs to society because of the resources consumed in treating illness and because of the lost productivity of affected individuals. These "primary" economic costs are referred to as direct and indirect costs. The direct cost category includes all costs for treatment, travel to obtain treatment, patient care, equipment and supplies; indirect costs include the losses due to the reduced productivity of the patient or his family. Such productivity losses may occur because the patient is too ill to work, the family is caring for the patient, the patient's functioning is permanently impaired, or the patient dies at a younger age than would have been likely without the radiation-induced health effect.

In addition to the primary costs of health effects, there are "secondary" costs that are not monetary in nature. These costs include the value of pain and suffering; the cost of family members' stress-induced illness precipitated by the illness or death of the patient; and the cost of depression or psychological stress due to actual or anticipated illness. While attempts have been made to measure some of these effects (Abt 1975), no rigorous estimates of secondary effects or their costs are available, either in absolute terms or relative to primary costs.

The types of health effects that may be induced by radiation are described briefly in Section 1.2.1. The relationship between the occurrence of health effects and the occurrence of economic costs is discussed in Section 1.2.2. Some of the difficulties in accounting for societal costs are discussed and the measurement approach taken by PNL is explained in Section 1.2.3.

1.2.1 Description of Health Effects

Three major types of health effects are of concern for any population sustaining a significant radiation exposure: acute radiation injuries, cancers and genetic effects. A broad review of current knowledge regarding these health effects was recently completed by the NRC (Evans, Moeller and Cooper 1985). That study is the source of much of the incidence-related information

(a) Societal cost includes all monetary and nonmonetary costs. The PNL health effects cost estimates include only the subset of costs that are monetary in nature.

in this report. Brief descriptions of the illnesses included in each of these categories are provided below. Further detail related to the incidence and treatment of these effects can be found in Chapters 4, 5 and 6.

Acute Radiation Injuries

The occurrence of acute radiation injuries in an exposed population is determined by the total dose, the rate at which the dose is received, and the quality of the radiation. A wide variety of biological effects may result from exposure to radiation. The possible injuries vary in intensity from negligible or undetectable to those that are more severe: from temporary discomfort to permanent impairment and life-threatening effects. Characteristics of the major types of radiation injuries are given below. For external sources of x-rays, gamma rays, and beta particles, the dose units "rad", and "rem" are equivalent. A Gray (Gy) equals 100 rad.

- **Prodromal Symptoms** - These flu-like symptoms may result from a combination of the effects of tissue damage and anxiety about the ultimate effects of the individual's radiation exposure (Blakely 1968, p. 35; Dalrymple 1973, p. 192). Symptoms begin within a few hours of exposure and generally subside in a few days. Affected individuals may experience nausea, loss of appetite, headache, diarrhea and weakness. Occasionally, individuals receiving a dose as low as 0.5 Gy may be affected and virtually everyone exposed at doses above 2 Gy would exhibit these symptoms (Blakely 1968, p. 35).
- **Gastrointestinal Syndrome** - At gastrointestinal doses above 15 Gy, cellular damage may result in fatal gastrointestinal symptoms for half the exposed populations. Symptoms include vomiting and diarrhea with severe fluid loss, failure of food absorption and hemorrhage. Intestinal ulceration may occur, accompanied by bacterial invasion (Blakely 1968, p. 41). Affected persons may be expected to die within 10 to 14 days or to survive to exhibit the bone marrow impairment described below.
- **Hematopoietic Syndrome** - This process is initiated by whole body exposures of 2 Gy or more. There is damage to the bone marrow, spleen and lymph nodes that, in turn, results in impairment of the body's blood-forming and immune functions (NRC 1975, Appendix VI, p. F-1). The illness is characterized by infections, hemorrhage and anemia, which may be fatal alone or in combination. With supportive treatment approximately 50 percent of exposed individuals may be expected to die within two months after acute doses greater than about 4.5 Gy (Evans, Moeller and Cooper 1985, Vol. 2, p. 20).
- **Pulmonary Syndrome** - Doses of about 4.8 Gy or more (Evans, Moeller and Cooper 1985, Vol. 2, p. 37) can result in impaired pulmonary function. There may be pulmonary infections, and shortness of breath may in turn affect heart function. Generally, injuries from lung exposure induce pneumonitis, followed by pulmonary fibrosis (Evans, Moeller and Cooper 1985, Vol. 2, p. 35).

- Prenatal Injury - The radiosensitivity of embryos is very high, resulting in deaths from doses as low as 0.5 Gy. Most such deaths would be unnoticed since they would occur in the early stage of pregnancy (NRC 1975, Appendix VI, p. F-17-20). In later stages of development the fatality rate decreases but the probability of abnormalities increases. These generally take the form of growth impairment, especially microcephaly and mental retardation. The data indicate that critical doses are in the range of 0.37 to 1.3 Gy (Evans, Moeller and Cooper 1985, Vol. 2, p. 54-60).
- Thyroid Disorders - Thyroiditis and hypothyroidism are impairments of thyroid function that can be induced by radiation exposure. These injuries are likely to result from inhalation rather than external doses. The dose threshold is about 200 Gy for occurrence of thyroiditis, an inflammation seen about two weeks after exposure. Hypothyroidism is a deficiency of thyroid function seen years after exposure. The median dose for brief exposure is 60 Gy (Evans, Moeller and Cooper 1985, vol. 2, p. 41).
- Skin Damage - The skin may also be affected by doses in the range of 6 to 20 Gy, resulting in radiation dermatitis. This condition has levels of severity comparable to first, second, and third degree thermal burns and in the most severe cases (due to doses of over 20 Gy) can result in permanent skin ulceration (Prasad 1974, p. 240-248; Evans, Moeller and Cooper 1985, Vol. 2, p. 48).
- Cataracts - Doses of 2 to 5 Gy to the lens of the eye may result in formation of cataracts after a latency period that varies with both dose and dose rate (Evans, Moeller and Cooper 1985, Vol. 2, p. 58).
- Sterility - Radiation-induced sterility may be either temporary or permanent. For males, temporary effects occur at a lower dose than for females, but a higher dose is required for permanent effects. Permanent sterility, in males or females, is unlikely below doses that are life threatening if whole body exposure is involved (NRC 1975, Appendix VI, p. 9-15).

Cancers

Cancers, induced by radiation exposure are indistinguishable from other cancers. As a result, the cause of any particular cancer is rarely, if ever, identifiable. Radiation-induced cancers may only be apparent as an increased statistical rate of cancer incidence in an affected population. The "excess" cancer may then be attributed to the radiation exposure of the population.

Susceptibility to cancer varies among organs and tissues, so that the rates at which excess cancers appear in various sites differ. Cancer induction is influenced by sex, age when irradiated, and type of radiation, among other factors (BEIR 1980, p. 84-85). The cancers that are most likely to be radiation induced are leukemia and cancers of the breast, bone, lung, skin and gastrointestinal tract. Both benign and malignant thyroid nodules may also be

induced. While it is possible for radiation-induced cancers to occur in other organs and tissues, the types mentioned above are the most likely and are the focus of concern in this study.

Genetic Effects

Genetic effects may affect many generations of the offspring of persons exposed to radiation, though at a decreasing rate over time. Radiation may increase the mutation rate, but does not affect the nature of mutations or the associated health effects. Thus, the health effects that occur in offspring of an exposed population are the same types that occur spontaneously.

Of the possible types of mutation, autosomal dominant disorders are most likely to increase in direct proportion with radiation exposure. Resulting disorders include chondrodystrophy, osteogenesis imperfecta, neurofibromatosis, eye anomalies, polydactylism and polycystic renal disease (NRC 1975, Appendix VI, Appendix I).

X-linked disorders act as dominants in the male population and as recessives in females. This type of mutation causes disorders such as hemophilia, albinism, and hydrocephalus (Evans, Moeller and Cooper 1985, Vol. 2, Appendix 3E).

Chromosome aberrations and anomalies are the third major category of genetic effect considered in this study. Aberrations involving unbalanced translocation of chromosomes generally result in severe congenital malformation or retardation. Chromosome anomalies, either missing or additional chromosomes, result in a variety of syndromes including Down's, Edward's and Hunter's.

1.2.2 Relationship of Health Effects to Costs

Acute radiation injuries and cancers affecting the exposed population, and genetic damage affecting future generations may each result in premature mortality, as well as morbidity (illness) and physical impairments, but the effects are likely to occur at different points in time. Most types of acute radiation injuries would become apparent within a few weeks of exposure and the resulting fatalities would generally occur within six months. With a few important exceptions such as leukemia, cancers would not be apparent until ten to fifteen years after radiation exposure and incidence might be spread over the remaining lifetime of the affected population. Genetic effects would occur in the offspring of the exposed population and then diminish in frequency over subsequent generations. As a result of the delayed impact of genetic effects, the economic costs of radiation-induced health effects could be spread over a substantial period of time. While secondary, nonmonetary, costs would also be associated with the health effects, they are not estimated in this study.

Although the details are complex, the basic process by which health effects result in economic costs is shown in its simplest form in Figure 1.1.

FIGURE 1.1. Diagram of Radiation-Induced Health Effects and Resultant Social Costs

The starting point is a population that has accidentally been exposed (1) to a source of radiation at some point in time. Depending on the dose received, and the dose rate, individuals may develop acute radiation injuries (2) of varying severity. If symptoms develop, society incurs direct costs for the treatment of the illness and indirect costs due to the decreased productivity (3) of the stricken individual. Those individuals for whom treatment is ineffective die (4) resulting in additional indirect costs (5) to society from the premature loss of their productive capacity.

Those who survive the radiation injuries, as well as those who were uninjured, may develop cancer (6) at some time after the latency period. Both direct costs for treatment and indirect costs due to lost work (7) are incurred by society as a result of the cancers. For those who succumb to cancer (8) there are additional indirect costs (9) of productivity loss due to their premature mortality.

The portion of an exposed population that is unaffected by, or survives, radiation injuries also would face the risk of bearing offspring with genetic disorders (10). Health impairment due to these genetic effects could result in direct costs for medical treatment and indirect costs due to reduced productivity (11) of the affected individuals and the families who care for them. Indirect costs may also result from early mortality due to genetic effects (12). The genetic effects and their economic costs may occur over several generations.

1.2.3 Composition of Costs

The value of avoiding radiation-associated illness can be measured conceptually in two different ways: by estimating the value that the public places on decreasing risks to health and safety, or by measuring the costs associated with higher levels of risk. A review of the relative merits of the two approaches is included in Chapter 3. The PNL health effects model focuses on monetary costs because they are more directly measurable and because they may account for a substantial part of the public's valuation of risk.

There are two basic approaches to estimating the cost of illness: a prevalence or an incidence perspective. Of the two, the prevalence approach is much more commonly employed. This is due both to its applications in budget allocation decisions and to the nature of available data. Essentially, a prevalence approach asks, "How much is an illness, e.g., cervical cancer, costing U.S. society annually?" It sums the costs in a given year of all cases of an illness, regardless of the cause or the timing of disease onset. In contrast, an incidence approach would focus on the question, "If a specified event occurred in a given year, what would be the resulting cost of induced cases of cervical cancer?" This approach permits evaluation of the benefits of changing the rate of development of new cases of disease. Because of its relevance to analyses involving changes in risk levels, the incidence approach is employed by PNL in estimating the costs of radiation-induced health effects.

The economic costs of illness represent both the value of resources consumed in diagnosing, treating, and adapting lifestyles to the illness, and the

value of goods that do not get produced because of morbidity or premature mortality from the illness. It has been the convention in health economics studies to label the consumed resources the direct costs of illness, the forgone production the indirect costs. Both the direct and indirect costs are measured in dollars. In addition to the economic costs, there are associated with illness and death a variety of social effects that constitute intangible costs (see Abt 1975 for an attempt to assess these costs). These elements of total social cost, such as pain and suffering, are not included in economic cost measures, since they do not divert resources from other productive uses, are difficult to quantify and do not have an observable value in the marketplace. However, it is clear that they are an important and appropriate matter of concern to the public in considering illness risks.

Direct costs are measurable in terms of monetary outlays both for health care and for other goods and services made necessary by the illness. Thus, direct costs include the costs of health care services on both an inpatient and outpatient basis for diagnosis and treatment. In addition, a full accounting of direct costs would include expenditures for such things as treatment-related travel and modification of housing (a wheelchair ramp, for example) and for population screening for illness. Unfortunately, the literature includes little information on the quantity of resources consumed, so nonmedical direct costs are not included in PNL's cost model. Direct costs of health care may represent a stream of outlays over a period of years. In this study, future streams of direct costs are represented in terms of their present value in the year of radiation exposure.

Indirect costs involve no monetary outlays, but reflect instead the value of lost productivity due to illness. Productivity losses may occur because the patient is too ill to work, the family is caring for the patient (and they are therefore unable to work), the patient's functioning is permanently impaired, or the patient dies at a younger age than would have been likely without the radiation-induced illness. In any of those cases society loses the goods and services that would have been produced had the patient (or family) been able to work. Estimating values of these productivity losses is similar to valuing current capital investments in terms of future output and is therefore generally known as a "human capital" approach to measuring indirect costs.

PNL employs the human capital approach in valuing indirect costs and includes among those costs the total present value of production lost because of radiation-related morbidity and mortality. One variant of the human capital approach includes among indirect costs only forgone net production; that is, the value of the person's production less his or her future consumption (see for example, Weisbrod 1961). Estimated in this way, net production measures only the value that the rest of society places on someone's life and ignores the value that that person derives from his or her own personal consumption, where personal consumption is understood to encompass all human mental and sensory phenomena, including those not resulting from marketplace transactions. The total production approach adopted by PNL comes closer to a full measure of the indirect costs in terms of human capital losses. However, this approach addresses only marketplace transactions. Furthermore, since marketplace transactions generally have a value to individuals in excess of the monetary

expenditure involved, this approach underestimates the value derived from marketplace transactions. Valuation of the extra market phenomena was beyond the scope of this project. (A discussion of alternative approaches to measuring indirect costs is provided in Chapter 7.)

Assuming workers are paid the value of their marginal product, the value of lost production is equal to the value of forgone future earnings. Following an incidence approach, as is employed for direct costs, indirect costs are measured in terms of the present value of each individual's forgone earnings.

In estimating the costs of health effects, we assume that in the event of population exposure, the change in demand for health care services would not be sufficient to affect the price structure. A similar assumption is made in regard to indirect costs, that the numbers of fatalities involved would be insufficient to affect wage rates or prices. Thus, only small, or marginal changes within our economic system are considered in estimating health effects costs.

1.3 MODELING APPROACH

The cost estimates by HECOM are expressed in real, or constant dollars, excluding strictly inflationary changes in costs. As a result of this approach, the cost estimates reflect comparable real resource costs regardless of the future year in which the costs may be incurred. All future costs are discounted to the base year of analysis so that the resulting HECOM estimates reflect the present value of costs that may actually be incurred in the future.

Because this cost simulation has been undertaken for analysis of future health risks, it is probabilistic in nature. Cost components are probabilistically applied to the age and sex cohorts of a population at risk for health effects. This means that cost projections are weighted by the normal probability of survival for each cohort as well as the age- and sex-related induced probabilities of health effects. In the case of cancer and genetic effects, costs are also probabilistically distributed over time. As a result of the approach taken, HECOM can project the societal impacts of health effects for which timing and population incidence are indeterminate.

1.3.1 Flexibility

HECOM is modular in structure and is designed for flexibility and ease in modifying and updating. It is expected that HECOM can be readily adapted to future changes in models for projecting health effects.

The modular construction enables a user to avoid gathering and using input data for calculations that are not of interest. For example, a user may wish to analyze the costs associated with treating radiation-induced cancers. The model's modular structure permits skipping the calculation of both direct and indirect costs for radiation injuries and genetic effects. Only those steps

essential to calculating the direct and indirect costs of cancers must be performed and only the data essential for performing these calculations are needed.

HECOM is designed to run with age and income data for user-specified time intervals. The data may be annual, or may cover five or ten year intervals, for instance. This allows HECOM to be run with available data at any level of aggregation.

The major HECOM modules relate to medical management of an exposed population, to early injuries, to cancers and to genetic effects. Each of these modules can be run separately or in combination with any of the others to project direct and indirect costs. In addition, HECOM can be run with a file of health effect incidence data to produce complementary cumulative distribution functions (CCDFs). Additional analytical capabilities are made available by altering various categories of input data. HECOM accepts health effect related output data from the CRAC (Calculations of Reactor Accident Consequences) Model and the MACCS (MELCOR Accident Consequence Calculation) Model and may be tied to either of these models.

Execution of HECOM is controlled by several parameters that define the number of years of costs to process, the types of cancers, radiation injuries and genetic effects to be included in cost calculations, and the number of age categories and sexes defined in the input data. The value of each of these parameters can be specified by the user. The input data file can be easily modified to alter various economic (i.e., income growth rates), demographic (i.e., cumulative life probabilities, labor force participation rates and population fractions) and health effects data. This enables a user to easily run different scenarios and thereby develop a range of estimated health effects costs.

1.3.2 Analytical Capabilities

One of HECOM's major applications is in siting analyses or site-specific safety evaluations. To carry out such analyses, a basic set of assumptions is needed regarding economic conditions, population distribution and characteristics, and treatment costs. Once these are entered in the input data file for a specific site, the user need only provide alternative scenarios of health effect incidence to estimate the economic costs associated with various risk levels. By changing only the data in the health effects input file, the user can estimate the marginal health-related benefit of reducing risk to alternative levels.

Clinical and Cost Information

HECOM's input file contains data that control the time periods over which health effect incidence is allocated, the method of incidence allocation and the direct costs. Subroutine MONITOR, for example, can provide cost estimates for alternative patient allocation and transportation plans. Data for hospital capacities, transportation distances and costs for each mode of transportation can be varied to suit the desired analysis. Similar flexibility is possible in

projecting population screening and decontamination or clinical monitoring costs under different scenarios of population management after exposure.

Various types of analyses related to radiation injuries are also possible. Radiation injuries are divided into several categories. The first category is injuries that are evidenced by clinical symptoms resulting in either death or recovery within the first year. Second, there are prenatal injuries resulting in lifetime treatment costs. Third, there are delayed symptoms requiring one-time treatment and, fourth, there are delayed symptoms requiring lifetime treatment. Within these categories, assumptions related to latency periods, or disability rates or costs may be varied. Additional types of injuries may be analyzed by HECOM simply by adding them to the appropriate category and supplying the requisite input data elements. (This is true for cancers and genetic effects as well.)

In the cancer module the rates at which cohorts are affected over time can be determined by either a relative or an absolute risk model. In addition, assumptions regarding latency periods, periods of risk, median survival time, treatment period and treatment cost can all be varied by the user.

In subroutine GENETIC the effects on costs of uncertainty in the projection of genetic effects can be analyzed by supplying alternative input data for the number of generations modeled and for effect elimination rates. Clinical assumptions such as incidence by sex cohort, period of impairment, disability rate, and period of life shortening can be varied. Initial and annual treatment costs are also variable inputs.

Economic Conditions

HECOM calculates health effect costs in "real," meaning inflation-free, terms. All cost outputs represent present values in the year's dollars of the input data. Cost estimates for the effects of acute radiation exposure in any given future year can be obtained by changing the base year of analysis in the input data file. The discount rate used in the analysis is also a variable, as are the real rates of change in treatment costs and income. Thus the parameters that simulate changes in real cost levels can be set to reflect expected long-term economic conditions.

Population Characteristics

HECOM can be used in generic analyses using data for typical U.S. population characteristics, site-specific studies using regional data, or analyses of subgroups such as utility employees. The model requires input data for population age and sex structure, labor force participation rates, income levels, and median ages for the age categories selected. The number of age cohorts included and the age interval covered by the data may be tailored to the desired analysis. By changing the input data, HECOM can be used in projecting health effect costs for ethnic or occupational subgroups or regional populations.

1.4 REPORT STRUCTURE

This report presents the conceptual and informational base from which PNL has developed health effects cost estimates. The methodology employed in estimating each component of these costs is described in detail. In addition, description and documentation are provided for the HECOM model developed to calculate the present value of possible future health effects costs. Conclusions and recommendations of the effort are presented in Chapter 2. This includes a discussion of the limitations of the cost estimates, the relative importance of the major cost components, and recommendations for further research.

Though the estimation of health effects costs is difficult, the difficulties stem mainly from incomplete medical and economic data and information, rather than from inadequacy of the conceptual basis for such cost estimates. Chapter 3 presents the conceptual basis and discusses the two major approaches to measurement of health effects costs: the individual preference approach and the human capital approach. Because of its greater tractability, the human capital approach has been employed in developing cost estimates.

In Chapter 4 we develop direct cost estimates for care of an exposed population and for early radiation injuries. Costs for medical management of a population are discussed first. These cover the activities involved in personal decontamination, identification of the injured, and transportation of the injured to treatment facilities. The remaining sections of Chapter 4 present the cost estimation procedures for gastrointestinal syndrome, hematopoietic syndrome, pulmonary impairment, prenatal injury, thyroid disorders, skin damage, cataracts and sterility.

Estimates for direct costs of cancers are developed in Chapter 5. Major aspects of projecting cancer incidence including models of cancer risk and the duration of latency periods are presented first. This is followed by presentation of the cost data and cost estimation methodology.

Genetic effect direct costs are the subject of Chapter 6. The first section presents descriptions of major categories of genetic damage and indicates the types of health impacts that may result. Remaining sections present the data and cost estimation methods.

The basis for calculating indirect costs is discussed in Chapter 7. Earnings losses associated with illness are the subject of Section 7.1 and losses due to early mortality are covered in Section 7.2. The cost estimation methodology for lost production is the same for each type of health effect.

An overview of HECOM structure is provided in Chapter 8. The utility sub-routines are presented first. The handling of population and earnings projections and of the time value of money is then discussed. The model structure is then presented as it pertains to each major health effect cost category.

Chapter 9 presents a sensitivity analysis of major parameters affecting the HECOM health effects cost estimates. Appendix A contains summaries and an explanation of the data used in the base case. Appendix B is a listing of the HECOM computer program.

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2.0 CONCLUSIONS

Preliminary conclusions from the cost estimation effort are presented below. An overview of the accomplishments in this first attempt to rigorously estimate health effects costs is presented in Section 2.1. The scope and focus of the study are indicated and some of the limitations are explained. Section 2.2 describes the sensitivity of HECOM cost estimates to major economic assumptions, apart from the uncertainty in the estimated numbers of health effects that are input to HECOM. The estimated ranges of costs for each type of health effect are then presented in Section 2.3. This is followed by suggestions for further research in regard to refinement of cost estimates and application of HECOM to risk analyses.

2.1 ACCOMPLISHMENTS

To improve the quantitative information used in evaluating actions that alter health risks, this study and the resulting health effects costs model (HECOM) provide estimates of the economic costs of the principal types of radiation-induced health effects. The study presents the conceptual basis for measuring direct and indirect economic costs and it describes in some detail likely medical treatment of radiation-related health impairments. PNL's cost model, HECOM, calculates the present dollar value of resources that would be consumed in treating radiation-induced health effects and the resources that would not be produced because of exposure-related morbidity and early mortality.

HECOM is a flexible computer code that uses health effects incidence input data to project the direct and indirect costs of acute radiation injuries and fatalities, cancers and genetic effects. HECOM allows for varying key parameters of the cost projection; by changing input data, the model can analyze effects of changes in incidence estimates, treatment costs, the discount rate, etc. Modules within HECOM can be run separately or in any combination permitting specialized analyses. In addition, health effect types may be deleted from or added to the modules. Because of its flexibility, HECOM will be adaptable over time as information improves regarding risks, treatment regimens and costs.

Use of HECOM estimates requires a clear understanding of the model's focus. Two general points are important in this regard: first, the model currently includes only the major forms of potential radiation-induced health impairments and second, the model centers on health effects costs and not on society's valuation of risk to life and health. HECOM calculates costs for acute radiation injuries and fatalities, cancers, and genetic disorders. However, it leaves uncounted other potential effects that may be important considerations to the public, such as psychological stress and sterility. The HECOM cost estimates do not measure the total value of life or health but only the value of resources that would be used or not produced because of ill health or early mortality.

The economic cost figures obtained from HECOM are useful as rigorous and documentable cost estimates for health effects potentially associated with population exposure to ionizing radiation. They constitute heretofore unavailable information that is appropriate for use in value-impact analyses and environmental impact statements for nuclear facility siting. While there is room for refinement of the health cost estimates, they provide an indication of the relative magnitude of health effects costs for use in regulatory decision making.

2.2 SENSITIVITY OF HEALTH EFFECTS COSTS

There is considerable uncertainty in the health effects incidence estimates that are currently available for input to HECOM for cost calculation. In addition, there is uncertainty regarding the distribution of cancers and genetic effects over time. In regard to genetic effects, there is also uncertainty as to the frequency of defects of various degrees of severity. Available information is inadequate for estimating the degree of uncertainty in incidence estimates. Compared with these uncertainties, there is considerably less uncertainty regarding the direct and indirect cost estimates we have developed for radiation injuries, cancers and genetic effects.

2.3 MAGNITUDE OF HEALTH EFFECTS COSTS

Since a probabilistic methodology was used in developing HECOM, the resulting health effects cost estimates do not represent the costs for any particular individual. Rather, the HECOM cost estimates are representative of costs for a population with a specified age and sex distribution, for whom both health effects risks and resulting costs vary with age, sex and other factors. The cost estimates for cancers and genetic effects are based on distributions of incidence and associated costs over long time periods. These cost estimates should not be confused with the average cost of a cancer or genetic effect occurring at any specific future time; they are statistical constructs that weight the probability and magnitude of costs in each year of the period modeled by HECOM and discount this stream to a base year. It is this characteristic of the HECOM estimates that makes them most suitable for use in evaluating changes in health effects risks.

Estimates from the HECOM base case are shown in Table 2.1 where direct, indirect and total costs are listed for acute radiation fatalities and injuries, cancers and genetic effects. The present-value cost estimates in Table 2.1 are for one case of each type of health effect probabilistically distributed over an exposed population and over time. Because the costs cover such a wide range due to the underlying variation in health effects severity (such as the difference between prodromal symptoms and prenatal injuries), an average cost for each health effect type (i.e., radiation injuries) would not be representative of the cost distributions.

For radiation injuries the total direct and indirect costs range from those for thyroiditis and transepithelial injuries (\$0.4K), through those for

TABLE 2.1. HECOM Present-Value^(a) Cost Estimates Per Radiation Injury, Cancer or Genetic Effect (1984 \$)

	<u>Direct Cost</u>	<u>Indirect Cost</u>	<u>Total Cost</u>
<u>Radiation Injuries</u>			
Prodromal Symptoms	1,500	200	1,700
Gastrointestinal Syndrome	28,000	128,500	156,500
Hematopoietic Syndrome	56,000	128,500	184,500
Pulmonary Syndrome	8,500	57,500	66,000
Prenatal Fatality	100	212,300	212,400
Thyroiditis	200	200	400
Transepithelial Injury	200	200	400
Hypothyroidism	2,400	100	2,500
Cataracts	3,200	100	3,300
Microcephaly	237,900	106,100	344,000
Mental Retardation	452,000	201,700	653,700
<u>Cancers</u>			
Leukemia	15,500	82,500	98,000
Lung	9,200	11,600	20,800
Breast	5,500	12,400	17,900
Bone	18,600	80,400	99,000
Skin	900	100	1,000
Gastrointestinal	9,000	8,400	17,400
All others	8,500	10,600	19,100
Thyroid	4,800	22,700	27,500
Thyroid - benign	1,100	100	1,200
In-utero leukemia	18,000	216,100	234,100
In-utero other	16,000	216,100	232,100
<u>Genetic Effects</u>			
Autosomal dominant	21,500	18,600	40,100
X-linked	58,500	53,100	111,600
Unbalanced translocation	99,200	76,600	175,800
Aneuploid	145,100	94,900	240,000

(a) Using a 4% discount rate, 1% earnings growth rate, and 2% treatment cost growth rate.

hematopoietic syndrome (\$184.5K), to the lifetime costs of prenatal injuries that are as high as \$653.7K per case. The extreme cost level of prenatal injuries results from the combined costs of lifetime care and loss of potential productivity. Since each radiation injury is qualitatively different in nature, as well as in costs, they are best considered as separate categories of effects.

Cancer costs cover a narrower range, from those for nonfatal thyroid nodules and skin cancers (\$1K and \$1.2K) to those for cancers caused by

in-utero exposures (\$232.1K and \$234.1K). The indirect costs of the latter are substantially higher than those of other cancers, due to the loss of all the affected individual's earning potential.

The cost estimates for genetic effects range from \$40.1K to \$240K. These estimates may be interpreted as the value of avoiding the risk of one individual's health impairment due to a genetic effect that would occur within the subsequent generations. Serious and minor effects are weighted in estimating the genetic effects costs so that the estimates apply to each broad category of genetic effects.

The health effects cost estimates given above may be applied to numbers of specific types of cancers or injuries (e.g., leukemia, prodromal symptoms) to evaluate total health effects costs for an affected population. Ways in which the above cost estimates could be improved are discussed below.

Concerning radiation injuries, there is uncertainty regarding the sensitivity of both mortality rates and costs to variations in the level of medical care provided. The Reactor Safety Study (NRC 1975) suggests that the lethality of radiation exposure can be avoided to an extent by sufficiently intensive levels of medical care. Currently HECOM applies the cost of "supportive" care in a well-equipped medical center to all bone marrow and gastrointestinal injuries. However, it does not treat the costs or the mortality implications of either minimal or heroic treatment. Emergency planning efforts could benefit from examination of the cost effects that would stem from the difference in mortality rates associated with various types of medical care.

An important next step is the application of HECOM to examples of hypothetical reactor accidents. The current output from the model shows the richness of information that can be obtained. In addition, application of the model may be expected to lend a new empirical basis to the enduring policy question concerning what safety levels should be required to prevent accidental irradiation.

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3.0 VALUING CHANGES IN HEALTH RISKS

Among the risks of exposure to acute radiation doses are increased illness and a lowered life expectancy. That is, compared with statistical norms, an exposed population faces a risk of higher morbidity rates and of excess (i.e., earlier) mortality. People are generally averse to risk: a decrease in risk is considered good; an increase in risk, bad. Concentrating, for simplicity, on the issue of excess mortality, this section provides a discussion of the difficult problem of evaluating (in dollar terms) the value of changes in risk levels.

It should be emphasized that the effort here is to evaluate an incremental change in risk, not to put a value on human life. Two general approaches have been followed to measure the cost of increased risk: measuring individual preferences for alternative risk levels and measuring consequences of risk on the value of human capital. A description of these general approaches is provided in Sections 3.1 and 3.2, along with an analysis of how comprehensive each is in capturing each of the components of the total cost of risk.

The total value to society of attaining a given risk level stems from individual preferences. There are at least five reasons why someone would prefer a lower societal risk of mortality to a higher one. The first three stem from valuing life per se:

1. If a lower societal risk level means the individual is at lower risk, he prefers that state of lower risk. Call the value of his preference in regard to his own life v1.
2. If a lower societal risk level means the individual's loved ones are at lower risk, he prefers that state. Call the value of his preference in regard to loved ones v2.
3. Even if neither the individual nor his loved ones benefit, he prefers a lower risk for other (anonymous) people purely out of beneficence. Call that v3.

Aside from beneficence or valuing life per se:

4. The individual would value a lower risk to anonymous others because it means a lower risk to benefits he derives from their net production. Call that v4.
5. The individual prefers lower risk because he values the resources that would otherwise be consumed in treating illness or in trying to avoid death. Call that v5.

These five components of the value society places on changes in risk levels are employed in the following discussion of risk valuation methods.

They are used to illustrate the extent to which each method captures the major aspects of society's valuation of changes in risk.

3.1 THE "HUMAN CAPITAL" APPROACH

The extent to which society is willing to forgo current consumption and to invest in productive plant and equipment depends on the value of the resulting output. That is, we value physical capital in terms of the goods and services produced with it. Similarly, we may value human capital in terms of the value of goods and services produced by labor. So a risk of losing productive years of labor, through increased morbidity and lower life expectancy, is also a risk of losing the value of the goods and services produced by that labor. Assuming that the value of the marginal product of labor is equal to the wages paid for that labor, lost wages (including the equivalent value of self-employment) are a measure of the value of health risk.

To employ the human capital approach, the cost of health risk is computed by multiplying a measure of the value of human capital by the change in the probabilistic risk of death. For example, consider an individual who expects to earn a discounted total of \$100,000 over his remaining life-time. That expectation depends to an extent on his life expectancy: he has some discrete probability of dying in each year. The level of his expected future earnings reflects both future wage levels and the probability of death in each subsequent year. Suppose a reactor accident imposes on that individual an increased probability of death every year in the future; now his risk-weighted, expected future earnings are, say, only \$90,000. Then the cost of the risk to that individual is estimated to be \$10,000 discounted to present value in the year of the accident.

With the human capital approach, both the increased level of risk and the value of the human capital at risk are considered. Each of the principal variants to human capital valuation is discussed briefly in the following paragraphs.

One commonly used measure of human capital is the share of each person's net production at risk of being lost to society, given risks of increased morbidity or early mortality. The value of a person's net production is the value of his or her total production (as measured by total earnings) less the value of what he or she consumes. It is a measure of the value of goods and services a person "gives" to society, over and above what he or she "takes away" through personal consumption. Weisbrod (1961) proposed this as the appropriate measure of human capital at risk.

This "net production" measure, however, evaluates only one component of the total value at risk; it corresponds only to the value v4 of the components listed at the beginning of this section. It ignores completely the value the individual places on the risk to his or her own life. And even from society's viewpoint, it takes no account of the beneficence that makes us prefer a lower risk to the lives of those whose net product is negative (that is, who consume more than they produce).

A more comprehensive approach to human capital valuation measures the value of total production, including personal consumption. This is the approach taken in cost of illness studies over the past twenty years by Rice and her associates (Rice 1966; Cooper and Rice 1976; Hodgson and Rice 1982). These studies compute the value of human capital from the total average earned income for a person in an age and sex cohort at risk. The total value of a loss from early mortality is measured over the period between the age at death and the year of normal life expectancy, and is equal to the present value of the stream of lost earnings (i.e., lost production). For Rice's purpose of estimating the annual cost of illness it is appropriate to discount this stream to its present value in the year of death; to apply such costs to a decision that affects risk (e.g., the risk of a reactor accident), it is appropriate to discount to present value for the year in which resources would be committed. The latter approach is followed by Hartunian, Smart and Thompson (1981) and by PNL in this study.

Further refinements are often made to both the total and net production measures, especially to account in different ways for the human capital of the nonwage-earning population. Since available data on earnings do not include values for nonmarket production, the value of household services, for example, must be imputed if the value of women's (and some men's) production is not to be significantly understated. (This is also true for other types of nonwage-earning labor, such as volunteer services, but data to carry it out are lacking.) Imputed values may be based on the market value of domestic services (Brody 1975) or on the opportunity cost principle, accounting for wages that could be earned in the marketplace as an alternative use of the homemaker's productive time (Prest and Turvey 1965). A problem with the latter approach is that it is difficult to determine likely wages that could be earned in the marketplace if a large number of homemakers not currently in the labor market entered it. Besides, the wage that could be earned in the market is, by observation, insufficient to reward the household for giving up the homemaker's services (Gronau 1973). In spite of the problems with the opportunity cost approach, we employ a modification of it in this study for practical ease of calculation. We compute the mean earnings of noninstitutionalized, wage-earning individuals in each age and sex cohort and apply that figure to all individuals in the cohort.

When refined to include an imputed value for household labor by those who are not otherwise employed, the measurement of human capital in terms of total production captures both net production (v4) and also some portion of v1, the value an individual places on a risk to his or her own life. This assumes that the dollar value of consumption is a rough measure of the satisfaction a person will receive out of life. Thus, an approximation of v1 is provided by the value of the person's future consumption.

The value of personal consumption is usually considered an underestimate of v1. In an argument requiring some theoretical rigor, Schulze et al. (1979) have shown that the principle of "risk aversion" is one reason why the value of consumption understates v1. In addition, consumers are often willing to pay more for a good than they actually end up paying in the market; therefore, they

get more satisfaction than is represented by the price they pay. Thus, expenditures on future consumption generally understate v1. Finally, personal consumption is understood here to include all human mental and sensory phenomena. To the extent that total production includes only those phenomena that result from market transactions, v1 will be underestimated.

The value a person places on his or her own life (v1) is an elusive measure. It is not constant over various risk levels; it varies among individuals; and for each individual it varies with circumstances and over time. Therefore, it is unclear just how much of v1 is measured by total production. Nevertheless, given the practical considerations of obtaining an estimate, in this study we add the value of direct treatment costs to the value of total production to develop an estimate of the total value at risk that includes v4, v5, and some measure of v1.

A number of problems with the human capital approach are evident, both in terms of particular methodological difficulties and more generally in terms of theoretical shortcomings. Particular methodological problems include the tendency of the approach to value risk to life based on earnings; those who have low earnings tend to be assigned low values (Mushkin and Dunlop 1979, p. 6). Mushkin and Dunlop list other problems involved in human capital valuation: changing trends in workforce participation rates at different ages and for males and females, changes in productivity growth rates, and changes in earning patterns over a working life (1979, p. 6).

Aside from the methodological problems, significant challenges have been raised against using the human capital approach in risk valuation on grounds of incompatibility with economic theory. Neoclassical economists are uniformly in agreement that a measure of human capital simply has no place in cost-benefit analysis. (See for example Mishan [1971].) Instead of using human capital, the benefits of a particular project should be measured in terms of individual preferences, according to economic theory.

In summary, for reasons both of problematic details in the valuation of human capital, and because of that approach's theoretical shortcomings, many economists have urged that risk to longevity be measured in terms of the value of individual preferences. (For general descriptions of the theoretical support for measuring individual preferences and for comparison of this approach with human capital valuation, see Schelling 1968; Mishan 1971; Acton 1973; Zeckhauser 1975; Jones-Lee 1976; Willig 1976; Rhoads 1978; Clarke 1979; Dorfman 1979; and Weinstein, Shepard and Pliskin 1980.)

3.2 THE "INDIVIDUAL PREFERENCE" APPROACH

When the total costs are accounted for, the introduction of a particular project (e.g., a project that lowers risk from a reactor accident) will make some members of the public better off, some worse off, and others will be indifferent (no change in well-being) to the change. For example, an investment in safety equipment may decrease public risk but require increased worker exposures. If in the aggregate, the total value of individual preferences

regarding the project is positive, there is a potential for improving overall public welfare by going ahead with the project. In the individual preference approach the value of that potential improvement is interpreted as the excess of benefits over costs arising from the introduction of the project. The value of the improvement is measured directly from the preferences of the public. Methods to observe individual preferences are discussed in detail below.

Economic theory suggests that the value of a change in an individual's perceived well-being can be measured by the amount of money the individual would be willing to accept (WTA) or willing to pay (WTP) to remain indifferent to the change. The benefit of a risk-reducing project can best be measured, in theory, by how much the community, in aggregate, would be willing to pay for a decrease in the level of risk, or would be willing to accept to face an increase in the existing level of risk.

Selection of the appropriate measure (WTA or WTP) depends upon the assignment of rights within the affected society. If consumers have a right to a lower-risk state, their willingness-to-accept payment to face a higher risk is the relevant measure. If consumers do not start with the right to a lower risk, then we should measure individuals' willingness to pay to obtain a lower risk. In practice, the distinction between WTP and WTA is often blurred, with the availability of information a more important criterion for the choice of either measure than the distribution of rights.

Among the attempts to evaluate individual preferences, three approaches stand out: measurement of WTP by questioning consumers directly (Acton 1973; Jones-Lee 1976), measurement of WTA by wage differentials paid to workers in risky occupations (Thaler and Rosen 1976), and measurement of WTP by public budgets for life-saving programs (Cohen 1980). We ignore the last here because the factors in a program's success in political battles over budgets do not appear to be directly related to society's valuation of the risks averted by that program.

Acton (1973) describes the use of a questionnaire to elicit willingness-to-pay responses directly from the public. While concerned more with general patterns of responses and with the applicability of the technique than with numerical estimates, Acton concludes that the questionnaire method yields results that are reasonably consistent internally. He finds that when confronted with a hypothetical situation involving risks to themselves, people are generally willing to pay more for larger reductions in risk than for smaller ones (p. 105). He notes also, however, that this relationship is nonlinear, varying directly with the absolute level of risk faced by a respondent. Because people face and perceive different levels of risk, the nonlinearity of responses means a single "willingness-to-pay" measure cannot be expected from such studies (p. 108). Acton reports that his respondents were willing to pay an average of \$43 to reduce annual mortality risk by one death per 1000 people, and \$56 to reduce risk by one death in 500 people (p. 109). (Both figures are in 1971 \$). These values are for risk to a group of which the respondents were members.

It is important to note here that Acton and other investigators of individual preferences measure individuals' valuations of risk directly. These risk valuations are often discussed in the context of "the value of a life." In that use, it is necessary to perform a calculation from the risk value to obtain what Freeman (1979) calls the value of a "statistical life." For example, if the average individual willingness to pay for a program that reduced the mortality rate of a given group from seven deaths per 100,000 to six deaths per 100,000 were \$5, the "value of statistical life" would be \$500,000 (Freeman 1979, p. 168). Thus Acton's results are commonly presented in terms of a "value of life" ranging from \$28,000 to \$43,000, depending on the risk change evaluated. For most policy purposes, however, it is the value of risk that is relevant, not the secondary calculation of value of life.

In an approach generally similar to Acton's, Jones-Lee (1976) also uses a questionnaire to elicit willingness-to-pay information. Posing a hypothetical situation in which the respondents themselves are at risk, Jones-Lee finds that, effective over relatively short periods of time, the average reported value of a decrease in risk of one death per 500,000 people is about 6 pounds sterling (1975£) (or about \$10 at 1975 exchange rates).

Thaler and Rosen (1976) seek a measure of willingness to accept (WTA) in an alternative to the questionnaire approach. They reason that the wage differentials paid to individuals in high-risk industries constitute a measure of those individuals' valuation of risk. This assumes, of course, that such individuals are fully informed of the risks they face and have less risky employment alternatives. Controlling for a variety of nonrisk-related characteristics of laborers, Thaler and Rosen present four equations that yield risk valuation estimates in a range from \$136 to \$260 (in 1967 \$) for reducing risk from one death per 1000 people to zero.

Just as the human capital approach can be faulted for ignoring certain components of the cost of risk, so can the empirical studies undertaken to measure individual preferences. The risk values reported by Acton correspond only to v1, the value an individual places on risk to his or her own life. Acton attempts measurement of v2, an individual's valuation of risk to loved ones, but does not quantify the responses in dollar terms.

Jones-Lee (1976) suggests that v4, the risk of losing a share of net production, and v5, the risk of having to share in treatment costs, should be added to v1 for a full valuation of the cost of risk. He acknowledges that he has not accounted for v2, the value put on a loved one's life, and he ignores altogether what we have labeled v3, the preference for lower risk stemming purely from beneficence.

Kneese and Schulze (1977) employ Thaler and Rosen's high estimate in a rough approximation of the costs of cancer associated with selected environmental hazards. However, they reason that even that high estimate is "probably seriously biased downward." They argue first that workers in risky jobs are less risk averse than the general population, and therefore accept risk at a lower wage differential. Second, they suggest that people may be more willing to take risks voluntarily than to have risks imposed externally. To the extent

that risks from environmental carcinogens are accepted involuntarily, people may demand more compensation for that acceptance. Finally, they argue that job-associated death risks may not entail the particularly unpleasant pain and suffering of cancers, for which people would seek higher compensation (Kneese and Schulze 1977, p. 331).

Neither wage differentials, as used by Thaler and Rosen for a measure of WTA, nor other similar marketplace valuations are capable of including values other than v1, an individual's concern for risk to his or her own life. Thaler and Rosen concentrate only on v1.

Table 3.1 provides a summary of the value components measured by each of the approaches assessed in this section. As can be seen in Table 3.1, none of the approaches quantifies adequately all of the components of risk value.

Depending on the age, sex, and kinship relationship of the person(s) being considered, Needleman (1976) suggests adding to v1 a value ranging between 25 and 100 percent of v1 to account for v2. If any value were added for v3 in that scheme it would be less than 25 percent of v1.

TABLE 3.1. Extent to Which Selected Methods Measure the Various Components of Value

Methods	<u>v1</u>	<u>v2</u>	<u>v3</u>	<u>v4</u>	<u>v5</u>
Human Capital	Partial	0	0	Full	0
Human Capital plus Direct Costs ^(a)	Partial	0	0	Full	Full
Acton	Full	(b)	0	0	0
Jones-Lee	Full	(b)	0	(c)	(c)
Thaler-Rosen	Partial ^(d)	0	0	0	0

(a) Approach taken in this study.

(b) This component is considered, but not quantified.

(c) Addition of this component is recommended, but the study does not attempt it.

(d) The critique of Kneese and Schulze (1977) indicates several reasons why this wage differential measure may understate v1.

That still leaves the question of whether the other components could be appropriately added together. Perhaps, as Jones-Lee suggests, one may add WTP or WTA to other component values of risk costs. However, that approach is neither practicable nor desirable in the present PNL effort.

3.3 CONCLUSIONS

The human capital approach is not ideal; it measures only a portion of the probable "true" value of risk reduction. And it measures that portion in a way inconsistent with certain principles of economic theory.

However, the individual preference approach, while firmly rooted in economic theory, is difficult and costly to implement. Mishan (1971) suggests that a "contingent market" study (i.e., measurement through surveys) is a proper vehicle for measuring WTP or WTA. Cronin (1982) shows, however, that such studies must be rigorously designed to avoid several kinds of respondent bias. While such an approach may be implemented in the future, no broadly based studies are presently available.

The valuation of individual preferences through WTP or WTA depends to a significant degree on how the risk valuation question is asked, on the perceived risk levels, and on the pain and suffering expected. (See Currie and Kidd [1980] for a demonstration of how WTP and WTA values may vary depending on how the question is asked.) It is not appropriate, therefore, simply to transfer a WTP or WTA estimate from one study to another. Instead, it would be necessary to perform a special survey to explore individual preferences regarding the risks of radiation-associated morbidity and mortality. And it would still be useful to pursue both the human capital valuation and the direct cost valuation for risk-weighted measures of v4 and v5, respectively, to provide a baseline.

To gain an understanding of the magnitude of the value of risk reduction with minimum investment, the human capital approach is adopted in this study. A contingent market survey would offer greater potential for a full valuation of health effects risks but it could be implemented only after substantial investment in survey design and testing.

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4.0 DIRECT COST ESTIMATION FOR EARLY RADIATION INJURIES

If one measures the values of life and livelihood by the human capital approach, an accounting of the direct costs of treating an illness is necessary to measure the total benefit achievable by risk reduction. Conceptually, in a consumer's response that he is willing to pay \$X for some risk-reducing program, there is implied both a value of life and limb and an assessment of the actual monetary outlays he will face if the risk is not reduced. Since the consumer is unlikely to know the total value of the monetary outlays, the questioner should be expected to provide an estimate. Thus even in a willingness-to-pay approach, an estimate of actual outlays (direct costs) is necessary.

Direct costs of radiation-induced health effects include all of the costs of hospitalization, physicians' care, drugs, nursing, special equipment, transportation required for medical treatment, medical supplies, etc. Regardless of whether these costs are paid by individuals, private insurance, or government programs, or represent bad debts that are paid indirectly by other users of medical services, they involve costs to society for medical treatment and should be counted.

The rest of this section describes the bases for developing direct cost estimates for radiation injuries, including the hematopoietic, gastrointestinal, and pulmonary syndromes; thyroid disorders; cataracts; skin damage; sterility; and prenatal injuries. We review information about the incidence and treatment of the main categories of early radiation injuries to provide the basis for cost estimates. This includes experimental and epidemiological studies of dose and effect relationships, information on the clinical symptoms associated with each type of illness, and the treatments likely to be required for each. Cases of radiation injuries such as acute radiation syndrome have occurred too infrequently for specific cost estimates to be readily available. However, specialists in radiation medicine have reached relatively close agreement about the clinical manifestations of radiation illness. Given the clinical information, we estimate the costs of treatment from the cost of treating patients with similar clinical symptoms. From this information estimates are developed for the direct costs of treating each type of injury.

Depending on dose levels and on individual sensitivities, exposure to significant amounts of radiation may result in acute symptoms that could range from mild nausea to death. Treatment required for recovery may range from a few days of bed rest at home to heroic intervention in a well-equipped regional medical center. If a large population were accidentally exposed, procedures would be required for decontamination, identification of the injured and provision of medical care. The medical management of an exposed population is discussed in Section 4.1. The following sections cover each of the major injury categories and Section 4.11 provides a summary of estimated costs.

4.1 MEDICAL MANAGEMENT OF AN EXPOSED POPULATION

In the event of a severe reactor accident resulting in release of radionuclides constituting a threat to public health, the immediate goal would be

effective sheltering or evacuation of the population. After the evacuation or sheltering action has been taken, a need for two types of population screening would emerge, both directed toward minimizing health impacts. First, individual radiation surveys to detect possible external and internal contamination would be needed. Once decontamination, if necessary, was carried out, medical screening would be the next priority. This would involve an effort to identify those individuals who, because of location, age, existing illness, etc., may have received a dose sufficient to cause a radiation-related injury. These individuals would require medical testing and monitoring to diagnose the likely severity of the illness and to allow planning for adequate medical care during the acute phase of the illness. If the number of persons with acute radiation illnesses is greater than the capacity of local hospitals for care of such patients, transportation to other facilities would be needed to ensure provision of care that would increase the chances of survival.

4.1.1 Radiation Screening

General procedures for radiation screening, responsible agencies, available personnel, equipment, and facilities are discussed in this section. Since there has not been an accident requiring mass radiation screening (and one is highly unlikely), there is considerable uncertainty about procedural details. Population screening and transportation costs are constructed from the available information.

In the event of an accidental radionuclide release from a nuclear reactor, the responsibility for care of the exposed population lies with state and local governmental units. Before a reactor is licensed for operation by the Nuclear Regulatory Commission (NRC), it must have Federal Emergency Management Agency (FEMA) approval of a plan to coordinate state, local, and utility resources to deliver needed screening and medical care (USNRC and FEMA 1980, Section L). Radiological screening personnel and equipment may be provided by utility employees, by private firms under contract to utilities, and by state and local agencies. Facilities and personnel trained to detect and to care for radiation injuries are generally available at the hospitals closest to nuclear reactors. In addition, access to specialized laboratory and hospital facilities has been contracted for by many utilities (Linnemann and Thiessen 1971).

If the size of the exposed population were to overwhelm the screening resources in a state, federal assistance could be requested. The federal response would be coordinated by FEMA following procedures set out in the Federal Radiological Emergency Response Plan (FEMA 1984). While several federal agencies have responsibilities pertaining to public health, the Department of Energy (DOE) has the major responsibility for radiological monitoring during the emergency phase of an accident (FEMA 1984, p. 35907). Each DOE field office can dispatch a team equipped to assist with population screening. In addition, DOE has personnel and equipment available at its national laboratories, and airplanes and helicopters to facilitate their rapid deployment (NCRP 1982, p. 203).

The first step in the health effect management of a population with suspected exposure to radionuclides consists of physical dosimetry and decontamination. If an individual's proximity to the reactor or the plume makes

contamination highly likely, decontamination would probably take precedence over dosimetry, even though the level of contamination might be very low. His clothing would be removed and bagged; he would be showered or scrubbed and then provided with clean clothing. The physical dosimetry procedures for this group and for those whose exposure is only suspected are similar (ICRP 1978; NCRP 1982).

A survey of the skin for external contamination would be the initial dosimetry step. If evidence of contamination were found, efforts to remove it by washing would be repeated as necessary. Once instrument readings indicated an absence of external contamination, portable instruments could be used to survey for inhalation and thyroid uptake. If indications of internal contamination were found, more precise monitoring with a whole-body counter would probably follow. These instruments are available at many reactors, hospitals, and nuclear laboratories. In 1970, there were whole-body counters in 55 locations around the U.S. (NCRP 1982, p. 56). In addition, there are several mobile counters available from commercial sources.

During the screening process a history should be taken, including location of the individual during the release, existing illnesses, and current symptoms. This, along with the physical evidence, could be critical in determining whether the person should be released or given medical follow-up (ICRP 1978; NCRP 1982).

Many hospitals have designated areas for radiation screening and decontamination. Under emergency conditions the procedures could be carried out in almost any type of facility that had showers and a layout such that one-way traffic and control points could be established. For instance, various types of athletic facilities could be readily adapted for use.

The radiation screening could be carried out by health physics or radiological protection technicians (and others with capability to interpret instrument readings). Individuals with such training would probably be drawn from hospitals, nuclear power plants, and the DOE national laboratories.

4.1.2 Medical Monitoring

Medical monitoring, consisting of laboratory tests and symptom evaluation, is desirable for three major groups of individuals that may be identified in the initial screening process. First, follow-up is recommended for anyone whose exposure is suspected (due to location or dosimetry) to have exceeded 1 Gy (ICRP 1982, p. 16). Second, anyone experiencing nausea or diarrhea should be monitored in case these symptoms are indicative of a significant exposure. Third, people whose pre-existing illnesses make them particularly sensitive to any depression of the immune system should be monitored. This group could include patients with cancer or infectious diseases.

Ideally, monitoring should begin within a few hours after an accidental radiation exposure. Early testing provides a baseline against which changes in blood components during the first 48 hours after exposure can be compared to project the likely course of the radiation illness. For anyone who is likely to experience the hematopoietic or gastrointestinal syndromes, early diagnosis

can increase survival probabilities. It facilitates both treatment of some potential disease manifestations before symptoms develop and placement of the patient in the most appropriate facility (Andrews 1980; NCRP 1982; IAEA 1978). See the document on response to the Chernobyl disaster for a detailed description of Soviet efforts to medically monitor the exposed population.^(a)

Total and differential whole blood counts are needed for medical monitoring. These should be carried out every 3 to 12 hours for 48 hours after exposure to detect changes in granulocytes and lymphocytes. Typically, the granulocytes increase during the first day to a peak that indicates the severity of coming illness and the lymphocytes decrease to a minimum in 2 to 3 days that is also a predictor of illness severity (ICRP 1978, p. 4). Machines suitable for conducting the blood tests have a capacity ranging from 50 to 135 samples per hour and, thus, could handle tests for about 2500 people over a 48-hour period if dedicated to that use. If the patient has an internal exposure, radiochemical assays of excreta can provide useful diagnostic information, so that the treatment can be appropriately tailored to the isotopes involved (NCRP 1982, p. 51).

The monitoring procedures described above are requisites for providing the types of medical care designated "supportive," and "intensive" or "heroic" in health effects assessments (USNRC 1975, Appendix VI, p. F-1,2; Evans, Moeller and Cooper 1985, Vol. 2, p. 19). If the circumstances of a reactor accident were such that radiation screening and medical monitoring were not available to the exposed population until several days afterward, the most severely injured would be more difficult to identify and treatment would have to be determined symptomatically. This would probably result in some decrease in the recovery rate for those with serious but survivable injuries.

4.1.3 Patient Transportation

Medical care to supplement that available in close proximity to the reactor site may be available through mutual assistance compacts among hospitals and among states. When the National Disaster Medical System (NDMS) is fully operational, it will provide administrative mechanisms for distribution of mass casualties across a broad network of hospitals (NDMS Task Force 1984). In addition, for treatment of serious radiation injuries, there are several specialized medical facilities across the country (Ricks 1980).

The cost of transporting patients to medical care facilities will depend on both the number of people injured and the severity of their injuries. People without any injury symptoms and thought to have been exposed to less than 1 Gy would not require transportation since they could be followed up on an outpatient basis (Andrews 1980, p. 303; Blakely 1968, p. 49).

Those exposed to over 1 Gy are likely to be assigned to hospitals based on the diagnosed severity of their injuries, the total number of patients

(a) USSR State Committee on the Utilization of Atomic Energy. 1986. The Accident at the Chernobyl Nuclear Power Plant and Its Consequences. Annex 7, Draft.

requiring care, and the bed availability in hospitals with superior-to-adequate treatment facilities. The most likely treatment locations are those with physicians specially trained to treat burn and cancer patients. The most severely injured patients are likely to be treated at hospitals with facilities designed for treating radiation injuries. These we term "specialized care centers." If the number of patients exceeds the combined capacity of these centers and the better equipped hospitals, additional hospitals whose facilities can be readily adapted for care of radiation injuries are likely to be called upon. In this second category, we assume that capacity in the immediate region would be filled before patients are transported nationwide.

People exposed to more than about 4 Gy would almost certainly be transported to a hospital prepared for specialized care of radiation injuries. If only a few people receive this high an exposure, just one center might be involved. In the unlikely event that such injuries numbered in the hundreds, transportation would be needed to most of the specialized care centers nationwide. There are between 50 and 80 of these centers; the number depends on the criteria used to categorize facilities and expertise. Assuming that these centers could accommodate an average of 10 patients with radiation injuries, (a) their combined capacity is 500 to 800 patients.

If patients exposed to from 1 Gy to about 4 Gy were few in number and constituted the most severely injured group, they would probably be transported to specialized care centers. If beds in definitive care centers were filled, the patients would probably be transported to a second tier of hospitals capable of providing "supportive" care. The required services for supportive care are generally available in hospitals with active hematology and oncology programs (Andrews 1980, p. 304) and possibly those with specialized burn care units. Nationwide there are about 100 hospitals with the required combination of capabilities (including those considered specialized care centers); these are generally located in metropolitan areas and have an average capacity of over 600 beds (Applied Management Sciences 1982). Using less stringent criteria, Andrulis Corporation (1982) concluded that a total of 550 hospitals nationwide could provide "supportive" care. These hospitals could employ reverse isolation techniques to treat bone marrow depression if they lack laminar air flow capabilities. (In a survey of New York hospitals, Andrulis identified availability of blood product supplies, especially platelets, rather than facilities as the most uncertain factor in treatment.) Excluding those counted as specialized care centers, the Andrulis Corporation estimate indicates that there may be a maximum of 500 additional hospitals with adequate treatment resources. If these admit an average of 10 patients each, their combined incremental capacity is about 5,000. (This implies a total capacity nationwide of about 5,500 beds in the specialized care centers and second tier of hospitals combined.)

(a) This assumption is based on findings of Andrulis Corporation (1982); an estimate prepared by Drs. Neil Wald and Joseph Watson for the Reactor Safety Study (USNRC 1975); and conversations with Dr. Bryce Breitenstein of the Hanford Environmental Health Foundation and Dr. Robert Ricks of the Radiation Emergency Assistance Center and Training Site.

Since there is about a two-week lag between acute radiation exposure and onset of serious symptoms, there should be adequate planning time available to coordinate patient distribution and transportation. This relatively symptomless period should also permit the use of normal, rather than emergency, modes of transportation. Transportation to regional hospitals (within 200 to 300 miles) could be provided by automobiles or buses, with planes used for longer distances. If the situation warranted, the Armed Services Medical Regulating Office at Scott Air Force Base, Illinois, could coordinate patient transportation. The Air Force has a C-9 Nightingale and various cargo planes designed for aeromedical evacuation that are employed on a daily basis to distribute military patients nationwide (NDMS Task Force 1984, p. 6).

4.1.4 Cost Estimation

This section describes in turn the cost estimation methods employed for radiation screening, medical monitoring and patient transportation. Radiation survey cost estimates are composed of three components: initial survey costs, resurvey costs incurred in seeking to identify individuals with possible internal body burdens, and whole-body counter costs to measure internal exposures. It is assumed that the entire population that is within a 90° sector downwind at the time of an accident is both surveyed initially and then resurveyed. Costs of decontaminating persons, clothing and decontamination facilities are not included.

To develop screening cost estimates, survey times of 10 minutes for a contaminated individual and 2 to 3 minutes for others are assumed. Assuming that roughly half the population were found to have some external contamination, the initial survey could be conducted at a rate of 8 to 10 persons per hour, or a maximum of about 80 per 8-hour day. Working 16-hour days at this rate, about 25 technicians would be required to process 10,000 people in less than three days. The cost per technician would be about \$600 per 8-hour day, consisting of \$400 for labor and overhead costs, plus travel, subsistence, and equipment costs.

Following decontamination, individuals would be resurveyed for internal contamination. Assuming the same processing rate, 5000 people could be processed by an additional 12 technicians within a 3-day period. Those with suspected internal body burdens would be surveyed in a whole-body counter. Most of these can survey 4 to 5 people per hour and one recently developed unit can provide less precise results at a rate of 50 to 60 per hour under ideal conditions.^(a) Since conditions would not be ideal, we assume that one regular and one high-speed unit could process 20 people per hour or about 1000 people in three 16-hour days. The cost of leasing a whole body counter, accompanied by operators, is about \$2000 per day for each type of unit. The cost of a whole-body count is assumed to apply to the population that is evacuated in connection with a radionuclide release. Since both speed and precision would be required, it is assumed that both a high speed and a regular unit would be

(a) This information was provided by Elizabeth Fothergill of Helgeson Scientific Services, Inc.

employed, with a combined processing rate of about 300 people per day. These costs are summarized in Table 4.1.

Medical monitoring costs have two components, medical service and laboratory costs. To develop cost estimates, it is assumed that medical monitoring would involve at a minimum, a history taking, an initial examination, and review of the patient's symptoms and laboratory test results after 48 hours. Since patients need not be hospitalized during this period, costs are likely to be in the range for similar services rendered in a clinic or office by a general practitioner, \$50 to \$75. A cost of \$65 is estimated for the examination, history taking and diagnostic evaluation. The cost of each complete blood count required lies in a range of about \$15 to \$25. Assuming these tests are carried out every six hours at a cost of \$20 each, the cost of testing would total about \$160. The total estimated cost for people exposed to over 1 Gy is \$225 each.

Transportation costs may vary considerably depending on whether the mode of transportation is military or civilian, public or private, group or individual. Costs per mile for private motor vehicle transportation are available

TABLE 4.1. Radiation Screening and Medical Monitoring Cost Summary (1984 \$)

Procedures	Population Affected	Processing Rate	Labor/Equipment Costs	Cost/Unit
I. Radiation Screening:				
Initial Survey	Persons within 90° sector downwind	10 persons/hour	\$600/8-hour day	\$600/80 persons
Resurvey	(a)	10 persons/hour	\$600/8-hour day	\$600/80 persons
Whole Body Count ^(b)	All persons evacuated	20 persons/hour	\$2000/16-hour day	\$4000/300 persons
II. Medical Monitoring:				
Medical Services ^(c)	Persons w/ greater than 1 Gy exposure	2 persons/hour	\$65/person	\$65/person
Laboratory Tests	Persons w/ greater than 1 Gy exposure	50-135 tests/hour	\$20/test	\$160/person

(a) Some fraction of persons in 90° section downwind. Fraction is user-selectable with default value of 0.5.

(b) Assumes use of one high-speed and one regular unit.

(c) Exam, history-taking and review of laboratory tests.

from the U.S. Department of Commerce for various vehicle types and ownership periods (USDOC 1984, p. 621). The automobile cost estimate employed in the HECOM data file represents a mid-range value over standard and compact car sizes for a 12-year ownership period.

Motor vehicle costs are used in estimating transportation costs to regional hospitals, defined as lying within a 200- to 300-mile radius of the accident site. Air transportation costs per passenger mile are based on Civil Aeronautics Board data for 700- through 1000-mile domestic coach fares (CAB 1980). The estimated average roundtrip distance to hospitals, including specialized care centers, nationwide is based on the assumption that the locations of nuclear reactors and major hospitals are both influenced by population density. Both appear to be concentrated east of the Mississippi River. To provide an estimate of average air transportation distance, the average distance to all reactors, from a reactor centroid location, of approximately 800 miles is used. This distance is applied to air transportation costs.

Table 4.2 provides a summary of patient transportation cost elements for an exposed population. The capacities shown for specialized care centers, regional hospitals and hospitals nationwide are not additive since specialized and regional hospitals are subsets of the total. In estimating total transportation costs for the population, we assume that specialized care centers would be the first placement choice for all patients, with priority given to the most severely injured. After these beds are filled, regional hospitals capable providing supportive care are assumed to be the second choice. The assumption of 50 hospitals within a 200- to 300-mile radius as shown in Table 4.2 is much more representative of the distribution east of the Mississippi than west. If the injured fill these beds as well, we assume that remaining patients would be transported to hospitals nationwide that are capable of providing supportive care. In the unlikely event that even these are filled, additional patients are assumed to be placed in regional area hospitals with less adequate facilities.

TABLE 4.2. Patient Transportation Cost Summary (1984 \$)

Type of Facilities	Approximate Number	Approximate Capacity ^(a)	Average Round-Trip Travel Distance (miles)	Cost Per Mile	Cost Per Person
Specialized Care	50-80	500-800	1600	\$0.13 ^(b)	\$208
Regional Hospitals	50	500	400-600	\$0.29 ^(c)	\$145
Hospitals Nationwide	550	5500	1600	\$0.13 ^(b)	\$208

(a) First two categories are subsets of the national total.

(b) Air transportation costs are based on Civil Aeronautics Board 1980. "Domestic Rate Structure Cost Analysis, 1979, Trucks and Local," Office of Economic Analysis.

(c) Automobile costs based on 12-year average in 1981 dollars from Statistical Abstract of the United States 1984, Table No. 1078.

4.2 PRODROMAL SYMPTOMS

Whole-body irradiation can cause gastrointestinal symptoms ranging from mild and transient discomfort to potentially fatal vomiting and diarrhea. The dose effect relationships, treatments and direct costs for mild morbidity are discussed in this section and more serious gastrointestinal morbidity and mortality are discussed separately in Section 4.3.

Prodromal symptoms of radiation exposure may include nausea, loss of appetite, headache, diarrhea, and weakness. The higher the radiation dose and the shorter the exposure duration, the sooner these symptoms occur (Blakely 1968, p. 35 and USNRC 1975, p. F-13). Blakely (p. 35) reports that prodromal symptoms may occur occasionally after a dose as low as 0.5 Gy, but are more likely at 1 Gy and are seen in all cases at 2 Gy and above. They generally occur within a few hours of exposure and persist for a few days.

In its milder forms prodromal symptoms may be treated like a case of the flu, and are not serious in themselves, except perhaps for the very young, the old, and those with prior illness or injury (Dalrymple et al. 1973, p. 191). The appearance of prodromal symptoms, however, serves to identify persons who may have received sufficient exposure to result in more serious radiation injuries, such as bone marrow syndrome. Because closely monitoring prodromal symptoms provides a way to detect the existence of serious injury, we assume that people would be treated as though seriously injured until evidence develops to the contrary. Such treatment could involve two or three days of hospitalization, with the administration of fluids and medications and the performance of numerous laboratory tests.

Following the prodromal symptoms there may be a latent period before the manifestation of more serious injury. The duration of the latency period varies inversely with the dose and dose rate. Table 4.3 summarizes the typical progression of acute radiation symptoms for various whole-body dose levels. In the less serious cases the latency period lasts from 1 to 3 weeks, during which time the individual may experience weakness and fatigue and should have both mental and physical rest to minimize the severity of the hemorrhage and infection that may follow (Blakely 1968, p. 50). This is a time when preparations can be made at regional medical centers for the treatment of severe cases and a time when patients can be transported to centers with adequate facilities.

Treatment costs are estimated from likely hospital charges. In 1981 the average total hospital charge for inpatient services was approximately \$300 per day (Health Care Financing Administration, June 1982). This amount is \$385 in 1984 dollars. If physicians' fees average some one-third of hospital charges, as they do for cancer patients (Scotto and Chiazze 1976), then they will add another \$125 for each day of care. We assume a 3-day stay in the hospital, resulting in an estimate of about \$1,500 per case of pronounced prodromal symptoms. While provision of such high-quality care may be unlikely in the event of an accident affecting a large population, lack of it would probably increase fatality rates and, hence, societal losses. Unless the injured are quickly identified and isolated to prevent infection, fatalities may occur even among those exposed to as little as 1.5 Gy to 1.75 Gy (USNRC 1975, Appendix VI, F-1).

TABLE 4.3. Clinical Progression of Acute Radiation Syndrome^(a) Following Whole-Body Exposure

Approximate Whole-Body Dose Levels (Gy)	Clinical Progression						
0.5-2			prodromal symptoms	+	recovery		
2-4	prodromal symptoms	+	latency 2-3 weeks	+	mild bone marrow crisis 2-8 weeks	+	most recover
4-6	prodromal symptoms	+	latency 1 week	+	severe bone marrow crisis 2-6 weeks	+	about 50 percent recover ^(b)
6-10	prodromal symptoms	+	latency few days	+	gastrointes- tinal injury 1-2 weeks	+	probable death
10	death within hours from cerebrovascular crisis						

- (a) USNRC (1975) considers dose ranges from 3.5 to 5.5 Gy as critical for the bone marrow. That study distinguishes between whole-body doses and locus-specific doses to gastrointestinal tract and lungs. Evans, Moeller and Cooper (1985, Vol. 2, p. 30) suggest that in the absence of bone marrow complications mortality from gastrointestinal injury alone would not occur below 10 Gy.
- (b) At dose levels of about 3.4 Gy, 50 percent of the exposed population are expected to die within 60 days if they received only minimal treatment. Evans, Moeller and Cooper (1985) suggest that 50 percent could survive whole-body doses as high as 11 Gy with heroic treatment (i.e., with a bone marrow transplant).

4.3 GASTROINTESTINAL SYNDROME

At whole-body doses over approximately 6 Gy the symptoms of gastrointestinal syndrome are likely to precede those of bone marrow damage (Saenger 1982; Blakely 1968). Symptoms appear after a shorter latency period (a few days to a week) than at lower doses. The symptoms include vomiting and diarrhea of a severity that is qualitatively different from that experienced in the prodromal phase. Death is probable within a week or two of exposure (Blakely 1968, p. 41; Evans, Moeller and Cooper 1985, Vol. II, p. 36). For local irradiation of the gastrointestinal tract without a high whole-body dose, the median-lethal dose may be closer to 15 Gy for acute exposures (Evans, Moeller and Cooper 1985, Vol. 2, p. 34).

For either local or whole-body irradiation, treatment involves the replacement of fluids and electrolytes. Such treatment may keep the patient alive long enough for the intestinal lining to heal (Blakely 1968, p. 41). However, with whole-body exposure, the recovered patient will face severe bone marrow syndrome a short time later. Because of this threat of bone marrow syndrome in patients who survive the gastrointestinal symptoms, we assume that gastrointestinal patients would be treated from the start in the same conditions of isolation prescribed for bone marrow patients.

Under this assumption, the treatment costs are likely to be of the same magnitude as for hematopoietic syndrome since the major cost component is the cost of maintaining sterile isolation. For two weeks of treatment, costs are estimated to total \$28,000. Details of the cost estimation procedure are described below in Section 4.4.

4.4 HEMATOPOIETIC SYNDROME

Failure of the blood producing function of the bone marrow system would be the primary cause of serious illness or death as a result of whole-body radiation exposure in a reactor accident. Hematopoietic syndrome is characterized by impairment of the blood forming system, with the degree of impairment depending on the dose. The clinical manifestations include severe susceptibility to infection, hemorrhage, and anemia. Blakely (1968, p. 37) places the lower threshold for hematopoietic syndrome at about 2 Gy. Milder manifestations result from doses between 2 and 4 Gy and more severe symptoms from doses between 4 and 6 Gy.

Evans, Moeller and Cooper (1985) Vol. 2, p. 25) present dose-response curves for bone marrow damage depending on the extent of medical intervention. That study predicts 50 percent of the people exposed to 3.4 Gy would die within 60 days if they were given only minimal treatment. With heroic or "intensive" treatment the report asserts that the 50 percent lethal dosage may be as high as 11 Gy for whole-body exposure. Heroic treatment would involve a bone marrow transplant as well as the measures applied in "supportive" treatment.

With supportive treatment, as assumed in this cost estimate, it is estimated that 4.5 Gy would be lethal within 60 days for 50 percent of those exposed (Evans, Moeller and Cooper 1985, Vol. 2, p. 26). Treatment would be focused on keeping the patient free from complications until bone marrow function is regained. Supportive treatment involves sterile isolation, controlling infection by employing special air filtration systems and sterilizing everything that comes into the room (Andrews 1980, p. 306; Blakely 1968, p. 61; USNRC 1975, Appendix VI p. 9-3). Administration of antibiotics is prescribed (Saenger 1982; USNRC 1975), as well as continual monitoring with laboratory tests (Andrews 1980; Saenger 1982) and replacement of blood components as needed.

For purposes of outlining the probable course of treatment and its costs, we suggest there are relevant similarities between the characteristics of bone marrow syndrome and those of burn trauma. Both are potentially lethal threats,

with infection as the immediate concern. In addition, a possibility of severe hemorrhage is present in either condition. Because of the clinical similarities, we assume that the services involved in providing "supportive treatment" are similar to those given a nonsurgical burn trauma patient. Many of the patients from the Chernobyl accident required simultaneous treatment for hematopoietic syndrome and severe radiation burns.^(a)

To control infection, burn patients are placed in reverse sterile isolation, usually employing special air filtration systems. Because of these special precautions, a regional burn care center charges \$1255 per day for "room and board" alone.^(b) That is the cost for nonsurgical burn patients; those requiring surgery receive additional precautionary measures, and pay up to \$2000 per day for a room in sterile isolation. Patients with radiation-induced bone marrow syndrome would require somewhat similar precautions to avoid infection (Andrews 1980, p. 306; Blakely 1968, p. 61). Therefore we apply a cost for hospital room of about \$1250 per day.

In addition to hospital room charges, a typical nonsurgical burn patient may pay \$200 per day for medications, \$180 per day for laboratory tests, and \$50 for each blood transfusion.^(b) Saenger (1982) suggests both prophylactic and systemic antibiotic therapy should be used to fight infection in the bone marrow syndrome patient. He advises the use of antibiotic and antifungal agents such as neomycin, oxacillin, and nystatin. This aggressive approach to medication is probably not very different from that followed for a burn patient, so we include \$200 per day for medications in the total cost of treating bone marrow syndrome.

Similarly the continual monitoring of blood counts along with laboratory cultures results in high laboratory costs for a hematopoietic syndrome patient (see Andrews 1980 and Saenger 1982). The daily costs could easily reach levels similar to those of a burn patient, about \$180 per day for laboratory tests.

Each bone marrow patient can expect a number of transfusions both to replace white blood cells, in moderate forms of bone marrow failure, and to replace whole blood and platelets, required by hemorrhage in severe cases. We add another \$25 per day to account for cost of a transfusion approximately every second day.

Based on these estimates, total daily cost of hospital services for bone marrow syndrome may run approximately \$1650. Because of the relatively high cost of the hospital services component of this care, physicians' charges may not amount to the full 33 percent we have applied to other estimates based on the experience with cancer care. If physicians' fees amount to about one-fifth of hospital costs in this case, they may total some \$350 per day, resulting in

(a) USSR 1986 (draft).

(b) Communication with staff at Harborview (Seattle) Medical Center's burn care unit, March 1983. This cost estimate was corroborated by multiplying the average number of tests per patient from Chernobyl by an average cost per test, resulting in approximately the same total cost level.

of hospital costs in this case, they may total some \$350 per day, resulting in a total cost close to \$2000 per day. Depending on the severity of injury, patients may be hospitalized for from two to six weeks. Costs could range, therefore, from \$28,000 to \$84,000 for bone marrow syndrome. These costs do not include the cost of a bone marrow transplant, which prior to the Chernobyl experience, was often recommended for patients with severe bone marrow syndrome, especially for those who received a probable fatal dose (Blakely 1968; Dalrymple 1973; USNRC 1975; Saenger 1982). Exception to this recommendation was taken by Andrews (1980). Based on the lack of transplant survivors after Chernobyl, the Soviets concluded that "the proportion of patients in whom transplantation of allogeneous bone marrow is absolutely indicated and for whom this treatment will obviously be beneficial is very small."^(a) The cost of a bone marrow transplant is approximately \$70,000 (Gale 1979, p. 1923; Thomas 1983, p. 2536). Although bone marrow syndrome is not the most severe manifestation of acute radiation injury, it is probably the most costly, since more severe injuries are almost certain to end in death before large amounts of medical resources can be expended.

4.5 PULMONARY SYNDROME

Pulmonary impairment can be expected to result from high radiation doses to the lung from inhalation or whole-body external radiation. It is possible to receive a high lung dose with relatively low whole-body doses. Acute lung doses of 9.5 Gy may cause either mortality or morbidity to 50 percent of adults. Half of that dose may cause similar injury to children (Evans, Moeller and Cooper 1985, Vol. 2, p. 40). Fatalities are most likely to occur among children and the elderly (Vol. 2, p. 37).

Symptoms of pulmonary injury include pneumonitis in the first two months after irradiation and development of pulmonary fibrosis from two to nine months after exposure. Chronic lesions may persist for the remaining lifetime. In the absence of bone marrow syndrome, we assume these symptoms could be treated in typical hospital accommodations. Corticosteroid therapy is recommended for symptoms of pneumonitis (Evans, Moeller and Cooper 1985, Vol. 2, p. 37). In the case of higher dose levels, corticosteroid therapy does not halt the progression to fibrosis (Phillips and Wyatt 1980).

Typical hospital charges are used to develop a treatment cost estimate. The average 1981 hospital room charge was \$300 per day. At 33 percent of hospital charges, physicians' fees may add another \$100 per day. Thus, pulmonary impairment may cost about \$400 per day for hospital and medical services. In 1982 the average length of stay in acute-care hospitals was eight days for pneumonia, and ten days for pulmonary fibrosis (National Center for Health Statistics 1983). Assuming that radiation-induced pulmonary injuries would require similar treatment, we sum the data for these similar diseases and assume an 18-day length of stay. That leads to a total cost estimate for pulmonary impairment of approximately \$10,000 in 1984 dollars.

(a) USSR 1986, p. 33, (draft).

Cost estimates for pneumonia and pulmonary fibrosis were also developed from the National Medical Care Utilization and Expenditure Survey (NMCUES), 1980 (National Center for Health Statistics 1983b). The data include costs of hospitalization, physicians' fees, drugs and other medical expense. In 1984 dollars, the average total treatment cost for a case of pneumonia was \$4130 and for pulmonary fibrosis, \$3240. Summing these to reflect the clinical progression of pulmonary syndrome results in total costs of \$7370. Given the range of costs produced by the two estimation methods, we use a rough mid-point of \$8500 as the best estimate.

4.6 PRENATAL INJURY AND MORTALITY

A category qualitatively different from other radiation injuries is in-utero or prenatal effects. At doses of 0.5 Gy injuries would be induced mainly by irradiation during the second trimester of pregnancy (10 to 18 weeks gestation), with spontaneous abortion likely for embryos in earlier gestational stages (Evans, Moeller and Cooper 1985, Vol. 2, p. 56). The nervous system is particularly sensitive to injury during the second trimester and effects such as growth impairment, microcephaly and mental retardation have been observed at doses as low as 0.1 to 0.2 Gy (USNRC 1975, Appendix VI, p. F-18). Microcephaly, which is generally associated with severe retardation, is estimated to occur in about 50 percent of fetuses exposed to 0.37 Gy as a result of acute exposures (Evans, Moeller and Cooper 1985, Vol. 2, p. 59).

Long-term institutionalization may be required for individuals injured by prenatal irradiation. The care provided may be similar to that given to individuals who are severely affected by Down's Syndrome or mental retardation. Lacking specific information to guide our cost estimates for prenatal radiation injuries, we rely on the probable similarities with these two other prenatal-onset disorders resulting in lifetime impairment.

Cost estimates for institutional care of individuals with congenital defects, similar in effect to the retardation and nervous system anomalies induced by prenatal radiation injury, are used to estimate the cost of caring for individuals with prenatal injuries. Two studies provide estimates of the annual cost that may be incurred in the institutional care of persons with Down's Syndrome or other mental retardation. Conley (1973, p. 298) estimates an annual cost of \$10,300 in 1984 dollars. Intagliata, Wilder and Cooley (1979, p. 155) provide estimates that range from \$10,000 to \$15,000 in 1984 dollars when normal consumption expenditures are subtracted. We use a rough average of those estimates, \$12,500, as the annual cost of a prenatal injury. The annual cost is applied to each year of the injured person's life and is weighted by the degree of impairment [e.g., 50 percent, estimated for individuals with chromosomal defects such as Down's syndrome (see Section 6.1.3)]. Life span is assumed to be normal.

In the case of prenatal mortality, mainly occurring in the first trimester of pregnancy, there may be costs associated with after care of the mother. Costs for an examination, laboratory tests and, in a few cases, a dilation and curettage procedure are estimated to average \$100 per case.

4.7 THYROID DISORDERS

Two types of thyroid disorders, acute thyroiditis and hypothyroidism, may result from high levels of exposure to ^{131}I . These disorders are not likely to result from whole-body irradiation alone since the dosage required would result in a lethal case of hematopoietic syndrome.

4.7.1 Thyroiditis

Thyroiditis is an inflammation of the thyroid gland that can result in partial or total cell destruction. The symptoms of glandular pain and tenderness begin within two weeks of exposure and generally last for two to four weeks. A thyroid dose of about 1200 Gy is required to induce thyroiditis in 50 percent of cases (Evans, Moeller and Cooper 1985, Vol. 2, p. 43). The acute or thyrotoxic phase may be treated with fluids, rest and anti-inflammatory drugs such as aspirin. Steroids are administered in severe cases (Silverberg 1985, pp. 516-17).

Data from NMCUES 1980 (National Center for Health Statistics 1983b) provide a treatment cost estimate. The average cost of medical office visits reported was \$136 and the average cost for medication was \$10. These total \$207 in 1984 dollars.

4.7.2 Hypothyroidism

Hypothyroidism, which is a deficiency or loss of thyroid function can be induced in 50 percent of cases by a thyroid dose of 60 Gy (Evans, Moeller and Cooper 1985, Vol. 2, p. 44). A year or more may elapse before symptoms develop. Symptoms of this condition include fluid retention, muscle cramps, mental sluggishness, and reduced bodily functions. Oral doses of thyroid hormone are used to correct this condition. These must be continued over the patient's remaining lifetime to avoid symptom recurrence. The average cost reported in the NMCUES data (National Center for Health Statistics 1983b) for medical office visits was \$91. A cost of \$18 was reported for medications. The total of \$155 in 1984 dollars is assumed to be an annual expense.

4.8 SKIN DAMAGE

Loss of hair occurs two to three weeks after external doses in excess of 3 Gy. This is likely to be temporary unless the dose exceeds 6 Gy (USNRC 1975, Appendix VI, p. F-13). The skin may also be affected by doses in this range, resulting in radiation dermatitis. Survivable whole-body doses are unlikely to cause more severe injuries than hair loss and skin reddening. Skin damage has levels of severity comparable to first, second and third degree thermal burns and in the most severe cases (skin doses of over 20 Gy) can result in permanent skin ulceration (Prasad 1974, p. 240-248).

The mildest form, erythema, involves skin reddening occurring from hours to weeks after exposure to 6 Gy in 50 percent of cases. This is much like a sunburn and does not require medical care. Transepithelial injury is

equivalent to a second degree burn, and blisters which appear within a week or two after exposure require treatment to prevent infection. This condition may result from a dose of 20 Gy in 50 percent of cases (Evans, Moeller and Cooper 1985, Vol. 2, p. 51). Information regarding the most severe form of damage, dermal necrosis is inadequate for development of dose effect relationships. Cost estimates are developed from NMCUES 1980 data for external burns, other than eyes (National Center for Health Statistics 1983b). An average of \$200 per case (1984 dollars) is estimated for obtaining outpatient medical care and medications.

4.9 CATARACTS

Doses of 2 to 5 Gy to the lens of the eye may result in formation of cataracts after a latency period that varies with both dose and dose rate (USNRC 1975, Appendix VI, p. 9-18). The average latency period is two to three years (Evans, Moeller and Cooper 1985, Vol. 2, p. 60). Generally, cataracts are removed surgically and the lens of the eye is replaced. Average costs for treatment were estimated from data contained in NMCUES 1980 (National Center for Health Statistics 1983b). In 1980 dollars, the charges averaged \$1691 for hospitalization, \$1671 for in-hospital treatment by physicians, \$162 for visits to the doctor's office, and \$82 for drugs and other medical expenses. The total is equivalent to \$5122 in 1984 dollars.

4.10 STERILITY

Radiation-induced sterility may be either temporary or permanent. Temporary effects may occur in males at lower doses than in females but males require higher doses for permanent effects. Permanent sterility, in males or females, is unlikely below doses that are life-threatening if whole-body exposure is involved, as is likely in accidental exposures (USNRC 1975, Appendix VI, p. 9-15).

Temporary sterility of females may be caused by doses of 1.5 to 2 Gy and doses above 3.2 Gy may cause permanent sterility. These effects result from killing of the oocyte cells. In males, temporary sterility may be caused by doses as low as 0.1 Gy and recovery may require several years. Permanent sterility requires doses of from 2 to 6 Gy (Evans, Moeller and Cooper 1985, Vol. 2, p. 53).

Since permanent sterility is not amenable to treatment, no treatment cost estimate is developed. While temporary sterility may be amenable to treatment, the desirability of doing so is questionable due to the possibility of genetic damage. The major impacts of sterility are nonmonetary in nature.

4.11 COST SUMMARY

Treatment costs for radiation injuries are summarized in Table 4.4. Some of these costs, such as the cost of prodromal symptoms, are for morbidity only, while others apply equally to fatal and nonfatal cases. The cost shown for mortality resulting from prenatal injury applies only to care of the pregnant woman, not to the fetus or embryo. Costs shown for treatment of hematopoietic syndrome are higher than for gastrointestinal syndrome, a more severe injury. This difference is due to the greater potential for survival of those with hematopoietic syndrome.

TABLE 4.4. Radiation Injury Direct Costs (1984 \$)

<u>Radiation Injuries</u>	<u>Treatment Costs</u>	
	<u>Morbidity</u>	<u>Mortality</u>
Prodromal Symptoms	1,500	--
Gastrointestinal Syndrome	28,000	28,000
Hematopoietic Syndrome	56,000	56,000
Pulmonary Syndrome	8,500	8,500
Prenatal Injury/Mortality	12,500 ^(a)	100
Acute Thyroiditis	200	--
Hypothyroidism	150 ^(a)	--
Cataracts	5,100	--
Transepithelial Injury	200	--

^(a) Annual expense.

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5.0 DIRECT COST ESTIMATION FOR CANCERS

Two different approaches have been employed in the past to measure the direct costs of treatment for cancers: prevalence and incidence. The prevalence approach, which was discussed in Section 1.2.3, has been employed by Rice and her associates (Rice 1966; Cooper and Rice 1976; Rice and Hodgson 1981; Hodgson and Rice 1982). This approach is well-suited to an aggregate accounting of illness costs, in which total national expenditures for selected health services are allocated to the various illness categories. Direct costs thus computed are of little use, however, in evaluations of actions that affect the risk of illness.

The incidence approach asks, conceptually, "Given a certain event--a reactor accident, for example--what will be the total cost of treating the associated health effects?" The incidence approach requires a "bottom-up" measurement of treatment costs based on scenarios of expected treatment.

Cromwell et al. (1976) and Hartunian, Smart and Thompson (1981) provide the only incidence-based measures of direct costs across a range of cancer types presently available. A number of other studies have undertaken "bottom-up" measurements of costs for particular types of cancer; examples are, Scitovsky and McCall (1977) for breast cancer, and Schneider and Twigg (1972) for cervical carcinoma. Unfortunately, these studies concentrate typically on patients with specific cancers, and are not representative of treatment regimens and costs for a broad range of cancer types.

The treatment regimens underlying in the two principal studies of costs of cancer incidence by Cromwell et al. (1976) and Hartunian, Smart and Thompson (1981) are based on information regarding treatment as reported in the Third National Cancer Survey. The PNL HECOM direct cost estimates for various types of cancer mirror the basic approach taken by both Cromwell and Hartunian: given the treatment regimens reported in the Third National Cancer Survey (TNCS), current costs are computed by inflating TNCS costs to current dollars (with a few adjustments).

Because the cost estimates are being developed to evaluate changes in the risks of accidental irradiation, cancers are grouped into broad categories related to the organ irradiated. These categories are leukemia, lung, breast, bone, gastrointestinal, skin, thyroid and a miscellaneous category of "other" cancers. Information regarding the methods used in projecting incidence of these cancers is presented in Section 5.1. Because the cost data for all of the cancer categories drawn from one source, the cost data for all categories are discussed in Section 5.2 and the cost estimation methodology is described in Section 5.3.

5.1 INCIDENCE AND FATALITY PROJECTIONS

There is wide consensus among scientists that an association exists between ionizing radiation and cancer. Nevertheless, there is considerable

uncertainty regarding dose-effect relationships, to the extent, as Land (1980, p. 1197) reports, that scientists contributing to BEIR 80 differed by as much as a factor of 100 in their assessment of the risk from exposures to a single rad of ionizing radiation. Because there are basic disagreements about central features of the techniques used to estimate dose-effect relationships, and because scientific knowledge concerning the risks from radiation is changing, the major source of uncertainty in the cancer cost estimates generated by HECOM is the incidence projections. Discussion of issues related to incidence projection may be found in Evans, Moeller and Cooper (1985) and in BEIR (1980).

Since cancer incidence may occur over the remaining lifetime of an exposed population, costs will be distributed similarly. The risk models, latency periods and periods of risk used to project treatment costs are discussed in the following sections.

5.1.1 Models for Projecting Risks Over Time

An absolute risk model posits that for a given exposure the number of induced cancers per population unit is constant over time. In contrast, a relative risk model maintains a constant ratio over time between induced and spontaneously occurring cancers for each exposed age group. Since most cancers occur spontaneously at higher rates in older age groups, the relative risk model projects increasing numbers of induced cancers over time. New data led Evans, Moeller and Cooper to employ a relative risk model for the central risk estimates for the breast, lung, gastrointestinal and other cancer categories. An absolute risk model is used for leukemia, bone, thyroid, and skin cancers and cancers due to in utero exposures (1985, Vol. 2, p. 99). These modeling approaches are also employed in the HECOM base case.

5.1.2 Latency Periods

Studies have found that the minimal latency periods for cancers may be affected by age at exposure (Evans, Moeller and Cooper [1985], Vol. 2, p. 111). The latency periods used in the HECOM base case are those supported by the BEIR 80 analysis and used in Evans, Moeller and Cooper: two years for leukemia and bone cancer, five years for thyroid cancer and ten years for the other categories. In addition, based on new data for Japanese atomic bomb survivors, Evans, Moeller and Cooper (1985, Vol. 2, p. 111) and HECOM assume that breast cancers are not induced before the age of 30 and lung cancers not before the age of 40.

5.1.3 Periods of Risk

Based on recent information, Evans, Moeller and Cooper have adopted a model in which cancer risks, except for leukemia, bone cancer and cancers due to in-utero exposure, persist over remaining lifetimes (1985, Vol. 2, p. 109). A risk period of 25 years (2 to 27 years following exposure) is employed for bone cancer and leukemia. For cancers due to in-utero exposure the risk period is estimated to persist from birth to 12 years for leukemia and to 10 years for other cancers (Evans, Moeller and Cooper 1985, Vol. 2, p. 123).

5.2 COST DATA

Because the Third National Cancer Survey (TNCS) (Scotto and Chiazze 1976) is the primary source of information, both on services rendered and on costs, it is useful to review the strengths and limitations of the TNCS data. The TNCS collected data from several sources. As part of the TNCS, a sample of approximately 8500 cancer patients, newly diagnosed in the years 1969-71, were interviewed in depth with a Patient Interview Booklet (PIB). (That study represented slightly less than 10 percent of the full TNCS sample). The PIB elicited details of both the services received by each patient and the payments for those services. In addition to the PIB, information on hospital charges was extracted from patient records for 6332 of the TNCS patients (Scotto and Chiazze 1976). Cromwell et al. (1976) used the hospital records sample to analyze payments from patients to hospitals and to other health providers as reported on the PIB. As Cromwell shows (pp. 66-68), the difference between the two data sources is small in terms of average hospital cost per cancer case. Across the various types of cancer, however, Cromwell shows that there are significant differences between the two data sources (differing by as much as 50 percent). Cromwell concludes that the self-reported data from the PIB may be an unreliable source of hospital costs data by cancer type.

Because the PIB is the major source available for other treatment costs, Cromwell et al. (1976) cancer cost study presents hospital costs as reported on the PIB. In comparison, Hartunian, Smart and Thompson (1981) make use of the stronger data from Scotto and Chiazze (1976) to measure hospital costs. Then they construct the ratio of hospital costs to other service costs, as reported on the PIB, to estimate the cost of all nonhospital services. We employ a similar methodology.

In addition to the weakness of particular data collection instruments, there are other limitations to the cost data from the TNCS. Cromwell (1976, pp. 56-73) identifies biases in that the high cost Northeastern states are not represented, nonresponse occurred more heavily among patients with the more aggressive cancers, and interviewees exhibited selective memory.

A structural limitation of the TNCS data particularly worth mentioning is that the PIB data cover a time interval between the onset of symptoms and the date of the interview. This time interval varied widely (Cromwell 1976, p. 72) and the wide range of time spans makes it difficult to interpret the cost data. Theoretically, direct costs would include monetary outlays for the entire course of the illness, discounted to present value in the year of decision making. Lacking such data, direct costs should be measured over a standard time frame, such as considered for hospital costs (but not for the costs of other services) by Scotto and Chiazze. Their data include hospital costs over the first two years after diagnosis. Because the available cost data are not comprehensive, the direct cost estimates are almost certainly biased downward.

Because the TNCS data were collected for the period 1969 through 1971, they also represent the treatment modes in use at that time. Medical advances have lowered cancer mortality rates since 1970 and treatment is now more intensive. To the extent that increases in real costs have been required to make

treatment gains, direct costs may be underestimated by use of the TNCS data. A study of changes in treatment cost from 1951 to 1971 showed both cost reductions and increases (Scitovsky and McCall 1977).

5.3 COST ESTIMATION

The direct costs of cancer include all of the costs of hospitalization, physicians' care, drugs, nursing, special equipment, transportation, radiation treatments, chemotherapy, etc. Data from the TNCS (and PIB survey) are used to create the following cost categories:

Hospital/inpatient--includes physicians' and nurses' services, laboratory, diagnostic, radiotherapy and surgical charges as well as hospital bed charges, supplies, and special services.

Outpatient/doctor--office, home and clinic, outpatient visits, and surgical and other physician inpatient costs.

Other nonhospital--nursing home, private nurse, in-home nursing, drugs, and rehabilitation.

These direct cost components are then used to construct a direct cost estimate for each cancer category. Since the TNCS data are categorized partially by cancer type and partially by cancer site, these categories are combined where necessary to correspond with the cancer categories as shown in Table 5.1. For the categories of "gastrointestinal tract" and "other," the TNCS and PIB data are aggregated using the proportional incidence of the major types or sites of cancers within the category as weights. Those cancers constituting less than five percent of the total incidence are not included. Thus, based on the distribution in Table 5.1, the cost estimate for gastrointestinal cancers is a weighted average of the costs for the five major types of cancers falling within that category.

Average total hospital cost is calculated from the TNCS data for each of the cancer categories, using the proportional weighting for TNCS categories described in Table 5.1 for the gastrointestinal and other cancer categories. These cost estimates, representing first and second year hospital costs, are shown in Table 5.2. While treatment of some patients may extend over several years, the brevity of median survival periods makes application of two years' costs to all cases a reasonable approximation of total costs. In calculating benign thyroid nodule costs, the HECOM base case assumed that 75 percent of the benign nodules are diagnosed without surgery and that only outpatient costs are incurred in these cases.^(a)

Cromwell et al. (1976, p. 55) have summarized the direct cost components reported on the PIB and adjusted them for nonresponse. The resulting

(a) Communication with Oncology Department staff, University of Washington.

TABLE 5.1. Corresponding Cancer Categories in HECOM and the Third National Cancer Survey Hospitalization and PIB Data

<u>HECOM Category</u>	<u>Third National Cancer Survey Category</u>	<u>TNCS Percent</u>	<u>PIB Percent (a)</u>
Leukemia	Leukemias	--	--
Lung	Lung and Bronchus	--	--
Breast	Breast	--	--
Bone	Bone	--	--
Skin	NA	NA	NA
Thyroid	Thyroid	--	--
Gastrointestinal	Colon	45	46
	Rectum	20	22
	Pancreas	13	14
	Stomach	18	15
	Esophagus	4	3
Other Cancers	Bladder	44	38
	Brain	15	18
	Multiple Myelomas	0	11
	Lymphomas	41	33

(a) Population in each TNCS and PIB category (by site) shown as a percentage of the corresponding, broader HECOM category.

proportional relationships between hospital costs and outpatient-doctor and other nonhospital costs are applied to the TNCS (Scotto and Chiazze 1976, pp. 65-69) hospital cost data in Table 5.2 to estimate the outpatient-doctor and other nonhospital cost components.

Hospital data were collected by TNCS over a two-year period but are only reported for first and second admissions and total. To allocate costs over time, we assume that the first admission occurs in the first year and that all subsequent admissions occur in the second. The resulting hospital cost estimates are shown in Table 5.3. Average total nonhospital costs from Table 5.2 are then allocated proportionally to hospital costs in Table 5.3.

Total direct cancer care costs shown in Table 5.4 are converted to 1984 dollars using the all medical care cost component of the Consumer Price Index (CPI). Once the direct cost estimates are calculated in this form, they are applied to cancer fatality and morbidity projections to calculate the total direct cost of cancer care over time.

TABLE 5.2. Average Hospital Costs and Computation of Average Nonhospital Costs by Cancer Type from TNCS and PIB Data (1970 \$)

Cancer Type	Average Total Hospital Cost (a)	Nonhospital Cost		Average Total Nonhospital Cost
		Outpatient Doctor (b)	Other (c)	
Leukemia	3813	1788	892	2680
Lung	3627	1701	849	2550
Breast	2148	1007	503	1510
Bone	4622	2168	1082	3250
Skin	NA	NA	NA	NA
Gastrointestinal	3603	1690	843	2533
Other	3366	1579	788	2367
Thyroid-malignant	1768	829	414	1243
Thyroid-benign	442	207	--	207

- (a) Average total hospital costs are from Scotto and Chiazze, Table 10A, increased by 20 percent to affect nonpayment bias.
- (b) Outpatient/doctor costs are 46.9 percent of hospital costs as adjusted for nonresponse by Cromwell et al., Table 3.1B.
- (c) Other costs are 23.4 percent of hospital costs as adjusted for non-response by Cromwell et al., Table 3.1B.

Direct costs of treating skin cancer other than melanomas were unavailable from the TNCS data. Instead, skin cancer costs are estimated from National Medical Care Utilization and Expenditure Survey data (NMCUES) (National Center for Health Statistics 1983). Average cost of hospitalization for skin cancer was \$726, doctors fees \$295, office visits \$195 and drugs \$17 in 1980 dollars. The total in 1984 dollars is \$1723. Since basal and squamous cell carcinomas are the major cancer types involved, it is unlikely that further treatment costs would be incurred.

The NMCUES data were also used to calculate treatment cost estimates for gastrointestinal, lung and breast cancers. For lung and breast cancers the NMCUES cost estimates varied from the TNCS first year costs by ten percent or less. For gastrointestinal cancers the difference was nearly 16 percent. There was no evidence of bias in the differences between estimates.

Since the cancers would not occur immediately after radiation exposure but would generally occur over the remaining population lifetime after minimum latency periods of from two to 10 years, the direct costs must be discounted to a present value estimate. This is accomplished by projecting the cancer rate for each age group in each year of remaining lifetime and discounting the associated cost to a base year estimate. For cancers to which the absolute

TABLE 5.3. Computation of Cancer Direct Costs by Year from TNCS
Hospitalization Cost Data (1970 \$)

Cancer Type	First Year			Second Year		
	Hospital (a)	Nonhospital (b)	Total	Hospital (c)	Nonhospital (b)	Total
Leukemia	1682	1179	2861	2131	1501	3632
Lung	2170	1530	3700	1457	1020	2477
Breast	1361	951	2312	787	559	1346
Bone	1670	1170	2840	2952	2080	5032
Skin	NA	NA	NA	NA	NA	NA
Gastrointestinal (d)	2484	1748	4232	1119	785	1904
Other (d)	1821	1278	3099	1545	1089	2634
Thyroid-malignant	1348	945	2293	420	298	718
Thyroid-benign	337	157	494	105	50	155

(a) TNCS cost for first admission is increased by 20 percent to eliminate nonpayment bias.

(b) The ratio of first-year hospital costs to total hospital costs is used to allocate nonhospital costs calculated in Table 5.2 between treatment years.

(c) Costs for the second year represent average total costs less average first admission costs. Costs are increased by 20 percent to eliminate nonpayment bias.

(d) TNCS costs are weighted as shown in Table 5.1 to create these cost estimates.

Source: Scott, J. and L. Chlazze. 1976. Third National Cancer Survey: Hospitalizations and Payments to Hospitals. HEW Publ. No. NIH 76-1094. Table 110A.

TABLE 5.4. Direct Costs of Cancer Care by Cancer Type (1984 \$)

Cancer Type	First Year	Second Year
Leukemia	9,003	11,429
Lung	11,643	7,795
Breast	7,275	4,236
Bone	8,937	15,835
Skin(a)	1,723	0
Gastrointestinal	13,317	5,991
Other	9,752	8,289
Thyroid-malignant	7,216	2,259
Thyroid-benign	1,555	488

(a) Developed from National Medical Utilization and Expenditure Survey, 1980 (National Center for Health Statistics, 1983).

Source: Table 5.3 and the Consumer Price Index all medical care components from the U.S. Bureau of Labor Statistics. (Monthly)

risk model applies, cancer incidence and fatalities are allocated to the exposed age groups in proportion to the relative number of expected person-years at risk for each age group in the exposed population^(a). Then the probability of developing cancer is calculated for each year after the minimum latency period until each age group reaches the maximum age at the end of the risk period, whichever occurs sooner. Using the absolute risk model, the induced cancer rate per surviving person per year is constant over the remaining lifetime of the exposed population.

Cancer fatalities and incidence of the types to which the relative risk model applies are distributed to age and sex cohorts proportionally to the spontaneous cases expected over the number of person-years at risk. The number of years at risk for each cohort is a function of their age at exposure, the latency period and the maximum age simulated. In allocating cancers affecting a cohort to particular years, proportionality is maintained between the induced cancer rate and the spontaneous rate over time.

The cost estimates shown in 1970 dollars in Table 5.3 have been inflated to 1984 dollars using the all medical care component of the CPI. Table 5.4 presents the resulting estimates for each cancer category. These are the costs presently being used in the base case of PNL's Health Effects Cost Model (HECOM). They can be converted from 1984 dollars to any other year's dollars using the medical care cost component of the CPI.

(a) Exceptions occur for breast cancer which is only allocated to females and also for thyroid cancer and benign nodules. Thyroid neoplasms are allocated to those under age 18 at twice the adult rate and to females at 2.5 times the rate for males. Incidence data supporting this allocation are summarized by Evans, Moeller and Cooper (1985, Vol. 2, p. 194-195).

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6.0 DIRECT COST ESTIMATION FOR GENETIC EFFECTS

Estimating the costs of radiation-induced genetic disease is a task made difficult by both conceptual issues and limited information. In this section we examine some relevant conceptual issues, describe an approach to estimating costs, and apply limited data within that approach to construct a cost estimate.

In Section 3.0 we suggested several reasons why individuals would prefer lower health risks: because they value life itself, for themselves (v1), for their loved ones (v2), and for anonymous others (v3); because they prefer not to lose the net production of others' labor (v4); and because they prefer not to bear the resource costs of treating others' illness (v5). In estimating the costs of radiation injuries and radiation-related cancers, we have proposed that since the sum of direct and indirect costs accounts for most of v1, v4, and v5, that sum is a reasonable approximation of total costs.

With respect to genetic disease, the rationale for use of direct and indirect cost measures is similar, though there are difficult conceptual issues involved. For instance, if genetic disease affects only future generations and not this one, does an estimate of future direct costs measure v5 for this present generation? And does an estimate of the loss of future earnings measure either v1 or v4 to this generation? Those living are not at risk from genetic disease and need bear the cost of those health effects little, if at all; why then, should we value either resources consumed by future generations (v5), or net production (earnings) forgone (v4)? And if we are not at risk, why include a measure of forgone consumption (v1) as a measure of loss from genetic mortality?

The answer lies to some extent in the fact that generations overlap; people now living will actively share in v4 and v5 for the next generation and to a lesser, but still positive, extent in that of the second generation hence. In addition, the satisfaction ("utility") of this generation is usually considered to depend not only on one's own opportunities but on the income and consumption opportunities of future generations. Thus, the welfare of future generations affects this generation directly to the extent that they will soon co-exist with us, and indirectly to the extent that our levels of satisfaction depend partly on theirs.

Employing direct and indirect costs as a measure of this generation's valuation of future health effects goes even further than mere concern for the future. It treats future generations in an egalitarian way, valuing their health effects as though they were our own. That is, if v1 is a measure of how much an individual values his own life (because it is a measure of his future consumption), then it is an appropriate component of the valuation of health risk only if the individual is among those at risk. Therefore, for this generation to consider direct and indirect costs, i.e., v1 + v4 + v5, as the valuation of genetic effects means that this generation evaluates those health effects on the same basis as if we were the ones at risk.

In practice, these future costs are discounted to present value, just as costs incurred later in this generation would be. Discounting results in a measure of the funds that would need to be placed in an annuity at the time of a reactor accident in order to pay the costs occurring at some future date.

Given that future direct costs are an appropriate measure of this generation's valuation of genetic effects, there remain a number of problems in estimating those direct costs. This section presents an approach to estimating the direct costs of genetic disease; the associated indirect costs are examined in Chapter 7. The major types of genetic effects that may be induced by radiation exposure of reproductive cells are described in Section 6.1. Some clinical manifestations of disease and disability are described and estimates are presented of the degree of life impairment resulting from each category of effect. Section 6.2 provides cost information on medical and institutional care. The methodology described in Section 6.3 is then used to develop cost estimates. Assumptions made to project future genetic effect costs are summarized in Section 6.4.

6.1 INCIDENCE AND IMPACTS

There are several impediments to estimating the genetic effects of increased radiation levels. First, evidence of the linkage between radiation and genetic damage in humans is weak. Since radiation causes identifiable mutation in other mammals, geneticists generally agree that radiation has the potential to cause harmful mutations in humans. However, difficult questions remain concerning what kinds of genetic disorders may be caused by radiation and how the dose-response relationship may be quantified. Even if the effects in terms of genetic material changes were identified and quantified, there remains an imposing problem of predicting the nature and severity of clinical manifestations (observable diseases) for each type of genetic damage. In this study we are interested in the clinical manifestations of genetic damage as observable, impact-producing health effects. The effects of inherited disorders, such as blindness, muscular dystrophy, chorea, and kidney disease, produce costs for society through requirements for treatment and losses in productivity.

To project the impact of genetic disease, both the types of diseases that may occur and their relative frequency of occurrence must be known. Information about the nature of genetic-related disease has been expanding rapidly. For example, in 1966 McKusick cataloged 169 diseases categorized as autosomal dominant disorders; by 1978 a total of 736 were listed (with another 753 not yet fully confirmed) (McKusick 1978). Similar growth in knowledge has occurred for the other types of genetic disorders as well. Stevenson (1959), Trimble and Doughty (1974) and Carter (1977) all provide lists of clinical diseases classified according to category of genetic disorder. Those lists are usually employed to estimate the current incidence of genetic effects. Estimates of the relative frequency of various genetic diseases vary depending on the disease classifications used as the basis for enumeration and on the population studied. It is apparent that different populations have widely varying rates for some genetic diseases.

There are four major categories of genetic effects that may be caused by radiation-induced mutation: dominant single-gene effects, recessive single-gene effects, multifactorial diseases, and chromosomal anomalies or aberrations (Evans, Moeller and Cooper 1985, Vol 2, p. 143). Of these, only autosomal dominant, X-linked recessive and chromosomal effects have been considered in developing cost estimates. The impact of induced, regularly inherited recessive genetic damage is too uncertain and too remote in time (over 3000 years) for analysis (Vol. 2, p. 145). Multifactorial diseases involve interaction of a genetic factor with environmental conditions to produce disease or a combination of mutant genes. While these constitute the majority of spontaneously occurring genetic effects, the uncertainties regarding induction rates are too great to permit their projection to each future generation (Evans, Moeller and Cooper, 1985, Vol. 2, p. 149). Major subcategories of dominant and chromosomal disorders for which we develop cost estimates are described below.

6.1.1 Autosomal Dominant Disorders

Among the categories of genetic damage, autosomal dominant disorders have special importance in radiation genetics: the relationship between the mutation rate and the frequency of birth defects is relatively direct, and a radiation-induced increase in the mutation rate is expressed most strongly in early generations (Carter 1977). The collective incidence of autosomal dominant disorders is roughly one percent of persons born (Stevenson 1959; Carter 1977; Oftedal and Searle 1980). Trimble and Doughty (1974) estimate the incidence at only 0.1 percent, but they ignore late-onset diseases.

Dominant disorders may affect the quality of life, the length of life, or both. Some, such as congenital deafness, retinoblastoma, and osteogenesis imperfecta, are evident at birth or early in childhood. Others involve progressive impairments or, like Huntington's chorea, develop in adult life. Most involve some shortening of the potential life span. Abrahamson, et al. weighted estimates of life shortening and impairment by the frequency of disorder occurrence to estimate impacts of dominant disorders as a category (Evans, Moeller and Cooper, 1985, Vol. 2, p. 155). Their estimate for dominant disorders is 25 years of life with a 33 percent disability level and a 13-year reduction in life expectancy (p. 155).

6.1.2 X-Linked Disorders

Another category of genetic disorders that would almost directly reflect a radiation-induced increase in the mutation rate are X-linked disorders. These mutations involve genes located on the X chromosome and behave as dominants in males. Their numbers are typically included with the dominants in a single category in estimates of radiation-induced genetic effects. As with the dominants, these disorders appear most frequently in the early generations after a one-time increase in the mutation rate. The current incidence of X-linked disorders is approximately 0.8 per 1000 liveborn males. (Stevenson 1959; Trimble and Doughty 1974; Carter 1977).

The major types of X-linked disorders become apparent early in life and involve both life shortening and functional impairment. Frequency weighted average estimates are 28 years of life lost plus 40 years of life with a 40 percent disability level (Evans, Moeller and Cooper 1985, Vol. 2, p. 155). Hemophilia and Duchenne's dystrophy are among the more frequent types of X-linked disorders.

6.1.3 Chromosome Aberrations and Anomalies

In addition to the genetic mutations discussed above, radiation exposure may cause a broad class of chromosome anomalies. This class includes two types of disorders: numerical changes (anomalies) and translocations or structural changes (aberrations) (Evans, Moeller and Cooper, (1985, Vol. 2, p. 146). Chromosome deletions may have effects indistinguishable from those of single-gene mutation and they are included among those disorders. The numerical anomalies (called aneuploids) contribute heavily to very early prenatal mortality, accounting for approximately 50 percent of spontaneous abortions, often so early that pregnancy is undetected (Denniston 1982, p. 331). They also result in genetic diseases such as Down's syndrome, Turner's syndrome, and Klinefelter's syndrome. Unbalanced translocations generally result in severe deformity and early mortality. As a class, chromosome anomalies lead to impairment in approximately 0.6 percent of livebirths, according to Denniston (p. 331).

The impacts in terms of life shortening are estimated to be 50 years for unbalanced translocation disorders and 24 years for aneuploidy. Disability may average 95 percent during their 20-year life span for those with unbalanced translocation disorders and 50 percent over 44 years for aneuploidy (Evans, Moeller and Cooper 1985, Vol. 1, p. 11).

6.2 CDST DATA

Information on the costs of treating disabilities and diseases that are genetic in origin is very limited. In this section we rely on data from four studies to estimate the magnitude of the direct costs of genetic effects.

Hall et al. (1978) present data on the treatment at one urban medical center of children with genetic disorders. That study reports an average cost per hospital admission of approximately \$1415 (1984 dollars) for children with diseases unambiguously attributable to genetic causes. Those children had been admitted to the hospital an average of 5.3 times each over an unspecified time period when records were reviewed for Hall's study. If that were the total number of admissions per child, the total hospital cost per child would average approximately \$7500 (1984 dollars). Of course, there is no reason to assume that the end of the study coincided with the end of hospitalizations for the children sampled, so \$7500 is doubtless an underestimate of the lifetime average total hospital costs.

Analysis of National Medical Care Utilization and Expenditure Survey, 1980 (National Center for Health Statistics 1983) data produced a cost estimate of

\$6073 in 1984 dollars. This represents average total cost annually for treatment of conditions attributable to congenital anomalies. No data are available on what percentage of individuals with genetic effects incur this level of costs.

In addition to the costs of acute care, a portion of the genetically diseased population also will incur costs for long-term institutional care. Conley and Milunsky (1975) examine the cost of institutional care for individuals with Down's and Hunter's Syndromes. Those syndromes are related to chromosome anomalies and would account for a small percentage of the genetic diseases associated with radiation exposure (UNSCEAR 1977 and BEIR III 1980). However, costs for those two syndromes may be somewhat representative of the costs for long-term institutionalization of other genetically impaired individuals. Assuming the costs are representative, the cost of institutional care would be approximately \$12,800 annually in 1984 dollars (inflating Conley and Milunsky's 1972 estimates by the medical care component of the Consumer Price Index).

Conley and Milunsky report that approximately 20 percent of the cost of institutionalization is comprised of normal personal consumption and should not be considered to be a result of disease. Subtracting that amount, the incremental costs of illness are \$10,300 per year.

This cost estimate is corroborated by a New York State study of institutionalization costs for mentally retarded persons. That study estimated annual costs that, when similarly adjusted, range from \$10,000 to \$15,500 (1984 dollars) (Intagliata, Wilder and Cooley 1979. p. 155).

6.3 COST ESTIMATION

Impacts of genetic effects associated with increases in radiation exposure may range in severity from color blindness to mortal or debilitating diseases. Obviously the direct and indirect costs will vary as well.

The direct costs of genetic effects result from requirements for both medical services and physical care. We employ the Hall et al. (1978) medical cost estimate of \$7500 since it is the better documented of the two estimates. These costs are based on treatment of defects requiring hospitalization, while less severe disorders are also included in the range embodied in incidence estimates. Because measures of medical care requirements for various genetic disorders are lacking, we assume the medical cost estimate applies to all cases. Since information on the timing of medical treatment is also unavailable, we assume that the medical costs are incurred in the first year of life. These two assumptions offset to some degree the understatement of medical costs by the available data.

Institutional care costs are applied to genetic effect incidence in proportion to the average degree of impairment estimated by Evans, Moeller and Cooper (1985, Vol. 1, p. 11). An annual cost of \$12,500 is used to represent

the range of cost estimates in the literature. We project that the years in which the impairment occurs will occur toward the end of the individual's life time.

In the HECOM base case we allocate affected births over five generations after the hypothetical reactor accident. The defects projected for the first generation are allotted to the first 30 years, those for the second generation the second 30 years, and so forth. Within each generation genetically impaired births are distributed evenly over the 30-year period.

Medical and institutional care costs are applied to each birth distributed over the generations considered. These costs are then discounted to their present value in the year of the hypothetical accident. That process yields an estimate of the funds that could be placed in an annuity at the time of a reactor accident to pay for future direct costs of genetic effects. There is an enduring question in economic theory concerning the appropriate discount rate for analysis of intergenerational cost streams. Because the discount rate must be treated as an important factor in any evaluation of future costs, a sensitivity analysis including the application of different discount rates is presented in Chapter 9.

6.4 SUMMARY

The uncertainty inherent in genetic-related disease projections is very great because of major information gaps in each stage of the projection process. Given the state-of-the-art, PNL uses the following assumptions to construct genetic effect cost estimates:

1. Most genetic effects would occur within five generations.
2. Genetic effects have an equal probability of affecting births in each year of a generational period.
3. Medical care costs are incurred in the first year of life.
4. Institutional care costs are incurred in the last years of life in proportion to disability levels.
5. Autosomal dominant and X-linked disorders are eliminated from the population at a rate of 20 percent per generation and chromosomal disorders at a rate of 33 percent (Evans, Moeller and Cooper 1985, Chapter 5). The elimination rate for unbalanced translocations is 100 percent and 40 percent for aneuploidy.

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7.0 ESTIMATION OF INDIRECT COSTS OF HEALTH EFFECTS

In addition to the direct costs of treating radiation-induced illnesses, there are potentially much larger indirect costs associated with those health effects. Indirect costs do not involve monetary outlays, but rather represent other losses incurred by society as a result of the health effects. In Section 3.0 we presented a conceptual discussion of how those other societal losses might be valued. Using a "human capital" approach, societal losses due to increases in illness and premature mortality are measured in terms of the value of lost production. That is, when an individual is too sick to work or when he or she dies earlier than might be expected, that person produces less. Because wages are a measure of the value of a person's marginal product, the value of the lost production is measured in terms of the value of lost earnings.

The value of earnings lost due to increased morbidity or premature mortality provides an approximate measure of two components of the societal losses due to illness. Lost earnings mean lost consumption to the individual. (That corresponds to v1 in the taxonomy employed in Chapter 3. However, as explained there, lost earnings seriously underestimate v1). The rest of society incurs a loss as well, consisting of the value of what the individual would have produced over and above what he or she would consume. (That is a measure of net production and it corresponds to v4 in Chapter 3).

The value of lost earnings is an underestimate of the full indirect costs because it ignores both the loss to loved ones (v2) and the loss society in general feels purely out of beneficence (v3). Furthermore, using an individual's lost earnings as the measure of lost production ignores the production lost by family and friends who take time out to care for the stricken individual.

There is another way in which the use of earnings underrepresents the full indirect costs: earnings data do not reflect the value of services performed in the home. In this study we employ two measures to avoid that shortcoming. First, we consider the population for whom indirect costs occur to be all non-institutionalized individuals, not just persons in the labor force. Second, within each age and sex cohort, we apply the average earnings of employed individuals to all noninstitutionalized persons in the cohort. That is, the production of a female homemaker, aged 35, is considered to be equal in value to that of an employed woman of the same age. (This method treats all males equally as well, although it does not treat men and women equally.)

The following sections relate how lost earnings measures of indirect costs are applied to evaluate both morbidity and mortality related to radiation-induced health effects. In general, several causes of lost production are associated with health effects: inability to work during acute phases of radiation injury or cancer, reduction in capabilities as a result of the illness, inability to work because of mental or physical impairment from prenatal injury or genetic defect, and permanent cessation of work due to early mortality. In this study, we explicitly calculate costs related to each cause except

illness-related reduction in capabilities, for which information on degree of impairment was unavailable for most of the illnesses considered.

7.1 INDIRECT COSTS OF MORBIDITY

For all types of health effects, the indirect cost of morbidity is estimated from the amount of work lost, valued by expected earnings. Estimates of work weeks lost for each type of health effect are inputs to HECOM. Lost production time is valued by projecting average earnings for individuals of a particular age and sex in each post-exposure time period. The incidence of illness resulting in lost work time is allocated across age and sex cohorts in proportion to their relative numbers and relative risks during the time period in which they would face health effects risks. The projected stream of future costs is then discounted to present value as of the year of a hypothetical reactor accident.

Estimates of work weeks lost are developed for each type of health effect from several different data sources. Values for acute radiation injuries and for latent cancers are given in Appendix A.4.3.

Indirect costs of morbidity associated with genetic effects are based on estimates of duration and degree of impairment rather than lost work weeks. Estimates of the time period and percent of disability are inputs to HECOM. These are applied to projections of expected earnings for each age cohort, given prior mortality probabilities. HECOM considers the incidence of genetic disorders over the number of generations specified by the user. (The base case is five generations.) Calculated indirect cost streams are then discounted and summed to a present value estimate at the time of population exposure.

7.2 INDIRECT COST OF MORTALITY

The indirect cost of mortality is valued by the earnings lost as a result of exposed individuals dying earlier than would be expected in an unexposed population. The basic computation is most easily seen in an example: for an individual who dies at the age of 30, the indirect cost would be the discounted sum of his or her expected future earnings given prior mortality probabilities. It is assumed that in each potential year of life after age 30 the individual would have produced (and therefore earned) a value equal to the average for his or her age and sex. The average earnings in each future year are weighted by the probability that the individual would have survived to that age, had he or she not died at age 30 due to radiation exposure.

Fatalities from acute radiation injuries are assumed to affect individuals of each age and sex cohort in proportion to their relative numbers in the total population. These fatalities are assumed to occur in the first year after exposure. Thus, mortality-related economic losses begin in the year of the accident and continue for many years, until all those exposed would have been dead of other causes. The total indirect cost is the sum of the discounted stream of future earnings for each fatally exposed individual.

Cancer-related mortality costs are calculated in a similar manner, except that cancer fatalities, and therefore the onset of losses, occur over a period of years. Estimates of cancer fatalities are assigned to age and sex cohorts in proportion to their risks of radiation-induced cancers and to their relative numbers in the population. Each type of cancer has a specific minimum latency period (see Appendix A.2.2 for data) between exposure and the onset of cancer symptoms. After the latency period has passed, individuals are expected to develop cancers and to die from those cancers over what would have been their normal lifetime given their age at exposure. That is, not all individuals will show cancer symptoms in the years immediately following the end of the latency period, and even after the onset of symptoms, some people will not die for many years.

Two methods are used to distribute cancer fatalities over time, an absolute and a relative risk model. With the absolute risk model cancer fatalities are allocated so that the induced cancer mortality rate in each year after the latency period plus median survival time until the end of a normal life span is constant. For example, consider that two persons in the 30 year-old age group at time of exposure will contract a fatal bone cancer. After a 10-year minimum latency period between exposure and bone cancer symptoms and a two-year median survival period, the probability of fatality in each succeeding year is treated as being proportional to the probability of survival in each remaining year of normal life expectancy. The resulting fatality rate due to bone cancer is constant over the remaining lifetime of the 30 year-old cohort. This probability of death in each succeeding year is applied to the value of the earnings that would be lost if an individual from that age cohort died in that year. The total indirect cost is the sum of the discounted stream of probabilistically weighted future losses for each projected fatality.

The relative risk model treats cancer fatalities as occurring in a constant proportional relationship to the mortality rate for spontaneously generated cancers. Projected bone cancer fatalities are allocated to each year after the end of the latency period plus median survival time in a constant proportion to the naturally occurring bone cancer fatality rate. The economic losses associated with the radiation-induced cancer mortality are then discounted to the year of exposure and summed.

In the case of genetic disorders, indirect costs are projected for the average period of life shortening for each genetic effect type. These cost streams for persons born in future generations are then discounted to a present value indirect cost estimate.

8.0 OVERVIEW OF HECOM STRUCTURE

This chapter provides an overview of the Health Effects Costs Model (HECOM) structure and processes. The equations underlying each major subroutine and function of the code are presented and discussed. (The code itself is listed in Appendix B.) Descriptions of the input data and file structure are contained in HECOM: Health Effects Cost Model Version 2.0 User's Manual (Pacific Northwest Laboratory 1986).

In the remainder of this chapter the subroutine and function descriptions are grouped by topical area. Section 8.1 describes the overall model structure and briefly indicates the purpose of the utility subroutines. The method of developing population and earnings projections is presented in Section 8.2 and the treatment of time-value of money in Section 8.3. Direct costs are covered in the next four sections. Section 8.4 describes the cost model for medical management of an exposed population. Section 8.5 does the same for radiation injuries, Section 8.6 for cancers and 8.7 for genetic effects. Section 8.8 discusses modeling of the indirect costs for illness and Section 8.9 the indirect costs of early mortality.

8.1 UTILITY SUBROUTINES

HECOM consists of a main program of call statements and a number of subroutines and functions that are either called directly by the main program or by a subroutine. A brief description is provided below for each of the utility subroutines. The health effect subroutines are grouped in modules and the calling of these modules is controlled by the user through input parameters. This structure permits the user to estimate costs for any one or combination of the medical management, radiation injury, cancer or genetic effect modules.

Subroutine READER

This subroutine opens files and reads in data for each of the HECOM variables.

Subroutine SKIP

As data are read in, this subroutine skips over comments in the input file.

Subroutine INIT

This subroutine initializes values of all calculated variables at zero.

Subroutine SETCCD

Creation of a file of HECOM output for up to 200 case runs is permitted by this subroutine. This is for use in developing a complementary, cumulative distribution function for health effects costs.

Subroutine SUMTOT

This subroutine sums costs by categories and calculates total costs.

Subroutine WRITER

HECOM output is written by this subroutine.

8.2 POPULATION AND EARNINGS PROJECTIONS

HECOM simulates health effect costs based on demographic characteristics such as survival probabilities, existing cancer morbidity and mortality rates, labor force participation, and earnings. Section 8.2.1 presents the functions used to project population characteristics. The subroutine and function used in the earnings projections are described in Section 8.2.2.

8.2.1 Projection of Cohort Survival Probabilities

This section describes the projection of cohort survival probabilities subject to the baseline mortality rates of the population considered. The existing pattern of mortality is used as the background against which radiation-induced health effects are projected. An array of conditional survival probabilities by age category, sex and year after the base year is projected to indicate the probability that a person of any age category and sex will attain each subsequent age.

Function LPRBRL

This subroutine calculates the prior probability that a person of a given sex and age category in the year of exposure would live to any subsequent age. Annual survival probabilities by age (defined in single years) and sex are the only input data.

$$\text{LPRBRL}(n)_{a,s} = \text{LIFEP}(a+1)_{a,s} \cdot \text{LIFEP}(a+2)_{a+1,s} \cdots \cdot \text{LIFEP}(a+n)_{a+n-1,s}$$

where $\text{LPRBRL}(n)_{a,s}$ = prior probability that a person of age a in the base year and of sex s will live to year n

$\text{LIFEP}(a+1)_{a,s}$ = probability that a person of age a and sex s will live to age $a+1$

n = year subsequent to base year, or birth year in the case of genetic effects.

Function AGECA

This function determines the age category associated with any given age, as required for the simulated aging of the exposed populations. For instance,

labor force participation rate categories and cancer mortality rate categories change as a cohort ages. If the age equals zero, the prenatal age category is used. AGECAT is used to maintain consistent data intervals for variables related to population characteristics.

8.2.2 Earnings Projections

Labor value projections by sex and age category are used to calculate indirect illness and fatality costs. Median labor value in each future year, for each cohort, is calculated from base year median earnings and the rate of real income growth.

Subroutine LVALUE

This subroutine calculates labor value by age category and sex in each year after the base year. Inputs to the subroutine include earnings in the year of exposure by age category and sex, labor force participation rates and the rate of real earnings growth. The following equation is used to calculate labor value in each future year:

$$LVAL(n)_{a,s} = MERN_{a,s} \cdot LFPR_{a,s} (1 + REG/100)^{n-1}$$

where $LVAL(n)_{a,s}$ = future value of earnings by age (at exposure) category a and sex s in year n after exposure

$MERN_{a,s}$ = median earnings in the base year for age category a and sex s

$LFPR_{a,s}$ = labor force participation rate of age category a and sex s

REG = rate of real earnings growth.

Within this function the age category of the group being processed is determined using the function INCCAT and real income in the year being processed is calculated using the function FV, described in Section 8.3.2.

Function INCCAT

This function determines the earnings category associated with each age. The function checks to see if current age is zero (in utero). If the age is not zero, the income category is determined by comparing the age to the upper age boundary of each income category. If the age is determined to be less than the upper age boundary of an income category, the function returns the number of that income category. A separate comparison is made for the in-utero category because, unlike the other age categories, all its members are of the same age and do not change income categories in the same years as the other age categories. If the age is zero, INCCAT returns an income category of one (1) since the in-utero age category is assigned to the first income category.

8.3 TIME VALUE OF MONEY

HECOM is designed to project money expenditures occurring at different times in real (i.e., uninflated) terms. The model employs two functions to do this: PV and FV. In function PV a real discount rate is used to calculate the equivalent present value of monetary expenditures occurring in future years. Future values are calculated in function FV using real rates of increase in costs or incomes over time. The formulas used in each of these functions are explained below. They are employed in the direct and indirect cost subroutines.

8.3.1 Function PV

This function determines the present value of a number based on the discount rate and number of years to be simulated. The following equation is used:

$$PV = V(n) / (1 + R/100)^{n+b-1}$$

where PV = present value of number

V(n) = value of variable in year n

R = real discount rate

n = years subsequent to the base year

b = difference between base year and years in which dollars of the input data are stated.

This equation is designed to permit simulation of costs for accidental exposures occurring at various points in time. The base year can be specified in terms of time lapse from the year's dollars in which the input data are expressed.

8.3.2 Function FV

The time dimension of potential health effects also necessitates modeling of changes in the real values of treatment of costs and productivity losses. Function FV calculates the future value of a number given the initial value, real rate of growth and number of future years simulated. The expected costs of morbidity or mortality are calculated using the following equation:

$$FV = V(i) \cdot (1 + G/100)^{n+b-1}$$

where FV = future value of a number

V(i) = initial value of a variable

G = real rate of growth.

8.4 DIRECT COSTS OF MEDICAL MANAGEMENT OF EXPOSED POPULATIONS

As discussed in Section 4.1, accidental exposure of a population would entail commitment of resources to assess possible contamination and to identify individuals who require treatment. If there were any injuries, transportation to treatment facilities would be required. Costs of these efforts are projected by three subroutines and then summed by a fourth. The screening and decontamination subroutine RADSCREEN is described in Section 8.4.1. This is followed in Section 8.4.2 by the MEDMON subroutine which estimates costs of clinically monitoring individuals at greatest risk for radiation injuries. Section 8.4.3 presents the medical transportation costs subroutine MEDTRAN. The cost summary, MONITOR is described in Section 8.4.4.

8.4.1 Subroutine RADSCREEN

This subroutine projects costs for radiation screening of a population that may have been accidentally exposed to radiation. Input data are required for physical survey and resurvey costs per person and also for costs of a whole body count. These costs per person are applied to two population groups for which input data are required. Of course, definitions of the population groups receiving screening may be varied as desired. The equation for radiation screening cost is shown below.

$$\text{RADCST} = \text{ASC} \cdot (\text{PSD} + \text{BSC}) \cdot (\text{PSD} + \text{ISC}) \cdot \text{PE}$$

where RADCST = radiation screening cost

PSD = population in the sector 90° downwind

PE = population evacuated

ASC = initial survey cost per person

BSC = resurvey cost per person

ISC = internal survey cost (whole body count) per person.

8.4.2 Subroutine MEDMON

This subroutine calculates medical monitoring costs for an exposed population exhibiting early symptoms of illness or considered at risk for development of illness. The cost components are medical services costs and laboratory

costs per person. The population input data value may represent those exposed to over one Gy or any other relevant group.

$$\text{RADMNC} = (\text{MSC} + \text{LTC}) \text{ PSE}$$

where RADMNC = medical monitoring cost

MSC = medical services cost per person

LTC = laboratory testing cost per person

PSE = population exposed to over one Gy.

8.4.3 Subroutine MEDTRAN

The transportation cost calculation requires input data for capacities at definitive care centers, regional hospitals and hospitals nationwide. Average distances to each of these types of facilities are required as well as air and motor vehicle transportation costs per person per mile. Costs are calculated in the equation shown below.

$$\begin{aligned} \text{TRNSCT} = & (\text{PHE} \leq \text{DCC}) \text{ DCD} \cdot \text{ATC} + [(\text{PHE} - \text{DCC}) \leq \text{RHC}] \text{ RHD} \cdot \text{MVC} \\ & + [(\text{PHE} - \text{DCC} - \text{RHC}) \leq \text{NHC}] \cdot \text{NHD} \cdot \text{ATC} + (\text{PHE} - \text{DCC} \\ & - \text{RHC} - \text{NHC}) \text{ RHD} \cdot \text{MVC} \end{aligned}$$

where TRNSCT = patient transportation cost

PHE = population exposed to over 2 Gy

ATC = air transportation cost per mile

MVC = motor vehicle transportation cost per mile

DCD = average round trip miles to definitive care centers

RHD = average round trip miles to regional hospitals

NHD = average round trip miles to hospitals nationwide, outside the region

DCC = patient capacity of definitive care center

RHC = patient capacity of regional hospitals

NHC = patient capacity nationwide, excluding the region.

The subroutine for transportation costs assumes that the first choice of treatment facility would be the definitive care centers so long as the number injured does not exceed their capacity. If the number of injured is larger, it is assumed that the less severely injured would be sent to regional hospitals with adequate facilities or if more capacity is needed, to similar hospitals nationally. In the highly unlikely event that national capacity in adequate facilities is exceeded, it is assumed that the less severely injured would be sent to less well-equipped regional hospitals.

8.4.4 Subroutine MONITOR

Medical management costs for an exposed population are summarized in the equation below. This includes radiation screening, medical monitoring and medical transportation costs. These costs are not differentiated by sex or age category.

$$MGTCST = RADCST + RMDMNC + TRNCST$$

where MGTCST = total cost of medical management

RADCST = radiation screening cost

RMDMNC = medical monitoring cost

TRNCST = patient transportation cost

8.5 DIRECT COSTS OF RADIATION INJURIES

Direct costs of radiation injuries are composed of the costs of treatment for both the injured who survive and for fatalities. The flow of information involved in calculating radiation treatment costs is shown in Figure 8.1. As the diagram illustrates, costs are calculated separately for early and for later and continuing effects.

These costs are developed from data for the fraction of the population in each age cohort, treatment costs by injury type, and radiation injury and fatality incidence projections. All direct radiation injury costs are assumed to occur in the first year for those injuries that are immediately evident. Costs of caring for persons with mental retardation and microcephaly, on the other hand, are assumed to continue for a lifetime. The costs of early effects of radiation injuries are modeled in Section 8.5.1. Both morbidity- and mortality-related costs are included. Later and continuing effects are presented in Section 8.5.2.

8.5.1 Early Effects

The health impacts of radiation exposure that take the form of prodromal symptoms, hematopoietic syndrome, gastrointestinal syndrome, pulmonary

FIGURE 8.1. HECOM Calculation of the Direct Costs of Radiation Injuries

syndrome, prenatal death, acute thyroiditis, and skin damage would occur entirely within the first year after exposure. These radiation injuries are modeled as a group, with separate subroutines for morbidity and mortality.

Subroutine RADEMORB

This subroutine allocates morbidity occurring in the first year after exposure to population cohorts. Population fractions by age and sex and numbers of radiation injuries by type are the input variables.

$$RMPYE_{a,s,r} = RADIE_r \cdot POPFE_{a,s}$$

where $RMPYE_{a,s,r}$ = morbidity in year $n = 1$ for age group a , sex s and radiation injury type r

$RADIE_r$ = early radiation injury type r

$POPFE_{a,s}$ = population fraction (excluding in utero) in age category a and sex s .

Pulmonary injuries are allocated to all age categories, unlike pulmonary fatalities which are restricted to young and older age groups in RADEFAT.

Subroutine RADEFAT

This subroutine allocates fatalities occurring in the first year after exposure to population cohorts. Population functions by age and sex and numbers of fatalities by type are the input variables.

$$RFPYE_{a,s,r} = RADFE_r \cdot POPFE_{a,s}$$

where $RFPYE_{a,s,r}$ = fatalities per year in year $n = 1$ for age group a , sex s and radiation injury type r

$RADFE_r$ = radiation-related fatalities of type r .

In the case of pulmonary syndrome, the equation above is modified so that all fatalities occur among children under age 5 and adults over age 50.

For prenatal deaths the fatalities are allocated to each sex by the equation:

$$RFPYE_{p,s,r} = RADPFE_r / SEX$$

where $RADPFE_r$ = radiation-related prenatal/neonatal fatality of type r

p = prenatal

SEX = number of sex categories

Subroutine RADECOST

This subroutine calculates the cost of treating early fatal or nonfatal radiation injuries. Input items are the cost of treating each type of radiation injury and the incidence of fatal and nonfatal radiation injuries by cohort developing in the first year after exposure. The equation for nonfatal early radiation injury treatment costs is:

$$RECSTM_{s,r} = \sum_{a=1}^{AC} [RMPYE_{a,s,r} \cdot CPROADE_r (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}]$$

where $RECSTM_{s,r}$ = direct cost for early, nonfatal radiation injuries of type r affecting sex s

$CPROADE_r$ = treatment cost for an early radiation injury of type r

RHG = real treatment cost escalation rate

R = discount rate.

AC = number of age categories

For early fatalities including prenatal deaths the direct cost equation is:

$$RECSTF_{s,r} = \sum_{a=1}^{AC+1} [RFPYE_{a,s,r} \cdot CPROADE_r (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}]$$

where $RECSTF_{s,r}$ = direct cost for early fatal radiation injuries of type r affecting sex s .

8.5.2 Later and Continuing Effects

Some radiation injuries become evident more than a year after exposure or have effects that continue over time. Because of their similarities, hypothyroidism and cataracts are modeled together. A separate equation is employed for the continuing effects of microcephaly and mental retardation.

Subroutine RADLSICK

This subroutine allocates morbidity occurring after the first year to population cohorts in proportion to their remaining life years.

$$RMORB_{a,s,r} = RADLI_r \cdot POPFE_{a,s} \cdot \sum_{n=z}^{YEARS} LPRBRL(n)_{a,s} / \sum_{s=1}^2 \sum_{a=1}^{AC}$$

$$(POPFE_{a,s} \cdot \sum_{n=z}^{YEARS} LPRBRL(n)_{a,s})$$

where $RMORB_{a,s,r}$ = morbidity of radiation injury type r affecting age group a and sex s

$RADLI_r$ = late and continuing radiation injuries of type r

$LPRBRL(n)_{a,s}$ = prior survival probability for year n of age category a and sex s

$YEARS$ = maximum attainable age

$MAGE_a$ = median age of age category a

LPR_r = latency period for radiation injury type r

$z = MAGE_a + LPR_r$.

For prenatal injuries such as microcephaly and mental retardation, the equation allocating injuries by sex is:

$$RMORBP_{p,s,r} = RADLPI_r / SEX$$

where $RADLPI_r$ = prenatal radiation injury of type r

SEX = the number of sex categories.

Subroutine SUMRLM

This subroutine calculates the sum used in Function RMPYL to distribute later and continuing radiation injuries to future years. The equation follows:

$$SMRMRB_{a,s,r} = \sum_{n=z}^{YEARS} (LPRBRL(n)_{a,s} \cdot POPFE_{a,s})$$

where $SMRMRB_{a,s,r}$ = intermediate summation used in distribution of later radiation injuries for each age category a, sex s and radiation injury type r

$$z = MAGE_a + LPR_r.$$

Function RMPYL

Later radiation injuries are distributed to future years by this function.

$$RMPYL(n)_{a,s,r} = RMORB_{a,s,r} \cdot LPRBRL(n)_{a,s} \cdot POPFE_{a,s} / SMRMRB_{a,s,r}$$

where $RMPYL(n)_{a,s,r}$ = later radiation morbidity of type r occurring in year n for age category a and sex s.

Subroutine RADLCOST

Costs of treating later and continuing radiation injuries are calculated in this subroutine. The cost of treating later occurring radiation injuries with one-time treatment is calculated by the equation:

$$RLCSTM_{s,r} = \sum_{a=1}^{AC} \sum_{n=z}^{YEARS} [RMPYL(n)_{a,s,r} \cdot CPRADL_r (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}]$$

where $RLCSTM_{s,r}$ = total direct cost of later radiation-induced morbidity of type r affecting sex s

$CPRADL_r$ = treatment cost for later radiation injury of type r

$$z = MAGE_a + LPR_r.$$

Costs of continuing effects are calculated in the equation:

$$RLCSTM_{s,r} = \sum_{a=1}^{AC \text{ YEARS}} \sum_{n=z}^{YEARS} (RMPYL(n)_{a,s,r} \cdot \sum_{n=j}^{YEARS} [CPRADL_r (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}])$$

where j = year of onset after $MAGE_a + LPR_r$

Like continuing effects, prenatal injuries result in an annual direct cost. The cost equation is:

$$RLCSTM_{s,r} = \sum_{n=1}^{YEARS} [RMORB_{p,s,r} \cdot LPRBRL(n)_{a,s} \cdot RDISP_r / 100 \cdot CPRADL_r (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}]$$

where $RDISP_r$ = disability percentage for injury type r

$CPRADL_r$ = annual treatment cost for radiation injury type r .

8.6 DIRECT COSTS OF CANCERS

Direct costs of treating cancers are projected in a four-step process. First, input data for numbers of fatal and nonfatal cancers are allocated to age and sex cohorts. This allocation follows either an absolute or a relative risk model depending on the cancer type. In the HECOM base case the absolute risk model is applied to leukemia, bone, thyroid and skin cancers and cancers caused by in-utero exposure, while the relative risk model is employed for the remaining cancer types. Second, an intermediate sum is calculated for use in distributing cancers to future years. Then morbidity and mortality are projected to the years at risk for each population cohort. Finally, treatment costs are projected for the years in which cancers would be diagnosed and the following year, and then are discounted to present value cost estimates. Figure 8.2 shows the relationships between the subroutines and functions employed.

FIGURE 8.2. HECOM Calculation of the Direct Costs of Cancers, by Sex and Cancer Type

Section 8.6.1 presents the absolute and relative risk models employed to distribute cancer morbidity first to age and sex cohorts and then over years. The comparable models for cancer fatalities are described in Section 8.6.2 and the cost calculation in Section 8.6.3.

8.6.1 Nonfatal Cancer

Cancer morbidity is defined here as including only nonfatal cancers. Treatment costs of fatal cancers are included in the mortality subroutines. Allocation of input data for nonfatal cancers by cancer type to population cohorts is carried out by two similar subroutines employing different risk models. The absolute risk model subroutine is described in detail and differences in the relative risk model are noted.

Subroutine SICKAB

This subroutine distributes morbidity data by cancer type to age (at exposure) and sex cohorts in the exposed population using the absolute risk model. Input variables include the number of nonfatal cancers, minimal latency periods and risk periods, population fractions excluding the in-utero cohort, and conditional survival probabilities computed in the LPRBRL function. Cancers are allocated to cohorts based on each cohort's person-years at risk relative to those of the total population. The number of person-years at risk for each cohort depends on age-specific conditional survival probabilities, age at exposure, latency period and period of risk. Females and those under 18 years of age are assigned higher probabilities of developing thyroid cancer than the general population.

$$\text{MORB}_{a,s,c} = \text{CNONF}_c \cdot \text{POPFE}_{a,s} \cdot \sum_{n=z}^N \text{LPRBRL}(n)_{a,s} / \sum_{s=1}^2 \sum_{a=1}^{\text{AC}}$$

$$[\text{POPFE}_{a,s} \cdot \sum_{n=z}^N \text{LPRBRL}(n)_{a,s}]$$

where

N = the lesser of YEARS or $\text{MAGE}_a + \text{LPC}_c + \text{POR}_c$

$\text{MORB}_{a,s,c}$ = cases of nonfatal cancer for age (at exposure) category a , sex s and cancer type c

$\text{POPFE}_{a,s}$ = population fraction of age category a and sex s excluding in utero

$\text{LPRBRL}(n)_{a,s}$ = prior probability of survival to year n for members of age (at exposure) category a and sex s

CNONF_c = nonfatal cancer cases of type c

AC = number of age categories

YEARS = maximum attainable age

MAGE_a = median age of age category a

LPC_c = minimal latency period for cancer type c

POR_c = period of risk for cancer type c

$z = \text{MAGE}_a + \text{LPC}_c$.

Subroutine SICKREL

In this subroutine nonfatal cancers are allocated to age (at exposure) and sex cohorts proportionally to their spontaneously occurring cancer risks.

$$\text{MORB}_{a,s,c} = \text{CNONF}_c \cdot \text{POPFE}_{a,s} \cdot \sum_{n=z}^{\text{YEARS}} (\text{SPONMR}(n)_{a,c} \cdot \text{LPRBRL}(n)_{a,s}) / \sum_{s=1}^2 \sum_{a=1}^{\text{AC}}$$

$$[\text{POPFE}_{a,s} \cdot \sum_{n=z}^{\text{YEARS}} (\text{SPONMR}(n)_{a,c} \cdot \text{LPRBRL}(n)_{a,s})]$$

where $SPONMR(n)_{a,c}$ = spontaneous cancer morbidity rate in year n for age category a and cancer type c

$$z = MAGE_a + LPC_c.$$

Subroutine SUMAB

The first step is the calculation of an intermediate sum required for projection of cancers to future years by Function MPY. Population fractions and conditional survival probabilities from Function LPRBRL are inputs. The period of time covered is controlled by the latency period and period of risk for each cancer type.

$$SUMMRB_{a,s,c} = \sum_{n=z}^N (LPRBRL(n)_{a,s} \cdot POPFE_{a,s})$$

where N = the lesser of years or $MAGE_a + LPC_c + POR_c$

$SUMMRB_{a,s,c}$ = intermediate sum used to project morbidity of cancer type c, age a, and sex s

$$z = MAGE_a + LPC_c.$$

Subroutine SUMREL

A similar calculation is also carried out for the relative risk model of cancer distribution over time. Lifetime risks are assumed.

$$SUMMRB_{a,s,c} = \sum_{n=z}^{YEARS} (SPONMR(n)_{a,c} \cdot LPRBRL(n)_{a,s})$$

where $z = MAGE_a + LPC_c.$

These intermediate calculations are used in Function MPY.

Function MPY

In the absolute risk model, cancers are projected to future years at a constant rate per surviving person per year over the risk period.

$$MPY(n)_{a,s,c} = MORB_{a,s,c} \cdot LPRBRL(n)_{a,s} \cdot POPFE_{a,s} / SUMMRB_{a,s,c}$$

where $n = \text{MAGE}_a + \text{LPC}_c \text{ to } \text{MAGE}_a + \text{LPC}_c + \text{POR}_c$

$\text{MPY}(n)_{a,s,c}$ = number of nonfatal cancers occurring in year n of cancer type c in age group a and sex s .

In the relative risk model the induced cancer rate for the surviving population in each year is a constant proportion of the spontaneous cancer morbidity rate over time.

$$\text{MPY}(n)_{a,s,c} = \text{MORB}_{a,s,c} \cdot \text{LPRBRL}(n)_{a,s} \cdot \text{SPONMR}(n)_{a,c} / \text{SUMMRB}_{a,s,c}$$

where $n = \text{MAGE}_c + \text{LPC}_c \text{ to } \text{YEARS}$.

8.6.2 Cancer Mortality

Like morbidity, cancer mortality is simulated in a multi-stage process. Fatalities are first allocated to age and sex cohorts and then projected to future years in the third stage. The end result is a matrix of fatality projections by age and sex cohort, cancer type and year. These projections are used to calculate indirect illness and fatality costs as well as direct cancer costs. Absolute and relative risk model use in allocating fatal cancer input data parallels the morbidity case. A separate equation is developed for cancer mortality from in utero exposure.

Subroutine FATAB

This subroutine allocates mortality data by cancer type to age and sex cohorts using an absolute risk model. A higher probability of developing thyroid cancer is assigned to females and those under 18 years old. Additional input variables include population fractions excluding the in-utero cohort, conditional survival probabilities from function LPRBRL, latency periods, median survival periods and risk periods.

$$\text{FAT}_{a,s,c} = \text{CF}_c \cdot \text{POPFE}_{a,s} \cdot \sum_{n=z}^N \text{LPRBRL}(n)_{a,s} / \sum_{s=1}^2 \sum_{a=1}^{AC}$$

$$(\text{POPFE}_{a,s} \cdot \sum_{n=z}^N \text{LPRBRL}(n)_{a,s})$$

where $N = \text{the lesser of } \text{YEARS} \text{ or } \text{MAGE}_a + \text{LPC}_c + \text{POR}_c + \text{MS}_c - 1$

$z = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c$.

For the in-utero age group the equation is:

$$FAT_{p,s,c} = CFP_c / SEX$$

where $FAT_{a,s,c}$ = fatalities of cancer type c affecting age group a
(p = prenatal) and sex s

CF_c = fatal cancers of type c

MS_c = median survival period for cancer type c

CFP_c = cancer fatalities of type c due to in-utero exposure.

Subroutine FATREL

This subroutine distributes mortality to age and sex cohorts using the relative risk model. Cancers are allocated to cohorts in proportion to their risk of spontaneously occurring fatal cancers.

$$FAT_{a,s,c} = CF_c \cdot POPFE_{a,s} \cdot \sum_{n=z}^{YEARS} (SPONFR(n)_{a,c} \cdot LPRBRL(n)_{a,s}) / \sum_{s=1}^2 \sum_{a=1}^{AC}$$

$$[POPFE_{a,s} \cdot \sum_{n=z}^{YEARS} (SPONFR(n)_{a,c} \cdot LPRBRL(n)_{a,s})]$$

where $SPONFR(n)_{a,c}$ = spontaneous cancer fatality rate in year n for age group a and cancer type c

$$z = MAGE_a + LPC_c + MS_c.$$

Subroutine SUMAB

This subroutine calculates an intermediate sum for use in projecting cancer fatalities to future years using the absolute risk model. The equation is:

$$SUMDTH_{a,s,c} = \sum_{n=z}^N (LPRBRL(n)_{a,s} \cdot POPFE_{a,s})$$

where $N = \text{lesser of YEARS or } \text{MAGE}_a + \text{LPC}_c + \text{POR}_c + \text{MS}_c - 1$

$\text{SUMDTH}_{a,s,c}$ = intermediate summation used in projecting cancer fatalities to future years

$$z = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c.$$

A separate equation is employed for in-utero exposures:

$$\text{SUMDTH}_{p,s,c} = \sum_{n=z}^{\text{PPOR}_c + \text{MS}_c - 1} \text{LRRBRL}(n)_{p,s} \cdot \text{POPFE}_{a,s}$$

where PPOR_c = period of risk for cancer type c for in-utero cohort

$$z = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c.$$

Subroutine SUMREL

A similar calculation is carried out for the relative risk model in the following equation:

$$\text{SUMDTH}_{a,s,c} = \sum_{n=z}^{\text{YEARS}} (\text{LPRBRL}(n)_{a,s} \cdot \text{SPONFR}(n)_{a,c})$$

where $z = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c.$

In the next stage the cancer fatalities are distributed over years by functions employing absolute or relative risk models. The first fatalities of each cancer type are projected to occur in the year after the end of both the latency period and the median survival period. The last fatality from each cancer type occurs in the year that the youngest age cohort reaches either the maximum age specified by the user or the end of the period of risk. The end result of this process is a matrix of fatality projections by age and sex cohort, year of death, and cancer type.

Function FPY

This function projects cancer mortality in subsequent years. For the absolute risk model, input variables are incidence of cancer mortality by age and sex cohort from subroutine FATAB, conditional survival probabilities from subroutine LPRBRL, latency periods and median survival periods.

$$FPY(n)_{a,s,c} = FAT_{a,s,c} \cdot LPRBRL(n)_{a,s} \cdot POPFE_{a,s} / SUMDTH_{a,s,c}$$

where $n = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c$ to the lesser of YEARS or $\text{MAGE}_a + \text{LPC}_c + \text{POR}_c + \text{MS}_c - 1$.

A separate equation is used for the in-utero cohort:

$$FPY(n)_{p,s,c} = FAT_{p,s,c} \cdot LPRBRL(n)_{a,s} \cdot POPFE_{p,s} / SUMDTH_{p,s,c}$$

where n is less than $\text{MAGE}_{(p)} + \text{PPOR}_c + \text{LPC}_c + \text{MS}_c - 1$, and p equals prenatal.

With the relative risk model the induced cancer fatality rate for the surviving population in each year is a constant proportion of the spontaneous cancer fatality rate.

$$FPY(n)_{a,s,c} = FAT_{a,s,c} \cdot SPONFR(n)_{a,c} \cdot LPRBRL(n)_{a,s} / SUMDTH_{a,s,c}$$

where $n = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c$ to YEARS

8.6.3 Cost Calculation

Direct costs of cancer treatment are calculated in the subroutine CANCOST. The projected morbidity or mortality in each future year is multiplied by first and second year treatment costs escalated to the years in which treatment would occur. Resulting costs are then discounted to a present value estimate.

Subroutine CANCOST

This subroutine calculates a present value estimate of total cancer treatment costs over time. Inputs to the subroutine include morbidity and mortality over time, the first and second year treatment costs for each type of cancer, the real (inflation free) rate of treatment cost growth, and the discount rate. The equation below is used to calculate cancer treatment cost for all population cohorts and cancer types.

$$\begin{aligned}
CCOSTS_{s,c} = & \sum_{a=1}^{AC+1} \sum_{n=y}^{YEARS} [MPY(n)_{a,s,c} \cdot CPCF_c (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}] \\
& + \sum_{a=1}^{AC+1} \sum_{n=y}^{YEARS} [MPY(n)_{a,s,c} \cdot CPCS_c (1 + RHG/100)^n / (1 + R/100)^n] \\
& + \sum_{a=1}^{AC+1} \sum_{n=z}^{YEARS} [FPY(n)_{a,s,c} \cdot CPCF_c (1 + RHG/100)^{n-2} / (1 + R/100)^{n-2}] \\
& + \sum_{a=1}^{AC+1} \sum_{n=z}^{YEARS} [FPY(n)_{a,s,c} \cdot CPCS_c (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}]
\end{aligned}$$

where $CCOSTS_{s,c}$ = present value of the cost of treating sex s for cancer type c

$CPCF_c$ = first year cost of treating one case of cancer type c

$CPCS_c$ = second year cost of treating one case of cancer type c

RHG = rate of real treatment cost growth

R = discount rate

$y = MAGE_a + LPC_c$

$z = MAGE_a + LPC_c + MS_c$

8.7 DIRECT COSTS OF GENETIC EFFECTS

Estimating the direct costs of genetic effects requires first projecting genetic effect incidence to future years. The method is described in Section 8.7.1. Costs are then calculated as described in Section 8.7.2.

8.7.1 Projection of Genetic Effects

Genetic effect incidence estimates and genetic effect elimination rates are inputs to the genetic effect projection process. Elimination rates are used to allocate genetic effects to each affected generation. The births in each generation are then allocated equally to each year within the generational period. The genetic effect projections are used to calculate both the direct and the indirect costs of illness due to genetic effects.

Subroutine GENDIST

This subroutine distributes genetic effects to each year after radiation exposure until the end of a user-specified genetic effect period. Inputs to the subroutine include total genetic effects by type, incidence rates by sex, and elimination rates per generation by type of genetic effect. The number of generations is a user-specified parameter. The first step in the subroutine is to calculate first generation effects.

$$\text{GENPG}_{g,s,t} = \text{GENI}_g \cdot \text{GSRATE}_s / 100 \cdot \sum_{t=1}^{\text{NGEN}} (1 - \text{ERATE}_g)^{t-1}$$

where $\text{GENPG}_{g,s,t}$ = births with major genetic effects of type g occurring in generation t for sex s

GENI_g = total genetic effect incidence of type g

GSRATE_s = fraction of genetic effects affecting sex s

NGEN = maximum number of generations considered

ERATE_g = elimination rate for genetic effect g .

The second step is to project genetic effects occurring in each remaining generation using the following equation:

$$\text{GENPG}_{g,s,t} = \text{GENPG}_{g,s,t-1} (1 - \text{ERATE}_g / 100)$$

The final step is to allocate the effects for each generation equally to the years within each generational period.

$$\text{GENPY}(n)_{g,s} = \text{GENPG}_{g,s,t} / \text{YPG}$$

where $\text{GENPY}(n)_{g,s}$ = genetic effect births in year n of sex s affected by genetic disorder g

YPG = years per generation.

The output of GENDIST subroutine is a matrix of genetic effects by birth year, type of effect and sex.

8.7.2 Direct Costs of Genetic Effects

Direct costs of genetic effects consist of the lifetime cost (over and above normal consumption) of treating persons born with genetic defects. Direct costs are calculated from initial medical costs plus the sum of lifetime expected costs for each person born with a genetic defect. Expected annual costs are derived from the percent of disability and the real costs of institutionalization in each year an individual is incapacitated. These costs are all discounted back to the base year using the discount rate.

Subroutine GENCOST

This subroutine calculates the present value of direct costs attributable to the genetic effects allocated in GENDIST. Inputs to the process include the number of persons born with genetic effects in each year, the costs of initial treatment and institutionalization, the period and percent of disability, and years of life shortening. Costs are calculated by the following equation:

$$GCOST_{g,s} = \sum_{n=1}^{GYEARS} [GENPY(n)_{g,s} \cdot (MEDCOST_g (1 + RHG/100)^{n-1} / (1 + R/100)^{n-1}) \\ + \sum_{j=z}^{70 - LS_g} (INCOST_g \cdot DISP_p (1 + RHG/100)^{n+j-1} / (1 + R/100)^{n+j-1})]$$

where $GCOST_{g,s}$ = cost of caring for individuals of sex s having genetic effects of type g

LS_g = years of life shortening due to genetic effect g

IMY_g = years of impaired functioning due to genetic effect g

$MEDCOST_g$ = cost of initial medical treatment for genetic effect g

$INCOST_g$ = cost of institutional care per year (excluding normal consumption expenditures) for genetic effect g

$DISP_g$ = disability percentage associated with genetic effect g

$z = 70 - LS_g - IMY_g$.

The medical treatment costs are assumed to occur in the year of birth and the institutional care cost portion to occur during the period of impaired function.

8.8 INDIRECT COSTS OF ILLNESS

Indirect costs of illness are estimated from the value of labor lost due to illness. Data from intermediate calculations of labor value over time, and projections of morbidity and fatalities over time are combined with data for weeks of work missed to calculate indirect illness costs. Costs for radiation injuries, cancers and genetic effects are developed in separate equations.

8.8.1 Radiation-Injury-Related Work Loss

In the case of early injuries, work loss related to radiation injury is assumed to occur within the year of morbidity onset. For early fatalities, the value of work loss during illness is included in the indirect cost calculation for premature mortality (RLOSTV).

An estimate of work weeks lost due to each type of radiation injury is applied to the value of labor for each cohort to calculate labor value lost. Separate though similar equations are employed for early and later occurring injuries.

Subroutine RWORKE

Early radiation injury work loss costs are calculated in the following equation:

$$LWMRB_{s,r} = \sum_{a=1}^{AC} [RMPYE_{a,s,r} \cdot LVALUE(1)_{a,s} / 52 \cdot LWRKRE_r / (1 + R/100)^{n-1}]$$

where $LWMRB_{s,r}$ = present value of the indirect cost of work loss for sex s and radiation injury type r

$RMPYE_{a,s,r}$ = morbidity from early radiation injury type r among age group a and sex s

$LVALUE(1)_{a,s}$ = the projected value of earnings in year 1 for age group a and sex s

$LWRKRE$ = lost work weeks for early radiation injury r

AC = number of age categories

R = discount rate.

Subroutine RWORKL

Indirect costs of work loss associated with later and continuing injuries are calculated in this subroutine. Work loss is assumed to occur in the year of injury onset. The equation for later injuries such as cataracts and hypothyroidism is as follows:

$$RILIND_{s,r} = \sum_{a=1}^{AC} \sum_{n=z}^{YEARS} [RMPYL(n)_{a,s,r} \cdot LVALUE(n)_{a,s} / 52 \cdot LWRKRL_r / (1 + R/100)^{n-1}]$$

where $RILIND_{s,r}$ = indirect costs of work loss for later radiation injury type r affecting sex s

$RMPYL(n)_{a,s,r}$ = later radiation morbidity type r occurring in year n for age a and sex s

$LWRKRL_r$ = lost work weeks for later radiation injury type r

$YEARS$ = maximum attainable age

$MAGE_a$ = median age of age category a

LPR_r = latency period for radiation injury type r

$z = MAGE_a + LPR_r$

For continuing injuries such as microcephaly and mental retardation, the equation is:

$$RILIND_{s,r} = \sum_{n=z}^{YEARS} [RMORB_{p,s,r} \cdot LPRBRL(n)_{a,s} \cdot DISP_r / 100 \cdot LVALUE(n)_{a,s} / (1 + R/100)^{n-1}]$$

where $RMORB_{p,s,r}$ = incidence of continuing injury type r affecting the prenatal group p and sex s

$LPRBRL(n)_{a,s}$ = prior survival probability in year n for age group a (p) and sex s

$DISP_r$ = disability percent for injury type r

$z = MAGE_a + LPR_r$

8.8.2 Cancer-Related Work Loss

For cancers, labor productivity loss is assumed to occur in the year prior to death. Projections of morbidity and mortality in each year are combined with labor value projections for each year and the number of weeks of work lost for each cancer type to calculate labor value loss by sex and type of cancer.

Subroutine CWORK

This subroutine calculates the value of lost work time. Inputs to the subroutine include morbidity and mortality incidence per year, the number of weeks of work missed for each cancer type, and future labor values.

The value of work lost due to cancer morbidity is calculated using the following equation:

$$LWMRB_{c,s} = \sum_{a=1}^{AC+1 \text{ YEARS}} \sum_{n=z} [MPY(n)_{a,s,c} \cdot LVALUE(n)_{a,s} / 52 \cdot LWORKC_c / (1 + R/100)^{n-1}]$$

where $LWMRB_{c,s}$ = present value of lost work time due to cancer-related illness by sex s and cancer type c

$MPY(n)_{a,s,c}$ = morbidity occurring in year n for age group a , sex s and cancer type c

$LWORKC_c$ = lost work weeks due to cancer type c

$z = MAGE_a + LPC_c$

The cost equation for cancer fatality related work loss during illness is:

$$LWFAT_{c,s} = \sum_{a=1}^{AC+1 \text{ YEARS}} \sum_{n=z} [FPY(n)_{a,s,c} \cdot LVALUE(n)_{a,s} / 52 \cdot LWORKC_c / (1 + R/100)^{n-1}]$$

where $LWFAT_{c,s}$ = present value of lost work by sex s due to cancer fatalities of type c

$FPY(n+1)_{a,s,c}$ = fatalities in year n + 1 affecting age a and sex s due to cancer type c

MS_c = median survival time for cancer type c

$z = MAGE_a + LPC_c + MS_c$.

8.8.3 Genetic-Effect-Related Work Loss

Genetic effects of different types may result in various ranges and periods of disability. Estimates of the period and percent of disability typically associated with each genetic effect type are combined with future labor value to project indirect costs.

Subroutine GWORK

$$GENINM_{s,g} = \sum_{n=1}^{GYEARS} [GENPY(n)_{s,g} \cdot \sum_{j=z}^{70-LS_g} [MERN(j)_{a,s} \cdot LFPR(j)_{a,s} \cdot DISP_g / 100 (1 + REG/100)^{j-1} / (1 + R/100)^{j-1}] \cdot (1 + REG/100)^{n-1} / (1 + R/100)^{n-1}]$$

where $GENINM_{s,g}$ = present value of productivity loss by sex s due to genetic effect g

$GYEARS$ = period of births with genetic effects

$GENPY(n)_{s,g}$ = births in year n of sex s affected by genetic disorder g

LS_g = years of life shortening due to genetic effect g

IMY_g = years of impaired functioning due to genetic effect g

$DISP_g$ = disability percent associated with genetic effect g

$MERN(j)_{a,s}$ = median earnings at age j for age group a and sex s

$LFPR(j)_{a,s}$ = labor force participation rate at age j for age group a and sex s

REG = real rate of earnings growth

$$z = 70 - LS_g - IMY_g.$$

8.9 INDIRECT COSTS OF FATALITIES

Indirect costs of fatalities represent the value of labor lost to society because of premature death. Data from intermediate projections of cohort survival probabilities, labor value over time, fatalities by age cohort, cause of death and sex are used to calculate indirect costs.

The labor value lost because of a fatality is the sum of projected annual labor values from the year of death to the maximum expected age of the individual specified by the user. The calculation of labor value lost is based on projections of fatalities in each year by age category, cause of death and sex. Labor value losses are discounted to estimate the present value of indirect costs of fatalities.

8.9.1 Radiation-Injury-Related Mortality

Mortality due to radiation injury is assumed to occur within the year of exposure. There are no fatalities associated with later-occurring injury types. Indirect costs are calculated from potential earnings had the individual achieved normal life expectancy.

Subroutine RLOSTV

Inputs to this subroutine include Subroutine RADEFAT projections of fatalities, Subroutine LVALUE and Function LPRBRL. The equation is as follows:

$$RIEIND_{s,r} = \sum_{a=1}^{AC+1} RFPYE_{a,s,r} \cdot \sum_{n=MAGE_a}^{YEARS} [LPRBRL(n)_{a,s} \cdot LVALUE(n)_{a,s} / (1 + R/100)^{n-1}]$$

where $RIEIND_{s,r}$ = present value of indirect costs of mortality for sex s due to injury type r

$RFPYE_{a,s,r}$ = early radiation-induced fatalities of type r affecting age group a and sex s

AC = number of age categories

YEARS = maximum age attainable

MAGE_a = median age of age category a

R = discount rate.

8.9.2 Cancer-Related Mortality

Projection of indirect cancer costs is similar to that for radiation injuries except that the deaths occur in future years.

Subroutine CLOSTV

Indirect costs of cancer-related mortality are calculated in Subroutine CLOSTV. Inputs to the subroutine include Function FPY, Function LPRBRL and Subroutine LVALUE. The equation is:

$$\text{CANIND}_{s,c} = \sum_{a=1}^{AC+1} \sum_{n=z}^{\text{YEARS}} (\text{FPY}(n)_{a,s,c} \cdot \sum_{n=z}^{\text{YEARS}} [\text{LPRBRL}(n)_{a,s} \cdot \text{LVALUE}(n)_{a,s} / (1 + R/100)^{n-1}])$$

where $\text{CANIND}_{s,c}$ = present value of indirect costs due to cancer mortality of type c affecting sex s

$\text{FPY}(n)_{a,s,c}$ = fatalities in year n of cancer type c affecting age group a and sex s

LPC_c = latency period for cancer type c

MS_c = median survival period for cancer type c

$z = \text{MAGE}_a + \text{LPC}_c + \text{MS}_c$

8.9.3 Genetic-Effect-Related Mortality

The early mortality of those born with genetic effects in the future also results in indirect costs. These are calculated in Subroutine GLOSTV.

Subroutine GLOSTV

Inputs to this subroutine include periods of life shortening by genetic effect type, Subroutine GENDIST, Subroutine LVALUE and Function LPRBAB. The equation is:

$$\begin{aligned}
 \text{GENINF}_{s,g} = & \sum_{n=1}^{\text{GYEARS}} [\text{GENPY}(n)_{s,g} \cdot \sum_{j=70 - \text{LS}_g}^{70} (\text{MERN}(j)_{a,s} \cdot \text{LFPR}(j)_{a,s} \cdot \\
 & \text{LPROBRL}(j)_{a,s} / (1 + R/100)^{j-1}) (1 + R/100)^{n-1}]
 \end{aligned}$$

where $\text{GENINF}_{s,g}$ = present value of indirect costs of genetic effect g life shortening affecting sex s

GYEARS = period of genetic effect births

$\text{GENPY}(n)_{s,g}$ = genetic effect births in year n of type g for sex s

LS_g = years of life shortening due to genetic effect g.

9.0 SENSITIVITY ANALYSIS

HECOM has applications in many types of risk-related analyses, including siting analyses and evaluations of safety goals and standards. A wide range of analyses are possible by varying health effect, clinical, economic or population data input to HECOM. Section 9.1 presents an example of HECOM output using the base case input data contained in Appendix A. Since some level of uncertainty underlies the data for each of the input variables used in estimating health effects costs, a sensitivity analysis was carried out to ascertain its significance. The results are discussed in Section 9.2.

9.1 BASE CASE

The base case discussed in this section was run for ten cases of each type of potential fatal and nonfatal health effect likely to be associated with acute radiation exposure. This was done to permit examination of the cost projections on the basis of cost per case. The costs are shown here as they appear in the HECOM output. A summary on a cost per case basis is included in Chapter 2.

9.1.1 Assumptions

Population and injury numbers used in running the base case were selected solely for ease in analyzing cost per person or cost per case. Sources of the input data used in the base case are documented in Appendix A. This data base represents a distillation of the most current information available regarding impacts of radiation-induced health effects. Development of the treatment cost estimates in the data base has been described in previous chapters of this report. The costs are expressed in 1984 dollars and 1984 is used as the base year of analysis.

A real cost escalation rate of 1 percent is assumed for incomes and 2 percent for medical costs. While increases, especially in medical costs, have been higher than this in recent years, a relatively low growth rate is assumed because of the long run nature of HECOM estimates. A discount rate of 4 percent is used in the base case for similar reasons.

9.1.2 Output

Computer output for the base case assumptions described above is shown in the tables that follow. Direct and indirect cost estimates are given for each health effect type and major category, such as cancers, as well as by sex and total population.

Table 9.1 shows the direct costs of medical management for an exposed population. The radiation screening costs are based on estimates of 100,000 people located in the sector 90° downwind from the radiation release, and 10,000 people evacuated. Medical monitoring costs assume 1,000 people are exposed to over one Gy. For medical transportation costs, 100 people are

TABLE 9.1. Direct Costs of Medical Management

RADIATION SCREENING	1.5140E+07
MEDICAL MONITORING	2.2500E+05
MEDICAL TRANSPORTATION ...	2.0800E+04
TOTAL COSTS	1.5386E+07

assumed to be exposed to over two Gy. These numbers are only assumed to provide a basis for testing the model.

Radiation injury costs are shown in Table 9.2. For the injury types that are likely to result in both morbidity and mortality, the estimates are based on 20 cases (ten fatal, ten nonfatal). Those which result in only morbidity or only mortality, such as prodromal symptoms, gastrointestinal syndrome and prenatal fatalities, are based on ten cases.

Of the various types of injuries, the most costly to treat are the prenatal injuries: microcephaly (\$237.9K) and mental retardation (\$452.0K). These are also most costly when total direct and indirect costs are compared (\$344.0K and \$653.7K respectively). This is due both to the relatively high cost of institutional care and the major degree of disability involved. Prenatal fatalities are next in total cost per case due to high indirect costs (\$212.4K), followed by the hematopoietic and gastrointestinal syndromes (\$184.5K and \$156.5K). Costs of nonfatal effects (other than prenatal injuries) consist mainly of treatment costs and are low in comparison: prodromal symptoms (\$1.7K); thyroiditis (\$0.4K), transepithelial injury (\$0.4K), hypothyroidism (\$2.5K) and cataracts (\$3.3K).

Cancer costs are presented in Table 9.3. Costs of skin cancers, benign thyroid nodules, and leukemia and other cancers due to in-utero exposure are based on ten cases each; the rest are based on 20. Direct costs vary from \$0.9K for skin cancer to \$18.6K for bone cancer. Indirect costs are, of course, lowest for the effects that are not associated with fatalities, skin cancer and benign thyroid nodules, and highest for early fatalities due to in-utero exposures. The relatively high indirect costs of leukemia (\$82.5K) and bone cancer (\$80.4K) are due to the relatively early induction of deaths by these cancers.

Table 9.4 shows the direct and indirect cost estimates for genetic effects. These estimates are based on ten cases of each genetic effect type distributed probabilistically over the first five generations after exposure. The costs of aneuploidy are highest (\$240.0K) because they all occur in the first generation. Total costs for the other genetic effects (\$40.1K to \$175.8K) vary with the degree of disability and the degree to which they are weighted toward earlier generations.

TABLE 9.2. Radiation Injury Costs

DIRECT COSTS:

INJURY TYPE	MALE	FEMALE	TOTAL
PRODROMAL SYMPTOMS	7.2900E+03	7.7100E+03	1.5000E+04
GASTROINTESTINAL SYN	2.7216E+05	2.8784E+05	5.6000E+05
HEMATOPOIETIC SYN	5.4432E+05	5.7568E+05	1.1200E+06
PULMONARY SYNDROME	7.9448E+04	9.0552E+04	1.7000E+05
PRENATAL FATALITY	5.0000E+02	5.0000E+02	1.0000E+03
THYROIDITIS	9.7200E+02	1.0280E+03	2.0000E+03
TRANSEPIHELIAL INJU	9.7200E+02	1.0280E+03	2.0000E+03
HYPOTHYROIDISM	1.1991E+04	1.2299E+04	2.4291E+04
CATARACTS	1.5189E+04	1.6492E+04	3.1681E+04
MICROCEPHALY	1.1605E+06	1.2181E+06	2.3787E+06
MENTAL RETARDATION	2.2050E+06	2.3145E+06	4.5195E+06
TOTAL	4.2983E+06	4.5258E+06	8.8241E+06

INDIRECT COSTS:

INJURY TYPE	MALE	FEMALE	TOTAL
PRODROMAL SYMPTOMS	1.0953E+03	6.3146E+02	1.7268E+03
GASTROINTESTINAL SYN	1.6455E+06	9.2612E+05	2.5716E+06
HEMATOPOIETIC SYN	1.6455E+06	9.2612E+05	2.5716E+06
PULMONARY SYNDROME	6.3835E+05	5.1079E+05	1.1491E+06
PRENATAL FATALITY	1.3638E+06	7.5884E+05	2.1227E+06
THYROIDITIS	1.0953E+03	6.3146E+02	1.7268E+03
TRANSEPIHELIAL INJU	1.0953E+03	6.3146E+02	1.7268E+03
HYPOTHYROIDISM	7.5176E+02	4.2238E+02	1.1741E+03
CATARACTS	7.4369E+02	4.1729E+02	1.1610E+03
MICROCEPHALY	6.8192E+05	3.7942E+05	1.0613E+06
MENTAL RETARDATION	1.2956E+06	7.2090E+05	2.0165E+06
TOTAL	5.5546E+06	9.1451E+06	1.1501E+07

The final table, Table 9.5, provides a summary of direct and indirect costs due to morbidity and mortality associated with each health effect category, as well as total costs. Only a total is shown for medical management costs since these costs apply to the entire population at risk.

9.2 RESULTS OF PARAMETER VARIATION

Model sensitivity is generally measured by varying the value of one input variable within the bounds of a range of uncertainty, while holding all other input variables constant. To evaluate sensitivity, we examine health effect, economic condition and cost variables.

TABLE 9.3. Cancer Costs

DIRECT COSTS:

CANCER TYPE	MALE	FEMALE	TOTAL
LEUKEMIA	1.5112E+05	1.5892E+05	3.1003E+05
LUNG	8.3299E+04	1.0159E+05	1.8489E+05
BREAST	0.0000E+00	1.0919E+05	1.0919E+05
BONE	1.8096E+05	1.9030E+05	3.7126E+05
SKIN	4.4511E+03	4.8430E+03	9.2942E+03
GASTROINTESTINAL	7.8013E+04	1.0114E+05	1.7915E+05
ALL OTHERS	7.6178E+04	9.4093E+04	1.7027E+05
THYROID	3.4994E+04	6.0777E+04	9.5771E+04
THYROID-BENIGN	4.3898E+03	6.8244E+03	1.1214E+04
IN-UTERO LEUKEMIA	8.9984E+04	8.9982E+04	1.7997E+05
IN-UTERO OTHER	7.9941E+04	7.9940E+04	1.5988E+05
TOTAL	7.8333E+05	9.9759E+05	1.7809E+06

INDIRECT COSTS:

CANCER TYPE	MALE	FEMALE	TOTAL
LEUKEMIA	1.0523E+06	5.9711E+05	1.6494E+06
LUNG	1.0893E+05	1.2266E+05	2.3159E+05
BREAST	0.0000E+00	2.4895E+05	2.4895E+05
BONE	1.0250E+06	5.8238E+05	1.6074E+06
SKIN	6.3154E+02	3.5510E+02	9.8664E+02
GASTROINTESTINAL	7.4182E+04	9.4650E+04	1.6883E+05
ALL OTHERS	1.0300E+05	1.0924E+05	2.1224E+05
THYROID	1.9134E+05	2.6349E+05	4.5484E+05
THYROID-BENIGN	5.1763E+02	4.1013E+02	9.2775E+02
IN-UTERO LEUKEMIA	1.3902E+06	7.7041E+05	2.1606E+06
IN-UTERO OTHER	1.3903E+06	7.7047E+05	2.1608E+06
TOTAL	5.3364E+06	3.5601E+06	8.8965E+06

9.2.1 Health Effect Variables

HECOM does not deal with any possible economies or diseconomies of scale that might occur in treating an exposed population. The model assumes that resources and capacity are made available to provide adequate population management after exposure and supportive care to those with radiation injuries. Less intensive care would result in higher morbidity and mortality rates. As a result of this assumption, HECOM cost estimates are linearly related to the number of health effect cases.

TABLE 9.4. Genetic Effect Costs

DIRECT COSTS:

GENETIC EFFECT TYPE	MALE	FEMALE	TOTAL
AUTOSOMAL DOMINANT	1.0747E+05	1.0747E+05	2.1495E+05
X-LINKED	5.8522E+05	0.0000E+00	5.8522E+05
UNBALANCED TRANSLOCA	4.9620E+05	4.9620E+05	9.9240E+05
ANEUPLOID	7.2569E+05	7.2569E+05	1.4514E+06
TOTAL	1.9146E+06	1.3294E+06	3.2439E+06

INDIRECT COSTS:

GENETIC EFFECT TYPE	MALE	FEMALE	TOTAL
AUTOSOMAL DOMINANT	1.2173E+05	6.4366E+04	1.8610E+05
X-LINKED	5.3134E+05	0.0000E+00	5.3134E+05
UNBALANCED TRANSLOCA	4.9557E+05	2.6999E+05	7.6556E+05
ANEUPLOID	6.1891E+05	3.3054E+05	9.4945E+05
TOTAL	1.7676E+06	6.6490E+05	2.4325E+06

TABLE 9.5. Summary of Direct and Indirect Health Effects Costs

DIRECT COST SUMMARY	MORBIDITY	MORTALITY	TOTAL
MEDICAL MANAGEMENT COSTS .	NA	NA	1.5386E+07
RADIATION INJURY COSTS ...	7.8981E+06	9.2600E+05	8.8241E+06
CANCER COSTS	7.4071E+05	1.0402E+06	1.7809E+06
GENETIC EFFECT COSTS	3.2439E+06	NA	3.2435E+06
TOTAL	1.1883E+07	1.9662E+06	2.9235E+07
INDIRECT COST SUMMARY	MORBIDITY	MORTALITY	TOTAL
RADIATION INJURY COSTS ...	3.2201E+06	8.2804E+06	1.1501E+07
CANCER COSTS	1.0812E+05	8.7884E+06	8.8965E+06
GENETIC EFFECT COSTS	9.3715E+05	1.4953E+06	2.4325E+06
TOTAL	4.2654E+06	1.8564E+07	2.2829E+07
TOTAL COSTS			
MEDICAL MANAGEMENT COSTS	1.5386E+07		
RADIATION INJURY COSTS	2.0325E+07		
CANCER COSTS	1.0677E+07		
GENETIC EFFECT COSTS	5.6764E+06		
TOTAL	5.2064E+07		

9.2.2 Economic Condition Variables

To measure sensitivity in HECOM, we examined four variables: the discount rate, the rate of earnings growth, the rate of medical cost growth, and the base year. Of the three rates, the model is most sensitive to the discount rate.

Table 9.6 shows the effect of different discount rates on the indirect, direct and total costs calculated by HECOM. The 10 percent discount rate is mandated for use by the U.S. Office of Management and Budget and is used by the Nuclear Regulatory Commission. A 4 percent discount rate is used to represent the social rate of discount. Rates of 7 percent and 1 percent were also tested to explore fully the sensitivity of HECOM. As shown in Table 9.6, the model is clearly sensitive to the discount rate. Use of a 7 or 10 percent rate significantly lowers total costs because costs occurring in the years after initial exposure are given much less weight than similar costs occurring in the base year. A discount rate of 1 percent substantially increases costs because costs in the more distant future are given nearly the same weight as costs in the near future.

The effect on indirect and total costs of varying the rate earnings growth from its base case rate of 1 percent to rates of -1, 0.5 and 3 percent is shown in Table 9.7. The 3 percent rate results in a more than 60 percent increase in

TABLE 9.6. Sensitivity of HECOM Estimates to the Discount Rate

<u>Discount Rate %</u>	<u>Direct Cost % Δ from Base</u>	<u>Indirect Cost % Δ from Base</u>	<u>Total Cost % Δ from Base</u>
10	-28	-61	-53
7	-20	-42	-37
4 (Base)	0	0	0
1	100	131	124

TABLE 9.7. Sensitivity of HECOM Estimates to the Rate of Earnings Growth

<u>Rate Productivity Growth (%)</u>	<u>Indirect Cost % Δ from Base</u>	<u>Total Cost % Δ from Base</u>
3	66	50
1 (Base)	0	0
0.5	-10	-8
-1	-33	-25

indirect costs and a 50 percent increase in total costs. The higher labor productivity growth rate causes the share of indirect costs as a percentage of total costs to rise substantially.

Table 9.8 shows relative costs of treatment calculated using the 2 percent base rate, and alternative rates of 4 percent, 0.5, and -1 percent for real treatment cost escalation. Increasing the rate to 4 percent raises direct costs by over 40 percent.

Effects of shifting the base year of analysis forward in time are shown in Table 9.9. Indirect costs are more strongly affected than direct costs. Costs decrease as the date of accidental exposure is shifted into the future because the discount rate used is higher than the treatment cost or earnings growth rates.

TABLE 9.8. Sensitivity of HECOM Estimates to the Rate of Treatment Cost Escalation

<u>Rate of Treatment Cost Growth (%)</u>	<u>Direct Cost % Δ from Base</u>	<u>Total Cost % Δ from Base</u>
4	43	11
2 (Base)	0	0
0.5	-13	-3
-1	-21	-5

TABLE 9.9. Sensitivity of HECOM Estimates to the Base Year

<u>Base Year</u>	<u>Direct Cost % Δ from Base</u>	<u>Indirect Cost % Δ from Base</u>	<u>Total Cost % Δ from Base</u>
10	-8	-26	-22
5	-4	-14	-11
2	-2	-6	-5
0	0	0	0

9.2.3 Cost Variables

Sensitivity to the base level of costs used as model input was tested for all of the cost variables. The base cost level was varied by plus and minus 10 percent. Resulting cost estimates were shifted proportionally by these factors.

APPENDIX A

HEALTH EFFECTS COST MODEL INPUT DATA

APPENDIX A

HEALTH EFFECTS COST MODEL INPUT DATA

The data used as input to HECOM can be grouped in four general categories: population and facilities characteristics, health effects incidence, direct costs of treatment, and indirect costs of lost productivity. In this section, data sources related to each of these categories are described and some of the merits and the limitations of particular data sources are explained.

A.1 POPULATION AND FACILITIES CHARACTERISTICS

HECOM employs the following descriptions of the population at risk: population proportions by age and sex cohorts; survival probabilities; and morbidity and mortality rates for some types of cancers. In addition, data on study area population and medical facilities nationwide are required.

A.1.1 Population by Age and Sex

Population counts, distributed into cohorts by sex and by age intervals, are used both in allocating health effects and in estimating indirect costs. On the national level, the most recent data of this type are from the 1980 Census. These data can be used if detailed local or regional data are unavailable. The proportional distribution of the U.S. 1980 population by age and sex cohorts is shown in Table A.1. Proportions for ages above the maximum age modeled in a particular HECOM run must be added and the sum used as the data input for the highest age category.

The proportion of the population in utero at the time of exposure must also be supplied to HECOM. This is assumed to be approximately 1.2 percent of the population, divided equally between the sexes. A more exact estimate may be supplied if desired.

A.1.2 Survival Probabilities

Data on survival probabilities are used to describe the life expectancies of individuals in the unexposed population by age and sex cohorts. These are shown in Table A.2. Annual life probabilities are computed from the data in Vital Statistics of the United States 1978 (National Center for Health Statistics 1980). These annual data are interpreted as the probability of living an additional year given attainment of the indicated age.

A.1.3 Cancer Morbidity and Mortality Rates

Use of a relative risk model in projecting cancers requires information on spontaneous cancer incidence rates. Morbidity rates by age group for the

TABLE A.1. U.S. Population Distribution by Age and Sex, 1980

Ages	Population Proportion ^(a)	
	Male	Female
All ages	48.6	51.4
0 - 4	3.7	3.5
5 - 9	3.8	3.6
10 - 14	4.1	3.9
15 - 19	4.7	4.6
20 - 24	4.7	4.7
25 - 29	4.3	4.3
30 - 34	3.8	3.9
35 - 39	3.0	2.5
40 - 44	2.5	2.6
45 - 49	2.4	2.5
50 - 54	2.5	2.7
55 - 59	2.4	2.7
60 - 64	2.1	2.4
65 - 69	1.7	2.2
70 - 74	1.3	1.7
75 - 79	0.8	1.3
80 - 84	0.4	0.8
85+	0.4	1.5

(a) Percentages by age for each sex represent the age distribution for that sex within the total population.

Source: U.S. Bureau of the Census.
1983. Table 4, pp. 1-23.

relevant cancer types are shown in Table A.3. These are derived from Cancer Incidence and Mortality in the United States, 1973-77 (National Cancer Institute 1983). Comparable data for cancer mortality from Vital Statistics of the U.S. (National Center for Health Statistics 1978) are shown in Table A.4.

A.1.4 Study Area Population

To simulate medical management costs for an accidentally exposed population, HECOM requires data for various components of the population. The data elements are listed in Table A.5. Values would depend on the region and scenario analyzed.

TABLE A.2. Annual Survival Probabilities by Age and Sex

Annual Survival Probabilities					
Age Attained	Male	Female	Age Attained	Male	Female
0	0.984730	0.987760	45	0.995862	0.997660
	0.998924	0.999210		0.995463	0.997435
	0.999177	0.999362		0.995026	0.997176
	0.999349	0.999483		0.994550	0.996894
5	0.999460	0.999574	50	0.994000	0.996600
	0.999531	0.999645		0.993374	0.996291
	0.999572	0.999695		0.992668	0.995969
	0.999602	0.999726		0.991880	0.995280
10	0.999653	0.999756	55	0.991039	0.995526
	0.999694	0.999787		0.990200	0.994805
	0.999735	0.999807		0.989372	0.994388
	0.999724	0.999807		0.988519	0.993964
15	0.999653	0.999776	60	0.987661	0.993522
	0.999469	0.999725		0.986691	0.993017
	0.999233	0.999654		0.985464	0.992401
	0.998957	0.999573		0.983891	0.991637
20	0.998710	0.999481	65	0.982045	0.990743
	0.998493	0.999420		0.979991	0.989733
	0.998326	0.999389		0.977886	0.988694
	0.998200	0.999368		0.975865	0.987750
25	0.998094	0.999357	70	0.974051	0.986985
	0.997977	0.999336		0.972363	0.986288
	0.997921	0.999326		0.970654	0.985576
	0.997906	0.999315		0.968726	0.984677
30	0.997943	0.999315	75	0.966427	0.983528
	0.998106	0.999294		0.963624	0.982008
	0.997945	0.999293		0.960355	0.980180
	0.998098	0.999272		0.956796	0.978131
35	0.998115	0.999261	80	0.953070	0.976120
	0.998133	0.999240		0.948933	0.973437
	0.998140	0.999209		0.944360	0.970258
	0.998136	0.999177		0.939338	0.966540
40	0.998101	0.999125		0.933900	0.962448
	0.998034	0.999073		0.928072	0.958091
	0.997924	0.999010		0.921904	0.953535
	0.997803	0.998947		0.915400	0.948829
	0.997660	0.998864		0.908621	0.943930
	0.997484	0.998738		0.901618	0.938756
	0.997296	0.998612		0.894615	0.933185
	0.997075	0.998455		0.887832	0.927129
	0.996830	0.998276		0.881742	0.920361
	0.996539	0.998086		0.877178	0.912674
	0.996224	0.997884			

Source: Calculated from National Center for Health Statistics, 1980, Vol II, Section 5, Table 5-1.

TABLE A.3. 1973-1977 Cancer Incidence Rates (new cases/year per 100,000 population)

Age	Gastro-intestinal Cancer	Lung Cancer	Breast Cancer (Females)	Other Cancer ^(a)
0 - 4	0.7	0	0	9.5
5 - 9	0.2	0	0	5.6
10 - 14	0.3	0.1	0	6.1
15 - 19	0.5	0.2	0.2	10.7
20 - 24	1.3	0.2	1.1	18.3
25 - 29	2.4	0.7	8.3	25.9
30 - 34	5.5	2.3	26.7	34.1
35 - 39	11.9	7.1	57.2	45.3
40 - 44	24.9	20.4	106.2	70.6
45 - 49	50.2	47.7	173.8	113.7
50 - 54	89.4	79.8	195.9	187.2
55 - 59	155.5	130.2	228.9	277.6
60 - 64	240.5	185.6	251.2	351.8
65 - 69	351.2	235.5	282.9	420.1
70 - 74	475.2	258.5	302.0	489.6
75 - 79	617.9	255.9	338.0	564.4
80 - 84	708.9	211.4	350.0	586.2
85 - 89	795.6	166.0	376.3	611.3

(a) Excluding leukemia, skin, bone, prostate, thyroid, gastrointestinal, lung, and breast cancers.

Source: National Cancer Institute. 1983.

A.1.5. Medical Facilities

To project patient transportation costs HECOM requires information pertaining to medical facility capacities and distances. The data elements and estimated values are shown in Table A.6. These are developed in Section 4.1.

A.2 HEALTH EFFECTS

Projection of health effects occurrence requires data on incidence, latency periods, survival times and periods of risk. Related data inputs are described in this section.

A.2.1 Incidence Categories

HECOM processes incidence data for radiation injuries, cancers, and genetic effects. The morbidity and mortality data inputs accepted for each health effect category are shown in Table A.7. Genetic effect-related morbidity and mortality impacts are projected using somewhat different methods from radiation

TABLE A.4. 1978 Cancer Mortality Rates (deaths/year per 100,000 population)

Age	Gastro- intestinal Cancer	Lung Cancer	Breast Cancer (Females)	Other Cancer ^(a)
0 - 4	0.2	0	0	2.9
5 - 9	0.1	0	0	2.1
10 - 14	0.1	0	0	1.7
15 - 19	0.2	0	0	2.7
20 - 24	0.4	0.1	0.2	3.9
25 - 29	1.0	0.3	1.2	5.9
30 - 34	2.4	1.3	5.6	8.2
35 - 39	5.2	4.8	11.7	12.3
40 - 44	11.8	15.1	22.9	23.6
45 - 49	25.0	36.2	41.4	41.6
50 - 54	48.1	70.6	60.1	69.5
55 - 59	79.1	110.2	75.9	103.9
60 - 64	133.1	166.4	91.4	157.1
65 - 69	184.8	201.3	89.9	196.8
70 - 74	266.8	238.2	110.7	260.0
75 - 79	376.3	245.0	128.4	340.8
80 - 84	467.4	218.3	139.9	394.4
85 - 89	513.3	147.1	157.2	408.6

(a) Excluding leukemia, skin, bone, prostate, thyroid, gastrointestinal, lung, and breast cancers.

Source: National Center for Health Statistics. 1978.

TABLE A.5. Study Area Population Data Requirements

Population Component	Value ^(a)
Population in the sector 90° downwind	?
Population evacuated	?
Population exposed to over 1 Gy	?
Population exposed to over 2 Gy	?

(a) User-specified value.

injuries and cancers. Only total incidence data are required for each type of genetic effect: dominant and X-linked single gene disorders, aneuploidy and unbalanced translocations. (An incomplete data set is provided for multifactorial diseases, which are not currently estimated in HECOM because elimination rate data are lacking.)

TABLE A.6. Medical Facility Characteristics

	<u>Miles</u>
Average Round-Trip Distance:	
To definitive care centers	1600
To regional hospitals	500
To nationwide hospitals (excluding region)	1600
	<u>Beds</u>
Patient Capacity:	
Definitive care centers	500
Regional hospitals	500
Hospitals nationwide (excluding region)	5500

TABLE A.7. Categories of Morbidity and Mortality Incidence Data Inputs

	<u>Morbidity</u>	<u>Mortality</u>
Radiation Injuries:		
Prodromal symptoms	X	--
Gastrointestinal syndrome	X	X
Hematopoietic syndrome	X	X
Pulmonary syndrome	X	X
Prenatal injury	X	X
Hypothyroidism	X	--
Thyroiditis	X	--
Cataracts	X	--
Skin damage	X	--
Cancers:		
Leukemia	X	X
Lung	X	X
Breast	X	X
Bone	X	X
Skin	X	--
Gastrointestinal	X	X
Other	X	X
Thyroid	X	X
Thyroid - benign nodules	X	--
In utero - leukemia	--	X
In utero - other	--	X

A.2.2 Latency Periods

Evans, Moeller and Cooper (1985, Vol. 2, p. 100) is the source of data on typical minimum latency periods, by health effect type, for the population in utero and for all other ages. These are based on BEIR III and more recent analyses of Japanese data. See Table A.8 for a listing of latency periods for cancers and radiation injuries.

TABLE A.8. Latency Periods for Selected Health Effects

	Age at Exposure	
	<u>Prenatal</u>	<u>All Other</u>
Cancers:		
Leukemia	0	2
Lung	--	10 ^(a)
Breast	--	10 ^(b)
Bone	--	2
Skin	--	10
Gastrointestinal	--	10
Other	0 ^(c)	10
Thyroid - malignant	--	5
Thyroid - benign nodules	--	10
Radiation Injuries: ^(d)		
Hypothyroidism	--	1
Cataracts	--	2

(a) Latency period extends to age 40 for younger age groups.

(b) Latency period extends to age 30 for younger age groups.

(c) Modeling of "other" cancers due to in-utero exposure follows the bone cancer pattern employing an absolute risk model.

(d) All other radiation injuries are assumed to be evident in the first year.

Source: Evans, Moeller and Cooper. 1985.

A.2.3 Disability Levels

The continuing radiation injuries, microcephaly and mental retardation, result in lifelong disability. This impairment is estimated to be 50 and 95 percent, respectively (Evans, Moeller and Cooper 1985, Vol. 2, p. 54).

A.2.4 Cancer Risk Models

The risk models used to calculate central estimates of health effects by Evans, Moeller and Cooper (1985, Vol. 2, p. 99) are applied to each cancer type. These are listed in Table A.9.

A.2.5 Period of Risk

Estimates of the time period an individual exposed to radiation would be at risk for cancers are listed in Table A.10. These risk periods are used to allocate fatalities to the years after radiation exposure.

TABLE A.9. Risk Model Applied to Each Cancer Type

<u>Cancer Type</u>	<u>Risk Model</u>
Leukemia	Absolute
Lung	Relative
Breast	Relative
Bone	Absolute
Skin	Absolute
Gastrointestinal	Relative
Other	Relative
Thyroid	Absolute
In utero - leukemia	Absolute
In utero - other	Absolute

Source: Evans, Moeller and Cooper. 1985.

TABLE A.10. Period of Cancer Risk After Exposure

<u>Cancer Type</u>	<u>Period of Risk (years after exposure)</u>
Leukemia	2 - 27
Lung	10 - lifetime
Breast	10 - lifetime
Bone	2 - 27
Skin	10 - lifetime
Gastrointestinal	10 - lifetime
Other	10 - lifetime
Thyroid - malignant	5 - lifetime
Thyroid - benign nodules	10 - lifetime
In utero - leukemia	0 - 12
In utero - other	0 - 10

Source: Evans, Moeller and Cooper. 1985. Vol. 2, pp. 100-101.

A.2.6 Cancer Survival Periods

Median survival times are calculated by cancer site from data in summary Tables 1 and 2 in Cancer Patient Survival, Report Number 5. (National Institutes of Health 1976.) The median survival times input to the HECOM model are averages of the data reported for black and white population subgroups, weighted by the proportion of each cancer type affecting that subgroup. The NIH data do not differentiate survival times by sex. Median survival times are presented in Table A.11. These apply only to cancer types for which mortality is modeled.

TABLE A.11. Median Survival Time, 1960-1973

<u>Cancer Type</u>	<u>Median Survival^(a) Time (years)</u>
Leukemia	1
Lung	1
Breast	6
Bone	2
Gastrointestinal	2
All Other	4
Thyroid - malignant	15

(a) Averages for data for blacks and whites, weighted by proportion of each cancer type attributed to the racial subgroup.

Source: National Institutes of Health, USOHHS. 1976.

A.2.7 Genetic Effect Elimination Rates

HECOM requires input estimates of the total number of genetic effects of each type considered. The rate per generation at which genetic defects are eliminated from the population must also be input to distribute incidence over time. Estimated rates are shown in Table A.12.

TABLE A.12. Genetic Effects Elimination Rates

<u>Genetic Effect Type</u>	<u>Elimination Rate per Generation</u>
Autosomal dominant	20%
X-linked	20%
Unbalanced translocation	100%
Aneuploidy	40%

Source: Evans, Moeller and Cooper. 1985.
Vol. 1, p. 40.

A.2.8 Genetic Effect Distribution by Sex

The rate at which each sex is affected must be supplied to distribute genetic effect incidence. Table A.13 shows estimated rates for each type of genetic effect modeled.

TABLE A.13. Genetic Effect Distribution by Sex

<u>Genetic Effect Type</u>	<u>Percent Male</u>
Autosomal dominant	50
X-linked	100
Unbalanced translocation	50
Aneuploidy	50

A.2.9 Genetic Effect Impacts

To project direct costs associated with providing physical care, the years of impaired life and the degree of disability for each type of genetic effect must be estimated. Estimates of the years of impaired functioning and the percent of disability are given in Table A.14.

TABLE A.14. Impacts of Genetic Effects on Functioning

<u>Genetic Effect Type</u>	<u>Impaired Years</u>	<u>Percent Disability</u>
Autosomal dominant	25	33
X-linked	40	40
Unbalanced translocations	20	95
Aneuploidy	44	50
Multifactorial	20	25

Source: Evans, Moeller and Cooper. 1985. Vol. 1, p. 11.

A.3 DIRECT COSTS

Input data for direct costs of health effects are required by health effect type. Methods used to develop the HECOM data base shown are described for radiation injuries in Chapter 4, for cancers in Chapter 5, and for genetic effects in Chapter 6. We have inflated the estimates to 1984 dollars using the appropriate medical care components of the Consumer Price Index.

A.3.1 Population Management

The medical management of an exposed population would result in direct costs for screening, monitoring and transporting any injured. Cost estimates are developed in Section 4.1. Input data used in HECOM are summarized in Table A.15.

A.3.2 Radiation Injuries

Direct cost data for each category of radiation injury are required by HECOM. Treatment cost estimates developed in Chapter 4 are presented in Table A.16.

TABLE A.15. Direct Costs of Population Management (1984 \$)

<u>Activity</u>	<u>Cost</u>
Initial survey/person	75.00
Resurvey/person	75.00
Internal survey/person	14.00
Medical services/person	65.00
Laboratory testing/person	160.00
Air transportation/mile	0.13
Motor vehicle transportation/mile	0.29

TABLE A.16. Direct Costs of Radiation Injuries (1984 \$)

<u>Injury Type</u>	<u>Cost</u>
Prodromal symptoms	1,500
Gastrointestinal syndrome	28,000
Hematopoietic syndrome	56,000
Pulmonary syndrome	8,500
Prenatal injury	12,500(a)
Prenatal fatality	100
Hypothyroidism	150(a)
Thyroiditis	200
Cataracts	5,100
Skin damage	200

(a) Annual cost.

A.3.3 Cancers

HECOM can accept input data for first and second year treatment costs for cancers. Direct cost estimates developed in Chapter 5 are presented in Table A.17.

A.3.4 Genetic Effects

Two direct cost inputs are required for all types of genetic effects: an initial medical treatment cost of \$7,500 and the annual cost of institutionalization, \$12,500 in 1984 dollars. Data on impaired years and the percent of disability are used to project the relevant costs for each type of genetic effect.

TABLE A.17. Direct Costs of Cancers (1984 \$)

Cancer Type	Costs	
	First Year	Second Year
Leukemia	9,000	11,400
Lung	11,600	7,800
Breast	7,300	4,200
Bone	8,900	15,800
Skin	1,700	0
Gastrointestinal	13,300	6,000
Other	9,800	8,300
Thyroid - malignant	7,200	2,300
Thyroid - benign nodules	1,600	500
In utero - leukemia	9,000	11,400
In utero - other	9,800	8,300

A.4 INDIRECT COSTS

The HECOM model estimates indirect costs related to lost productivity for periods of morbidity and for premature mortality. Calculation of indirect costs due to morbidity requires data on work weeks lost as well as the earnings data and labor force participation rates required for the mortality cost calculation. These data are discussed in the following section. For genetic effect mortality costs, data on life shortening are also presented.

A.4.1 Mean Earnings by Age and Sex

Mean earnings information is used in estimating lifetime expected earnings to calculate indirect costs. The input data are for five-year age intervals. Either site-specific or national data can be used in HECOM. Table A.18 provides an example of the recent national earnings data. Mean earnings figures in 1984 dollars are listed by five-year age increments and by sex (U.S. Dept. of Commerce 1982). The Consumer Price Index for "all items" for all urban consumers is used to inflate the 1982 earnings estimates to 1984 dollars. (U.S. Dept. of Labor, selected years.)

A.4.2 Labor Force Participation Rate

Based on an analysis by Hartunian, Smart and Thompson (1981, p. 49) these data are the average of employment and housekeeping participation rates for 1970 and 1975 published in Employment and Earnings by the Bureau of Labor Statistics. Because 1970 was a high employment year and 1975 was a post-recession year, the average of the two years' rates was used to estimate expected labor force participation rates. The computed rates are listed in Table A.19.

TABLE A.18. Mean Earnings of Employed Persons,
by Age and Sex (1984 \$)^(a)

<u>Ages</u>	<u>Male</u>	<u>Female</u>
15 - 19 ^(b)	4,436	3,547
20 - 24	15,211	8,902
25 - 29	17,933	10,902
30 - 34	22,659	11,308
35 - 39	26,475	11,302
40 - 44	26,959	11,396
45 - 49	27,928	11,335
50 - 54	26,929	11,401
55 - 59	26,340	11,247
60 - 64	21,942	10,789
65 - 69	14,000	7,205
70 - 74	13,495	6,440
75 - 79	10,720	5,674
80+	10,720	5,674

- (a) 1980 incomes inflated to 1984 dollars by "All Items" index, Consumers Price Index for all urban consumers, as published by Bureau of Labor Statistics.
- (b) Income for 18-24 year-olds was allocated to 15-19 and 20-24 year-olds based on the population weighted relationship between these categories and 18-24 year-olds' income in 1969. The same procedure was used to compute income for 65-69, 70-74, 75-79, 80-85 age categories.

Source: U.S. Department of Commerce, Bureau of the Census. 1982.

A.4.3 Work Weeks Lost

Indirect costs are incurred when people are unable to work due to illness. Data for work weeks lost due to radiation injuries are developed from several sources and those for cancers are developed from Hartunian, et al. The data are presented in Table A.20.

A.4.4 Genetic Effect Life Shortening

Calculating indirect costs of genetic effects requires estimates of the years of life shortening associated with each major type of genetic effect. Estimates are given in Table A.21. These are based on a 70-year life span.

TABLE A.19. Employment and Housekeeping Participation Rates
by Age and Sex (percent)

<u>Ages</u>	<u>Male</u>	<u>Female</u>
16 - 19	49.4	49.3
20 - 24	76.6	84.0
25 - 29	89.7	93.0
30 - 34	92.9	93.6
35 - 39	93.4	94.0
40 - 44	92.7	94.5
45 - 49	91.6	94.6
50 - 54	88.5	94.2
55 - 59	84.2	94.0
60 - 64	68.5	91.8
65 - 69	35.8	88.3
70 - 74	17.9	78.0
75 - 79	9.3	74.6
80 - 84	5.3	73.4
85+	3.5	73.0

Source: Hartunian, Smart and Thompson. 1981.

TABLE A.20. Mean Number of Work Weeks Lost Following Onset of Illness

<u>Health Effects</u>	<u>Work Weeks Lost</u>
Radiation Injuries:	
Prodromal symptoms	1
Gastrointestinal syndrome	--(a)
Hematopoietic syndrome	26
Pulmonary syndrome	26
Hypothyroidism	1
Thyroiditis	1
Cataracts	1
Skin damage	1
Cancers:(b)	
Leukemia	16.3
Lung	19.9
Breast	17.2
Bone	23.3
Skin	1
Gastrointestinal(c)	19.7
Other(c)	18.2
Thyroid	5.9
Thyroid - benign nodules	1
In utero - leukemia	0
In utero - other	0

(a) Value of lost work time is included in the cost of premature mortality.

(b) Simple mean for all types of leukemia.

(c) Mean for gastrointestinal and "other," weighted by relative share of component cancer sites in the Third National Cancer Survey data.

Source: (cancer data) Hartunian, Smart and Thompson. 1981. P. 236.

TABLE A.21. Early Mortality Due to Genetic Effects

<u>Genetic Effect Type</u>	<u>Life Shortening (years)</u>
Autosomal dominant	13
X-linked	28
Unbalanced translocations	50
Aneuploidy	24
Multifactorial	30

Source: Evans, Moeller and Cooper. 1985. Vol. 1, p. 11.

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APPENDIX B

HECOM MAIN PROGRAMS

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CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
CC  MAIN PROGRAM CONTAINING SUBROUTINE CALL STATEMENTS
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C-----
C  INSERT COMMON BLOCK AND PARAMETER INFORMATION FROM FILE 'CONTROL.FOR'
C-----
C      INCLUDE 'CONTROL.FOR'
C-----
C  CALL SUBROUTINE READER TO READ IN DATA
C-----
C      CALL READER
C-----
C  READ CCDF DATA IF FLAG SET
C-----
C      IF (ICCDF .NE. 0) CALL READCD
C-----
C  SET CASE COUNTER AND TERMINATION FLAG FOR MULTIPLE CASES
C-----
C      NCAS = 0
C      TERMIN = 0
100  CONTINUE
C-----
C  INITIALIZE VARIABLES
C-----
C      CALL INIT
C-----
C  READ IN FATALITY AND MORBIDITY DATA FOR THIS CASE
C-----
C      CALL READSN
C      IF (TERMIN.EQ.1) GO TO 200
C      NCAS = NCAS + 1
C-----
C  CALCULATE COST OF MEDICAL MONITORING
C-----
C      IF (MONFLG.GT.0) CALL MONITOR
C-----
C  CALCULATE COST OF TREATING RADIATION INJURIES
C-----
C      IF (RADFLG.GT.0) CALL RADINJ
C-----
C  CALCULATE COST OF TREATING CANCERS
C-----
C      IF (CANFLG.GT.0) CALL CANCERS
C-----
C  CALCULATE DIRECT GENETIC COSTS
C-----
C      IF (GENFLG.GT.0) CALL GENETIC
C-----

```

```

C  CALCULATE INDIRECT COSTS
C-----
C      CALL INDIRECT
C-----
C  SUM UP COST TOTALS
C-----
C      CALL SUMTOT
C-----
C  IF ICCDF NE 0, SET UP CCDF OUTPUT
C-----
C      IF (ICCDF .NE. 0) CALL SETCCD
C-----
C  PRINT OUT RESULTS
C-----
C      IF (WRTFLG .GT. 0) CALL WRITER
C-----
C  GO BACK AND READ NEXT CASE
C-----
C      GO TO 100
C-----
C  TERMINATE
C-----
200 IF (ICCDF .NE. 0) CALL WRITCD
      STOP
      END

```

```

C FILE INCLUDE.FOR
C
C CONTAINS ALL THE COMMON BLOCKS USED BY THE HEALTH EFFECTS
C COST MODEL (HECOM).
C
C MAXIMUM INDICES ARE AS FOLLOWS :
C     NUMBER OF CANCER TYPES :          12
C     NUMBER OF AGE CATEGORIES :        20
C     NUMBER OF YEARS :                 85
C     NUMBER OF SEXES :                 2
C     NUMBER OF INCOME CATEGORIES :     21
C     NUMBER OF EARLY RADIATION INJURIES : 8
C     NUMBER OF LATE RADIATION INJURIES : 5
C     NUMBER OF GENETIC EFFECTS :       6
C     NUMBER OF GENETIC GENERATIONS :    7
C     NUMBER OF GENETIC EFFECT YEARS :   150
C     NUMBER OF VARIABLES FOR CCDF:     40
C     MAX NUMBER OF OBSERVATIONS FOR CCDF: 200
C
C IMPLICIT DOUBLE PRECISION (A-H,J-Z)
C
C COMMON/INDICE/AINC,IINC,INR(12),YEARS,AC,IC,SEX,GYEARS,
1 CTYPES,NER,NLR,GTYPES,NGEN,RHG,R,ICCDF,MYEARS
C
C COMMON/BIT/ RDEBIT(8),RDLBIT(5),CANBIT(12),GENBIT(6)
C
C COMMON/ITERA/TERMIN,NCAS
C
C COMMON/DISCFN/ BASE
C
C COMMON/FLAGS/ CANFLG,RADFLG,GENFLG,MONFLG,WRTFLG
C
C COMMON/NAMES/ CNAME(12),RENAME(8),RLNAME(5),GNAMES(6),
1 CASE
C
C COMMON/SPECA/ PRENAT,BREAST,LUNG,MALES,FEMALE,PULMON,
1 MICROC,RETARD,THYRID,THYBEN
C
C COMMON/COMIN/ POPFE(20,2),POPF(20,2),MAGE(20),ACAGE(20),
1 LIFE(85,2),ICAGE(21)
C
C COMMON/EARN/ MERN(20,2),LFPR(20,2),REG
C
C COMMON/CANIN/ LPC(12),MS(12),SPONFR(20,12),CF(12),
1 SPONMR(20,12),CNONE(12),POR(12),PPOR(12),CPCF(12),
2 CPCS(12),LWORKC(12),CFP(12)
C
C COMMON/CANWRK/ SUMDTH(12,20,2),SUMMRB(12,20,2)
C
C COMMON/CANOUT/ FAT(12,20,2),MORB(12,20,2),CCOSTS(12,2),
1 TOTCC(12),CCSTMR,CCSTFT,TCANC(3)

```

```

C      COMMON/INCANO/ CANIND(12,2),CANINC(12),CANINM,CANINF,
1      CANINT(3)
C
C      COMMON/MONIN/ ASC,RSC,PSD,ISC,PE,MSC,LTC,PSE,PHE,DCC,
1      DCD,ATC,RHC,RHD,MVC,NHC,NHD
C
C      COMMON/MONOUT/ RAD CST,RADMNC,TRNCST,MGTCST
C
C      COMMON/RADIN/ RADIE(8),RADFE(8),RADPFE(8),LPR(5),
1      RADLI(5),RADLPI(5),CPRADE(8),CPRADL(5),LWRKRE(8),
2      LWRKRL(5),RDISP(5),LRADCT(5)
C
C      COMMON/RADOUT/ RMPYE(20,2,8),RFPYE(20,2,8),RMORR(20,2,5),
1      RECAST(8,2),TOTREC(8),RECSTM,RECSTF,RLCOST(5,2),
2      TOTRLC(5),RLCSTM,SMRMRB(5,20,2),TERADC(3),TLRAOC(3)
3      ,RTCSTM,TTRADC(3),RTCSTF
C
C      COMMON/INRDEO/ RIEIND(8,2),RIEINS(8),RIEINM,RIEINF,
1      RIEINT(3),RILIND(5,2),RILINS(5),RILINM,RILINT(3)
2      ,RITINT(3),RITINM
C
C      COMMON/GENIN/ ERATE(6),GENI(6),GSRATE(6,2),LS(6),IMY(6),
1      INCOST(6),DISP(6),MEDCST(6)
C
C      COMMON/GENOUT/ GENPY(6,150,2),GCOST(6,2),GTCOST(6),
1      GMCOST,GENPG(6,7,2),TGENC(3)
C
C      COMMON/INGENO/ GENIND(6,2),GENINS(6),GENINM,GENINT(3),GENINF
C
C      COMMON/TOTALS/ TOTDRM,TOTDRF,TOTDIR,TOTINM,TOTINF,TOTIND,
1      TOTRAD,TOTCAN,TOTGEN,TOTAL
C
C      COMMON/CCDF/ CCDDAT(40,200),CDNAM(40),INC,INVAR
C
C      REAL*8  SUML(20,2),SUMC,IVL,NL,MPY,FPY,RMPYL,SUMG(150),
1      LVALUE,LPRBRL,LPRBAB,PV,FV,LWMRB,LWFAT,
2      RLP,SUMGL,ISC,IBF,IEF,ICAGE,IMY,INCOST,SUMLN(85,20,2)
C
C      INTEGER CANFLG,RADFLG,GENFLG,MONFLG,AC,SEX,CTYPES,AINC,
1      IINC,YEARS,GYEARS,NER,NLR,GTYPES,NGEN,AGE,
2      PRENAT,BREAST,LUNG,MALES,FEMALE,PULMON,MICROC,RETARD,
3      N,I,J,K,IB,A,S,C,G,RT,THYRID,THYBEN,AGECAT,MYEARS,
4      TERMIN,NCAS,RDEBIT,RDLBIT,CANBIT,GENBIT,WRTFLG
C
C      CHARACTER*20 CNAME,RENAME,RLNAME,GNAMES
C      CHARACTER*50 CASE
C      CHARACTER*8  CDNAM
C
C      END OF INCLUDE.FOR

```

```

C *****
C SUBROUTINE MONITOR
C *****
C INCLUDE 'CONTROL.FOR'
C CALLS THE SUBROUTINES TO CALCULATE THE MONITORING COSTS
C
C RADGST = 0.000
C RADMNC = 0.000
C TRNCST = 0.000
C
C IF(PSD.GT.0.000) CALL RADSCREEN
C IF(PHE.GT.0.000) CALL MEDTRAN
C IF(PSE.GT.0.000) CALL MEDMON
C
C CALCULATE TOTAL MEDICAL MONITORING COST
C
C MGTCST = RADGST + RADMNC + TRNCST
C
C RETURN
C END
C
C *****
C SUBROUTINE RADSCREEN
C *****
C INCLUDE 'CONTROL.FOR'
C
C CALCULATES RADIATION SCREENING COSTS IF POPULATION 9D DEGS
C DOWNWIND (PSD) IS GREATER THAN ZERO
C
C RADGST = ASC*PSD + BSC*PSO + ISC*PE
C RETURN
C END
C
C *****
C SUBROUTINE MEDMON
C *****
C CALCULATES MEDICAL MONITORING COSTS IF POPULATION EXPOSED
C TO OVER 1 GY (PSE) IS GREATER THAN ZERO
C
C INCLUDE 'CONTROL.FOR'
C RADMNC = (MSC + LTC) * PSE
C RETURN
C END
C
C

```

```

C *****
C      SUBROUTINE MEDTRAN
C *****
C      INCLUDE 'CONTROL.FOR'
C      REAL*8 VAL,LOV
C
C      CALCULATES MEDICAL TRANSPORTATION COSTS IF PHE IS
C      GREATER THEN ZERO
C
C      TRNCST = DMIN1(PHE,DCC) * DCD * ATC
C      IF( (PHE-DCC).LE.0.0) GO TO 999
C      TRNCST = TRNCST + (DMIN1(PHE-DCC,RHC) * RHD * MVC)
C      VAL = PHE-DCC-RHC
C      IF(VAL.LE.0.0) GO TO 999
C      TRNCST = TRNCST + (DMIN1(VAL,NHC) * NHD * ATC)
C      LOV = PHE-DCC-RHC-NHC
C      IF(LOV.LE.0.0) GO TO 999
C      TRNCST = TRNCST + (LOV * RHD * MVC)
999  CONTINUE
      RETURN
      END
C

```



```

C *****
C      SUBROUTINE RADINJ
C *****
C
C      RUNS THE CALCULATIONS FOR RADIATION INJURIES
C
C      INCLUDE 'CONTROL.FOR'
C
C      DO 10 RT=1,NER
C          IF(RDEBIT(RT) .LE. 0) GO TO 10
C          CALL RADEMORB(RT)
C          CALL RADEFAT(RT)
C          CALL RADECOST(RT)
10  CONTINUE
C
C      DO 20 RT=1,NLR
C          IF(RDLBIT(RT) .LE. 0) GO TO 20
C          CALL RADLSICK(RT)
C          CALL SUMRLM(RT)
C          CALL RADLCOST(RT)
20  CONTINUE
C
C      RETURN
C      END
C
C *****
C      SUBROUTINE RADEMORB(RT)
C *****
C
C      ASSIGNS SICKNESS(MORBIDITY) DUE TO RADIATION
C      INJURIES TO POPULATION COHORTS FOR THOSE INJURY TYPES THAT
C      OCCUR IN THE FIRST YEAR ONLY
C
C      INCLUDE 'CONTROL.FOR'
C
C      DO 100 A=1,AC
C      DO 100 S=1,SEX
100  RMPYE(A,S,RT) = RADIE(RT) * POPFE(A,S)
C
C      RETURN
C      END
C
C *****
C      SUBROUTINE RADEFAT(RT)
C *****
C
C      ASSIGNS FATALITIES DUE TO RADIATION INJURY
C      TYPES THAT OCCUR WITHIN THE FIRST YEAR TO POPULATION COHORTS

```

```

C      INCLUDE 'CONTROL.FOR'
C
C      SEPARATE EQUATION NEEDED FOR PULMONARY SYNDROME
C      IF(RT.EQ.PULMON) GO TO 120
C
C      DO 100 A=1,AC
C      DO 100 S=1,SEX
C
C      RFPYE(A,S,RT) = RADFE(RT) * POPFE(A,S)
C
C 100  CONTINUE
C      GO TO 999
C
C  FOR PULMONARY SYNDROME :
C  ALLOCATE ALL CASES TO THOSE UNDER 5 AND OVER 50 YEARS OLD
C
C 120  CONTINUE
C
C      RT = PULMON
C
C      SET YOUNGER BOUND
C      IYB = 5 - 1
C      SET OLDER BOUND
C      IOB = 50
C
C      SUMP = 0.0
C      DO 390 S=1,SEX
C  SUM POPFE OVER THE AGE CATEGORIES TO BE USED
C
C      DO 310 A=1,AGECAT(IYB)
C      SUMP = SUMP + POPFE(A,S)
C 310  CONTINUE
C      DO 320 A=AGECAT(IOB),AC
C      SUMP = SUMP + POPFE(A,S)
C 320  CONTINUE
C 390  CONTINUE
C
C  CALCULATE EARLY RADIATION INJURIES FOR PULMONARY SYNDROME
C
C      DO 391 S=1,SEX
C      DO 350 A=1,AGECAT(IYB)
C      RFPYE(A,S,RT) = RADFE(RT) * (POPFE(A,S)/SUMP)
C 350  CONTINUE
C
C      DO 360 A=AGECAT(IOB),AC
C      RFPYE(A,S,RT) = RADFE(RT) * (POPFE(A,S)/SUMP)
C 360  CONTINUE
C
C 391  CONTINUE
C

```

```

999    CONTINUE
C
C    FOR PRENATAL FATALITIES
C
      RS = DFLOAT(SEX)
      DO 200 S=1,SEX
      RFPYE(PRENAT,S,RT) = RADPFE(RT) / RS
200    CONTINUE
C
      RETURN
      END
C
C *****
C    SUBROUTINE RADECOST(RT)
C *****
C
C    CALCULATE DIRECT COSTS FOR THE INJURY TYPES THAT OCCUR IN THE 1ST YEAR
C
      INCLUDE 'CONTROL.FOR'
      REAL *8 SUM(2)
C
      DO 120 S=1,SEX
C
C
      DO 10 I=1,2
10    SUM(I) = 0.0
      DO 110 A=1,AC+1
C
      N=1
      SUM(1) = SUM(1) + (RMPYE(A,S,RT) *
X    PV(FV(CPRADE(RT),RHG,N),R,N))
C
      SUM(2) = SUM(2) + (RFPYE(A,S,RT) *
X    PV(FV(CPRADE(RT),RHG,N),R,N))
C
110    CONTINUE
C
      RECSTM = RECSTM + SUM(1)
      RECSTF = RECSTF + SUM(2)
      RECOST(RT,S) = SUM(1) + SUM(2)
      TOTREC(RT) = TOTREC(RT) + RECOST(RT,S)
C
120    CONTINUE
C
200    CONTINUE
C
      DO 300 S=1,SEX
      TERADC(S) = TERADC(S) + RECOST(RT,S)
300    CONTINUE
      TERADC(SEX+1) = RECSTM + RECSTF
C

```

```

        RETURN
        END
C
C
C *****
      SUBROUTINE RADLSICK(RT)
C *****
C
C   ALLOCATES MORBIDITY TO POPULATION COHORTS FOR THOSE INJURY TYPES
C   THAT OCCUR AFTER THE FIRST YEAR
C
C   INCLUDE 'CONTROL.FOR'
C
C
C   DO 10 A=1,AC+1
C   DO 10 S=1,SEX
10    SUML(A,S) = 0.0
      SUMC = 0
C
C   ASSIGN MICROCEPHALY AND MENTAL RETARDATION ONLY TO PRENATAL
C   AGE CATEGORY
C
C   IF(RT.EQ.MICROC .OR. RT.EQ.RETARD) GO TO 555
C
C   DO 110 A=1,AC
C   DO 110 S=1,SEX
      IVL = MAGE(A) + LPR(RT)
      IBT = IDINT(IVL)
      IB = MIN(IBT,YEARS)
      DO 120 N=IB,YEARS
120    SUML(A,S) = SUML(A,S) + LPRBRL(IVL,S,N)
      CONTINUE
C
      SUMC = SUMC + POPFE(A,S) * SUML(A,S)
110    CONTINUE
C
      IF(SUMC .LE.0.000) GO TO 555
C
C   DO 111 A=1,AC
C   DO 111 S=1,SEX
C
      RMORB(A,S,RT) = RADLI(RT) * POPFE(A,S) * SUML(A,S) / SUMC
111    CONTINUE
C
C
C
555    CONTINUE
C
C   FOR PRENATAL AGE CATEGORY
C
      RS = DFLOAT(SEX)

```

```

      DO 210 S=1,SEX
      RMORB(PRENAT,S,RT) = RADLPI(RT) / RS
210  CONTINUE
C
C 100  CONTINUE
C
      RETURN
      END
C
C
C *****
C      FUNCTION RMPYL(N,A,S,RT)
C *****
C      CALCULATES THE MORBIDITIES PER YEAR FOR RADIATION INJURY TYPES THAT
C      HAVE EFFECTS PAST THE FIRST YEAR
C
C      INCLUDE 'CONTROL.FOR'
C
C      RMPYL = 0.000
C      IF(SMRMRB(RT,A,S).LE.0.000) GO TO 120
C
C      RMPYL = RMORB(A,S,RT) * LPRBRL(MAGE(A)+LPR(RT),S,N) * POPFE(A,S)
X    / SMRMRB(RT,A,S)
C
120  CONTINUE
C
      RETURN
      END
C
C
C *****
C      SUBROUTINE SUMRLM(RT)
C *****
C      CALCULATES THE SUM NEEDED TO CALCULATE MORBIDITIES PER YEAR FOR
C      THOSE INJURY CATEGORIES THAT HAVE EFFECTS PAST THE FIRST YEAR
C
C      INCLUDE 'CONTROL.FOR'
C
C      DO 10 A=1,AC+1
C      DO 10 S=1,SEX
10    SMRMRB(RT,A,S) = 0.0
C
C      DO 130 A=1,AC+1
C      DO 130 S=1,SEX
C          IVL = MAGE(A) + LPR(RT)
C          IBT = IDINT(IVL)
C          IB = MIN(IBT,YEARS)
C      DO 130 N=IB,YEARS
C          SMRMRB(RT,A,S) = SMRMRB(RT,A,S) + (LPRBRL(IVL,S,N)*POPFE(A,S))

```

```

130    CONTINUE
C
      RETURN
      END
C
C
C *****
      SUBROUTINE RADLCOST(RT)
C *****
C
C   CALCULATES THE DIRECT COSTS FOR THOSE INJURY TYPES THAT HAVE
C   EFFECTS PAST THE FIRST YEAR
C
C   INCLUDE 'CONTROL.FOR'
C   REAL*8 SUM
C
C   DO 150 S=1,SEX
C
C   SUM = 0.0
C
C   COST EQUATION TYPE ONE   E.G. CATARACTS
C
C   IF(LRADCT(RT).EQ.1) THEN
C
C
C   DO 110 A=1,AC
C     IM = IDINT(MAGE(A))
C     BT = MAGE(A) + LPR(RT)
C     IBT = IDINT(BT)
C     IB = MIN(IBT,YEARS)
C     DO 110 N=IB,YEARS
C
C     J = N - IM
C     SUM = SUM + (RMPYL(N,A,S,RT) *
X    PV(FV(CPRADL(RT),RHG,J),R,J))
C
C 110    CONTINUE
C
C
C   COST EQUATION TYPE THREE   E.G. PRENATAL CASES
C
C   ELSE IF(LRADCT(RT).EQ.3) THEN
C
C   A = PRENAT
C   IM = IDINT(MAGE(A))
C   RT = MAGE(PRENAT) + LPR(RT)
C   IBT = IDINT(BT)
C   IB = MIN(IBT,YEARS)
C   RIB = DFLOAT(IB)
C   IF(IB.LE.0) IB = 1
C   DO 115 N=IB,YEARS

```

```

C      J = N - IM
C
C      SUM = SUM + (RMORB(A,S,RT) * LPRBRL(RIB,S,N)
X      * (RDISP(RT)/100) * PV(FV(CPRADL(RT),RHG,J),R,J))
C
115  CONTINUE
C
C
C      COST EQUATION TYPE TWO    E.G. HYPOTHYROIDISM
C
C      ELSE IF(LRADCT(RT).EQ.2) THEN
C
C      DO 400 A=1,AC
C      IBF = MAGE(A) + LPR(RT)
C      IBT = IDINT(IBF)
C      IB = MIN(IBT,YEARS)
C      IF(IB.LE.0) IB = 1
C      IM = IDINT(MAGE(A))
C
C      DO 400 N=IB,YEARS
C      SUMC = 0.0
C      DO 401 IONS=N,YEARS
C      J = IONS - IM
C      SUMC = SUMC + PV(FV(CPRADL(RT),RHG,J),R,J)
401  CONTINUE
C
C      SUM = SUM + (RMPYL(N,A,S,RT) * SUMC)
400  CONTINUE
C
C      ENDIF
C
C      RLCSTM = RLCSTM + SUM
C      RLCOST(RT,S) = SUM
C      TOTRLC(RT) = TOTRLC(RT) + RLCOST(RT,S)
C
150  CONTINUE
200  CONTINUE
C
C      DO 300 S=1,SEX
C      TLRADC(S) = TLRADC(S) + RLCOST(RT,S)
300  CONTINUE
C      TLRADC(SEX+1) = RLCSTM
C
C      RETURN
C      END

```

```

C *****
C SUBROUTINE CANCERS
C *****
C INCLUDE 'CONTROL.FOR'
C
C DO 10 I=1,CTYPES
C IF(CANBIT(I) .LE. 0) GO TO 10
C
C RELATIVE RISK MODEL FOR CANCER TYPE I
C
C IF(INR(I) .GT. 0) THEN
C   CALL FATREL(I)
C   CALL SICKREL(I)
C   CALL SUMREL(I)
C ELSE
C
C ABSOLUTE RISK MODEL FOR CANCER TYPE I
C
C   CALL FATAB(I)
C   CALL SICKAB(I)
C   CALL SUMAB(I)
C   ENDIF
10 CONTINUE
C
C CALCULATE DIRECT CANCER COSTS
C
C CALL CANCOST
C
C RETURN
C END
C
C *****
C SUBROUTINE SICKAB(C)
C *****
C INCLUDE 'CONTROL.FOR'
C
C ABSOLUTE RISK MODEL
C ALLOCATES MORBIDITIES OF CANCER TYPE(C) TO AGE CATEGORY(A),AND
C SEX(S)
C
C DO 10 A=1,AC
C DO 10 S=1,SEX
10 SUML(A,S) = 0.
C SUMC = 0
C
C WAYT = 1.0
C
C DO 100 A=1,AC
C DO 100 S=1,SEX

```



```

      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
      IVL = MAGE(A) + LPC(C) + POR(C)
      IV = IDINT(IVL)
      IE = MIN(YEARS,IV)
      BT = MAGE(A) + LPC(C)
      IBT = IDINT(BT)
      IB = MIN(IBT,YEARS)
      RIB = DFLOAT(IB)
      DO 110 N=IB,IE
110      SUML(A,S) = SUML(A,S) + LPRBRL(RIB,S,N)
C
C      SET THYROID AGE AND SEX WEIGHTS
C
      IF(C.EQ.THYRID.OR.C.EQ.THYBEN) THEN
        IF(S.EQ.FEMALE) WAYT = 2.5
        IF(ACAGE(A).LT.18) WAYT = 2.0
        IF(S.EQ.FEMALE .AND. ACAGE(A).LT.18) WAYT = 5.0
      ENDIF
C
      SUMC = SUMC + (WAYT * POPFE(A,S) *SUML(A,S))
100      CONTINUE
C
      WAYT = 1.0
      IF(SUMC.LE.0.0) GO TO 23
      DO 200 A=1,AC
      DO 200 S=1,SEX
C
      IF(C.EQ.THYRID.OR.C.EQ.THYBEN) THEN
        IF(S.EQ.FEMALE) WAYT = 2.5
        IF(ACAGE(A).LT.18) WAYT = 2.0
        IF(S.EQ.FEMALE .AND. ACAGE(A).LT.18) WAYT = 5.0
      ENDIF
C
      MORB(C,A,S) = CNONF(C) * WAYT * POPFE(A,S) * SUML(A,S)/SUMC
200      CONTINUE
C
23      RETURN
      END
C
C *****
C      SUBROUTINE SICKREL(C)
C *****
C      INCLUDE 'CONTROL.FOR'
C
C      RELATIVE RISK MODEL
C      ALLOCATES MORBIDITIES OF CANCER TYPE(C) TO AGE CATEGORY(A),AND
C      SEX(S)
C
      DO 10 A=1,AC
      DO 10 S=1,SEX
10      SUML(A,S) = 0.

```

```

      SUMC = 0
C
      DO 100 A=1,AC
      DO 100 S=1,SEX
      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
      IBF = (MAGE(A) + LPC(C))
      IRT = IDINT(IBF)
      IB = MIN(IRT,YEARS)
      DO 110 N=IB,YEARS
110      SUML(A,S) = SUML(A,S) + ((SPONMR(AGECAT(N),C)/100000) *
X          LPRBRL(IBF,S,N))
C
      SUMC = SUMC + POPFE(A,S) * SUML(A,S)
100      CONTINUE
C
      IF(SUMC.LE.0.0) GO TO 23
      DO 200 A=1,AC
      DO 200 S=1,SEX
      MORB(C,A,S) = CNONF(C) * POPFE(A,S) * SUML(A,S)/SUMC
200      CONTINUE
C
23      RETURN
      END
C
C *****
C      SUBROUTINE SUMAB(C)
C *****
C      INCLUDE 'CONTROL.FOR'
C
C      ABSOLUTE RISK MODEL
C      CALCULATES THE INTERMEDIATE SUM VALUES NEEDED FOR THE CALCULATION
C      OF FATALITY AND MORBIDITY PER YEAR
C
C      SUM NEEDED TO CALCULATE FATALITIES PER YEAR
C
      DO 10 A=1,AC+1
      DO 10 S=1,SEX
10      SUMDTH(C,A,S) = 0.
C
      DO 100 A=1,AC
      DO 100 S=1,SEX
      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
      IVL = MAGE(A) + LPC(C) + POR(C) + MS(C) - 1.0
      IV = IDINT(IVL)
      IE = MIN(YEARS,IV)
      BT = MAGE(A) + LPC(C) + MS(C)
      IBT = IDINT(BT)
      IB = MIN(IRT,YEARS)
      RIB = DFLOAT(IB)
      DO 110 N=IB,IE

```

```

AGE = N
IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 110
IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 11D
SUMDTH(C,A,S)=SUMDTH(C,A,S)+(LPRBRL(RIB,S,N)*POPFE(A,S))
110 CONTINUE
100 CONTINUE
C
C
IF(CFP(C).LE.0.0) GO TO 205
DO 200 S=1,SEX
IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 200
IVL = MAGE(PRENAT) + LPC(C) + PPOR(C) + MS(C) - 1.0
IV = IDINT(IVL)
IE = MIN(IV,YEARS)
BT = MAGE(PRENAT) + LPC(C) + MS(C)
IBT = IDINT(BT)
IB = MIN(IBT,YEARS)
RIB = DFLOAT(IB)
DO 210 N=IB,IE
AGE = N
IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 210
IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 210
SUMDTH(C,PRENAT,S) = SUMDTH(C,PRENAT,S) + (LPRBRL(RIB,S,N) *
X POPFE(PRENAT,S))
210 CONTINUE
200 CONTINUE
205 CONTINUE
C
C
C SUM TO CALCULATE MORBIDITIES PER YEAR
C
DO 30 A=1,AC
DO 30 S=1,SEX
30 SUMMRB(C,A,S) = 0.0D0
C
DO 300 A=1,AC
DO 300 S=1,SEX
IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 300
IVL = MAGE(A) + LPC(C) + POR(C)
IV = IDINT(IVL)
IE = MIN(YEARS,IV)
BT = MAGE(A) + LPC(C)
IBT = IDINT(BT)
IB = MIN(IBT,YEARS)
RIB = DFLOAT(IB)
DO 310 N=IB,IE
AGE = N
IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 310
IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 310
SUMMRB(C,A,S)=SUMMRB(C,A,S)+(LPRBRL(RIB,S,N)*POPFE(A,S))
310 CONTINUE

```

```

300    CONTINUE
C
      RETURN
      END
C
C
C *****
      SUBROUTINE SUMREL(C)
C *****
      INCLUDE 'CONTROL.FOR'
C
C    RELATIVE RISK MODEL
C    CALCULATES THE INTERMEDIATE SUM VALUES NEEDED FOR THE CALCULATION
C    OF FATALITY AND MORBIDITY PER YEAR
C
C    CALCULATE SUM USED TO CALCULATE FATALITIES PER YEAR
C
      DO 10 A=1,AC
      DO 10 S=1,SEX
10      SUMDTH(C,A,S) = 0.
C
      DO 100 A=1,AC
      DO 100 S=1,SEX
      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
      IBF = (MAGE(A) + LPC(C) + MS(C))
      IBT = IDINT(IBF)
      IB = MIN(IBT,YEARS)
      RIB = DFLOAT(IB)
      DO 110 N=IB,YEARS
      AGE = N
      IAGETM = N
      IAGECT = AGECACT(N)
      IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 110
      IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 110
      SUMDTH(C,A,S)=SUMDTH(C,A,S)+(LPRBRL(RIB,S,N)
X      * (SPONFR(AGECACT(N),C)/100000))
110      CONTINUE
100      CONTINUE
C
C
C
C    SUM TO CALCULATE MORBIDITIES PER YEAR
C
      DO 20 A=1,AC
      DO 20 S=1,SEX
20      SUMMRB(C,A,S) = 0.
C
      DO 200 A=1,AC
      DO 200 S=1,SEX
      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 200

```

```

        IBF = (MAGE(A) + LPC(C))
        IBT = IDINT(IBF)
        IB = MIN(IBT,YEARS)
        RIB = DFLOAT(IB)
        DO 210 N=IB,YEARS
            AGE = N
            IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 210
            IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 210
            SUMMRB(C,A,S)=SUMMRB(C,A,S)+(LPRBRL(RIB,S,N)
X      * (SPONMR(AGECAT(N),C)/100000))
210    CONTINUE
200    CONTINUE
C
        RETURN
        END
C
C
C
C *****
      FUNCTION MPY(C,A,N,S)
C *****
      INCLUDE 'CONTROL.FOR'
C
      ALLOCATES MORBIDITIES OF CANCER TYPE(C) , AGE CATEGORY(A),
      AND SEX(S) TO YEAR(N) : MORBIDITIES PER YEAR
C
      MPY = 0.0
      AGE = N
C
      IF(INR(C) .GT.0) THEN
C
C      CALCULATE USING THE RELATIVE RISK MODEL
C
        IF(SUMMRB(C,A,S).LE.0.000) GO TO 100
        IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 100
        IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 100
        MPY = MORB(C,A,S) * LPRBRL(MAGE(A)+LPC(C),S,N)
X      * (SPONMR(AGECAT(N),C)/100000) / SUMMRB(C,A,S)
100    CONTINUE
C
        ELSE
C
C      CALCULATE USING THE ABSOLUTE RISK MODEL
C
        IF(SUMMRB(C,A,S).LE.0.000) GO TO 200
        TI = MAGE(A) + LPC(C) + POR(C)
        IT = IDINT(TI)
        IF(AGE.GT.IT) GO TO 200
        IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 200
        IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 200

```

```

      MPY=MORB(C,A,S)*LPRBRL(MAGE(A)+LPC(C),S,N)*POPFE(A,S)
X    / SUMMRB(C,A,S)
200  CONTINUE
      ENDIF
C
      RETURN
      END
C
C
C *****
      SUBROUTINE FATAB(C)
C *****
      INCLUDE 'CONTROL.FOR'
      INTEGER IV
C
C      ABSOLUTE RISK MODEL
C      ALLOCATES FATALITIES OF CANCER TYPE(C) TO AGE CATEGORY(A),AND
C      SEX(S)
C
      DO 10 A=1,AC
      DO 10 S=1,SEX
10    SUML(A,S) = 0.
      SUMC = 0
C
      WAYT = 1.0
C
      DO 100 A=1,AC
      DO 100 S=1,SEX
      IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
      IVL = MAGE(A) + LPC(C) + POR(C) + MS(C) - 1.0
      IV = IDINT(IVL)
      IE = MIN(YEARS,IV)
      BT = MAGE(A) + LPC(C) + MS(C)
      IBT = IDINT(BT)
      IB = MIN(IBT,YEARS)
      RIB = DFLOAT(IB)
      DO 110 N=IB,IE
110    SUML(A,S) = SUML(A,S) + LPRBRL(RIB,S,N)
C
C      SET THYROID AGE AND SEX WEIGHTS
C
      IF(C.EQ.THYRID.OR.C.EQ.THYBEN) THEN
      IF(S.EQ.FEMALE) WAYT = 2.5
      IF(ACAGE(A).LT.18) WAYT = 2.0
      IF(S.EQ.FEMALE .AND. ACAGE(A).LT.18) WAYT = 5.0
      ENDIF
C
      SUMC = SUMC + (WAYT * POPFE(A,S) *SUML(A,S))
100  CONTINUE
C
      WAYT = 1.0

```

```

DO 205 S=1,SEX
C
IF(C.EQ.THYRID .OR. C.EQ.THYBEN) THEN
  WAYT = 2.0
  IF(S.EQ.FEMALE) WAYT = 5.0
ENDIF
C
RS = DFLOAT(SEX)
FAT(C,PRENAT,S) = (CFP(C) * WAYT) / RS
CC
IF(SUMC.LE.0.0) GO TO 205
DO 200 A=1,AC
C
IF(C.EQ.THYRID.OR.C.EQ.THYBEN) THEN
  IF(S.EQ.FEMALE) WAYT = 2.5
  IF(ACAGE(A).LT.18) WAYT = 2.0
  IF(S.EQ.FEMALE .AND. ACAGE(A).LT.18) WAYT = 5.0
ENDIF
C
FAT(C,A,S) = CF(C) * WAYT * POPFE(A,S) * SUML(A,S)/SUMC
200 CONTINUE
205 CONTINUE
RETURN
END
C
C *****
C SUBROUTINE FATREL(C)
C *****
C INCLUDE 'CONTROL.FOR'
C
C RELATIVE RISK MODEL
C ALLOCATES FATALITIES OF CANCER TYPE(C) TO AGE CATEGORY(A),AND
C SEX(S)
C
DO 10 A=1,AC
DO 10 S=1,SEX
10 SUML(A,S) = 0.
SUMC = 0.0
C
DO 100 A=1,AC
DO 100 S=1,SEX
IF(C.EQ.BREAST.AND.S.EQ.MALES) GO TO 100
IBF = (MAGE(A) + LPC(C) + MS(C))
IBT = IDINT(IBF)
IB = MIN(IBT,YEARS)
DO 110 N=IB,YEARS
110 SUML(A,S) = SUML(A,S) + ((SPONFR(AGECAT(N),C)/100000) *
X LPRBRL(IBF,S,N))
C
SUMC = SUMC + POPFE(A,S) * SUML(A,S)
100 CONTINUE

```

```

C      IF(SUMC.LE.0.0) GO TO 23
      DO 200 A=1,AC
      DO 200 S=1,SEX
      FAT(C,A,S) = CF(C) * POPFE(A,S) * SUML(A,S)/SUMC
200    CONTINUE
C
C      23    RETURN
      END
C
C
C      *****
      FUNCTION FPY(C,A,N,S)
C      *****
      INCLUDE 'CONTROL.FOR'
C
C      ALLOCATES FATALITIES OF CANCER TYPE(C) , AGE CATEGORY(A),
C      AND SEX(S) TO YEAR(N) : FATALITIES PER YEAR
C
C
C      FPY = 0.0
      AGE = N
C
C      IF(INR(C) .GT. 0) THEN
C
C      CALCULATE USING RELATIVE RISK MODEL
C
      IF(SUMOTH(C,A,S).LE.0.000) GO TO 100
      IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 100
      IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 100
      FPY = FAT(C,A,S) * LPRBRL(MAGE(A)+LPC(C)+MS(C),S,N)
      X * (SPONFR(AGECAT(N),C)/100000) / SUMDTH(C,A,S)
100    CONTINUE
C
      ELSE
C
C      CALCULATE USING ABSOLUTE MODEL
C
      IF(A.EQ.PRENAT) GO TO 201
      IF(SUMDTH(C,A,S).LE. 0.000) GO TO 130
      TI = MAGE(A) + LPC(C) + POR(C) + MS(C) - 1.0
      IT = IOINT(TI)
      IF(AGE.GT.TI) GO TO 130
      IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 130
      IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 130
      FPY=FAT(C,A,S)* LPRBRL(MAGE(A)+LPC(C)+MS(C),S,N)*POPFE(A,S)
      X / SUMDTH(C,A,S)
130    CONTINUE
      GO TO 211
C
201    IF(SUMDTH(C,PRENAT,S).LE.0.000) GD TO 200

```



```

        TI = MAGE(A) + PPOR(C) + LPC(C) + MS(C) - 1.0
        IT = IDINT(TI)
        IF(AGE.GT.IT) GO TO 200
        IF(C.EQ.BREAST .AND. AGE.LT.30) GO TO 200
        IF(C.EQ.LUNG .AND. AGE.LT.40) GO TO 200
        FPY=FAT(C,PRENAT,S) * LPRBRL(MAGE(PRENAT)+
X   LPC(C)+MS(C),S,N)*POPFE(PRENAT,S) / SUMDTH(C,PRENAT,S)
C
200    CONTINUE
211    CONTINUE
        ENDIF
        RETURN
        END
C
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE CANCOST
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C   THIS SUBROUTINE CALCULATES THE PRESENT VALUE COST OF TREATING
C   CANCERS RESULTING FROM RADIATION EXPOSURE.
C
        INCLUDE 'CONTROL.FOR'
        REAL*8 SUM(4),SUMFPY(20,2)
C
        DO 100 C=1,CTYPES
        DO 100 S=1,SEX
C
        DO 10 I=1,4
10    SUM(I) = 0.0
C
        DO 110 A=1,AC+1
        IM = IDINT(MAGE(A))
        IF(A.EQ.PRENAT) GO TO 125
        BT = MAGE(A) + LPC(C)
        IBT = IDINT(BT)
        IB = MIN(YEARS,IBT)
        IF(IB.LE.0) IB = 1
        DO 120 N=IB,YEARS
        J = N-IM+1
        SUM(1) = SUM(1) + MPY(C,A,N,S)* PV( FV(CPCF(C),RHG,J),
X   R,J)
C
        SUM(2) = SUM(2) + MPY(C,A,N,S) * PV(FV(CPCS(C),RHG,J+1),
X   R,J+1)
C
120    CONTINUE
125    CONTINUE
C
CC

```

```

      BT = MAGE(A) + LPC(C) + MS(C)
      IBT = IDINT(BT)
      IB = MIN(IBT, YEARS)
      IF (IB, LE, 0) IB = 1
      IF (C, EQ, 1) SUMFPY(A, S) = 0.0
      DO 111 N=IB, YEARS
        J = N - IM + 1
        SUM(3) = SUM(3) + FPY(C, A, N, S) * PV(FV(CPCF(C), RHG, J-1),
X      R, J-1)
C
      SUM(4) = SUM(4) + FPY(C, A, N, S) * PV(FV(CPCS(C), RHG, J), R, J)
C
      IF (C, EQ, 1) SUMFPY(A, S) = SUMFPY(A, S) + FPY(C, A, N, S)
111    CONTINUE
C
110    CONTINUE
C
      CCSTMR = CCSTMR + SUM(1) + SUM(2)
      CCSTFT = CCSTFT + SUM(3) + SUM(4)
      CCOSTS(C, S) = SUM(1) + SUM(2) + SUM(3) + SUM(4)
C
100    CONTINUE
C
      DO 200 S=1, SEX
      DO 200 C=1, CTYPES
      TOTCC(C) = TOTCC(C) + CCOSTS(C, S)
      TCANC(S) = TCANC(S) + CCOSTS(C, S)
200    CONTINUE
      TCANC(SEX+1) = CCSTMR + CCSTFT
C
      RETURN
      ENO
C

```

```

C *****
C SUBROUTINE GENETIC
C *****
C
C CALLS THE ROUTINES TO RUN THE GENETIC EFFECTS COST CALCULATIONS
C
C INCLUDE 'CONTROL.FOR'
C
C DO 10 G=1,GYPES
C   IF(GENBIT(G) .LE. 0) GO TO 10
C   CALL GENDIST(G)
C   CALL GENCOST(G)
10  CONTINUE
C
C RETURN
C END
C
C *****
C SUBROUTINE GENDIST(G)
C *****
C
C THIS SUBROUTINE DISTRIBUTES GENETIC EFFECTS TO EACH YEAR AFTER
C EXPOSURE
C
C INCLUDE 'CONTROL.FOR'
C INTEGER YPG,L
C   J = G
C -----
C CALCULATE THE NUMBER OF YEARS PER GENERATION
C -----
C   YPG=GYEARS/NGEN
C   SUM = 1
C -----
C CALCULATE FIRST GENERATION EFFECTS
C -----
C   DO 2000 I=1,SEX
C   DO 1000 K=2,NGEN
C -----
C SUM UP DECAY DIVISOR
C -----
C   SUM=SUM+(1-(ERATE(J)/100))**(K-1)
1000 CONTINUE
C -----
C USE SUM TO COMPUTE FIRST YEAR EFFECTS
C -----
C   GENPG(J,1,I)=GENI(J)*(GSRATE(J,I)/100)/SUM
C -----
C SET SUM TO 1 (X**0)
C -----

```

```

      SUM=1
2000 CONTINUE
C-----
C   CALCULATE EFFECTS IN REMAINING GENERATIONS
C-----
      DO 3000 I=1,SEX
      DO 3000 K=2,NGEN
      GENPG(J,K,I)=GENPG(J,K-1,I)*(1-(ERATE(J)/100))
3000 CONTINUE
C-----
C   ALLOCATE GENERATIONAL EFFECTS EQUALLY TO EACH YEAR WITHIN GENERATION
C-----
      DO 4000 I=1,SEX
      DO 4000 K=1,NGEN
      DO 4000 L=1,YPG
      GENPY(J,((K-1)*YPG)+L,I)=GENPG(J,K,I)/YPG
4000 CONTINUE
      RETURN
      END
C
C *****
C   SUBROUTINE GENCOST(G)
C *****
C
C
C   INCLUDE 'CONTROL.FDR'
C
      DO 10 I=1,GYEARS
10    SUMG(I) = 0.000
C
      RM = DFLOAT(MYEARS)
      IBF = (RM-LS(G)-IMY(G))
      IEF = (RM-LS(G))
      IB = IDINT(IBF)
      IE = IDINT(IEF)
C
      DO 110 N=1,GYEARS
      DO 110 J=IB,IE
      SUMG(N)=SUMG(N)+PV(FV((INCGST(G)*(DISP(G)/100)),RHG,N+J),R,N+J)
110    CONTINUE
C
      DO 130 S=1,SEX
      DO 120 N=1,GYEARS
      GCOST(G,S)=GCOST(G,S) + (GENPY(G,N,S)* SUMG(N))
      X   + (PV(FV(MEDCST(G),RHG,N),R,N) * GENPY(G,N,S))
120    CONTINUE
      GTCOST(G) = GTCOST(G) + GCOST(G,S)
130    CONTINUE
C
      GMCOST = GMCOST + GTCOST(G)
C

```

```

100    CONTINUE
C
      DO 300 S=1,SEX
        TGENC(S) = TGENC(S) + GCOST(G,S)
300    CONTINUE
        TGENC(SEX+1) = GMCOST
C
      RETURN
      END
C

```

```

C *****
C      SUBROUTINE INDIRECT
C *****
C      CALLS THE ROUTINES TO CALCULATE THE INDIRECT COSTS
C
C      INCLUDE 'CONTROL.FOR'
C
C      IF(RADFLG.GT.0) THEN
C      DO 10 RT=1,NER
C          IF(RDEBIT(RT) .LE. 0) GO TO 10
C          CALL RWORKE(RT)
C          CALL RLOSTV(RT)
10      CONTINUE
C
C      DO 11 RT=1,NLR
C          IF(RDLBIT(RT) .GT. 0) CALL RWORKL(RT)
11      CONTINUE
C
C      ENDIF
C
C      IF(CANFLG.GT.0) THEN
C      DO 20 C=1,CTYPES
C          IF(CANBIT(C) .LE. 0) GO TO 20
C          CALL CWORK(C)
C          CALL CLOSTV(C)
20      CONTINUE
C
C      ENDIF
C
C      IF(GENFLG.GT.0) THEN
C      DO 30 G=1,GTYPES
C          IF(GENBIT(G) .LE. 0) GO TO 30
C          CALL GWORK(G)
C          CALL GLOSTV(G)
30      CONTINUE
C
C      ENDIF
C
C      RETURN
C      END
C
C *****
C      SUBROUTINE RWORKE(RT)
C *****
C
C      CALCULATES THE VALUE OF THE WORK WEEKS LOST DURING ILLNESS
C      DUE TO EARLY RADIATION INJURIES
C
C      INCLUDE 'CONTROL.FOR'

```

```

C      N = 1
C
C      DO 100 S=1,SEX
C
C      LWMRB = 0.0
C      DO 110 A=1,AC+1
C
C          LWMRB = LWMRB + (RMPYE(A,S,RT) *
X      PV((LVALUE(N,A,S)/52.),R,N) * LWRKRE(RT))
C
C      110  CONTINUE
C
C      RIEIND(RT,S) = LWMRB
C      RIEINS(RT) = RIEINS(RT) + RIEIND(RT,S)
C
C      RIEINM = RIEINM + LWMRB
C
C      100  CONTINUE
C
C      RETURN
C      END
C
C
C      *****
C      SUBROUTINE RWORKL(RT)
C      *****
C
C      CALCULATES THE VALUE OF THE WORK WEEKS LOST DURING ILLNESS
C      DUE TO LATE RADIATION INJURIES
C
C      INCLUDE 'CONTROL.FOR'
C
C      DO 100 S=1,SEX
C
C      IF(RT.EQ.MICROC .OR. RT.EQ.RETARD) GO TO 200
C      DO 110 A=1,AC
C          BT = MAGE(A) + LPR(RT)
C          IBT = IDINT(BT)
C          IB = MIN(IBT,YEARS)
C          IM = IDINT(MAGE(A))
C      DO 110 N=IB,YEARS
C

```

```

      J = N - IM
      RILIND(RT,S) = RILIND(RT,S) + (RMPYL(N,A,S,RT) *
X   PV( (LVALUE(J,A,S)/52.),R,J) * LWRKRL(RT))
C
110   CONTINUE
C
      GO TO 299
C
200   CONTINUE
C
C   PRESUME MEDIAN AGE OF PRENATAL AGE CATEGORY EQUALS ZERO FOR FOLLOWING
C
      A = PRENAT
      BT = MAGE(A) + LPR(RT)
      IBT = IDINT(BT)
      IB = MIN(IBT,YEARS)
      IF (IB.LE.0) IB = 1
      IM = IDINT(MAGE(A))
      DO 210 N=IB,YEARS
      J = N - IM
      RILIND(RT,S) = RILIND(RT,S)+(RMORB(A,S,RT)*LPRBRL(MAGE(A)+
X   LPR(RT),S,N)*(RDISP(RT)/100) * PV(LVALUE(J,A,S),R,J))
210   CONTINUE
C
299   CONTINUE
C
      RILINS(RT) = RILINS(RT) + RILIND(RT,S)
C
      RILINM = RILINM + RILIND(RT,S)
C
100   CONTINUE
C
      DO 300 S=1,SEX
      RILINT(S) = RILINT(S) + RILIND(RT,S)
300   CONTINUE
      RILINT(SEX+1) = RILINM
C
C
      RETURN
      END
C
C
C   *****
C   SUBROUTINE CWORK(C)
C   *****
C
C   CALCULATES THE VALUE OF THE WORK LOST DURING ILLNESS
C   DUE TO CANCER FATALITY AND MORBIDITY
C
      INCLUDE 'CONTROL.FOR'
C

```



```

C      DO 100 S=1,SEX
C          LWMRB = 0.0
C          LWFAT = 0.0
C
C      DO 110 A=1,AC+1
C          BT = MAGE(A) + LPC(C)
C          IBT = IDINT(BT)
C          IB = MIN(IBT,YEARS)
C          IF(IB.LE.0) IB = 1
C          IM = IDINT(MAGE(A))
C      DO 112 N=IB,YEARS
C
C          J = N- IM + 1
C          LWMRB = LWMRB + (MPY(C,A,N,S) *
X          (PV((LVALUE(J,A,S)/52.),R,J) * LWORKC(C)))
112      CONTINUE
C
C          BT = MAGE(A) + LPC(C) + MS(C)
C          IBT = IDINT(BT)
C          IB = MIN(IBT,YEARS)
C          IF(IB.LE.0) IB = 1
C      DO 111 N=IB,YEARS
C          J = N - IM + 1
C          LWFAT = LWFAT + (FPY(C,A,N+1,S) *
X          (PV((LVALUE(J,A,S)/52.),R,J) * LWORKC(C)))
C
111      CONTINUE
C
110      CONTINUE
C
C          CANIND(C,S) = LWMRB + LWFAT
C          CANINC(C) = CANINC(C) + CANIND(C,S)
C
C          CANINM = CANINM + LWMRB
C          CANINF = CANINF + LWFAT
100      CONTINUE
C
C      RETURN
C      END
C
C
C      *****
C      SUBROUTINE GWORK(G)
C      *****
C
C      CALCULATES THE VALUE OF THE WORK LOST DUE TO GENETIC EFFECTS
C
C      INCLUDE 'CONTROL.FOR'
C
C
C

```

```

      RM = DFLOAT(MYEARS)
C
      IBF = (RM-LS(G)-IMY(G))
      IEF = (RM-LS(G))
      IB = IDINT(IBF)
      IE = IDINT(IEF)
      IF(IB.LE.0) IB = 1
      IF(IE.LE.0) IE = 1
C
      DO 110 S=1,SEX
C
      SUMGL = 0.0
      DO 130 J=IB,IE
      RJ = DFLOAT(J)
      SUMGL = SUMGL + (PV(FV((MERN(INCCAT(RJ),S) * LFPR(AGECAT(J),S)
X      * DISP(G)/100),REG,J),R,J))
130  CONTINUE
C
      DO 120 N=1,GYEARS
      GENIND(G,S) = GENIND(G,S) + (GENPY(G,N,S) *
X      PV(FV(SUMGL,REG,N),R,N))
C
120  CONTINUE
C
      GENINS(G) = GENINS(G) + GENIND(G,S)
C
110  CONTINUE
      GENINM = GENINM + GENINS(G)
C
      RETURN
      END
C
C
C *****
C      SUBROUTINE RLOSTV(RT)
C *****
C
C      CALCULATES VALUE OF LABOR LOST DUE TO PREMATURE FATALITY
C      DUE TO AN EARLY RADIATION INJURY
C
C      INCLUDE 'CONTROL.FOR'
C      REAL*8 SUM,SUMR
C
C      DO 10 A=1,AC+1
C      DO 10 S=1,SEX
10  SUML(A,S) = 0.0
C
C      RY = DFLOAT(YEARS)
C      DO 110 A=1,AC+1
C      IM = IDINT(MAGE(A))
C      DO 110 S=1,SEX

```

```

C      RLP = RY - MAGE(A)
      IF(RLP.LE.0.0) RLP = 1.0
      IRLP = IDINT(RLP)
      DO 110 N=1,IRLP
        J = IM + N
        SUML(A,S) = SUML(A,S) + (PV(LVALUE(N,A,S),R,N) *
X      (LPRBRL(MAGE(A),S,J)))
110    CONTINUE
C
      SUMT = 0.0

      SUMR = 0.0
      DO 111 S=1,SEX
        SUM = 0.0
        DO 120 A=1,AC+1
C
          SUM = SUM + (RFPYE(A,S,RT) * SUML(A,S))
C
120    CONTINUE
C
      RIEIND(RT,S) = RIEIND(RT,S) + SUM
      RIEINS(RT) = RIEINS(RT) + SUM
      SUMR = SUMR + SUM
111    CONTINUE
C
      RIEINF = RIEINF + SUMR
      RETURN
      END

C
C
C *****
C      SUBROUTINE CLOSTV(C)
C *****
C      CALCULATES THE LOST LABOR VALUE DUE TO CANCER RELATED
C      PREMATURE MORTALITIES
C
C      INCLUDE 'CONTROL.FOR'
C      REAL*8 SUM,SUMT
C
C
C      SUMT = 0.0
C      RY = DFLOAT(YEARS)
C
C      DO 10 N=1,YEARS
C        DO 10 A=1,AC+1
C        DO 10 S=1,SEX
10    SUMLN(N,A,S) = 0.0
C

```

```

RDIF = LPC(C) + MS(C)
IRD = IDINT(RDIF)
C
DO 110 A=1,AC+1
DO 110 S=1,SEX
RB = MAGE(A) + LPC(C) + MS(C)
IB = IDINT(RB)
IB = MIN(IB,YEARS)
IF (IB.LE.0) IB = 1
C      RLP = RY - MAGE(A)
IM = IDINT(MAGE(A))
C
DO 110 N=IB,YEARS
C
RN = DFLOAT(N)
C
DO 113 J=N-IM,YEARS-IM
IDIS = J + IM
      SUMLN(N,A,S) = SUMLN(N,A,S) + (PV(LVALUE(J,A,S),R,J)
X      * LPRBRL(RN,S,IDIS))
113 CONTINUE
C
110 CONTINUE
C
SUMC = 0.0
DO 111 S=1,SEX
SUM = 0.0
C
DO 120 A=1,AC+1
BT = MAGE(A) + LPC(C) + MS(C)
IBT = IDINT(BT)
IB = MIN(IBT,YEARS)
IF (IB.LE.0) IB = 1
DO 120 N=IB,YEARS
C
SUM = SUM + (FPY(C,A,N,S) * SUMLN(N,A,S))
120 CONTINUE
C
CANIND(C,S) = CANIND(C,S) + SUM
SUMC = SUMC + SUM
111 CONTINUE
C
CANINC(C) = CANINC(C) + SUMC
CANINF = CANINF + SUMC
SUMT = SUMT + SUMC
C
RETURN
END
C
C

```

```

C *****
C SUBROUTINE GLOSTV(G)
C *****
C
C CALCULATE THE VALUE OF THE LABOR LOST DUE TO PREMATURE FATALITY
C DUE TO GENETIC EFFECTS
C
C INCLUDE 'CONTROL.FOR'
C REAL*8 SUM,SUMGT
C
C RM = DFLOAT(MYEARS)
C SUMGT = 0.0
C
C IBF = (RM-LS(G))
C IB = IDINT(IBF)
C IF(IB.LE.0) IB = 1
C IE = MYEARS
C
C DO 110 S=1,SEX
C
C SUM = 0.0
C SUMGL = 0.0
C DO 130 J=IB,IE
C RJ = DFLOAT(J)
C SUMGL = SUMGL + (PV(FV(MERN(INCCAT(RJ),S)*LFPR(AGECAT(J),S),
X REG,J),R,J) * LPRBRL(MAGE(A),S,J))
C
C 130 CONTINUE
C
C DO 120 N=1,GYEARS
C SUM = SUM + (GENPY(G,N,S) * PV(FV(SUMGL,REG,N),R,N))
C 120 CONTINUE
C
C GENIND(G,S) = GENIND(G,S) + SUM
C GENINS(G) = GENINS(G) + SUM
C SUMGT = SUMGT + SUM
C
C 110 CONTINUE
C
C GENINF = GENINF + SUMGT
C
C RETURN
C END
C

```

```

CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C   SUBROUTINE READER
C
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C   SUBROUTINE READER
C
C   THIS SUBROUTINE READS ALL INPUT DATA FOR EXECUTION OF HECOM
C
C   FIRST INCLUDE CONTROL FILE
C       INCLUDE 'CONTROL.FOR'
C-----
C       OPEN THE MAIN INPUT FILE
C-----
C       OPEN (UNIT=2,FILE='HECOM18.DAT',STATUS='OLD')
C-----
C       READ IN DATA
C-----
C       CALL SKIP(2)
C       READ(2,30) CASE
C       CALL SKIP(2)
C       READ(2,*) RHG
C       CALL SKIP(2)
C       READ(2,*) REG
C       CALL SKIP(2)
C       READ(2,*) R
C       CALL SKIP(2)
C       READ(2,*) BASE
C       CALL SKIP(2)
C
C       READ IN SIZE PARAMETERS, INDICES, AND SPECIAL CASES
C
C       READ(2,*) ICCDF
C       CALL SKIP(2)
C       READ(2,*) WRTFLG
C       CALL SKIP(2)
C       READ(2,*) MONFLG
C       CALL SKIP(2)
C       READ(2,*) RADFLG
C       CALL SKIP(2)
C       READ(2,*) CANFLG
C       CALL SKIP(2)
C       READ(2,*) GENFLG
C       CALL SKIP(2)
C
C
C       READ(2,*) AC
C       CALL SKIP(2)
C       READ(2,*) IC
C       CALL SKIP(2)

```

```

      READ(2,*) AINC
      CALL SKIP(2)
      READ(2,*) IINC
      CALL SKIP(2)
      READ(2,*) YEARS
      CALL SKIP(2)
      READ(2,*) SEX
      CALL SKIP(2)
      READ(2,*) CTYPES
      CALL SKIP(2)
      READ(2,*) NER
      CALL SKIP(2)
      READ(2,*) NLR
      CALL SKIP(2)
      READ(2,*) GTYPES
      CALL SKIP(2)
      READ(2,*) GYEARS
      CALL SKIP(2)
      READ(2,*) NGEN
      CALL SKIP(2)
      READ(2,*) MYEARS
      CALL SKIP(2)

```

C

```

      READ(2,*) (MERN(I,1),I=1,IC)
      CALL SKIP(2)
      READ(2,*) (MERN(I,2),I=1,IC)
      CALL SKIP(2)
      READ(2,*) (LFPR(I,1),I=1,AC+1)
      CALL SKIP(2)
      READ(2,*) (LFPR(I,2),I=1,AC+1)
      CALL SKIP(2)
      READ(2,*) (POPFE(I,1),I=1,AC+1)
      CALL SKIP(2)
      READ(2,*) (POPFE(I,2),I=1,AC+1)
      CALL SKIP(2)
      READ(2,*) (MAGE(I),I=1,AC+1)
      CALL SKIP(2)

```

C

C

MONITOR DATA

C

```

      READ(2,*) ASC
      CALL SKIP(2)
      READ(2,*) BSC
      CALL SKIP(2)
      READ(2,*) ISC
      CALL SKIP(2)
      READ(2,*) MSC
      CALL SKIP(2)
      READ(2,*) LTC
      CALL SKIP(2)
      READ(2,*) ATC

```

```

      CALL SKIP(2)
      READ(2,*) MVC
      CALL SKIP(2)
      READ(2,*) DCC
      CALL SKIP(2)
      READ(2,*) DCD
      CALL SKIP(2)
      READ(2,*) RHC
      CALL SKIP(2)
      READ(2,*) RHD
      CALL SKIP(2)
      READ(2,*) NHC
      CALL SKIP(2)
      READ(2,*) NHD
      CALL SKIP(2)
C-----
C   READ IN RADIATION INJURY DATA
C-----
      DO 1300 I=1,NER
      READ(2,20) RENAME(I)
1300 CONTINUE
      CALL SKIP(2)
C
      READ(2,*) (RDEBIT(I),I=1,NER)
      CALL SKIP(2)
      READ(2,*) (CPRADE(I),I=1,NER)
      CALL SKIP(2)
      READ(2,*) (LWRKRE(I),I=1,NER)
      CALL SKIP(2)
C
C   READ IN LATE RADIATION INJURIES DATA
C
      DO 1302 I=1,NLR
      READ(2,20) RLNAME(I)
1302 CONTINUE
      CALL SKIP(2)
C
      READ(2,*) (RDLBIT(I),I=1,NLR)
      CALL SKIP(2)
      READ(2,*) (LPR(I),I=1,NLR)
      CALL SKIP(2)
      READ(2,*) (CPRADL(I),I=1,NLR)
      CALL SKIP(2)
      READ(2,*) (LWRKRL(I),I=1,NLR)
C
      CALL SKIP(2)
      READ(2,*) (RDISP(I),I=1,NLR)
      CALL SKIP(2)
      READ(2,*) (LRADCT(I),I=1,NLR)
      CALL SKIP(2)
C

```



```

C   READ IN CANCER DATA
C
C-----
C   READ IN CANCER TYPE NAMES
C-----
      DO 1000 I=1,CTYPES
        READ(2,20) CNAME(I)
1000 CONTINUE
C
      CALL SKIP(2)
      READ(2,*) (CANBIT(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (INR(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (LPC(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (MS(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (POR(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (PPDR(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (CPCF(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (CPCS(I),I=1,CTYPES)
      CALL SKIP(2)
      READ(2,*) (LWORKC(I),I=1,CTYPES)
      CALL SKIP(2)
C
C-----
C   READ IN GENETIC EFFECTS DATA
C-----
      DO 1400 I=1,GTYPES
        READ(2,20) GNAME(I)
1400 CONTINUE
C
      CALL SKIP(2)
      READ(2,*) (GENBIT(I),I=1,GTYPES)
      CALL SKIP(2)
      DO 3000 G=1,GTYPES
        READ(2,*) (GSRATE(G,I),I=1,SEX)
3000 CONTINUE
      CALL SKIP(2)
      READ(2,*) (ERATE(I),I=1,GTYPES)
      CALL SKIP(2)
      READ(2,*) (LS(I),I=1,GTYPES)
      CALL SKIP(2)
      READ(2,*) (IMY(I),I=1,GTYPES)
      CALL SKIP(2)
      READ(2,*) (DISP(I),I=1,GTYPES)
      CALL SKIP(2)

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```

      READ(2,*) (MEDCST(I),I=1,GTYPES)
      CALL SKIP(2)
      READ(2,*) (INCCOST(I),I=1,GTYPES)
C-----
C   CLOSE FILE
C-----
      CLOSE (UNIT=2)
C-----
C   OPEN FILE CONTAINING DEATH DISTRIBUTION DATA AND READ IT IN
C-----
      OPEN (UNIT=3,FILE='DIST.DAT',STATUS='OLD')
CC
      DO 2000 I=1,YEARS
      READ(3,*) (LIFEP(I,J),J=1,SEX)
2000 CONTINUE
C-----
C   CLOSE FILE
C-----
      CLOSE (UNIT=3)
C-----
C   OPEN FILE CONTAINING SPONTANEOUS MORBIDITY AND FATALITY RATES
C-----
      OPEN(10,FILE='SPONT18.DAT',STATUS='OLD')
C-----
C   READ SPONTANEOUS MORBIDITIES AND FATALITIES
C-----
      CALL SKIP(10)
      DO 4000 J=1,CTYPES
4000   READ(10,*) (SPONMR(I,J),I=1,AC)
      CALL SKIP(10)
      DO 4010 J=1,CTYPES
4010   READ(10,*) (SPONFR(I,J),I=1,AC)
C
C-----
C   CLOSE FILE
C-----
      CLOSE(UNIT=10)
C
20   FORMAT(A20)
30   FORMAT(A50)
      RETURN
      END
C
C
C *****
C   SUBROUTINE READSN
C *****
C   READ IN THE FATALITY FOR EACH CASE FROM THE FILE HECOMVAR.DAT
C

```

```

      INCLUDE 'CONTROL.FOR'
C
C   OPEN THE DATA FILE HECOMVAR.DAT
C
      OPEN(3,FILE='HECOMVAR.DAT',STATUS='OLD')
C   READ IN THE POPULATION IN SECTOR 90 DEGREES DOWNWIND
      READ(3,*,END=99) PSD
C
C   READ IN THE POPULATION EVACUATED
      READ(3,*,END=99) PE
C
C   READ IN THE POPULATION EXPOSED TO OVER 1 GY
      READ(3,*,END=99) PSE
C
C   READ IN THE POPULATION EXPOSED TO OVER 2 GY
      READ(3,*,END=99) PHE
C
C
C   NON-FATAL RADIATION INJURIES FOR EACH TYPE OF EARLY RADIATION INJURY
      READ(3,*,END=99) (RADIE(I),I=1,NER)
C
C   FATAL RADIATION INJURIES FOR EACH EARLY RADIATION INJURY TYPE
      READ(3,*,END=99) (RADFE(I),I=1,NER)
C
C   FATAL RADIATION INJURIES FOR EACH TYPE FOR PRENATAL AGE CATEGORY
      READ(3,*,END=99) (RADPFE(I),I=1,NER)
C
C   NON-FATAL INJURIES FOR EACH TYPE OF LATE RADIATION INJURIES
      READ(3,*,END=99) (RADLI(I),I=1,NLR)
C
C   RADIATION INJURIES FOR EACH LATE RADIATION INJURY TYPE FOR THE
      PRENATAL AGE CATEGORY
      READ(3,*,END=99) (RADLPI(I),I=1,NLR)
CC
CC
C   NUMBER OF FATALITIES FOR EACH CANCER TYPE
      READ(3,*,END=99) (CF(I),I=1,CTYPES)
C
C   NUMBER OF PRENATAL CANCER FATALITIES FOR EACH CANCER TYPE
      READ(3,*,END=99) (CFP(I),I=1,CTYPES)
C
C   NUMBER OF NON-FATAL CANCERS FOR EACH CANCER TYPE
      READ(3,*,END=99) (CNONF(I),I=1,CTYPES)
CC
CC
C   GENETIC EFFECT INCIDENCE FOR EACH TYPE OF GENETIC EFFECT
      READ(3,*,END=99) (GENI(I),I=1,GTYPES)
C
      GO TO 100
99   TERMIN = 1
100  CONTINUE

```

```

C      RETURN
C      END

C      *****
C      SUBROUTINE INIT
C      *****
C      INITIALIZE THE VARIABLES USED TO CALCULATE SUMS OR FINAL PRICES
C      TO ZERO, SET CALCULATED VARIABLES USED LATER.
C
C      INCLUDE 'CONTROL.FOR'
C      REAL*8 ZERO
C
C      SET SPECIAL CASE INDICES
C
C      PRENAT = AC + 1
C      MALES = 1
C      BREAST = 3
C      LUNG = 2
C      THYRID = 8
C      THYBEN = 9
C      PULMON = 4
C      MICROC = 3
C      RETARD = 4
C
C      CALCULATE BEGINNING AGE OF EACH AGE CATEGORY
C
C      DO 60 I=1,AC
60      ACAGE(I) = AINC * (I-1)
C      ACAGE(PRENAT) = 0.0
C
C      CALCULATE BEGINNING AGE OF INCOME CATEGORIES
C
C      DO 70 I=1,IC
70      ICAGE(I) = IINC * (I-1)
C
C      SET FEMALES FLAG
C
C      IF(SEX.NE.2) GO TO 80
C      IF(MALES.EQ.1) THEN
C          FEMALE = 2
C      ELSE
C          FEMALE = 1
C      ENDIF
80      CONTINUE
C
C      ZERO = 0.000

```

```

C
C      ZERO OUT CANCER VARIABLES
C
      CANINM = ZERO
      CANINF = ZERO
      CCSTMR = ZERO
      CCSTFT = ZERO
C
      DO 101 S=1,SEX+1
        CANINT(S) = ZERO
        TCANC(S) = ZERO
101    CONTINUE
C
      DO 100 C=1,CTYPES
        CANINC(C) = ZERO
        TOTCC(C) = ZERO
C
      DO 100 S=1,SEX
        CANIND(C,S) = ZERO
        CCOSTS(C,S) = ZERO
C
      DO 100 A=1,AC+1
        FAT(C,A,S) = ZERO
        MORR(C,A,S) = ZERO
        SUMDTH(C,A,S) = ZERO
        SUMMRB(C,A,S) = ZERO
100    CONTINUE
C
C      ZERO OUT RADIATION INJURY VARIABLES
C
      RIEINM = ZERO
      RIEINF = ZERO
      RILINM = ZERO
      RECSTM = ZERO
      RECSTF = ZERO
      RLCSTM = ZERO
      RTCSTM = ZERO
      RTCSTF = ZERO
      RITINM = ZERO
C
      DO 201 S=1,SEX+1
        RIEINT(S) = ZERO
        RILINT(S) = ZERO
        TTRADC(S) = ZERO
        TERADC(S) = ZERO
        TLRADC(S) = ZERO
        RITINT(S) = ZERO
201    CONTINUE
C
      DO 200 RT=1,NER
        RIEINS(RT) = ZERO

```

```

      TOTREC(RT) = ZERO
      DO 200 S=1,SEX
        RIEIND(RT,S) = ZERO
        RECOST(RT,S) = ZERO
      DO 200 A=1,AC+1
        RMPYE(A,S,RT) = ZERO
        RFPYE(A,S,RT) = ZERO
200    CONTINUE
C
      DO 210 RT=1,NLR
        RILINS(RT) = ZERO
        TOTRLC(RT) = ZERO
      DO 210 S=1,SEX
        RILIND(RT,S) = ZERO
        RLCOST(RT,S) = ZERO
      DO 210 A=1,AC+1
        RMORB(A,S,RT) = ZERO
210    CONTINUE
C
C
C    ZERO OUT GENETIC EFFECTS VARIABLES
C
      GENINF = ZERO
      GENINM = ZERO
      GMCOST = ZERO
C
      DO 301 S=1,SEX+1
        GENINT(S) = ZERO
        TGENC(S) = ZERO
301    CONTINUE
C
      DO 300 G=1,GTYPES
        GENINS(G) = ZERO
        GTCOST(G) = ZERO
      DO 300 S=1,SEX
        GENIND(G,S) = ZERO
        GCOST(G,S) = ZERO
      DO 310 N=1,GYEARS
        GENPY(G,N,S) = ZERO
310    CONTINUE
      DO 320 N=1,NGEN
        GENPG(G,N,S) = ZERO
320    CONTINUE
C
300    CONTINUE
C
      RETURN
      END
C
C

```

```

C *****
C SUBROUTINE SUMTOT
C *****
C SUM FOR THE TOTAL INOIRECT COSTS AND OVER ALL THE COST
C CATEGORIES TO GET TOTALS BY CATEGORY AND THE OVERALL TOTAL
C COST.
C INCLUDE 'CONTROL.FOR'
C
C INDIRECT COSTS
C DO 100 C=1,CTYPES
C DO 100 S=1,SEX
100 CANINT(S) = CANINT(S) + CANIND(C,S)
CANINT(SEX+1) = CANINF + CANINM
C DO 200 RT=1,NER
C DO 200 S=1,SEX
200 RIEINT(S) = RIEINT(S) + RIEIND(C,S)
RIEINT(SEX+1) = RIEINM + RIEINF
C DO 300 G=1,GTYPES
C DO 300 S=1,SEX
300 GENINT(S) = GENINT(S) + GENIND(G,S)
GENINT(SEX+1) = GENINM + GENINF
C SUM LATE AND EARLY RAOIATION INJURY COSTS
C DO 400 S=1,SEX+1
C TTRADC(S) = TERADC(S) + TLRADC(S)
C RITINT(S) = RIEINT(S) + RILINT(S)
400 CONTINUE
C RTCSTM = RECSTM + RLCSTM
C RTCSTF = RECSTF
C RITINM = RIEINM + RILINM
C
C TOTAL DIRECT COSTS
C TOTDRM = RECSTM + RLCSTM + CCSTM + GMCOST
C TOTDRF = RECSTF + CCSTF
C TOTDIR = TOTDRM + TOTDRF + MGTOST
C
C TOTAL INDIRECT COSTS
C TOTINM = RIEINM + RILINM + CANINM + GENINM
C TOTINF = RIEINF + CANINF + GENINF

```

```

      TOTIND = TOTINM + TOTINF
C
C   TOTAL COSTS FOR EACH CATEGORY AND THE TOTAL
C
      TOTRAD = TERADC(SEX+1) + TLRADC(SEX+1) + RIEINT(SEX+1)
1      + RILINT(SEX+1)
      TOTCAN = TCANC(SEX+1) + CANINT(SEX+1)
      TOTGEN = TGENC(SEX+1) + GENINT(SEX+1)
      TOTAL = MGTCST + TOTRAD + TOTCAN + TOTGEN
C
      RETURN
      END
C
C *****
C   SUBROUTINE READCD
C *****
C   READ THE CCDF DATA FROM PREVIOUS RUNS INTO THE ARRAYS CCDDAT FOR THE
C   DATA, AND INTO CCDNAM FOR THE NAMES. INVAR = NUMBER OF VARIABLES.
C   INC = THE NUMBER OF CASES.
C   IF ICCDF LESS THAN ZERO, THIS IS PRESUMED TO BE THE FIRST CASE AND
C   ANY EXISTING DATA IN THE FILE IS THROWN OUT.
C   IF ICCDF IS GREATER THAN ZERO, THE DATA FOR THIS RUN WILL BE APPENDED
C   ONTO THE EXISTING FILE BY SUBROUTINES SETCCD AND WRITCD.
C
      INCLUDE 'CONTROL.FOR'
C
      OPEN(9,FILE='CCDFIN.DAT',STATUS='OLD',RECL=132)
C
      READ(9,*) INC
      IF(ICCDF.LT.0) INC = 0
      READ(9,*) INVAR
C
      DO 10 I=1,INVAR
        READ(9,100) CDNAM(I),(CCDDAT(I,J),J=1,INC)
10      CONTINUE
100     FORMAT(1X,A,1X,23(9(1PE12.4,1X)/9X))
C
      CLOSE(UNIT=9)
C
      RETURN
      END
C
C *****
C   SUBROUTINE SETCCD
C *****
C   SETS UP THE VALUES GENERATED BY THIS RUN INTO THE ARRAY FOR
C   CCDF EVALUATION LATER
C   MAX NUMBER OF VARIABLES (INVAR) SET AT 40, WITH 40 BEING USED

```



```

C      AT PRESENT.
C      MAX NUMBER OF OBSERVATIONS SET AT 200.
C
C      INCLUDE 'CONTROL.FOR'
C
C      INC = INC + 1
C
C      CCDDAT(1,INC) = MGT CST
C      CCDDAT(2,INC) = TTRADC(1)
C      CCDDAT(3,INC) = TTRADC(2)
C      CCDDAT(4,INC) = TTRADC(3)
C      CCDDAT(5,INC) = RITINT(1)
C      CCDDAT(6,INC) = RITINT(2)
C      CCDDAT(7,INC) = RITINT(3)
C      CCDDAT(8,INC) = TCANC(1)
C      CCDDAT(9,INC) = TCANC(2)
C      CCDDAT(10,INC) = TCANC(3)
C      CCDDAT(11,INC) = CANINT(1)
C      CCDDAT(12,INC) = CANINT(2)
C      CCDDAT(13,INC) = CANINT(3)
C      CCDDAT(14,INC) = TGENC(1)
C      CCDDAT(15,INC) = TGENC(2)
C      CCDDAT(16,INC) = TGENC(3)
C      CCDDAT(17,INC) = GENINT(1)
C      CCDDAT(18,INC) = GENINT(2)
C      CCDDAT(19,INC) = GENINT(3)
C      CCDDAT(20,INC) = RTCSTM
C      CCDDAT(21,INC) = RTCSTF
C      CCDDAT(22,INC) = CCSTMR
C      CCDDAT(23,INC) = CCSTFT
C      CCDDAT(24,INC) = GMCOST
C      CCDDAT(25,INC) = TOTDRM
C      CCDDAT(26,INC) = TOTDRF
C      CCDDAT(27,INC) = TOTDIR
C      CCDDAT(28,INC) = RITINM
C      CCDDAT(29,INC) = RIEINF
C      CCDDAT(30,INC) = CANINM
C      CCDDAT(31,INC) = CANINF
C      CCDDAT(32,INC) = GENINM
C      CCDDAT(33,INC) = GENINF
C      CCDDAT(34,INC) = TOTINM
C      CCDDAT(35,INC) = TOTINF
C      CCDDAT(36,INC) = TOTIND
C      CCDDAT(37,INC) = TOTRAD
C      CCDDAT(38,INC) = TOTCAN
C      CCDDAT(39,INC) = TOTGEN
C      CCDDAT(40,INC) = TOTAL
C
C      RETURN

```

```

      END
C
C *****
C      SUBROUTINE WRITCD
C *****
C      WRITE THE CCDF DATA BACK TO THE CCDF DATA FILE.
C      IF ICCDF GREATER THAN ZERO, DATA FROM PREVIOUS RUNS WAS INCLUDED.
C      IF ICCDF LESS THAN ZERO, ONLY DATA FROM THIS RUN WAS SAVED TO BE
C      WRITTEN.
C
C      INCLUDE 'CONTROL.FOR'
C
C      OPEN(8,FILE='CCDFIN.DAT',STATUS='NEW',RECL=132)
C
C      WRITE(8,*) INC
C      WRITE(8,*) INVAR
C
C      DO 20 I=1,INVAR
C          WRITE(8,100) CONAM(I),(CCDDAT(I,J),J=1,INC)
20      CONTINUE
100     FORMAT(1X,A,1X,23(9(1PE12.4,1X)/9X))
C
C      CLOSE THE FILE
C
C      CLOSE(UNIT=8)
C
C      RETURN
C      END
C
C *****
C      SUBROUTINE WRITER
C *****
C      WRITES OUT THE FINAL PRICES CALCULATED BY HECOM
C
C      INCLUDE 'CONTROL.FOR'
C      CHARACTER*11 NOTA
C
C      OPEN(12,FILE='HECOM.OUT',RECL=80,STATUS='NEW',
X      CARRIAGECONTROL='FORTRAN')
C
C
C      NOTA = '      NA      '
C
C      IF(NCAS.EQ.1) THEN
C          WRITE(12,5) CASE
C
C          WRITE(12,101) 'COST PROJECTION PARAMETERS '
C          WRITE(12,99)

```

```

        WRITE(12,110) 'RATE OF REAL EARNINGS GROWTH .....',REG
        WRITE(12,110) 'RATE OF REAL HEALTH COST GROWTH ...',RHG
        WRITE(12,110) 'REAL DISCOUNT RATE .....',R
        WRITE(12,110) 'BASE YEAR .....',BASE
    ELSE
        WRITE(12,6) NCAS
    ENDIF
C
    IF(MONFLG.LE.0) GO TO 501
    WRITE(12,100) 'MEDICAL MANAGEMENT COSTS'
    WRITE(12,99)
    WRITE(12,120) 'RADIATION SCREENING .....',RADGST
    WRITE(12,120) 'MEDICAL MONITORING .....',RADMNC
    WRITE(12,120) 'MEDICAL TRANSPORTATION .....',TRNCST
    WRITE(12,99)
    WRITE(12,120) '          TOTAL COSTS          ',MGTCST
C
501  IF(RADFLG .LE. 0) GO TO 502
C
    WRITE(12,100) 'RADIATION INJURY COSTS'
    WRITE(12,201)
    WRITE(12,140)
    DO 10 RT=1,NER
10    WRITE(12,145) RENAME(RT),(RECAST(RT,S),S=1,SEX),TOTREC(RT)
    DO 11 RT=1,NLR
11    WRITE(12,145) RLNAME(RT),(RLCOST(RT,S),S=1,SEX),TOTRLC(RT)
C
    WRITE(12,99)
    WRITE(12,145) '          TOTAL          ',(TTRADC(S),S=1,SEX+1)
C
    WRITE(12,99)
    WRITE(12,202)
    WRITE(12,140)
    DO 20 RT=1,NER
20    WRITE(12,145) RENAME(RT),(RIEIND(RT,S),S=1,SEX),RIEINS(RT)
C
    DO 21 RT=1,NLR
21    WRITE(12,145) RLNAME(RT),(RILIND(RT,S),S=1,SEX),RILINS(RT)
C
    WRITE(12,99)
    WRITE(12,145) '          TOTAL          ',(RITINT(S),S=1,SEX+1)
C
C
502  IF(CANFLG .LE. 0) GO TO 503
    WRITE(12,100) 'CANCER COSTS'
    WRITE(12,201)
    WRITE(12,150)
C
    DO 14 C=1,CTYPES
14    WRITE(12,145) CNAME(C),(CCOSTS(C,S),S=1,SEX),TOTCC(C)
C

```

```

        WRITE(12,99)
        WRITE(12,145) '          TOTAL          ',(TCANC(S),S=1,SEX+1)
C
        WRITE(12,99)
        WRITE(12,202)
        WRITE(12,150)
        DO 15 C=1,CTYPES
15      WRITE(12,145) CNAME(C),(CANIND(C,S),S=1,SEX),CANINC(C)
C
        WRITE(12,99)
        WRITE(12,145) '          TOTAL          ',(CANINT(S),S=1,SEX+1)
C
C
503      IF(GENFLG.LE.0) GO TO 504
        WRITE(12,100) 'GENETIC EFFECT COSTS'
C
        WRITE(12,201)
        WRITE(12,160)
C
        DO 17 G=1,GTYPES
17      WRITE(12,145) GNAME(G),(GCOST(G,S),S=1,SEX),GTCOST(G)
C
        WRITE(12,99)
        WRITE(12,145) '          TOTAL          ',(TGENC(S),S=1,SEX+1)
C
        WRITE(12,99)
        WRITE(12,202)
        WRITE(12,160)
C
        DO 18 G=1,GTYPES
18      WRITE(12,145) GNAME(G),(GENIND(G,S),S=1,SEX),GENINS(G)
C
        WRITE(12,99)
        WRITE(12,145) '          TOTAL          ',(GENINT(S),S=1,SEX+1)
C
C
504      WRITE(12,100) 'DIRECT COST SUMMARY'
        WRITE(12,301)
C
        IF(MONFLG.GT.0)WRITE(12,347) 'MEDICAL MANAGEMENT COSTS ..',
X      NOTA,NOTA,MGTCST
        IF(RADFLG.GT.0) WRITE(12,345) 'RADIATION INJURY COSTS ...',
X      RTCSTM,RTCSTF,TTRADC(SEX+1)
        IF(CANFLG.GT.0) WRITE(12,345) 'CANCER COSTS .....',
X      CCSTMR,CCSTFT,TCANC(SEX+1)
        IF(GENFLG.GT.0) WRITE(12,346) 'GENETIC EFFECT COSTS .....',
X      GMCOST,NOTA,TGENC(SEX+1)
        WRITE(12,99)
        WRITE(12,345) '          TOTAL          ',TOTDRM,TOTDRF,
X      TOTDIR
C

```

```

C      WRITE(12,101) 'INDIRECT COST SUMMARY'
      WRITE(12,301)
C
      IF(RADFLG.GT.0)WRITE(12,345)'RADIATION INJURY COSTS ...',
X      RITINM,RIEINF,RITINT(SEX+1)
      IF(CANFLG.GT.0)WRITE(12,345)'CANCER COSTS .....',
X      CANINM,CANINF,CANINT(SEX+1)
      IF(GENFLG.GT.0)WRITE(12,345)'GENETIC EFFECT COSTS .....',
X      GENINM,GENINF,GENINT(SEX+1)
C
      WRITE(12,99)
      WRITE(12,345) '      TOTAL              ',TOTINM,TOTINF,
X      TOTIND
CC
C      WRITE(12,99)
      WRITE(12,101) 'TOTAL COSTS'
      WRITE(12,99)
C
      IF(MONFLG.GT.0) WRITE(12,445) 'MEDICAL MANAGEMENT COSTS ',
X      MGTCSST
      IF(RADFLG.GT.0) WRITE(12,445) 'RADIATION INJURY COSTS ',
X      TOTRAD
      IF(CANFLG.GT.0) WRITE(12,445) 'CANCER COSTS ',
X      TOTCAN
      IF(GENFLG.GT.0) WRITE(12,445) 'GENETIC EFFECT COSTS ',
X      TOTGEN
C
      WRITE(12,445) '      TOTAL              ',TOTAL
C
C
C      FORMAT STATEMENTS
C
      5      FORMAT('1',15(/),29X,'HEALTH EFFECTS COST MODEL',//,
X      1X,28X,'U.S. NUCLEAR REGULATORY COMMISSION',////,
X      1X,28X,A50)
      6      FORMAT('1',10X,'CASE NUMBER = ',13)
C
      100     FORMAT('1',5(/),5X,A)
      101     FORMAT(7(/),5X,A)
C
      110     FORMAT(/,10X,A35,F5.2)
      120     FORMAT(/,15X,A30,1PE11.4)
      99      FORMAT(/)
C
      201     FORMAT(/,8X,'DIRECT COSTS : ')
      202     FORMAT(/,8X,'INDIRECT COSTS : ')
C
      140     FORMAT(/,17X,'INJURY TYPE',12X,'MALE',8X,'FEMALE',9X,'TOTAL')
      145     FORMAT(12X,A20,4X,2(1PE11.4,2X),2X,1PE11.4)

```

```

150  FORMAT(/,17X,'CANCER TYPE',12X,'MALE',8X,'FEMALE',9X,'TOTAL')
160  FORMAT(/,10X,'GENETIC EFFECT TYPE',12X,'MALE',8X,'FEMALE',
X    9X,'TOTAL')
C
301  FORMAT(/,42X,'MORBIDITY',4X,'MORTALITY',7X,'TOTAL',/)
345  FORMAT(12X,A26,2X,2(1PE11.4,2X),2X,1PE11.4)
346  FORMAT(12X,A26,2X,1PE11.4,2X,A11,4X,1PE11.4)
347  FORMAT(12X,A26,2X,2(A11,2X),2X,1PE11.4)
C
445  FORMAT(12X,A26,7X,1PE11.4)
C
      RETURN
      END

```

```

CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
    FUNCTION LVALUE(N,A,S)
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
    INCLUDE 'CONTROL.FOR'
    INTEGER B
C
C-----
C    DETERMINE LABOR VALUE CATEGORY OF SOMEONE WHO IS N+MAGE YEARS OLD
C-----
    RN = DFLOAT(N)
    ACT = RN + MAGE(A)
    B=INCCAT(ACT)
    IAC = IDINT(ACT)
    IA = AGECAT(IAC)
C-----
C    CALCULATE LABOR VALUE IN YEAR N
C-----
    LVALUE = FV(MERN(B,S)*LFPR(IA,S),REG,N)
    RETURN
    END
C
C *****
C    FUNCTION LPRBRL(RIA,S,N)
C *****
C
C    FINDS THE PROBABILITY OF A PERSON RIA YEARS OLD SURVIVING TO BE
C    N YEARS OLD.
C
    INCLUDE 'CONTROL.FOR'
    REAL*8 PROD,RIA
C
    IB = IDINT(RIA)
    IF(IB.GE.YEARS .OR. IB.GE.N) THEN
        PROD = 0.0
        GO TO 11
    ENDIF
    PROD = LIFEP(IB+1,S)
C
    DO 10 I=IB+2,N
        PROD = PROD * LIFEP(I,S)
    10 CONTINUE
C
    11 CONTINUE
        LPRBRL = PROD
C
    RETURN
    END
C

```



```

C
C      K=0
C-----
C      CHECK FIRST TO SEE IF IINC EQUALS ZERO
C-----
C      IF (IINC.NE.0) GOTO 1000
C      K=1
C      GOTO 5000
C-----
C      AGE CATEGORY EQUALS 18. CHECK FIRST FOR IN UTERO.
C-----
1000 IF (AGEI.GT.0.0) GOTO 2000
C      K=1
C      GO TO 5000
C-----
C      NOW STEP THROUGH EACH YEAR. K WILL COUNT INCOME CATEGORY OF AGE.
C-----
2000 CONTINUE
C      DO 3000 I=1,IC
C          K=K+1
C-----
C      CHECK TO SEE IF THIS IS IN UTERO AGE CATEGORY IF IT IS
C      THEN ASSIGN IT THE PROPER AGE CATEGORY AS SOON AS ITS AGE
C      REACHES THE MINIMUM BOUNDARY FOR A CATEGORY. IF IT IS NOT
C      IN UTERO WAIT UNTIL AGE IS ABOVE MEDIAN AGE FOR A CATEGORY.
C-----
C      IF(AGEI.LT.ICAGE(I+1)) GO TO 5000
3000 CONTINUE
5000 INCCAT=K
C      RETURN
C      END
CC
C
C *****
C      FUNCTION AGECAT(N)
C
C      INCLUDE 'CONTROL.FOR'
C
C      K = 0
C
C      IF(AINC.NE.0) GO TO 100
C      K=1
C      GO TO 500
C
100  IF(N.NE.0) GO TO 200
C      K = PRENAT
C      GO TO 500
C
200  CONTINUE
C      DO 300 I=1,AC

```

```

        K = K + 1
        IF(N.LT.ACAGE(I+1)) GO TO 500
300    CONTINUE
C
500    CONTINUE
C
        AGECAT = K
C
        RETURN
        END
C
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
    FUNCTION FV(P,R,Y)
    REAL*8 P,R,BASE,EXN,RY
    INTEGER Y
        COMMON/DISCFN/BASE
        RY = DFLOAT(Y)
        EXN = RY + BASE - 1.0
        FV=P*(1.0+R/100.)**(EXN)
        RETURN
        END
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
    FUNCTION PV(P,R,N)
    REAL*8 P,R,BASE,EXN,RY
    INTEGER N
        COMMON/DISCFN/BASE
        RY = DFLOAT(N)
        EXN = RY + BASE - 1.0
        PV=P/( (1.0+R/100.)**(EXN) )
        RETURN
        END

```

CCDF.FOR

```

C      MAIN PROGRAM FOR CCDF GENERATION
C
C
C
C      CHARACTER*1 A
C      CHARACTER*8 VNAME(40)
C      REAL PROBAB(40,75),VECT(40,200),VTEM(200),PRT(75),TABLE(41,70)
C      INTEGER INVEC(40),ZERO(40)
C
C      OPEN(9,FILE='CCDFIN.DAT',STATUS='OLD')
C
C      READ(9,*) INC
C      READ(9,*) INVAR
C      DO 10 I=1,INVAR
C          READ(9,100) VNAME(I),(VECT(I,J),J=1,INC)
10      CONTINUE
100     FORMAT(1X,A,1X,23(9(E12.4,1X)/9X))
C
C      OPEN(10,FILE='CCDFLG.DAT',STATUS='OLD')
C
C      DO 22 J=1,4
22     READ(10,101) A
C
101    FORMAT(A1)
C
C      DO 20 I=1,INVAR
C      DO 21 J=1,4
21     READ(10,101) A
C
C      READ(10,*) INVEC(I)
20     CONTINUE
C
C
C      DO 31 I=1,INVAR
31     ZERO(I) = 0
C
C      INUM = 0
C      DO 11 I=1,INVAR
C      IF(INVEC(I) .NE. 1) GO TO 11
C      INUM = INUM + 1
C      DO 12 J=1,INC
12     VTEM(J) = VECT(I,J)
C
C      CALL CALCDC(INC,VTEM,PRT,ZERO(INUM))
C
C      DO 13 J=1,75
13     PROBAB(INUM,J) = PRT(J)
11     CONTINUE
C

```

```

      CALL TABCCD(PROBAB,INUM,TABLE,NET,ZERO)
      CALL TABWT(TABLE,NET,VNAM,INUM,INVAR,INVEC)
      STOP 'CCDF COMPLETE'
      END
C
C *****
C      SUBROUTINE CALCDC(NVC,VECT,PROB,Z)
C
C      REAL VECT(200),PROB(75),TVAL,PRNUM(75)
C      INTEGER MAGI(5),Z
C      DATA MAGI /1,2,3,5,7/
C
C      NMA = 5
C      Z = 0
C      IALLZ = 0
C
C      TMIN = 1E+15
C      TMAX = 1E-15
C      RMIN = TMIN
C      RMAX = TMAX
C
C      DO 11 I=1,NVC
C         IF(VECT(I).LT. rmin .AND. VECT(I).NE. 0.0) rmin = VECT(I)
C         IF(VECT(I).GT.rmax .AND. VECT(I).NE. 0.0) rmax = VECT(I)
11      CONTINUE
C
C      IF(RMAX .GE. 1.0) THEN
C         MAXEXP = ALOG10(RMAX)
C      ELSE IF(RMAX.EQ.TMAX) THEN
C         MAXEXP = 0
C         IALLZ = 1
C         NEI = 0
C         GO TO 99
C      ELSE
C         MAXEXP = NINT( ALOG10(rmax) - 0.4999999)
C      ENDIF
C      IF(RMIN .GE. 1.0) THEN
C         MINEXP = ALOG10(RMIN)
C      ELSE IF(RMIN.EQ.TMIN) THEN
C         MINEXP = 1
C      ELSE
C         MINEXP = NINT(ALOG1D(rmin) - .4999999)
C      ENDIF
C
C      NGR = MAXEXP - MINEXP + 1
C      NEI = NGR * NMA
C
C      DO 15 I=1,75
C         PRNUM(I) = 0.0
15      PROB(I) = 0.0
C

```

```

DO 20 K=1,NVC
  IF(VECT(K) .EQ. 0) THEN
    Z = 1
    GO TO 20
  ENDIF
DO 10 I=1,NGR
DO 10 J=1,NMA
  IND = (I-1)*NMA + J
  TVAL = FLOAT(MAGI(J)) * (10.0**((MINEXP-1)+I))
  IF(TVAL.GT.VECT(K)) GO TO 20
  PRNUM(IND) = PRNUM(IND) + 1
10 CONTINUE
20 CONTINUE
C
99 CONTINUE
C
  PROB(1) = NEI
  DO 25 I=2,NEI+1
25  PROB(I) = PRNUM(I-1) / NVC
C
  IF(IALLZ .EQ. 1) Z = -1
  PROB(NEI+2) = RMAX
  PROB(NEI+3) = RMIN
  PROB(NEI+4) = MAXEXP
  PROB(NEI+5) = MINEXP
  RETURN
  END
C
C *****
C   SUBROUTINE TABCCD(PROBAB,IFCCO,TABLE,NEI,ZERO)
C
C   TO PUT THE CALCULATED PROBABILITIES INTO AN ARRAY FOR PRINTING
C
C   REAL PROBAB(40,75) , TABLE(41,70)
C   INTEGER NEL(40),MAGI(5),MAXEXP(40),MINEXP(40),INDV(40),ZERO(40)
C   DATA MAGI /1,2,3,5,7/
C
C   NMA = 5
C   ITNM = 15
C   MINT = ITNM
C   TMAX = 1E-15
C   MAXT = - ITNM
C
C   DO 11 I=1,IFCCD
C   NEL(I) = NINT(PROBAB(I,1))
C   IF(ZERO(I) .LT. 0) THEN
C     MINEXP(I) = MINT
C     GO TO 11
C   ENDIF
C
C   MAXEXP(I) = NINT(PROBAB(I,NEL(I)+4))

```

```

MINEXP(I) = NINT(PROBAB(I,NEL(I)+5))
IF(MAXEXP(I) .GT. MAXT) MAXT = MAXEXP(I)
IF(MINEXP(I) .LT. MINT) MINT = MINEXP(I)
11  CONTINUE
C
    NGR = MAXT - MINT + 1
    NEI = NGR * NMA
C
    DO 13 K=1,IFCCD
    DO 13 I=1,NGR
    DO 13 J=1,NMA
        IND = (I-1)*NMA + J
        TABLE(1,IND) = FLOAT(MAGI(J)) * (10.0**((MINT-1) + I))
13    IF(MINEXP(K) .EQ. (MINT-1+I)) INDV(K)= (I-1)*NMA+1
C
    DO 14 I=1,IFCCD
C
    DO 16 J=2,NEL(I) + 1
16    TABLE(I+1,INDV(I)+J-2) = PROBAR(I,J)
C
    DO 17 J=INDV(I)+NEL(I)+1 , NEI
17    TABLE(I+1,J) = 0.0
C
    DO 15 J=1,INDV(I)-1
        IF(ZERO(I) .LE. 0) THEN
            TABLE(I+1,J) = 1.0
        ELSE
            TABLE(I+1,J) = TABLE(I+1,INDV(I))
        ENDIF
15    CONTINUE
C
14    CONTINUE
C
    RETURN
    END
C
C *****
C    SUBROUTINE TABWT(TABLE,NET,VNAM,IFCCD,INVAR,INVEC)
C
C    WRITES OUT THE TABLE OF PROBABILITIES
C
C    REAL TABLE(41,70)
C    INTEGER INVEC(40),IGLAB(40)
C    CHARACTER*8 VNAM(40)
C    CHARACTER*12 MAG
C
C    OPEN(8,FILE='TABOUT.OUT',STATUS='NEW',RECL=132,
X    CARRIAGECONTROL='FORTRAN')
C
C    MAG = 'MAGNITUDE '
C

```

```

      IT = 1
      DO 31 I=1,INVAR
        IF(INVEC(I) .GT. 0) THEN
          IGLAB(IT) = I
          IT = IT + 1
        ENDIF
31      CONTINUE
C
      NUMTAB = INT(IFCCD/10)
      IF(MOD(NUMTAB,IFCCD).GT.0) NUMTAB = NUMTAB + 1
C
      IB = 2
      IFT = IFCCD + 1
C
      DO 20 IT=1,NUMTAB
        IET = IB + 9
        IE = MIN(IET,IFT)
C
        IBL = IB-1
        IEL = IE -1
C
        WRITE(8,100)
100      FORMAT(//)
C
        WRITE(8,110) MAG,(VNAM(IGLAB(I)),I=IBL,IEL)
110      FORMAT(2X,A12,10(A8,4X))
        WRITE(8,101)
C
        DO 10 I=1,NET
          WRITE(8,120) TABLE(1,I),(TABLE(J,I),J=IB,IE)
120      FORMAT(1X,1PE8.1,3X,10(1PE10.3,2X))
          ISP = MOD(I,5)
          IF(ISP.EQ.0) WRITE(8,101)
101      FORMAT(/)
          10      CONTINUE
C
          IB = IE+1
          WRITE(8,102)
102      FORMAT('1')
C
          20      CONTINUE
          RETURN
          END
C
C

```


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