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ESTIMATING THE ECONOMIC COSTS OF RADIATION-INDUCED HEALTH EFFECTS

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SUMMARY

The research effort covered by this report was performed by Pacific Northwest Laboratory (PNL) for the Division of Risk Analysis, and the Division of Health, Siting and Environment, both within the Office of Nuclear Regulatory Research of the NRC. 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 flexible computer model, Health Effects Cost Model (HECOM), which utilizes the output of an accident consequences model (CRAC2) to calculate the discounted sum of the economic costs associated with 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 market value.

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1.0 INTRODUCTION

This report was prepared by the Pacific Northwest Laboratory (PNL) for the Division of Risk Analysis and the Division of Health, Siting and Environment, both within the Office of Nuclear Regulatory Research (RES) 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. 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 economic costs for the major types of potential radiation-induced health effects, and 3) developed a flexible computer code for calculating costs that could result over time due to a single nuclear incident. As a result of this effort, quantitative estimates of the economic costs of health effects risks will be available for inclusion in environmental impact statements for nuclear facility siting and for evaluation of safety-related actions. The introduction covers the need for health effects cost estimates, the nature of the radiation-induced health effects, the composition of resulting costs, and the scope of the PNL effort. 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 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 in part due 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 an economic model.

The lack of economic treatment of health effects has also been due to the argument that it is inappropriate, or even immoral, to place an explicit value on human life and health. This study does not attempt to estimate the value of human life or health; it estimates the economic losses to society that could occur due to radiation-induced illness and injury. Although 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 available information 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 NRC. They may be used in developing health effects impact 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.

1.2 OVERVIEW OF SOCIETAL[a] COSTS OF RADIATION-INDUCED HEALTH EFFECTS

The value of avoiding radiation exposure, whether for the general population or for 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 the illness and because of the lost productivity of the affected individuals. These primary economic costs are referred to as direct and indirect costs. Direct costs include all costs for treatment, travel to obtain treatment, patient care, equipment and supplies, while indirect costs are 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 nonmonetary 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; the cost of depression or psychological stress due to actual or anticipated illness. While recent attempts have been made to measure some of these effects, no rigorous estimates of secondary costs are available, either in absolute terms or relative to primary costs.

The relationship between the occurrence of health effects and the occurrence of economic costs is discussed in Section 1.2.1. In Section 1.2.2 which follows, the types of health effects that may be induced by radiation are described briefly. 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 Relationship of Health Effects to Costs

Three major types of health impairments may result from accidental radiation exposure: acute radiation injury and cancer affecting the exposed population, and genetic damage affecting future generations. Each of these may result in premature mortality, as well as morbidity (illness) and physical impairments. 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. The genetic effects of concern 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 of radiation exposure, the costs of the health effects would be spread over a substantial period

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

of time. While secondary, nonmonetary, costs would be associated with the health effects, they are not estimated and are not included in this discussion.

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. The starting point is a population that has been exposed (1) to a source of radiation at some point in time. Depending on the dose received, and the period of exposure, 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) accrue to 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 would face the risk of bearing offspring with dominant or recessive genetic damage (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. The health effects and their economic costs may continue for many generations.

1.2.2 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. 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 Chapter 3.

Acute Radiation Injuries

The occurrence of acute radiation injuries among 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 possibilities vary in intensity from negligible or undetectable to those that are more severe: temporary discomfort, 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.

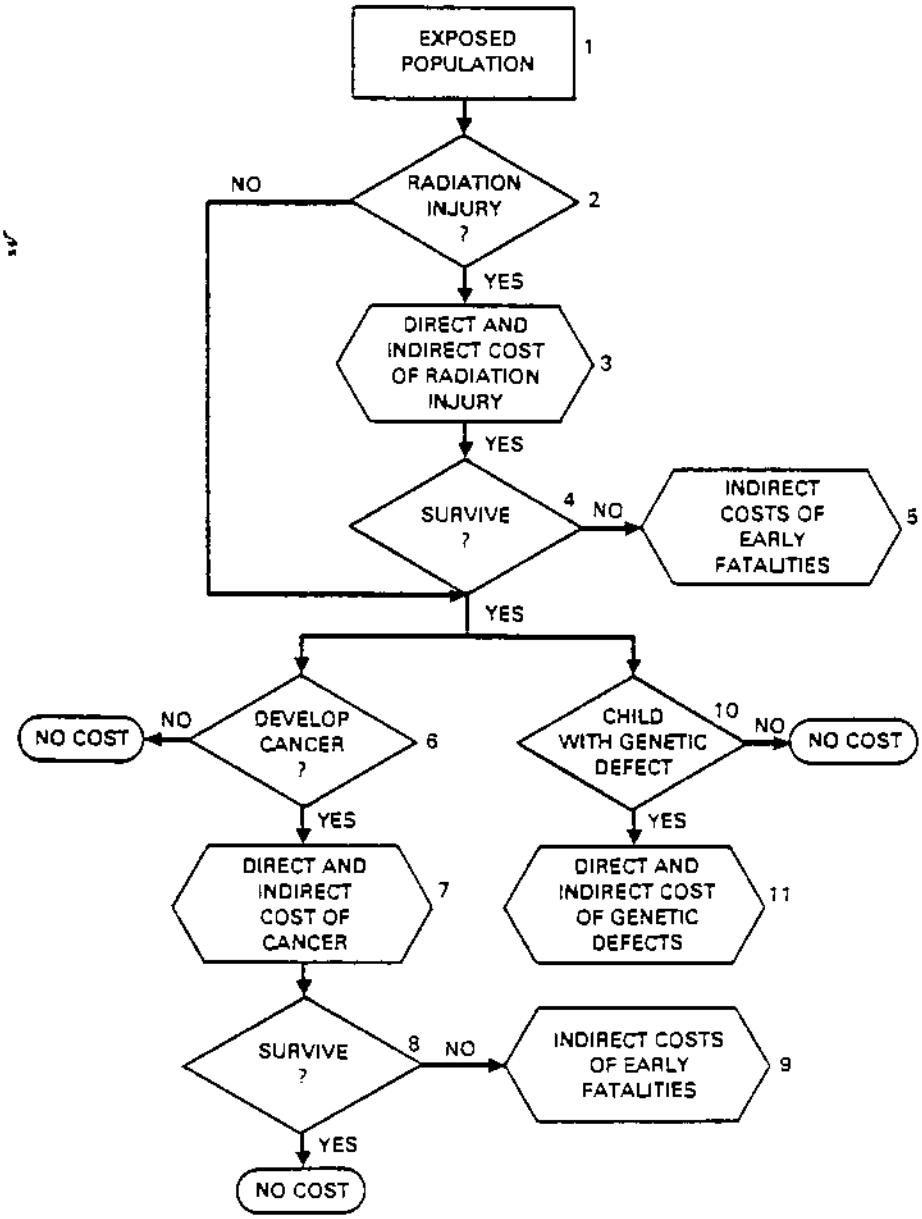

FIGURE 1.1. Diagram of Radiation-Induced Health Effects and Resultant Social Costs

- 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 50 rads may be affected and at doses above 200 rads virtually everyone would exhibit these symptoms (Blakely 1968, p. 35).

- Bone Marrow Syndrome - This process is initiated by whole body exposures of 200 rads or more. There is damage to the bone marrow, spleen and lymph nodes which 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. Approximately 50 percent of exposed individuals may be expected to die within two months of exposure at doses greater than about 450 rads (NRC 1975, Appendix VI, F-3).

- Gastrointestinal Syndrome - At whole-body doses above 600 to 1000 rads, cellular damage may result in gastrointestinal symptoms. 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 above.

- Pulmonary Syndrome - Doses of about 750 rads or more (Cooper, et al. 1982, p. 4-6) 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 (NRC 1975, Appendix VI, p. F-3).

- Hypothyroidism - This is an impairment of thyroid function which can be induced by radiation exposure. Oral medication is effective and inexpensive (NRC 1975, Appendix VI, p. 9-13).

- 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).

- Cataracts - Doses of 200 to 500 rads to the lens of the eye may result in formation of cataracts after a latency period that varies with both dose and dose rate (NRC 1975, Appendix VI, p. 9-18).

- Skin and Hair Damage - Loss of hair occurs two to three weeks after external doses in excess of 300 rads. This is likely to be temporary unless the dose exceeds 600 rads (NRC 1975, Appendix VI, p. F-13). The skin may also be affected by doses in this range, 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 2000 rads) can result in permanent skin ulceration (Prasad 1974, p. 240-248). Survivable whole-body acute doses are unlikely to cause more severe injuries than hair loss and skin reddening.

- Prenatal Injury - The radiosensitivity of embryos is very high, resulting in deaths from doses as low as ten rads. Most such deaths would be unnoticed due to the early stage of the pregnancy. In later stages of development the fatality rate decreases but the probability of abnormalities increases. These generally take the form of growth impairment and mental retardation, especially microcephaly. As in the case of prenatal mortality, cases have been documented after exposures of about ten rads (NRC 1975, Appendix VI, p. F-17-20).

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 differ at which excess cancers appear in various sites. Cancer induction is influenced by sex, age when irradiated, and type of radiation, among other factors (BEIR 1980, p. 84-5). The cancers that are most susceptible to radiation induction are leukemia and cancers of the breast, bone, lung 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 the Calculation of Reactor Accident Consequences Model (CRAC) as well as in this study.

Genetic Effects

Genetic effects, in the form of abnormalities and diseases, 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 the mutations or the associated health effects. Thus, the health effects that occur are of the same type that occur spontaneously. Of the possible types of mutation, autosomal dominant disorders are most likely to increase in direct proportion with radiation exposure. These disorders may cause chondrodystrophy, osteogenesis imperfecta, neurofibromatosis, eye

anomalies, polydactylism and polycystic renal disease. Other types of health effects due to autosomal dominant mutations occur much less frequently (NRC 1975, Appendix VI, Appendix I).

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 4. The PNL health effects model focuses on costs because they are more directly measurable and because they account for a substantial part of the public's evaluation of risk.

There are two ways to estimate the cost of illness, from either a prevalence or an incidence perspective (Hartunian, Smart and Thompson 1981). Essentially, a prevalence approach asks, "How much is an illness, e.g., cervical cancer, costing U.S. society in 1983?" 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 1983, 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. PNL employs the incidence approach 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 (Abt 1975). These elements of social costs, 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 a market value. However, it is clear that they are an 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 these nonmedical direct costs; they 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 measured 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 forgoes the goods and services that would have been produced had the patient (or family) been able to work. Valuing these productivity losses is similar to valuing 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 forgone because of radiation-related morbidity and mortality. A variant of the human capital approach would include among indirect costs only the forgone net production; that is, the value of the person's production less his or her future consumption (for example, Weisbrod 1961). However, net production measures only the value that the rest of the public places on someone's life and ignores the value that person derives from his or her own personal consumption. The total production approach comes closer, therefore to a full measure of the indirect costs in terms of human capital. (A fuller discussion of alternative approaches to measuring indirect costs is provided in Chapter 6.)

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 present value in the year of exposure.

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 estimation of health effects costs.

1.3 SCOPE OF THE STUDY

Radiation-induced health effects may result in both economic costs and nonmonetary impacts on society. PNL cost estimates are limited to the economic costs: 1) the direct costs of health care provision and 2) the indirect costs of productivity losses resulting from illness or premature mortality. Other measures of health effects impacts, such as the value of pain and suffering, are beyond the scope of this effort.

The PNL cost estimates represent the present value of probable future costs that are likely to be associated with each of the major types of radiation-induced health effects. In the case of acute radiation injuries, PNL

estimates the costs of bone marrow syndrome, gastrointestinal syndrome, pulmonary impairment, prodromal symptoms and prenatal injuries.[a] For cancers, the PNL cost estimates cover the same categories projected by the CRAC2 model: leukemia, lung, breast, bone, gastrointestinal tract, thyroid and all others. In addition, direct and indirect costs are considered for radiation-induced genetic effects occurring in future generations.

The cost estimation methodology is designed to use the health effects output of the CRAC2 model, but also to accommodate health effects projections from other sources as well. PNL has developed a Health Effects Cost Model (HECOM) for implementation of this methodology. The model is modular in structure and is designed for flexibility and ease in modification and updating. It is expected that HECOM can be readily adapted to future changes in CRAC2 and related models for projecting health effects.

1.4 REPORT STRUCTURE

This report presents the conceptual and informational base from which PNL has developed health effects cost estimates. It describes in detail the methodology employed in estimating each component of these costs. In addition, it provides a description and documentation of the model (HECOM) 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.

In Chapter 3 we review the major health effects studies and models which provide the basis for cost estimation. Assumptions as to health effects incidence and timing that affect cost estimates are discussed, as are the uncertainties involved in the health effects projection.

Though the estimation of health effects costs is difficult, the difficulties stem from incomplete medical and economic data and information, rather than inadequacy of the conceptual basis for such cost estimates. Chapter 4 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, PNL employs the human capital approach in developing cost estimates.

The methodology used in this cost estimation is detailed in Chapters 5 and 6. In Chapter 5 the direct costs of radiation-induced morbidity are discussed. Costs for radiation injuries are developed in Section 5.1, costs for cancers in Section 5.2, and costs for genetic effects in Section 5.3. These sections present information as to likely treatments and the associated costs, and describe the methods used to calculate each cost component. Similar

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(a) Other types of radiation injuries, such as cataracts, are not included because they are dominated by the effects of acute whole-body exposure.

information is presented in Chapter 6 for the indirect costs of morbidity. The same cost estimation methodology applies to each type of health effect.

An overview of PNL's Health Effects Cost Model (HECOM) is provided in Chapter 7. The general approach employed to develop a flexible health effects costs model is presented in Section 7.1. HECOM will accept input data from various sources, will allow simulation of alternative health effects incidence assumptions and can easily be modified or updated. The model structure is described in Section 7.2 and use of CRAC2 data as inputs to HECOM is discussed in Section 7.3. The sensitivity of health effects cost estimates to various data and model parameters is explored in Section 7.4.

Chapter 8 presents an example of the application of HECOM health effects cost estimates to the evaluation of reactor accidents risks. The estimated health effects costs are based on one hypothetical accident scenario.

Documentation of the model appears in Appendix A, along with summaries of the data used in the base case. The computer code is listed in Appendix B.

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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 level of uncertainty inherent in the HECOM cost estimates, 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, improvement of health effects incidence 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 health effects cost 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 combines health effects incidence and timing with streams of treatment costs and lost productivity values to approximate the sum of direct and indirect costs of potential acute radiation injuries and fatalities, cancers and genetic effects. The flexibility of HECOM allows analysis of costs while varying key parameters. The model can accept changes in incidence estimates, in treatment costs, in the discount rate and in real growth rates. Because of its flexibility, it 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 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. Thus, 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 BOUNDING ESTIMATES 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. For cancers the choice of an absolute versus a relative risk model has a major effect on cost estimates. We are currently using an absolute risk model to distribute cancer incidence over time in HECOM. In regard to genetic effects, there is uncertainty as to the frequency of defects of various degrees of severity. We have made a number of assumptions to develop cost estimates, however, but available information regarding genetic defect severity is inadequate for estimating the level of uncertainty in our severity estimates.

There is considerably less uncertainty regarding the direct and indirect cost estimates we have developed for radiation injuries, cancers and genetic effects. Using the HECOM base case parameters of a four percent discount rate and one percent growth rates for medical costs and labor productivity, the level of uncertainty in total costs due to the uncertainties in the direct and indirect cost components is about 25 percent.

2.3 RELATIVE 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. For instance, cost estimates for cancers and genetic effects are based on probability 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 occuring at any specific future time; they are statistical constructs that weight the probability and magnitude of costs in each year of the period modelled 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.

Results of 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. For total costs, a ±25 percent range of uncertainty is shown, based on a sensitivity analysis of HECOM cost estimates. The present-value cost estimates in Table 2.1 are for one case of each type of

TABLE 2.1. HECOM Present-Value[a] Cost Estimates Per Radiation Injury, Cancer or Genetic Effect (1981 $)

	Direct Cost (000 $)	Indirect Cost (000 $)	Total Cost (000 $)	
			Base	±25 Percent
Radiation Injuries				
Prodromal	1.0	0.1	1.1	0.8 - 1.4
Bone Marrow	56.0	96.1	152.1	114.1 - 190.1
Lung	3.6	96.1	99.7	74.8 - 124.6
Gastrointestinal	28.0	96.1	124.1	93.1 - 155.1
Prenatal	100.0	25.8[b]	125.8[b]	94.4 - 157.3
Cancers				
Leukemia	6.3	30.6	36.9	27.7 - 46.1
Lung	2.4	4.0	6.4	4.8 - 8.0
Gastrointestinal	3.5	3.5	7.0	5.3 - 8.8
Breast	0.9	2.8	3.7	2.8 - 4.6
Bone	9.9	18.4	28.3	21.2 - 35.4
All others	2.1	3.3	5.4	4.1 - 6.8
Thyroid	0.2	0.1	0.3	0.2 - 0.4
Genetic Effects	5.5	0.1	5.6	4.2 - 7.0

(a) Using a 10% discount rate.
(b) Because of the HECOM aggregation procedures, this figure includes some indirect costs of cancers affecting individuals irradiated in utero.

health effects 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 would not be representative of the cost distributions.

For radiation injuries the total costs range from those for prodromal injuries ($0.8K to $1.4K), through those for bone marrow ($114K to $190K) injuries and for manifestations of acute radiation syndrome, to the costs of prenatal injuries that are over $200K per injury. Since each of these injuries are qualitatively different in nature, as well as in costs, they are best considered as five separate categories of effects rather than as a single category, radiation injuries.

Cancer costs cover a narrower range, from those for nonfatal thyroid nodules and thyroid cancers ($0.2K to $0.4K) to those for leukemia ($27.7K to $46.1K). The indirect costs of leukemia and bone cancer are substantially higher than those of other cancers, due to the higher risk to younger people and the potential brevity of the latency period.

The cost estimate for a genetic effect has a range from $4.2K to $7.0K. This cost estimate 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 ten generations. Serious and minor effects are weighted in estimating the genetic effects costs so the estimate applies to the broad category of genetic effects.

The individual 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.

Regarding the estimation of cancer risks, there is reason to believe that recent data from the Japanese A-bomb survivors may lead to increased use of relative risk models to model some cancer risks (Cooper et al. 1982, Section 5). Currently, HECOM employs an absolute risk model to distribute cancer fatalities over time; this is consistent with the CRAC2 methodology. HECOM is designed to accommodate a relative risk model option, that has not yet been implemented. We recommend that this option be developed.

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 question arises partly from the Reactor Safety Study's (NRC 1975) suggestion 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 relatively intensive 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 would benefit from examination of the cost effects that would stem from the difference in mortality rates associated with various types of medical care?

An effort to assess the costs of the principal diseases associated with mutation would entail first the identification of those diseases and second the gathering of relevant cost data. To distribute genetic diseases according to severity would be a simpler task that could employ, perhaps, a panel of experts.

Changes in the estimation of particular health effects costs as discussed in the preceding paragraphs would add increased precision to HECOM. Regardless of whether those changes are made, an important next step is the application of the model to examples of hypothetical reactor accidents. The current output from the model shows the richness of information that can be obtained. Application of the model may be expected, in addition, to lend a new empirical basis to the enduring policy question concerning the potential costs associated with irradiation.

Aside from improvements to and application of the current model, beneficial advances could be made in the valuation of risk by further conceptual and empirical work toward the development of a contingent market study of the public's risk valuation. The ideal approach to estimation of the value of a

change in risk is to measure individuals' willingness to exchange income for that risk change. A carefully designed contingent market survey can provide information about individuals' preferences toward nuclear risk; from a rigorous theoretical perspective, such information about individual valuation is most appropriate in measuring the benefits of risk reduction.

2.0 REFERENCES

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3.0 REVIEW OF HEALTH EFFECTS PROJECTIONS

In this chapter we review the information about the incidence of health effects that provides the bases 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. Radiation injuries are discussed in Section 3.1, cancers in Section 3.2 and genetic effects in Section 3.3.

3.1 RADIATION INJURY INCIDENCE AND TREATMENT

Depending on dose levels and on individual sensitivities, exposure to significant amounts of radiation may result almost immediately in acute symptoms that could range from 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. It is convenient to consider the range of possible acute effects by grouping radiation injuries into three categories: 1) prodromal symptoms, which last only a few days; 2) bone marrow syndrome, gastrointestinal syndrome, and pulmonary impairment, which are all potentially life-threatening; and 3) in-utero effects, which cause severe and permanent impairment to the irradiated fetus. In this section we provide a review of how each category of injury relates to radiation dosage and how the clinical signs of the injury are likely to progress. We also suggest parallels with more common diseases in order to estimate the levels of treatment that may be involved for each injury category.

3.1.1 Prodromal Symptoms

Prodromal symptoms may include nausea, loss of appetite, headache, diarrhea, and weakness. The higher the radiation dose and the shorter the time over which exposure occurs, the sooner these symptoms occur and the longer they persist (Blakely 1968, p. 35 and NRC 1975, p. F-13). Blakely (1968, p. 35) reports that prodromal symptoms may occur occasionally after a dose as low as 50 rads, but are more likely at 100 rads and are seen in all cases at 200 rads and above.

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 recent illness or injury (Dalrymple 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 is the only 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 is a latent period before the manifestation of more serious injury. The duration of the latency period varies inversely with the dose rate. Table 3.1 provides a summary of the progression of acute radiation symptoms for various whole body dose levels. In the less serious cases this latency period lasts from 1-3 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. The cost estimates developed in this study assume that facilities are available locally. If unusual efforts were required to deliver medical care, the costs could be substantially higher.

3.1.2 Bone Marrow Syndrome

Failure of the bone marrow system would be the primary cause of serious illness or death as a result of radiation exposure in a reactor accident. Blakely (1968, p. 37) places the lower threshold for bone marrow syndrome at about 200 rads, with milder manifestations resulting from doses between 200 and 400 rads and severe symptoms at doses between 400 and 600 rads.

The Reactor Safety Study (NRC 1975, Appendix VI pp. F-1 - F-3) presents dose-response curves for bone marrow damage depending on the extent of medical intervention. That study predicts 50 percent of the people exposed to 340 rads would die within 60 days if they were given only minimal treatment. With supportive medical treatment, the estimate is that 510 rads would be a lethal dose within 60 days to 50 percent of those exposed. Supportive treatment is described later in this section. With heroic treatment the report asserts that the 50 percent lethal dosage may be as high as 1050 rads for whole-body exposure. Heroic treatment would involve bone marrow transplantation. We consider transplants to be an unlikely form of treatment because of the difficulties of finding a compatible donor for most patients, a problem that may be accentuated in the aftermath of a reactor accident. In addition, at least one researcher (Andrews 1980) advises that marrow transplant may not be helpful.

Bone marrow 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. Treatment is centered around 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; NRC 1975, Appendix VI p. 9-3). Administration of antibiotics is prescribed (Saenger 1982; NRC 1975), as well as continual monitoring with laboratory tests (Andrews 1980; Saenger 1982) and use of blood transfusions.

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,

TABLE 3.1. Clinical Progression of Acute Radiation Syndrome[a]

Approximate Dose Levels (whole-body rads)	Clinical Progression						
50-200		prodromal symptoms	→	recovery			
200-400	prodromal symptoms	→ latency 2-3 weeks	→	mild bone marrow crisis 2-3 weeks	→	most recover	
400-600	prodromal symptoms	→ latency 1 week	→	severe bone marrow crisis 4-6 weeks	→	about 50 percent recover[b]	
600-1000	prodromal symptoms	→ latency few days	→	gastrointestinal injury 1-2 weeks	→	probable death	
1000's		death within hours from cerebrovascular crisis					

(a) Blakely (1968) describes a similar pattern of disease progression, except that he predicts near 100 percent mortality at 600 rads, with cerebrovascular crisis occurring at around 1400 rads. NRC (1975) considers dose ranges from 350 to 550 rads as critical for the bone marrow. That study distinguishes between whole body doses and locus-specific doses to gastrointestinal tract and lungs. NRC 1975 (Appendix VI p. I-7) suggests that in the absence of bone marrow complications mortality from gastrointestinal injury alone would not occur below 1000 rads.

(b) At dose levels of about 450-500 rads 50 percent of the exposed population are expected to die within 60 days even with supportive treatment. At 600 rads the death rate may be close to 100 percent without heroic intervention. NRC (1975) suggests that 50 percent could survive whole body doses as high as 1050 rads with heroic treatment (i.e., with a bone marrow transplant).

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 the provision of "supportive treatment" are similar to those given a nonsurgical burn trauma patient. The costs of such services are estimated in Section 5.1.2.

3.1.3 Gastrointestinal Syndrome

At whole-body doses over approximately 600 rads the symptoms of gastrointestinal syndrome are likely to precede those of bone marrow damage (Saenger 1982; Blakely 1968). The onset of symptoms comes after a shorter latency

period than at lower doses (a few days to a week). 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). For local irradiation of the gastrointestinal tract without a high whole-body dose, the lethal dose may be closer to 3500 rads (NRC 1975).

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 healing of the intestinal lining (Blakely 1968, p. 41). However, recovery will result in the patient facing severe bone marrow syndrome a short time later. Because of this threat of bone marrow syndrome in patients who survive the gastrointestinal problems, we assume that gastrointestinal patients would be treated from the start in the same isolation prescribed for bone marrow patients.

3.1.4 Pulmonary Impairment

Pulmonary impairment can be expected in approximately five percent of cases after inhalation doses of 3000 rads and in 100 percent after inhalation of 6000 rads (NRC 1975, Appendix VI, p. F-6). Depending on the source of the radioactivity, 100 percent mortality can be expected from lung doses of 15,000 to 30,000 rads. Although it is possible to receive that high an inhalation dose with relatively low whole body doses, at any given distance from the reactor the probability of death from lung dose would always be substantially lower than that from the associated bone marrow dose (NRC 1975, Appendix VI, p. 9-5).

Symptoms of pulmonary injury include pneumonitis and pulmonary fibrosis. In the absence of bone marrow syndrome, we assume these symptoms could be treated in an average hospital room.

3.1.5 In-Utero Injury

A category qualitatively different from other radiation injuries is in-utero or prenatal effects. Injuries and deaths would be due mainly to irradiation during the second trimester of pregnancy, with spontaneous abortion likely for embryos in earlier gestation. The nervous system is particularly sensitive to injury and effects such as growth impairment, microcephaly and mental retardation have been observed at doses as low as 10 to 20 rads (NRC 1975, Appendix VI, p. F-18). Microcephaly, which is generally associated with severe retardation, occurred in about 50 percent of fetuses exposed to 150 rads as a result of atomic bomb exposures (p. F-36). Using information about the age structure of the potentially exposed population and dose rates, the number of in-utero injuries can be estimated, though it is not by CRAC2.

Long-term institutionalization may be required for individuals irradiated in utero. The care provided may be similar to that given to individuals who are severely affected by Down's Syndrome or spina bifida. For lack of information specific to in-utero radiation injuries, we rely on the probable similarities with those two other prenatal-onset diseases with long-term impairment to guide our cost estimates.

3.1.6 Other Radiation Injuries

There are other possible forms of injury from irradiation that are of less concern than those outlined above, either because they cause relatively minor problems or because they become serious only at doses high enough to preclude probable survival:

- Hypothyroidism - This is an impairment of thyroid function that can be induced by radiation exposure. Oral medication is effective and inexpensive (NRC 1975, Appendix VI, p. 9-13).

- Sterility - Radiation-induced sterility may be either temporary or permanent. Males may have temporary effects at lower doses than females but 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 (NRC 1975, Appendix VI, p. 9-15).

- Cataracts - Doses of 200 to 500 rads to the lens of the eye may result in formation of cataracts after a latency period that varies with both dose and dose rate (NRC 1975, Appendix VI, p. 9-18).

- Skin and Hair Damage - Loss of hair occurs two to three weeks after external doses in excess of 300 rads. This is likely to be temporary unless the dose exceeds 600 rads (NRC 1975, Appendix VI p. F-13). The skin may also be affected by doses in this range, 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 2000 rads) can result in permanent skin ulceration (Prasad 1974, p. 240-248). Survivable whole body doses are unlikely to cause more severe injuries than hair loss and skin reddening.

3.1.7 The CRAC2 Projections of Radiation Injuries

The CRAC2 output includes estimates of early fatalities and injuries; i.e., those occurring within one year of accidental radiation exposure. (In actuality, most of these effects would occur within the first three months.) For exposures of less than 1000 rads, which includes most hypothetical accident scenarios, the primary cause of early fatalities would be dose to the bone marrow. In some cases, however, pulmonary exposure could also be instrumental in inducing mortality. To estimate fatalities the CRAC2 computer code calculates population exposures and then applies a probabilistic fatality rate to the estimated exposure level of each segment of the population. The dose and associated mortality rates used in these calculations are shown in Table 3.2.

The methodology used is documented in the Reactor Safety Study (NRC 1975, Appendix VI) and in the CRAC2 user's manual (Sandia 1981). Mortality rates for dose levels between those listed are developed within the model by linear interpolation. Early fatalities, as estimated by the CRAC2 model, are the sum

TABLE 3.2. Dose Values and Associated Mortality Rates Used in CRAC2 (Sandia 1981)

Organ	Dose (rem)	Mortality Rate
Bone Marrow	320	0
	400	.03
	510	.5
	615	1.00
Small Intestine Lining	2000	0
	5000	1.00
Lung	5000	0
	14,800	.24
	22,400	.73
	24,000	1.00

of probable fatalities for the entire exposed population; double counting of fatalities due to multiple fatal organ doses is avoided in the model.

CRAC2 use of the mortality rates shown in Table 3.2 is based on the assumption that all of the injured would receive a level of medical treatment designated as "supportive" by the Reactor Safety Study (Appendix VI, p. 9-3). Unfortunately, an estimate of the total number of people who would require this treatment is not available from the CRAC2 output. While the fatalities are counted, the survivors of bone marrow exposure are not explicitly included in the category of "early injuries" and their number cannot be derived from the number of fatalities. It would be advantageous to indicate the population receiving doses within 100 rem intervals, so that cost estimates could be linked to the severity of the injuries.

Injuries evident in the immediate post-accident period are calculated by the CRAC2 model from the information in Table 3.3. As in calculating fatalities, the injury rate is applied to the population projected to have received each dose level and the resulting estimates are summed. The threshold for injuries is approximately 50 rads. Injury rates at intermediate dose levels are derived by linear interpolation within the model. At the levels of possible doses to offsite population developed in most accident scenarios, it is whole-body dose that is primarily responsible for injuries.

People receiving whole-body doses above 50 rads may experience prodromal symptoms such as nausea, vomiting, anorexia and diarrhea within a few hours of exposure and continuing for a day or two. While CRAC2 calculates the number of people likely to experience actual prodromal symptoms, it does not provide any indication of the number likely to require medical care. As noted by Dalrymple (1973, p. 192), people in the vicinity of an accident may experience circulatory system or gastrointestinal system symptoms that are due to anxiety rather

TABLE 3.3. Dose Values and Associated Morbidity Rates Used in CRAC2 (Sandia 1981)

Organ	Dose (rem)	Morbidity Rate
Whole Body	55	0
	150	.3
	280	.8
	370	1.00
Lung	3000	0
	3000.1	.05
	6000	1.00
Small Intestine Lining	1000	0
	1000.1	.05
	2500	1.00

than radiation exposure. Thus, both injured and uninjured individuals may initially experience identical symptoms. In the event of an accident where the occurrence of significant population exposures is suspected, a major population screening and treatment effort would be required. The number of people who would require treatment for prodromal symptoms and screening for more severe injuries would be at least as large as the number of early injuries calculated by the CRAC2 code. There is a high probability that the actual number would be substantially larger.

The present form of CRAC2 output for early injuries is ill-suited to projection of direct costs. Only an aggregate measure of early injuries is available, one that includes transient, prodromal symptoms on the same basis as life-threatening pulmonary and gastrointestinal effects and that omits bone marrow injuries. Major types of potential injuries and their status in the CRAC2 calculation are shown in Table 3.4. If those effects that are included in the CRAC2 calculations were available by organ (e.g., lower intestine lining), the estimates could be used directly in calculating costs. No technical reason for the exclusion of bone marrow syndrome from the estimate of early injuries has been identified. CRAC2 modifications required to calculate numbers of bone marrow injuries are discussed in Section 7.3.

There is an additional category of health effects that is omitted from the CRAC2 calculations but which may have substantial impacts. That is in-utero fatalities and injuries. An analysis of the numbers of fatalities potentially involved indicated that "embryonic and fetal deaths would be fewer than 10 to 5 percent, respectively, of the early fatalities..." (NRC 1975, p. 9-11). The rationale given for excluding them from reported early fatalities is that the embryonic (first trimester) deaths would not be noticed and the fetal (second and third trimesters) deaths fall within the range of uncertainty of the CRAC estimates.

TABLE 3.4. Summary of Early Injury-Related Information

Major Injuries Categories	Included in CRAC2 Estimate	Duration of Acute Symptoms
Prodromal symptoms	yes	2 days
Bone marrow syndrome	no	4 to 8 weeks
Gastrointestinal syndrome	yes	(a)
Lung effects	yes	1 year(b)
In-utero injuries	no	lifetime

(a) Patients who die generally do so within 10 to 14 days. No estimate of the recovery period was noted in the literature but it is likely to be several months.
(b) No information on treatment or likely length or recovery period was found.

While projection of fetal injuries would not have much effect on the total number of early injuries calculated by CRAC2, it is important in the calculation of accident costs since these injuries are the most costly type of health effects. (See the discussion of the in-utero injury treatment costs in Section 5.1.5). Sufficient information to project in-utero injuries is available from the Reactor Safety Study (NRC 1975) and other sources.

3.2 CANCER INCIDENCE AND TREATMENT

There is wide consensus among scientists that an association exists between ionizing radiation and cancer. In fact, scientists may know more about the carcinogenic effects of ionizing radiation than about those of any other environmental agent (Land 1980). 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 is rapidly changing concerning the risks from radiation, there are several issues to be raised pertinent to the CRAC2 estimates of cancer effects. In this section we do not attempt to provide resolution of those issues, but rather to explain how reasonable estimates may vary from those used as inputs in this study.

In regard to estimates of incidence, there are reasons to suggest that the CRAC2 estimates may be too high, and other reasons why they may be too low. In addition to questions of dose-effect relationships, changes in treatment may also have an important influence on the cost estimates provided by this study. Questions are raised relevant both to incidence and to treatment.

3.2.1 Incidence Assumptions

There are at least two general issues of current concern in regard to cancer incidence estimates, each relevant to the Reactor Safety Study (NRC 1975) and CRAC2 projections. First, dosimetry data and incidence estimates for the Japanese atomic bomb casualties have come into question. Second, uncertainty about the shape of the dose-response curve may have an important impact on the estimates of responses to low-dose radiation.

The issue regarding the accuracy of dosimetry data for the Japanese A-bomb casualties is central to dose-effect estimates because BEIR 72 and BEIR 80 (and therefore the Reactor Safety Study and CRAC2) base their projections of incidence on the Japanese data. Each of those incidence projections employs dosimetry estimates computed in 1965 and labeled "temporary" (the "T65" dose). Further study now suggests that the neutron component of the Hiroshima bomb may have been lower than previously calculated, with some corresponding increase in the gamma component (Loewe 1981). The net result may be that some risk estimates will be doubled (Beebe 1981).

In addition to changes in dose estimates, other new information on the Japanese casualties suggests that cancer incidence and related mortalities may be higher than previously estimated (Wakabayashi et al. 1983). Consideration of the new estimates reinforces a conclusion that earlier incidence estimates based on the Japanese data may be significantly too low.

Unlike the new Japanese data that suggest current dose-effect estimates are too low, the dual problems of inadequate sample size and uncertainty regarding the shape of the dose-response function result in an ambiguous conclusion that current estimates could be either too high or too low. A problem arises in estimating the effects of low-level radiation because such an estimate requires a study with very large sample size. Land (1980, p. 1197) describes the problem with an example: "If the excess risk is proportional to dose, and if a sample of 1000 persons is necessary to determine the effect of a 100-rad exposure, a sample of 100,000 may be needed for a 10-rad exposure, and about 10 million for 1 rad." The Japanese Life Span Study sample includes data on 110,000 people, some from Hiroshima and some from Nagasaki, with exposure to a very different mix of radiation types in the two cities. While the sample may be adequate for projection of high-dose effects, it is unlikely that the Japanese data can provide estimates of risk in the low-dose region except with the assumption of a specific dose-response function (Beebe 1981). Since the sample is too small and too diverse to derive estimates at low doses, the experience at high doses must be extrapolated to obtain low-dose estimates. The critical question is, on what basis should the extrapolation be made: is the dose-response function linear or of some curvilinear form?

Extrapolation assuming a linear dose-response curve may overestimate low-dose responses, if the true function actually curves up more steeply at high doses. Conversely, adjustments that imply a curvilinear (positive second derivative) dose response curve may cause an underestimate of the response if the function is linear.

BEIR 72 assumed a linear dose-response function for all types of cancer. BEIR 80 subsequently asserted a curvilinear (linear-quadratic) dose-response function for all cancers, against a dissent from the Committee Chair (BEIR 80, pp. 227-253) who argued for a linear form. Beebe (1981, pp. 780-781) supports the use of the linear form for its ease of application and its interpretability as an upper boundary. Land (1980, p. 1202) observes that the linear model appears to overestimate leukemic effects of low-dose radiation, although it fits reasonably well the evidence of breast cancers associated with low doses.

Basing its "upper bound" estimate on the linear extrapolations of BEIR 72, the Reactor Safety Study offers a "central estimate" for all cancers other than breast cancer to account for "the ameliorating effects of dose protraction and the lesser effectiveness of very small acute doses." The central estimate is not a representation of a curvilinear dose-response function but in modifying the linear function it has a similar effect. Cooper et al. (1982, p. 5-4) cite more recent studies that suggest that fractionation by dose protraction may make low doses even more effective at low dose rates. Cooper et al. conclude that such studies would argue against dose reduction factors (such as used in the Reactor Safety Study to adjust from the BEIR 72 functions to the central estimates). In fact, they observe that those studies support dose factors that would result in higher dose-effectiveness at low, protracted doses. (Cooper et al. 1982, p. 5-4)

CRAC2 (Sandia 1981) employs the central estimate from the Reactor Safety Study. As discussed above, there are some reasons to suspect that projection is too high, others to consider it too low. It is likely that the central estimate adopted in CRAC2 lies within the band of uncertainty. The data and models that provide the basis for CRAC2 estimates are currently being reviewed (Cooper et al. 1982). Completion of this review is expected during 1983.

3.2.2 Treatment Assumptions

Due to lack of more recent information, this study of health effects costs assumes cancer treatment effectiveness to be the same today as it was in the early 1970s. First, the estimates of cancer mortality input to the PNL model via CRAC2 are derived from BEIR 72. Therefore, the estimation of fatality costs is based on fatality rates that do not consider any medical progress since 1972. Second, the direct costs of cancer treatment included in this study are based on information obtained through the Third National Cancer Survey completed in 1974. That information includes the recollections of survivors and of nonsurvivors' kin regarding treatment received in the early 1970s.

In its effects on cost estimation, the assumption of unchanging treatment modes yields an ambiguous result. On the one hand, to the extent that medical advances have lowered cancer mortality rates since 1972, the projection of early mortalities should be adjusted downward. The indirect costs would be expected to be lower as a result of such an adjustment. On the other hand, medical advances have been obtained only with increases in real costs. Cancer treatment is more intensive than in the early seventies, and consequently, the direct costs may be higher in real terms. We are unable to discern whether these changes have led to increased or decreased economic costs.

3.3 NATURE AND INCIDENCE OF GENETIC EFFECTS

In the 1950s, as government and public attention focused on the possible risks of radiation, genetic risks were the predominant concern (Denniston 1982). Over time, attention has shifted to radiation-induced cancers. This shift may be due partly to the perception that cancers pose a more immediate threat, and also partly to the fact that science has displayed a greater facility in quantifying cancer risks than in estimating genetic effects. In this section, we review selected relevant literature for a discussion of the difficulties in predicting radiation-induced genetic disease.

There are several enduring impediments to the estimation of the genetic effects of increased radiation levels. First, evidence is weak regarding the linkage between radiation and genetic damage in humans. Since radiation causes identifiable mutations in other mammals, geneticists generally agree that radiation can cause harmful mutations in humans. However, there remain difficult questions 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 are identified and quantified, there remains an imposing problem of predicting the nature and severity of clinical manifestations (observable diseases) of each type of genetic damage.

3.3.1 Kinds of Genetic Damage Associated with Radiation

Among the categories of genetic damage, autosomal dominant disorders have special importance in radiation genetics: the relationship between the mutation rate and birth defect frequency is relatively direct and 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.

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 are expressed almost exclusively in males. These disorders behave as dominants in males. Estimates of 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 disorder is approximately 0.8 per 1000 liveborn males. (Stevenson 1959; Trimble and Doughty 1974; Carter 1977).

Unlike dominant and X-linked disorder that require the presence of only one mutant gene for their expression, autosomal recessive disorders appear only when two mutant genes are inherited, one from each of the parents. There is a very low probability of a newly induced recessive mutation pairing up with a previously existing mutant allele in a way that will express a deleterious condition in the first or early generations (Oftedal and Searle 1980).

Instead, the interval between the induction of a recessive mutation in a gene and the birth of an affected individual may be centuries or even millenia (Ash, Vennert, and Carter 1977). For that reason the category of recessive genetic disorders is usually considered to be negligibly affected by increased radiation (UNSCEAR 1977; BEIR 1980). In contrast, Edwards (1979) holds that the recessive disorders are particularly severe and that over a very long time they represent the "main hazard in man" (p. 467). Estimates of the current incidence of recessive disorders vary from 1.1 per 1000 liveborn (Trimble and Doughty 1974) through 2.1 per 1000 (Stevenson 1959) to 2.5 per 1000 (Carter 1977).

In addition to the disorders discussed thus far, all of which have unambiguously genetic causes, there is a large category of multifactorial disorders (also called irregularly inherited disorders.) These may stem partly from dominant mutations and partly from environmental causes. In order to predict their increased incidence, it is necessary to estimate their "mutation component," the proportion of their frequency that depends on the mutation rate. Each multifactorial disorder has its own mutation component and very little is known about these components (Denniston 1982). UNSCEAR (1977) estimates the mutation component to be 5 percent; the BEIR (1980) estimate is 50 percent. Estimates of current incidence range from 4 per 100 liveborns (Stevenson 1959) to 9 per 100 (Trimble and Doughty 1974).

In addition to the genetic mutations discussed above, radiation exposure may cause a broad class of chromosome anomalies. This class includes three types of disorders: numerical aberrations, rearrangements, and deletions (Denniston 1982). The deletions may have effects indistinguishable from those of single gene mutation and they are included among those disorders. The numerical aberrations 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 (Denniston p. 331). As a class, chromosome anomalies lead to impairment in approximately 0.6 percent of liveborns, according to Denniston (p. 331).

3.3.2 Estimation Methods

There are two principal ways to estimate the effect of increased radiation dosages in terms of the incidence of genetic and chromosomal disorders. Both involve extrapolation to humans from experience with irradiated mice and other mammals.

The doubling dose method is based on the equation (Dennison 1982)

$$\text{Induced burden per rad} = \frac{\text{spontaneous burden}}{\text{doubling dose}} \times \text{mutational component}$$

The spontaneous burden is estimated from human population studies such as Stevenson (1959), Trimble and Doughty (1974) and Carter (1977), as reported above for each type of genetic disorder. The mutational component is the part of the existing burden expected to increase in proportion to the mutation

rate. It is 100 percent for autosomal dominant disorders and open to question for others. The doubling dose itself is calculated from nonhuman data, generally from the mouse. The increase in dominant disorders in humans is estimated from the induced mutation rates of recessive genes in the mouse. Each of the variables in the doubling dose equation is dependent on interpretation of evidence that permits widely divergent estimates.

The direct method of dose-response estimation relies on skeletal structure anomalies in the offspring of irradiated mice. This method requires extrapolation of skeletal effect rates to other body systems and then projection of the experimental findings in mice to effects in humans. The method also calls for adjustment by various "correction factors" to compensate for high dose rates and for fractionation and to estimate a total population incidence from experimentation with males alone (Denniston 1982).

3.3.3 The Risk Estimates

There are three major studies of primary relevance to the estimation of radiation-induced genetic disorders, the two reports from the National Academy of Sciences Committees, BEIR I in 1972 and BEIR III in 1980, and one from a United Nations Committee, UNSCEAR, in 1977. Table 3.5 shows these committees' estimates for an average population exposure of 1 rad. Estimates are given both for the first generation following exposure and for equilibrium, which is the level at which, after several generations, the incidence rate would level off and be sustained if there were no further changes in exposure (i.e., a new steady state.)

TABLE 3.5. Estimated Increase in Genetic Disorders per Million Liveborn, from an Average Population Exposure of One Rad

	Current	BEIR 72		UNSCEAR 77		BEIR 80	
Disease Type	Incidence	1st[a]	Eq[a]	1st	Eq	1st	Eq
Dominant and X-linked	10,000	10-100	50-500	20	100	5-65	40-200
Recessives	2,500	slight	very slow increase	slight	slow increase	very few	slow increase
Unbalanced Rearrangements	4,000	12	15	40	40	<10	increase only slightly
Aneuploids		1	1	--	--	0	0
Irregularly Inherited Disorders	90,000	1-100[b]	10-1,000[b]	5	45	--	20-900
Totals	106,500	25-215	75-1,500	65	185	--	60-1,100

(a) First generation; equilibrium.
(b) Used a current incidence of 40,000.
Source: Adapted from Denniston 1982.

Both UNSCEAR 1977 and BEIR 1980 employed the doubling dose method for estimation of single gene effects in equilibrium, and the direct method for first generation estimates. UNSCEAR used the doubling dose method for estimating chromosomal rearrangements, while BEIR 1980 used human and marmoset data for direct estimation (That is the reason for the divergence in the committees' estimates regarding rearrangements.) BEIR 1972 employed the direct method for estimating first generation incidence of induced chromosomal aberration, but used a doubling dose method throughout for gene mutation.

There are additional reasons why the estimates vary. We concentrate here on the differences between UNSCEAR 1977 and BEIR 1980 as reflections of the current state of the art. The UNSCEAR Committee accepted a doubling dose of 100 rad; BEIR 1980 considered it to be in the range of 50-250 rem (Selby 1979). BEIR 1972 had placed it in the range of 20 to 200 rem using the direct method to forecast the effects in the first generation. The committees used different estimates of both the mouse-human relationship and the skeleton-whole body relationship. The UNSCEAR Committee accepted an estimate that about one-half of the dominant mutations found in mice would cause serious disorders if found in humans; the BEIR Committee felt the true range to be from one-quarter to three-quarters of the mouse disorders. The UNSCEAR Committee multiplied the skeletal disorders by five to estimate the whole body effects; BEIR preferred a range from five to 15.

Scientific interpretation causes estimates of increased genetic disorders to vary even though, as Denniston (1982) observes, the UNSCEAR and BEIR Committees have overlapping memberships and they used the same data (p. 332). In order to estimate genetic effects in terms of their clinical manifestations instead of as genetic disorders, a further interpretive step must be taken.

3.3.4 Clinical Manifestations of Genetic Disorders

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 in the calculation of genetic effects: they provide the estimates of current incidence to which the doubling dose is applied. In this study we are interested in the clinical manifestations as final outcomes, as the observable, impact-producing health effects related to radiation-induced genetic damage. It is the effects of inherited disorders, such as blindness, muscular dystrophy, chorea, and kidney disease, that produce costs for society.

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 catalogued 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.

Estimates of the relative frequency of various genetic diseases varies 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. Of the major population studies, Trimble and Doughty's (1974) for British Columbia probably most closely represents the U.S. population. This study could be used as the basis for identifying the relative frequency of genetic diseases with different levels of impact for society. At the present time, information is unavailable as to the frequency of severe genetic diseases relative to those that create little or no cost.

3.3.5 The Reactor Safety Study and CRAC Model

In the Reactor Safety Study (NRC 1975) information from BEIR 1972 is modified to some extent, to estimate the genetic effects of a reactor accident. Instead of documenting each step, we include here a brief discussion of the differences and similarities between the Reactor Safety Study's assertions and those of BEIR 1972 and BEIR 1980:

- In order to make the estimates of genetic effects comparable to the estimates of other health effects, the Reactor Safety Study makes several computational changes from BEIR 1972: effects are calculated per million in the population, not per million liveborn; effects are calculated per rem instead of per 5-rem dose.

- BEIR 1972 employed a doubling dose in the range 20-200 rem, BEIR 1980 in the range of 50-250 rem. The Reactor Safety Study uses a point estimate of 100 rem for the doubling dose.

- The Reactor Safety Study uses the BEIR 1972 range for mutation component of multifactorial disorders: 5 to 50 percent.

In general, the Reactor Safety Study indicates there are reasons to consider the estimates from BEIR 72 to be too high. And, as shown in Table 3.5, BEIR 1980 supports that assessment, lowering very slightly the estimates of the previous BEIR Committee.

Genetic effects have been estimated by the CRAC model, though they are neither included in the CRAC2 version nor documented in the user's manual (Sandia 1981). While the discussion of genetic effects in the Reactor Safety Study indicates an approach to projection based on BEIR 1972, the CRAC model actually uses a simple calculation of 260 genetic effects per million person-rem.[a] This procedure is currently being revised as part of a larger NRC risk modelling effort.

3.3.6 Summary

The level of uncertainty inherent in genetic-related disease projections is very high due to the major information gaps in each stage of the projection

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(a) Conversation with Roger Blond, Division of Risk Analysis, Office of Research, NRC, April 5, 1983.

process. Given the state-of-the-art and the recent rapid expansion of information regarding genetic disease, PNL currently uses the following assumptions to provide a basis for genetic effect cost estimates:

1. Genetic effects are expressed within ten generations.

2. Half of all effects are due to autosomal dominant and half are due to multifactorial genetic disorders and chromosomal damage.(a)

3. Autosomal dominant disorders are eliminated from the population at a rate of 20 percent per generation and multifactorial (and chromosomal) disorders at a rate of 10 percent (NRC 1975, Appendix VI, p. 9-30).

4. Genetic diseases are equally distributed between those types that are most disabling and those that have little or no impact.(b)

Because advances in the state-of-the-art are expected, PNL's cost model (HECOM) has been designed for ease of modification of these assumptions regarding genetic effects incidence.

(a) This is based on the midpoint of the range of uncertainty regarding incidence of multifactorial disorders (NRC 1975, Appendix VI, p. I-11).
(b) This assumption is made in the absence of an empirical information base.

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4.0 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 more morbidity and of excess (i.e., earlier) mortality. People are generally averse to risk: a decrease in risk is considered a good, and to be sought; an increase in risk is a bad, and to be avoided. Concentrating, for simplicity, on the issue of excess mortality, this section provides a discussion of the difficult problem of evaluating (in dollar terms) the cost of an increase in risk.

It is useful to begin the discussion by emphasizing 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 and measuring the risk to the value of human capital. A description of these general approaches is provided in Sections 4.1 and 4.2, along with an analysis of how comprehensive each is in terms of capturing each of the components of the cost of risk.

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 lower societal risk means he himself 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 lower societal risk means his 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 he 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. He would value a lower risk to anonymous others because it means a lower risk to his claim on their net production. Call that v4.

5. He 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.

4.1 THE "HUMAN CAPITAL" APPROACH

Society is willing to forgo current consumption and to invest in productive plant and equipment to an extent that 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.

Employing the human capital approach in practice, 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 lifetime. 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. Now 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.

In computing the cost of risk this approach considers both the increased level of risk and the value of the human capital at risk. This section discusses the several ways in which the value of human capital can be measured. 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 in 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 exclude 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 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, suddenly 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 non-institutionalized, 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 probably understate v1.

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 one 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 have been observed, both in terms of particular methodological troubles 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 changing 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 the 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; Rhoads 1978; Clarke 1979; Dorfman 1979; and Weinstein, Shepard and Pliskin 1980.)

4.2 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 on balance, some worse off on balance, and others will be indifferent to the project. For example, an investment in safety equipment may decrease public risk but require increased worker exposures. If in the aggregate the total 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 the battle 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 non-linear, 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 estimate willingness-to-pay. 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. 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 4.1 provides a summary of the value components measured by each of the approaches assessed in this section. As can be seen in Table 4.1, none of the approaches quantifies adequately all of the components of risk value.

TABLE 4.1. Extent to Which Selected Methods Measure the Various Components of Value

Methods	v1	v2	v3	v4	v5
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.

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.

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.

4.3 CONCLUSION

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 in order 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 $\underline{v4}$ and $\underline{v5}$, respectively, to provide a baseline.

To gain an understanding of the magnitude of the value of risk reduction with minimum investment, we have adopted the human capital approach 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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5.0 ESTIMATION OF THE DIRECT COSTS OF HEALTH EFFECTS

If one measures the values of life and livelihood by the human capital approach, an additional 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, cancers, and genetic effects.

5.1 DIRECT COSTS OF RADIATION INJURIES

Depending on dose levels and on individual sensitivities, exposure to significant amounts of radiation may result almost immediately in acute symptoms that could range from 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. Cases of acute radiation syndrome have occurred too infrequently to result in the development of information regarding treatment practices and costs. However, specialists in radiation medicine have reached relatively close agreement about the clinical manifestations of radiation illness. We estimate the costs of treatment from information on the cost of treating patients with similar clinical problems. For this analysis, radiation injuries are grouped into three categories: 1) prodromal symptoms, which last only a few days; 2) bone marrow syndrome, gastrointestinal syndrome, and pulmonary impairment, which are all potentially life-threatening; and 3) in-utero effects, which cause severe and permanent impairment to the irradiated fetus.

5.1.1 Prodromal Symptoms

Prodromal symptoms, consisting of nausea, vomiting, and diarrhea may occur within a few hours of whole-body exposures over about 50 rads and may continue for a few days. Andrews (1980) suggests that individuals displaying prodromal symptoms should be kept at home, partly to avoid the infectious environment of a hospital and partly to avoid undue apprehension. However, because closely monitoring prodromal symptoms is the only 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. In 1981 the average total hospital charge for inpatient services was approximately $300 per day (Health Care Financing Administration, June 1982). If physicians' fees average some one-third of hospital charges, as they do for cancer patients (Scotto and Chiazze 1976), then they will total another $100 for each day of care. We assume a 2.5-day stay in the hospital, resulting in an estimate of about $1,000 per case of prodromal symptoms. While provision of such high-quality care may be unlikely in the event of a major accident, 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 150 to 175 rads (NRC 1975, Appendix VI, F-1).

5.1.2 Bone Marrow Syndrome

Bone marrow syndrome is characterized by impairment of the blood forming system; depending on the extent of damage, the clinical manifestations include severe susceptibility to infection, hemorrhage and anemia. 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. In both cases the most immediate concern is the threat of infection. In addition, patients suffering from either face a threat of severe hemorrhage.

To control infection, burn patients are placed in reverse sterile isolation, usually employing special air filtration systems and sterilizing everything that comes into the room. Because of all these special precautions, a regional burn care center charges $1255 per day for "room and board" alone.[a] That is the cost for nonsurgical burn patients; those requiring surgery receive additional precautionary measures, and pay up to $2,000 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 for about 3 weeks for those patients with bone marrow syndrome.

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.[a] 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. That aggressive approach to

(a) Communication with staff at Harborview (Seattle) Medical Center's burn care unit March 1983.

medication is probably not very different from that followed for a burn patient, so we include the full $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 bone marrow syndrome patient (see Andrews 1980 and Saenger 1982). The daily costs could easily reach levels similar to those of a burn patient. So we add $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, in case of hemorrhage in severe cases. We add another $20 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 services 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 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. This does not include the cost of a bone marrow transplant, which is often recommended for patients with severe bone marrow syndrome, especially for those who have received a probable fatal dose (Blakely 1968; Dalrymple 1973; NRC 1975; Saenger 1982). The cost of a bone marrow transplant is approximately $70,000.(a) We have not included bone marrow transplant as a likely form of treatment because of the difficulties of finding a compatible donor for most patients, a problem that may be more difficult in the aftermath of a reactor accident. In addition, at least one researcher (Andrews 1980) advises that marrow transplant may not be helpful. Although bone marrow syndrome is not the most severe manifestation of acute radiation injury, it is probably the most costly, since other severe forms are almost certain to end in death before large amounts of medical resources can be used.

5.1.3 Gastrointestinal Syndrome

Symptoms of gastrointestinal syndrome include severe diarrhea and vomiting. Patients are likely to die within two weeks of the onset of these symptoms. There is some chance that treatment involving replacement of fluids and electrolytes may assist the patient to recover from the associated symptoms. However, a radiation dose high enough to cause gastrointestinal injury is also

(a) Communication with staff at the Fred Hutchinson Cancer Research Center, Seattle, March 1983.

probably high enough to damage the bone marrow; a patient surviving the former will almost surely suffer the latter. For that reason, we consider it plausible that patients with gastrointestinal injury will be treated from the start with infection-preventing measures similar to the treatment given bone marrow patients. However, since they are likely to die within two weeks, we apply to these patients a treatment cost for only two weeks: $28,000.

5.1.4 Pulmonary Impairment

Symptoms of pulmonary injury include pneumonitis and pulmonary fibrosis. We assume that (in the absence of bone marrow syndrome) these symptoms could be treated in an average hospital room at the average 1981 charge of $300 per day. At 33 percent of hospital charges, physicians' fees may add another $100 per day. Thus, pulmonary impairment may cost some $400 per day for all hospital and medical services. In 1977 the average length of stay in acute-care hospitals was 8.0 days for pneumonia, and 9.8 days for emphysema (National Center for Health Statistics 1982). Lacking similar statistics for radiation-induced pulmonary complications, we average the data for those similar diseases and assume a nine-day length of stay. That leads to a total cost for pulmonary impairment of approximately $3600.

5.1.5 In-Utero Injury

Cost estimates for direct care of individuals with congenital defects, similar in effect to the retardation and nervous system anomalies induced by in-utero radiation injury, are applied to all in-utero injuries. Two studies provide estimates of the present value of streams of costs that can be incurred in the care of Down's Syndrome (Conley and Milunsky 1975) and spina bifida (Layde, Allmen and Oakley 1979). The studies' cost estimates are $116,000 and $86,500, respectively, in 1981 dollars. We are currently using a rough average of those estimates, $100,000, as the cost of an in-utero injury.

In summary, the resulting cost estimates are used in the HECOM Model base case for different manifestations of radiation injury:

TABLE 5.1. Radiation Injury Cost Estimates (1981 $)

Prodromal	1,000	
Bone marrow syndrome	56,000	(a mean value)
Gastrointestinal injury	28,000	
Pulmonary injury	3,600	
In-utero injury	100,000	

5.2 DIRECT COSTS OF CANCERS

Two different perspectives have been employed in the past in measuring the direct costs of treatment for selected diseases including cancers: prevalence and incidence. The prevalence approach asks, conceptually, "What is (for example) cancer of the cervix costing the nation this year in terms of direct

outlays for treatment? It is this approach that has been followed by Rice and her associates (Rice 1966; Cooper and Rice 1976; Hodgson and Rice 1982). The prevalence approach is well-suited to an aggregate or "top down" 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.

In practice, the treatment regimens used in the two principal studies of costs of cancer incidence (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), compute current costs by inflating TNCS costs to current dollars (with a few adjustments).

Cromwell and Hartunian provide the only incidence-based measures of direct cancer 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, for example, Scitovsky and McCall (1976), Kodlin (1972), and Schneider and Twiggs (1972). Unfortunately, those studies concentrate typically on patients with specific cancers, and are unrepresentative of treatment regimens and costs for a broad range of cancer types.

5.2.1 Cancer Cost Data

Because the TNCS 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. 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 both on the services received by each patient and on 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) report hospital charges as contained in the hospital records sample of the TNCS. Cromwell et al. (1976) uses 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. Among 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 by cancer type.

Nevertheless because the PIB is also the source for other treatment costs, Abt (1975) records hospital costs as on the PIB. In comparison, Hartunian, Smart and Thompson (1981) use data from Scotto and Chiazze (1976) to measure hospital costs. Then they use the ratio of hospital costs to other service costs, as reported on the PIB, in order to estimate the cost of all nonhospital services.

In addition to the details 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 those with the more aggressive cancers, and interviewees exhibited selective memory.

A final 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. Ideally, 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.

5.2.2 Cost Estimation Methodology

The direct costs of cancer include all of the costs of hospitalization, physicians' care, drugs, nursing, special equipment, transportation, radiation treatments, chemotherapy, etc. Disaggregate data from the TNCS 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.

Nursing home - includes daily room charges, nursing costs, and supplies.

Private nurse - costs of in-hospital private nursing, billed separately.

In-home nursing - includes nursing and supply costs.

Drugs - includes everything from prescription drugs used in chemotherapy to over-the-counter medications.

Rehabilitation - includes physical therapy, special equipment, and prosthetics.

These direct cost components are then used to construct a direct cost estimate for each CRAC2 cancer category. Since the TNCS data are categorized partially by cancer type and partially by cancer site, these categories are combined to correspond with the CRAC2 output as shown in Table 5.2. For the CRAC2 categories of "gastrointestinal tract" and "other," the TNCS data are aggregated using the proportional incidence of the major types or sites of cancers as weights for the costs. Those cancers constituting less than five percent of the total incidence are not included. Thus, based on the distribution in Table 5.2, the cost estimate for gastrointestinal cancers is a weighted average of the costs for the seven major types of cancers falling within that category.

TABLE 5.2. Corresponding Cancer Categories in CRAC2 and the Third National Cancer Survey

CRAC2 Category	Third National Cancer Survey Category[a]	Percent[b]
Leukemia	Leukemias	--
Lung	Lung	--
Breast	Breast	--
Bone	Bone	--
Gastrointestinal	Colon	34%
	Bladder	14%
	Rectum	11%
	Pancreas	11%
	Stomach	11%
	Oral cavity	12%
	Kidney	7%
Other	Larynx	44%
	Cervix	10%
	Uterine corpus	11%
	Prostate	26%
	Lymphomas	10%

(a) The table excludes TNCS categories that constitute fewer than 5 percent of the corresponding CRAC2 cases.
(b) TNCS category (by site) shown as a percent of the corresponding, broader CRAC2 category.

An estimate of each cost component, such as "hospital/inpatient," is then calculated for each of the CRAC2 cancer categories, using the proportional weighting for TNCS categories described in Table 5.2 for gastrointestinal and "other" cancers. These cost estimates, representing first and second year

treatment costs (Cromwell 1976, p. 70) for the eight categories of direct costs, are shown in Table 5.3. 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. Shown along with each cost estimate is the percentage of patients surveyed who incurred this type of cost. These percentages are applied to each cost category to calculate the weighted total cost shown in the last column for each type of cancer. In calculating benign thyroid nodule costs the base case assumes that 75 percent of the benign nodules are diagnosed without surgery and that only outpatient costs are incurred in these cases.(a)

The weighted total of cancer care costs is converted to 1981 dollars using the hospital room and medical care cost components of the Consumer Price Index (CPI). Once the direct cost estimates are calculated in this form, they are used with the CRAC2 health effects projections to calculate the total direct cost of cancer care over time. CRAC2 health effects estimates, which except for thyroid are for fatalities only, are converted to incidence estimates by application of the ratios shown in Column 2 of Table 5.4. Since the thyroid health effects estimate produced by CRAC2 reflects incidence (NRC 1975, Appendix VI, p. 9-27), it needs only to be partitioned between benign and malignant cases. The resulting estimate of thyroid cancer (and benign thyroid nodule) incidence can then be allocated across age groups and time to calculate total direct cost due to exposure.

Since the cancers would not occur immediately after radiation exposure but would generally have minimum latency periods of from two to 15 years, the direct costs must be discounted to a present value estimate. This is accomplished by discounting the costs that are projected to occur over the remaining lifetime of the exposed population. First, cancer incidence is allocated to age groups in proportion to the size of each age group in the exposed population and the relative risks for people in each age and sex category. Members of each age group are then assigned a probability of developing cancer in each year after the minimum latency period until they reach the maximum age considered.

The preliminary cost estimates shown in 1970 dollars in Table 5.3 have been inflated to 1981 dollars using the appropriate components of the CPI. Table 5.5 presents the resulting PNL estimates for each CRAC2 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 1981 dollars to any other year's dollars using the medical care cost component of the CPI (see Table A.13).

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

TABLE 5.3. Direct Costs of Cancer Care for First Two Years of Treatment by Cancer Type (1970 $)

CRAC2 Categories	Hospital/ Inpatient[a]	% [b]	Outpatient/ Doctor	%	Nursing Home	%	Private Nurse	%	In-Home Nursing	%	Drugs	%	Rehabili-tation	%	Other	%	Weighted Total
Leukemia	3,914	100	881	100	1,421	13	317	7	677	10	198	68	186	18	106	70	5,312
Lung	3,905	100	1,376	100	1,369	12	608	6	572	17	134	75	122	36	67	81	5,814
Breast	1,745	100	996	100	3,272	8	458	10	464	5	104	71	50	100	47	69	3,228
Bone	7,908	100	2,041	100		--		--	200	19	181	90	2,395	100	132	100	12,677
Gastro-intestinal	3,140	100	1,138	100	1,067	5	694	6	990	14	120	73	115	15	81	30	4,822
Other	2,366	100	1,132	100	1,144	9	266	6	941	4	138	100	81	14	72	53	3,842
Thyroid-benign	1,516	25	1,108	100		--		--		--		--		--		--	1,487
Thyroid-malignant	1,516	100	1,108	100	1,250	5	234	21	187	14	81	100	153	24	34	100	2,914

Source: Cromwell, J., et al. 1976. The Measurement of the Cost of Cancer Care. Abt Report No. 76-152; Abt Associates, Inc, and Boston University Cancer Research Center, Cambridge, Mass. Hospital and Outpatient/Doctor costs are from Table 3.4, p. 60; all other costs are from Table 3.3, pp. 58-59.

(a) Hospital costs are increased by 20 percent to reflect uncollected charges. According to Scotto and Chiazze (1976) an investigation of selected survey cases showed that 20 percent of actual hospital charges were not reflected in the Third National Cancer Survey data since they were not paid by the patient, private insurance, Medicare, or Medicaid.

(b) Percentages represent the proportion of patients with a given type cancer, who receive each type of service. For each type of service, patient totals are adjusted for missing data, as suggested by Cromwell et al.

TABLE 5.4. Calculation of Cancer Incidence Based on CRAC2 Fatality Estimates

Health Effect	CRAC 2 Estimates	X	Incidence to Fatality Ratio	=	Health Effect Incidence
	Fatalities				
Leukemia	X_1		1.00		Y_1
Lung	X_2		1.00		Y_2
Breast	X_3		2.00		Y_3
Bone	X_4		1.25		Y_4
Gastrointestinal	X_5		1.20		Y_5
Other	X_6		2.00		Y_6
	Incidence				
Thyroid-benign[a]	X_7		0.6[b]		Y_7
Thyroid-malignant[a]	X_7		0.4[b]		Y_8

Source: U.S. Nuclear Regulatory Commission. 1975, Appendix VI, pp. G18-G23.

(a) CRAC2 provides a single incidence estimate for all thyroid effects.

(b) Proportion of nodules that are benign or are malignant.

TABLE 5.5. Direct Costs of Cancer Care by Cancer Type (1981$)[a]

CRAC2 Categories	Weighted Total Cost
Leukemia	16,300
Lung	17,400
Breast	9,400
Bone	37,600
Gastrointestinal	14,000
Other	11,400
Thyroid-benign	7,700
Thyroid-malignant	8,400

Source: Table 5.3 and Consumer Price Index inflators from the US Bureau of Labor Statistics. (Monthly)

(a) To convert from 1970 to 1981 dollars, hospital costs are inflated using the hospital room cost component of the CPI; all others are inflated by the all medical care cost component.

5.3 DIRECT COSTS OF 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 preliminary cost estimate.

In Section 4.0 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's 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 measures is similar, albeit more difficult to see. 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? That is, we (this generation) are not the ones at risk from genetic disease and we need not bear the cost of those health effects 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; this generation 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 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. (Choice of the discount rate for intergenerational valuations is a methodological issue in itself which we address briefly in this section.)

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. The remainder of this section presents an approach to estimating the direct costs of genetic disease. The associated indirect costs are examined in Chapter 6.

5.3.1 Genetic Effects Cost Data

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

Hall et al. (1978) present data on the hospital treatment at one urban medical center of children with genetic disease. That study reports an average cost per hospital admission of approximately $1100 (1981 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 at the time 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 $5830 (1981 dollars). Of course, there is no reason to surmise that the end of the study coincided with the end of hospitalizations for the children sampled, so $5830 is doubtless an underestimate of the average total hospital costs.

Assuming that physicians' fees average approximately one-third of hospital costs, as is the experience with cancer patients (Scotto and Chiazze 1976), the average total costs for acute care of childhood-onset genetic diseases may be as low as $7775 (1981 dollars), but are most likely higher because of multiple hospitalizations. We treat the total acute care costs as if they were incurred in the first year of life.

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 aberrations and would account for a very 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 institutionalizations of other genetically impaired individuals. Assuming the costs are representative, for an individual born in 1981 and institutionalized for the next 70 years the cost would be approximately $14,000 annually in 1981 dollars (inflating Conley and Milunsky's 1972 estimates by the medical care component of the CPI). 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. We subtract that amount so that only the incremental costs of illness are considered, $11,600 per year. These costs are distributed over the person's lifetime.

5.3.2 Cost Estimation Methodology

The genetic effects associated with increases in radiation exposure may range in severity from color blindness to mortal or debilitating diseases. Obviously the costs vary as well.

The two studies cited in the previous section result in rough estimates of $8,000 for acute care and $11,600 per year for long-term care for individuals affected by severe genetic diseases. The problem is to determine what proportion of genetic effect result in costs of this magnitude.

McKusick (1978) lists 736 "diseases" that can be traced to autosomal dominant genetic defects. Along with X-linked defects, the autosomal dominant would be a major category of genetic effects likely to result from radiation exposure. Some of those "diseases" cause little or no symptomatic problems; others are life-threatening or totally debilitating. The genetic effect may be obvious at birth in some cases and disability onset may occur in adulthood in others. Unfortunately, we are unaware of any studies that provide the frequency of genetic effects classified by severity. Lacking any information as to the frequency of genetic effects with no economic costs relative to those resulting in maximum cost, we assume, for current working purposes, that the median point in the range is representative. To implement this assumption we treat half of the genetic effects as resulting in maximum costs and half as resulting in no treatment or institutionalization cost.

The resulting estimated lifetime costs for treatment are discounted to present value at the time of each affected individual's birth. We allocate those births over 10 generations after the hypothetical reactor accident. The defects projected for the first generation are allotted to the first 30 years, the second 30 years for the second generation, and so forth. Occurrences of genetically impaired births projected for the first generation are distributed evenly over the first 30 years post-accident. The number of affected births projected for the second generation are distributed evenly over the years 30 through 59 and so forth.

After applying the estimates of average lifetime costs to each birth distributed over the appropriate generations, it is necessary to discount those costs 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 Section 7.4.

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6.0 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 4.0 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 Section 4.0). 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 Section 4.0).

The value of lost earnings should be considered 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 lost production experienced in addition by family and friends who take time out to care for the stricken individual.

There is another way in which the use of earnings often underrepresents the full indirect costs: earnings data do not reflect the value of services performed in the home. In this study we avoid that shortcoming by two steps. First, we consider the population incurring indirect costs 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 non-institutionalized 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. (The method treats all males equally as well, although it does not treat men and women equally.)

The following sections relate how lost earnings measures 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 due to mental or physical impairment as a result of prenatal injury or genetic defect, and permanent cessation of work due to early mortality. In this study, we explicitly calculate costs related to all those causes except those due to

illness-related reduction in capabilities; the average earnings data used implicitly reflect a low rate of handicaps among workers.

6.1 INDIRECT COSTS OF MORBIDITY

Lost production during illness is estimated based on weeks of missed work for each type of illness. The value of that loss is measured by average earnings, for individuals of a particular age and sex, in each post-exposure time period. The incidence of illness is assumed to fall across age and sex cohorts in proportion to age- and sex-related risks of radiation-induced illness and to each cohort's relative numbers in the exposed population. The estimate takes into account the individual's age at the time of illness and also accounts for the fact that normal probabilities of death lead to an expectation that some exposed individuals would die of other causes before latent cancer can result in any lost production.

For cancers, we apply an estimate of lost work ranging from about 6 weeks to more than 23 weeks depending on the cancer type. (See Section A.2.4.). Among the cases of radiation injury, prodromal symptoms are assumed to cause one lost week of work; all other types of radiation sickness are assumed to result in a loss of six months of work. Individuals disabled by growth impairments and mental retardation resulting from prenatal exposure to more than 200 rem are assumed to suffer a 100 percent income loss, beginning at the age of 15 and continuing over the person's expected lifetime. Among individuals afflicted with genetic defects, we currently assume 50 percent to suffer a 100 percent income loss similar to those injured in utero. The remaining 50 percent are currently assumed either to have no handicap as a result of genetic disease or are considerd to have been successfully treated before age 15.

The model considers the incidence of genetic effects through ten generations (300 years). The indirect costs of genetic effects are calculated in a manner similar to those for premature mortality due to illness. A review of the literature, unfortunately, does not disclose any estimate of the rate at which productivity impairment results from genetic effects. We currently assume that one-half of the individuals experiencing genetic effects will never be productively employed and that the remainder have no impairments. Applying this assumption, the expected earnings of each age cohort (given normal mortality probabilities) provide the basis for estimating the stream of potential indirect costs for a genetically damaged individual born in each year after population exposure. The rate at which such individuals are born is calculated as it is for the direct costs, allocating first generation effects equally across the first 30 years post exposure, the second generation effects across the next 30 years, and so forth. The resulting indirect cost streams are then discounted and summed to the present value at the time of population exposure.

For all types of health effects, the indirect cost of morbidity is estimated from the amount of work lost, valued by expected earnings. These costs are computed for the specific age and sex cohorts in the population and the

time period in which they would face health effects risks. To apply those costs to the year of exposure, the projected stream of future costs is discounted to present value as of the year of a hypothetical reactor accident.

6.2 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. 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. For those who suffer fatalities from acute injury, the fatality is assumed to come in the first year after exposure. Thus, expected losses begin in the year of the accident and extend out 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 losses 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. CRAC2 estimates of cancer fatalities are assigned to age and sex cohorts in proportion to their risks of radiation-induced cancers and relative numbers in the population. Each type of cancer has a specific minimum latency period (see Section A.2.2) between exposure and the onset of cancer symptoms. After the latency period has passed, individuals are expected to show signs of cancer and to die from those cancers over a time period distributed over what would have been their normal lifetime. 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. Thus, cancer fatalities are treated as having an equal probability of occurring in each year after the latency period and continuing for a normal life span.

For example, CRAC2 may project that two persons in the 30-year-old age group will contract a fatal bone cancer. After a 10-year minimum latency period between exposure and bone cancer symptoms, 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 loss that would occur 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 individual.

7.0 HECOM STRUCTURE AND DEVELOPMENT

This section provides a conceptual overview of the Health Effects Cost Model (HECOM) structure and processes. A more detailed, user-oriented discussion of the data base, subroutines and processes is contained in Appendix A and the computer code is listed in Appendix B.

In Section 7.1, the general approach used in developing HECOM is discussed. Aspects of the model's flexibility and the treatment of future cost streams in present-value, real terms are emphasized. An overview of the HECOM structure is then provided in Section 7.2. This is followed in Section 7.3 by a discussion of the steps required to modify CRAC2 output for use as input to HECOM. The sensitivity of HECOM to both input data and parameters has been examined and this analysis is presented in Section 7.4.

7.1 MODELING APPROACH

The general approach employed in developing HECOM was dictated by the need to develop a flexible model that could be easily updated or modified. The ways in which this flexibility have been achieved are discussed in Section 7.1.1 and the method used to discount future cost streams is explained in Section 7.1.2.

HECOM is a probabilistic model designed for analysis of changes in population health risks. The cost estimates calculated by the model are dependent on population distribution by age and sex, cohort survival probabilities, excess health effects risk estimates by cohort, and probabilistic distributions of incidence over time. As a result of this approach, HECOM can project the societal impacts of health effects for which timing and population incidence are indeterminate.

The cost estimates calculated 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.[a] Detail of the discounting method employed is provided in Section 7.1.2.

7.1.1 Flexibility of HECOM

HECOM has been designed to be as flexible as possible, subject to the limitations imposed by the computer code used to develop the model. This

(a) To analyze the consequences of exposures in years after the base year of analysis, costs and wages can be escalated to the level of the year of exposure before being input to HECOM. HECOM cost-estimate output can then be discounted back to the base year.

flexibility enables the model to use input data in several different forms and to easily calculate cost estimates for a variety of population exposure scenarios. Flexibility has been achieved through the model's modular construction, through user-specified control parameters, input data files that can be easily modified, the use of real costs and growth rates, and the ability of the model to aggregate and report costs in a variety of ways.

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 study the costs of treating radiation-induced cancers. The model's modular construction enables him to skip the calculation of radiation injury and genetic effect treatment costs, as well as the calculation of indirect costs. Only those steps essential to calculating the direct cost of cancers must be performed and only the data essential for performing these calculations is needed.

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 and 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 in addition to a point estimate.

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

Costs calculated by HECOM are stored in the lowest level of aggregation possible. This enables the model (with minor algorithm modifications) to aggregate costs in a variety of ways. For example, health effects costs could be aggregated and reported by age cohort, type of illness, year of occurrence and sex depending on the specific needs of the user.

7.1.2 Treatment of Costs Over Time

The effects of radiation exposure are long-term, with both direct and indirect costs occurring over the lifetime of the affected population and succeeding generations. To evaluate the merits of various measures that affect health effects risks, the cost stream must be reduced to a single current dollar estimate for the base year, the year in which action would be taken. This is accomplished by discounting the costs expected in each future year back to the base year. A present value estimate of both direct and indirect costs of health effects projected in future years is calculated using the following basic approach:

$$\text{Present Value of Costs} = \sum_{n=1}^{A} \frac{EC(a-j+1)_{a,s}}{(1 + R/100)^{n-1}}$$

where

a = ages of the affected individual, from 0 to maximum (A) considered

j = age at onset of morbidity or mortality

$EC(a-j+1)_{a,s}$ = expected cost in current dollars for the (a-j+1) year after morbidity onset (given direct or indirect cost levels, real escalation rates and estimated survival probabilities) for an individual of age a and sex s

s = sex

R = real discount rate (in integer form)

n = year after population exposure.

The real discount rate used is an input parameter, thus facilitating sensitivity analyses.

The time dimension of potential health effects also necessitates accommodation of changes in the levels of direct and indirect costs relative to the general rate of inflation, that is, changes in the real value of treatment costs and productivity losses. This is handled by specifying the real escalation rates for treatment costs and productivity losses as input parameters. Expected costs of morbidity or mortality occurring in any given year are projected as follows:

$$EC(n)_{a,s} = C_{a,s} \cdot P(a+1)_{a,s} \cdot (1+E)^{n-1}$$

where

$EC(n)_{a,s}$ = expected cost, or loss, in year (n) for an individual of age a and sex s

$C_{a,s}$ = average cost, or loss, for an individual of age a and sex s

$P(a+1)_{a,s}$ = probability that an individual of age a and sex s would normally survive to age a+1

E = real cost escalation rate.

7.2 OVERVIEW OF HECOM STRUCTURE

The algorithm developed to estimate the direct and indirect costs of health effects is described in this section. Figures that identify major computational processes and the types of data used to carry them out are provided to present a conceptual overview of the algorithm. Figure 7.1 shows the relationships among the major algorithm processes. Each box represents a process and each line represents a flow of information. The remaining figures describe the individual processes shown in Figure 7.1 in more detail. Since the same processes appear in various figures, they are always shown in the same position on the page.

7.2.1 Major HECOM Processes

Health effects costs are calculated for five cost components: direct costs of cancer, radiation injuries and genetic effects, and indirect costs of illness and fatalities. These cost calculations are represented by the five boxes in the middle row of Figure 7.1. Four intermediate processes are necessary to calculate these health effects costs: projection of genetic effect incidence, of cohort survival probabilities, of labor value over time and of fatalities over time. These intermediate processes are represented in Figure 7.1 by the four boxes in the top row. The final step in the algorithm is to aggregate direct and indirect cost estimates into a form usable for analysis. This step is represented by the bottom box in Figure 7.1. The number of the figure which provides detail on each process is shown in parenthesis in each box.

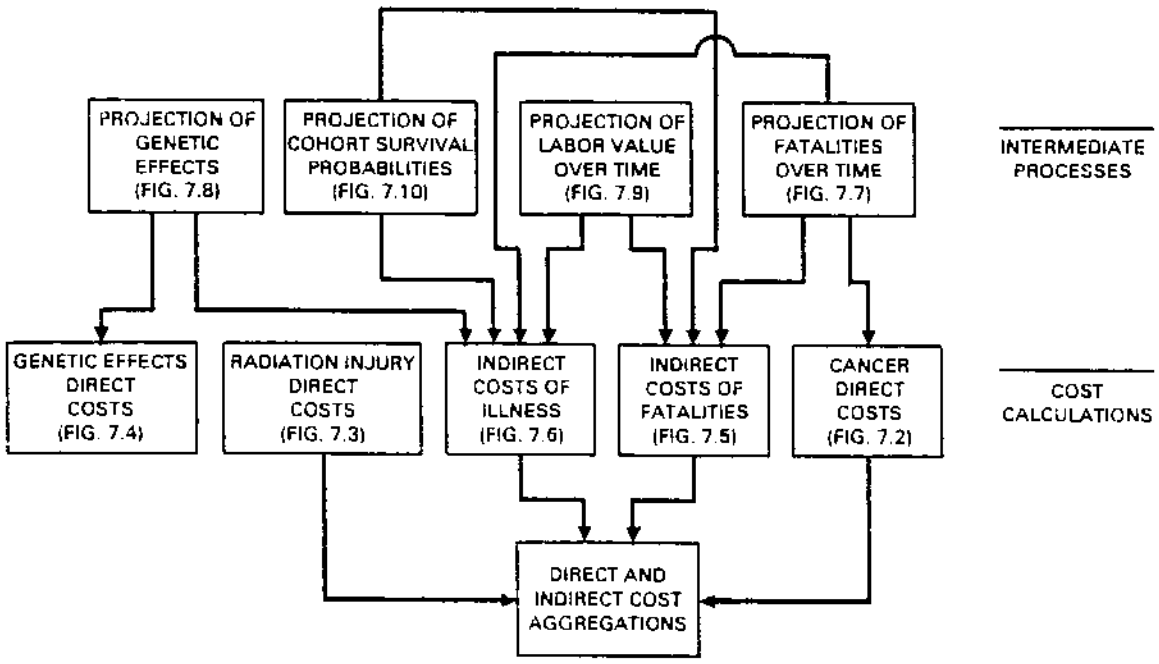

FIGURE 7.1. Overview of Health Effects Cost Model Processes

The cost calculations shown in the middle row of Figure 7.1 each represent a component of the total costs of health effects. Direct cancer costs are shown in the righthand box of the middle row. These are the costs of providing medical care to affected individuals at the point when the cancer develops and is diagnosed. While this cost component is referred to as cancer direct costs, it also includes the cost of treating benign thyroid nodules. CRAC2 fatality projections for other cancers are converted to incidence by HECOM. Since the costs of treating cancer vary with age and sex, due to differing mortality probabilities, the model is designed to calculate direct costs by age, sex and cancer type. The process is described in Section 7.2.2.

Radiation injury direct costs, shown in the second box from the left, consist of the costs of providing medical care to persons with bone marrow, gastrointestinal, or pulmonary injuries or with prodromal symptoms. Costs of providing care to persons with growth and mental retardation due to prenatal exposure are also included. The calculational process is described in Section 7.2.3.

Direct costs of genetic effects are shown on the far left. The calculation of these costs is explained in Section 7.2.4. While costs of caring for persons with genetic effects may stretch into the indefinite future, the costs are calculated as though all future effects would occur within the first ten generations after population exposure. Direct costs for the portion of individuals assumed to be disabled by genetic effects include both acute medical care and institutional care costs.

Indirect costs of fatalities are covered by the second box from the right. While these indirect costs should include the value of all of an individual's productive activities, earnings data are presently being used in the HECOM base case, with only a partial correction for nonwage-earning labor. The indirect costs of fatalities depend on the sex and age of the deceased as well as other factors such as the rate of labor productivity increase over time. The computational elements and general process for calculating indirect costs of fatalities are presented in Section 7.2.5.

The indirect costs of illness, shown in the center box, are similar to the indirect costs of fatalities except that generally they are of shorter duration. There is an exception in the case of prenatal injuries, which are assumed to prevent productive employment over the individual's lifetime. Indirect costs are calculated for the total incidence of cancers, rather than just the cancer fatalities projected by CRAC2. They also include losses during the period of illness for those with radiation injuries. The calculation procedure is explained in Section 7.2.6.

The top row of Figure 7.1 shows the major processes that prepare the input data for use in the cost calculations. On the right-hand side, the projection of fatalities over time involves the calculation of cancer fatality probabilities in each subsequent year for each age and sex cohort depending on its remaining expected lifetime. Based on these probabilities, the cancer fatality

incidence from CRAC2 is distributed over time. Acute fatalities are assigned to age and sex cohorts in proportion to their fraction of the population and are treated as occurring in the base year. Additional details of the procedure are given in Section 7.2.7.

On the far left is a box representing the projection of genetic effects over time. Procedures used to allocate genetic effects are explained in Section 7.2.8. Different types of genetic damage are treated as being eliminated from the population at different rates across generations. The genetic effects allocated to each succeeding generation, however, are treated as having an equal probability of occurrence in each year of the 30-year generational period.

The projection of labor value over time is shown to right of center. A full description of the process is provided in Section 7.2.9. It is based on data for the median income of any specified number of median age categories. When five-year age intervals are used, the cohorts are "aged" through successive median age and income levels with labor value changing at some real rate over time.

To the left of center is the box representing the projection of cohort survival probabilities. This process is discussed in Section 7.2.10. Annual survival probabilities by sex and age are used to develop the cumulative survival probability for each cohort as of the base year. These estimates are then applied to future labor value to calculate probable earnings in each year for each cohort.

7.2.2 Calculation of Cancer Direct Costs

Cancer direct costs are composed of the cost of treating cancers induced by radiation exposure. The information used to perform this calculation is shown in Figure 7.2. To calculate total cancer direct costs by cancer type and sex, data from the intermediate process (which projects fatalities over time) are combined with data on the real treatment cost escalation rate, the discount rate, cancer treatment costs by cancer type, cancer incidence per fatality and duration of treatment.

The cost of treating each type of cancer in each subsequent year is determined using base year treatment costs and the treatment cost escalation rate. Incidence of cancer in each year after exposure is based on projected fatalities by cancer type and the ratio of cancer incidence to fatalities for each type of cancer. With this information, direct cancer costs are determined for each year. These costs are then discounted back to the base year, using the discount rate. In the final process these data are aggregated to totals by sex and type of cancer.

7.2.3 Calculation of the 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 7.3.

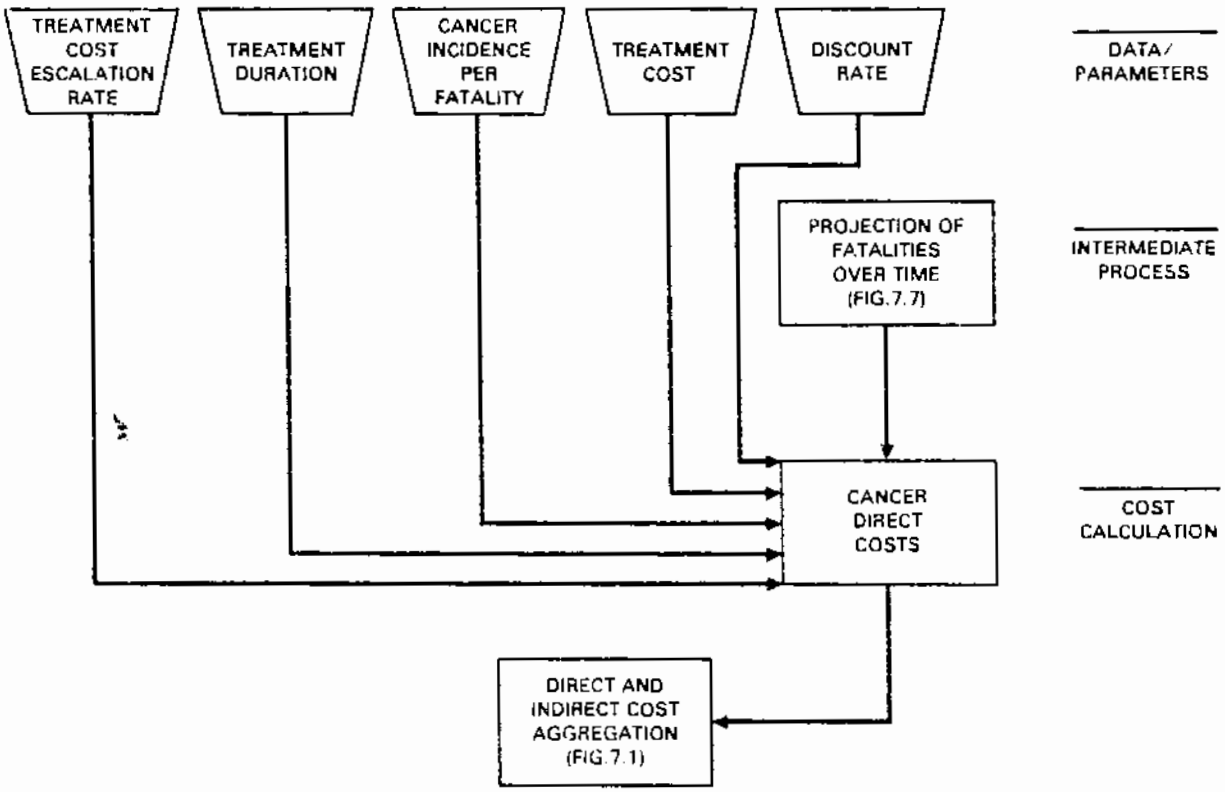

FIGURE 7.2. HECOM Calculation of the Direct Costs of Cancers, by Sex and Cancer Type

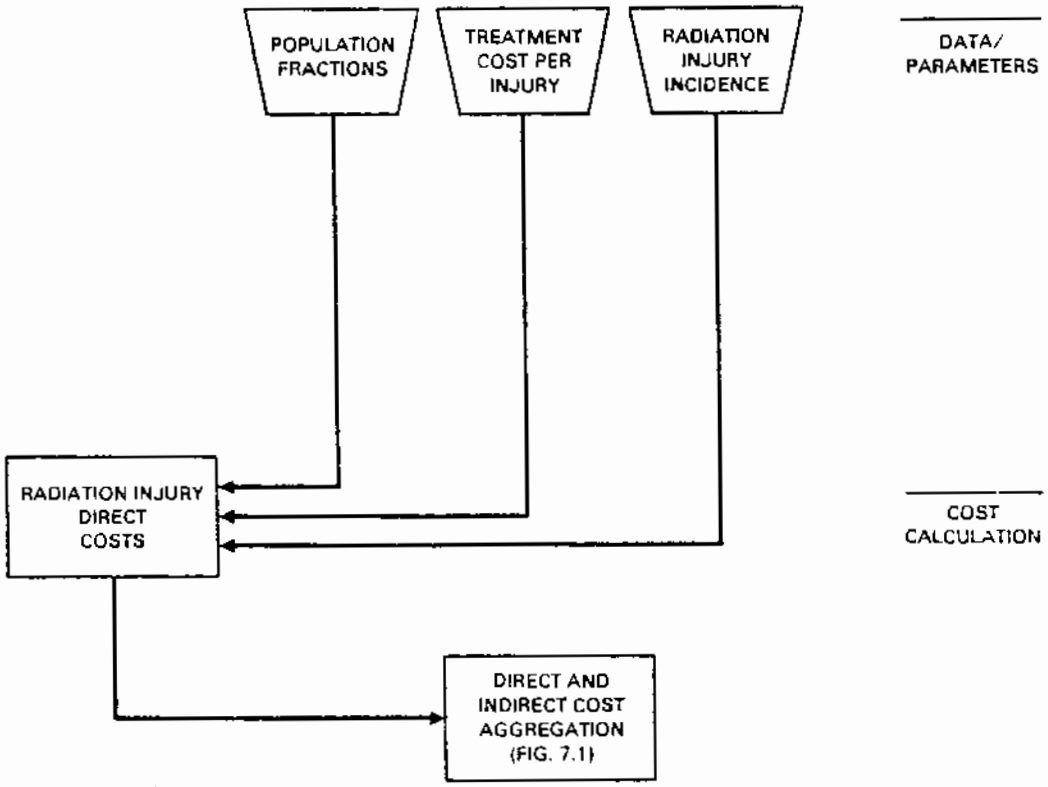

FIGURE 7.3. HECOM Calculation of the Direct Costs of Radiation Injuries, by Sex and Injury Type

These costs are based on data for the fraction of the population in each age cohort, treatment costs by injury type, and radiation injury incidence projected by CRAC2. Direct radiation injury costs are assumed to occur only in the first year. Radiation injury incidence, allocated according to each cohort's relative size in the population, is combined with the treatment cost for each injury to estimate treatment costs by injury type. Finally, direct costs by sex and injury type are calculated.

7.2.4. Calculation of the Direct Costs of Genetic Effects

Direct costs of genetic effects consist of the cost of treating persons born with severe genetic defects and institutionalizing them over their lifetime. Inputs to the process include the number of persons requiring care in each year, the cost of treatment and institutionalization, the discount rate and the rate of treatment cost escalation.

Direct costs are calculated as the sum of lifetime expected institutionalization and treatment costs for each person born with a severe genetic defect. Expected institutionalization and treatment costs are based on cohort survival probabilities and the real costs of treatment and institutionalization in each year an individual is incapacitated. These costs are all discounted back to the base year using the discount rate. An overview of the process is provided in Figure 7.4.

7.2.5 Calculation of Indirect Costs of Fatalities

Indirect costs of fatalities represent the value of labor lost to society because of premature death. The flow of information used to perform this calculation is pictured in Figure 7.5. Data from intermediate projections of cohort survival probabilities, of labor value over time, and of fatalities by age cohort, type of death and sex are used to calculate indirect costs.

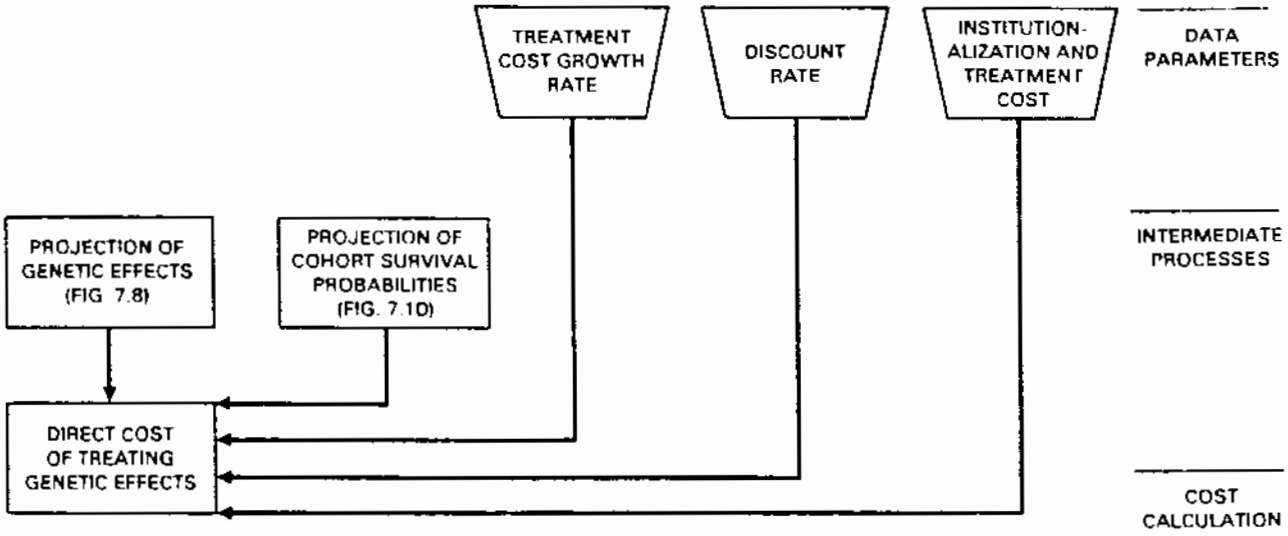

FIGURE 7.4. HECDM Calculation of the Direct Costs of Genetic Effects

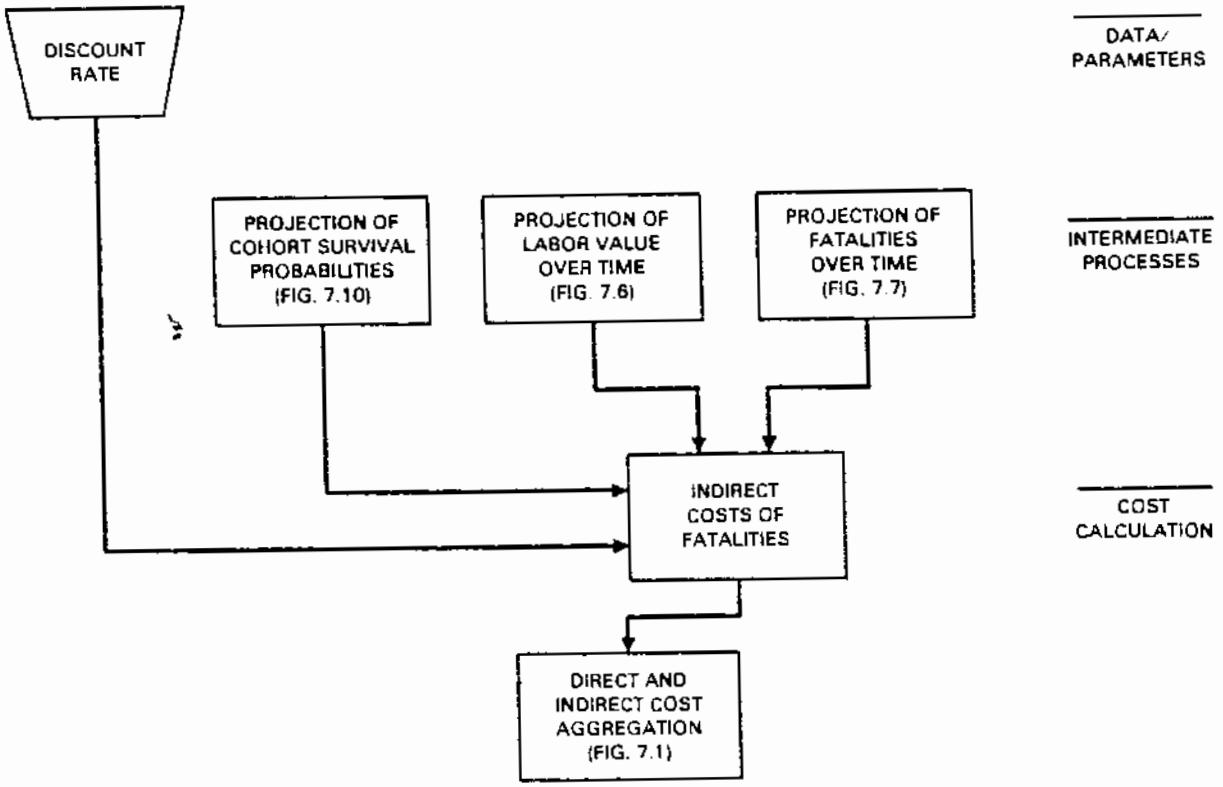

FIGURE 7.5. HECOM Calculation of Indirect Costs of Premature Mortality, by Age, Sex, and Cause of Death

The labor value lost because of a fatality is the sum of projected annual labor values from the year of death to the maximum specified age of the individual. The calculation of labor value lost is based on projections of fatalities in each year by age category, cause of death and sex. These labor value losses are discounted back to the base year to approximate the indirect costs of fatalities. These indirect costs are then aggregated by age cohort, sex and cause of death.

7.2.6 Calculation of Indirect Costs of Illness

Indirect costs of illness represent the value of labor lost due to illness. The flow of information in this calculation is presented in Figure 7.6. Data from intermediate calculations of projected cohort survival probabilities, labor value over time and fatalities over time are combined with data for the fraction of the population in each age cohort, radiation injury incidence, weeks of work missed, treatment duration, and the discount rate to calculate indirect illness costs.

Labor productivity loss is assumed to occur in the year prior to death. Projections of fatalities in each year are combined with incidence to fatality ratios, labor value projections in each year and the number of weeks of work

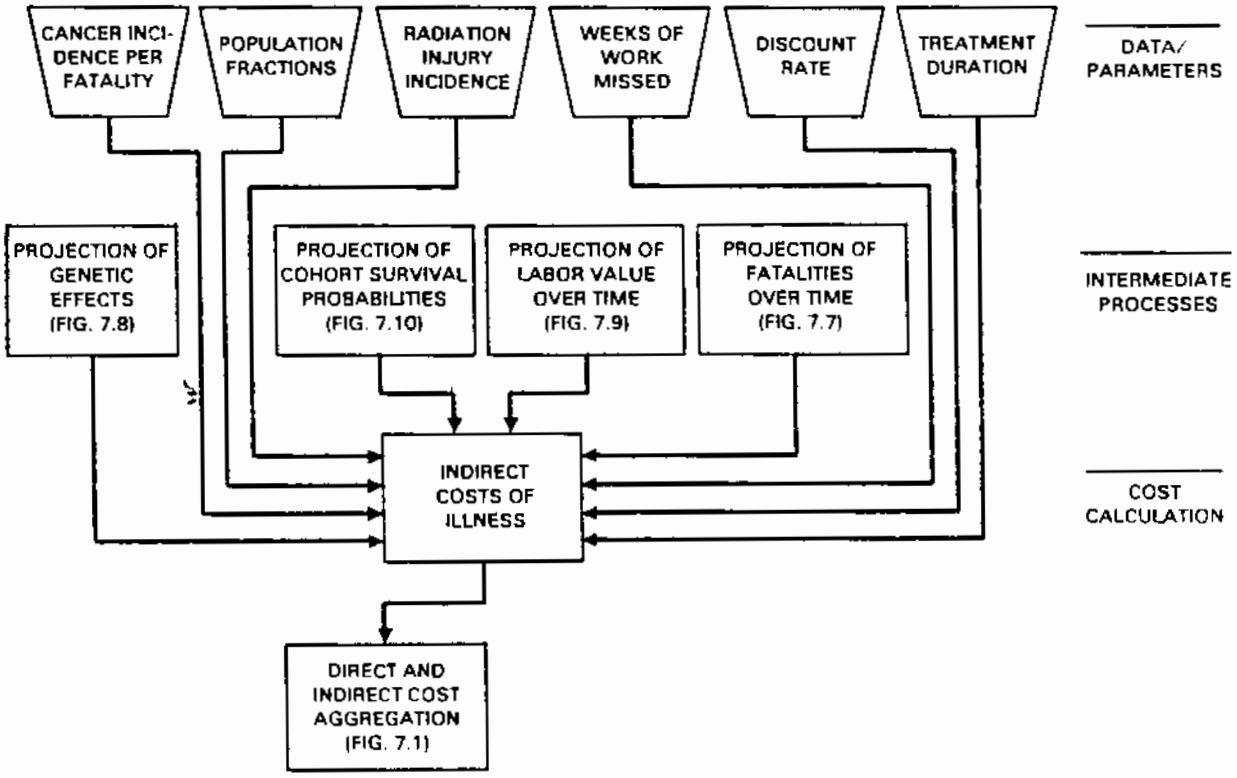

FIGURE 7.6. HECOM Calculation of Indirect Costs of Illness, by Age, Sex, and Cause of Death

lost for each type of cancer to calculate labor value loss by age category, year of illness, type of health effect and sex. Projections of cohort survival probabilities are used to adjust these loss estimates for the possibility that an individual will die from causes other than radiation-induced cancer.

Radiation injuries are assumed to occur in the base year only. Radiation injuries, allocated by sex, are apportioned to each age cohort according to its population fraction. The estimate of work weeks missed due to each type of radiation injury is applied to the value of labor for each cohort to calculate labor value lost due to radiation injuries.

Indirect costs attributable to genetic effects represent the lifetime productivity loss for each person born with a severe genetic defect. Projections of persons born with several genetic effects in each year are combined with labor value projections to estimate the expected value of genetic effect productivity loss.

The indirect costs associated with genetic effects, cancers and radiation injuries are discounted to the base year using the discount rate. In the final step, indirect illness costs for cancer are summarized by sex, cause of death, and age category.

7.2.7. Projection of Fatalities

The information used to project fatalities over time and the subsequent use of the fatality projections is presented in Figure 7.7. Input data to the calculation include population fractions with and without the in-utero age cohort, projections of cohort survival rates over time, fatality incidence from CRAC2, period of risk estimates, risk weighting factors, median survival times after diagnosis and minimum latency periods for each type of cancer. The fatality projections are used to calculate indirect illness and fatality costs and direct cancer costs.

The CRAC2 cancer fatality estimates are apportioned to age categories based on each cohort's fraction of the total population and each cohort's risk weighting factor. Acute fatality estimates from CRAC2 are apportioned to age categories using population fractions excluding the in-utero age category. All acute fatalities are treated as occurring in the first year after exposure. Using the absolute risk model option, cancer fatalities are distributed so that the annual fatality rate is constant over each age cohort's years at risk. The first fatality is projected to occur in the year after the end of both the latency period and the median survival period. The last fatality occurs in the year that the age cohort reaches the maximum age specified or the end of the period of risk. The end result of this process is a matrix of fatality projections by age cohort, year of death, cause of death and sex.

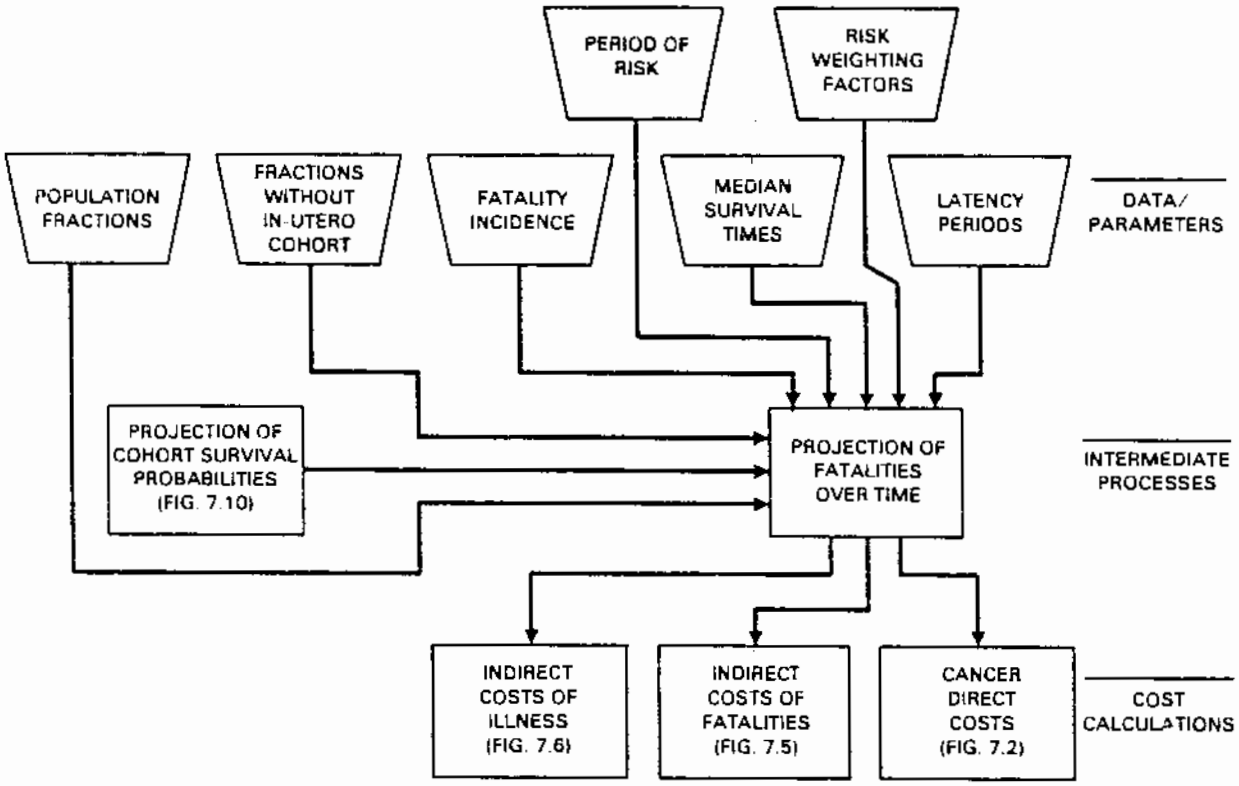

FIGURE 7.7. HECOM Projection of Fatalities, by Age, Sex, and Cause of Death

7.2.8 Projection of Genetic Effects

Figure 7.8 presents the flow of information into, and out of, the genetic effect projection process. Genetic effect incidence estimates, institutionalization rates, and genetic effect elimination rates are inputs to the process. The genetic effect projections are used to calculate both the direct and the indirect costs of illness due to genetic effects.

Institutionalization rates are used to determine the number of genetic effects that are so severe they will require treatment and institutional care. The elimination rates are used to allocate these genetic effects to each affected generation. The incidence in each generation is then allocated equally to each year within the generations.

7.2.9 Projection of Labor Value Over Time

The flow of information into, and out of, the labor value projection process is presented in Figure 7.9. Inputs to the process are the rate of labor productivity growth, median earnings or other labor value data for each age category and the median age of each age cohort. The labor value projections by sex and age category are used to calculate indirect illness and fatality costs.

Labor value projections for each year after exposure are calculated for each age cohort by sex. Median labor value in each future year, for each age cohort, is calculated from base year median earnings by age cohort, the rate of real income growth, labor force participation rates and the earnings categories the original cohorts will belong to in each year after the base year. When a

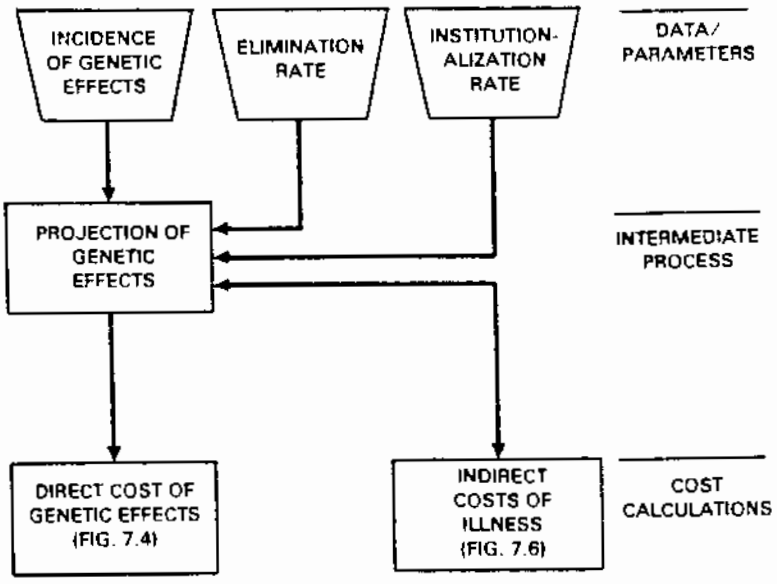

FIGURE 7.8. HECOM Projection of Genetic Effects

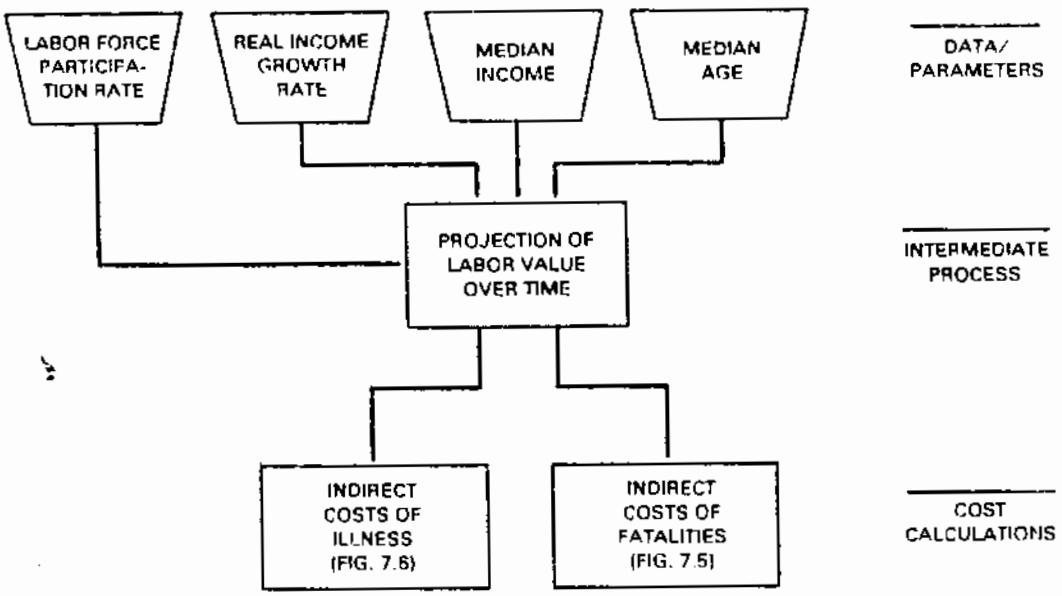

FIGURE 7.9. HECOM Projection of Labor Value, by Age and Sex

cohort ages over a time interval (i.e., five years), it is assigned the median earnings level of the cohort five years older with five years of labor productivity growth applied.

7.2.10 Projection of Cohort Survival Probabilities

Figure 7.10 presents the flow of information for projection of cohort survival probabilities and the cost calculations which use this information. Data on annual survival probabilities by sex and the median age of each cohort are inputs to the process, which produces an array of life probabilities by age category, sex and year after the base year. Data on annual survival probabilities (the probability that a person of any age and sex will live to the subsequent year) and the median age of each cohort are combined to calculate the probability that a person in each cohort at the time of exposure will live over subsequent years.

7.3 MODIFICATION OF CRAC2 OUTPUT FOR USE AS HECOM INPUT

Since the CRAC2 output was not designed to facilitate calculation of health effects costs, some intermediate steps are required to create compatible health effects and cost categories. The definitions of health effects projected by CRAC2 and the steps required to use them are described below.

7.3.1 Acute Effects

The CRAC2 projection of acute fatalities includes all deaths due to bone, lung, or gastrointestinal tract exposure. The projection is available as an aggregate, not by organ involved. The CRAC2 categories of acute fatalities and acute injuries are mutually exclusive and individuals are not double-counted.

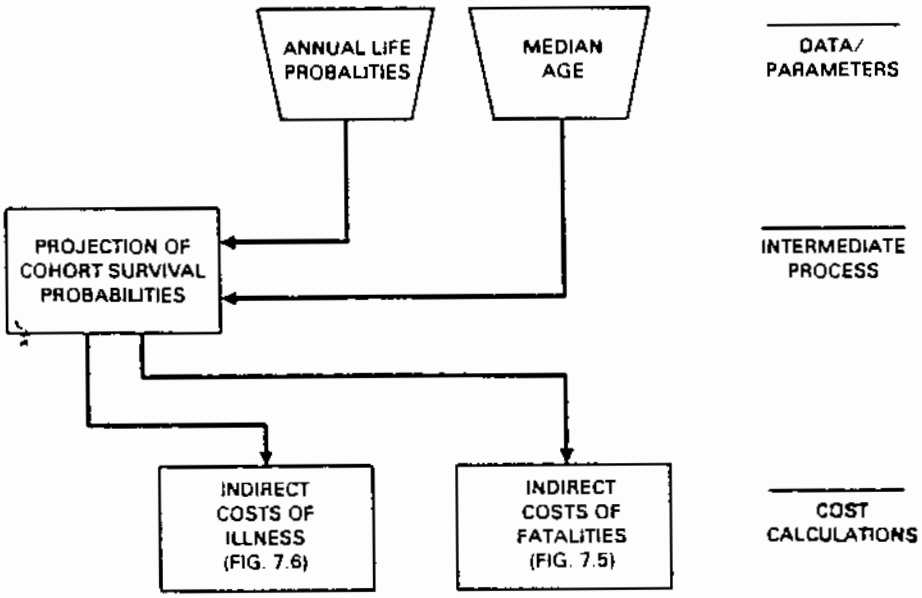

FIGURE 7.10. HECOM Projection of Cohort Survival Probabilities, by Age and Sex

Since all acute fatalities occur within the first year, the indirect costs due to a fatality do not depend on the type of injury. Therefore, the HECOM indirect cost computation is based directly on the CRAC2 acute fatalities estimate and is an aggregate for fatalities resulting from all of the types of radiation injuries.

Calculation of treatment costs for acute injuries is less straightforward because of the aggregate nature of CRAC2 injury estimates. CRAC2 does not provide estimates of serious injuries by type so that approximate treatment cost estimates can be applied. CRAC2 injury estimates do not include those who are injured (thus incurring costs) but die. Since all injured people would require treatment, this total is needed as the basis for the direct cost estimates. Projections of acute radiation injuries produced by CRAC2 represent the number of persons likely to have either prodromal symptoms, gastrointestinal syndrome or lung impairment. These effects are not double-counted, though in actuality, people may have multiple injuries. Bone marrow and prenatal injuries are not included in the CRAC2 projection of acute injuries.

Since the effects of radiation injuries range from minor to life-threatening, their treatment costs also vary considerably. To weight these costs, estimates are needed of the incidence of each type of radiation injury. This is calculated internally by CRAC2 for all injuries except bone marrow and prenatal injuries, but disaggregated output is not available as an option.

PNL has modified the standard CRAC2 code to provide disaggregated estimates of acute injuries and fatalities. The modifications use the CRAC2 health effects data set for acute exposure in its present form. Fatalities are estimated for each exposure type as follows:

$$F_i = PE \cdot PB_i$$

where

F_i = fatalities due to exposure type i
PE = population exposed, as calculated by CRAC2
PB_i = fatality probability given the exposure level
i = exposure type (i.e., bone marrow, etc.).

This modified calculation is performed for each exposure type for each evacuation scenario. Total fatalities for each start time are calculated as a weighted average over each evacuation scenario (as CRAC2 does currently for other early effects).

Injuries occurring in the population with exposures exceeding the fatality threshold are estimated as follows:

$$I_i = PE\ (1.0 - PB_i)$$

where

I_i = injuries of type i for people who are exposed above the fatality threshold but do not die.

The injuries are only calculated if PB_i is greater than zero. (If equal to zero, the fatality threshold was not reached.) The injuries are weighted by each evacuation scenario probability to estimated total injuries for the start time.

The calculation of injuries occurring in the population exposed to less than the fatality threshold also excludes people who die from fatal effects. The calculation is:

$$I_j = PEI \cdot PB_j$$

where

I_j = nonfatal injuries of type j
PEI = population exposed above the injury threshold
PB_j = injury probability given the exposure level.

Total injuries (by type) are estimated as a weighted sum over all evacuation scenarios.

To project prenatal injuries, the assumption is made that the distribution of population age groups exposed to greater than 200 rems is the same as their proportions in the general population. The proportion of the general population "in-utero" is multiplied by the number of individuals with an exposure of over 200 rem to estimate the size of the group at risk for prenatal injury. Based on the Reactor Safety Study (NRC 1975, Appendix VI p. F-21) an incidence rate for prenatal injuries of 50 percent is applied to the group at risk.

7.3.2 Cancers

CRAC2 estimates of cancers are available in the form of fatality projections for leukemia, lung, bone, breast, gastrointestinal, and other cancers. These fatality estimates are used directly in the HECOM calculation of indirect cancer costs. To calculate direct costs, the CRAC2 fatality estimates must first be converted to estimates of cancer incidence. This conversion is carried out within HECOM using the fatality/ incidence relationships documented in the Reactor Safety Study (NRC 1975, Appendix VI). These ratios are listed in Appendix A, Table A.6. The resulting incidence estimates provide the basis for the direct cost projections.

The thyroid effects projected by CRAC2 are an incidence, rather than fatality, estimate that includes both benign and malignant nodules. Since the costs of treating these nodules differ, the CRAC2 thyroid projection is allocated by HECOM to the two types of effects in proportion to the relative spontaneous incidence of benign thyroid nodules and malignancies in the population (NRC 1975, Appendix VI p. 9-27).

7.3.3 Genetic Effects

When the option of calculating genetic effects with CRAC2 is implemented, the resulting projection is an aggregate of all types of genetic disorders. Since different types of effects are eliminated from the population at different rates, HECOM allocates each type of genetic effect across generations separately. To accommodate this level of disaggregation, the CRAC2 estimates must be allocated between genetic effect types before being input to HECOM. Currently, we are assuming two treatment categories and an allocation of 50 percent of effects to each one.

7.4 HECOM SENSITIVITY ANALYSIS

Some level of uncertainty exists in each of the input variables used to estimate health effects costs. A sensitivity analysis was performed to provide an indication of the significance of these uncertainties; it illustrates how costs would change in response to variation in input estimates.

Sensitivity is generally measured by systematically varying the value of one input variable within the bounds of a range of uncertainty, while holding all other input variables constant. To measure sensitivity in HECOM we examined seven variables: the discount rate, rate of labor productivity growth, rate of real growth in treatment costs, base year earnings, base year treatment costs, weeks of work missed due to illness, and labor force participation rates.

The model appears to be most sensitive to changes in the discount rate, the rate of treatment cost escalation, and the rate of labor productivity growth. The effect is most significant when the discount rate is assumed to be equal to the real growth rates of labor productivity and treatment costs. In that case there is effectively no discounting of costs over time.

Regarding HECOM sensitivity to cost input data, both variations in earnings and treatment costs cause substantial changes in the HECOM cost estimates; variations in the weeks of illness cause almost no effect. In the sections that follow, the results of each sensitivity test are examined separately.

7.4.1 Sensitivity to the Discount Rate

Table 7.1 shows the effect of different discount rates on the indirect, direct and total costs calculated by HECOM. The ten 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 four percent discount rate is used to represent the social rate of discount. Rates of seven percent and one percent are also tested to explore fully the sensitivity of HECOM. As shown in Table 7.1, the model is clearly sensitive to the discount rate. Use of a seven or ten 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 one percent substantially increases costs because costs in the more distant future are given nearly the same weight as costs in the near future. All cost categories are strongly affected by use of a one percent discount rate because the costs of treating genetic effects over 300 years become relatively large.

TABLE 7.1. Sensitivity of HECOM Estimates to the Discount Rate

Oiscount Rate %	Indirect Cost % Δ From Base	Direct Cost % Δ From Base	Total Cost % Δ From Base
10	-73.1	-44.0	-58.4
7	-53.7	-31.7	-42.6
4 (Base)	0.0	0.0	0.0
1	299.8	264.6	282.0

7.4.2 Sensitivity to Labor Productivity Growth Rates

The effect on indirect and total costs of varying the rate of labor productivity growth from its base case rate of one percent to a rate of three percent is shown in Table 7.2. The three percent rate results in a more than 100 percent increase in indirect costs and more than 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 7.2 Sensitivity of HECOM Estimates to the Rate of Labor Productivity Growth

Rate of Labor Productivity Growth (%)	Indirect Cost % Δ From Base	Total Cost % Δ From Base
3	114.0	56.4
1 (Base)	0.0	0.0

7.4.3 Sensitivity to Treatment Cost Escalation

Table 7.3 shows relative costs of treatment calculated using the one percent base rate, and alternative rates of three percent and five percent for real treatment cost escalation. Increasing the rate to three percent raises direct costs by over 80 percent, and further increasing the rate to five percent results in an increase of over 1400 percent for direct costs and 700 percent for total costs. This dramatic increase occurs because the rate of treatment cost growth exceeds the base case discount rate of four percent, resulting in very large genetic effect treatment costs over the 300 years following exposure. Because there is significant uncertainty regarding the future rate of growth for real treatment costs, HECOM estimates must be interpreted carefully. Over the modeled period of 300 years, real costs of medical care for genetic disorders could either rise or fall and may well have a complex pattern of change.

TABLE 7.3 Sensitivity of HECOM Estimates to the Rate of Treatment Cost Escalation

Rate of Treatment Cost Growth	Direct Cost % Δ From Base	Total Cost % Δ From Base
5%	1,446.5	742.3
3%	81.4	41.3
1% (Base)	0.0	0.0

7.4.4 Sensitivity to Earnings Levels

Table 7.4 shows the effects on indirect and total costs of a 20 percent variation in base year earnings levels. Indirect costs change in direct

TABLE 7.4 Sensitivity of HECOM Estimates to Earnings Levels

Earnings	Indirect Cost % Δ From Base	Total Cost % Δ From Base
Base Income plus 20%	20.0	9.8
Base Income	0.0	0.0
Base Income minus 20%	-20.0	-9.8

proportion to levels of base year earnings. The potential error in total health effects costs resulting from uncertainties in base year earnings estimates is approximately 10 percent.

7.4.5 Sensitivity to Treatment Costs

The effect of uncertainties in treatment cost estimates is presented in Table 7.5. The range of uncertainty in treatment costs is estimated to be 30 percent. Varying treatment costs by 30 percent results in an identical percentage change in direct costs and a 15.2 percent variation in total health effects costs.

TABLE 7.5 Sensitivity of HECOM Estimates to Treatment Costs

Treatment Costs	Direct Cost % Δ From Base	Total Cost % Δ From Base
Base plus 30%	30.0	15.2
Base	0.0	0.0
Base minus 30%	-30.0	-15.2

7.4.6 Sensitivity to Weeks of Illness

The uncertainty in estimates of weeks of work missed due to illness is estimated to be about 50 percent. Table 7.6 presents the effects on indirect and total health effects costs of a 50 percent variation in estimated weeks of illness. The results indicate that this variable is of only minor importance in determining indirect costs and that the high level of uncertainty in this variable leads to only a 1.9 percent margin of uncertainty in total health effects cost estimates.

TABLE 7.6 Sensitivity of HECOM Estimates to Weeks of Illness

Weeks of Illness	Indirect Cost % Δ From Base	Total Cost % Δ From Base
Base plus 50%	3.9	1.9
Base	0.0	0.0
Base minus 50%	-3.9	-1.9

7.4.7 Sensitivity to Labor Force Participation Rates

Estimates of labor force participation rates are used by HECOM to determine the expected value of population earnings. Labor force participation rates for each cohort were analyzed at a 100 percent level, and at the base case values given in Appendix A, Table A.4. The results of this variation on cost estimates are shown in Table 7.7. The results indicate that 100 percent participation in the labor force would increase indirect costs by about 20 percent and total costs by slightly over ten percent.

TABLE 7.7 Sensitivity of HECOM Estimates to Labor Force Participation Rates

Labor Force Participation Rates	Indirect Cost % Δ From Base	Total Cost % Δ From Base
100%	20.6	10.2
Base	0.0	0.0

7.4.8 Comparison of Median and Interval Data Results

Table 7.8 compares HECOM estimates based on median and interval case data. The median case represents the national median income, while the interval data case uses 18 age category-specific income estimates. Total costs in the median case are eight percent higher than the interval case. Direct costs in both cases are almost equal. Most of the difference in cost estimates occurs in the estimation of indirect costs where the median case estimate is 16 percent higher than the interval estimate.

TABLE 7.8. Comparative Results of Median and Interval Data Cases

Case	Indirect Cost % Δ From Interval	Direct Cost % Δ From Interval	Total Cost % Δ From Interval
Median	16.2	0.0	8.0
Interval	0.0	0.0	0.0

7.0 REFERENCE

U.S. Nuclear Regulatory Commission. 1975. Reactor Safety Study. Appendix VI. WASH-1400, National Technical Information Service, Springfield, Virginia.

8.0 HEALTH EFFECTS COSTS FOR A HYPOTHETICAL REACTOR ACCIDENT

HECOM has applications in various types of siting analyses, evaluations of safety goals and standards, and many decisions related to the management of nuclear power. An example of the output of HECOM for use in these types of applications is provided in this section. The numbers shown are derived from only one hypothetical accident scenario at a representative reactor, and until further research is undertaken, it cannot be determined whether the order of magnitude of the costs is typical for other reactors, or even other accident scenarios at the same reactor. Thus, the estimates given should be treated only as illustrative examples of HECOM's calculational capabilities.

8.1 HEALTH EFFECTS ESTIMATES

Probabilistic estimates of health effects from a CRAC run for a given hypothetical reactor accident scenario were used as inputs to the HECOM cost calculation. The projected numbers of each type of health effect are shown in Table 8.1. These estimates are based on PNL's modification of the standard CRAC code; the modification provides estimates for each of the categories of acute fatalities and injuries. Bone marrow injuries are included in the PNL modification, although omitted from standard CRAC analyses. An explanation of the code modification is provided in Section 7.3.1. Prenatal effects are calculated as a function (explained in Section 7.3.3) of the number of people exposed to over 200 rem, in this case, 3360. Genetic effects are estimated on the basis of 260 per million rem of population exposure.

8.2 COST ESTIMATES

To project health effects costs, the HECOM user must specify the desired real growth and discount rates. The following estimates assume one percent annual growth rates for real income and health care costs and a discount rate of ten percent. Health effects costs are all shown in 1981 dollars.

HECOM projects costs for direct (treatment) costs and indirect (lost productivity) costs. These are shown for each category of health effect in Table 8.2. Health effects costs for this reactor accident scenario total $\$7.6 \times 10^7$. Other categories of costs for accident consequences at this reactor are: evacuation, $\$3.14 \times 10^6$; agricultural losses, $\$1.56 \times 10^7$; relocation, $\$3.64 \times 10^8$; land interdiction, $\$1.53 \times 10^9$; and decontamination, $\$1.80 \times 10^8$. Judging from these estimates, the health costs are a substantial portion of the total potential economic impact of a reactor accident.

TABLE 8.1. Projected Numbers of Health Effects for One Hypothetical Reactor Accident Scenario Used as Input to the Sample HECOM Calculation

Health Effect	Number
Cancers:	
Leukemia	18.8
Lung	23.5
Breast	69.2
Bone	5.9
Gastrointestinal	6.0
Other	16.3
Thyroid	43.7
Acute Fatalities:	
Bone Marrow	331.2
Lung	13.8
Gastrointestinal	0
Acute Injuries:	
Bone Marrow	198.8
Prodromal	222.6
Lung	564.5(a)
Gastrointestinal	8.0
Prenatal	20.2
Genetic	616.2

(a) The relatively large proportion of acute lung injuries is due to meteorological conditions in the single scenario analyzed.

TABLE 8.2. Projected Health Effects Costs for One Hypothetical Reactor Accident Scenario (Thousands of 1981 $)

Health Effect	Cost Component		Total Cost
	Direct	Indirect	
Cancers	404	1,056	1,460
Acute Fatalities and Injuries	34,223	36,535	70,758
Genetic	3,414	584	3,998
Total	38,041	38,175	76,216

APPENDIX A

HEALTH EFFECTS COST MODEL

APPENDIX A

HEALTH EFFECTS COST MODEL

The Health Effects Cost Model (HECOM) calculates the direct and indirect costs resulting from radiation-induced health effects. The model is written in FORTRAN-77 and is being maintained on a Digital Equipment Corporation VAX 11/780. An overview of HECOM is given in Section A.1, a description of input data and input file structure in Section A.2, a detailed explanation of subroutine and function operation in Section A.3 and a description of outputs in Section A.4. The FORTRAN source code is listed in Appendix B.

A.1 OVERVIEW

This section presents an overview of HECOM. A description of each file within the model is provided along with a description of the process used in calculating health effects cost estimates.

A.1.1 HECOM Files

HECOM consists of seven files: three FORTRAN files (source, object and executable), three data files and a FORTRAN control file. The function of each file is described below:

- HECOM.FOR. FORTRAN source code containing all subroutines and functions.
- HECOM.OBJ. Object code produced by the FORTRAN compiler.
- HECOM.EXE. Executable file produced by linker.
- HECOM1.DAT. Data file containing median case data.
- HECOM18.DAT. Data file containing interval case data by five-year age cohorts.
- INDIST18.DAT. Data file containing risk weighting factors by five-year age cohorts.
- INDIST1.DAT. Data file containing risk weighting factors for the median case.
- DIST.DAT. Data file containing survival probability data by year and sex.
- CONTROL.FOR. Source code containing PARAMETER, COMMON, REAL, INTEGER and CHARACTER statements. This file is used to control execution of the model and is accessed by each subroutine. It is incorporated by the FORTRAN compiler into HECOM.OBJ.

A.1.2 Description of Program Operation

Execution of HECOM is controlled by the file CONTROL.FOR. Parameters are assigned in the first two lines of this file and are used to dimension all arrays and to control processing of all loops within the main program and its subroutines. Health effects data inputs, except for cancer fatalities, are identified by specifying the array index of either acute and thyroid death types or prenatal radiation injuries. The parameters that must be set by the user are listed in Table A.1. All input data must be consistent with these parameters. The file CONTROL.FOR is accessed by each subroutine using the system command "INCLUDE 'CONTROL.FOR'".

Figure A.1 shows the structure of the model. The main program consists of a series of sequential subroutine call statements. The program begins by reading in data and ends by writing out health effects costs to an output file. The functions of each subroutine and function shown in Figure A.1 are briefly described below. A detailed description of each subroutine and function is given in Section A.3 and the program listing in Appendix B.

- READER. Reads in input data from DIST.DAT and either HECOM1.DAT or HECOM18.DAT depending on the number of age categories set in CONTROL.FOR.

- SPROB. Calculates the probability a person of either sex and a given age at time of exposure will be alive in any year after exposure. Based on the survival probabilities contained in DIST.DAT.

- LATENCY. Calculates minimum latency periods for each cancer type.

TABLE A.1. HECOM Parameters

Parameter	Definition
AC	Number of age categories
IC	Number of income categories
GAC	Number of genetic effect age categories
IINC	Income data interval (number of years)
AINC	Age data interval (number of years)
GINC	Genetic effect age category data interval (number of years)
YEARS	Maximum age affected population can attain
GYEARS	Maximum years to project genetic effects
NGEN	Number of generations
DTYPES	Number of death causes
SEX	Number of sex categories (1 or 2)
HTYPES	Number of cancer types
RTYPES	Number of radiation injuries
ACUTE	Set to acute death type
THYROID	Set to thyroid death type
PRENAT	Set to prenatal radiation injury type

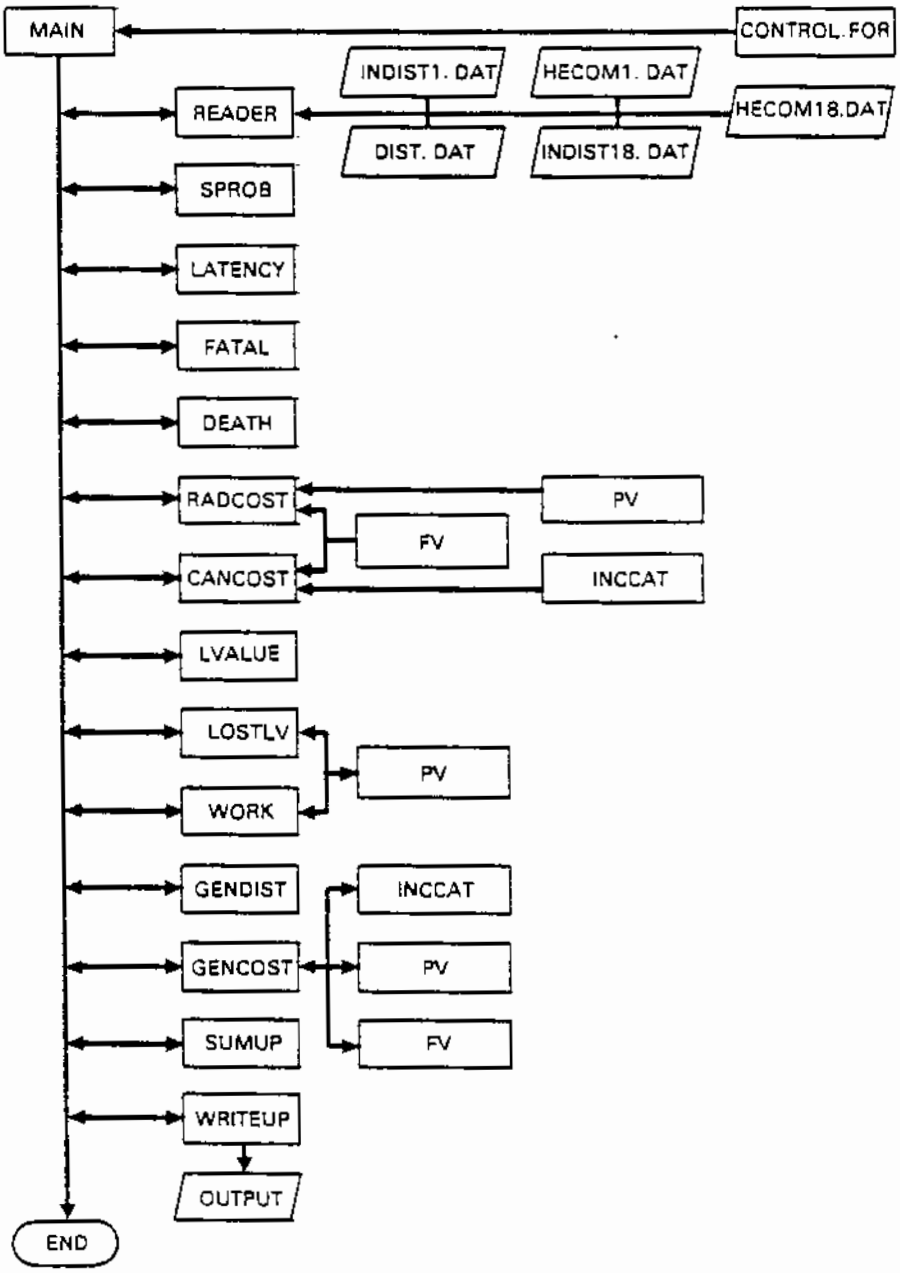

FIGURE A.1. HECOM Structure

- FATAL. Distributes fatalities to age categories.

- DEATH. Calculates fatalities per year by cause of death using optional constant absolute risk distribution model.

- RADCOST. Cost of treating radiation injuries is calculated based on incidence and population fractions by age cohort.

- CANCOST. Cost of treating cancer is calculated based on the incidence to fatality ratios and the number of fatalities of each cancer type per year calculated in DEATH.

- LVALUE. Labor value by age cohort and year after radiation exposure is calculated based on wages by age cohort and the rate of real income growth.

- LOSTLV. Lifetime discounted earnings loss is calculated for a person dying in each year after exposure. Earnings calculated in LVALUE are multiplied by the probability that a person will be alive in any year and summed over the time period between the year of death and the maximum age specified in CONTROL.FOR.

- WORK. Calculates the value of work lost due to illness. Cost is based on fatalities per year calculated in DEATH, life probabilities calculated in LIFE, earnings calculated in LVALUE, incidence to fatality ratios, and weeks of work lost due to illness by health effects type.

- GENDIST. Distributes genetic effects to the years after radiation exposure.

- GENCOST. Calculates the present value of treating and institutionalizing individuals with genetic defects.

- SUMUP. Calculates total income loss based on fatalities calculated in DEATH, and income loss in LOSTLV. This subroutine also calculates summary arrays used in printing results.

- WRITER. Prints out summary variables calculated in SUMUP.

- PV. Calculates present value of a number given the discount rate and the number of years to be included.

- FV. Calculates future value of a number given the growth rate and number of years to be included.

- INCCAT. Determines earnings category of any age cohort.

A.2 DESCRIPTION OF INPUT DATA FILES

The data used as input to HECOM can be grouped in four general categories, including information on population characteristics, health effects incidence, direct costs of treatment, and indirect costs of lost productivity. In this section, we describe sources for data in each of those categories, explain some of the merits and the limitations of particular data sources and describe the structure of HECOM input files.

A.2.1 Population Characteristics

HECOM employs descriptions of the population at risk: by age and sex categories, by mean earnings for the two sexes at each age, and by life expectancy at different ages.

- Population by age and sex: Population counts, distributed into cohorts by sex and by age intervals, are used both in the allocation of health effects and in the estimation of indirect costs. On the national level, the most recent data of this type are from the 1970 Census. These data can be used if detailed local or regional data are unavailable. See Table A.2 for the proportional distribution of the US 1970 population by age and sex cohorts.

TABLE A.2. U.S. Urban and Rural Population Distribution by Age and Sex, 1970

Ages	(Percentage)[a] Total	Male	Female
All ages	100	48.7	51.3
In utero	1.1	0.6	0.6
1 - 4	7.3	3.8	3.5
5 - 9	9.9	5.0	4.8
10 - 14	10.3	5.1	5.1
15 - 19	9.4	4.8	4.7
20 - 24	7.9	3.8	4.1
25 - 29	6.6	3.2	3.4
30 - 34	5.6	2.8	2.9
35 - 39	5.5	2.7	2.8
40 - 44	5.9	2.9	3.0
45 - 49	6.0	2.9	3.1
50 - 54	5.5	2.6	2.8
55 - 59	4.9	2.3	2.6
60 - 64	4.3	2.0	2.3
65 - 69	3.4	1.5	1.9
70 - 74	2.7	1.1	1.5
75 - 79	1.9	0.8	1.1
80+	2.1	0.7	1.2

Source: U.S. Bureau of the Census. 1973. Census of Population: 1970 Detailed Characteristics. Final Report PC(1)-D1 United States Summary. U.S. Government Printing Office, Washington, D.C. Table 1, p. 591.

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

Statistics from the 1980 Census are not yet available to describe the characteristics of the US population as a whole. A compendium of "provisional estimates" is now available, but those estimates describe the population in grosser schema (for example, by 10-year age increments instead of 5-year) and the estimates include little information on income characteristics. The HECOM model can easily be run with information from the 1980 Census when appropriate data are available.

- Mean earnings by age and sex: This information is used in the estimation of lifetime expected earnings and thereby in the calculation of indirect costs. The input data may either be median or for any width age interval. In addition, either site-specific or national data can be used in HECOM. Table A.3 provides an example of the most recent national earnings data available. Mean earnings figures in 1981 dollars are listed by 5-year age increments and by sex (U.S. Dept. of Commerce 1982.) We use the Consumer Price Index for "all items" for all urban consumers, to inflate the 1980 earnings estimates to 1981 dollars. (US Dept. of Labor, selected years.)

TABLE A.3. Mean Earnings of Employed Persons, by Age and Sex (1981 $)[a]

Ages	Male	Female
18 - 24[b]	7,431	5,211
25 - 29	15,696	9,542
30 - 34	19,833	9,898
35 - 39	23,173	9,892
40 - 44	23,597	9,975
45 - 49	24,445	9,921
50 - 54	23,570	9,979
55 - 59	23,055	9,844
60 - 64	19,205	9,443
65+	9,080	4,590

Source: US Department of Commerce, Bureau of the Census. 1982. Money Income of Households and Families and Persons in the United States: 1980. Current Population Reports, Series P-60, No. 132. U.S. Government Printing Office, Washington, D.C.

(a) 1980 incomes inflated to 1981 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 15-20 and 20-25 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.

- Life expectancy: Data on cumulative life probabilities are used to describe the life expectancies of individuals in the unexposed population, in cohorts distinguished according to age and sex. Annual life probabilities are computed from the data in Table 5-1, Vital Statistics of the United States 1978, Volume II-Section 5, "Life Tables," p. 5.9. (National Center for Health Statistics 1980.) The 1978 life tables are the most recent currently available; the vital statistics life table data are typically 2 to 3 years old at the time of publication.

- Labor force participation rate: Based on an analysis by Hartunian, et al. (1982, 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.4.

TABLE A.4. Employment and Housekeeping Participation Rates by Age and Sex (in %)

Ages	Male	Female
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: N. S. Hartunian, C. A. Smart and M. S. Thompson. 1982. The Incidence and Economic Costs of Major Health Impairments. Lexington Books, Lexington, Massachusetts, p. 49.

A.2.2 Health Effects

Calculation of health effects costs requires data on incidence, latency periods, survival times, period of risk and relative risk by age and sex. Data for cancers and for radiation injuries and fatalities are described first.

- Incidence: HECOM requires incidence data for mortality and morbidity for each type of health effect. These data must be entered by sex if the model is run using two sex categories. Proportional allocation of the data by sex is computed prior to data entry. Incidence data for fatalities, injuries and cancers are taken directly from CRAC2 output, when available, and calculated based on CRAC2 output in the remaining cases. Table A.5 shows the source and method used for each portion of the data. The incidence to fatality ratios applied to the CRAC2 cancer fatality estimates to calculate total cancer incidence are shown in Table A.6. These are the same ratios that are assumed by CRAC2 in projecting fatalities. The process by which CRAC2 acute injury estimates are disaggregated by type is described in Section 7.3.2.

TABLE A.5. Health Effects Incidence Data Sources

	Latent Effects		
	Cancers	Thyroid	Acute Effects
Morbidity Data	Computed by HECOM from CRAC2 output using incidence to fatality ratios	CRAC 2 output	Computed by a modified CRAC2 process
Mortality Data	CRAC2 output	None	CRAC2 output

TABLE A.6. Incidence/Fatality Ratios Applied to CRAC2 Fatality Projections

Cause of Death	Ratio
Leukemia	1.00
Lung	1.00
Breast	2.00
Bone	1.25
Gastrointestinal	1.20
Other	2.00
Acute	1.00

Source: U.S. Nuclear Regulatory Commission. 1975. Reactor Safety Study. Appendix VI. WASH-1400. National Technical Information Service, Washington, D.C., pp. G18-G23.

- Latency periods: Appendix VI of the Nuclear Regulatory Commission's Reactor Safety Study (Section G, p. G-23) is the source of data on minimum latency periods, by cancer site, for the population in utero and for all other ages. (NRC 1975.) See Table A.7 for a listing of the values used. There is no latency period for acute health effects.

TABLE A.7. Period of Latency for Selected Cancer Types

Cancer Type	In Utero	All Other
Leukemia	0	2
Lung	15	15
Gastrointestinal	15	15
Breast	15	15
Bone	10	10
All Other	0	15
Thyroid	10	10

Source: US Nuclear Regulatory Commission. 1975. Reactor Safety Study, Appendix VI. Wash-1400. Government Printing Office, Washington, D.C., p. G-23.

- Survival time: 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 attributable to that subgroup. The NIH data do not distinguish survival times by sex. Median survival times are presented in Table A.8. For radiation injuries the survival time for all fatal cases is less than a year.

TABLE A.8. Median Survival Time, 1960-1973

Cancer Type	Median Survival[(a)] Time (years)
Leukemia	1
Lung	1
Gastrointestinal	2
Breast	6
Bone	2
All Other	4
Thyroid	15

Source: National Institutes of Health, USDHHS. 1976. Cancer Patient Survival, Report Number 5. NIH Publication No. 81-992. Government Printing Office, Washington, D.C.

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

- Period of Risk: Estimates of the time period an individual exposed to radiation would be at risk for cancers are listed in Table A.9. These risk periods are used to allocate fatalities to the years after radiation exposure.

TABLE A.9. Period of Risk of Incurring Cancer After Exposure

Cancer Type	Period of Risk
Leukemia	30 years
Lung	lifetime
Breast	lifetime
Bone	30 years
Gastrointestinal	lifetime
Other	lifetime
Thyroid	lifetime

Source: Committee on the Biological Effects of Ionizing Radiation. 1980. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. National Academy of Sciences, Washington, D.C., p. 243.

- Risk Weighting Factors: Risk weighting factors are used in conjunction with population fraction data to allocate cancer incidence to each age and sex cohort. These data are BEIR III estimates of excess cancer incidence resulting from radiation exposure. Values used are listed in Table A.10.

TABLE A.10. Risk Weighting Factors by Age and Sex

	MALES Age at Exposure				
Cancer Types	0 - 9	10 - 19	20 - 34	35 - 49	50+
Leukemia	3.98	1.85	2.60	1.92	4.32
Lung	0.00	0.54	2.45	5.10	6.79
Gastrointestinal	0.33	0.33	0.65	1.06	2.79
Breast	0.00	0.00	0.00	0.00	0.00
Bone	3.98	1.85	2.60	1.92	4.32
Other	0.62	0.38	1.12	1.40	2.90
Thyroid	2.20	2.20	2.20	2.20	2.20
	FEMALES				
Leukemia	2.54	1.19	1.67	1.24	2.76
Lung	0.00	0.54	2.45	5.10	6.79
Gastrointestinal	0.33	0.33	0.65	1.06	2.79
Breast	0.00	7.30	6.60	6.60	6.60
Bone	2.54	1.19	1.67	1.24	2.76
Other	0.62	0.38	1.12	1.40	2.90
Thyroid	5.80	5.80	5.80	5.80	5.80

Source: Committee on the Biological Effects of Ionizing Radiation. 1980. The Effects on Populations of Exposure to Low Levels of Ionizing Radiation. National Academy of Sciences, Washington, D.C. Table V-14 and V-17, pp. 250 and 256.

HECOM requires estimates of the total number of genetic effects of each type considered. Currently the base case treats only autosomal dominant and multifactorial defects as shown in Table A.11. An estimate of the proportion of cases that are severe is input to assign costs. The rate per generation at which genetic defects are eliminated from the population must also be input to distribute incidence over time.

TABLE A.11. Genetic Effects Incidence

Genetic Effect type	Percentage of Total that are Severe	Elimination Rate Per Generation
Autosomal dominant	50%	20%
Multifactorial	50%	10%

A.2.3 Direct Costs

Input data for direct costs of cancers and radiation injuries are required by cancer and injury type. Methods used to develop the HECOM data base shown in Table A.12 are described for radiation injuries in Section 5.1 and for cancers in Section 5.2. We have inflated estimates to 1981 dollars using The Consumer Price Index "hospital room" component for hospital costs and the more general "medical care" component for other treatment costs. (U.S. Department of Labor, various years.) See Table A.13 for relevant components of the Consumer Price Index, for selected years.

TABLE A.12. Direct Costs of Health Effects (1981 $)

Cancers	Treatment Cost
Leukemia	16,300
Lung	17,400
Breast	9,400
Bone	37,600
Gastrointestinal	14,000
Other	11,400
Thyroid-benign	7,700
Thyroid-malignant	8,400
Radiation Injuries	
Prodromal	1,000
Bone marrow	56,000
Gastrointestinal	28,000
Pulmonary	3,600
Prenatal (in utero)	100,000

TABLE A.13. Consumer Price Index, All Urban Consumers (1967 = 100)

	1970	1975	1980	1981
CPI, all items	116.3	161.2	247.0	272.3
CPI, all services	121.6	166.6	270.9	306.2
All Medical Care (services + commodities)	120.6	168.6	267.2	295.1
Medical care services	124.2	179.1	288.9	318.6
Hospital Room	145.4	236.1	416.3	476.8

Source: Bureau of Labor Statistics, US Department of Labor, Consumer Price Index: Detailed Statistics. Published monthly. U.S. Government Printing Office, Washington, D.C.

A.2.4 Indirect Costs

The HECOM model treats both cancers and radiation injuries as resulting in indirect costs related to lost productivity for periods of morbidity and because of premature mortalities. Calculation of indirect costs due to morbidity requires data on work weeks lost. Data presented in Hartunian et al. (1981, p. 236) are shown in Table A.14. The value associated with those lost weeks is computed using earnings data by age and sex category.

TABLE A.14. Mean Number of Work Weeks Lost by Cancer Patients During First Year After Onset of Illness

Cancer Type	Number of Work Weeks Lost
Leukemia[a]	16.3
Lung	19.9
Gastrointestinal[b]	18.5
Breast	17.2
Bone	23.3
All Others[b]	16.1
Thyroid	5.9

Source: Hartunian N.S., et al. 1981. The Incidence and Economic Costs of Major Health Impairments. Lexington Books, Lexington, Massachusetts, p. 236.

(a) Simple mean for all types of leukemia.
(b) Mean for gastrointestinal and "others," weighted by relative share of component cancer sites.

For radiation injuries, the period of productivity loss due to morbidity is estimated using information from Prasad 1974; Dalrymple 1973; and Blakely 1968. One week of work loss is assumed for prodromal injuries and 26 weeks each for bone marrow, gastrointestinal and pulmonary injuries.

A.3 DESCRIPTION OF SUBROUTINES AND FUNCTIONS

HECOM contains 15 subroutines and 3 functions as well as the main program. The main program contains only subroutine call statements. A description of each subroutine and function follows; including the calculations used, a brief description of subroutine or function operation and a listing of all variables and arrays.

The subscripts used in the calculations follow the following conventions:

a = age category of an individual in the year of exposure
s = sex
n = number of years after base year
d = cause of death (cancer and acute radiation injuries)
c = cancer type
r = radiation injury type
t = genetic effect type

The subroutine and function descriptions that follow are in the same order as used within HECOM (see Figure A.1). Section A.1 explains how each subroutine and function fits into the HECOM structure.

A.3.1 READER

This subroutine reads in input data. Lifetime probability data are read from the file DIST.DAT. If the median data case is being run, data are read from HECOM1.DAT and risk weighting factor data from INDIST1.DAT. If the interval data case is being run, data are read from HECOM18.DAT and risk weighting factor data from INDIST18.DAT.

A.3.2 SPROB

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 year. The only inputs to the subroutine are the probabilities that a person of age a will live to age a + 1. The probability of living to any given year n is calculated as follows:

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

where

$P(n)_{a,s}$ = prior probability that a person who was of age a in the year of exposure and of sex s will be alive after n years

$P(a+1)_{a,s}$ = probability that a person of age a and sex s will live to be age a + 1.

The subroutine processes nested loops for age category and sex. Within the inner loop the median age of the age category is determined and used to calculate remaining life period. A third loop processes years, and, nested within this loop is another that calculates the product of the conditional probabilities. This subroutine is used to compute the vector for a newborn infant. A separate vector of probabilities is calculated for use in determining the costs of genetic effects.

A.3.3 LATENCY

This subroutine generates a matrix of latency periods by age category and cause of death. Latency periods are assigned identically to each age category for each cause of death except for the in utero age category that is assigned separately. The latency period information is constructed from input data.

A.3.4 FATAL

This subroutine distributes fatalities by age category, cause of death and sex. Input items include population fractions by age category and sex (calculated for all age categories and again for all age categories except in utero); risk weighting factors by cause of death, age and sex; and total fatalities by cause of death and sex. Cancer fatalities are distributed based on both age category population fractions and risk weighting factors. Acute fatalities are distributed based on age category population fractions excluding in-utero. The in-utero category is treated separately. While in-utero fatalities are not currently estimated in the HECOM calculation, indirect costs of prenatal radiation exposure are calculated in this subroutine since these victims are too disabled to earn any income. Fatalities are distributed according to the following equations:

Cancer Fatalities

$$F_{a,s,d} = F_d(PF_{a,s} \cdot RWF_{a,s,d}) / \sum_{s=1}^{2} \sum_{a=1}^{AC} (PF_{a,s} \cdot RWF_{a,s,d})$$

Indirect Costs, In-Utero Age Category

$$F_{(\text{in utero}),s,(\text{Acute})} = RIF_{(\text{Prenatal}),s} + RINF_{(\text{Prenatal}),s}$$

Acute Fatalities

$$F_{a,s,d} = F_d \cdot PA_{a,s}$$

where

$F_{a,s,d}$ = fatalities for age category a, sex s and cause of death d

F_d = total fatalities by cancer or acute radiation injury fatality type d

$PF_{a,s}$ = population fraction of age category a and sex s

$RWF_{a,s,d}$ = risk weighting factor for age category a, sex s and cause of death d

AC = number of age categories

$PA_{a,s}$ = population fraction (excluding in-utero) of age category a and sex s

$RIF_{(prenatal),s}$ = fatal prenatal radiation injuries for sex s

$RINF_{(prenatal),s}$ = nonfatal prenatal radiation injuries for sex s.

A.3.6 DEATH (OPTION)

This subroutine calculates fatalities in each subsequent year by age category, cause of death and sex. A provision exists within the subroutine to distribute deaths using alternative epidemiological models. At present, a constant absolute risk model is used. Inputs to the subroutine are the distribution model option; fatalities by age category, cause of death and sex; life probabilities by age category and sex; periods of risk by cause of death; latency periods by age category and cause of death; mean survival times by cause of death and the median age of each age category. Except for acute fatalities, deaths are allocated to each year of a cohort's lifetime from the end of the minimum latent period and mean survival time to either the end of the risk period or the maximum age attainable. Thus, cancer fatalities are allocated to the years within this period, weighted by the probability that an individual will be alive in each year. Acute fatalities are allocated equally from the year after the latency period to the year of the latency period plus mean survival time. (In application, all acute fatalities occur in the base year.) The calculations used to allocate fatalities are

Acute Fatalities

$$F(n)_{a,s,d} = F_{a,s,d}/(MS_d)$$

subject to $LP_{a,d} < n \leq MS_d$

All Others

$$F(n)_{a,s,d} = F_{a,s,d} \cdot P(n)_{a,s} \Big/ \sum_{n=LM_{a,d}}^{M_{a,s}} P(n)_{a,s}$$

subject to $LP_{a,d} + MS_d < n \leq M_{a,s}$

and $LM_{a,d} = LP_{a,d} + MS_d$

where:

$F(n)_{a,s,d}$ = fatalities in year n, for age category a and sex s due to cause of death d

$P(n)_{a,s}$ = probability that an individual of age category a and sex s will be alive in year n

$F_{a,s,d}$ = total fatalities for age category a and sex s due to cause of death d

MS_d = median survival time for cause of death d

$LP_{a,d}$ = latency period for age category a and cause of death d

$M_{a,s}$ = remaining life period or period of risk, whichever is less, for age a and sex s at time of exposure ($M_{a,s}$ = A - median $age_{a,s}$).

The subroutine DEATH processes nested loops for sex, cause of death and age category. Within the innermost loop the cause of death is checked to determine whether or not it is acute. If it is, fatalities are distributed based on the equation described above for acute fatalities. If the death type is not acute, fatalities are distributed to each year using the constant absolute risk model shown above for all others.

A.3.6 RADCOST

This subroutine calculates the cost of treating radiation injuries. Input items are the cost of treating a radiation injury, the incidence of radiation injuries and the population fractions. Costs for all radiation injuries, except prenatal, are allocated to age categories based on their population fractions. Prenatal injuries are allocated entirely to the in-utero age cohort (except when running the median age case). The calculations for radiation injury treatment costs are

Direct Costs, All Acute Radiation Injuries but Prenatal

$$RC_{a,s,r} = (RIF_r + RINF_r) \cdot CPR_r \cdot PF_{a,s}$$

Direct Costs, Prenatal

$$RINF_p = EREM \cdot (PF_{p,male} + PF_{p,female}) \cdot 0.5$$

$$RC_{1,s,p} = (RIF_p + RINF_p) \cdot CPR_p \cdot PF_{p,s} / \sum_{s=1}^{2} PF_{p,s}$$

where

$RC_{a,s,r}$ = cost of treating a radiation injury of type r, for age category a and sex s

RIF_r = fatal radiation injury incidence of type r

$RINF_r$ = nonfatal radiation injury incidence of type r

CPR_r = treatment cost per case radiation injury r

$PF_{a,s}$ = population fraction for age category a, and sex s

EREM = population exposed to over 200 rem

p = prenatal.

A.3.7 CANCOST

This subroutine calculates the cost of treating cancers. Inputs to the subroutine include the incidence to fatality ratio (upon which CRAC2 fatality estimates are based) for each cancer type; the cost to treat each type of cancer; fatalities per year for each age category, death type and sex and an array for associating cancer treatment costs with death types. The equation used to calculate health effects is:

$$HC(n)_{a,s,c} = \sum_{n=1}^{A} \frac{CH_c \cdot IPF_c \cdot F(n)_{a,s,d} \cdot (1 + T/100)^{n-1}}{(1 + R/100)^{n-1}}$$

where

$HC(n)_{a,s,c}$ = present value of the cost of treating age category a and sex s for cancer type c in year n

CH_c = cost of treating one person for cancer type c

IPF_c = incidence to fatality ratio for cancer type c

$F(n)_{a,s,d}$ = fatalities in year n for age category a, sex s and death type d

R = real discount rate

T = rate of treatment cost growth.

The subroutine processes nested loops for sex, age category, cancer types, and years. Within these loops real cancer treatment costs for each year after exposure are calculated. In the next statement, these costs are discounted and added to the cost accumulator for each age category, sex and cancer type. The function FV is used to calculate the future value of base level treatment costs. The function PV is used to calculate the present value of future treatment costs.

A.3.8 LVALUE

This subroutine calculates labor value by age category and sex in each year after the base year. Inputs to the subroutine include income in the year of exposure by age category and sex, the median age of each cohort, labor force participation rates and the rate of labor productivity growth. The following equation is used to determine labor value in each year:

$$L(n)_{a,s} = L(1)_{a,s} \cdot PR_{a,s}(1 + W/100)^{n-1}$$

where

$L(n)_{a,s}$ = labor value in year n for age category a and sex s

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

W = rate of real earnings growth.

This calculation is controlled by three loops which process sex, age category and years, respectively. Within these loops 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.

A.3.9 LOSTLV

This subroutine calculates the present value of total lifetime labor value lost due to a premature fatality occurring in each year after population exposure. Inputs to the subroutine are median age by sex and age cohort; annual labor value by age category, sex, and number of years after exposure; life probabilities in each year by age and sex; and the discount rate. Lifetime labor value loss is calculated using the following equation:

$$LL(n)_{a,s} = \sum_{n=1}^{M_{a,s}} \frac{L(n)_{a,s} \cdot P(n)_{a-n,s}}{(1 + R/100)^{n-1}}$$

where

$LL(n)_{a,s}$ = lifetime labor value loss for a person dying n years after exposure of age category a in year of exposure, and sex s

$M_{a,s}$ = remaining life period or period of risk, whichever is less, for age category a and sex s

$L(n)_{a,s}$ = labor value in year n for a person of age category a in the year of exposure, and sex s

$P(n)_{a,s}$ = probability that a person of age category a and sex s will be alive in year n

R = real discount rate.

The subroutine processes nested loops for sex, age category and year after exposure. Within the inner loop the remaining life period is calculated and used as the termination year for accumulating real income loss. The function PV is used to discount each real income figure to the year of exposure as the income is accumulated. A person dying in a given year is assumed to lose all income in that year and all subsequent years until he would have reached an age equal to the maximum considered (A).

A.3.10 WORK

This subroutine calculates the value of lost work time. Inputs to the subroutine include fatalities per year, incidence to fatality ratios, the number of weeks of work missed for each cause of death, income for each age category and the prior probability that a person would be alive in each year after the year of exposure. The values of work lost due to cancers and due to radiation injuries are calculated separately using the following equations:

Radiation Injuries

$$RWC(1)_{a,s,r} = \frac{RINF_r \cdot LW_r \cdot PA_{a,s} \cdot L(1)_{a,s} \cdot P(1)_{a,s}}{52}$$

Cancers

$$WC(n)_{a,s,d} = \sum_{Ty=1}^{Td} \frac{F(n)_{a,s,d} \cdot IPF_d \cdot LW_d \cdot L(n)_{a,s} \cdot P(n)_{a,s}}{52 \cdot (1 + R/100)^{n-1} \cdot Ty}$$

where

$RWC(1)_{a,s,r}$ = value of lost work in year 1, for age category a, and sex s for radiation injury r

$RINF_r$ = incidence of nonfatal radiation injury r

LW_r = weeks of work lost for each type of radiation injury r

$PA_{a,s}$ = population fraction, excluding in utero, for age category a and sex s

$L(n)_{a,s}$ = income in year n for age category a, and sex s

$P(n)_{a,s}$ = probability that an individual in age category a, and sex s will be alive in year n

$WC(n)_{a,s,d}$ = value of lost work in year n, for age category a, and sex s for cause of death d

Td = years of treatment for cancer type d

Ty = sequential year of treatment

$F(n)_{a,s,d}$ = fatalities in year n for age category a, and sex s for cause of death d

IPF_d = incidence to fatality ratio for cause of death d

LW_d = total weeks of work lost for each cancer type d

R = real discount rate.

The cost of lost work is calculated by processing nested loops for sex, age category, death type, treatment time and years. Treatment time is included to spread lost work costs to more then one year if desired. The function PV is used to calculate the present value of income in any year. Radiation injury costs are calculated for the first year only.

A.3.11 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 fractions by

sex, and genetic effect elimination rates by type of genetic effect. The number of generations is user specified. The first step in the subroutine is to calculate first generation effects according to the following equation:

$$GE_{1,s,t} = (TG_t \cdot S_s \cdot IR_t) / \sum_{g=1}^{G} (1 - D_t)^{g-1}$$

where

$GE_{g,s,t}$ = major genetic effects of type t occuring in generation g for sex s (g = 1 for first generation)

TG_t = total genetic effects of type t

S_s = fraction of genetic effects allocated to sex s

IR_t = institutionalization rate for genetic effect t.

g = number of generations to a maximum of G

D_t = elimination rate for genetic effect t

The second step is to project major genetic effects for each remaining generation according to the following equation:

$$GE_{g,s,t} = GE_{g-1,s,t} \cdot (1-d_t)$$

The final step is to allocate the effects for each generation equally to the years within the generational period. The subroutine performs each of these steps separately. The output of the subroutine is a matrix of genetic effects by year, type of effect and sex.

A.3.12 GENCOST

This subroutine calculates the present value of direct and indirect costs attributable to the genetic effects allocated in GENDIST. Inputs to the subroutine include incidence of major genetic effects, median income, the cost of treating genetic effects, labor force participation rates and survival probabilities. Direct and indirect costs are calculated according to the following equations:

Direct

$$DG_{y,s,t} = \sum_{y+a=1}^{\text{max years}} \frac{IC_a \cdot P(a)_s \cdot GE_{y,s,t} \cdot (1 + T/100)^{y+a-1}}{(1 + R/100)^{y+a-1}}$$

Indirect

$$IG_{y,s,t} = \sum_{y+a=1}^{\text{max years}} \frac{L(1)_{a,s} \cdot PR_{a,s} \cdot P(a) \cdot GE_{y,s,t} \cdot (1 + W/100)^{y+a-1}}{(1 + R/100)^{y+a-1}}$$

where

$DG_{y,s,t}$ = present value of direct costs of genetic effect type t, for a person born in year y, of sex s

Σ

y = year of birth after year of population exposure

IC_a = cost of institutionalizing and treating genetic effects of an individual in age category a

$P(a)_s$ = probability that an individual of sex s will live to age a

$GE_{y,s,t}$ = genetic effects of type t, occurring in year y to sex s

T = rate of treatment cost growth

R = real discount rate

$y+a$ = years after birth in year y

$IG_{y,s,t}$ = present value of indirect costs of genetic effect type t, in a person born in year y, of sex s

$L(1)_{a,s}$ = median earnings in the base year for age category a and sex s

$PR_{a,s}$ = labor force participation rate for age category a and sex s

W = rate of real earnings growth

The subroutine processes nested loops for sex, genetic effect type and number of years in which effects occur. Within the innermost loop another loop accumulates the lifetime direct and indirect costs occurring to an individual born in each year. The subroutine assigns income and labor force participation rates in two ways, depending on the number of income categories being used.

A.3.13 SUMUP

This subroutine calculates total income loss and summarizes direct and indirect cost data for reporting purposes. Income loss due to fatalities is determined for all causes of death except thyroid cancer. The equation used to calculate income loss due to fatalities is

$$FC(n)_{a,s,d} = F(n)_{a,s,d} \cdot LL(n)_{a,s}$$

where

$FC(n)_{a,s,d}$ = lifetime, discounted real income loss in year n due to fatalities of type d, for age category a, and sex s

$F(n)_{a,s,d}$ = fatalities occurring in year n for age category a and sex s due to cause of death d

$LL(n)_{a,s}$ = discounted lifetime labor value loss of an individual in age category a and sex s dying n years after exposure.

Summary information is calculated by sex, age category, and health effects type, for direct and indirect costs.

A.3.14 WRITER

This subroutine prints out results. The following summary tables are printed with subtotals for each sex.

- indirect cost due to fatalities by age cohort
- indirect cost due to fatalities by cause of death
- indirect cost due to illness by age cohort
- indirect cost due to illness by cause of death
- indirect cost summary
- direct cost of radiation injuries by injury type
- direct cost of cancer by cancer type
- direct cost summary
- total cost summary.

A.3.15 PV

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

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

where

PV = present value of number

V(n) = value of number in year n

R = real discount rate

n = years to be discounted back to the year of exposure.

A.3.16 FV

This function calculates the future value of a number given the initial value, rate of growth and number of years in the future. The value is calculated using the following equation:

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

where

FV = future value of a number

V(b) = base level value of a number

G = real rate of growth

n = year of future value determination after the year of exposure.

A.3.17 INCCAT

This function determines the income category of each age cohort. The function checks first to see if the median data case is running. If it is, it returns an income category of one (1) since this case has only one income category. Otherwise, it checks to see if the current age of the cohort is zero (in utero). If it is, it also returns an income category of one (1) since in utero is the first income category. If the age is not zero the income category is determined by comparing the age to the upper age boundary of each income category. When the age is determined to be greater 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 income 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.

A.4 OUTPUT

Tables A.15 through A.24 provide samples of HECOM output for one case of each type of health effect. The health effects costs shown are those associated with the national data samples described in Section A.2. All costs are in 1981 dollars. The number of age categories; incidence of fatalities, radiation injuries, and cancers; rates of real income and health cost growth and discount rate are displayed, where appropriate, in the header above each table. Results are printed for each sex and for the entire exposed population. Indirect cost estimates are disaggregated by causes of death and age category. Direct costs are disaggregated by type of illness only.

TABLE A.15. HECOM Output: Indirect Costs Due to Fatalities

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

FATALITIES:

LEUKEMIA	1.0
LUNG	1.0
GI TRACT	1.0
BREAST	1.0
BONE	1.0
ALL OTHERS	1.0
THYROID	0.0
ACUTE	3.0
PRENATAL	1.0

INDIRECT COSTS DUE TO FATALITIES

DEATH CAUSE	MALE	FEMALE	TOTAL
LEUKEMIA	21597.	8227.	29824.
LUNG	2123.	1690.	3813.
GI TRACT	1799.	1366.	3155.
BREAST	0.	2651.	2651.
BONE	12862.	4715.	17577.
ALL OTHERS	1777.	1236.	3013.
THYROID	0.	0.	0.
ACUTE	192163.	111064.	303227.
TOTAL LOSS	232312.	130949.	363261.

TABLE A.16. HECOM Output: Indirect Costs Due to Fatalities by Age Category

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

FATALITIES:

LEUKEMIA	1.0
LUNG	1.0
GI TRACT	1.0
BREAST	1.0
BONE	1.0
ALL OTHERS	1.0
THYROID	0.0
ACUTE	3.0
PRENATAL	1.0

INDIRECT COSTS DUE TO FATALITIES

AGE CATEGORY	MALE	FEMALE	TOTAL
1	16295.	9469.	25765.
2	7337.	3226.	10563.
3	13721.	6321.	20042.
4	15400.	8654.	24054.
5	20732.	11410.	32141.
6	24324.	14043.	38367.
7	24075.	12853.	36928.
8	22430.	10855.	33285.
9	21078.	10114.	31192.
10	20693.	10265.	30958.
11	18098.	9773.	27871.
12	13864.	8523.	22387.
13	8794.	6660.	15455.
14	4082.	4477.	8559.
15	982.	2267.	3249.
16	309.	1237.	1546.
17	83.	596.	679.
18	15.	204.	219.
TOTAL LOSS	232312.	130949.	363261.

TABLE A.17. HECOM Output: Indirect Costs Due to Illness

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

ILLNESS AND INJURY INCIDENCE

CANCERS:	
LEUKEMIA	1.0
LUNG	1.0
GI TRACT	2.0
BREAST	1.3
BONE	1.2
ALL OTHERS	2.0
THYROID-BENIGN	0.6
THYROID-MALIGNANT	0.4
RADIATION INJURIES:	
PRODROMAL	1.0
BONE	1.0
LUNG	1.0
GI TRACT	1.0
PRENATAL	1.0

INDIRECT COSTS DUE TO ILLNESS

HEALTH EFFECT	MALE	FEMALE	TOTAL
CANCERS:			
LEUKEMIA	631.	252.	883.
LUNG	145.	110.	255.
GI TRACT	197.	167.	364.
BREAST	0.	156.	156.
BONE	628.	254.	882.
ALL OTHERS	165.	119.	284.
THYROID	15.	22.	36.
RADIATION:			
PRODROMAL	87.	51.	138.
BONE	2266.	1316.	3583.
LUNG	2266.	1316.	3583.
GI TRACT	2266.	1316.	3583.
PRENATAL	0.	0.	0.
TOTAL LOSS	8666.	5080.	13747.

TABLE A.18. HECOM Output: Indirect Costs Due to Illness by Age Category

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

ILLNESS AND INJURY INCIDENCE

CANCERS:

LEUKEMIA	1.0
LUNG	1.0
GI TRACT	2.0
BREAST	1.3
BONE	1.2
ALL OTHERS	2.0
THYROID-BENIGN	0.6
THYROID-MALIGNANT	0.4

RADIATION INJURIES:

PRODROMAL	1.0
BONE	1.0
LUNG	1.0
GI TRACT	1.0
PRENATAL	1.0

INDIRECT COSTS DUE TO ILLNESS

AGE CATEGORY	MALE	FEMALE	TOTAL
1	7.	4.	11.
2	47.	22.	69.
3	123.	53.	176.
4	111.	70.	181.
5	269.	175.	444.
6	691.	477.	1167.
7	895.	552.	1447.
8	972.	490.	1463.
9	1059.	480.	1539.
10	1120.	515.	1635.
11	1097.	518.	1615.
12	955.	506.	1460.
13	745.	449.	1194.
14	428.	368.	795.
15	99.	183.	282.
16	55.	104.	138.
17	10.	60.	71.
18	4.	56.	60.
TOTAL LOSS	8666.	5080.	13747.

TABLE A.19. HECOM Output: Indirect Cost Summary

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
NUMBER OF DEATH CATEGORIES= 8.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

INDIRECT COST SUMMARY

HEALTH EFFECT	MALE	FEMALE	TOTAL
CANCERS	41928.	20966.	62895.
RAD INJ+FATAL	199050.	115063.	314113.
GENETIC	596.	351.	947.
TOTAL LOSS	241574.	136380.	377954.

TABLE A.20. HECOM Output: Indirect Cost Summary by Age Category

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
NUMBER OF DEATH CATEGORIES= 8.0
RATE OF INCOME GROWTH=1.0
DISCOUNT RATE=10.0

INDIRECT COST SUMMARY

AGE CATEGORY	MALE	FEMALE	TOTAL
1	16303.	9473.	25775.
2	7384.	3248.	10632.
3	13944.	6375.	20219.
4	15511.	8724.	24235.
5	21001.	11585.	32586.
6	25015.	14520.	39535.
7	24970.	13405.	38375.
8	23402.	11345.	34747.
9	22137.	10595.	32731.
10	21812.	10780.	32593.
11	19195.	10290.	29486.
12	14819.	9028.	23847.
13	9540.	7109.	16649.
14	4510.	4845.	9355.
15	1081.	2450.	3532.
16	343.	1341.	1684.
17	93.	656.	749.
18	20.	260.	279.
TOTAL LOSS	240978.	136029.	377007.

TABLE A.21. HECOM Output: Direct Costs of Radiation Injuries

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF HEALTH COST GROWTH=1.0
DISCOUNT RATE=10.0

RADIATION INJURY INCIDENCE:

PRODROMAL	1.0
BONE	2.0
LUNG	2.0
GI TRACT	2.0
PRENATAL	1.0

DIRECT COSTS OF RADIATION INJURIES

INJURY TYPE	MALE	FEMALE	TOTAL
PRODROMAL	486.	514.	1000.
BONE	54432.	57568.	112000.
LUNG	3499.	3701.	7200.
GI TRACT	27216.	28784.	56000.
PRENATAL	50001.	50001.	100002.
TOTAL LOSS	135634.	140568.	276202.

TABLE A.22. HECOM Output: Direct Costs of Cancers

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
RATE OF HEALTH COST GROWTH=1.0
DISCOUNT RATE=10.0

CANCER INCIDENCE:

LEUKEMIA	1.0
LUNG	1.0
GI TRACT	2.0
BREAST	1.3
BONE	1.2
ALL OTHERS	2.0
THYROID-BENIGN	0.6
THYROID-MALIGNANT	0.4

DIRECT COST OF CANCERS

CANCER TYPE	MALE	FEMALE	TOTAL
LEUKEMIA	3755.	2529.	6284.
LUNG	1147.	1213.	2360.
GI TRACT	1701.	1826.	3528.
BREAST	0.	860.	860.
BONE	5935.	3997.	9932.
ALL OTHERS	1025.	1057.	2082.
THYROID-BENIGN	63.	162.	225.
THYROID-MALIGNANT	46.	118.	164.
TOTAL LOSS	13672.	11763.	25435.

TABLE A.23. HECOM Output: Direct Cost Summary

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
NUMBER OF CANCER TYPES= 8.0
NUMBER OF RADIATION INJURIES= 5.0
RATE OF HEALTH COST GROWTH=1.0
DISCOUNT RATE=10.0

DIRECT COST SUMMARY

HEALTH EFFECT	MALE	FEMALE	TOTAL
CANCERS	13672.	11763.	25435.
RAD INJURIES	135634.	140568.	276202.
GENETIC	2759.	2781.	5541.
TOTAL LOSS	152066.	155113.	307178.

TABLE A.24. HECOM Output: Total Cost Summary

HEALTH EFFECTS COST MODEL

NUMBER OF AGE CATEGORIES=18.0
NUMBER OF CANCER TYPES= 6.0
NUMBER OF RADIATION INJURY TYPES= 5.0
RATE OF INCOME GROWTH=1.0
RATE OF HEALTH COST GROWTH=1.0
DISCOUNT RATE=10.0

TOTAL COST SUMMARY

HEALTH EFFECT	MALE	FEMALE	TOTAL
CANCERS	55601.	32729.	88330.
RAD INJURIES	334684.	255631.	590315.
GENETIC	3355.	3132.	6488.
TOTAL LOSS	393640.	291493.	685132.

APPENDIX B

PRELIMINARY HECOM COMPUTER CODE

```
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
CC      MAIN PROGRAM CONTAINING SUBROUTINE CALL STATEMENTS
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C-------------------------------------------------------------------------
C       INSERT COMMON BLOCK AND PARAMETER INFORMATION FROM FILE 'CONTROL.FOR'
C-------------------------------------------------------------------------
        INCLUDE 'CONTROL.FOR'
C-----------------------------------------
C       CALL SUBROUTINE READER TO READ IN DATA
C-----------------------------------------
        CALL READER
C-----------------------------------------------------
C       CALCULATE LIFE PROBABILITIES FOR EACH AGE CATEGORY
C-----------------------------------------------------
        CALL SPROB
C-------------------------------
C       CALCULATE LATENCY PERIODS
C-------------------------------
        CALL LATENCY
C-------------------------------------------------
C       CALCULATE COST OF TREATING RADIATION INJURIES
C-------------------------------------------------
        CALL RADCOST
C-----------------------------------------------------
C       CALCUALTE FATALITIES BY AGE CATEGORY AND DEATH TYPE
C-----------------------------------------------------
        CALL FATAL
C-----------------------------------------------------------
C       CALCULATE FATALITIES IN EACH YEAR USING SUBROUTINE DEATH.
C-----------------------------------------------------------
        CALL DEATH(1)
C-----------------------------------------
C       CALCULATE COST OF TREATING HEALTH EFFECTS
C-----------------------------------------
        CALL CANCOST
C-------------------------------------------------------------
C       CALCULATE LABOR VALUE FOR EACH AGE CATAGORY IN EACH YEAR
C-------------------------------------------------------------
100     CALL LVALUE
```

```
C--------------------------------------------------------------------------
C       CALCULATE LABOR VALUE LOSS PER PERSON IN EACH YEAR A DEATH OCCURS
C--------------------------------------------------------------------------
        CALL LOSTLV
C--------------------------------------------------------------
C       CALCULATE COST DUE TO WORK LOSS IN YEAR BEFORE DEATH
C--------------------------------------------------------------
        CALL WORK
C-------------------------------------------
C       CALCULATE GENETIC EFFECTS PER YEAR
C-------------------------------------------
        CALL GENDIST
C---------------------------------------------------
C       CALCULATE DIRECT AND INDIRECT GENETIC COSTS
C---------------------------------------------------
        CALL GENCOST
C-----------------------
C       SUMMARIZE RESULTS
C-----------------------
        CALL SUMUP


C-----------------------
C       PRINT OUT RESULTS
C-----------------------
        CALL WRITER
200     STOP
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE GENDIST
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C       THIS SUBROUTINE DISTRIBUTES GENETIC EFFECTS TO EACH YEAR AFTER
C       EXPOSURE
C
        INCLUDE 'CONTROL.FOR'
        INTEGER YPG
```

```
C-------------------------------------------------
C       CALCULATE THE NUMBER OF YEARS PER GENERATION
C-------------------------------------------------
        YPG=GYEARS/NGEN
C---------------------------------------
C       CALCULATE FIRST GENERATION EFFECTS
C---------------------------------------
        DO 2000 I=1,SEX
        DO 2000 J=1,GTYPES
        DO 1000 K=1,NGEN
C-------------------------
C       SUM UP DECAY DIVISOR
C-------------------------
        SUM=SUM+(1-DRATE(J))**(K-1)
1000    CONTINUE
C-----------------------------------------
C       USE SUM TO COMPUTE FIRST YEAR EFFECTS
C-----------------------------------------
        GEPG(J,1,I)=(GI(J)*GSRATE(I)*INRATE(J))/SUM
C-----------------
C       ZERO OUT SUM
C-----------------
        SUM=0
2000    CONTINUE
C-------------------------------------------------
C       CALCULATE EFFECTS IN REMAINING GENERATIONS
C-------------------------------------------------
        DO 3000 I=1,SEX
        DO 3000 J=1,GTYPES
        DO 3000 K=2,NGEN
        GEPG(J,K,I)=GEPG(J,K-1,I)*(1-DRATE(J))
3000    CONTINUE
C-----------------------------------------------------------
C       ALLOCATE GENERATIONAL EFFECTS EQUALLY TO EACH YEAR WITHIN GENERATION
C-----------------------------------------------------------
        DO 4000 I=1,SEX
        DO 4000 J=1,GTYPES
        DO 4000 K=1,NGEN
        DO 4000 L=1,YPG
        GEPY(J,((K-1)*YPG)+L,I)=GEPG(J,K,I)/YPG
4000    CONTINUE
        RETURN
        END
```

```
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC



      SUBROUTINE GENCOST
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C     THIS SUBROUTINE CALCULATES THE DIRECT AND INDIRECT COST OF
C     GENETIC EFFECTS
C
      INCLUDE 'CONTROL.FOR'
      REAL LABOR
C
      DO 2000 I=1,SEX
      DO 2000 J=1,GTYPES
      DO 2000 K=1,GYEARS
      DO 2000 L=1,YEARS
C-----------------------------------------
C     DETERMINE AGE CATEGORY BEING PROCESSED
C-----------------------------------------
      AGE=FLOAT(L-1)
      KK=GINC
      KAT=INCCAT(AGE,1,KK)
C-------------------------------------------------------
C     SUM UP LIFETIME DIRECT COST OF INSTITUTIONALIZATION
C-------------------------------------------------------
      DGCOST(J,K,I)=DGCOST(J,K,I)+PV(FV(INCOST(KAT),RHG,K+L-1)
     &  *GLPROB(L,I)*GEPV(J,K,I),R,K+L-1)
C-------------------------------------------------------------
C     GET ST UP TO CALCULAT INDIRECT COSTS.  IF GAC AND AC ARE
C     EQUAL DETERMINE EARNINGS (EARN) AND LABOR FORCE PARTICIPATION
C     (LABOR) BASED ON GENETIC CATEGORIES.
C-------------------------------------------------------------
      IF (AC.NE.GAC) GOTO 1000
      AGE=FLOAT(L)
      KAT=INCCAT(AGE,1,KK)
      EARN=MI(KAT,I)
      LABOR=LFPR(KAT,I)
      GOTO 1800
```

```
C-----------------------------------------------------------------------
C       IF GAC AND AC ARE NOT EQUAL DETERMINE EARNINGS AND LABOR FORCE
C       PARTICIPATION RATES BASED ON INCOME CATEGORIES.
C-----------------------------------------------------------------------
1000    CONTINUE
        KK=IINC
        AGE=FLOAT(L)
        LL=INCCAT(AGE,1,KK)
        EARN=MI(LL,I)
        LABOR=LFPR(LL,I)
1800    CONTINUE
C-----------------------------------------------------------------------
C       SUM UP LIFETIME INDIRECT COST OF EXPECTED LABOR VALUE LOSS
C-----------------------------------------------------------------------
        IDGCOST(J,K,I)=IDGCOST(J,K,I)+PV(FV(EARN,RIG,K+L-1)
     &                  *LABOR*GLPROB(L,I)*GEPY(J,K,I),R,K+L-1)
        EARN=0
        LABOR=0
2000    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE RADCOST
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC



C
C       THIS SUBROUTINE CALCULATES THE COST OF TREATING RADIATION INJURIES
C
        INCLUDE 'CONTROL.FOR'
C-----------------------------------------------------------------------
C       THE PRENATAL RADIATION INCIDENCE VALUE REPRESENTS THE NUMBER OF
C       PEOPLE EXPOSED TO OVER 200 REMS.  THIS NUMBER MUST BE ADJUSTED TO
C       REPRESENT ONLY PRENATAL INJURIES.
C-----------------------------------------------------------------------
        IF (PRENAT.NE.0) RADINF(PRENAT)=RADINF(PRENAT)*
     &                   (POPF(1,1)+POPF(1,2))*0.5
```

```
C-----------------------------------------------------------------------
C       COST IS CALCULATED BY MULTIPLYING COST PER CASE BY THE NUMBER
C       OF CASES AND DISTRIBUTING THESE COSTS TO AGE CATEGORIES BASED
C       ON POPULATION FRACTIONS.  IF THE RADIATION INJURY IS PRENATAL THEN
C       ALL INJURIES ARE ASSUMED TO BE IN UTERO.
C-----------------------------------------------------------------------
        DO 100 I=1,RTYPES
        DO 100 J=1,AC
        DO 100 K=1,SEX
                IF (I.NE.PRENAT) RCOST(J,I,K)=(RADINF(I)+RADIF(I))
     &                          *CPRAD(I)*POPF(J,K)
                IF (I.EQ.PRENAT.AND.J.EQ.1) RCOST(J,I,K)=(RADIF(1)
     &           +RADINF(I))*CPRAD(I)*POPF(1,K)/(POPF(1,1)+POPF(1,2))
100     CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE CANCOST
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C       THIS SUBROUTINE CALCULATES THE PRESENT VALUE COST OF TREATING
C       CANCERS RESULTING FROM RADIATION EXPOSURE.
C
        INCLUDE 'CONTROL.FOR'
C-----------------------------------------------------------------------
C       TREATMENT COST IN A YEAR IS CALCULATED BY MULTIPLYING THE FATALITIES
C       IN THE NEXT YEAR BY THE INCIDENCE PER FATALITY AND THE REAL COST PER
C       INCIDENCE.  THIS COST IS THEN DISCOUNTED TO THE YEAR OF EXPOSURE.
C-----------------------------------------------------------------------
        DO 1000 I=1,SEX
        DO 1000 J=1,AC
        DO 1000 K=1,CTYPES
        DO 1000 L=1,YEARS-1
                Z=FV(CPI(K),RHG,L)*IPF(K)*FPY(CFCONV(K),J,L+1,I)
                CCOST(J,K,I)=CCOST(J,K,I)+PV(Z,R,L)
                Z=0
1000    CONTINUE
        RETURN
        END
```

```
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
      SUBROUTINE SUMUP
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C     THIS SUBROUTINE SUMMARIZES FATALITY AND WORK LOSS DATA
C
      INCLUDE 'CONTROL.FOR'
C-----------------------------------------------------------------------



C     CALCULATE TOTAL LABOR VALUE LOSSES AND SUMMARIZE LOSSES BY DEATH TYPE,
C     AGE CATEGORY AND YEAR.
C-----------------------------------------------------------------------
      DO 2000 L=1,SEX
      DO 2000 J=1,AC
      DO 1000 I=1,YEARS
      DO 1000 K=1,DTYPES
C-----------------------------------------------------------------------
C           CALCULATE TOTAL LABOR VALUE LOSS DUE TO FATALITIES.  IF DEATH
C           TYPE IS THYROID THEN DO NOT PERFORM COMPUTATION.
C-----------------------------------------------------------------------
            IF (K.NE.THYROID) TLVLOSS(J,I,K,L)=LVLOSS(J,I,L)*FPY(K,J,I,L)
C-----------------------------------------------------------
C           SUMMARIZE LABOR VALUE LOSS BY DEATH TYPE
C-----------------------------------------------------------
            SDTLOSS(K,L)=SDTLOSS(K,L)+TLVLOSS(J,I,K,L)
C-----------------------------------------------------------
C           SUMMARIZE LABOR VALUE LOSS BY AGE CATEGORY
C-----------------------------------------------------------
            SACLOSS(J,L)=SACLOSS(J,L)+TLVLOSS(J,I,K,L)
C-----------------------------------------------------------
C
C           CALCULATE SUMMARY STATISTICS FOR LOST WORK
C
C           SUMMARIZE LOSS BY DEATH TYPE
C-----------------------------------------------------------
            SDTLW(K,L)=SDTLW(K,L)+LWCOST(J,I,K,L)
```

```
C-----------------------------------------
C                SUMMARIZE LOSS BY AGE CATEGORY
C-----------------------------------------
                 SACCLW(J,L)=SACCLW(J,L)+LWCCOST(J,I,K,L)
1000     CONTINUE
C-------------------------------------------------------
C        CALCULATE SUMMARY STATISTICS FOR RADIATION TREATMENT
C        AND ILLNESS
C-------------------------------------------------------
         DO 1400 K=1,RTYPES
C-----------------------------------------
C                SUMMARIZE COST BY AGE CATEGORY
C-----------------------------------------
                 SACRAD(J,L)=SACRAD(J,L)+RCOST(J,K,L)
                 SACRLW(J,L)=SACRLW(J,L)+LWRCOST(J,1,K,L)
C-------------------------------------------------
C                SUMMARIZE COST BY RADIATION INJURY TYPE
C-------------------------------------------------
                 SRTRAD(K,L)=SRTRAD(K,L)+RCOST(J,K,L)
                 SRTLW(K,L)=SRTLW(K,L)+LWRCOST(J,1,K,L)
1400     CONTINUE
C-----------------------------------------------
C        CALCULATE SUMMARY STATISTICS FOR CANCER COSTS
C-----------------------------------------------
         DO 1500 K=1,CTYPES
C-----------------------------------------
C                SUMMARIZE COST BY AGE CATEGORY
C-----------------------------------------
                 SACCAN(J,L)=SACCAN(J,L)+CCOST(J,K,L)
C-----------------------------------------
C                SUMMARIZE COST BY HEALTH TYPE
C-----------------------------------------
                 SCTCAN(K,L)=SCTCAN(K,L)+CCOST(J,K,L)
1500     CONTINUE
2000     CONTINUE



C-------------------------------------------------------------
C        COMPUTE TOTALS FOR PRINT OUT FOR AGE CATEGORIES AND TOTALS BY SEX
C        AND FOR SEXES COMBINED
C-------------------------------------------------------------
```

```
      DO 3000 I=1,AC
      DO 3000 J=1,SEX
              TACLOSS(I)=TACLOSS(I)+SACLOSS(I,J)
              TACCLW(I)=TACCLW(I)+SACCLW(I,J)
              TACRLW(I)=TACRLW(I)+SACRLW(I,J)
              SACLW(I,J)=SACCLW(I,J)+SACRLW(I,J)
              SLWCOST(J)=SLWCOST(J)+SACCLW(I,J)+SACRLW(I,J)
              SCOST(J)=SCOST(J)+SACLOSS(I,J)
              TACLW(I)=TACLW(I)+SACLW(I,J)
              TACRAD(I)=TACRAD(I)+SACRAD(I,J)
              TACCAN(I)=TACCAN(I)+SACCAN(I,J)
              SDRAD(J)=SDRAD(J)+SALRAD(I,J)
              SDCAN(J)=SDCAN(J)+SACCAN(I,J)
3000  CONTINUE
C-------------------------------------------------------------
C     CALCULATE TOTAL LOSSES FOR FATALITIES AND ILLNESS BY DEATH TYPE
C-------------------------------------------------------------
      DO 3500 J=1,SEX
      DO 3600 I=1,DTYPES
              TDTLOSS(I)=TDTLOSS(I)+SDTLOSS(I,J)
              TDTLW(I)=TDTLW(I)+SDTLW(I,J)
3600  CONTINUE
C-------------------------------------------------------------
C     CALCULATE TOTAL LOSSES FOR ILLNESS AND RADIATION INJURY TREATMENT
C-------------------------------------------------------------
      DO 3700 I=1,RTYPES
              TRTRAD(I)=TRTRAD(I)+SRTRAD(I,J)
              TRTLW(I)=TRTLW(I)+SRTLW(I,J)
3700  CONTINUE
C-------------------------------------------------------------
C     CALCULATE TOTAL LOSSES FOR ILLNESS AND RADIATION INJURY TREATMENT
C-------------------------------------------------------------
      DO 3600 I=1,CTYPES
              TCTCAN(I)=TCTCAN(I)+SCTCAN(I,J)
3800  CONTINUE
3500  CONTINUE
C-------------------------------------------
C     CALCULATE TOTAL CANCER TREATMENT COSTS
C-------------------------------------------
      DO 3900 I=1,SEX
              TCOST=TCOST+SCOST(I)
              TLWCOST=TLWCOST+SLWCOST(I)
```

```
                TDRAD=TDRAD+SDRAD(I)
                TDCAN=TDCAN+SDCAN(I)
3900    CONTINUE
C*********************************
C
C       CALCULATE GENETIC SUMMARY
C
C*********************************
        DO 3950 I=1,SEX
        DO 3940 J=1,GTYPES
        DO 3930 K=1,GYEARS
        SDGTGEN(J,I)=SDGTGEN(J,I)+DGCOST(J,K,I)
        SIDGTGEN(J,I)=SIDGTGEN(J,I)+IDGCOST(J,K,I)
3930    CONTINUE
        SDGEN(I)=SDGEN(I)+SDGTGEN(J,I)
        SIDGEN(I)=SIDGEN(I)+SIDGTGEN(J,I)



3940    CONTINUE
        TDGEN=TDGEN+SDGEN(I)
        TIDGEN=TIDGEN+SIDGEN(I)
3950    CONTINUE
C************************************
C
C       COMPUTE INDIRECT COST SUMMARY
C
C************************************
C
C-------------------------------------------------------------------
C       CALCULATE INDIRECT CANCER COSTS BY SEX AND TOTAL.  ACUTE DEATHS ARE
C       NOT INCLUDED AS PART OF CANCER TOTAL.
C-------------------------------------------------------------------
        DO 4000 I=1,DTYPES
        IF (I.EQ.ACUTE) GOTO 4000
                TIDCAN=TIDCAN+TDTLW(I)+TDTLOSS(I)
                DO 4000 J=1,SEX
                        SIDCAN(J)=SIDCAN(J)+SDTLW(I,J)+SDTLOSS(I,J)
4000    CONTINUE
C-----------------------------------------------
C       ADD ILLNESS COSTS TO INDIRECT RADIATION COSTS
C-----------------------------------------------
```

```
      DO 4050 I=1,RTYPES
              TIDRAD=TIDRAD+THTLW(I)
              DO 4050 J=1,SEX
                      SIDRAD(J)=SIDRAD(J)+SRTLW(I,J)
4050  CONTINUE
C-----------------------------------------------------------
C     ADD IN ACUTE FATALITY COSTS TO INDIRECT RADIATION COSTS
C-----------------------------------------------------------
      TIDRAD=TIDRAD+TOTLOSS(ACUTE)
      DO 4060 I=1,SEX
              SIDRAD(I)=SIDRAD(I)+SOTLOSS(ACUTE,I)
4060  CONTINUE
C-----------------------------------------------------
C     CALCULATE TOTAL INDIRECT COSTS BY AGE CATEGORY
C-----------------------------------------------------
      DO 4100 I=1,AC
              TIDAC(I)=TACCLW(I)+TACRLW(I)+TACLOSS(I)
              DO 4100 J=1,SEX
                      SIDAC(I,J)=SACCLW(I,J)+SACRLW(I,J)+SACLOSS(I,J)
4100  CONTINUE
C-----------------------------------------------------
C     CALCULATE TOTAL AND SEX SPECIFIC INDIRECT COSTS
C-----------------------------------------------------
      DO 4200 I=1,SEX
              SACID(I)=SIDRAD(I)+SIDCAN(I)
              SID(I)=SIDRAD(I)+SIDCAN(I)+SIDGEN(I)
              TACID=TACID+SACID(I)
              TID=TID+SID(I)
4200  CONTINUE
C*********************************************************
C
C     COMPUTE DIRECT COST SUMMARY
C
C*********************************************************
C
C-----------------------------------------------------
C     CALCULATE TOTAL AND SEX SPECIFIC DIRECT COSTS
C-----------------------------------------------------
      DO 5000 I=1,AC
```

```
         TDAC(I)=TACCAN(I)+TACRAD(I)
         DO 5000 J=1,SEX
                  SDAC(I,J)=SACCAN(I,J)+SACRAD(I,J)
                  SD(J)=SD(J)+SDAC(I,J)
5000     CONTINUE
         DO 5400 I=1,SEX
                  SD(I)=SD(I)+SDGEN(I)
                  TD=TD+SD(I)
5400     CONTINUE
C***********************************************************
C
C        FINALLY, CALCULATE TOTAL HEALTH EFFECT COSTS
C
C***********************************************************
         DO 5500 I=1,SEX
                  SCAN(I)=SIDCAN(I)+SDCAN(I)
                  SRAD(I)=SIDRAD(I)+SDRAD(I)
                  SGEN(I)=SIDGEN(I)+SDGEN(I)
                  ST(I)=SD(I)+SID(I)
                  TCAN=TCAN+SCAN(I)
                  TRAD=TRAD+SRAD(I)
                  TGEN=TGEN+SGEN(I)
                  TT=TT+ST(I)
5500     CONTINUE
         RETURN
         END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
         SUBROUTINE WRITER
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C        SUBROUTINE TO WRITE OUT SUMMARY DATA
C
C        FIRST INCLUDE COMMON BLOCK AND CONTROL PARAMETERS
C
         INCLUDE 'CONTROL.FOR'
C
C        PRINT OUT HEADER
C
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CCCCCCCCCCCCCCC TEST WRITE SECTION CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
```

```
      GOTO 1234
      DO 666 I=1,SEX
      DO 666 J=1,AC
      WRITE(6,1002) J, (PI(K,J,1),K=1,8)
666   CONTINUE
      WRITE(6,1101)
      DO 667 I=1,SEX
      DO 667 J=1,AC
      WRITE(6,1002) J,(FAT(K,J,I),K=1,8)
667   CONTINUE
      GOTO 4321
      WRITE(6,1101)
      DO 771 I=1,YEARS
771   WRITE(6,1001) I,(LV(J,I,1),J=1,9)
1001  FORMAT(1X,I2,9F10.0)
      WRITE(6,1101)
      DO 772 I=1,YEARS
772   WRITE(6,1001) I, (LV(J,I,1),J=10,18)
1002  FORMAT(1X,I2,8F10.6)
```



```
      WRITE(6,1101)
      DO 773 I=1,YEARS
773   WRITE(6,1001) I,(LVLOSS(J,I,1),J=1,9)
      WRITE(6,1101)
      DO 774 I=1,YEARS
774   WRITE(6,1001) I,(LVLOSS(J,I,1),J=10,18)
      DO 775 I=1,YEARS
775   WRITE(6,1003) I,LIFEP(I,1),(LPROB(J,I,1),J=1,8)
1003  FORMAT(1X,I4,9F10.6)
      DO 776 I=1,AC
      WRITE(6,1101)
      DO 776 J=1,YEARS
776   WRITE(6,1004) I,J,(TLVLOSS(I,J,K,1),K=1,8)
4321  WRITE(6,1101)
      DO 779 I=1,AC
      WRITE(6,1101)
      DO 779 J=1,YEARS
779   WRITE(6,1006) I,J,(FPY(K,I,J,1),K=1,8)
      GOTO 5556
```

```
      WRITE(6,1101)
      DO 777 J=1,SEX
      DO 777 I=1,AC
777   WRITE(6,1005) I,J,(LWRCOST(I,1,K,J),K=1,5)
      WRITE(6,1101)
      GOTO 5556
      DO 778 I=1,AC
778   WRITE(6,1009) I,(CCOST(I,J,1),J=1,CTYPES)
1009  FORMAT(1X,I3,8F10.2)
1004  FORMAT(1X,2I4,8F10.2)
1005  FORMAT(1X,2I4,5F10.2)
1006  FORMAT(1X,2I4,6F10.5)
1101  FORMAT(////)
5556  CONTINUE
1234  WRITE(6,5) CASE
C*****************************************************
C
C     WRITE OUT FATALITY SUMMARY
C
C*****************************************************
      WRITE(6,10) AC,RIG,R
      WRITE(6,810)
      DO 1000 I=1,DTYPES
      IF (I.EQ.THYROID) WRITE(6,811) DTNAMES(I),0.0
      IF (I.NE.THYROID) WRITE(6,811) DTNAMES(I),CF(I)
1000  CONTINUE
      WRITE(6,811) RNAMES(PRENAT),(RADINF(PRENAT)+RADIF(PRENAT))
      WRITE(6,20)
C-----------------------------------------
C     WRITE OUT COSTS FOR EACH DEATH TYPE
C-----------------------------------------
      DO 1100 I=1,DTYPES
      WRITE(6,30) DTNAMES(I), (SUTLOSS(I,J),J=1,SEX),TOTLOSS(I)
1100  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,40) SCOST(1),SCOST(2),TCOST
C-----------------------------------------------------
C     NOW WRITE OUT COST BY AGE CATEGORY - FIRST HEADER
C-----------------------------------------------------
      WRITE(6,10) AC,RIG,R
      WRITE(6,810)
```

```
      DO 1150 I=1,DTYPES
      IF (I.EQ.THYROID) WRITE(6,811) DTNAMES(I),0.0
      IF (I.NE.THYROID) WRITE(6,811) DTNAMES(I),CF(I)
1150  CONTINUE
      WRITE(6,811) RNAMES(PRENAT),(RADINF(PRENAT)+RADIF(PRENAT))
C-----------------------------
C     WRITE OUT TABLE CAPTION
C-----------------------------
      WRITE(6,50)
C--------------------
C     WRITE OUT DATA
C--------------------
      DO 1200 I=1,AC
      WRITE(6,60) I,(SACLOSS(I,J),J=1,SEX),TACLOSS(I)
1200  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,70) SCOST(1),SCOST(2),TCOST
C*************************************************
C
C     WRITE OUT RESULTS FOR MISSED WORK SUMMARY
C
C*************************************************
      WRITE(6,10) AC,RIG,R
      WRITE(6,820)
      WRITE(6,821)
      DO 1300 I=1,CTYPES
      WRITE(6,845) CNAMES(I),(CF(CFCONV(I))*IPF(I))
1300  CONTINUE
      WRITE(6,825)
      DO 1400 I=1,RTYPES
      WRITE(6,811) RNAMES(I),RADINF(I)
1400  CONTINUE
C-------------------------------------------------
C     WRITE OUT TABLE CAPTIONS FOR DEATH TYPE SUMMARY
C-------------------------------------------------
      WRITE(6,120)
C---------------------------------------
C     WRITE OUT COSTS FOR EACH DEATH TYPE
C---------------------------------------
      WRITE(6,33) 'CANCERS:  '
```

```
      DO 2000 I=1,DTYPES
      IF (I.EQ.ACUTE) GOTO 2000
      WRITE(6,31) DTNAMES(I), (SDTLW(I,J),J=1,SEX),TDTLW(I)
2000  CONTINUE
      WRITE(6,33) 'RADIATION:'
      DO 2100 I=1,RTYPES
      WRITE(6,31) RNAMES(I), (SRTLW(I,J),J=1,SEX),TRTLW(I)
2100  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,40) SLWCOST(1),SLWCOST(2),TLWCOST
C-------------------------------------------------------
C     NOW WRITE OUT COST BY AGE CATEGORY - FIRST HEADER
C-------------------------------------------------------
      WRITE(6,10) AC,RIG,R
      WRITE(6,820)
      WRITE(6,821)
      DO 2150 I=1,CTYPES
      WRITE(6,845) CNAMES(I),(CF(CFCONV(I))*IPF(I))



2150  CONTINUE
      WRITE(6,825)
      DO 2160 I=1,RTYPES
      WRITE(6,811) RNAMES(I),RADINF(I)
2160  CONTINUE
C-----------------------------
C     WRITE OUT TABLE CAPTION
C-----------------------------
      WRITE(6,150)
C--------------------
C     WRITE OUT DATA
C--------------------
      DO 2200 I=1,AC
      WRITE(6,60) I,(SACLW(I,J),J=1,SEX),TACLW(I)
2200  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
```

```
      WRITE(6,70) SLWCOST(1),SLWCOST(2),TLWCOST
C*************************************
C
C     WRITE OUT INDIRECT COST SUMMARY
C
C*************************************
      WRITE(6,510) AC,DTYPES,RIG,R
C-------------------------------------------------
C     WRITE OUT TABLE CAPTIONS FOR SUMMARY DEATH TYPE
C-------------------------------------------------
      WRITE(6,520)
C---------------------------------
C     WRITE OUT COSTS FOR EACH DEATH TYPE
C---------------------------------
      WRITE(6,30) 'CANCERS     ', (SIDCAN(J),J=1,SEX),TIDCAN
      WRITE(6,34) 'RAD INJ+FATAL', (SIDRAD(J),J=1,SEX),TIDRAD
      WRITE(6,30) 'GENETIC     ', (SIDGEN(J),J=1,SEX),TIDGEN
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,40) SID(1),SID(2),TID
C-------------------------------------------------
C     NOW WRITE OUT COST BY AGE CATEGORY - FIRST HEADER
C-------------------------------------------------
      WRITE(6,510) AC,DTYPES,RIG,R
C---------------------------
C     WRITE OUT TABLE CAPTION
C---------------------------
      WRITE(6,550)
C--------------------
C     WRITE OUT DATA
C--------------------
      DO 3200 I=1,AC
      WRITE(6,60) I,(SIDAC(I,J),J=1,SEX),TIDAC(I)
3200  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,70) SACID(1),SACID(2),TACID
C*************************************************
C
C     WRITE OUT RESULTS FOR RADIATION TREATMENT COSTS
C
C*************************************************
```

```
      WRITE(6,11) AC,RHG,R
      WRITE(6,830)
      DO 3300 I=1,RTYPES
      WRITE(6,811) RNAMES(I),(RADIF(I)+RADINF(I))
3300  CONTINUE
C-------------------------------------------------------
C     WRITE OUT TABLE CAPTIONS FOR RADIATION TYPE SUMMARY
C-------------------------------------------------------
      WRITE(6,320)
C--------------------------------------
C     WRITE OUT COSTS FOR EACH DEATH TYPE
C--------------------------------------
      DO 4000 I=1,RTYPES
      WRITE(6,30) RNAMES(I), (SRTRAD(I,J),J=1,SEX),TRTRAD(I)
4000  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
      WRITE(6,40) SDRAD(1),SDRAD(2),TDRAD
C-------------------------------------------------------
C     NOW WRITE OUT COST BY AGE CATEGORY - FIRST HEADER
C-------------------------------------------------------
C     WRITE(6,11) AC,RHG,R
C     WRITE(6,830)
C     DO 4100 I=1,RTYPES
C     WRITE(6,811) RNAMES(I),(RADINF(I)+RADIF(I))
4100  CONTINUE
C-----------------------------
C     WRITE OUT TABLE CAPTION
C-----------------------------
C     WRITE(6,350)
C--------------------
C     WRITE OUT DATA
C--------------------
C     DO 4200 I=1,AC
C     WRITE(6,60) I,(SACRAD(I,J),J=1,SEX),TACRAD(I)
4200  CONTINUE
C----------------------
C     WRITE OUT TOTALS
C----------------------
C     WRITE(6,70) SDRAD(1),SDRAD(2),TDRAD
```

```
C*****************************************************
C
C       WRITE OUT RESULTS FOR CANCER TREATMENT COSTS
C
C*****************************************************
        WRITE(6,12) AC,RHG,R
        WRITE(6,840)
        DO 4500 I=1,CTYPES
        WRITE(6,845) CNAMES(I),(CF(CFCONV(I))*IPF(I))
4500    CONTINUE
C-------------------------------------------------------
C       WRITE OUT TABLE CAPTIONS FOR CANCER COST SUMMARY
C-------------------------------------------------------
        WRITE(6,420)
C--------------------------------------
C       WRITE OUT COSTS FOR EACH CANCER
C--------------------------------------
        DO 5000 I=1,CTYPES
        WRITE(6,450) CNAMES(I), (SCTCAN(I,J),J=1,SEX),TCTCAN(I)
5000    CONTINUE
C----------------------
```



```
C       WRITE OUT TOTALS
C----------------------
        WRITE(6,440) SDCAN(1),SDCAN(2),TDCAN
C-------------------------------------------------------
C       NOW WRITE OUT COST BY AGE CATEGORY - FIRST HEADER
C-------------------------------------------------------
C       WRITE(6,12) AC,RHG,R
C       WRITE(6,840)
C       DO 5100 I=1,CTYPES
C       WRITE(6,845) CNAMES(I),(CF(CFCONV(I))*IPF(I))
5100    CONTINUE
C-----------------------------
C       WRITE OUT TABLE CAPTION
C-----------------------------
C       WRITE(6,450)
C--------------------
C       WRITE OUT DATA
```

```
C--------------------
C       DO 5200 I=1,AC
C       WRITE(6,60) I,(SACCAN(I,J),J=1,SEX),TACCAN(I)
5200    CONTINUE
C----------------------
C       WRITE OUT TOTALS
C----------------------
C       WRITE(6,70) SCAN(1),SCAN(2),TCAN
C*************************************************************
C
C       WRITE OUT DIRECT COST SUMMARY
C
C*************************************************************
C
C----------------------
C       WRITE OUT HEADER
C----------------------
        WRITE(6,610) AC,CTYPES,RTYPES,RHG,R
C-----------------------------
C       WRITE OUT TABLE CAPTION
C-----------------------------
        WRITE(6,650)
C--------------------
C       WRITE OUT DATA
C--------------------
        WRITE(6,30) 'CANCERS      ', (SDCAN(J),J=1,SEX),TDCAN
        WRITE(6,30) 'RAD INJURIES', (SDRAD(J),J=1,SEX),TDRAD
        WRITE(6,30) 'GENETIC      ', (SDGEN(J),J=1,SEX),TDGEN
C----------------------
C       WRITE OUT TOTALS
C----------------------
        WRITE(6,40) SD(1),SD(2),TD
C---------------------------------
C       WRITE OUT TOTAL COST SUMMARY
C---------------------------------
        WRITE(6,710) AC,CTYPES,RTYPES,RIG,RHG,R
C-----------------------------
C       WRITE OUT TABLE CAPTION
C-----------------------------
        WRITE(6,750)
C--------------------
C       WRITE OUT DATA
C--------------------
```

```
      WRITE(6,30) 'CANCERS      ', (SCAN(J),J=1,SEX),TCAN
      WRITE(6,30) 'RAD INJURIES', (SRAD(J),J=1,SEX),TRAD



      WRITE(6,30) 'GENETIC      ', (SGEN(J),J=1,SEX),TGEN
C-----------------------
C     WRITE OUT TOTALS
C-----------------------
      WRITE(6,40) (ST(J),J=1,SEX),TT
C**********************************
C     FORMAT STATEMENTS FOR REPORTS
C**********************************
5     FORMAT('1',15(/),1X,40X,'HEALTH EFFECTS COST MODEL',//,1X,
     &  35X,'BATTELLE PACIFIC NORTHWEST LABORATORIES',////,1X,35X,A50)
10    FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,
     &  /,1X,'RATE OF INCOME GROWTH=',F5.1,/,1X,'DISCOUNT RATE=',F4.1)
11    FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,
     &  /,1X,'RATE OF HEALTH COST GROWTH=',F3.1,
     &  /,1X,'DISCOUNT RATE=',F4.1)
12    FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,
     &  /,1X,'RATE OF HEALTH COST GROWTH=',F3.1,
     &  /,1X,'DISCOUNT RATE=',F4.1)
20    FORMAT(1X,///,1X,15X,'INDIRECT COSTS DUE TO FATALITIES',//,
     &  1X,'DEATH CAUSE',9X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,11('-'),9X,4('-'),7X,6('-'),7X,5('-'))
30    FORMAT(1X,A12,2X,3F12.0)
31    FORMAT(1X,2X,A12,3F12.0)
32    FORMAT(1X,/)
33    FORMAT(1X,A10)
34    FORMAT(1X,A13,1X,3F12.0)
40    FORMAT(1X,17X,9('-'),3X,9('-'),3X,9('-'),
     &  /,1X,'  TOTAL LOSS',2X,3F12.0,///)
60    FORMAT(1X,4X,I2,10X,3F12.0)
50    FORMAT(1X,///,1X,15X,'INDIRECT COSTS DUE TO FATALITIES',//,
     &  1X,'AGE CATEGORY',10X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,12('-'),10X,4('-'),7X,6('-'),7X,5('-'))
70    FORMAT(1X,19X,9('-'),3X,9('-'),3X,9('-'),
```

```
     &  /,1X,'  TOTAL LOSS',4X,3F12.0,///)
120     FORMAT(1X,///,1X,15X,'INDIRECT COSTS DUE TO ILLNESS',//,
     &  1X,'HEALTH EFFECT',7X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,13('-'),7X,4('-'),7X,6('-'),7X,5('-'))
150     FORMAT(1X,///,1X,15X,'INDIRECT COSTS DUE TO ILLNESS',//,
     &  1X,'AGE CATEGORY',10X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,12('-'),10X,4('-'),7X,6('-'),7X,5('-'))
320     FORMAT(1X,///,1X,15X,'DIRECT COSTS OF RADIATION INJURIES',//,
     &  1X,'INJURY TYPE',9X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,11('-'),9X,4('-'),7X,6('-'),7X,5('-'))
350     FORMAT(1X,///,1X,15X,'DIRECT COST OF RADIATION INJURIES',//,
     &  1X,'AGE CATEGORY',10X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,12('-'),10X,4('-'),7X,6('-'),7X,5('-'))
430     FORMAT(1X,A20,2X,3F12.0)
440     FORMAT(1X,25X,9('-'),3X,9('-'),3X,9('-'),
     &  /,1X,'  TOTAL LOSS',10X,3F12.0,///)
420     FORMAT(1X,///,1X,15X,'DIRECT COST OF CANCERS',//,
     &  1X,'CANCER TYPE',17X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,11('-'),17X,4('-'),7X,6('-'),7X,5('-'))
450     FORMAT(1X,///,1X,15X,'DIRECT COST OF CANCERS',//,
     &  1X,'AGE CATEGORY',10X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,12('-'),10X,4('-'),7X,6('-'),7X,5('-'))
510     FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,/,
     &  1X,'NUMBER OF DEATH CATEGORIES=',F4.1,
     &  /,1X,'RATE OF INCOME GROWTH=',F3.1,/,1X,'DISCOUNT RATE=',F4.1)



520     FORMAT(1X,///,1X,15X,'INDIRECT COST SUMMARY',//,
     &  1X,'HEALTH EFFECT',7X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,13('-'),7X,4('-'),7X,6('-'),7X,5('-'))
550     FORMAT(1X,///,1X,15X,'INDIRECT COST SUMMARY',//,
     &  1X,'AGE CATEGORY',10X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,12('-'),10X,4('-'),7X,6('-'),7X,5('-'))
610     FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,
     &  /,1X,'NUMBER OF CANCER TYPES=',F4.1,
     &  /,1X,'NUMBER OF RADIATION INJURIES=',F4.1,
     &  /,1X,'RATE OF HEALTH COST GROWTH=',F3.1,
     &  /,1X,'DISCOUNT RATE=',F4.1)
```

```
650     FORMAT(1X,///,1X,15X,'DIRECT COST SUMMARY',//,
     &  1X,'COST TYPE',11X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,9('-'),11X,4('-'),7X,6('-'),7X,5('-'))
710     FORMAT('1',///,1X,'HEALTH EFFECTS COST MODEL',/,1X,25('-'),//,
     &  1X,'NUMBER OF AGE CATEGORIES=',F4.1,
     &  /,1X,'NUMBER OF CANCER TYPES=',F4.1,
     &  /,1X,'NUMBER OF RADIATION INJURY TYPES=',F4.1,
     &  /,1X,'RATE OF INCOME GROWTH=',F3.1,
     &  /,1X,'RATE OF HEALTH COST GROWTH=',F3.1,
     &  /,1X,'DISCOUNT RATE=',F4.1)
750     FORMAT(1X,///,1X,15X,'TOTAL COST SUMMARY',//,
     &  1X,'COST TYPE',11X,'MALE',7X,'FEMALE',7X,'TOTAL'/
     &  ,1X,9('-'),11X,4('-'),7X,6('-'),7X,5('-'))
810     FORMAT(1X,/,1X,'FATALITIES:')
811     FORMAT(1X,3X,A12,10X,F6.1)
820     FORMAT(1X,/,1X,'ILLNESS AND INJURY INCIDENCE')
821     FORMAT(1X,/,1X,'CANCERS:')
825     FORMAT(1X,/,1X,'RADIATION INJURIES:')
830     FORMAT(1X,/,1X,'RADIATION INJURY INCIDENCE:')
840     FORMAT(1X,/,1X,'CANCER INCIDENCE:')
845     FORMAT(1X,3X,A20,3X,F5.1)
5555    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C       SUBROUTINE READER
C
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
        SUBROUTINE READER
C
C       THIS SUBROUTINE READS ALL INPUT DATA FOR EXECUTION OF HECON
C
C       FIRST INCLUDE CONTROL FILE
C
        INCLUDE 'CONTROL.FOR'
C-----------------------------------------------------------------------
C       DETERMINE WHICH FILE SHOULD BE OPENED DEPENDING ON THE NUMBER
C       OF AGE CATEGORIES
C-----------------------------------------------------------------------
        IF (AC.EQ.18) OPEN (UNIT=2,FILE='HECON18.DAT',STATUS='OLD')
```

```
      IF (AC.EQ.1) OPEN (UNIT=2,FILE='RECOM1.DAT',STATUS='OLD')
C-----------------------------------------------------------------
C     READ IN DATA
C-----------------------------------------------------------------
      READ(2,30) CASE
      READ(2,*) RNG
      READ(2,*) RIG
      READ(2,*) R



      READ(2,*) (M1(I,1),I=1,AC)
      READ(2,*) (M1(I,2),I=1,AC)
      READ(2,*) (LFPR(I,1),I=1,AC)
      READ(2,*) (LFPR(I,2),I=1,AC)
      READ(2,*) (POPF(I,1),I=1,AC)
      READ(2,*) (POPF(I,2),I=1,AC)
      READ(2,*) (POPFA(I,1),I=1,AC)
      READ(2,*) (POPFA(I,2),I=1,AC)
      READ(2,*) (MAGE(I,1),I=1,AC)
      READ(2,*) (MAGE(I,2),I=1,AC)
      READ(2,*) (LPU(I),I=1,DTYPES)
      READ(2,*) (LPO(I),I=1,DTYPES)
      READ(2,*) (CF(I),I=1,DTYPES)
      READ(2,*) (POR(I),I=1,DTYPES)
      READ(2,*) (MS(I),I=1,DTYPES)
      READ(2,*) (LWORK(I),I=1,DTYPES)
      READ(2,*) (TREAT(I),I=1,DTYPES)
C-----------------------------
C     READ IN DEATH TYPE NAMES
C-----------------------------
      DO 1000 I=1,DTYPES
      READ(2,10) DTNAMES(I)
1000  CONTINUE
C----------------------
C     READ HEALTH DATA
C----------------------
      READ(2,*) (CPI(I),I=1,CTYPES)
      READ(2,*) (IPF(I),I=1,CTYPES)
      READ(2,*) (CFCONV(I),I=1,CTYPES)
      DO 1100 I=1,CTYPES
```

```
      READ(2,20) CNAMES(I)
1100  CONTINUE
C-------------------------------------
C     READ IN RADIATION INJURY DATA
C-------------------------------------
      READ(2,*) (LWORKR(I),I=1,RTYPES)
      READ(2,*) (RADINF(I),I=1,RTYPES)
      READ(2,*) (RADIF(I),I=1,RTYPES)
      READ(2,*) (CPRAD(I),I=1,RTYPES)
      DO 1300 I=1,RTYPES
      READ(2,10) RNAMES(I)
1300  CONTINUE
C-------------------------------------
C     READ IN GENETIC EFFECTS DATA
C-------------------------------------
      READ(2,*) (INCOST(I),I=1,GAC)
      READ(2,*) (GI(I),I=1,GTYPES)
      READ(2,*) (GSRATE(I),I=1,SEX)
      READ(2,*) (DRATE(I),I=1,GTYPES)
      READ(2,*) (INRATE(I),I=1,GTYPES)
      DO 1400 I=1,GTYPES
      READ(2,20) GNAMES(I)
1400  CONTINUE
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')
      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 INCIDENT DISTRIBUTION DATA
C-----------------------------------------------------------
         IF (AC.EQ.18) OPEN (UNIT=4,FILE='INDIST18.DAT',STATUS='OLD')
         IF (AC.EQ.1)  OPEN (UNIT=4,FILE='INDIST1.DAT',STATUS='OLD')
C---------------------------------------------
C        READ INCIDENT DISTRIBUTION DATA
C---------------------------------------------
         DO 3000 I=1,SEX
         DO 3000 J=1,DTYPES
                  READ(4,*) (IDIST(J,K,I),K=1,AC)
C                 WRITE(6,*) (IDIST(J,K,I),K=1,AC)
3000     CONTINUE
C------------------
C        CLOSE FILE
C------------------
         CLOSE (UNIT=4)
10       FORMAT(A12)
20       FORMAT(A20)
30       FORMAT(A50)
         RETURN
         END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
         SUBROUTINE LATENCY
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C        THIS SUBROUTINE CALCULATES THE LATENCY PERIOD FOR EACH
C        CAUSE OF DEATH AND AGE CATEGORY
C
         INCLUDE 'CONTROL.FOR'
C------------------------------------------------------
C        CALCULATE LATENCY PERIOD FOR EACH AGE OPTION
C
C        FIRST FOR AGE CATEGORY EQUAL 1
C------------------------------------------------------
         IF (AC.NE.1) GOTO 2000
         DO 1000 I=1,DTYPES
                  LP(I,1)=LPO(I)
1000     CONTINUE
         GOTO 3000
```

```
C-----------------------------------------------
C       COMPUTE LATENCY PERIOD FOR AGE CATEGORY=18
C-----------------------------------------------
2000    CONTINUE
        DO 2500 I=1,DTYPES
                LP(I,1)=LPU(I)
                DO 2500 J=2,AC
                        LP(I,J)=LPO(I)
2500    CONTINUE
3000    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC



        SUBROUTINE FATAL
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C       THIS SUBROUTINE CALCULATES FATALITIES BY AGE CATEGORY
C       AND CAUSE OF DEATH.
C
        INCLUDE 'CONTROL.FOR'
        REAL SUM(DTYPES)
C-------------------------------------------------------------------------------
C       FIRST, CALCULATE THE SUM OF (POPULATION FRACTION * INCIDENCE DIST)
C       FOR EACH CAUSE OF DEATH.
C-------------------------------------------------------------------------------
        DO 1000 I=1,DTYPES
        DO 1000 J=1,SEX
        DO 1000 K=1,AC
                SUM(I)=SUM(I)+POPF(K,J)*IDIST(I,K,J)
1000    CONTINUE
C-------------------------------------------------------------------------------
C       CALAULATE PERCENT INCIDENCE OF CANCER FOR EACH SEX AND AGE CATEGORY
C-------------------------------------------------------------------------------
        DO 3500 K=1,SEX
        DO 3500 I=1,AC
                DO 3500 J=1,DTYPES
                        PI(J,I,K)=(POPF(I,K)*IDIST(J,I,K))/SUM(J)
```

```
C-----------------------------------------------------------------------
C                       CHECK TO SEE IF ACUTE IS A DEATH TYPE.  IF IT IS
C                       THEN OVERRIDE POPULATION FRACTION WITH NUN IN UTERO
C                       POPULATION FRACTIONS.
C-----------------------------------------------------------------------
                        IF (J.EQ.ACUTE) PI(J,I,K)=(POPFA(I,K)*IDIST(J,I,K))
     &                                            /SUM(J)
3500    CONTINUE
C-----------------------------------------------------------------------
C       CALCULATE DEATHS FOR EACH CANCER TYPE AND AGE CATEGORY.
C-----------------------------------------------------------------------
        DO 4000 K=1,SEX
        DO 4000 J=1,DTYPES
                DO 4000 I=1,AC
                        FAT(J,I,K)=PI(J,I,K)*CF(J)
4000    CONTINUE
C-----------------------------------------------------------------------
C       ASSIGN PRENATAL RADIATION INJURIES TO IN UTERO, ACUTE FATALITES.
C       THIS IS DONE BECAUSE PRENATAL RADIATION VICTIMS ARE ASSUMED TO
C       NEVER BE ABLE TO WORK.
C-----------------------------------------------------------------------
        DO 5000 I=1,SEX
        IF (ACUTE.NE.0.) FAT(ACUTE,1,I)=(RADINF(PRENAT)+RADIF(PRENAT))*
     &                   POPF(1,I)/(POPF(1,1)+POPF(1,2))
5000    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE WORK
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
C       THIS SUBROUTINE CALCULATES THE LABOR VALUE LOST DUE TO
C       MISSED WORK
C
        INCLUDE 'CONTROL.FOR'
```

```
C
      DO 5000 L=1,SEX
      DO 5000 I=1,AC
      DO 4000 J=1,DTYPES
C-----------------------------------------------------------------------
C     IF DTYPE IS ACUTE THEN SKIP THE CALCULATION.  ACUTE-WORK LOSS IS
C     CALCULATED SEPARATELY FOR EACH RADIATION INJURY.
C-----------------------------------------------------------------------
      IF (J.EQ.ACUTE) GOTO 4000
C-----------------------------------------------------------------------
C     DEATH TYPE IS NOT ACUTE SO COMPUTE LOST WORK BASED ON FATALITIES.
C-----------------------------------------------------------------------
      DO 4000 M=1,TREAT(J)
      DO 4000 K=1,YEARS-M
C-----------------------------------------------------------------------
C     IF DEATH TYPE IS THYROID THEN FATALITIES ARE ACTUALLY INCIDENCE.
C     IF DEATH TYPE IS NOT THYROID THEN INCLUDE INCIDENCE TO FATALITY
C     RATIO IN COST CALCULATION.
C-----------------------------------------------------------------------
      IF (J.EQ.THYROID) GOTO 3000
            LWCCOST(I,K,J,L)=FPY(J,I,K+M,L)*IPF(J)*LWORK(J)/52.0*
     &      PV(LV(I,K,L),R,K)*LPROB(I,K+M,L)
      GOTO 4000
3000  CONTINUE
            LWCCOST(I,K,J,L)=FPY(J,I,K+M,L)*LWORK(J)/52.0*
     &      PV(LV(I,K,L),R,K)*LPROB(I,K+M,L)
4000  CONTINUE
C-----------------------------------------------------------------------
C     CALCULATE WORK LOSS FOR EACH RADIATION INJURY BY AGE CATEGORY
C-----------------------------------------------------------------------
      DO 5000 J=1,RTYPES
         LWRCOST(I,1,J,L)=RADINF(J)*LWORKR(J)/52.0*PI(ACUTE,I,L)*
     &                    PV(LV(I,1,L),R,1)*LPROB(I,1,L)
5000  CONTINUE
      RETURN
      END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
      SUBROUTINE SPROB
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
```

```
C
C       THIS SUBROUTINE CALCULATES THE PROBABILITY THAT A MEMBER OF
C       AN AGE CATEGORY WILL BE ALIVE IN A YEAR - INDEPENDENT OF ANY DEATHS
C       CAUSED BY RADIATION EXPOSURE.
C
        INCLUDE 'CONTROL.FOR'
        INTEGER RLP
C-------------------------------
C       THE FIRST LOOP PROCESSES SEX
C-------------------------------
        DO 5000 ISEX=1,SEX
C-----------------------------------------------
C       THE SECOND LOOP PROCESSES EACH AGE CATEGORY
C-----------------------------------------------
        DO 5000 I=1,AC
C-------------------------------------------------------------
C       CONVERT MEDIAN AGE AND REMAINING LIFE PERIOD TO INTEGERS
C-------------------------------------------------------------
        MA=INT(MAGE(I,ISEX))
        RLP=YEARS-MA
C-------------------------------------------------------


C       IF IN UTERO SET MA=1 TO MATCH IT UP WITH FIRST PROB
C-------------------------------------------------------
        IF (MA.EQ.0) MA=1
C-------------------------------
C       THE THIRD LOOP PROCESSES YEARS
C-------------------------------
        DO 5000 J=1,RLP
C-----------------------------------------------
C       INITILIZE CURRENT PROBABILITY OF LIVING TO 1.0
C-----------------------------------------------
        LPROB(I,J,ISEX)=1.0
C-------------------------------------------------------------
C       CALCULATE PRODUCT OF CONDITIONAL PROBABILITIES FROM YEAR OF
C       EXPOSURE TO CURRENT YEAR (J)
C-------------------------------------------------------------
                DO 5000 L=MA,MA+J-1
                        LPROB(I,J,ISEX)=LPROB(I,J,ISEX)*LIFEP(L,ISEX)
```

```
5000    CONTINUE
C
C
        DO 6000 I=1,SEX
        GLPROB(1,I)=LIFEP(1,I)
        DO 6000 J=2,YEARS
        GLPROB(J,I)=GLPROB(J-1,I)*LIFEP(J,I)
6000    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE LVALUE
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
        INCLUDE 'CONTROL.FOR'
        INTEGER A,B
C
        DO 2000 ISEX=1,SEX
        DO 2000 A=1,AC
        DO 2000 I=1,YEARS
C-------------------------------------------------------------------------------
C       DETERMINE LABOR VALUE CATEGORY OF SOMEONE WHO IS I+MAGE YEARS OLD
C-------------------------------------------------------------------------------
        B=INCCAT(I+MAGE(A,ISEX),A,IINC)
C-------------------------------------------
C       CALCULATE LABOR VALUE IN YEAR I
C-------------------------------------------
        LV(A,I,ISEX)=FV(MI(B,ISEX)*LFPR(B,ISEX),RIG,1)
2000    CONTINUE
        RETURN
        END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
        SUBROUTINE LOSTLV
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
        INCLUDE 'CONTROL.FOR'
        INTEGER Y,A,RLP
C-------------------------------------------
C       A IS COUNTER FOR INCOME CATEGORIES
C-------------------------------------------
```

```
      DO 3000 ISEX=1,SEX
      DO 3000 A=1,AC



C-------------------------------------
C     DETERMINE REMAINING LIFE PERIOD
C-------------------------------------
      RLP=YEARS-INT(MAGE(A,ISEX))
C-----------------------------------------------
C     DETERMINE LABOR VALUE LOSS FOR EACH YEAR
C-----------------------------------------------
      DO 3000 Y=1,RLP
C-----------------------------------------------
C     ADD UP PRESENT VALUE OF LOST LABOR VALUE
C-----------------------------------------------
      DO 3000 I=Y,RLP
              LVLOSS(A,Y,ISEX)=LVLOSS(A,Y,ISEX)+PV(LV(A,I,ISEX),R,I)
     *                          *LPROB(A,I,ISEX)
3000  CONTINUE
      RETURN
      END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
      SUBROUTINE DEATH(OPTION)
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
C
      INCLUDE 'CONTROL.FOR'
      INTEGER OPTION,RLP
C-----------------------------------------------------
C     DETERMINE DESIRED METHOD OF ALLOCATING FATALITIES
C-----------------------------------------------------
      IF (OPTION.NE.1) GOTO 5000
C-----------------------------------------------------------------
C     ALLOCATE FATALITIES EQUALLY TO ALL YEARS IN WHICH DEATHS OCCUR
C-----------------------------------------------------------------
      DO 2000 K=1,SEX
      DO 2000 J=1,DTYPES
      DO 2000 I=1,AC
```

```
C-------------------------------------------------
C               CHECK TO SEE IF DEATH TYPE IS ACUTE
C-------------------------------------------------
                IF (J.NE.ACUTE) GOTO 1500
C*************************************************
C
C               COMPUTE FATALITES FOR ACUTE DEATH TYPE
C
C*************************************************
C
C-----------------------------------------
C               COMPUTE FIRST YEAR OF FATALITIES
C-----------------------------------------
                M=INT(LP(J,I))
C---------------------------------------
C               COMPUTE LAST YEAR OF LIFE
C---------------------------------------
                L=M+INT(MS(J))
C---------------------------------------
C               CALCULATE ACUTE FATALITIES
C---------------------------------------
                IF (L.EQ.M) GOTO 2000
                DO 1400 I2=M+1,L
                FPY(J,I,I2,K)=FAT(J,I,K)/(L-M)
1400            CONTINUE
                GOTO 2000
C



1500            CONTINUE
C-------------------------------------------------------
C               COMPUTE FATALITIES FOR NON-ACUTE DEATH TYPE
C
C
C               CONVERT FIRST YEAR OF DEATH TO AN INTEGER AND CALCULATE
C               REMAINING LIFE PERIOD
C-------------------------------------------------------
                M=INT(LP(J,I)+MS(J))
                RLP=YEARS-INT(AAGE(I,K))
```

```
C-------------------------------------------------------------------
C                   DETERMINE NUMBER OF YEARS TO CALCULATE DEATHS.  THIS
C                   NUMBER IS EITHER THE REMAINING LIFE PERIOD OR PERIOD OF
C                   RISK FOR A CAUSE OF DEATH; WHICHEVER IS LESS.
C-------------------------------------------------------------------
                    IF (RLP.LE.POR(J)) L=RLP
                    IF (RLP.GT.POR(J)) L=INT(POR(J))
                    IF (M.GE.L) GOTO 2000
                    SUM=0
                    DO 1550 KK=M+1,L
                    SUM=SUM+LPROB(I,KK,K)
1550     CONTINUE
C----------------------------------------------------------
C                   IF YEAR IS GREATER THEN, OR EQUAL TO L START
C                   ON NEW DEATH TYPE
C----------------------------------------------------------
1600                IF (M.GE.L) GOTO 2000
C-------------------------------------------------------------------
C                   COMPUTE FATALITIES FOR DEATH TYPE J, AGE CATEGORY I AND
C                   YEAR M+1.  M+1 REPRESENTS THE FIRST YEAR OF DEATH DURING
C                   THE FIRST ITERATION.  IT IS THEN INCREMENTED BY ONE YEAR
C                   UNTIL THE REMAINING LIFE PERIOD HAS EXPIRED.
C-------------------------------------------------------------------
                    IF (SUM.EQ.0) WRITE(6,*)J,I,K,M+1
                    FPY(J,I,M+1,K)=FAT(J,I,K)*LPROB(I,M+1,K)/SUM
C---------------------------
C                   INCREMENT YEAR
C---------------------------
                    M=M+1
                    GOTO 1600
2000     CONTINUE
5000     CONTINUE
         RETURN
         END
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
         FUNCTION INCCAT(AGE,IC,IINC)
         INTEGER IINC,IC
         K=0
C-----------------------------------------
C        CHECK FIRST TO SEE IF IINC EQUALS ZERO
C-----------------------------------------
         IF (IINC.NE.0) GOTO 1000
```

```
         K=1
         GOTO 5000
C---------------------------------------------------------------
C        AGE CATEGORY EQUALS 18.  CHECK FIRST FOR IN UTERO.
C---------------------------------------------------------------
1000     IF (AGE.GT.0) GOTO 2000
         K=1
         GO TO 5000
C---------------------------------------------------------------------------
C        NOW STEP THROUGH EACH YEAR.  K WILL COUNT INCOME CATEGORY OF AGE.



C---------------------------------------------------------------------------
2000     CONTINUE
         DO 3000 I=1,86,IINC
                  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 BOUNDRY FOR A CATEGORY.  IF IT IS NOT
C                 IN UTERO WAIT UNTIL AGE IS ABOVE MEDIAN AGE FOR A CATEGORY.
C----------------------------------------------------------------------------
                  IF (IC.EQ.1.AND.AGE.LT.(I)) GOTO 5000
                  IF (IC.NE.1.AND.AGE.LT.(I+2)) GOTO 5000
3000     CONTINUE
5000     INCCAT=K
         RETURN
         END
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
         FUNCTION FV(P,R,Y)
         REAL P,R
         INTEGER Y
         FV=P*(1+R/100)**(Y-1)
         RETURN
         END
CC
CCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCCC
CC
```

```
FUNCTION PV(P,R,N)
REAL P,R
INTEGER N
PV=P/((1+R/100)**(N-1))
RETURN
END
```

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