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Critical Review of the Reactor-Safety Study Radiological Health Effects Model

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CRITICAL REVIEW OF THE
REACTOR-SAFETY STUDY RADIOLOGICAL-HEALTH-EFFECTS MODEL

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ABSTRACT

This review of the radiological health effects models originally presented in the Reactor Safety Study (RSS) and currently used by the U.S. Nuclear Regulatory Commission (NRC) was undertaken to assist the NRC in determining whether or not to revise the models and to aid in the revision, if undertaken. The models as presented in the RSS and as implemented in the CRAC (Calculations of Reactor Accident Consequences) Code are described and critiqued. The major elements analyzed are those concerning dosimetry, early effects, and late effects. The published comments on the models are summarized, as are the important findings since the publication of the RSS. We conclude that the following areas deserve priority for consideration in any revision: *dosimetry*: the likely particle size distributions and chemical forms of radioactive aerosols, the behavior of radioactive materials on and in soil, the uptake of radioactive material by various organs, the dosimetry of radioactive iodine in the thyroid; *early effects*: the existence of especially sensitive or insensitive population subgroups, the availability of various levels of medical treatment for accidents of different magnitudes, dose-rate effects, situations in which lung damage may contribute substantially to the number of fatalities, broadening the list of early morbidities evaluated, interactions among various types of injuries; *late effects*: re-evaluation of the risks of various cancer types in the light of the BEIR III (NAS, 1980) report, the mortality rate for thyroid cancer victims, the doubling dose for genetic effects, "non-specific aging," and interactions between radiation and other carcinogens, genetic resistance or predisposition to cancer.

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SECTION 1

INTRODUCTION

D.W. Cooper

The goal of Phase I of this project was to produce a critique of the health effects model (HEM) used in WASH-1400, the *Reactor Safety Study* (RSS), and to use this critique as the basis for selecting a Working Group to carry out a revision of the model, aided by the comments of an Advisory Group, to be selected, and other professionals. This critique includes: description of the health effects model and its role within the overall consequence model, review of the dosimetry model, analysis of the modeling of early effects and of the modeling of latent effects, discussion of the USNRC computer code (CRAC) based on this model, and an overview of the model. Special attention was paid to reviewing the literature from the time of publication of the RSS through 1981.

This review is intended to make more clear the nature, strengths, and weaknesses of the current health effects model used for analysis of the safety of light water reactors, to help in planning for the revision of the model, to aid those who perform that revision, and to serve as an aid in evaluating the outcome of the revision process. The process may result in a model appropriate to radiological releases throughout the entire fuel cycle, and that possibility is reflected in some of the comments presented.

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The outline for the final report is as follows, with the names of the individuals primarily responsible for the sections indicated:

1. Introduction (Cooper)
2. Background (Evans)
3. Dosimetry (Kase)
4. Early Health Effects (Maletskos)
5. Late Health Effects (Robertson)
6. Computer Implementation (Evans and Cooper)
7. Overview (Cooper and Evans)

SECTION 2

BACKGROUND

J.S. Evans

2.1 DESCRIPTION

The following description of the *Reactor Safety Study* health effects model (RSS HEM) is based almost entirely upon Appendix VI, WASH-1400. As such, we feel that it summarizes the "model" as it was available to the public. Section 6 of our report includes a summary of the CRAC computer code implementation of the "model." There are, in some cases, differences between the two, which are discussed in Section 6 of this report.

It should be noted at the outset that there are aspects of the model on which Appendix VI, WASH-1400 presents either vague or conflicting evidence. Thus, it is only natural that, in parts, our presentation of the model will be unclear. However, we have made every attempt to synthesize the information presented in Appendix VI, and where possible have attempted to resolve areas of conflict or vagueness through discussion with those responsible for the original model formulation.

2.1.1 Overview

The RSS health effects model is quite complex. The outputs include estimates of the expected number of deaths within one year of an accident and the expected number of illnesses within that year, the expected number of latent deaths and of severe genetic damage in the 544 model geographical cells surrounding the accident site. Input to the model includes: the duration of the release; the quantity of each of 54 radionuclides released; the amount of energy of the release; the meteorological conditions at the site (wind speed, stability, precipitation, and wind direction); the number of people living in each of the model cells; and certain emergency response data. The cells are formed by concentric circles and by radii usually chosen 22.5° apart. The innermost circle has a radius of 0.5 mile and the outermost, 500 miles.

The model can be understood more readily by considering a single geographical cell and by dealing with the fraction of the population in the cell which experiences the effects, rather than the number (which depends on population density). The entire discussion which follows uses this simplification; i.e., it pertains to calculations for one geographical cell and one nuclide, under one set of conditions. The meteorological modeling applied simply results in transfer coefficients of the form

$$\bar{A}(t) = \bar{a}(t)E \quad (2-1)$$

in which $\bar{A}(t)$ is the airborne concentration (for a specific release energy) in Ci/m³ at time t after the accident and $\bar{a}(t)$ is the expected transfer coefficient for a given release of a certain amount of emissions E (in Ci) and set of meteorological conditions. The model is shown qualitatively in Figure 2-1. The characteristics of the accident are combined with the meteorological model to produce

Figure 2-1. Highly simplified schematic diagram of RSS models for estimating health effects. Emissions, E (Ci), are estimated by an accidental release model. Atmospheric transfer coefficients, $\bar{a}(t)$, (Ci/m³ per Ci) are used in conjunction with emission estimates to yield estimates of atmospheric concentrations, $\bar{A}(t)$ (Ci/m³). From these concentrations environmental pathways models, dosimetry models, and dose-response models are used to estimate organ-specific (i) probabilities of morbidity and mortality, q_i .

air concentrations which are actually the product of the transfer coefficients and the emission amounts. The air concentrations are most important: with the deposition modeling and the associated dosimetry, they determine the doses which are then used to obtain health effects from the HEM.

The health effects model has a dosimetry element and a response element. The key to the dose element is the expected dose as a function of time $\bar{D}(t)$ for each organ of interest, such as the bone marrow, the lung, the gastro-intestinal tract, etc.

As is shown in Figure 2-2, the dosimetry model is used to calculate total doses for each organ, with the details of the dose calculation being determined by what is needed by the health response model (HRM) for the effect type (early death, early morbidity, latent cancer, genetic effects). The effects q_i are functions of doses which are in turn functions of air concentration (and thus of ground concentration, too). Thus, for each effect type i and for each organ, there is a relationship of the following form:

$$q_i = f_i(D_i) = h_i(\bar{A}(t)) \quad (2-2)$$

where $h_i(\bar{A}(t))$ is determined by transport and dosimetry. The health response models are outlined next. The response functions are the fractions of the population exposed that then develop the indicated symptoms.

2.1.2 Health Response Models

HRM 1: Early Mortality

The dose-response relationship for damage to the blood-forming system (bone marrow) is shown in Figure 2-3 (RSS Figure VI 9-1). The curve (B) for supportive treatment was used in the calculation of consequences given in RSS Chapter 13. The dose-response relationship for death due to respiratory illness is given in Figure 2-4 (RSS Figure VI 9-3). The dose-response relationship for death due to gastro-intestinal tract irradiation is shown in Figure 2-5 (RSS Figure VI 9-4), as a function of dose to the lower large intestine. Mathematically, the bone marrow dose-response function for supportive treatment is approximately a cumulative normal distribution with a mean of 510 rads and a standard deviation of 70 rads. For the lung, the dose-response function is, approximately $1.08 \times 10^{-11} D^{2.49}$, in which D is in rad also. For the lower large intestine (LLI), the response function is either 1.00 (doses above 5000 rad) or $(D - 2000)/3000$ (doses between 2000 and 5000 rad) or 0.00 (doses less than 2000 rad).

The probability of early death, q_1 , is calculated by the RSS model from the probability of early death due to the bone marrow syndrome, q_{11} , the respiratory syndrome, q_{12} , and the GI tract irradiation, q_{13} .

$$q_1 = q_{11} + (1 - q_{11})q_{12} + (1 - q_{11})(1 - q_{12})q_{13} \quad (2-3)$$

which is the probability one would calculate by assuming that these effects are independent (i.e., no interactions or synergisms) of one another:

Figure 2-2. Highly simplified schematic of environmental pathways, dosimetry, and dose-response models used in RSS estimation of health effects. The probabilities, q , of effects in five classes are estimated. These are early mortality, early morbidity, cancer mortality, incidence of genetic effects, and thyroid effects (both morbidity and mortality). All models are driven by the atmospheric concentrations, $\bar{A}(t)$.

Figure 2-3. Estimated dose-response curves for 50% mortality in 60 days with minimal treatment (curve A), supportive treatment (curve B), and heroic treatment (curve C).

(RSS Figure VI 9-1 with modified caption and axis labels)

Figure 2-4. Dose-response curve ("Criterion") for lung used in RSS HEM.

(RSS Figure VI 9-3 with modified caption and axis labels)

Figure 2-5. Dose-mortality criterion for irradiation of the lower large intestine.

(RSS Figure VI 9-4 with modified caption and axis labels)

$$1-q_1=(1-q_{11})(1-q_{12})(1-q_{13}) \quad (2-4)$$

In addition to these three causes of early death that were included in the consequence calculations presented in Chapter 13, Appendix VI, WASH-1400, deaths due to radiation thyroiditis and prenatal deaths were discussed in Chapter 9. The dose-mortality function for radiation thyroiditis was assumed to have a threshold of 25000 rad and to give a 0.00021 chance of death at any dose above this threshold. Since in a reactor accident anyone receiving a 25000 rad dose to the thyroid would also receive a lethal dose to the bone marrow, the RSS group concluded that no deaths due to radiation thyroiditis would be expected. The embryonic dose-mortality functions are given in Figure 2-6 (RSS Figure VI 9-5), which is reproduced here. The fetal dose-mortality function was assumed to be equal to the bone marrow dose-mortality function for minimal treatment, i.e., curve A in Figure 2-3 (RSS Figure VI 9-1). On the basis of these curves the RSS group concluded that the total number of prenatal deaths (i.e., embryonic plus fetal) would be about 15% of the deaths due to bone marrow, lung and GI tract exposures. These were not totalled in the consequence calculations, since almost 2/3 of them would be embryonic deaths... "and it is likely that the woman concerned would know neither that she was pregnant nor that the embryo had died." The remaining 5% were discarded as lying "within the uncertainty in the overall numbers."

HRM 2: Early Morbidity

In the HEM, the probability of early morbidity is calculated for respiratory impairment, q_{21} , due to lung exposure. The dose-response function used in the model is given by Figure 2-7 (RSS Figure VI 9-6). It is 0.00 probability for doses to the lung less than 3000 rad, $0.05 + (D - 3000) \cdot (0.95)/3000$ for doses from 3000 to 6000 rad, and 1.00 for doses above 6000 rad. For the purpose of making numerical estimates of early morbidity, the study defined morbidity as those illnesses requiring medical attention and possibly hospital admission. This would include respiratory impairment and hypothyroidism. (See RSS Appendix VI, Section 9.2.3.8, page 9-20).

However, in retrospect, there is great uncertainty about what was actually done. For example, immediately after stating that respiratory impairment and hypothyroidism should be included in this definition, the text goes on, saying that respiratory impairments are included in the calculated consequences presented in Chapter 13, Appendix VI, WASH-1400. A similar statement is made in RSS Appendix VI, Chapter 13, referring to the early illness estimates plotted in RSS Figure VI 13-31:

As stated in section 9.2.3.8, early illnesses are defined as those requiring medical treatment. The prime contributor would be cases of respiratory impairment *which constitute the cases shown in Figure VI 13-31.* (Emphasis added.) (See RSS Appendix VI, Section 13.4, pages 13-39.)

Nonetheless, in personal communication, two of the members of the group which developed Appendix VI of the Reactor Safety Study maintained that the consequence estimates given in Chapter 13 included both respiratory impairment and prodromal vomiting. As we mention in Chapter 8, this point of view is

Figure 2-6. Percent termination of pregnancy following brief external exposure during the first trimester of gestation.

(RSS Figure VI 9-5)

Figure 2-7. Dose-morbidity criterion for lung used in RSS HEM.

(RSS Figure VI 9-6 with modified caption and axis labels)

supported by our review of the CRAC Code. In fact, the total early illnesses computed by the CRAC Code include respiratory syndrome, GI syndrome, and prodromal vomiting. The dose-response curve used to predict the incidence of prodromal vomiting is a piecewise linear approximation to the curve shown here as Figure 2-11.

Gastro-intestinal morbidity was assumed to have a probability function which is 0.00 for doses less than 1000 rad, increasing linearly to 1.00 at 2500 rad and above. The RSS noted: "...the dose to the gastrointestinal tract would be approximately equal to that to the bone marrow. Since 1000 rad to the bone marrow would be lethal, gastrointestinal morbidity would never be a major concern."

Prodromal vomiting was considered by the authors of RSS Appendix VI, Chapter 9 to be of little concern. The RSS group noted: "Prodromal vomiting would be the cause of temporary discomfort.... It would unlikely be a source of permanent injury to the affected person." However, as noted above, prodromal vomiting may have been included in the consequence estimates given in Chapter 13.

Although in Appendix H to RSS Appendix VI a dose-response relationship for hypothyroidism occurring within one year of exposure was given (see RSS Appendix VI Table VI H-2, page H-17), the CRAC Code did not incorporate a dose-response function for early hypothyroidism. In addition, no cases of hypothyroidism were reflected in the consequence estimates given in Chapter 13. This is apparently due to the high levels of dose required to induce hypothyroidism within one year of exposure.

Although no dose-response curve for early hypothyroidism was included in the CRAC Code, provision was made for calculating latent cases of hypothyroidism using the dose-response of 110 cases per 10^8 person-rem of exposure. Again, there is some question as to whether these cases are included in the early illness consequence estimates of Chapter 13.

In addition to these four causes of early morbidity which either were intended to be included in the numerical consequence calculations or were included in the numerical consequence calculations, several other potential causes of early morbidity were discussed in Chapter 9. These include radiation thyroiditis, sterility, congenital malformations and growth retardation, and cataracts. Erythema from beta radiation was not discussed.

No cases of radiation thyroiditis were expected, since the doses (> 25000 rad) required to induce the effect would be accompanied by lethal doses to the bone marrow. The possible use of chemicals (such as KI) to reduce the thyroid uptake of iodine was not incorporated.

On the basis of the dose-response curves given in Figures 2-8 and 2-9 (RSS Figures VI 9-7 and VI 9-8), the RSS group estimated that the number of cases of temporary sterility in males might be eight times the number of early morbidities due to respiratory impairment, and that the number of cases of temporary sterility in females might be one-and-a-half times the number of early respiratory morbidities. No cases of permanent sterility would be expected because the doses necessary to induce permanent sterility would be accompanied by

Figure 2-8. Transient sterility (azoospermia) in males within 210 days after exposure.

(RSS Figure VI 9-7 with modified caption)

Figure 2-9. Dose-response curves for sterility in females.

(RSS Figure VI 9-8)

doses lethal to other organs. The RSS group noted: "...the vast majority of these cases would not be aware of their temporary sterility. For this reason, temporary sterility should be regarded as less serious than other early radiation illnesses...."

Congenital malformations and growth retardations are also discussed. It is estimated that the number of cases of fetal hypothyroidism might be about 1% of the total number of cases of hypothyroidism. An approximate calculation, based upon the dose-response function given in Figure 2-10 (RSS Figure VI 9-9) indicated that the total number of cases of microcephaly might be about 50% of the total number of early deaths from doses to the bone marrow, lung and GI tract. The RSS group made a very approximate estimate that the incidence of all other congenital malformations might be equal to the incidence of microcephaly.

The RSS group assumed that cataracts could be produced by doses above 200 to 500 rad. However, they concluded that "...cataract formation would be a consideration only for individuals who received preferential exposure to the head....unlikely in the event of a reactor accident. Therefore, eye cataracts in any survivors are very unlikely."

There was some consideration of the effect of radiation on the immunological system, particularly its influence on resistance to infection and on antibody formation. However, it was concluded that "quantitative estimates of the risks to man from radiation induced lesions in the immune system" could not be made.

HRM 3: Latent Cancer Deaths

Because of the uncertainty and controversy surrounding the quantitative relationship between radiation dose and the probability of inducing cancer, three estimates were produced by the RSS study group.

The upper (also called "expected," anomalously), central, and lower estimates all rely on the same dose estimation. The upper estimate assumes that effect and dose are proportionally related:

$$q_3 = b D_3 \quad (2-5)$$

in which the effect (q_3) is death due to cancer and the dose (D_3) is the dose during the time period of interest.

The central estimate assumes a piecewise linear relationship between dose and effect, in which the slopes of the line segments depend upon both dose and dose rate. The dose rate (DR) used to determine which dose-effectiveness factor (0.2, 0.4, or 1.0) to apply is the average dose rate during the first 30 days after the accident. For all organs except the breast and thyroid this central estimate is given by:

$$q_3 = 0.2bD_3 \text{ if } D_3 < 10 \text{ rem or if } DR < 1 \text{ rem/day} \quad (2-6a)$$

Figure 2-10. Incidence of small head circumference versus dose for individuals exposed between the 0 and 17 weeks of gestation in Hiroshima and Nagasaki.

(a) This data point also includes maternal doses exceeding 150 rads.

(RSS Figure VI 9-9)

Figure 2-11. Prodromal vomiting within 48 hours of exposure without pretreatment.

(RSS Figure VI F-10 with modified caption)

$$q_3 = 1.0bD_3 \text{ if } D_3 > 25 \text{ rem and if } DR > 10 \text{ rem/day} \quad (2-6b)$$

$$q_3 = 0.4bD_3 \text{ otherwise} \quad (2-6c)$$

For the breast the central estimate is equal to the upper estimate.

Finally, the lower estimate is found as follows:

For some organs:

$$q_3 = b(D_3 - 10) \text{ for } D_3 > 10 \text{ rem} \quad (2-7a)$$

$$q_3 = 0 \text{ for } D_3 \leq 10 \text{ rem} \quad (2-7b)$$

For other organs:

$$q_3 = b(D_3 - 25) \text{ for } D_3 > 25 \text{ rem} \quad (2-8a)$$

$$q_3 = 0 \text{ for } D_3 \leq 25 \text{ rem} \quad (2-8b)$$

For each organ, the value of b is found as follows:

$$b = \sum_{j=1}^{11} f_j y_j r_j \quad (2-9)$$

in which j indexes the age cohorts, taking the value $j=1$ for the fraction of the population in utero; $j=2$ for the group 0 to 1 years of age; $j=3$ for 1 to 10 years of age and the rest of the j values increase age by ten years, with $j=11$ being for ages 80 and above. The symbol f_j is for the fraction of the population in the particular age cohort (based on 1970 U.S. census). If a cancer-induction model with a plateau period p and a latency period l is used (see Figure 2-12, RSS Figure VI 9-10), then y_j is the smaller of p and the difference between the remaining life expectancy, e_j , and the latency period. Finally, the risk coefficient, r_j , is the number of cancer deaths per year during the expression period per 10^6 person-rem of radiation to the organ of interest for people in the j th cohort. The 1972 BEIR report was used (with minor modifications) to estimate the values of r , l , and p . Table 2-1 (RSS Table VI 9-3) has the population age distribution and the life expectancies used. It also presents an example calculation using the latent periods, the years at risk (plateau lengths) and the risk factor for each year at risk to develop the expected cases per 10^6 person-rem exposure for leukemia. Table 2-2 (RSS Table VI 9-4) gives the expected deaths per 10^6 man-rem resulting from similar calculations for the following cancers: leukemia, lung, stomach, breast, bone, alimentary canal, pancreas, and all others (except thyroid). Table 2-3 (RSS Tables 9-1 and 9-2) gives the latent and plateau periods and the risk coefficients given in the 1972 BEIR report as well as those used in the RSS HEM calculations of dose-mortality constants, b . The proportionality constants (b) are used in connection with the organ-specific dose which is received by external exposure and chronic internal exposure.

Figure 2-12. Basic model for latent cancer fatalities.

(RSS Figure VI 9-10)

Table 2-1. Calculation of Expected Leukemia Dose Mortality Constant (b) for External Exposure and Chronic Internal Exposure.

(RSS Table VI 9-3 with modified caption)

Age Cohort (years)	Fraction of Population	Life Expectancy (years)	Latent Period (years)	Years at Risk	Risk Factor ($10^6/\text{rem}/\text{year}$)	Expected Cases
<u>In utero</u>	0.011	71.0	0	10	15	1.65
0-0.99	0.014	71.3	2	25	2	0.70
1-10	0.146	69.4	2	25	2	7.30
11-20	0.196	60.6	2	25	1	4.90
21-30	0.164	51.3	2	25	1	4.10
31-40	0.118	42.0	2	25	1	2.95
41-50	0.109	32.6	2	25	1	2.73
51-60	0.104	24.5	2	22.5	1	2.34
61-70	0.080	17.1	2	15.1	1	1.21
71-80	0.044	11.1	2	9.1	1	0.40
80+	0.020	6.5	2	4.5	1	<u>0.09</u>
						b = 28.36

Table 2-2. Latent Cancer Dose-Mortality Constants (b) (Excluding Thyroid) for External Exposure and Chronic Internal Exposure.

(RSS Table VI 9-4 with modified caption)

Type of Cancer	Expected Deaths per 10 ⁶ Man-rem
	b
Leukemia	28.4
Lung	22.2
Stomach	10.2
Alimentary canal	3.4
Pancreas	3.4
Breast	25.6
Bone	6.9
All other	<u>21.6</u>
Total (excluding thyroid)	121.6

Table 2-3. Latency (l) and Plateau (p) Periods and Risk Coefficients (r_j)
Given in BEIR Report and Used in RSS HEM Calculations.

RSS Table VI 9-1. Parameters Given in BEIR Report (caption modified).

Type of Cancer	Age at Time of Irradiation	Latent Period (years)	Plateau Period (years)	Risk Coefficient (deaths/ 10^6 / yr/rem)
Leukemia	<u>In utero</u>	0	10	25
	0-9.9	2	25	2
	10+	2	25	1
Lung	10+	15	30 (a)	1.3
Gastrointestinal tract, including stomach	10+	15	30 (a)	1.0
Breast	10+	15	30 (a)	1.5 (b)
Bone	10+	15	30 (a)	0.2
All other (c)	<u>In utero</u>	0	10	25 (d)
	0-9.9	15	30 (a)	1 (d)
	10+	15	30 (a)	1 (e)

(a) Remaining life expectancy stated as an alternative plateau period.

(b) Includes males and an assumed 50% cure rate.

(c) Includes thyroid and skin.

(d) "All other" denotes all cancers except leukemia.

(e) "All other" denotes all cancers except those specified in table.

RSS Table VI 9-2. Parameters Used in RSS HEM Calculations (caption modified).

Type of Cancer	Age at Time of Irradiation	Latent Period (years)	Plateau Period (years)	Risk Coefficient (deaths/ 10^6 / yr/rem)
Leukemia	<u>In utero</u>	0	10	15
	0-9.9	2	25	2
	10+	2	25	1
Lung	10+	15	30	1.3
Gastrointestinal tract:				
Stomach	10+	15	30	0.6
Rest of ali- mentary canal	10+	15	30	0.2
Pancreas	10+	15	30	0.2
Breast	10+	15	30	1.5 (a)
Bone	0-19.9	10	30	0.4
	20+	10	30	0.2
All other	<u>In utero</u>	0	10	15 (b)
	0-9.9	15	30	0.6 (c)
	10+	15	30	1 (d)

(a) Includes males and an assumed 50% cure rate.

(b) "All other" includes all cancers except leukemia.

(c) "All other" includes all cancers except leukemia and bone.

(d) "All other" includes all cancers except those specified in table.

The model for early internal exposures (i.e., inhalation) is somewhat different. Only the population *living at the time of the accident* receives any appreciable internal exposure due to inhalation of radionuclides from the passing cloud. Since this population will decrease in size with time, due to death from natural causes, the proportionality constants are adjusted such that b_i has the same form as b but has $f_i = 0$ for any age group whose upper age limit is less than or equal to the number of years, t , which have elapsed since the accident. Table 2-4 (RSS Table 9-5) gives the values of b_i used in this health response model in terms of the proportionality constant between dose (in 10^6 person-rem) and fatalities due to cancer caused by internal exposure due to inhalation of radionuclides from the passing cloud. These weighted values may then be applied to a population without regard to the age or sex of the individuals.

The total probability of death due to latent cancer for each organ is given by the sum of b times the external dose and chronic internal dose and b_i times the early internal dose to the organ. These probabilities are those attributable to dose during the indicated time interval, not the probabilities of death during the time intervals.

HRM 3A: Thyroid Morbidity and Mortality

A separate dose calculation and dose-response model is used to calculate the chance of developing either a benign or a cancerous thyroid nodule. The dose used in these calculations, D_{3A} , is the expected dose (rem) to the adult thyroid and the dose-response functions are as follows:

a. The probability of developing a benign thyroid nodule is taken as:

$$0.0002 D_{3A} \text{ for } D_{3A} < 1500$$

$$0.0001 D_{3A} \text{ for } 1500 \leq D_{3A} < 5000$$

$$0 \text{ for } D_{3A} \geq 5000$$

b. The probability of developing a cancerous thyroid nodule is taken as:

$$0.000134 D_{3A} \text{ for } D_{3A} < 1500$$

$$0.000067 D_{3A} \text{ for } 1500 \leq D_{3A} < 5000$$

$$0 \text{ for } D_{3A} \geq 5000$$

These dose-effect relationships include constants of proportionality which were derived in a manner similar to that used to obtain the dose-mortality coefficients (b) applicable to other latent cancers. In making the calculations for the thyroid, a latency period, l , of ten years and a plateau period, p , of thirty years were assumed. The risk coefficient, r_j , for thyroid cancer was taken to be 4.3 cancers per year of expression (plateau) period per 10^6 person-rem of exposure, regardless of age at exposure. The risk coefficient, r_j , for benign thyroid nodules was taken to be 8.1 nodules per year of expression (plateau) period per 10^6 person-rem of exposure of people less than 20 years old, and 4.0 nodules per year of expression (plateau) period per 10^6 person-rem of exposure of people

Table 2-4. Latent Cancer Dose-Mortality Constants (b_{it}) (Excluding Thyroid) for Internal Doses Received in Various Time Intervals Due to Early Internal Exposure (i.e., Inhalation from the Plume).

(RSS Table VI 9-5 with modified caption)

Type of Cancer	Time Period (years) After Accident								
	0-1	1-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80
Leukemia	28.4	27.2	18.7	13.8	9.7	6.8	4.0	1.7	0.5
Lung	22.2	22.2	22.2	14.5	8.1	4.0	1.5	0.2	0
Gastrointestinal tract ^(a)	13.6	13.6	13.6	8.9	5.0	2.5	0.9	0.1	0
Pancreas	3.4	3.4	3.4	2.2	1.3	0.6	0.2	0	0
Breast	25.6	25.6	25.6	16.8	9.4	4.6	1.7	0.3	0
Bone	6.9	6.7	5.0	2.6	1.6	0.9	0.4	0.1	0
All other	21.6	19.8	17.1	11.2	6.3	3.1	1.2	0.2	0
Total	121.6	118.5	105.5	70.1	41.3	22.4	10.0	2.6	0.5

^(a) Includes stomach and rest of alimentary canal.

Note: The dose-mortality constants for the first time interval, b_{i1} , are the dose mortality constants used for external exposure and chronic internal exposure.

older than 20. As outlined in RSS Table VI 9-8, since the CRAC Code calculates doses only for the *adult* thyroid, it was necessary to modify the thyroid dose-effect coefficients to reflect the increased doses received by children. This was done by multiplying the risk coefficient applicable to children between the ages 1 and 10 by the "age-dose factor" 1.9, and multiplying the risk coefficient applicable to children between the ages 11 and 20 by the "age-dose factor" 1.6 prior to calculating their contributions to the population-weighted thyroid dose-effect coefficients. These "age-dose factors" were taken from RSS Table VI 8-5. Thus, application of the dose-effect factors to adult thyroid doses would be expected to reflect properly the contribution to risk of the higher doses to children's thyroids.

Further, it was estimated that one-tenth of the people who develop cancerous thyroid nodules die from them. Also noteworthy is that the thyroid dose is calculated a bit differently from the other organ doses used to predict "latent mortality," a point discussed more fully below.

HRM 4: Genetic Damage

The probabilities of four types of genetic damage are discussed in Appendix VI of the RSS. The types are: autosomal dominant disorders, multifactorial disorders, disorders due to chromosomal aberrations, and spontaneous abortions. For each, the risk could be calculated in such a way that the constants of proportionality apply to the average member of the population without regard to age or sex. The form of the calculation for each type of disorder is again the sum of the product of proportionality constants between internal dose and response and between external dose and response. These proportionality factors have two time arguments: time of expression, and age during exposure. The values of these proportionality constants are presented in Tables 2-5 and 2-6 (RSS Table VI 9-11 and Table VI 9-12).

The risk coefficients presented in Tables 2-5 and 2-6 are based largely upon those given in BEIR (1972). The assumptions upon which they are based include:

1. A doubling dose of 100 rem.
2. A live birth rate of $14000/10^6$ person-yr.
3. An interval between generations of 30 yr.

In addition for each type of genetic effect a baseline incidence and rate of elimination were assumed. The values chosen were:

Type of Effect	Baseline Incidence (#/yr- 10^6 person)	Rate of Elimination (per generation)
autosomal dominant	140	20%
multifactorial	580	10%
chromosomal	75	50%
spontaneous abortion	330	50%

Other single gene effects, such as autosomal recessive and x-chromosome linked effects were thought to be negligible. The variation in the risk coefficients for multifactorial disorders given in Tables 2-5 and 2-6 is due to uncertainty as to the fraction of the baseline incidence (5% to 50%) believed to be related to the

Table 2-5. Disorders and Spontaneous Abortions Attributable to Radiation from External Sources Derived from Releases at the Time of the Hypothetical Accident.

(RSS Table VI 9-11)

Postaccident Period over Which Dose is Accumulated (Years)	Genetic Effects (per Rem per Million Population) Expressed in the Two 30-Year Periods After the Accident and Expressed over All Time			
	0-30 Years	31-60 Years	Remaining to Be Expressed	Total (over All Time)
<u>Autosomal Dominant Disorders</u>				
0-1	8.15	6.45	24.59	39.19
1-30	4.2	7.39	27.60	39.19
31-60	--	8.15	31.04	39.19
61+	--	--	39.19	39.19
<u>Multifactorial Disorders</u>				
0-1	0.83-8.25	0.74-7.39	6.27-62.76	7.84-78.4
1-30	0.42-4.2	0.79-7.88	6.63-66.32	7.84-78.4
31-60	--	0.83-8.25	7.01-70.15	7.84-78.4
61+	--	--	7.84-78.4	7.84-78.4
<u>Disorders Due to Chromosomal Aberrations</u>				
0-1	4.8	0.8	0.6	6.2
1-30	2.7	2.7	0.8	6.2
31-60	--	4.8	1.4	6.2
61+	--	--	6.2	6.2
<u>Spontaneous Abortions</u>				
0-1	31.8	5.1	3.6	40.6
1-30	18.0	17.6	5.0	40.6
31-60	--	31.8	8.8	40.6
61+	--	--	40.6	40.6

Table 2-6. Disorders and Spontaneous Abortions Due to Radiation from Internal Sources Incorporated at the Time of the Hypothetical Accident.

(RSS Table VI 9-12)

Postaccident Period over Which Dose is Accumulated (Years)	Genetic Effects (per Rem per Million Population) Expressed in the Two 30-Year Periods After the Accident and Expressed over All Time			
	0-30 Years	31-60 Years	Remaining to Be Expressed	Total (over All Time)
<u>Autosomal Dominant Disorders</u>				
0-1	8.15	6.45	24.59	39.19
1-10	6.18	5.27	20.76	32.21
11-20	3.12	2.64	12.47	18.23
21-30	0.68	0.88	4.45	6.01
31-40	--	0.20	0.81	1.01
41-50	--	(a)	(a)	(a)
<u>Multifactorial Disorders</u>				
0-1	0.83-8.25	0.74-7.39	6.27-62.76	7.84-78.4
1-10	0.62-6.22	0.60-5.97	5.22-52.24	6.44-64.43
11-20	0.31-3.12	0.29-2.92	3.05-30.42	3.65-36.46
21-30	0.07-0.68	0.09-0.93	1.04-10.4	1.20-12.01
31-40	--	0.02-0.22	0.18-1.80	0.20-2.02
41-50	--	(a)	(a)	(a)
<u>Disorders Due to Chromosomal Aberrations</u>				
0-1	4.8	0.8	0.6	6.2
1-10	3.8	0.7	0.6	5.1
11-20	2.0	0.5	0.4	2.9
21-30	0.4	0.4	0.2	1.0
31-40	--	<0.1	<0.1	<0.2
41-50	--	(a)	(a)	(a)
<u>Spontaneous Abortions</u>				
0-1	31.8	5.2	3.6	40.6
1-10	25.5	4.7	3.2	33.4
11-20	13.4	3.5	2.0	19.0
21-30	2.9	2.5	0.9	6.3
31-40	--	0.9	0.2	1.1
41-50	--	(a)	(a)	(a)

(a) Negligibly small in comparison with preceding row.

mutation rate. Although these four types of genetic damage are discussed, it appears that the numerical estimates presented in Chapter 13 included only autosomal dominant disorders, multifactorial disorders and disorders due to chromosomal aberrations. Spontaneous abortions are not included in the estimates, since for the purposes of making numerical estimates the study adopted a definition of genetic effects as "live births with a genetically caused disorder that could be transmitted to their children." Autosomal recessive disorders were discarded as being negligible in comparison with the increased incidence of other disorders.

Summary of HRM's

In summary, while many potential health effects of reactor accidents were discussed by the RSS study group in Chapter 9, Appendix VI, WASH-1400, only those listed below were included in the numerical consequence estimates presented in Chapter 13:

HRM 1: probabilities of early deaths from bone marrow, from lung, and/or from GI tract irradiation;

HRM 2: probabilities of early illness due to respiratory impairment of lung function and hypothyroidism (some participants in the RSS maintain that the estimates excluded hypothyroidism but included prodromal vomiting, and we cannot rule this out);

HRM 3: probabilities of cancer deaths from leukemia, from cancers of lung, GI tract (stomach and alimentary canal), and/or any other cancer (including pancreas but nominally excluding thyroid);

HRM 3A: probabilities of developing a benign thyroid nodule, a malignant thyroid nodule, dying from a malignant thyroid nodule;

HRM 4: probabilities of genetic damage including multifactorial disorders, disorders due to chromosomal aberration, and autosomal dominant disorders, but excluding spontaneous abortion and autosomal recessive disorders.

2.1.3 Exposure Pathway and Dosimetry Models

Next is discussed the nature of the functions which convert air concentrations of radioactive material into the dose measures used in the health response models.

A brief review of the physical mechanisms and models involved in the RSS study is needed. In the broadest sense, the model allows for four basic sources of exposure:

1. external exposure from the passing cloud ("cloudshine");
2. internal exposure from material inhaled as the cloud passes or inhaled from material resuspended later;
3. external exposure ("groundshine") from the ground contamination due to "fall-out" or "rain-out";

4. internal exposure from ingestion of milk and other foods which have been contaminated either directly or indirectly.

Table 2-7 presents the 54 radionuclides used in the dosimetry calculations, the core inventory of each of the radionuclides, the half-life of each, and whether the radionuclide enters into the model for chronic exposure (and in which dosimetric submodel therein). All 54 radionuclides are used in the calculation of early exposures.

Passing Cloud Model - External Exposure

The model of the exposures due to the passing cloud has three elements. First, the time-integrated exposure (Ci-s/m^2) is obtained by multiplying the expected cloud concentration by the time of exposure to the cloud. This exposure (E_{1a}) is multiplied by the semi-infinite cloud dose conversion factor for the organ of interest, D_{∞}^c , to estimate the dose (rem) expected from immersion in a semi-infinite cloud:

$$E_{1b} = E_{1a} D_{\infty}^c \quad (2-10)$$

The dose conversion factors were calculated for each organ by the EXREM III code (Oak Ridge National Laboratory). The factors for the total bone marrow, the lung, the testes, and the whole body are given in Table 2-8 (RSS Table VI C-1).

Next, the final dose estimate E_{1c} is created by applying a finite-cloud correction factor, D^c / D_{∞}^c . These correction factors, based upon those of Slade for a spherical cloud, are dependent on position and width of the plume and are given in Table 2-9 (RSS Table VI 8-1). Note that for persons more than 25 mi from the reactor a 0.75 shielding factor is applied to these doses. The 0.75 shielding factor is used to reflect the composite time-weighted shielding offered by various structures. The weights are based upon "normal" activity patterns, e.g., those in RSS Table VI 11-11. Therefore a shielding factor of 1.0 is applied to the doses within 25 miles of the site, a factor appropriate to evacuation by automobile.

Inhalation Models

There are two inhalation models involved in the RSS HEM. The first model is used to estimate the dose received in each time period after the accident due to inhalation of radionuclides from the passing cloud. The second model is used to estimate comparable doses for each time period after the accident due to inhalation of resuspended radionuclides.

In the first, the calculations begin with integration of the time-averaged initial cloud concentration:

$$I_{1a} = \int_{t_0}^{t_b} \bar{A}(t) dt \quad (2-11)$$

Table 2-7. Nuclides Involved in Chronic Exposure Calculations.
(All 54 radionuclides enter the early exposure calculations.)

No.	Nuclide	Inventory (10^8 Ci)	Half-Life (days)	Chronic Exposure Pathway		
				Groundshine	Inhalation (Resuspension)	Ingestion
1	Cobalt-58	0.0078	71.0	x		
2	Cobalt-60	0.0029	1920.	x		
3	Krypton-85	0.0056	3950.			
4	Krypton-85m	0.24	0.183			
5	Krypton-87	0.47	0.0528			
6	Krypton-88	0.68	0.117			
7	Rubidium-86	0.00026	18.7			
8	Strontium-89	0.94	52.1			x
9	Strontium-90	0.0037	11030.		x	x
10	Strontium-91	1.1	0.403			
11	Yttrium-90	0.0039	2.67			
12	Yttrium-91	1.2	59.0			
13	Zirconium-95	1.5	65.2	x		
14	Zirconium-97	1.5	0.71			
15	Niobium-95	1.5	35.0	x		
16	Molybdenum-99	1.6	2.8			
17	Technetium-99m	1.4	0.25			
18	Ruthenium-103	1.1	39.5	x		
19	Ruthenium-105	0.72	0.185			
20	Ruthenium-106	0.25	366.	x	x	
21	Rhodium-105	0.49	1.50			
22	Tellurium-127	0.0	0.391			
23	Tellurium-127m	0.011	109.			
24	Tellurium-129	0.31	0.048			
25	Tellurium-129m	0.053	0.340			
26	Tellurium-131m	0.13	1.25			
27	Tellurium-132	1.2	3.25			
28	Antimony-127	0.061	3.93			
29	Antimony-129	0.33	0.179			
30	Iodine-131	0.85	8.05	x		x
31	Iodine-132	1.2	0.0958			
32	Iodine-133	1.7	0.875			x
33	Iodine-134	1.9	0.0366			
34	Iodine-135	1.5	0.280			
35	Xenon-133	1.7	5.28			
36	Xenon-135	0.34	0.384			
37	Cesium-134	0.075	750.	x		x
38	Cesium-136	0.030	13.0	x		x
39	Cesium-137	0.047	11000.	x		x
40	Barium-140	1.6	12.8			
41	Lanthanum-140	1.6	1.67			
42	Cerium-141	1.5	32.3			
43	Cerium-143	1.3	1.38			
44	Cerium-144	0.85	284.			
45	Praseodymium-143	1.3	13.7			
46	Neodymium-147	0.60	11.1			
47	Neptunium-239	16.4	2.35			
48	Plutonium-238	0.00057	32500.		x	
49	Plutonium-239	0.00021	8.9×10^6		x	
50	Plutonium-240	0.00021	2.4×10^6		x	
51	Plutonium-241	0.034	5350.		x	
52	Americium-241	0.000017	1.5×10^5		x	
53	Curium-242	0.050	163.		x	
54	Curium-244	0.000023	5630.		x	

Table 2-8. Photon Dose-Conversion Factors D_{∞}^c (rem per Ci - s/m³)
for Immersion in Contaminated Air.

(RSS Table VI C-1 with modified caption)

Radionuclide	Whole Body ^(a)	Total Marrow	Lung	Testes
CO-57	2.18E-01	2.40E-01	2.01E-01	2.11E-01
CO-60	6.20E-01	6.31E-01	5.47E-01	4.28E-01
KR-85	4.75E-04	5.78E-04	4.47E-04	5.22E-04
KR-85M	3.64E-02	5.50E-02	3.22E-02	4.78E-02
KR-87	1.81E-01	1.92E-01	1.72E-01	1.32E-01
KR-88	4.07E-01	4.83E-01	4.47E-01	2.83E-01
SR-86	2.27E-02	2.17E-02	1.84E-02	1.07E-02
SR-89	0.0	0.0	0.0	0.0
SR-90	0.0	0.0	0.0	0.0
SR-91	1.60E-01	1.83E-01	1.68E-01	1.64E-01
Y-90	0.0	0.0	0.0	0.0
Y-91	6.25E-04	6.36E-04	5.94E-04	4.67E-04
ZR-95	1.62E-01	1.87E-01	1.52E-01	1.81E-01
ZR-97	4.22E-02	4.72E-02	4.00E-02	3.26E-02
NR-95	1.86E-01	1.83E-01	1.56E-01	1.52E-01
MO-99	3.64E-02	4.44E-02	3.42E-02	3.02E-02
TC-99M	3.26E-02	5.42E-02	2.54E-02	2.04E-02
RU-103	1.11E-01	1.36E-01	1.05E-01	1.24E-01
RU-105	1.79E-01	2.21E-01	1.67E-01	1.97E-01
RU-106	4.31E-02	5.22E-02	4.06E-02	4.59E-02
RH-105	1.82E-02	2.74E-02	1.81E-02	2.36E-02
TE-127	9.36E-04	1.16E-03	8.78E-04	1.05E-03
TE-127M	1.10E-03	1.79E-03	5.61E-04	1.50E-03
TE-129	1.47E-02	1.31E-02	1.35E-02	1.61E-02
TE-129M	7.83E-03	9.02E-03	6.97E-03	7.36E-03
TE-131M	3.14E-01	3.56E-01	2.94E-01	2.25E-01
TE-132	4.75E-02	7.31E-02	4.19E-02	6.14E-02
SR-127	1.51E-01	1.84E-01	1.43E-01	1.63E-01
SR-129	2.68E-01	2.07E-01	2.53E-01	2.47E-01
I-131	8.72E-02	1.08E-01	8.22E-02	9.75E-02
I-132	5.11E-01	5.39E-01	4.83E-01	5.25E-01
I-133	1.54E-01	1.83E-01	1.46E-01	1.62E-01
I-134	5.33E-01	5.89E-01	5.00E-01	4.83E-01
I-135	4.19E-01	4.42E-01	4.00E-01	3.39E-01
XE-133	9.06E-03	1.59E-02	9.07E-03	9.38E-03
XE-135	5.07E-02	2.47E-02	5.06E-02	7.35E-02
CS-134	3.50E-01	4.03E-01	3.28E-01	3.50E-01
CS-136	4.78E-01	5.42E-01	4.44E-01	4.44E-01
CS-137	1.22E-01	1.40E-01	1.15E-01	1.35E-01
BA-140	4.44E-02	5.61E-02	4.14E-02	5.06E-02
LA-140	5.67E-01	6.06E-01	5.39E-01	4.58E-01
CE-141	1.83E-02	3.22E-02	1.50E-02	1.74E-02
CE-143	6.81E-02	9.36E-02	6.08E-02	7.89E-02
CE-144	4.31E-03	7.61E-03	3.44E-03	4.17E-03
PR-143	0.0	0.0	0.0	0.0
ND-147	3.14E-02	4.39E-02	2.78E-02	3.42E-02
HP-239	3.08E-02	4.97E-02	2.65E-02	3.53E-02
PU-238	5.25E-05	4.25E-05	9.58E-06	4.19E-05
PU-239	2.30E-05	2.17E-05	5.42E-06	1.94E-05
PU-240	4.64E-05	3.89E-05	9.17E-06	3.70E-05
PU-241	4.17E-10	0.53E-10	2.94E-10	3.55E-10
AM-241	4.56E-03	9.33E-03	3.22E-03	3.05E-03
CM-242	5.00E-05	3.89E-05	8.31E-06	3.99E-05
CM-244	1.42E-03	2.81E-03	1.07E-03	1.27E-03

(a) For organs not listed the dose-conversion factors are similar to those of the whole body.

Table 2-9. Finite-Cloud Dose Correction Factors (D^c/D_∞^c)^(a,b)
 (RSS Table VI 8-1)

Vertical Diffusion σ_z meters	Height of Cloud Centerline, z/σ_z					
	0	1	2	3	4	5
3	0.020	0.018	0.011	0.066	0.005	0.004
10	0.074	0.060	0.036	0.020	0.015	0.011
20	0.150	0.120	0.065	0.035	0.024	0.016
30	0.220	0.170	0.088	0.046	0.029	0.017
50	0.350	0.250	0.130	0.054	0.028	0.013
100	0.560	0.380	0.150	0.045	0.016	0.004
200	0.760	0.511	0.150	0.024	0.004	0.001
400	0.899	0.600	0.140	0.014	0.001	0.001
1000	0.951	0.600	0.130	0.011	0.001	0.001

(a) Data from Slade (1968, Fig. 7.14).

(b) $D^c/D_\infty^c = \frac{\text{dose from spherical cloud}}{\text{dose from semi-infinite cloud}}$

in which the integral is for the period from the time of arrival of the cloud, t_a , to the time it departs, t_b . The $\bar{A}(t)$ values during the initial passage of the cloud (plume) are given by the meteorological model. To obtain the expected quantity of radioactive material inhaled, I_{1a} is multiplied by an average breathing rate, B : $2.66 \times 10^{-4} \text{ m}^3/\text{s}$:

$$I_{1b} = BI_{1a} \quad (2-12)$$

The fraction of the inhaled activity, I_{1b} , which is deposited in each of the three pulmonary compartments is based upon the deposition model shown in Figure 2-13 (RSS Figure VI 8-6). To account for variation of breathing rate, organ masses, and metabolism with age, doses to children from certain nuclides are corrected using the ratios of children's dose to adult dose, D_{ch}/D_{ad} , given in Table 2-10 (RSS Table VI D-6). Predicted fractional depositions of 0.08 in the T-B (tracheobronchial) region, 0.25 in the P (pulmonary) region, and 0.32 in the N-P (nasopharyngeal) region result from the assumption of $1.0 \mu\text{m}$ aerodynamic diameter particles. [Note: These numbers are only approximate, as they were read from the figure in the RSS.] The final element converts deposited radioactive material values into dose (to each organ) accumulated in each of the time periods of interest for early dosimetry and in each of the ten time intervals relevant for chronic dosimetry. This is done using the dose conversion factors $DCF_2(t)$ calculated using a slightly modified version of the ICRP Task Group lung model to distribute inhaled activity to the organs and a computer program developed at Oak Ridge National Laboratory. (See Figure 2-14, RSS Figure VI 8-7 and Table 2-11, RSS Table VI D-2.)

The model for inhalation of resuspended radionuclides is a bit more complex. The $A(t)$ values result from application of models for wet and dry deposition, radioactive decay and resuspension. From the ground surface concentration, $G_g(t)$, estimated from models discussed in the groundshine section, the airborne concentration due to resuspension is estimated to be

$$A(t) = K(t)G_g(t) \quad (2-13)$$

where the resuspension factor $K \text{ (m}^{-1}\text{)}$ varies over time (t in years) according to the equation:

$$K(t) = 10^{-5} \exp(-0.667t) + 10^{-9} \quad (2-14)$$

The amount of a radionuclide inhaled due to resuspension of ground deposits is calculated for each 10-year interval after the accident by integration, e.g.,

$$I_{1b}(10-20\text{yr}) = B \int_{10}^{20} A(t) dt \quad (2-15)$$

In this calculation the value of the breathing rate, B , is $7300 \text{ m}^3/\text{yr}$. The incremental doses due to inhalation of resuspended radionuclides for each ten-year interval after the accident are calculated as a sum of products of appropriate time-interval-specific dose conversion factors, $DCF_2(t)$, and amounts inhaled,

Figure 2-13. Deposition model. The radioactive or mass fraction of an aerosol that is deposited in the nasopharyngeal, tracheobronchial, and pulmonary regions is given in relation to the activity of mass median aerodynamic diameter (AMAD or MMAD) of the aerosol distribution. The model is intended for use with aerosol distributions that have an AMAD or MMAD between 0.2 and 10 microns with geometric standard deviations of less than 4.5. Provisional deposition estimates further extending the size range are given by the broken lines. For the unusual distribution having an AMAD or MMAD greater than 20 microns, complete nasopharyngeal deposition can be assumed. The model does not apply to aerosols with AMADs or MMADs below 0.1 micron.

(RSS Figure VI 8-6)

Table 2-10. Dose Correction Ratios D_{ch}/D_{ad} for Various Ages and Radionuclides.^(a)
 (RSS Table VI D-6)

Radionuclide	Organ of Reference	Dose correction ratio D_{ch}/D_{ad}		
		Age 1 year	Age 5 years	Age 10 years
Iodine-131	Thyroid	5.4	4.6	2.4
Cesium-137	Total body	1.1	1.2	1.1
Strontium-89 or Strontium-90	Bone or red marrow	5	1	1

^(a) The dose correction ratios given here are per unit activity intake.

Figure 2-14. Retention model.

(RSS Figure VI 8-7 with modified caption)

Table 2-11. Dose-Conversion Factors for Inhaled Radionuclides (rem/Ci Inhaled)
Body Organ: Total Marrow.

(RSS Table VI D-2)

Radionuclide	0-2 Days	0-7 Days	0-30 Days	0-1 Year	0-10 Years	0-30 Years	0-50 Years
CO-58	2.3E+02	4.0E+02	1.1E+03	3.0E+03	3.1E+03	3.1E+03	3.1E+03
CO-60	5.5E+02	1.2E+03	2.3E+03	2.1E+04	5.5E+04	5.8E+04	5.3E+04
KR-35	6.0E-01	6.1E-01	6.1E-01	6.1E-01	6.1E-01	6.1E-01	6.1E-01
KR-85M	3.9E-01	3.9E-01	3.9E-01	3.9E-01	3.9E-01	3.9E-01	3.9E-01
KR-87	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00	1.3E+00
KP-38	3.1E+00	3.1E+00	3.1E+00	3.1E+00	3.1E+00	3.1E+00	3.1E+00
RB-86	5.0E+02	1.7E+03	4.8E+03	6.5E+03	6.5E+03	6.5E+03	6.5E+03
SR-39	5.2E+02	1.7E+03	5.0E+03	1.3E+04	1.3E+04	1.3E+04	1.3E+04
SP-90	3.8E+02	2.2E+03	1.0E+04	1.1E+05	5.3E+05	6.9E+05	7.3E+05
SR-91	1.9E+02	2.0E+02	2.3E+02	3.1E+02	3.2E+02	3.2E+02	3.2E+02
Y-90	2.0E+02	4.3E+02	5.1E+02	5.1E+02	5.1E+02	5.1E+02	5.1E+02
Y-91	1.6E+02	5.7E+02	2.3E+03	9.2E+03	9.3E+03	9.3E+03	9.3E+03
ZR-95	1.8E+02	4.0E+02	9.4E+02	3.5E+03	3.6E+03	3.6E+03	3.6E+03
ZR-97	1.7E+02	1.9E+02	1.9E+02	1.9E+02	1.9E+02	1.9E+02	1.9E+02
NB-95	1.3E+02	3.8E+02	7.7E+02	1.4E+03	1.4E+03	1.4E+03	1.4E+03
MO-99	7.5E+01	1.2E+02	1.3E+02	1.3E+02	1.3E+02	1.3E+02	1.3E+02
TC-99M	1.1E+01	1.1E+01	1.1E+01	1.1E+01	1.1E+01	1.1E+01	1.1E+01
RU-103	1.3E+02	2.6E+02	5.5E+02	1.1E+03	1.1E+03	1.1E+03	1.1E+03
RU-105	2.3E+01	2.4E+01	2.4E+01	2.4E+01	2.4E+01	2.4E+01	2.4E+01
RU-106	7.6E+01	2.2E+02	6.6E+02	3.6E+03	6.2E+03	6.2E+03	6.2E+03
RH-105	1.6E+01	2.3E+01	2.3E+01	2.3E+01	2.3E+01	2.3E+01	2.3E+01
TE-127	3.7E+00	3.9E+00	3.9E+00	3.9E+00	3.9E+00	3.9E+00	3.9E+00
TE-127M	2.6E+01	9.5E+01	2.7E+02	7.5E+02	8.0E+02	8.0E+02	8.0E+02
TE-129	1.1E+00	1.1E+00	1.1E+00	1.1E+00	1.1E+00	1.1E+00	1.1E+00
TE-129M	6.7E+01	2.2E+02	5.3E+02	8.3E+02	8.4E+02	8.4E+02	8.4E+02
TE-131M	2.1E+02	2.9E+02	3.1E+02	3.1E+02	3.1E+02	3.1E+02	3.1E+02
TE-132	4.8E+02	8.8E+02	1.0E+03	1.0E+03	1.0E+03	1.0E+03	1.0E+03
SB-127	1.7E+02	2.9E+02	3.3E+02	3.3E+02	3.3E+02	3.3E+02	3.3E+02
SB-129	4.5E+01	4.6E+01	4.6E+01	4.6E+01	4.6E+01	4.6E+01	4.6E+01
I-131	7.7E+01	1.2E+02	1.9E+02	1.9E+02	1.9E+02	1.9E+02	1.9E+02
I-132	5.0E+01	5.0E+01	5.0E+01	5.0E+01	5.0E+01	5.0E+01	5.0E+01
I-133	8.8E+01	9.3E+01	9.4E+01	9.4E+01	9.4E+01	9.4E+01	9.4E+01
I-134	2.0E+01	2.0E+01	2.0E+01	2.0E+01	2.0E+01	2.0E+01	2.0E+01
I-135	9.1E+01	9.1E+01	9.1E+01	9.1E+01	9.1E+01	9.1E+01	9.1E+01
XE-133	1.5E+00	1.6E+00	1.6E+00	1.6E+00	1.6E+00	1.6E+00	1.6E+00
XE-135	2.1E+00	2.1E+00	2.1E+00	2.1E+00	2.1E+00	2.1E+00	2.1E+00
CS-134	5.8E+02	2.0E+03	7.9E+03	4.3E+04	4.8E+04	4.8E+04	4.8E+04
CS-136	6.9E+02	2.1E+03	5.0E+03	6.0E+03	6.0E+03	6.0E+03	6.0E+03
CS-137	3.6E+02	1.3E+03	5.2E+03	3.1E+04	3.7E+04	3.7E+04	3.7E+04
BA-140	4.8E+02	1.4E+03	2.8E+03	3.4E+03	3.4E+03	3.4E+03	3.4E+03
LA-140	4.4E+02	6.6E+02	6.8E+02	6.8E+02	6.8E+02	6.8E+02	6.8E+02
CE-141	3.7E+01	7.6E+01	1.5E+02	2.7E+02	2.7E+02	2.7E+02	2.7E+02
CE-143	6.6E+01	9.1E+01	1.0E+02	1.1E+02	1.1E+02	1.1E+02	1.1E+02
CE-144	6.2E+01	1.2E+02	3.5E+02	3.6E+03	9.2E+03	9.2E+03	9.2E+03
PR-143	3.0E+00	9.6E+00	2.6E+01	3.4E+01	3.4E+01	3.4E+01	3.4E+01
ND-147	5.5E+01	1.1E+02	1.7E+02	1.9E+02	2.0E+02	2.0E+02	2.0E+02
HP-239	4.0E+01	6.0E+01	6.4E+01	6.4E+01	6.4E+01	6.4E+01	6.4E+01
PU-238	1.8E+01	6.3E+01	2.8E+02	6.0E+03	2.3E+05	8.7E+05	1.3E+06
PU-239	1.6E+01	5.3E+01	2.6E+02	5.6E+03	2.3E+05	9.2E+05	1.5E+06
PU-240	1.6E+01	5.8E+01	2.6E+02	5.6E+03	2.3E+05	9.2E+05	1.5E+06
PU-241	3.3E-03	1.2E-02	7.2E-02	6.1E+00	1.8E+03	1.5E+04	3.2E+04
AM-241	4.0E+01	1.1E+02	4.2E+02	7.2E+03	2.5E+05	9.9E+05	1.6E+06
CM-242	2.1E+01	7.6E+01	3.3E+02	3.2E+03	6.1E+03	8.5E+03	1.0E+04
CM-244	2.0E+01	7.3E+01	3.3E+02	6.6E+03	2.1E+05	5.8E+05	7.4E+05

Table 2-11. (Continued) Body Organ: Lung.

Radionuclide	0-2 Days	0-7 Days	0-30 Days	0-1 Year	0-10 Years	0-30 Years	0-50 Years
CO-53	1.9E+03	5.2E+03	1.7E+04	5.9E+04	6.1E+04	6.1E+04	6.1E+04
CO-60	4.8E+03	1.3E+04	4.9E+04	4.6E+05	1.2E+06	1.3E+06	1.3E+06
KR-85	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01	1.8E-01
KR-85M	2.1E-01	2.1E-01	2.1E-01	2.1E-01	2.1E-01	2.1E-01	2.1E-01
KR-87	9.6E-01	9.6E-01	9.6E-01	9.6E-01	9.6E-01	9.6E-01	9.6E-01
KR-88	2.0E+00	2.0E+00	2.0E+00	2.0E+00	2.0E+00	2.0E+00	2.0E+00
RB-86	7.2E+03	9.0E+03	1.2E+04	1.4E+04	1.4E+04	1.4E+04	1.4E+04
SR-89	6.0E+03	6.8E+03	7.2E+03	7.8E+03	7.8E+03	7.8E+03	7.8E+03
SR-90	4.6E+03	9.3E+03	1.2E+04	1.6E+04	1.8E+04	1.8E+04	1.8E+04
SR-91	3.4E+03	3.5E+03	3.8E+03	4.3E+03	4.3E+03	4.3E+03	4.3E+03
Y-90	1.7E+04	2.9E+04	3.3E+04	3.3E+04	3.3E+04	3.3E+04	3.3E+04
Y-91	1.3E+04	3.5E+04	1.0E+05	2.0E+05	2.0E+05	2.0E+05	2.0E+05
ZR-95	3.5E+03	9.7E+03	3.4E+04	1.3E+05	1.3E+05	1.3E+05	1.3E+05
ZR-97	1.4E+04	1.5E+04	1.5E+04	1.5E+04	1.5E+04	1.5E+04	1.5E+04
NB-95	1.8E+03	4.9E+03	1.5E+04	3.1E+04	3.1E+04	3.1E+04	3.1E+04
MO-99	7.7E+03	1.4E+04	1.6E+04	1.6E+04	1.6E+04	1.6E+04	1.6E+04
TC-99M	8.9E+01	8.9E+01	8.9E+01	8.9E+01	8.9E+01	8.9E+01	8.9E+01
RU-103	2.9E+03	7.6E+03	2.4E+04	5.4E+04	5.4E+04	5.4E+04	5.4E+04
RU-105	2.1E+03	2.2E+03	2.2E+03	2.2E+03	2.2E+03	2.2E+03	2.2E+03
RU-106	3.3E+04	9.1E+04	3.3E+05	2.5E+06	3.0E+06	3.0E+06	3.0E+06
RH-105	2.5E+03	3.5E+03	3.6E+03	3.6E+03	3.6E+03	3.6E+03	3.6E+03
TE-127	1.5E+03	1.6E+03	1.6E+03	1.6E+03	1.6E+03	1.6E+03	1.6E+03
TE-127M	5.2E+03	1.6E+04	5.2E+04	1.2E+05	1.2E+05	1.2E+05	1.2E+05
TE-129	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02
TE-129M	1.3E+04	3.5E+04	9.5E+04	1.5E+05	1.5E+05	1.5E+05	1.5E+05
TE-131M	6.6E+03	9.4E+03	1.1E+04	1.1E+04	1.1E+04	1.1E+04	1.1E+04
TE-132	1.3E+04	2.4E+04	3.0E+04	3.0E+04	3.0E+04	3.0E+04	3.0E+04
SB-127	9.1E+03	1.0E+04	2.4E+04	2.5E+04	2.5E+04	2.5E+04	2.5E+04
SB-129	3.1E+03	3.1E+03	3.1E+03	3.2E+03	3.2E+03	3.2E+03	3.2E+03
I-131	2.1E+03	2.3E+03	2.4E+03	2.4E+03	2.4E+03	2.4E+03	2.4E+03
I-132	1.0E+03	1.0E+03	1.0E+03	1.0E+03	1.0E+03	1.0E+03	1.0E+03
I-133	3.1E+03	3.1E+03	3.1E+03	3.1E+03	3.1E+03	3.1E+03	3.1E+03
I-134	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02	5.6E+02
I-135	2.5E+03	2.5E+03	2.5E+03	2.5E+03	2.5E+03	2.5E+03	2.5E+03
XE-133	4.1E-01	4.1E-01	4.1E-01	4.1E-01	4.1E-01	4.1E-01	4.1E-01
XE-135	9.4E-01	9.4E-01	9.4E-01	9.4E-01	9.4E-01	9.4E-01	9.4E-01
CS-134	3.0E+03	4.7E+03	1.1E+04	4.5E+04	5.1E+04	5.1E+04	5.1E+04
CS-136	2.9E+03	4.5E+03	7.3E+03	8.2E+03	8.2E+03	8.2E+03	8.2E+03
CS-137	3.2E+03	4.4E+03	8.3E+03	3.4E+04	4.0E+04	4.0E+04	4.0E+04
BA-140	4.6E+03	5.7E+03	6.1E+03	6.3E+03	6.3E+03	6.3E+03	6.3E+03
LA-140	1.0E+04	1.5E+04	1.6E+04	1.6E+04	1.6E+04	1.6E+04	1.6E+04
CE-141	4.0E+03	1.0E+04	3.1E+04	6.2E+04	6.2E+04	6.2E+04	6.2E+04
CE-143	7.0E+03	1.0E+04	1.2E+04	1.3E+04	1.3E+04	1.3E+04	1.3E+04
CE-144	2.9E+04	8.2E+04	3.0E+05	2.1E+06	2.9E+06	2.9E+06	2.9E+06
PR-143	6.9E+03	1.7E+04	4.0E+04	4.9E+04	4.9E+04	4.9E+04	4.9E+04
ND-147	6.1E+03	1.5E+04	3.1E+04	3.7E+04	3.8E+04	3.8E+04	3.8E+04
NP-239	5.0E+03	8.3E+03	9.2E+03	9.2E+03	9.3E+03	9.3E+03	9.3E+03
PU-238	1.3E+06	3.6E+06	1.3E+07	1.2E+08	3.1E+08	3.1E+08	3.1E+08
PU-239	1.2E+06	3.3E+06	1.2E+07	1.2E+08	2.9E+08	2.9E+08	2.9E+08
PU-240	1.2E+06	3.3E+06	1.2E+07	1.2E+08	2.9E+08	2.9E+08	2.9E+08
PU-241	1.3E+02	3.7E+02	1.8E+03	6.4E+04	5.3E+05	5.7E+05	5.9E+05
AM-241	1.3E+06	3.6E+06	1.3E+07	1.3E+08	3.1E+08	3.1E+08	3.2E+08
CM-242	1.4E+06	3.9E+06	1.4E+07	7.6E+07	8.7E+07	8.7E+07	8.7E+07
CM-244	1.4E+06	3.8E+06	1.4E+07	1.3E+08	3.1E+08	3.1E+08	3.1E+08

Table 2-11. (Continued) Body Organ: Lower Large Intestine Wall.

RADIONUCLIDE	0-2 Days	0-7 Days	0-30 Days	0-60 Days
CO-58	3.2E+03	6.9E+03	7.1E+03	7.2E+03
CO-60	8.9E+03	1.8E+04	1.8E+04	1.9E+04
KR-85	1.8E-01	1.8E-01	1.8E-01	1.8E-01
KR-85M	2.2E-01	2.2E-01	2.2E-01	2.2E-01
KR-87	1.0E+00	1.0E+00	1.0E+00	1.0E+00
KR-88	2.3E+00	2.3E+00	2.3E+00	2.3E+00
RE-P6	1.2E+03	2.7E+03	5.8E+03	7.1E+03
SR-89	9.2E+03	1.4E+04	1.4E+04	1.4E+04
SR-90	7.3E+03	1.4E+04	1.6E+04	1.6E+04
SR-91	2.4E+03	2.6E+03	2.6E+03	2.6E+03
Y-90	2.2E+04	4.9E+04	4.9E+04	4.9E+04
Y-91	2.0E+04	4.6E+04	4.9E+04	5.0E+04
ZP-95	6.1E+03	1.4E+04	1.4E+04	1.5E+04
ZR-97	1.5E+04	1.9E+04	1.9E+04	1.9E+04
HR-95	3.2E+03	6.9E+03	7.0E+03	7.1E+03
HO-99	1.2E+04	2.9E+04	2.1E+04	2.1E+04
TC-99M	1.1E+01	1.1E+01	1.1E+01	1.1E+01
RU-103	4.8E+03	1.0E+04	1.1E+04	1.1E+04
RU-105	9.1E+02	1.2E+03	1.2E+03	1.2E+03
RU-106	5.5E+04	1.2E+05	1.3E+05	1.3E+05
RH-105	3.3E+03	5.1E+03	5.1E+03	5.1E+03
TE-127	7.3E+02	7.8E+02	7.3E+02	7.3E+02
TE-127M	6.9E+03	1.7E+04	2.0E+04	2.1E+04
TE-129	5.7E+00	5.7E+00	5.7E+00	5.7E+00
TE-129M	1.3E+04	3.4E+04	3.6E+04	3.7E+04
TE-131M	5.8E+03	8.5E+03	8.5E+03	8.5E+03
TE-132	3.3E+03	6.0E+03	6.1E+03	6.1E+03
SD-127	1.3E+04	2.6E+04	2.6E+04	2.6E+04
SD-129	7.9E+02	8.1E+02	8.1E+02	8.1E+02
I-131	2.6E+02	3.3E+02	3.5E+02	3.6E+02
I-132	6.9E+01	6.9E+01	6.9E+01	6.9E+01
I-133	3.9E+02	3.3E+02	3.3E+02	3.3E+02
I-134	2.9E+01	2.9E+01	2.9E+01	2.9E+01
I-135	2.2E+02	2.2E+02	2.2E+02	2.2E+02
XE-133	4.2E-01	4.2E-01	4.2E-01	4.2E-01
XE-135	9.9E-01	9.9E-01	9.9E-01	9.9E-01
CS-134	9.0E+02	2.8E+03	1.9E+04	1.9E+04
CS-136	9.9E+02	2.8E+03	6.4E+03	7.5E+03
CS-137	6.8E+02	1.9E+03	6.3E+03	1.1E+04
BA-140	9.4E+03	1.6E+04	1.6E+04	1.6E+04
LA-140	1.3E+04	2.1E+04	2.1E+04	2.1E+04
CE-141	6.7E+03	1.5E+04	1.5E+04	1.5E+04
CE-143	9.8E+03	1.6E+04	1.6E+04	1.6E+04
CE-144	5.2E+04	1.2E+05	1.2E+05	1.2E+05
PR-143	1.2E+04	2.5E+04	2.5E+04	2.5E+04
ND-147	1.0E+04	2.1E+04	2.2E+04	2.2E+04
HP-239	7.4E+03	1.3E+04	1.3E+04	1.3E+04
PU-238	2.2E+04	5.0E+04	5.1E+04	5.2E+04
PU-239	2.0E+04	4.6E+04	4.7E+04	4.8E+04
PU-240	2.1E+04	4.7E+04	4.8E+04	4.8E+04
PU-241	2.1E+02	4.7E+02	4.8E+02	4.8E+02
AM-241	2.3E+04	5.2E+04	5.4E+04	5.5E+04
CM-242	2.4E+04	5.5E+04	5.6E+04	5.7E+04
CM-244	2.3E+04	5.2E+04	5.4E+04	5.5E+04

Table 2-11. (Continued) Body Organ: Skeleton.

Radionuclide	0-2 Days	0-7 Days	0-30 Days	0-1 Year	0-10 Years	0-30 Years	0-50 Years
CO-58	1.5E+02	3.3E+02	8.4E+02	2.5E+03	2.6E+03	2.6E+03	2.6E+03
CO-60	3.5E+02	8.0E+02	2.2E+03	1.8E+04	4.3E+04	5.0E+04	5.7E+04
KR-85	1.5E-01	1.5E-01	1.5E-01	1.5E-01	1.5E-01	1.5E-01	1.5E-01
KR-85M	1.7E-01	1.9E-01	1.9E-01	1.9E-01	1.9E-01	1.9E-01	1.9E-01
KR-87	8.3E-01	8.3E-01	8.3E-01	8.3E-01	8.3E-01	8.3E-01	8.3E-01
KR-88	1.8E+00	1.8E+00	1.8E+00	1.8E+00	1.8E+00	1.8E+00	1.8E+00
RB-86	5.8E+02	1.7E+03	4.8E+03	6.5E+03	6.5E+03	6.5E+03	6.5E+03
SR-90	7.2E+02	3.0E+03	1.0E+04	3.3E+04	3.0E+04	3.0E+04	3.0E+04
SR-90	5.3E+02	3.0E+03	2.1E+04	2.0E+05	1.6E+06	2.4E+06	2.3E+06
SR-91	1.9E+02	2.0E+02	2.3E+02	3.4E+02	3.4E+02	3.4E+02	3.4E+02
Y-90	4.1E+02	8.6E+02	1.0E+03	1.0E+03	1.0E+03	1.0E+03	1.0E+03
Y-91	3.4E+02	1.2E+03	4.7E+03	1.7E+04	1.2E+04	1.9E+04	1.9E+04
ZR-95	1.2E+02	2.0E+02	7.0E+02	3.3E+03	3.4E+03	3.4E+03	3.4E+03
ZR-97	1.2E+02	1.3E+02	1.3E+02	1.3E+02	1.3E+02	1.3E+02	1.3E+02
HR-95	1.2E+02	2.6E+02	6.2E+02	1.2E+03	1.2E+03	1.2E+03	1.2E+03
MO-99	6.2E+01	1.0E+02	1.1E+02	1.1E+02	1.1E+02	1.1E+02	1.1E+02
TC-99M	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01	1.0E+01
RU-103	7.7E+01	1.0E+02	4.3E+02	8.8E+02	8.8E+02	8.8E+02	8.8E+02
RU-105	1.6E+01	1.7E+01	1.7E+01	1.7E+01	1.7E+01	1.7E+01	1.7E+01
RU-106	5.8E+01	1.9E+02	6.7E+02	3.4E+03	5.9E+03	5.9E+03	5.9E+03
RH-105	1.1E+01	1.6E+01	1.6E+01	1.6E+01	1.6E+01	1.6E+01	1.6E+01
TE-127	5.0E+00	5.2E+00	5.2E+00	5.2E+00	5.2E+00	5.2E+00	5.2E+00
TE-127M	3.1E+01	1.3E+02	4.4E+02	1.8E+03	2.9E+03	2.0E+03	2.0E+03
TE-129	1.2E+00	1.2E+00	1.2E+00	1.2E+00	1.2E+00	1.2E+00	1.2E+00
TE-129M	7.5E+01	2.6E+02	7.5E+02	1.4E+03	1.4E+03	1.4E+03	1.4E+03
TE-131M	1.6E+02	2.3E+02	2.5E+02	2.5E+02	2.5E+02	2.5E+02	2.5E+02
TE-132	4.4E+02	8.0E+02	9.1E+02	9.1E+02	9.1E+02	9.1E+02	9.1E+02
SB-127	1.2E+02	2.1E+02	2.5E+02	2.6E+02	2.6E+02	2.6E+02	2.6E+02
SB-129	3.8E+01	3.8E+01	3.8E+01	3.9E+01	3.9E+01	3.9E+01	3.9E+01
I-131	7.6E+01	1.2E+02	2.0E+02	2.1E+02	2.1E+02	2.1E+02	2.1E+02
I-132	4.7E+01	4.7E+01	4.7E+01	4.7E+01	4.7E+01	4.7E+01	4.7E+01
I-133	8.7E+01	9.2E+01	9.2E+01	9.2E+01	9.2E+01	9.2E+01	9.2E+01
I-134	1.9E+01	1.9E+01	1.9E+01	1.9E+01	1.9E+01	1.9E+01	1.9E+01
I-135	8.7E+01	8.7E+01	8.7E+01	8.7E+01	8.7E+01	8.7E+01	8.7E+01
XE-133	3.5E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01	3.6E-01
XE-135	7.2E-01	7.2E-01	7.2E-01	7.2E-01	7.2E-01	7.2E-01	7.2E-01
CS-134	5.7E+02	2.0E+03	7.8E+03	4.2E+04	4.7E+04	4.7E+04	4.7E+04
CS-136	6.6E+02	2.0E+03	4.9E+03	5.9E+03	5.9E+03	5.9E+03	5.9E+03
CS-137	3.6E+02	1.3E+03	5.1E+03	3.1E+04	3.6E+04	3.6E+04	3.6E+04
BA-140	6.1E+02	2.0E+03	4.2E+03	5.2E+03	5.2E+03	5.2E+03	5.2E+03
LA-140	4.2E+02	6.7E+02	7.0E+02	7.0E+02	7.0E+02	7.0E+02	7.0E+02
CE-141	2.5E+01	6.1E+01	1.6E+02	3.2E+02	3.2E+02	3.2E+02	3.2E+02
CE-143	4.7E+01	7.1E+01	9.4E+01	1.1E+02	1.1E+02	1.1E+02	1.1E+02
CE-144	1.0E+02	2.0E+02	6.3E+02	7.2E+03	1.9E+04	1.7E+04	1.7E+04
PR-143	7.6E+00	2.4E+01	6.5E+01	8.6E+01	8.6E+01	8.6E+01	8.6E+01
ND-147	3.8E+01	8.5E+01	1.0E+02	1.9E+02	2.3E+02	2.4E+02	2.4E+02
NP-239	3.0E+01	4.9E+01	5.4E+01	5.4E+01	8.0E+01	1.6E+02	2.3E+02
PU-238	9.9E+03	3.5E+04	1.6E+05	3.4E+06	1.3E+08	4.9E+08	7.6E+08
PU-239	9.2E+03	3.3E+04	1.5E+05	3.1E+06	1.3E+08	5.2E+08	8.7E+08
PU-240	9.2E+03	3.3E+04	1.5E+05	3.1E+06	1.3E+08	5.2E+08	8.7E+08
PU-241	2.4E-01	1.2E+00	1.3E+01	2.7E+03	9.8E+05	8.3E+06	1.7E+07
AM-241	1.1E+04	3.9E+04	1.8E+05	3.6E+06	1.4E+08	5.5E+08	9.0E+08
CM-242	1.2E+04	4.3E+04	1.8E+05	1.9E+06	3.4E+06	4.8E+06	5.8E+06
CM-244	1.1E+04	4.1E+04	1.9E+05	3.7E+06	1.2E+08	3.3E+08	4.2E+08

Table 2-11. (Continued) Body Organ: Thyroid.

Radionuclide	0-2 Days	0-7 Days	0-30 Days	0-60 Days
CO-58	1.1E+02	2.9E+02	9.5E+02	1.6E+03
CO-60	2.4E+02	6.5E+02	2.3E+03	4.4E+03
KR-85	1.8E-01	1.8E-01	1.8E-01	1.8E-01
KR-85M	2.0E-01	2.0E-01	2.0E-01	2.0E-01
KR-87	9.7E-01	9.7E-01	9.7E-01	9.7E-01
KR-88	2.0E+00	2.0E+00	2.0E+00	2.0E+00
RS-86	5.9E+02	1.7E+03	4.8E+03	6.1E+03
SR-89	2.0E+02	5.7E+02	9.2E+02	1.2E+03
SR-90	1.8E+02	7.0E+02	1.9E+03	2.8E+03
SR-91	1.3E+02	1.4E+02	1.6E+02	1.7E+02
Y-90	8.2E+00	1.7E+01	2.1E+01	2.1E+01
Y-91	7.1E+00	2.5E+01	9.8E+01	1.3E+02
ZR-95	7.9E+01	2.2E+02	8.0E+02	1.5E+03
ZR-97	7.7E+01	8.6E+01	8.6E+01	8.6E+01
HO-95	8.1E+01	2.1E+02	6.4E+02	9.5E+02
HO-99	9.4E+01	1.4E+02	1.5E+02	1.5E+02
TC-99M	4.5E+01	4.6E+01	4.6E+01	4.6E+01
RU-103	5.2E+01	1.4E+02	4.3E+02	6.6E+02
RU-105	1.4E+01	1.5E+01	1.5E+01	1.5E+01
RU-106	4.8E+01	1.7E+02	6.2E+02	1.9E+03
RH-105	6.4E+00	9.6E+00	9.9E+00	9.9E+00
TE-127	2.9E+00	3.0E+00	3.0E+00	3.0E+00
TE-127M	1.6E+01	6.3E+01	1.6E+02	1.9E+02
TE-129	8.1E-01	8.1E-01	8.1E-01	8.1E-01
TE-129M	4.3E+01	1.4E+02	3.2E+02	3.7E+02
TE-131M	4.5E+03	3.4E+04	8.7E+04	9.5E+04
TE-132	4.8E+04	8.8E+04	9.7E+04	9.7E+04
SB-127	1.0E+02	1.8E+02	2.2E+02	2.2E+02
SB-129	3.7E+01	3.7E+01	3.8E+01	3.8E+01
I-131	1.3E+05	4.8E+05	1.9E+06	1.1E+06
I-132	6.6E+03	6.6E+03	6.6E+03	6.6E+03
I-133	1.2E+05	1.8E+05	1.3E+05	1.8E+05
I-134	1.1E+03	1.1E+03	1.1E+03	1.1E+03
I-135	4.3E+04	4.4E+04	4.4E+04	4.4E+04
XE-133	3.9E-01	4.0E-01	4.0E-01	4.0E-01
XE-135	9.1E-01	9.1E-01	9.1E-01	9.1E-01
CS-134	5.8E+02	2.0E+03	7.9E+03	1.4E+04
CS-136	6.9E+02	2.1E+03	5.9E+03	5.9E+03
CS-137	3.6E+02	1.3E+03	5.1E+03	9.5E+03
BA-140	2.2E+02	5.9E+02	1.9E+03	1.2E+03
LA-140	1.5E+02	2.2E+02	2.3E+02	2.3E+02
CE-141	6.0E+00	1.6E+01	4.6E+01	6.9E+01
CE-143	1.8E+01	2.4E+01	2.5E+01	2.5E+01
CE-144	5.1E+00	1.3E+01	4.5E+01	8.4E+01
PR-143	9.3E-02	2.0E-01	7.9E-01	9.9E-01
HO-147	1.2E+01	2.8E+01	6.9E+01	6.8E+01
HP-239	8.2E+00	1.4E+01	1.5E+01	1.5E+01
PU-238	1.8E+01	6.2E+01	2.8E+02	6.0E+02
PU-239	1.6E+01	5.8E+01	2.6E+02	5.6E+02
PU-240	1.6E+01	5.8E+01	2.6E+02	5.6E+02
PU-241	1.7E-03	6.9E-03	4.5E-02	1.4E-01
AM-241	2.0E+01	7.3E+01	3.3E+02	6.9E+02
CM-242	2.1E+01	7.5E+01	3.3E+02	6.5E+02
CM-244	2.0E+01	7.3E+01	3.3E+02	7.0E+02

$I_{1b}(t)$. For example, the incremental dose during the third ten-year time interval after the accident ($t = 20$ to 30 yr) would be:

$$I_{1b}(0-10\text{yr})DCF_2(20-30\text{yr}) + I_{1b}(10-20\text{yr})DCF_2(10-20\text{yr}) \\ + I_{1b}(20-30\text{yr})DCF_2(0-10\text{yr}) \quad (2-16)$$

the sum of the doses due to materials inhaled in the first thirty years after the accident.

Groundshine

There are two groundshine models, one for early exposure and another for chronic exposure. First the initial concentration of material deposited on the ground, G_0 (Ci/m^2) is calculated. The initial ground concentration is the sum of dry deposition and wet deposition, each integrated over the interval during which the cloud (plume) is passing over the area of interest; i.e., from its time of arrival, t_a , to its time of departure, t_b . The deposition velocity V_d is set to zero for noble gases and to 1 cm/s for the other nuclides. The dry deposition component of the ground concentration is

$$\int_{t_a}^{t_b} V_d A(t) dt \quad (2-17)$$

and the wet deposition is modeled such that the airborne concentration, $A(t)$, during rain decreases by $1/e$ ($e = 2.718$) in 10^4 seconds for stable conditions and in 10^3 seconds in convective storms. (Again, the noble gases are not assumed to be deposited.)

The model for early exposure is relatively simple. The dose is found simply by multiplying the initial ground concentration (Ci/m^2) by a shielding factor, SF^g , and a dose conversion factor (rem per Ci/m^2), D_g^g :

$$E_g = G_0 D_g^g SF^g \quad (2-18)$$

The dose conversion factors for time periods of 1 day and 7 days are given in Table 2-12 (RSS Table VI C-2) for the whole body, total marrow, lung and testes. Shielding factors for groundshine vary from 0.02 for an office or large apartment building to 0.7 above ordinary ground. A shielding factor of 1.0 would apply to an infinite, smooth surface. The ground shielding factors used are 0.5 for persons within 25 miles of the nuclear power plant and 0.33 at greater distances. The 0.5 shielding factor for persons within 25 miles of the site is based on the assumption that they would be evacuating the area in autos, see RSS Table VI 11-8. The 0.33 factor for others is based upon "normal" time-weighted activity patterns and the shielding offered by representative structures; see RSS Table VI 11-12.

The model for chronic exposure from groundshine is a bit more complicated. The initial ground concentration is depleted by radioactive decay,

Table 2-12. Photon Dose-Conversion Factors for Exposure to Contaminated Ground (rem per Ci/m²) (Time-Integral Dose to N Days). (a)

(RSS Table VI C-2)

Radionuclide	Whole Body ^(b)		Total Marrow		Lung		Testes	
	1 Day	7 Days	1 Day	7 Days	1 Day	7 Days	1 Day	7 Days
CO-58	3.29E+02	2.24E+03	3.67E+02	2.50E+03	3.08E+02	2.10E+03	3.08E+02	2.09E+03
CO-60	8.43E+02	5.82E+03	8.89E+02	6.22E+03	7.39E+02	5.58E+03	6.01E+02	4.80E+03
KR-85	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
KR-85M	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
KR-87	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
KR-88	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
RS-86	2.93E+01	1.85E+02	3.22E+01	2.02E+02	2.75E+01	1.73E+02	2.66E+01	1.66E+02
SR-89	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SR-90	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
SR-91	1.63E+02	2.05E+02	1.96E+02	2.32E+02	1.59E+02	1.73E+02	1.70E+02	2.07E+02
Y-90	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Y-91	8.82E+01	5.91E+00	8.92E+01	6.05E+01	8.39E+01	5.66E+00	6.61E+01	4.44E+00
ZR-95	2.47E+02	1.77E+03	2.85E+02	2.04E+03	2.32E+02	1.67E+03	2.46E+02	1.76E+03
ZR-97	3.33E+02	5.38E+02	4.02E+02	6.52E+02	3.14E+02	5.10E+02	3.59E+02	5.39E+02
NS-95	2.49E+02	1.54E+03	2.73E+02	1.87E+03	2.33E+02	1.54E+03	2.25E+02	1.49E+03
PO-99	7.73E+01	3.25E+02	1.07E+02	4.63E+02	6.95E+01	2.91E+02	7.95E+01	3.32E+02
TC-99M	1.52E+01	1.62E+01	2.70E+01	2.82E+01	1.25E+01	1.35E+01	1.41E+01	1.51E+01
RU-103	1.75E+02	1.16E+03	2.14E+02	1.42E+03	1.65E+02	1.09E+03	1.92E+02	1.30E+03
RU-105	7.23E+01	7.94E+01	9.04E+01	9.92E+01	6.77E+01	7.37E+01	8.03E+01	8.91E+01
RU-106	6.51E+01	4.56E+02	7.96E+01	5.54E+02	6.17E+01	4.30E+02	7.15E+01	4.97E+02
RU-107	2.20E+01	5.67E+01	3.32E+01	8.55E+01	1.95E+01	5.01E+01	2.07E+01	7.37E+01
TE-127	6.77E+01	8.13E+01	8.43E+01	1.01E+02	6.30E+01	7.67E+01	7.52E+01	9.14E+01
TE-127M	7.90E+00	5.84E+01	1.27E+01	9.20E+01	4.40E+00	3.37E+01	1.07E+01	7.77E+01
TE-129	1.98E+00	1.90E+00	2.44E+00	2.44E+00	1.83E+00	1.83E+00	2.17E+00	2.17E+00
TE-129M	3.63E+01	2.46E+02	4.54E+01	3.07E+02	3.20E+01	2.22E+02	4.04E+01	2.74E+02
TE-131M	3.31E+02	9.60E+02	4.36E+02	1.20E+03	3.56E+02	8.90E+02	3.47E+02	8.86E+02
TE-132	6.77E+02	3.08E+03	8.04E+02	3.53E+03	6.36E+02	2.82E+03	6.07E+02	3.05E+03
SB-127	2.12E+02	9.20E+02	2.57E+02	1.11E+03	2.00E+02	8.65E+02	2.29E+02	9.95E+02
SB-129	1.00E+02	1.04E+02	1.12E+02	1.16E+02	9.47E+01	9.78E+01	9.25E+01	9.57E+01
I-131	1.28E+02	7.03E+02	1.59E+02	8.73E+02	1.21E+02	6.55E+02	1.43E+02	7.55E+02
I-132	1.07E+02	1.77E+02	1.23E+02	1.33E+02	1.01E+02	1.01E+02	1.05E+02	1.05E+02
I-133	1.63E+02	3.11E+02	1.93E+02	3.75E+02	1.54E+02	2.91E+02	1.70E+02	3.22E+02
I-134	4.14E+01	4.14E+01	4.56E+01	4.56E+01	3.88E+01	3.88E+01	3.76E+01	3.76E+01
I-135	2.52E+02	2.55E+02	2.77E+02	3.10E+02	2.38E+02	2.69E+02	2.13E+02	2.52E+02
XE-133	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
XE-135	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
CS-134	5.30E+02	3.60E+03	6.10E+02	4.26E+03	4.97E+02	3.47E+03	5.27E+02	3.67E+03
CS-136	6.04E+02	4.10E+03	7.79E+02	4.88E+03	6.37E+02	3.92E+03	6.33E+02	3.92E+03
CS-137	1.86E+02	1.31E+03	2.28E+02	1.60E+03	1.76E+02	1.24E+03	2.08E+02	1.46E+03
BA-140	2.13E+02	3.65E+03	2.42E+02	3.60E+03	2.02E+02	3.46E+03	1.92E+02	3.11E+03
LA-140	6.40E+02	1.80E+03	6.80E+02	1.92E+03	6.12E+02	1.71E+03	5.23E+02	1.46E+03
CE-141	2.77E+01	1.92E+02	4.93E+01	3.24E+02	2.28E+01	1.50E+02	2.63E+01	1.73E+02
CE-143	9.00E+01	2.24E+02	1.24E+02	3.00E+02	2.00E+01	2.00E+02	1.05E+02	2.59E+02
CE-144	1.72E+01	1.20E+02	2.30E+01	1.67E+02	1.52E+01	1.07E+02	1.46E+01	1.03E+02
PR-143	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
ND-147	5.20E+01	3.05E+02	7.28E+01	4.26E+02	4.63E+01	2.70E+02	5.71E+01	3.34E+02
NP-239	5.39E+01	2.02E+02	9.53E+01	3.26E+02	5.09E+01	1.74E+02	6.87E+01	2.35E+02
PU-238	8.95E+01	6.20E+00	7.17E+01	5.02E+00	1.62E+01	1.14E+00	7.10E+01	4.97E+00
PU-239	3.76E+01	2.63E+00	3.56E+01	2.40E+00	8.88E+00	6.22E+01	3.10E+01	2.23E+00
PU-240	7.84E+01	5.47E+00	6.53E+01	4.57E+00	1.55E+01	1.02E+00	6.25E+01	4.38E+00
PU-241	4.52E+05	2.21E+03	9.23E+05	4.52E+03	3.18E+05	1.56E+03	3.05E+05	1.94E+03
AM-241	2.06E+01	1.43E+02	4.21E+01	2.05E+02	1.45E+01	1.02E+02	1.80E+01	1.26E+02
CN-242	7.92E+01	5.46E+00	6.14E+01	4.25E+00	1.31E+01	9.05E+01	6.29E+01	4.35E+00
CN-244	4.96E+00	3.46E+01	9.73E+00	6.81E+01	3.73E+00	2.61E+01	4.40E+00	3.08E+01

(a) Note: noble gases do not deposit on the ground.

(b) For organs not listed the dose-conversion factors are similar to those of the whole body.

according to the half-life of the nuclide, T_h :

$$G_d(t) = G_0 \exp(-0.693/T_h t) \quad (2-19)$$

Weathering also depletes the ground concentration of the radionuclides, and the following formula is used to approximate the ground concentration during depletion:

$$G_w(t) = G_d(t) [0.63 \exp(-1.13t) + 0.37 \exp(-0.0075t)] \quad (2-20)$$

In this weathering equation t is given in years. The exposures are modified to account for shielding. The ground shielding factor actually used in the RSS for chronic groundshine was 0.33. The shielded exposure factors, $G_s(t)$, are obtained from the shielding factors (SF) by:

$$G_s(t) = G_w(t) SF \quad (2-21)$$

Finally, these shielded exposure factors are converted into organ-specific dose increments in each of the 10-year intervals after the accident using the dose conversion factors DCF_o (rem per year per Ci/m^2) calculated by EXREM-III and integrating over time intervals of interest; e.g.,

$$\begin{aligned} \bar{E}_2(0\text{yr} - 1\text{yr}) &= \int_0^1 G_s(t) DCF_o dt \\ \bar{E}_2(10\text{yr} - 20\text{yr}) &= (1/3) \int_1^{30} G_s(t) DCF_o dt \end{aligned} \quad (2-22)$$

Ingestion

The ingestion model considers two basic pathways: milk consumption (m) and consumption of "all other" food (o). Contaminated vegetation is the source of nuclides for both of these pathways. The model considers two ways in which the vegetation may become contaminated: direct deposition (dd) and root uptake (ru). Figure 2-15 summarizes the process schematically. Direct deposition affects only the crops being cultivated at the time of the accident, whereas root uptake is a continuing source of contamination for many years. If the accident does not occur during the growing season, the dose due to direct deposition is set to zero. Estimated doses are calculated, as indicated next, then compared with radiation protection guidelines, which are assumed to limit the dose which individuals would receive.

Thus the ingested dose, I_2 , reduces to a sum of four components:

$$I_2 = I_{2mdd} + I_{2odd} + I_{2mru} + I_{2oru} \quad (2-23)$$

In the ingestion model, each of these terms is related to the initial ground

Figure 2-15. Simplified schematic of ingestion model. Initial ground concentrations, G_0 (Ci/m²), are transferred to vegetation by direct deposition and root uptake. This transfer and the subsequent transfer from vegetation to humans via milk and "all other" foods are modeled with concentration factors, CF (Ci per Ci/m²). Finally, human doses are estimated using dose conversion factors, DCF_i (rem/Ci), applicable to each organ, i .

concentration, G_0 , as indicated below:

$$I_{2...}(t) = CFG_0 DCF_i(t) \quad (2-24)$$

Here, CF is a nuclide-specific concentration factor for either root uptake or direct deposition that is applicable either to consumption of milk or "all other" foods. The concentration factors, given in Tables 2-13 and 2-14 (RSS Tables VI E-5 and VI E-8), relate ground concentration (C_i/m^2) to amounts ingested (C_i) over a lifetime by individuals. Although Appendix VI of WASH-1400 is not clear on the details of time integration involved in these concentration factors, it appears that they are integrated over all time, i.e., from $t=0$ to $t=\infty$. This practice is "conservative" as it treats the lifetime ingestion as if it were ingested at the time of the accident. In addition, the milk concentration factors used in the model are "conservative" in that they have been derived from data for the most sensitive segment of the population; e.g., for I-131 in milk the data are for young children. The $DCF_i(t)$ are internal dose conversion factors calculated by program provided by Oak Ridge National Laboratory. The dose conversion factors for ingestion are given in Table 2-16 (RSS Table VI 8-4). Ingestion is modeled by using the inhalation model, assuming no retention in the lungs but direct passage to the G.I. tract. These calculations are organ-and-nuclide-specific.

The input to the ingestion model is the initial ground surface concentration, G_0 , determined by the deposition model. The first element of the ingestion model postulates that 50 percent of this initial ground surface concentration is the concentration that ends up on plant foliage:

$$\bar{F}_0 = 0.50 \bar{G}_0 \quad (2-25)$$

This concentration is assumed to be reduced by radioactive decay:

$$\bar{F}_d(t) = \bar{F}_0 \exp((-0.693/T_h)t) \quad (2-26)$$

Weathering is also incorporated, assumed to have a fourteen-day half-life:

$$\bar{F}_w(t) = \bar{F}_d(t) \exp((-0.693/14)t) \quad (2-27)$$

As mentioned above, the transfer of radionuclides from vegetation to humans is modeled through four pathways.

Intake of radionuclides in milk, contaminated via the "direct deposition" pathway, is estimated by taking into account grazing habits of cows, milk consumption rates of humans, and the processes governing transfer of radionuclides from the cows' digestive systems into the milk. The calculations assume that: each cow grazes on 45 m^2 /day of pasture area with a forage density of 0.25 kg/m^2 ; the cows always graze on contaminated pasture, never returning to previously grazed areas; three days elapse between milk production and consumption; human milk consumption rates are about 0.7 L/day per person; and all milk comes from cows grazing on contaminated land. The calculations are performed for seven radionuclides: I-131, I-133, Sr-89, Sr-90, Cs-134, Cs-136, and

Table 2-13. Total Intake by a Critical Individual as a Result of Direct Deposition. (a)

(RSS Table VI E-5)

Radionuclide	CF via milk (curies)	CF via "other" Pathways (curies)
Iodine-131	0.692	--
Iodine-133	0.0042	--
Strontium-89	0.402	0.397
Strontium-90	0.588	0.505
Cesium-134	4.22	8.44
Cesium-136	1.42	2.84
Cesium-137	4.22	8.44

(a) Values normalized to an initial surface deposition level of 1 curie per square meter.

Table 2-14. Total Ingestion of Strontium and Cesium Resulting from Root Uptake. (a)

(RSS Table VI E-8)

Radionuclide	CF via Milk (curies)	CF via "other" pathways (curies)
Strontium-89	0.00682	0.0136
Strontium-90	0.669	1.34
Cesium-134	0.0547	0.164
Cesium-137	0.0835	0.251

(a) Assuming an initial surface deposition of 1 curie per square meter.

Table 2-16. Dose-Conversion Factors for Ingestion of Radionuclides
(Rem per Curie Ingested).

(RSS Table VI 8-4.)

	<u>0-10 Yr</u>	<u>0-20 Yr</u>	<u>0-30 Yr</u>	<u>0-40 Yr</u>	<u>0-50 Yr</u>
<u>Whole Body</u>					
Cs-134	7.14×10^4	7.14×10^4	7.14×10^4	7.14×10^4	7.14×10^4
Cs-136	8.96×10^3	8.96×10^3	8.96×10^3	8.96×10^3	8.96×10^3
Cs-137	5.49×10^4	5.49×10^4	5.49×10^4	5.49×10^4	5.49×10^4
Sr-89	1.91×10^3	1.91×10^3	1.91×10^3	1.91×10^3	1.91×10^3
Sr-90	5.52×10^4	7.55×10^4	8.29×10^4	8.37×10^4	8.40×10^4
I-131	8.79×10^2	8.79×10^2	8.79×10^2	8.79×10^2	8.79×10^2
I-133	2.70×10^2	2.70×10^2	2.70×10^2	2.70×10^2	2.70×10^2
<u>Total Marrow</u>					
Cs-134	7.34×10^4	7.34×10^4	7.34×10^4	7.34×10^4	7.34×10^4
Cs-136	9.29×10^3	9.29×10^3	9.29×10^3	9.29×10^3	9.29×10^3
Cs-137	5.61×10^4	5.61×10^4	5.61×10^4	5.61×10^4	5.61×10^4
Sr-89	5.26×10^3	5.26×10^3	5.26×10^3	5.26×10^3	5.26×10^3
Sr-90	2.08×10^5	2.61×10^5	2.74×10^5	2.84×10^5	2.87×10^5
I-131	2.87×10^2	2.87×10^2	2.87×10^2	2.87×10^2	2.87×10^2
I-133	1.48×10^2	1.48×10^2	1.48×10^2	1.48×10^2	1.48×10^2
<u>Bone (Mineral)</u>					
Cs-134	7.24×10^4	7.24×10^4	7.24×10^4	7.24×10^4	7.24×10^4
Cs-136	9.10×10^3	9.10×10^3	9.10×10^3	9.10×10^3	9.10×10^3
Cs-137	5.56×10^4	5.56×10^4	5.56×10^4	5.56×10^4	5.56×10^4
Sr-89	1.19×10^4	1.19×10^4	1.19×10^4	1.19×10^4	1.19×10^4
Sr-90	6.15×10^5	8.72×10^5	9.70×10^5	1.08×10^6	1.08×10^6
I-131	3.10×10^2	3.10×10^2	3.10×10^2	3.10×10^2	3.10×10^2
I-133	1.46×10^2	1.46×10^2	1.46×10^2	1.46×10^2	1.46×10^2
<u>Thyroid</u>					
Cs-134	7.33×10^4	7.33×10^4	7.33×10^4	7.33×10^4	7.33×10^4
Cs-136	9.23×10^3	9.23×10^3	9.23×10^3	9.23×10^3	9.23×10^3
Cs-137	5.55×10^4	5.55×10^4	5.55×10^4	5.55×10^4	5.55×10^4
Sr-89	5.81×10^2	5.81×10^2	5.81×10^2	5.81×10^2	5.81×10^2
Sr-90	3.18×10^3	3.24×10^3	3.26×10^3	3.26×10^3	3.26×10^3
I-131	1.68×10^6	1.68×10^6	1.68×10^6	1.68×10^6	1.68×10^6
I-133	3.21×10^5	3.21×10^5	3.21×10^5	3.21×10^5	3.21×10^5
<u>Lung</u>					
Cs-134	7.31×10^4	7.31×10^4	7.31×10^4	7.31×10^4	7.31×10^4
Cs-136	8.82×10^3	8.82×10^3	8.82×10^3	8.82×10^3	8.82×10^3
Cs-137	5.59×10^4	5.59×10^4	5.59×10^4	5.59×10^4	5.59×10^4
Sr-89	5.81×10^2	5.81×10^2	5.81×10^2	5.81×10^2	5.81×10^2
Sr-90	3.18×10^3	3.72×10^3	3.74×10^3	3.74×10^3	3.74×10^3
I-131	3.56×10^2	3.56×10^2	3.56×10^2	3.56×10^2	3.56×10^2
I-133	1.58×10^2	1.58×10^2	1.58×10^2	1.58×10^2	1.58×10^2
<u>Lower Large Intestine Wall</u>					
Cs-134	9.33×10^4	9.33×10^4	9.33×10^4	9.33×10^4	9.33×10^4
Cs-136	1.35×10^4	1.35×10^4	1.35×10^4	1.35×10^4	1.35×10^4
Cs-137	6.64×10^4	6.64×10^4	6.64×10^4	6.64×10^4	6.64×10^4
Sr-89	8.53×10^4	8.53×10^4	8.53×10^4	8.53×10^4	8.53×10^4
Sr-90	8.12×10^4	8.12×10^4	8.12×10^4	8.12×10^4	8.12×10^4
I-131	1.91×10^3	1.91×10^3	1.91×10^3	1.91×10^3	1.91×10^3
I-133	1.82×10^2	1.82×10^2	1.82×10^2	1.82×10^2	1.82×10^2

(a) Rem per curie ingested.

Cs-137.

Intake of radionuclides in "all other foods", contaminated via the "direct deposition" pathway, is evaluated only for strontium and cesium. For both Sr-89 and Sr-90 the concentration factors are based upon: fresh vegetable consumption of 0.12 kg/day; crop yields of 2.4 kg/m²; no loss of radionuclides during harvesting or food preparation; and the assumption that all vegetables consumed will come from contaminated farming areas. For all three isotopes of cesium the "all other food" concentration factors are taken to be two times as large as the concentration factors for milk contaminated via "direct deposition".

The model used to estimate the concentration factors applicable to milk contaminated via root uptake is shown in Figure 2-16 (RSS Figure VI E-14). It involves differential equations describing the transfer of radionuclides from pasture soil to pasture grass, from pasture grass to milk, from milk to man, and the permanent transfer of radionuclides from pasture soil to a "soil sink". Again, it is assumed that an average individual consumes 0.7 L/day of milk and that a cow consumes 11.3 kg/day of forage from a pasture with grass density of 0.25 kg/m². In these calculations each cow is assumed to graze on an area of 8500 m². Finally, it is assumed that loss of radionuclides from pasture soil to the "soil sink" is 10% per year for strontium and 61% per year for cesium, and that the uptake of radionuclides from pasture soil to pasture grass is 5% per year for strontium and 0.23% per year for cesium.

Once values for intake due to milk contaminated via "root uptake" are calculated, the model assumes that strontium intake through the "all other food" path will be twice as great and for cesium that intake through the "all other food" path will be three times as great.

If these estimated doses from the ingestion model are above the allowable guidelines, the milk or the crops or the land will be taken out of use ("interdicted") or the cows placed on stored feed. The dose criteria used in the model to determine the necessity of interdiction are given in Table 2-15 (RSS Table VI 11-6). The crops are interdicted if they are excessively contaminated by direct deposition or if the land is interdicted because the groundshine is excessive, but the crops are not interdicted based on contamination levels due to root uptake. The use of a larger external dose criterion for urban areas is said to be consistent with policies for the use of the Protective Action Guides (PAG's) of the Federal Radiation Council and with the Emergency Reference Levels (ERL's) of the Medical Research Council of Great Britain. Thus, the actual doses through ingestion will be the smaller of the estimated dose and the allowed dose:

$$I_2 = \min[I_{total}, I_{allowed}] \quad (2-28)$$

Dose Integration

Using the four exposure pathways, doses to each organ are calculated for two broad time periods, early exposure and chronic exposure. For calculation of early dose only cloudshine, groundshine, and inhalation of nuclides from the passing cloud are considered. In calculating the chronic dose inhalation of resuspended particles and ingestion of contaminated foods are also considered.

Figure 2-16. Compartment model describing radionuclide movement to man via the root uptake-milk consumption pathway. Each transfer is modeled using a rate constant, e.g., λ_{rg} , λ_{gc} , λ_{cm} , λ_{rd} .

(RSS Figure VI E-14)

Table 2-15. Dose Criteria Used by Reactor Safety Study to Evaluate Necessity of Interdiction of Milk, Crops and Land.

(RSS Table VI 11-6)

Exposure	Dose
External irradiation:	
Low-population-density areas	10 rem to the whole body in 30 years
Urban areas	25 rem to the whole body in 30 years
Ingestion via milk:	
Strontium	3.3 rem to the bone marrow in first year
Cesium	3.3 rem to the whole body
Iodine	10.0 rem to the thyroid
Ingestion via "other" pathways:	
Strontium	2.0 rem to the bone marrow in first year
Cesium	2.0 rem to the whole body

The early dose is calculated uniquely for each organ of concern. For all organs the external doses from the passing cloud are included. The groundshine doses are included, but are truncated: after four hours for persons within 25 miles of the nuclear power plant; after one day for persons living beyond this distance who are "immediately" relocated due to locally high dose rates; and after seven days for others living beyond this distance who are relocated later. For the bone marrow to this are added the internal dose due to inhalation for the first 7 days and one-half of the internal dose due to inhalation received between the 8th and 30th day after the accident. For the lung the internal dose due to inhalation is integrated over the first year after the accident. For the GI tract, the inhaled dose calculation is truncated seven days after the accident. For calculations of early thyroid effects the inhaled dose is truncated thirty days after the accident.

Chronic doses to each organ are calculated for each of nine time periods after the accident: 0-1, 1-10, 11-20, 21-30, 31-40, 41-50, 51-60, 61-70, and 71-80 years. (Actually the 41-50 year ingestion and inhalation doses are used for all later time intervals). Calculation of the relevant dose for latent thyroid effects (i.e., nodules) is slightly different. The calculated dose includes only: external doses from passing cloud and from groundshine plus the internal dose from inhalation of all nuclides except I-131 during the first 30 days plus one-tenth of the I-131 inhalation dose during this same period.

Evacuation and Relocation

Evacuation in the RSS refers to a short-term movement of people from the immediate vicinity of the reactor after the accident on the basis that a core meltdown is expected. (The model is somewhat different from current USNRC policy for emergency response planning.) It is assumed that the evacuees (those people residing within 5 miles of the nuclear power plant in any direction, or within 25 miles of the nuclear power plant in the 45° arc centered on the direction of plume movement) will move radially outward from the reactor site at a constant effective speed until the cloud reaches them and then to move at this same speed in a circumferential direction. The distribution of evacuation speeds was taken to be: 0 mph ($p=0.3$); 1.2 mph ($p=0.4$); and 7 mph ($p=0.3$); with a mean of 4.7 mph. This alters the initial doses due to the passing cloud, groundshine, and intake due to inhalation to people living in the evacuation zone. In the health effects model evacuation is accounted for by truncating the groundshine doses for people within 25 miles of the nuclear power plant after 4 hours and truncating the groundshine doses of people within 25 miles after 24 hours for those immediately relocated and after 7 days for those relocated later. The early cloudshine and inhalation doses of the evacuees are based on the total time they are immersed in the cloud. This is dependent upon the specific evacuation model in use.

In the RSS, interdiction refers to long-term (several years) movement of people to limit doses due to groundshine and inhalation of resuspended materials. The area over which relocation occurs is determined by comparing the estimated groundshine dose to the allowable groundshine dose. The interdiction criteria for land used in the RSS HEM were 10 rem in 30 years for rural areas and 25 rem in 30 years for urban areas. In areas where the estimated dose exceeds the allowable dose, the following actions are contemplated:

1. decontamination of contaminated ground, pavement, and roofs (note the RSS assumes that ground concentrations may be reduced on the average by a factor of twenty in this way),
2. relocation
3. interdiction (restricting the use) of:
 - a. crops
 - b. milk
 - c. land

Graphically, the zones where these would occur immediately after the accident are shown in Figures 2-17 and 2-18 (RSS Figures VI 11-1 and VI 11-6).

Figure 2-17. Simplified interdiction model.

(RSS Figure VI 11-1)

Figure 2-18. Illustrative interdiction model for ground level release.

(RSS Figure VI 11-6)

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SECTION 3

DOSIMETRY

K.R. Kase

3.1 RELATIONSHIP OF DOSIMETRY TO HEALTH EFFECTS

The Reactor Safety Study health effects model described in Section 2 is designed to produce a series of estimates of health effects resulting from accidents in nuclear power plants. A primary influence in the analysis is the radiation dose received by populations and individuals exposed to the radioactive materials released during the accident. Consequently, a review of the dosimetric models and assumptions is an integral part of any review of the health effects model.

The acute radiation effects described in the Reactor Safety Study (RSS) are generally characterized by sigmoidal dose-response relationships that indicate few, if any, effects below certain radiation doses. For each, there is a fairly narrow dose range in which the effect increases rapidly to its maximum expression. As a result, estimates of acute radiation doses to a population will influence greatly the projected effects, and small uncertainties in calculated dose may result in large uncertainties in predicted effects, especially for individuals.

Late radiation effects, such as cancer induction, will be influenced by acute dose as well as chronic dose, and the influence of uncertainties in acute dose estimates may be as great for late effects as for acute effects. Thus, realistic and accurate estimates of acute radiation dose are important also for late effects. The effects of uncertainties in the estimates of chronic radiation dose to populations are less clear, because of uncertainties in the relationship of late effects to protracted low-dose-rate radiation. However, it is clear that small uncertainties in dose rate can lead to large uncertainties in total dose over a long period of time and any effect which is dependent on total dose received will certainly be influenced by these factors.

In summary, if the goal is to achieve realistic estimates of health effects, realistic dose estimates must be made. Any review of the health effects model must ascertain that realistic radiation dose estimates have been made and applied correctly in the estimates of health effects.

3.2. RELEVANT SECTIONS OF RSS APPENDIX VI

The assessment of dosimetry related to the health effects model involved a thorough review of the Dosimetric Models (RSS Section 8) and related RSS Appendices C and D on external and internal dosimetry, respectively. These portions of the RSS describe the models used to calculate the radiation dose parameters and models which are subsequently used in various accident situations to determine radiation doses from which health effects estimates are made. In addition, the health effects model (RSS Section 9) and "Calculated Results with Consequence Model" (Section 13) were reviewed together with Appendices E, "Chronic Exposure Model," and F, "Early and Continuing Somatic Effects." From these portions of the report and selected references cited in these sections, some understanding of the dosimetric model and its application

was gained. Presented below is an assessment of the adequacy of the dosimetric model used to estimate dose to individuals from both external and internal radiation sources, the uncertainty of the dose parameters, and the application of these models to the estimates of health effects.

3.3. ADEQUACY OF THE DOSE-EFFECT MODEL

To relate the dosimetric model to subsequent health effects, a large number of parameters must be specified, evaluated and factored into the dose assessment calculations. These parameters are tabulated below.

Dose Calculation Parameters:

1. Relative amounts of each radionuclide released.
2. Release rate.
3. Air concentration (meteorology).
4. Time dependence of air concentration (dependent on release rate, deposition rate, meteorology).
5. Deposition rates of radionuclides (function of particle size and precipitation).
6. Radioactive decay schemes for each radionuclide considered.
7. Location and movement of people.

External Dose

8. Dose rate and airborne concentration for each nuclide for semi-infinite cloud.
9. Dose rate and surface concentration for each nuclide for infinite plane.
10. Shielding by buildings and surface irregularities.
11. Weathering rate (rate of disappearance of radionuclide from ground surface, or increased ground shielding with time).

Internal Dose

12. Breathing rate.
13. Particulate deposition in various portions of the pulmonary system, i.e. lung model (requiring assumptions about the particle size distribution).
14. Transfer of radionuclides from pulmonary system to other organs, retention and biological half-life (including transfer to and uptake from GI tract).
15. Transfer from the ground through the food chain to humans (including rates of intake from various sources of food).
16. Transfer of radionuclides from GI tract to other organs (i.e., GI model); retention and biological half-lives.
17. Resuspension of ground-deposited radionuclides.
18. Internal dosimetry model, i.e. specific organ doses resulting from deposition of a given radionuclide in a given organ (dose rate to organ A and concentration in organ B).

The relative dose contributions for each of the radionuclides released in an accident can then be calculated over the time period of interest. Total doses would then be determined by factoring in the total activity released, which will depend on accident type. Health consequences could be determined based on the dose-related Health Effects Model.

3.3.1. External Dose Model

The dosimetric model can be subdivided into dose from external radiation and dose from internally deposited radionuclides. The external dosimetry model is described in Appendix VI-C of the RSS and includes exposure to radionuclides deposited on the ground as well as exposure to a passing radioactive cloud. The description of the model for external dose calculations is very incomplete, inconsistent and in some instances inaccurate. It appears, however, that the dose conversion factors given in both Tables VI C-1 and VI C-2 are correct in spite of the fact that equations VI C-1 and VI C-2 are not correct. These equations are taken directly from Trubey and Kaye (1973), but their development is not explained in that report.

A check of Table VI C-2 for a few radionuclides (Co-60, Cs-137, Te-132, I-131, I-132) shows that the values tabulated are consistent with the model given in equation VI C-1 only if the constant in that equation is 1.066 and not 827 as indicated. Also, two interaction coefficients should be applied. The σ_{in} in the argument of E should be the attenuation coefficient for air while the σ_{in} used elsewhere should be the mass energy absorption coefficient for tissue.

A check of Table VI C-1 for Co-60, Cs-137, and I-131 shows that the values tabulated are consistent with the model:

$$D = 1.066 C_i \sum_{n=1}^{N_i} (R_{in} \sigma_{in} f_{in} E_{in}) \quad (3-1)$$

with C_i in Ci/cm^3 and $R_{in} = 5/\sigma_{in}^{\text{air}}$ (5 times the mean free path of the gamma photon in air). It is obvious that equation VI C-2 is incomplete. In the sum, σ_{in} is again the mass energy absorption coefficient for tissue.

Once the accuracy of Tables VI C-1 and VI C-2 was verified, a number of radionuclides were used to test the relative external dose contribution from a release such as is shown in RSS Figure 13-1. Scaling from the figure is rather difficult, but the relative positions and contributions of the various radionuclides appear to be correct within a factor of two. The validity of the dose contributions displayed in figures such as Figure 13-1 depends on the validity of the assumptions concerning core inventories and fraction of the inventory released in various accident situations. Table VI 8-1 lists core inventories for radionuclides of concern used for the RSS calculations. The source of this table is unclear and there are some significant differences between relative values given for various nuclides here and those derived, for example, from *Meteorology and Atomic Energy*, Table 7-1 (Slade, 1968).

3.3.2 Internal Dose Model

A few general comments need to be made about the internal dose model. Corrections were made for some radionuclides for dose versus age at exposure (Section 8.4.3 of RSS). These should be checked.

A second element which requires reevaluation relates to the calculation of dose to the thyroid for assessing the incidence of thyroid nodules and cancers. The evidence that dose from internally deposited I-131 is only 0.1 or less effective than dose from external radiation has been called into question (Lee et al., 1979). This question must be thoroughly reviewed.

The dosimetric model for internally deposited radionuclides is subdivided into exposure by inhalation and exposure by ingestion. Detailed checks were made by us only for the inhalation portion of the internal dose. Table VI D-2 was compared with a calculation of dose to lung (0 to 50 yr.) from radionuclides deposited in lung for 10 different radionuclides. The following model was used to determine the dose from beta particles emitted by radionuclides within the lung.

$$D(\text{rad} / \text{Ci inhaled}) = K T_{eff} \sum_{i=1}^N f_i E_{i\beta} \quad (3-2)$$

where

T_{eff} is the effective mean life in days for the radionuclide in lung;

$\sum_{i=1}^N f_i E_{i\beta}$ is the sum of the abundance of the i th beta particle multiplied by its mean energy in MeV;

$$K = \{2.13(\text{g rad} / \mu\text{Ci hour MeV}) 1. \times 10^6 (\mu\text{Ci} / \text{Ci}) 24(\text{hour} / \text{day}) 0.25\} / 950(\text{g});$$

$$K = 1.35 \times 10^4 (\text{rad} / \text{day Ci MeV}).$$

Our assumption of 0.25 as the fraction of activity inhaled which deposits in the lung is consistent with a $1 \mu\text{m}$ particle size. In addition, some long-lived radionuclides will move to other organs and contribute significant dose to the lung from their gamma radiation. For the 10 radionuclides we considered, dose to the lung from material deposited in other organs was calculated from the absorbed dose per cumulated activity data in MIRD Report No. 11 and information on deposition and biological half life from ICRP Committee II (1960). In general the agreement with values given in Table VI D-2 was quite good except for Cs-134, where the table is high by a factor of 2.5. Since the publication of the RSS, updated metabolic and internal dosimetry data have been published in ICRP Publication 30, and the NCRP has published a report on cesium.

A further test was made comparing RSS Table VI D-2 with values derived from Table 7 in a recent Battelle report PNL 3257 (1980). If one assumes a breathing rate of $2.66 \times 10^{-4} \text{ m}^3/\text{s}$ and an exposure time of 1/2 hour, differences between these two tables are generally less than a factor of 2 for the same 10 radionuclides, suggesting no major calculation errors were made.

As with the external dose model, a comparison was made with Fig. VI 13-2 on the basis of the relative dose contribution from inhaled radionuclides. In general the relative importance of the various radionuclides appears to be accurately represented by that figure.

The dose from inhaled radioactive material depends on the chemical form and particle size distribution of the radioactive aerosol. In Appendix VI-D the

general dose equation used to derive the values for Table VI D-2 is not presented and some questions concerning tabulated values arise because of this. For example: Sr-89 is a D-category nuclide for lung deposition and so has an effective half-life in the lung of 0.5 day. It emits only a beta particle and thus will not contribute dose to the lung once it has left the lung. Yet the table indicates that 12% of the total lung dose is delivered at times exceeding 7 days (14 half-lives).

Detailed calculations of dose from ingested radionuclides were not made for comparison with Table VI 8-4. However, several problems with the internal dosimetry model relative to ingestion did arise. It is questionable whether the dose to the gastro-intestinal (GI) tract has been calculated to the appropriate tissue in the appropriate manner. The dose calculations were simplified by applying the highest effective beta particle energy in the decay chain to all particles. This will tend to overestimate the beta radiation dose at any given depth in tissue. Since a large fraction of the total dose to critical intestinal cells results from the beta radiation, the above simplification may lead to large overestimation of dose to these cells. Also, the choice of the lower large intestine (LLI) as the critical portion of the GI tract should be re-evaluated. It is generally agreed that the residence time of material is much longer in the LLI than in the small intestine (SI) (about 4 times longer), so that if the dose rate to the critical cells is the same for both organs, the LLI would receive a much larger total dose. However, the separation between material in the intestinal lumen and the mitosing cells is about 140 μm in the SI and about 420 μm in the LLI (Eve, 1966). The dose rates in Table VI 8-3 are calculated to a depth of 500 μm . Depending on the beta particle energy, the dose rate at 100 μm depth can be of the order of 5 times the dose rate at 500 μm (Unnikrishnan et al., 1973). Thus, for some radionuclides the total dose to mitosing cells of the SI may be as great, or greater than the dose to mitosing cells of the LLI in spite of the longer residence time of material in the LLI.

Parts of the discussion of the ingestion model are incomplete, so it is not clear that the same assumptions were made for the calculation of dose to the LLI in both the inhalation and ingestion models. Iodine-129 and some of the long-lived transuranic radionuclides might be important for consideration also in the chronic ingestion situation, although representing small fractions of the core inventory. Screening for radionuclides important to chronic ingestion apparently involved consideration of metabolic characteristics of humans and other animals but not uptake by plant material.

Finally, equation VI E-21 includes a term, C, defined as the first day's intake of radioiodine by a cow, but also terms R and L which account for radioactive decay and loss from pasture grass in the time between deposition and start of milk production. On page E-22, C is defined as the intake assuming no decay or weathering, but R and L are defined as the decay and weathering losses from the start of ingestion, not the time of deposition. There seems to be some inconsistency in the definitions and use of the terms C, R and L unless it is assumed that no decay or weathering occurs between deposition and start of ingestion by the cow. In addition, it seems excessively conservative to assume that cows continuously graze on the contaminated pasture when calculating intakes of long-lived radionuclides such as Sr-90 and Cs-137.

3.4. UNCERTAINTY IN DOSE PARAMETERS

Uncertainties in the assumptions concerning the various parameters which enter into the dose calculations will, of course, influence the uncertainty of the subsequent dose assessment and ultimately affect the estimated health effects consequences. Some of the most important variables are:

1. Breathing rate;
2. Particle size distribution of airborne radionuclides;
3. Uptake and retention of radionuclides;
4. The time over which people are exposed;
5. Shielding factors;
6. Resuspension of deposited radionuclides;
7. Deposition rates (including influence of precipitation);
8. Behavior of radionuclides in the food chain.

Breathing rate will have an important effect on the quantity of radionuclide inhaled, but it also can affect the amount of material deposited in various portions of the respiratory tract. In this regard, particle size will also be important. The dosimetric model seems to consider an age-dependent inhalation rate ranging from 0.5 L/min for newborns to 16 L/min for adults. These values agree quite well with those in a recent article by Kaul and Roedler (1980) which gives age-dependent breathing rates ranging over a factor of 5 from 3 L/min for infants to 15 L/min for adolescents. However, the Task Group on Lung Dynamics (1966) gives values of 11.75 L/min and 21.75 L/min for moderate and moderately active activity. Persons in an agitated state could be breathing at an elevated rate which may result in intakes 30%-50% greater than those estimated from average inhalation rates, unless these higher rates have offsetting decreases in lung deposition.

Particle size distribution together with breathing rate can affect the deposition of material in the various portions of the respiratory tract. Deposition in the lung itself does not appear to be dependent on breathing rate for particle sizes ranging from 0.1 to 1.0 μm , but it can vary by a factor of two for larger particles and a factor of 50% for smaller particles (Morrow et al., 1966). Particle size affects the percentage deposition in the lung, but the dosimetric model seems to assume that airborne radionuclide particles will fall in the size range of 0.1 to 1.0 μm where deposition is reasonably independent of particle size and breathing rate.

Radionuclide uptake and retention will be greatly variable depending on dietary habits and age, among other things. The analyses used in the dosimetric model are reasonable, but should be reevaluated based on recent information. The effects on dose of the possible variations in these factors should be discussed. The model uses an intake of 0.7 L/day for milk in the analysis of iodine uptake and states that it is high by a factor of two for the population average. Kaul and Roedler (1980) give milk intake values of 0.5 L/day for 2 month-old infants and adults, and 0.8 L/day for ages in between. This would indicate that the 0.7 L/day value is not high by a factor of two. In addition, Kaul and Roedler indicated that fractional uptake of iodine from blood to thyroid can vary between 10% and 80%, depending on dietary iodine intake, and normally ranges between 30% and 60%. These variations can lead to significant uncertainties in individual doses.

The effect of exposure time on dose does not require much discussion.

Perhaps the area in which the largest uncertainties occur is in the calculation of doses from internally deposited radionuclides. In that case dose will be dependent on not only the time over which the intake occurs, but also on the biological half-life of the material. Again using the example of radioiodine, we note that the health effects model apparently assumes that intake occurs continuously from the time of deposition until the iodine has decayed away. The variation in dose should be considered for exposures over a more limited time.

The dosimetric model assumes shielding factors for ground-deposited radionuclides ranging from 0.02 for people in large buildings to 0.7 for people standing outside on the ground. Based on an article by Burson and Profio (1977), we find these appear to be reasonable assumptions and the resulting dose variations are taken into account in the model. Similarly, shielding of the dose from airborne radionuclides seems to have been adequately accounted for, if only the dwelling is between those inside and the radioactive material.

Resuspension of deposited material is very dependent on surface and atmospheric conditions. Resuspension factors have been shown to vary over eight orders of magnitude. The dosimetric model uses a factor of 10^{-5} decreasing with time according to a reasonable function. Lassey (1980) argues that a factor of 10^{-4} is more reasonable for the initial value of resuspension factor. The exact effect on the ultimate calculated dose to an individual caused by this large uncertainty is not clear.

The distribution of deposited radionuclides in the soil will affect both the shielding effect of the soils as well as potential resuspension. Appendix VI E deals with this problem in a way which raises questions concerning the uncertainties of the distributions and the attenuation effect of the soil. An exponential distribution is assumed with a relaxation depth of 3 cm, but this does not appear to hold for Sr-90, and, therefore, may not apply to certain other radionuclides as well. It also would not apply in disturbed soil.

The attenuation of photons is determined based on a model described by Beck (1966). It is not immediately clear how the effect of the soil on attenuation is handled, and this should be studied in more detail.

Deposition rates of airborne particles are also affected by atmospheric conditions and by precipitation, as well as by particle size and chemistry. The assumption of a deposition velocity of 1 cm/s in the model is not explained. The effects of potential variations in deposition rates should be addressed.

The passage of radionuclides through the food chain from soil to people is affected by many variables. There are large variations in behavior of radionuclides in soil and vegetation depending on land use, soil type and meteorology. Data accumulated since 1975 on radionuclide behavior in soil and vegetation are likely to be sparse because of the continuing decrease in radionuclide fallout from atmospheric weapons tests. Consequently, it may be difficult to make major improvements in the models and reduce the uncertainties in dose resulting from variability in the food chain. There are also uncertainties introduced because of variations in intake of various foods, depending on the age and diet of individuals. It would be useful to have a more complete discussion of the effect that variations in the food chain might have in the overall dose assessment.

3.5. APPLICATION OF DOSE ASSESSMENT TO ESTIMATES OF HEALTH EFFECTS

The desire to simplify the dosimetry where possible has led to the possibility that the radiation doses used to assess certain health effects were not appropriate. The doses to the total bone marrow, testes and lung were each calculated for exposure to both external radiation and radiation from internally deposited radionuclides. However, it is not clear that the doses to other organs were dealt with adequately. Appendix C, "External Dosimetry," implies in Tables VI C1 and C2 that specific organ doses for external radiation were determined only for total marrow, lung, and testes and that all other organ doses were equated to the whole body dose. Appendix D, "Internal Dosimetry," states (p. D-2) that specific organ doses for internally deposited radionuclides were determined for lungs, bone marrow, bone, GI tract, thyroid, ovaries and testes. In Section 13, "Calculated Results with Consequence Model," graphs are shown which give the dose to bone marrow, GI tract, lung and thyroid (RSS Figures VI 13-1 through 13-6) and dose to bone marrow, lung, bone, breast and testes (RSS Figures VI 13-8 through 13-17). Dose to the breast from external radiation is likely to be significantly different from dose to the GI tract, especially when considering relatively low energy gamma photons. For estimates of cancer induction in specific organs, a more accurate estimate of dose to those organs should be made. Similarly, dose to the fetus should be calculated rather than using the dose to the GI tract when assessing effects on the developing fetus.

Once the doses were determined, the best dose-response information available at the time was used to assess both the acute and long-term effects of the radiation exposures. Looking to the future, recent reports of acute radiation effects must be carefully evaluated for their applicability to the reactor accident situation. Long-term effects can be estimated using the recent report of the BEIR III Committee, but close attention must be paid to any reassessments required as a result of new dose calculations for the Japanese bomb exposures.

3.6. SUMMARY

The dosimetric model applied in the Reactor Safety Study was carefully developed and took into consideration the complexities of environmental transport and metabolism of radionuclides. Some simplifications were made and details of the model were sparse in the final report. In the years since the publication of the RSS, some new, or more up-to-date, information has become available. These factors lead to a number of issues which should be addressed when reassessing the radiation dosimetry relative to the Health Effects Model. These issues fall into two categories: (a) assumptions and development of the model and (b) uncertainties in dose calculations caused by environmental and biological variations. These issues have been discussed in this section and are summarized here.

Assumptions and development of the model:

1. Development of the external dose model must be improved and clarified.
2. The inhalation dose model must be justified with regard to assumptions about aerosol particle size and chemical nature.
3. The ingestion dose model requires a more complete discussion, particularly with respect to the dose calculations for the GI tract.

4. Assumptions made when calculating dose to the GI tract must be reassessed.
5. The dependence of organ dose on age at exposure deserves reevaluation.
6. A reevaluation is needed of the model for the pasture-cow-milk pathway.
7. Doses should be calculated specifically for appropriate organs such as active marrow and breast when evaluating specific organ responses.
8. Exposure/dose/effect relations for internally deposited I-131 require a thorough review.
9. Assumptions about core inventory, release fraction and radionuclides considered important for radiation exposures require a thorough review.
10. Uncertainties in dose calculations require a more detailed evaluation with regard to the effects of:
 - a. Breathing rate.
 - b. Resuspension factor.
 - c. Radionuclide distribution in soil.
 - d. Milk intake.
 - e. Iodine retention.

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3.8. APPENDIX; SUPPLEMENTARY DOSIMETRY REFERENCES

This is a list of references, primarily from *Health Physics*, which should be considered for the revision of the Dosimetry Model used to assess health effects in the Reactor Safety Study. These references cover the years 1975 through 1981 and they are listed chronologically in six groups:

- I. General dose calculations.
- II. General environmental pathways.
- III. Dose calculations for external sources.
- IV. Dose calculations for internal sources.
- V. Environmental pathways for internal sources.
- VI. Uptake and retention for internal sources.

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SECTION 4

EARLY HEALTH EFFECTS

C.J. Maletskos

4.1 INTRODUCTION

One of the difficulties in conducting a new evaluation of the RSS early health effects models is the paucity of additional information since the publication of the study in 1975. The kinds of questions that remain were partially addressed in the report, and some have begun to be addressed since publication.

Our literature search included a computer search using key words such as "biological effects," "health effects," "acute," "early," "human," "mortality," "morbidity," "whole body," "mean lethal dose," "thyroid," "intestinal tract," "lung," "bone marrow," "prenatal growth," "prenatal development," "sterility," "cataracts," "azoospermia," "amenorrhea," "internal," "external," " ≥ 1975 ," but produced only a few references, most of which are discussed below. The BEIR III and UNSCEAR reports (NAS, 1980; UNSCEAR, 1977) were also reviewed. However, neither of these reports addressed the acute effects of radiation, except from the standpoint of prenatal development discussed in a later subsection.

The RSS made a remarkably good first evaluation of the probability of early mortality and continuing illnesses from potential accidents in nuclear power plants. Even with the limited new information, there is opportunity, however, to make the analysis more rigorous, and to consider a few more aspects in greater detail.

This review follows the order, more or less, of RSS Section 9 ("Health Effects"), Appendix F ("Early and Continuing Effects") and Appendix H ("Thyroid Effects").

4.2 GENERAL COMMENTS

The comments discussed in this section are not related to the dose-response relationships as such, but they are so directly involved in the health effects calculations that they are brought up here because they apply to all the effects.

The first comment concerns the population distribution in the U.S. The RSS used the 1970 data of the U.S. Census Bureau. The results of the 1980 census are available now and should be used.

In the mitigation of the consequences of an accident, evacuation of the appropriate population is an important method. The RSS uses a reasonable evacuation model, except that the population is assumed to move outward radially until the plume overtakes the people and then they are moved out of the plume in a perpendicular direction, i.e., circumferentially. It would seem that the better strategy would be to have the population move away not only radially but also from the sector of the plume right from the start of evacuation. Evacuation tactics have been improved since the RSS, partly due to the use of computer analyses and simulations for developing more efficient movement of people by

accounting for actual existing road networks, stop lights, traffic jams, etc.. Improved evacuation methods could result in lower radiation doses than the model predicts.

On the other hand, projected dose level criteria used in the RSS to determine the areas of relocation and interdiction are different from the current Protective Action Guides (PAG's). If evacuation were based on the current PAG's, this would change the number of the persons exposed and the magnitude of their exposure. The PAG's are not necessarily the levels above which evacuation would be recommended, however.

In the process of evacuation, a small group of people were assumed to remain behind and were classified as a non-participating minority. Such people voluntarily choose not to evacuate. The RSS estimated the size of the group at 5%. There is another group of people which might not be evacuated: the sick and infirm, those that take care of them, and those that perform important functions during an emergency. Thus, the size of the nonparticipating minority needs to be reviewed in further detail and in light of current emergency planning practices.

Finally, any dose-response relations for early effects based in part on the Japanese survivor studies may change somewhat as a result of the new dose calculations made at Lawrence Livermore National Laboratory (Loewe and Mendelsohn, 1981) as well as at the Los Alamos National Laboratory and the Oak Ridge National Laboratory. The changes in the dose-response curves for acute effects that these new calculations might make, however, are not expected to alter the curves significantly.

4.3 COMMENTS ON HEALTH EFFECTS

4.3.1 Bone Marrow

The dose-response relationship for mortality under minimal treatment reflects quite well the information available to the RSS for a young-adult healthy population. Newer information on a few recent radiation accidents occurring since the study, described in the 1979 REAC/TS International Conference (Hubner et al., 1980) is still consistent with the selected dose-response relationship.

A peculiarity of these dose-response curves is that there is a break or "knee" in them at the 10% mortality value for the minimal and supportive treatment curves and at the 20% value for the heroic treatment curve. The unusual shapes in these curves are even used in the computer code. (See Section 6.) A review of the shapes would be useful.

The study assumes a uniform young-adult population, but it would be useful to apply some sensitivity-specific population distribution (including intrinsic sensitivity, age effects and state of health but excluding prenatal effects that are discussed separately). There are problems of estimating such a distribution and of applying an appropriate dose-response curve to it.

In an effort to represent a truly heterogeneous population, Lushbaugh et al. (1981) proposed a dose-response curve that is parallel to the RSS curve with

minimal treatment (without the break in the curve) and that has an $LD_{50/60}$ of about 250 rads. Such a curve may represent an oversimplified and conservative approach to the needs of the RSS, but some attempt to follow the suggestion that one use the sensitivity-specific population distribution, discussed above, would be more realistic than the current model.

The report of Lushbaugh et al. (1981) also suggests a very strong dose-rate effect that is applicable to external whole-body exposure during the first one or two days following the accident. Such a dose-rate effect would serve to increase the effective $LD_{50/60}$ at doses that are still high but protracted over a period of a day or two instead of a few hours. This is a significant effect that would lead to decreased mortality and deserves further study. The recommendations in the "Penalty Table" of NCRP Report No. 42 (NCRP, 1974) imply a similar dose-rate effect, but, not as much as that suggested by Lushbaugh et al.

Another consideration relates to the use of the different dose-response curves associated with the medical treatment to be administered: the ability to treat may not be realistic. Heroic treatment requires appropriate hospital centers and bone marrow transplantation from well-matched donors. Depending on the number of persons involved, the hospital centers may or may not be available at the time required. However, centers practicing whole-body irradiation therapy have had difficulty locating suitable transport donors. Thus, heroic treatment may not be a viable option. This dose-response curve was not used, and typically is not used, in calculations predicting consequences of nuclear reactor accidents, however.

In the case of supportive treatment, the RSS suggests that 2500-5000 persons can be transported to the appropriate hospitals in the United States (mainly teaching hospitals). In the confusion of a large accident, identification of those needing supportive treatment is possible but may not be easy and the number of those needing help who can really be helped may not be so large. A more realistic estimate would be to apply some sliding scale to supportive treatment whereby a few seriously exposed persons can all be given supportive treatment, but as the number of persons increases, the fraction of persons who can be given supportive treatment decreases in some realistic manner.

As an alternative or further means of providing supportive treatment, the usefulness of sending military field hospitals to the region of the accident should be considered. Field hospitals might be more effective for treating people "on site" rather than transporting them to hospitals throughout the country, but this would depend in part on how long it took to mobilize such field hospitals. For purposes of reducing exposures from inhalation and ingestion, a number of decorporation methods have been used for reducing the amount of radioactive material within the body. Current practice is summarized in the recent NCRP Report No. 65 (NCRP, 1980). Also see Section 4.4.3 for more current information on decorporation.

4.3.2 Lung

We found no new data on lung mortality. New information that was found relates to lung morbidity and is discussed under that heading.

Although all the information described in the subsection on lung morbidity comes from the exposure of sick subjects rather than healthy ones, the sensitivity of the human lung may be high enough that a pulmonary effect could be a contributing synergistic factor to acute whole-body bone marrow mortality. (See also the discussion on synergism in Section 4.4.1.)

The RSS deemphasizes acute pulmonary mortality since acute bone marrow mortality would almost always be overriding given its assumptions about the particle size distribution and solubility of the radioactive aerosol. The RSS dose-response curve for lung mortality uses the results from the Lovelace Inhalation Toxicology Research Institute (ITRI) that were obtained from experiments with dogs inhaling Y-90 and Y-91. Dose-response curves were developed for each of those exposures and an intermediate curve was chosen. (The slopes of the experimental curves were identical.) However, these dog experiments also included exposures to Sr-90 and Ce-144, data not used by the RSS. In addition, the Battelle Pacific Northwest Laboratory (PNL) has performed inhalation studies with animals, including baboons, primarily with the alpha-emitting plutonium nuclides, which studies also were not used, perhaps because such nuclides are not expected in significant amounts in the releases postulated.

Both laboratories joined forces to produce a common data base with all their combined results, and each laboratory proceeded to develop models and analyze the results separately. The aim of this work was to produce better estimates of acute effects, especially from inhalation of radionuclides, and to determine what new experiments could provide information that is necessary but not available. The results of those efforts are available as Phase I studies by the two laboratories: Hahn (1979) from ITRI and Filipy et al. (1980) from PNL.

The model in Hahn's report involves a hazard function method that leads to a Weibull distribution of response versus dose. Total lung dose over 18 months is used for each of the effective half-lives of the four radionuclides. The model in Filipy's report uses a linear combination of log-log relationships between survival time and dose to the lung over one year from the different exposures. Both models use all the animal data now available for all inhalation exposures. Both reports compare their results to the RSS study and to other models also recently developed, but, except for the RSS, the models chosen for comparison, surprisingly, are different. Thus, there are now about one-half dozen newly-developed models since the RSS. All the models lead to somewhat different answers, and a very careful and detailed study of these results will be necessary.

4.3.3 Gastrointestinal Tract

New information on the early effects on the gastrointestinal tract is quite limited. The results from radiation therapy on human beings continue to show RSS overstatement of risk. In a summary paper, Novak et al. (1979) indicate that the small bowel will escape clinically significant injury if whole abdominal radiation is kept below a fractionated dose of 2100 rads. Some indications from Maruyama et al. (1975) and Potish (1980) suggest that certain classes of persons are predisposed or more susceptible to intestinal radiation injury; i.e., persons with small stature, hypertension, diabetes. Such information, as qualitative as it is, would serve to change the slope of the dose-response relationship or lower the threshold or both.

Recent experiments of Sullivan et al. (1978) using Ru-106 - Rh-106 and Pm-147 indicate that the LD₅₀ of crypt cells is about 3500 rads in dogs and rats, the same value assumed in the RSS. These results suggest that the RSS dose-response curve is realistic.

4.3.4 Radiation Anxiety

The accident at TMI showed a strong psychological response by the public to a radiation accident. While the radiation exposures were low enough as to practically rule out any biological effects, the psychological response was large. Such a response is in contrast to the public's attitude to releases of toxic chemicals, where evacuations have been made quite calmly by comparison.

Severe anxiety responses of this type may be confused with early radiation illness (non-fatal). The number of people needing some attention would increase significantly and could lead to a decrease in the medical assistance to those affected physiologically by the radiation exposure. Thus, "radiation anxiety" could lead both to greater mortality because of confusion regarding who should be given appropriate medical support and to more numerous early illnesses, psychogenic though they might be.

Some effort could usefully be made to estimate these effects using the TMI experience, the experience of other applicable accidents or incidents of war, as well as expertise in psychology. Apparently, sociologists, psychologists and psychiatrists are sharply divided on the possible mental health effects of nuclear disasters (Kreps, 1981), and the issue may not be easily resolvable. Nevertheless, it would be advisable to consider some scenarios to see how this effect, at the least, might influence the treatment of those that have actually been exposed to significant amounts of radiation. A good quantitative analysis of this phenomenon is unlikely, but some estimate of the possible impact could be useful.

4.3.5 Morbidity Effects

The scarcity of new references on morbidity precludes our making many comments. Cataract, thyroid and lung effects are discussed briefly and a general comment is made.

In regard to the lens of the eye, a report by Sonneveld et al. (1979) indicates that, after whole body irradiation of 850-900 rads, cataracts form in monkeys at an incidence of 17% in 3 years and 100% in 10-15 years, while at 400 rads, no cataracts are formed. This result is consistent with the RSS evaluation of cataract production.

The preparation of NCRP Report 55 (NCRP, 1977) on the protection of the thyroid from releases of radioiodine continued the analyses of the dose-response relationships (further amplified by Maxon et al., 1977) that were initiated for the RSS and uncovered no significant new information. The RSS study pointed out that the dose-response for hypothyroidism is based primarily on patients receiving I-131 doses greater than 2500 rem and that more work is necessary to clarify the relationship at lower doses. No new information has been found since 1977 to modify the RSS conclusions. A recent report by Book (1980) indicates that short-lived I-132 is about 10 times more effective, rad for rad, in suppressing rat

thyroid weight increase than is the longer-lived I-131. Book also finds some age radiosensitivity of guinea pig thyroids, with fetal and weanling thyroids (after 100 days of exposure to I-131) more sensitive and neonatal thyroids less sensitive than adult thyroids, but each by a factor of less than 2.

Five new items of information on exposure of the human lung have been found. The evidence of Prato et al. (1976) suggests that pneumonitis can be observed at an incidence of a few percent following a single acute exposure of approximately 800 rads. This result is in essential agreement with that of Wara et al. (1973) (a reference of the RSS) who concluded that an incidence of 5% occurs at 770 rets (a protracted dose converted to a nominal single dose and expressed as rads equivalent therapeutic, "ret"). The second item (Braun et al., 1975) describes the death from pneumonitis of one person at an exposure of 750 rets although this person's lungs were presumably sensitized from previous long-term actinomycin-D therapy. Fukushima et al. (1977) observed that all 23 of their patients treated for bronchial carcinoma developed radiation pneumonitis at an average of ~ 3 months following a protracted dose of ≥ 4500 rad. (This information is taken from a translated abstract; details not available.) Tefft (1977) in a review of the results of the National Wilms' Tumor Study indicates that 7 of 24 subjects showed morbidity (pulmonary toxicity or pneumonitis) with doses starting at 800-700 rets.

The fifth item, a recent report by Van Dyk et al. (1981), provides the most definitive dose-response curve for pneumonitis. The results are based on a retrospective calculation of lung dose to patients who had single exposure half-body irradiation and who had no previous lung disease and no known lung tumors. Lung dose was determined from the dose to the patient by a dose-correction factor obtained on other patients using a treatment planning system assisted by computed tomography. The final results indicate a sigmoid curve with an incidence of pneumonitis of 5, 50 and 95% at lung doses of 820, 930 and 1060 rads, respectively. The threshold value is about 750 rads (which might be as low as about 700 rads) and 100% incidence occurs at about 1150 rads. Revision of the model of lung morbidity should incorporate these new results, which are different from the RSS dose-response relationship.

The RSS defines early morbidities as those requiring medical attention and hospital treatment. Respiratory impairment and hypothyroidism are included in this category. Bone marrow morbidity also falls in this category, but it is not even mentioned in the RSS. It would be better to make an estimate of more nearly all early illnesses, separating them into two groups: serious (bone marrow, lung and thyroid) and less serious. In this way, a more realistic estimate can be made of the number of persons actually affected by the radiation and for whom medical attention will be needed. The onset of symptoms will vary with the effect and with the individual, and might extend over a period of several months after the accident. A realistic estimate could be useful for logistical purposes, indicating how many might be sick after an accident.

4.3.6 Prenatal Development

Only a limited amount of good quantitative information on the effects of radiation on the developing human embryo and fetus was available to the RSS. Since then, the BEIR III and UNSCEAR reports (NAS, 1980; UNSCEAR, 1977) and a report by Hoffman et al. (1981) from the Bureau of Radiological Health have

reviewed this subject in detail, with each report containing about a dozen new references dated 1975 and later, many of them the same. The dates of most of these references are 1975 and 1976, suggesting that, with normal publication delays, some of the information was available to the RSS. These reports make no attempt to recommend any dose-response relationships, but the reports do indicate that the threshold for some effects might be as low as 5 rads. The UNSCEAR report indicates that the best documented malformation is microcephaly with an incidence of about 10^{-3} /rad for an acute dose in the range of 10 to 150 rads. Data are becoming available from recent studies on radiation's other impacts on fetal mental development.

The new references should be reviewed in detail to determine whether the RSS estimate might be modified, especially in the light of the new 1980 census, the possible lower threshold, and the new dose calculations for the Japanese survivor studies.

The RSS makes some estimates of prenatal developmental effects. The results are not used in the consequence calculations in RSS Section 13.

4.4 ADDITIONAL CONSIDERATIONS

4.4.1 Interactions or Synergisms From Doses to More Than One Organ

The RSS combines early health effects by assuming that each effect occurs independently. Possible interaction (i.e., cooperation) between effects is omitted from the calculations. A few experiments on synergism are discussed in the RSS, but it is stated that these results cannot be used in any quantitative synergistic application.

Interactions are discussed in more detail in the reports of Hahn (1979) and Filipy et al. (1980), especially from the standpoint of what experiments are needed to determine the importance of various possible synergistic combinations. These interactions include, for example, combined high and low dose rates, combined external and internal radiation, combined internal radiation of several organs and tissues, combined alpha and beta radiation, etc. An example of the combination of high- and low-LET radiation at the cellular level (hamsters) is described in the report by Ngo et al. (1981); neon ions and x-rays each produced damage that interacted with the damage of the other irrespective of which radiation was administered first. Until the new experiments at the ITRI and PNL laboratories are performed, it is not likely that data on synergism can be used to modify the incidences of early effects.

Another approach to multiple organ effects (late effects) is taken in ICRP Publication 26 (ICRP, 1977), which recommends the use of weighting factors for individual organs in order to determine the combined contributions to the effective dose equivalent for an exposed person, for stochastic (latent) effects. An analogous approach may be applicable to estimating early effects. It may be possible to use some kind of weighting factors for combining exposures as an alternative to awaiting the results of the experiments on synergism, which may not be available for several years.

4.4.2 Modeling

The development of new models was introduced in the subsection on lung effects. The reports of Hahn (1979) and Filipy et al. (1980) discuss models with respect to bone marrow effects and gastrointestinal tract effects. Each of their models, and the other models used for comparison in the reports, combine health effects on the basis of independence between effects, as did the RSS. Each model used a somewhat different method, however. As indicated in the lung subsection, considerable effort will be necessary to evaluate all these models in order to determine which ones are most applicable.

The hazard function concept, described by Hahn (1979), has applicability to late effects as well and has been used to reanalyze both the Japanese leukemia results (Groer et al., 1981a) and the MIT radium toxicity results (Groer et al., 1981b). In both these studies, the hazard function is different from that used by Hahn (1979). Application of the hazard function concept for both early and late effects may be possible and may lead to the ability to express all effects combined, possibly even as a function of time.

A new approach to modeling early bone marrow mortality has been developed by Jones (1981). Jones found that a log-log model provided a superior fit, in comparison with other models (including normal and log-normal distributions), to dose-response data for several mammals, for death due to the bone marrow syndrome. Further, the dose-response data were quite similar among the species tested, once the response was plotted against the ratio of the dose to the LD₅₀. Jones assumed that human dose-response would likewise fit such a log-log model. He related percentage mortality of mice to the percentage survival of their blood cell forming populations, and then argued that humans should also have much the same relationship between mortality and percentage survival of the blood cell forming units. The model permits the evaluation of mortality for various radiation exposures and conditions involving different radiations, mixed radiations, partial body irradiations, etc. This model may be useful, therefore, in determining, for example, the combined effect from external gamma and internal beta radiation of bone marrow.

For further details on models, see Section 6.

4.4.3 Decorporation

Health effects from internal deposition of radionuclides can be reduced in some cases by the administration, usually intravenously, of chelating agents that combine with specific radioactive atoms within organs and tissues and eliminate them from the body by excretion through the urine. The impact of such techniques on early fatalities is unclear but they would reduce chronic doses as well. Familiar chelating agents are EDTA and DTPA, the latter being generally more effective and better tolerated over a period of daily or near daily administration. Recently, new chelating compounds have been developed and tested that are more specific for certain radionuclides while reducing considerably the side effects (Durbin et al., 1981; Lloyd et al., 1981; Durbin and Raymond, 1982). Tetrameric catechol amides, in which the functional group is orthodihydroxybenzoic acid, have been shown to promote, in mice and dogs, the excretion of circulating plutonium and to accelerate biliary excretion of plutonium in the liver. Terminal alkane chains increase the lipophilicity and, hence, prolong the residence time of these chelating agents (ligands) in the body. This latter property suggests that these agents may be useful for removal of plutonium from

the lung, although plutonium is not a major contributor to risk, generally. Additionally, it may be possible to administer these agents orally rather than intravenously. Furthermore, the administered quantities and the frequency of administration can be reduced for use in long-term chronic administration. Preliminary toxicity screening studies indicate no acute toxic side effects of the most promising of these ligands, 3,4,3-LICAM carboxylate, at a dosage of 30 μ moles/kg body weight.

4.5 SUMMARY

The review of the RSS early health effects models has indicated a number of items that should be studied in greater detail. These items are summarized as follows:

1. As a general comment, the 1980 census should be used; current evacuation tactics and current protective action guides should be considered; and results from the new dose calculations for the Japanese survivor studies should be incorporated.

2. A dose-response curve for bone marrow mortality (hematopoietic syndrome) should be developed that considers the general population rather than a young-adult healthy population. Heroic treatment appears not to be a viable option, and the ability to provide reasonable supportive treatment should be related to the number of persons requiring such treatment. The influence of dose rate, even within a day or two after exposure, may be significant enough to decrease mortality below that anticipated by the RSS.

3. New information indicates that acute lung and gastrointestinal tract mortality estimates by the RSS need not be changed significantly. On the other hand, the sensitivity of the human lung may be high enough to contribute synergistically to bone marrow mortality as well as morbidity. Lung mortality due to protracted exposure from inhaled radionuclides needs further study, especially in light of the work of ITRI and PNL.

4. Radiation anxiety is a potential illness that would add to the number of persons affected by the accident and might confuse the identification of those physiologically affected by the radiation. Some effort to estimate this effect and its consequences, on the basis of plausible scenarios, would be useful.

5. All significant morbidity effects should be considered in the calculations, rather than the few in the RSS, to obtain a more complete and realistic measure of all persons who become ill with the different possible illnesses. A separation could be made of the more serious (bone marrow, lung and thyroid) possibly better quantified and the less serious (all the other illnesses) less well quantified. A new definitive pneumonitis morbidity dose-response curve should be used.

6. Synergism between effects and conditions of exposure should be considered to the extent possible.

7. New radiological health effects models by ITRI, PNL and others should be reviewed carefully. The hazard function used by the ITRI model for acute effects and by others for late effects may be applicable to developing a joint incidence of the combined early and late health effects.

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SECTION 5

LATE HEALTH EFFECTS

J.B. Robertson

5.1 INTRODUCTION

One would have expected that the vast amount of new data which have become available since the RSS was published would make critiquing the model a relatively straightforward task. For the late effects portion, this has not quite been the case. The BEIR (1980) report, which seeks to define, as accurately as possible, the quantitative relationships between doses and effects from ionizing radiation, seems to pose as many questions as answers. The problem is exacerbated by the possibility that the estimates given in the BEIR (1980) report may have to be modified when the reassessment of the dosimetry of the Japanese atomic bomb survivors is completed. Despite this development, BEIR (1980) is still the most current evaluation of the available data, and it is referred to frequently in this critique. When confronted with a choice between different interpretations of the data, both of which have some viability, our decision has been to use the more cautious one. This position is not inflexible and seems reasonable until the reevaluation of the Japanese atomic bomb survivor data is completed.

Thus a reexamination of the models for the upper, central and lower estimates of excess cancer deaths seems to be warranted, because of the present combination of circumstances, viz., the identification of susceptible fractions of the human population (Taylor et al., 1975), unexpected responses of cultured cells to fractionated exposures of low-LET radiation in cell transformation experiments (Miller and Hall, 1978; Borek, 1979; Little, 1979), and reevaluation of the neutron and gamma-ray doses received by Japanese atomic bomb survivors.

5.2 SPECIFIC COMMENTS

5.2.1 Changes to BEIR (1972) Risk Factors

In several instances, values chosen by the advisory group on health effects for the RSS risk factors differed from those of the then-recent BEIR (1972) report, as noted below.

The BEIR (1972) coefficient of 25 leukemia deaths per year per rem per million children irradiated in utero was not used by the RSS. The value 15 was used, instead, and this does not seem to be warranted in light of the recent update of the Oxford survey (Bithill and Stewart, 1975; Kneale and Stewart, 1976). [Webster (1981), on the other hand, argued that the fetus is no more sensitive than the adult.]

The second difference with respect to the BEIR (1972) coefficients was a different treatment of the gastrointestinal (GI) tract by the RSS. BEIR (1972) used a figure of 1 death/per million persons per rem per year (10^6 person-rem-yr), subdivided into 0.6 for stomach and 0.4 for the rest of the GI tract. The authors of the RSS chose to subdivide the 1 death/ 10^6 person-rem-yr into 0.6 for stomach, 0.2 for the rest of the alimentary tract, and 0.2 for the pancreas.

Since then, new data have become available on the atomic bomb survivors that might require reevaluation of these values (Miller and Hall, 1978). Beebe et al. (1978) report a death rate of 0.39 death per rad per year per million for esophageal cancer, and 0.81 excess death/ 10^6 person-rem-yr for stomach cancer. BEIR (1980) also suggests a value of 0.4 excess death/ 10^6 person-rem-yr for colon cancer based upon all of the current data. If these risks were summed up in a manner analogous to that of the RSS, one would have 0.8 death/ 10^6 person-rem-yr for stomach cancer and 0.8 death/ 10^6 person-rem-yr for the rest of the GI tract.

The value used in the RSS of 0.2 death/million per year per rad for pancreatic cancer is even more open to serious reconsideration. BEIR (1980) does not choose to report an induction rate for pancreatic cancer but states that it "appears to be low," and gives a range of induction rates. The Hanford studies (Hutchison et al., 1979; Gilbert and Marks, 1979), all of which show a correlation between pancreatic cancer and radiation exposure, yield risk values much larger than the value of 0.2 excess death per rad per million per year used in RSS.

If these greater values of risk were used, it would also seem more appropriate to calculate the doses to each portion of the GI tract rather than to use the dosimetry used in the RSS, where the dose to the large intestine was assigned to all portions of the alimentary tract. It would also be reasonable to calculate a pancreatic dose rather than to assume it equal to that of "other tissues."

Since additional years of follow up on the Japanese atomic-bomb survivors have shown the increase in leukemia risk persists for at least 25 years and that other cancer risks appear to persist for life (BEIR, 1980), consideration might be given to using plateaus of 30 years for leukemia and of lifetime for other cancer.

5.1.2 Central Estimate for Latent Cancer Fatalities

The central estimate used in the RSS (Section 9.3.3) simply takes the calculated latent cancer fatalities from the upper bound model and modifies them by the dose effectiveness factors listed in Table 9-7. For reasons outlined above in the introduction and defended below in the critique of Appendix G, a reevaluation of these dose factors might be considered.

5.1.3 Lower Bound for Latent Cancer Fatalities

In Section 9.3.4, the RSS suggested using a threshold of 10 or 25 rem as a basis for their lower-bound estimate. It is not clear whether the RSS applies this to in utero exposures. Such a threshold would not appear to be tenable for in utero exposures and, indeed, can be questioned for all exposures.

5.2 THYROID NODULES AND CANCERS

The mortality rate of 10% for thyroid cancer used in the RSS (Section 9.3.5) calculations seem a little high, since 89% of the radiation induced cancers are of the papillary carcinoma type and have a cure rate of at least 95% (BEIR, 1980). BEIR (1980) also suggests a lower risk rate of $4.0/10^6$ person-rem-yr for carcinoma, but their risk rate of $12.0/10^6$ person-rem-yr for benign nodules is three times greater than the rate used in the RSS model. BEIR (1980) does not

recommend age-specific rates. BEIR (1980) also notes the increased risk for women and notes that ethnic factors may also increase risk. Jewish females had a 17-fold increase in risk in one study population (BEIR, 1980).

5.3. GENETIC EFFECTS. POPULATION CHARACTERISTICS AND EXPOSURES.

In calculating the genetic dose it would seem to be a contradiction to say that individuals born after an accident would be exposed to external radiation from contaminated ground, and then in the next sentence state that they would not be exposed to internal radiation from incorporated radionuclides. If the ground were contaminated one would expect that food and water supplies would likely be contaminated also, although the food and water might be interdicted. Resuspension could occur. While the internal contribution from such sources might be small, it would seem better to demonstrate this to be the case before dismissing the dose as insignificant. This same statement is made in RSS Appendix I in Sections I3.1 and I6.5 without justification.

5.3.1 Single-Gene Disorders

The choice of 100 rem as the doubling dose by the RSS group should be reconsidered in light of the BEIR (1980) report, which used 50-250 rem as the likely range for the doubling dose for genetic damage. One could argue that upper, central and lower estimates of genetic damage should be made. This would relieve the reader of the responsibility of expanding the range of reported values as is suggested in Section I.6.2.

5.4 RISK ESTIMATES FOR CANCERS

Rather than use a fixed age distribution that is based on a national average, as the RSS now does in Appendix G, it would be advantageous if the model would have the capability of selecting a population that will accurately reflect regional differences in age and ethnic background, especially if the models are used for site-specific analysis.

5.4.1 Central Estimate

In explaining the choice of dose-effectiveness factors used in making the central estimate of late cancer deaths (section G1.4.2), the RSS identified a considerable body of radiobiological data that supported its position. Since its publication even more data have become available, not all of which support the original position.

The new studies leave little room for discussion about the existence of such a shoulder on in vitro cell survival curves, but a remarkable amount of leeway still remains in explaining its meaning. Since publication of the RSS, a great deal of work on in vitro malignant transformation utilizing mammalian cells has become available. Such tissue culture studies are considered reasonable models for examining the basic events responsible for transformation of a "normal" cell into a malignant one. Extrapolation of the in vitro data to in vivo situations is difficult, since it is almost impossible to duplicate all of the host factors that would effect whether such a transformed cell would develop into an actual tumor. These studies again confirm the existence of a shoulder in cell survival curves and demonstrate the reduced effectiveness of fractionated or low-dose-

rate, low-LET radiation with respect to cell killing, but they have not shown any indication of reduction in transformation frequency when the dose was fractionated. Indeed, several studies (Miller and Hall, 1978; Borek, 1979; Little, 1979; Bouk and Hall, 1974) have shown that fractionated doses become more effective than acute doses at transforming cells as the total dose is reduced. These types of studies would argue against dose reduction factors and, indeed, could be used to support an argument for dose factors with values greater than one at low, fractionated doses (Little, 1979).

Furthermore, the role of DNA repair alluded to with RSS has been extensively investigated since the RSS was published. The additional data, however, have far from settled the original question. It has been reported (Little, 1977) that the DNA repair processes that lead to cell survival are different from those that repair potentially transforming events.

Finally, the identification of subpopulations of humans with an apparently heritable sensitivity to cancer induction by radiation (Taylor et al., 1975) forces one to consider the possibility that such populations might dominate the dose-response curve at low doses. The Hanford studies (Hutchison et al., 1979; Gilbert and Marks, 1979) might identify yet another population that could dominate the dose response curve at low doses. Rather than having a heritable sensitivity to ionizing radiation, the Hanford workers with pancreatic cancers could be experiencing some sort of synergistic effect between ionizing radiation and some other agent as yet unidentified. Such agents could be distributed among the population, yielding subpopulations with an increased sensitivity to cancer induction by ionizing radiation. Such considerations require a reexamination of the dose reduction factors used in the central estimate.

5.4.2 Leukemia Upper Bound Estimate

The basic model, taken from BEIR (1972), might require updating since new data led to a revision of this model in the BEIR (1980) report. Along with those changes it is believed that the update of the Oxford survey (Bithill and Stewart, 1975; Kneale and Stewart, 1976) would require that the value for the plateau for those children irradiated in utero be reconsidered. Additionally, the alpha dose to the blood forming tissue might require more consideration than it is receiving, if the model is to be extended to other aspects of the nuclear fuel cycle.

5.4.3 Lung Cancer

The relatively large amount of new data on lung cancer would seem to require an entire reworking of the lung cancer portions of Appendix G. The data indicate that a life-time plateau might be more appropriate than the 30-year plateau used. The data also justify consideration of age-specific lung cancer risk estimates and possibly age-specific latent periods. Postulated accidents imply relatively little alpha radiation, but the extensive new data on alpha-particle-induced lung cancer would seem to demand more attention to possible consequences of the alpha dose. Also, the recent data do not lend support to the concept of a threshold for lung cancer.

5.4.4 Breast Cancer

Recent reports summarized in BEIR (1980) suggest that the latent period

and risk factors should be a function of age, rather than fixed, as is currently the case in the RSS model.

5.4.5 Other Cancers

Information which has become available since the RSS was published would seem to require a revision of the rate estimates for the "other cancers" category. In particular, the new information with respect to liver cancers should be noted. BEIR (1980) reports a risk rate of 13 and 0.7 death/ 10^6 person-rem-yr for high- and low-LET radiation respectively, again requiring a closer examination of the alpha increment of the total dose. Finally the threshold value of 500 rem and the risk estimate of $0.1/10^6$ person-rem-yr suggested for skin cancers in the RSS should be reexamined. BEIR (1980) estimates a risk factor of $1.0/10^6$ person-rem-yr for skin cancer. One study (Hempelmann et al., 1975) yielded a risk factor of $0.4/10^6$ person-rem-yr for irradiated infants where the average skin dose was only 330 rads.

5.5 HYPOTHYROIDISM

BEIR (1980) suggests that external thyroid radiation may be 2.5 times as effective as internal radiation in inducing hypothyroidism. This differs considerably from the equal effect approach used in the RSS, Section H6, for calculating the central estimate of the risk of hypothyroidism. Also the data cited (Section H3.4.1 and Table VI, H-3) to demonstrate a threshold of 20 rem for hypothyroidism induction do not appear to be compelling. Finally, hypothyroidism after in utero exposure to iodine isotopes should be considered.

5.6 AREAS REQUIRING ADDITIONAL STUDY AND CONSIDERATION

These areas are:

1. Genetic hazard of internal alpha emitters, if model is to be extended to entire nuclear fuel cycle.
2. Late lung effects other than neoplasia (e.g., fibrosis).
3. Synergistic effects between radiation and other environmental agents.
4. Nonspecific aging.
5. In vitro transformation studies at low doses and low dose rates.
6. Hypothyroidism and other thyroid effects after in utero exposure.
7. Variation in the sensitivity of the skin at different anatomical sites to the induction of skin cancer by radiation.

5.7 SUMMARY

The major elements needing reconsideration have been identified above as:

1. The risk coefficient and plateau period for leukemia.

2. Risk estimates for the GI tract and for pancreatic cancer.
3. Use of plateaus for cancers other than leukemia.
4. Assessment of likelihood of in utero effects.
5. Risk rate for thyroid cancer and assumption that ten percent of such cancers are fatal.
6. Influence of sensitive populations on risk estimation.
7. Doubling dose for genetic effects.
8. Dose-rate effects.
9. Age-specific cancer estimates.
10. Significance of alpha dose.
11. Effectiveness of internal and external radiation in producing thyroid damage.
12. Synergisms.

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SECTION 6

COMPUTER CODE IMPLEMENTATION

J.S. Evans and D.W. Cooper

6.0 INTRODUCTION

This section of our report includes three major subsections. The first, 6.1, is an introductory description of the computer code implementation of the RSS health effects model. The second, 6.2, reviews the differences between the computer implementation and the RSS health effects model described in Section 2 of our report. The third, 6.3, discusses error and sensitivity analyses.

6.1 DESCRIPTION - COMPUTER IMPLEMENTATION: RSS HEALTH EFFECTS MODEL

This outlines the RSS health effects model as implemented by the CRAC (Calculation of Reactor Accident Consequences) code and as described in the CRAC Code User's Manual.

The total number of deaths due to an accident is the sum of the early deaths and the latent deaths. The CRAC code uses different methods for estimating each of these components of the death toll. Early fatalities are calculated by applying non-linear organ-specific dose-response functions to the organ-specific doses estimated to be received by representative individuals. For the purpose of estimating doses, the population surrounding the reactor is divided into 544 geographical zones and doses are estimated at the midpoint of each zone. Latent fatalities are calculated by applying constants of proportionality to the aggregate population doses. As described in Sections 6.1.1.1 and 6.1.1.2 the Code allows for both a proportional and a piecewise-linear relationship between dose and response for latent fatalities. The details of each of these methods are described below.

6.1.1 Latent Fatalities

The total number of latent fatalities attributable to an accident under a specific set of meteorological conditions is given by the computer code (CRAC) implementation of the model as a summation of two components - latent fatalities due to *early exposure*, n_{le} , and latent fatalities due to *chronic exposure*, n_{lc} :

$$n_l = n_{le} + n_{lc} \quad (6-1)$$

Early exposure includes the exposure which occurs as the plume passes over the affected region, and from groundshine within the first few days after the accident. No exposure occurring more than seven days after the accident is included. We use the exposure index $t=0$ to designate this exposure interval. Chronic exposure is divided into ten time intervals following the accident. The first chronic exposure interval includes the period from the time of the accident until the first anniversary of the accident. We designate this interval as $t=1$. The second chronic exposure time interval runs from the first anniversary of the accident to the tenth anniversary. This is designated as $t=2$. The next seven chronic exposure intervals are each ten years in length, together covering the

period from the tenth to the eightieth anniversary of the accident. These are designated $t=3$ to $t=9$. The final chronic exposure interval, $t=10$, runs from the eightieth anniversary of the accident to infinity. Note that inherent in this scheme is a certain amount (likely to be quite small) of double counting of groundshine exposure. This occurs since the early exposure interval, $t=0$, is also included in the first chronic exposure interval, $t=1$.

The number of latent fatalities due to *early exposure* is itself a sum of two components - latent fatalities due to *early external exposure*, $n_{le,ext}$, and latent fatalities due to *early internal exposure*, $n_{le,int}$:

$$n_{le} = n_{le,ext} + n_{le,int} \quad (6-2)$$

To find the number of latent fatalities due to *early external exposure*, the total early external dose to a representative individual in the s th radial sector and r th concentric ring is found by summation of the dose due to groundshine ($j=1$) and the dose due to cloudshine ($j=2$). These doses are found by multiplying radionuclide(k)- and organ(i)-specific dose conversion factors, DCF_{ijk1} , by the pathway(j)- and radionuclide-specific exposures, $EXPO_{jkr0}$, received in the first time interval ($t=1$) after the accident in that sector and ring. Note that for any single set of meteorological conditions only a small subset of the sectors and rings have non-zero exposures. Organ-specific population doses (person-rem) are found by summing over all exposed sectors and rings the product of the sector and ring specific population (POP_{sr}) and sector-ring- and organ-specific doses. The total number of latent deaths due to early external exposure is found by summing over all organs the products of these organ specific population doses and organ-specific constants of proportionality, $MRCON_{i1}$. (Note $MRCON_{i1}$ is the same as the b_{i1} discussed in Chapter 2).

$$n_{le,ext} = \sum_{i=1}^6 [MRCON_{i1} \sum_{s=1}^{16} \sum_{r=1}^{34} POP_{sr} (\sum_{j=1}^2 \sum_{k=1}^{54} DCF_{ijk1} EXPO_{jkr0})] \quad (6-3)$$

The latent fatalities due to *early internal exposure* are due solely to inhalation of radionuclides from the passing cloud ($j=3$). The time interval (t)-organ- and radionuclide-specific internal doses are found by multiplying time-organ- and radionuclide-specific dose conversion factors, DCF_{i3kt} , by the radionuclide-specific exposures, $EXPO_{3krs1}$, received in the first time interval after the accident in that sector and ring. Time interval and organ-specific population doses (person-rem) are found by summing over all exposed sectors and rings the product of sector and ring specific population (POP_{sr}) and sector-ring-organ- and time-interval-specific doses. The total number of latent deaths due to early internal exposure is found by summing over all time intervals and organs the products of time-interval- and organ-specific constants of proportionality, $MRCON_{it}$.

$$n_{le,int} = \sum_{t=1}^{10} \sum_{i=1}^6 [MRCON_{it} \sum_{s=1}^{16} \sum_{r=1}^{34} POP_{sr} (\sum_{k=1}^{54} DCF_{i3kt} EXPO_{3krs0})] \quad (6-4)$$

The number of latent fatalities due to *chronic exposure* is also a sum of two components - latent fatalities due to *chronic external exposure*, $n_{lc,ext}$, and

latent fatalities due to *chronic internal exposure*, $n_{lc,int}$.

$$n_{lc} = n_{lc,ext} + n_{lc,int} \quad (6-5)$$

The latent fatalities due to *chronic external exposure* are all due to groundshine ($j=9$). The groundshine dose in any time interval after the accident to a representative individual in any sector and ring is found by summing over the ten isotopes ($m=1$ to 10) important for this pathway the products of isotope-organ-time-interval-specific dose conversion factors, DCF_{i9kt} , and isotope-time-interval-specific exposures for the sector and ring, $EXPO_{9mrst}$. Table 2-1 in Section 2 of our report lists the specific radionuclides which are considered in each of the chronic exposure pathways. The total organ-specific population dose due to chronic external exposure is found by summing these individual doses over sectors, rings and time intervals. And finally, the number of latent deaths due to chronic external exposure is found by summing over the six organs the product of organ-specific population doses and organ-specific constants of proportionality, $MRCON_{i1}$.

$$n_{lc,ext} = \sum_{t=1}^8 [MRCON_{i1} \sum_{s=1}^{18} \sum_{r=1}^{34} (\sum_{t=1}^{10} POP_{srt} \sum_{m=1}^{10} DCF_{i9mt} EXPO_{9mrst})] \quad (6-6)$$

Although equation 6.6 reflects a summation over ten time intervals, the CRAC Code User's Manual is not altogether clear on this point. However, a review of the CRAC Code indicated that for chronic groundshine ($j=9$) the ground exposures are integrated over four time intervals: $0 \leq t \leq 1$ yr, $1 \leq t \leq 30$ yr, $30 \leq t \leq 60$ yr and $60 \leq t < \infty$ yr. The first year integrated chronic ground exposure is $EXPO_{9mrs1}$. The second period ($1 \leq t \leq 10$ yr) integrated chronic ground exposure $EXPO_{9mrs2}$ is estimated as $1/3$ of the integral of the ground exposure over the period $1 \leq t \leq 30$ yr. The third ($10 \leq t \leq 20$) through tenth ($t \geq 80$) period integrated chronic groundshine exposures are estimated similarly.

The calculation of latent fatalities due to *chronic internal exposure* is a bit more complicated. There are five pathways involved: inhalation of resuspended materials ($j=4$), and four ingestion pathways ($j=5$ to 8). The four ingestion pathways include: ingestion of milk contaminated due to fallout ($j=5$), and of all other foods contaminated due to fallout ($j=6$), and ingestion of milk contaminated due to root uptake ($j=7$), and of all other foods contaminated due to root uptake ($j=8$). For the chronic inhalation pathway nine isotopes are involved ($n=1$ to 9), and for the ingestion pathways seven isotopes are involved ($p=1$ to 7). In addition to summation over radionuclides, organs, sectors, and rings, multiple summations over time periods are necessary to account for the contributions to each time interval dose of exposures in all previous time intervals.

$$n_{lc,int} = n_{lc,inhalation} + n_{lc,ingestion} \quad (6-7)$$

$$n_{lc,inhalation} = \sum_{t=1}^8 MRCON_{i1} \sum_{s=1}^{18} \sum_{r=1}^{34} \sum_{t=1}^{10} POP_{srt} \sum_{T=1}^t \sum_{n=1}^9 DCF_{i4n(t-T+1)} EXPO_{4nrst} \quad (6-8)$$

$$n_{ic, ingestion} = \sum_{i=1}^6 MRCON_{i1} \sum_{s=1}^{16} \sum_{r=1}^{34} \sum_{t=1}^{10} POP_{srt} \sum_{j=5}^8 \sum_{p=1}^7 DCF_{tjpt} \sum_{T=1}^{10} EXPO_{jprsT} \quad (6-9)$$

As indicated by comparison of equations 6-8 and 6-9, the details of handling the time intervals are different for the two major chronic internal exposure pathways. Again, the CRAC Code User's Manual was not clear on this point. However, a review of the CRAC code indicated that for chronic inhalation of resuspended matter ($j=4$) the amount inhaled (C_i) is integrated for each of six time intervals: $0 \leq t \leq 10$ yr, $10 \leq t \leq 20$ yr, $20 \leq t \leq 30$ yr, $30 \leq t \leq 40$ yr, $40 \leq t \leq 50$ yr, and $50 \leq t < \infty$ yr. The total dose in each of these six time intervals due to inhalation in that interval, or any of the previous intervals, is calculated using time dependent dose conversion factors (rem/Ci). The doses due to inhalation of resuspended matter in each of the ten time intervals of interest are then estimated from the doses in the six time intervals. The first year dose is taken to be one-tenth of the $0 \leq t \leq 10$ yr dose. The dose for the second time interval of interest ($1 \leq t \leq 10$ yr) is taken to be nine-tenths of the $0 \leq t \leq 10$ yr dose. For each of the final four time intervals ($50 \leq t \leq 60$ through $80 \leq t < \infty$) the dose is taken to be one-quarter of the $50 \leq t < \infty$ dose. For chronic ingestion a different approach is taken. First the exposure is integrated over all time to obtain "lifetime" ingestion (C_i). The "lifetime" ingestion is treated as if it all occurred at $t=0$. Doses (rem) in each of five ten-year time intervals and a sixth time interval covering $50 \leq t < \infty$ yr following the accident are estimated by applying time specific dose conversion factors (rem/Ci) to this total "lifetime" ingestion. The first year dose commitment is estimated as one-tenth of the integrated dose over the first ten years. The second period ($1 \leq t \leq 10$) dose commitment is estimated as nine-tenths of this integrated dose for the first ten years. For each of the final four time intervals ($50 \leq t \leq 60$ through $80 \leq t < \infty$) the dose is taken to be one-quarter of the $50 \leq t < \infty$ dose.

One other detail of calculation involves the methods of determining the size of the exposed population, POP_{srt} , for each of the four chronic ingestion pathways. Although equation 6-9 indicates that the same estimate of exposed population was used for all of the chronic ingestion pathways and that this was the same estimate of exposed population which was used for all other chronic exposure pathways, this is not the case. In fact, the code calculates a different exposed population for each ingestion pathway in a rather complex manner - reflecting the share of crops grown locally and other factors which would be thought to influence exposure to contaminated foods.

6.1.1.1 Upper Bound (1972 BEIR) Model for $MRCON_{it}$

The upper bound on dose-response for deaths due to latent effects (cancers) was derived from the estimates of the 1972 BEIR report. Total latent deaths are the sum of latent deaths from early exposure and latent deaths from chronic exposure. For either early or chronic exposure the population dose to an organ i in a time interval t is the sum over exposed sectors and rings of the products of population and dose for each specific sector and ring. The number of latent cancer deaths is the sum of the cancer deaths for the six organs (i) over the ten periods of dose (t). The ten dose intervals are the first year and then ten-year intervals after that followed by the period beyond eighty years. The organs are (in order of i): bone marrow, lung, breast, bone, GI tract (including stomach and alimentary canal), other (including the pancreas but excluding

the thyroid). The values of $MRCON_{it}$ are crucial, being the relationship between internal dose due to early exposure in the time interval t to organ i and the probability of death (deaths per person-rem). Because as time passes persons alive at the time of the accident will make up a smaller and smaller fraction of the population living in any sector and ring, the $MRCON_{it}$ factors used in the model for latent deaths from early internal exposure (which are applied to the population-dose, calculated as if the entire population had been alive to receive the early inhalation exposure as the cloud passed) are decreased over time. The values for three such periods of dose are given in Table 6-1. For external exposure and chronic internal exposure the values of $MRCON_{i1}$ are used in all calculations.

These show generally less than an order of magnitude difference among the organs and show a substantial decrease in the coefficient $MRCON_{it}$ with time. (As will be shown later, these $MRCON_{it}$ coefficients are themselves dependent on assumptions about, among other things, the latency and plateau periods assigned to each cancer type.)

6.1.1.2 Central and Lower Bound Models for $MRCON_{it}$

Central estimates of latent cancer fatalities are obtained by modifying the $MRCON$ values given above. The $MRCON$ values for all organs except the breast are multiplied by "dose effectiveness factors" varying from 0.2 to 1.0 depending upon the total dose received and the mean dose rate in Table 6-2.

Lower-bound estimates of latent cancer fatalities were obtained in the RSS report's health effects model by applying the upper-bound (BEIR, 1972) $MRCON$ values to population doses which have been modified as if there were a 10 rem or 25 rem threshold, depending upon the organ of interest. However, the CRAC Code does not permit the use of the lower-bound estimates.

6.1.2 Early Fatalities

The model for early fatalities may also be written as a multiple summation:

$$n_e = \sum_{r=1}^{18} \sum_{s=1}^{34} POP_{sr} \left[1 - \prod_{i=1}^3 (1 - f_i \left(\sum_{k=1}^{54} \sum_{j=1}^3 (DCF_{ijk} EXPO_{jkrs}) \right) \right] \quad (6-10)$$

a summation, over the annular rings (r) and the radial sectors (s) exposed to the plume, of the product of the population in that sector and ring (POP_{sr}) times one minus the product, over the three body organs (i) contributing to early deaths, of one minus the probability of death due to that organ (f_i). The calculation of exposure ($EXPO_{jkrs}$) and the conversion of exposure to organ-specific dose, using dose conversion factors (DCF_{ijk}), is done in the same manner as for latent deaths.

The dose-response (mortality) functions used in the CRAC code calculation of early deaths are piecewise-linear approximations to the functions described in Section 9.2, Appendix VI of WASH 1400. For early mortality three organs are modeled: bone marrow, lung, and GI tract. Each has a lower threshold dose for which the probability of death is taken to be zero, and an upper threshold dose for which the probability of death is taken to be unity. Between these two

Table 6-1. Values for $MRCON_u$

	t=1 (< 1 year)	t=5 (31-40 years)	t=8 (61-70 years)
marrow	28	9.7	1.7
breast	26	9.4	0.3
other	25	8.5	0.2
lung	22	8.1	0.2
intestines	14	5.0	0.1
bone	6.9	1.6	0.1

[Note: For external exposures and chronic internal exposures $MRCON_i = MRCON_{i1}$]

Table 6-2. Dose Effectiveness Factors

Dose(rem)	Dose Rate (rem/day)		
	<1	1-10	>10
<10	0.2	0.2	0.2
10-25	0.2	0.4	0.4
25-300	0.2	0.4	1.0

thresholds the function may have as many as two intermediate breakpoints, at which its slope changes. Graphs of these piecewise-linear dose-response functions are presented below. The bone marrow dose-response function approximates curve "B" (supportive treatment) from Figure VI 9-1, WASH 1400. The lung dose-response function approximates the "Criterion" curve from Figure VI 9-3, WASH-1400.

6.1.3 Morbidity

Early morbidity was calculated for two organs, the respiratory system and the GI tract, and for prodromal vomiting. These calculations are based upon the early doses described in section 6.1.2 and piecewise-linear dose-response functions with thresholds. For the lung, the modeled probability of response is: zero for lung doses less than 3000 rad; 0.05 for 3000 rad; 1.0 for 6000 rad; and linear between these values in the range of lung doses between 3000 and 6000 rad. For the GI tract, the modeled probability of response is: zero for GI tract doses less than 1000 rad; 0.05 for 1000 rad; 1.0 for 2500 rad; and linear between these values in the range of GI tract doses between 1000 and 2500 rad. For prodromal vomiting, the modeled probability of response is: zero for whole body doses up to 55 rad; 1.0 for 370 rad; 0.3 for 150 rad; 0.8 for 280 rad; and piecewise linear between these values in the range of whole body doses between 55 and 370 rad.

6.1.4 Thyroid Effects

According to the CRAC code user's guide three thyroid effects are modeled: benign nodules, malignant nodules and hypothyroidism. The probability of each of these effects is based solely upon early exposure to the thyroid. The early dose to the thyroid is calculated on the assumption that I-131 is only one-tenth as effective in inducing thyroid nodules as external x-rays. The incidence of hypothyroidism is proportional to the dose with incidence estimated at $110/10^6$ person-rem. Up to doses of 1500 rem, the incidence of thyroid nodules is also proportional to dose. The constant of proportionality applied to benign nodules is $200/10^6$ person-rem. For cancerous nodules the constant is $134/10^6$ person-rem. For doses between 1500 rem and 5000 rem, the constants of proportionality for thyroid nodules, both benign and malignant, is reduced by a factor of 2. Above 5000 rem, no nodules are induced, due to ablation of the thyroid.

6.1.5 Exposure Pathways

The early exposure due to groundshine (Ci/m^2) is given by:

$$EXPO_{1kr} = GC_{1kr} SHFAC1'2 \quad (6-11)$$

in which GC_{kr} is the initial ground concentration of nuclide k in ring r (Ci/m^2) and $SHFAC1'2$ is the ground shielding factor with (1) or without (2) evacuation. The ground concentration is obtained by considering the air concentration and the wet and dry deposition rates.

The air concentration AC_{kr} is calculated using a Gaussian model for atmospheric dispersion, incorporating vertical and lateral dispersion (as functions of distance and stability), release height and building wake effects. Concentrations are modified to reflect the influences of wet and dry deposition and radioactive

Figure 6-1. Early Lung Dose-Response (Death) Function
Used in CRAC Code.

Figure 6-2. Early G.I. Tract (LLI Wall) Dose-Response (Death)
Function Used in CRAC Code.

Figure 6-3. Early Total Marrow Dose - Response (Death)
Function Used in CRAC Code.

decay. Actual meteorological sequences may be sampled to obtain a probability distribution of consequence magnitudes for a postulated release.

The factors that enter into the values for $EXPO_{1kr}$, the ground exposure, have been outlined. The second mode is that for external exposure to the cloud ("cloudshine") ($Ci\text{-sec}/m^3$):

$$EXPO_{2kr} = AC_{kr} t_r SHFAC3'4[FCCV] \quad (6-12)$$

the product of the air concentration, the duration of cloud passage, t_r , the shielding factors assuming evacuation (3) or no evacuation (4), and the finite cloud conversion factor ($FCCV$). The exposure due to inhalation (Ci) is

$$EXPO_{3kr} = AC_{kr} t_r BRATE \quad (6-13)$$

the product of the air concentration, the duration of the cloud passage, and the breathing rate of the typical person.

The dose conversion factors are then used to obtain doses from exposures for the various nuclides and exposure modes in each sector and ring. The dose conversion factor for groundshine exposures is DCF_{i1kt} for organ i and nuclide k during times t , taken as 8 hours and 7 days. The dose conversion factor for cloudshine is DCF_{i2kt} . That for inhalation dose is DCF_{i3kt} .

To account for latent deaths due to chronic exposures, the second (pathway) index takes the values 4 through 9 for, respectively: resuspension of particulate material, ingestion of all food other than milk contaminated by radionuclides, which were directly deposited on crops, ingestion of milk by cows grazing on crops contaminated by direct deposition, ingestion of milk by cows grazing on crops contaminated by root uptake, ingestion of all other foods containing radioactive materials due to root uptake, and exposure to groundshine from contaminated ground.

6.1.6 Evacuation and Relocation/Interdiction

All of the doses used in the calculations described above may be modified extensively as a result of modeled evacuation, relocation, and interdiction. The fundamentals of these models were described in Section 2 of our report.

6.2 DIFFERENCES BETWEEN CRAC AND RSS HEM

This discussion addresses the differences between the apparent RSS HEM, as outlined in Appendix VI of WASH-1400 and the models actually used in the CRAC code, as outlined in the draft CRAC Code User's Manual. Differences may logically be grouped into two categories:

- (1) response functions for early health effects
- (2) response functions for latent health effects

6.2.1 Response Functions for Early Health Effects

The early health effects calculated by CRAC are death due to exposure of bone marrow, lung, and GI tract, disease (impairment) of the respiratory system and GI tract and prodromal vomiting. The piecewise-linear dose-response functions used by CRAC closely approximate the dose-response functions discussed in the RSS HEM.

However, the RSS HEM also discusses early effects (such as radiation thyroiditis, early hypothyroidism, prenatal and neonatal deaths (due to exposure of fetuses and/or embryos), temporary sterility, congenital malformations and growth retardation (due to exposure of fetuses and/or embryos), and cataracts) that are not included in the CRAC calculations. In most cases the omissions seem to be justified by the conclusions of preliminary analyses done during the development of the code. For example, section 9.2.2.5 of Appendix VI, WASH 1400 concludes that the number of prenatal deaths would be only about 5% of the early fatalities. And similarly, section 9.2.2.4 reasons that anyone receiving a lethal dose for radiation thyroiditis would have already died due to bone marrow exposures. Section 9.2.3.6 of the RSS HEM argues that no cataracts would be expected among survivors. However, the incidence of temporary sterility might be quite high. More troublesome are the omissions of congenital malformations and growth retardation from the CRAC computations. Section 9.2.3.5 of Appendix VI, WASH-1400 estimates that the number of cases of microcephaly might be 50% of the early fatalities due to the bone, lung and GI tract doses and that, as a very rough estimate, the number of other congenital effects might equal the number of cases of microcephaly.

8.2.2 Response Functions for Latent Health Effects

The constants of proportionality used by CRAC to estimate latent cancers are identical to those given in Table VI 9-5 of Appendix VI, WASH-1400. With CRAC it is possible to make estimates of latent cancer fatalities using either the "upper bound" BEIR model or the "central estimate" model employing the same dose reduction factors given in Table VI 9-7 of Appendix VI, WASH-1400. However, the CRAC Code is not set up to allow calculation of the "lower bound" latent cancer estimates. The work of Wall et al. appears to indicate that latent cancer estimates produced with 10 and 25 rem thresholds (i.e., the "lower bound" model) are within about 20% of those produced using the "central" model, for the most severe reactor accidents.

The constants of proportionality for cancer deaths developed by BEIR (1972) and modified for use in the RSS HEM assume that not all cancers will prove fatal within 5 years of diagnosis. The risk estimates are based on only those cases which are likely to be fatal within five years of diagnosis. Section G8 of Appendix VI, WASH-1400 indicates that the following assumptions were used in the estimation of risk factors for cancer mortality:

organ	cases/death
leukemia	1
lung	1
stomach	1
colon, rectum	3
pancreas	1

breast	2
bone	1.25

However, the CRAC code does not provide estimates of the non-fatal cancers expected to result from the accident. Wall et al. indicate that "typically" breast cancer would be expected to be responsible for 27% of the total cancer deaths from PWR-1 releases and 39% of the total cancer deaths from PWR-2 releases. The cases/deaths ratio of 2 indicates there will be as many non-fatal breast cancer cases as there are fatal breast cancer cases. The GI tract and bone combined are expected to account for approximately 8% and 11%, respectively, of the total cancer deaths for these two classes of releases. Thus, latent morbidity, a potentially significant component of the overall health impact of the accident, was ignored in the CRAC consequence estimates.

There are small differences between the constants of proportionality used by CRAC to calculate latent genetic effects and those given in Tables 2-5 and 2-6 (RSS Tables VI 9-11 and VI 9-12). The constants of proportionality (total number of effects in all future generations/ 10^6 person-rem) used by CRAC for internal exposures due to inhalation of material from the plume are given below:

Exposure Interval	Effect Type			
	Autosomal Dominant	Multifactorial	Chromosomal	Spontaneous Abortion
0-1 yr	45.8	85.4	5.1	35.9
1-10 yr	34.5	68.6	4.1	28.8
11-20 yr	17.7	35.4	2.1	14.9
21-30 yr	4.4	8.8	0.5	3.7
31-40 yr	0.7	1.3	0.1	0.6
41-50 yr	0.0	0.0	0.0	0.0

For early external exposures and all chronic exposures the 0-1 yr constants of proportionality were used. For multifactorial disorders these constants are based on the assumption that 50% of the baseline incidence is related to the mutation rate. None of the constants differ by more than 50% from those given in the RSS HEM and most are within 20% of the RSS HEM values.

6.3 ERROR AND SENSITIVITY ANALYSES

Many uncertainties are involved in modeling the health consequences resulting from a known specified temporal and spatial pattern of environmental exposures due to an accidental release of radionuclides. These uncertainties are compounded by uncertainties in the relationships between releases and exposures and by variability in meteorological conditions. One might reasonably ask what influence each of these uncertainties has on the overall uncertainty in the predicted health consequences and/or what part of the overall uncertainty in the predicted health consequences of an accidental release is due to uncertainty in the dosimetry and dose-response models.

These questions are best answered by two types of investigations. One form

of investigation assumes that the correct functional relationships (e.g., proportional, exponential, linear-quadratic) have been used in the models and looks only at the uncertainty induced by uncertainty in the selection of model parameters (e.g., slope, exponent). The second form of analysis asks how model predictions are influenced by the choice of model (e.g., proportional, exponential) form or structure (e.g., level of aggregation in time and space).

In this section the major sources of uncertainty in each of the health response sub-models are summarized, and the results of the major sensitivity analyses of the RSS HEM dose-response models are described. Appendix 3 contains a brief discussion of sensitivity analysis.

6.3.1 Sources of Uncertainty in RSS HEM Dose-Response Models

6.3.1.1 Early Effects

As described in Section 6.1.2, the five dose-response models for early effects in the CRAC Code are all piecewise-linear functions. Since the code allows each function to have three line segments, theoretically thirty parameters would be required to completely specify the model. In practice since some of the dose response functions involve only one or two segments, only twenty-two parameters are needed to specify the CRAC early effects model. Thus, it might appear that a complete parametric error analysis would need to look at the significance of uncertainties in each of these twenty-two model parameters.

A more intuitively meaningful understanding of the sources of uncertainty is obtained by considering each dose-response function as cumulative normal (or lognormal) and asking how much uncertainty there is in both the LD_{50} and the LD_{10} (or any equivalent second parameter). Using these functions all uncertainties are rooted in uncertainties in only ten model parameters (i.e., LD_{50} and LD_{10} for each of the five responses).

Other issues deserving attention in an error analysis of the early effects model would be:

1. The potential importance of effects due to doses to organs that are not considered in these models.
2. The error, if any, introduced by treating the effects upon individual organs as independent, i.e., the importance of synergisms and/or antagonisms.

6.3.1.2 Latent Effects

The RSS HEM latent effects model approached the issue of uncertainty in the form of the dose-response function by providing the three alternate forms (i.e., proportional, piecewise-linear, and linear-threshold).

Cast in this framework the analysis of the parameter uncertainties involves the selection of:

- a. The six organ-specific constants of proportionality for use in the proportional model, and the ten (only nine independent) fractions of

dose-response functions.

Full-fledged studies of propagation of errors and/or model sensitivity were outside the scope and budget of our work. As was indicated in previous sections, the model is quite complex and therefore not completely suitable for analytical study. However certain insights are possible through simplified analyses, and review of the work of others. (The discussion which follows applies only to use of the RSS HEM for prediction of health consequences of reactor accidents. Further, the validity of these remarks is contingent upon the validity of the results of models used in the RSS to simulate the dispersion and environmental transport of radionuclides; to estimate organ-specific doses from environmental contamination levels; and to simulate evacuation, interdiction and population relocation.)

6.3.2.1 Preliminary Considerations

The discussion in section 6.3.1 of this report indicates that a thorough parametric sensitivity and error analysis of the RSS HEM would involve determination of the partial derivatives of the predicted health effects with respect to each of twenty-two parameters of the early effects model and each of the thirty (or more) parameters of the latent effects model for a variety of conditions representative of the ranges of releases, atmospheric dispersion conditions (wind speed, stability, and precipitation), and plausible evacuation scenarios (lag time and speed).

However, two strategies are potentially useful in limiting the scope of such analyses. First, one may scrutinize the results of previous model calculations in an attempt to find organs which are not, in most cases, responsible for a significant fraction of the early consequence or late consequence predictions. These may be ignored in the sensitivity analysis unless:

- a. Their parameter uncertainty is so great that for certain plausible parameter values they could produce appreciable effects.
- b. The few cases (scenarios) in which they do produce appreciable effects are fairly likely, or the magnitude of the effects produced in these cases is extremely large.
- c. Cases in which they might be expected to contribute to consequences have not been examined.

Secondly, one may examine the reported parameter uncertainties for each of the remaining parameters in an attempt to find parameters for which the uncertainties are small. These may be ignored in the sensitivity analysis unless they are parameters of dose-response functions responsible for a large fraction of the predicted consequences.

6.3.2.2 Reported Sensitivity Studies: Description

The recently published (September, 1981) review draft of NUREG/CR-2300, *PRA Procedures Guide*, section 9.6.7, notes that the health effects modeling area "is another area in which there is a relative paucity of sensitivity studies." In contrast to the many error and sensitivity studies (primarily by Aldrich et al.)

in the area of evacuation modeling, and several studies of the pathways and dosimetry models, there are very few studies directly concerned with the dose-response relationships used in the RSS HEM. The three studies considered in our work were those by Kelly, Murfin, and Wall et al. Their methodologies are described below and their results are summarized in section 6.3.2.3.

Kelly reported on the results of a series of sensitivity studies performed by him and his colleagues at the National Radiological Protection Board in Great Britain. Their primary goal was to relate this work to safety analysis of the liquid metal fast breeder reactor (LMFBR), but many of their results were relevant to light water reactors (LWR) as well. Studies were done assuming 0%, 1%, 5%, or 10% of the molten core vaporized. The results pertinent to the RSS model (i.e., those with 0% vaporization of the core) were done assuming releases of the following fractions of the inventory of radionuclides:

- Noble gases: 0.9
- Halogens: 0.9
- Rb-Cs: 0.8
- Sb-Te: 0.15
- Ru group: 0.03
- (Mo, Tc, Ru, Rh)
- La Group: 0.003
- (Y, Zr, Nb, La, Ce, Pr, Nd, Pm)
- Actinides: 0.003
- Sodium: 0.05

Except for sodium these releases were "based on the release of elements from uranium-oxide fuel melted in environments typical of LWR accident conditions (USNRC, 1975)."

The material was assumed to be released in 1000 s to an effective height of 100 m, under a weighted average of atmospheric conditions (stability classes C and D at a 1 to 2 ratio), with particles of 1 μm AMAD (activity median aerodynamic diameter), released to the most populated 30 degree sector at a remote "semi-urban" site. A Gaussian plume dispersion model was used, accounting for dry deposition and radioactive decay. Exposure pathways in the model included: cloudshine, inhalation from the plume, and both early and chronic groundshine.

The LD-50's and SD-50 for early effects chosen were:

Mortality

- Marrow: 350 rad
- Lung: 25000
- GI: 3500

Morbidity

- Lung: 4250

For latent cancer deaths a simple proportional dose-mortality model was used. The constants of proportionality (deaths/ 10^6 person-rem) used in the study were: leukemia (20); lung (20); bone (10); liver (10); GI tract (20); breast (20); thyroid (5); and others (20). The whole population was assumed to be

outdoors during the passage of the plume. "Those individuals who potentially could receive doses from exposure to deposited activity sufficient to lead to early effects were assumed to be evacuated after 24 hours and thus received no further external exposure. At greater distances down-wind it was assumed that measures such as evacuation or decontamination would be undertaken to limit the exposure from deposited activity; the dose from deposited activity was limited to that accumulated in the first day if the exposure, when integrated over 50 years, would otherwise have exceeded 10 rad (low LET)."

Walter B. Murfin prepared in October 1979 a draft report entitled "A Sensitivity Study of the Reactor Safety Study Consequence Model" for the Probabilistic Analysis Staff of the U.S. Nuclear Regulatory Commission. The report is cited here with an added degree of caution because it has not yet been formally issued or published. Section 3.2.4 of Murfin's draft report was entitled "Influence of Dose-Response Relationships". Murfin concluded that the models for latent cancer fatalities due to chronic exposure were too complex to permit any simple determination or presentation of their sensitivity to changes in the dose-response parameters. Thus his analysis of the model for latent cancers from chronic exposure was very limited and is not discussed further here.

Although not strictly a sensitivity analysis, the data reported by Ian B. Wall et al. in NUREG-0340, *Overview of the Reactor Safety Study Consequence Model*, October 1977, is also useful in the identification of organs, pathways, and isotopes responsible for large fractions of the total health consequences.

6.3.2.3 Results of Studies Pertaining to Model Sensitivity

Early Effects

The work of Wall et al., Kelly, and Murfin indicates that dose to the bone marrow is responsible for almost all of the early deaths predicted to result from a reactor accident under the great majority of weather conditions. There are certain, relatively infrequent, circumstances (e.g., several hours of brisk winds with stability class A followed by a near calm or rain) in which the risk of death due to lung dose competes with the risk from bone marrow exposure.

In Kelly's sensitivity study the LD-50 for bone marrow irradiation was set at a lower bound of 300 rad and at an upper bound at 525 rad, the RSS level for supportive treatment. (The slopes of these curves were not indicated, but it is assumed they were similar to those of the RSS.) Lung irradiation LD-50's were taken as twice and half the base values given above. It was noted that sensitivity depended in part on the meteorological conditions, evacuation assumptions, etc.

Kelly noted that effect of changing the LD-50 for bone marrow irradiation was substantial:

"For releases from molten fuel alone the incidence of early death is determined almost solely from irradiation of the bone marrow which is relatively more highly irradiated compared to the lung than with releases in which fuel is vaporized; the sensitivity to variation in the dose-mortality relationship for lung irradiation is therefore minimal while that to the relationship for bone marrow irradiation is enhanced with the range of variation approaching an order of magnitude."

"If the upper bound dose-mortality relationship for bone marrow irradiation is considered representative of the possible influence of simple supportive medical treatment following irradiation, such treatment would appear to have the potential to reduce the incidence of early deaths by a factor of between 2 and 10, depending upon the composition and magnitude of the release."

Murfin found that variations of up to 50% in the "dose limits" for the lower large intestine wall made no difference in the probability of early fatality. Similarly, Murfin stated that unless the dose-mortality curve for the lung is "nonconservative" by a factor of 1.5, little change in the probability of death will be produced by correcting the curve. (Although Murfin's terminology, i.e., "nonconservative," is ambiguous it appears that he meant that unless the lung dose assumed to produce a stated probability of death was 1.5 (or more) times as high as the true dose required to produce that probability of death, the impact on the modeled total probability of death would be negligible.) In contrast, his analysis indicated that there were circumstances in which a 10% change in the total marrow "dose limits" could result in a twofold change in the predicted early fatalities. He also found that only in atypical circumstances with very low doses was the probability of early death insensitive to the total marrow "dose limits."

Wall et al. showed that for a BWR-1 release the total early dose to the marrow is approximately evenly divided among the three early exposure pathways: cloudshine, groundshine, and inhalation. This result was based upon the RSS HEM and the basic evacuation plan described in Section 2 of our report. Other emergency protective actions and other reactor accident types could lead to different dose apportionment. Further, they noted that the principle radionuclides responsible for the early bone marrow dose are Te-132, I-132, I-133, I-135, Sr-89, Ba-140 and their radioactive daughter products.

In section 13.5 of Appendix VI, WASH-1400, it is estimated that a reduction of the LD_{50/60} for deaths from bone marrow exposure from 510 rad to 340 rad could be expected to lead to a three- or fourfold increase in predicted early deaths.

Section 13.4, Appendix VI, WASH-1400 indicates that the primary cause of predicted early morbidity is respiratory impairment. Kelly noted that the dose-effect relationship for lung fibrosis was based on "very limited data." Further, it was noted, there was no allowance for the variation in degree of fibrosis.

In summary, it would appear that further work on the early effects model should concentrate on uncertainties in (and due to) the parameters of the dose-mortality function(s) for the bone marrow and the lung and on the parameters of the dose-morbidity function for the lung.

(As the predicted early health consequences are strongly dependent upon the efficacy presumed by the evacuation model, the reported sensitivities must also be viewed as strictly applicable for the particular evacuation efficacy which was studied.)

Latent Effects

Kelly's analysis indicated that for all meteorological conditions tested the vast majority of deaths due to an accident are expected to be latent cancer deaths:

"Fatal cancers make by far the greatest contribution to the total incidence of biological effects. The incidence of hereditary effects is significantly lower, and that of early deaths lower by some two orders of magnitude."

Based upon studies using the central estimate model for latent cancer deaths, Wall et al. note that for a PWR-1 release lung cancer accounts for most (43%) of all fatal cancers, with breast cancer second (27%), and leukemia third (12%), leaving only 18% to other sites. For a PWR-2 release breast cancer would account for most (39%) of all fatal cancers, leukemia second (18%), and lung cancer third (16%), leaving 27% due to other sites. In both cases groundshine was predicted to account for more than half of the total exposure. Wall et al. also noted that the central and lower bound (with a 10 rem threshold) models produce virtually identical estimates of predicted latent cancer deaths for severe accidents. This would seem to support the view that a linear-quadratic model with variable parameters could adequately reflect modeling uncertainty.

In his analysis of latent cancer deaths due to early exposure, Murfin demonstrated that, for circumstances leading to moderate (~50% of worst case) and low (~10% of worst case) doses, the total latent cancer deaths due to early exposure would be predicted to nearly double if the lung cancer constant of proportionality ($MRCON_2$) doubled. However, for these same circumstances, doubling the constants of proportionality applicable to the breast, marrow, or lower large intestine wall would be expected to increase total latent deaths due to early exposure by less than 5%. It must be noted that Murfin's analysis was based upon the upper bound proportional model for latent cancer deaths and that Murfin was considering the extremes of the distribution of doses. At these extreme points his analysis indicated that lung cancers were responsible for more than 84% of total cancers due to early exposure. In addition, it is not clear that Murfin adequately accounted for the influence of early fatalities on the population available for latent effects.

In summary it would appear that further study of the parameter sensitivity of the latent effects model should concentrate upon the uncertainties in (and due to) the parameters of the models for lung cancer, breast cancer, and possibly leukemia.

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SECTION 7

OVERVIEW

D.W. Cooper and J.S. Evans

7.1 INTRODUCTION

This section discusses evaluation of the model, using as a guide a model evaluation methodology presented elsewhere (Shaeffer, 1979). Comments from other reviewers of the Reactor Safety Study health effects model are summarized here (and given in more detail in Appendix 1), as are ours.

7.2 MODEL EVALUATION METHODOLOGY

Figure 7-1 (Shaeffer, 1979) presents a flow chart for evaluation of environmental assessment models. It includes six major tasks, described as: "1) model examination, 2) algorithm examination, 3) data evaluation, 4) sensitivity analyses, 5) validation studies and 6) code comparison studies. *Model examination* involves questioning whether anything fundamental was omitted in the initial conceptualization of the model. *Algorithm examination* attempts to determine whether appropriate numerical schemes have been adopted to represent the model in the form of a computer code. *Data evaluation* is intended to ascertain the quality and the quantity of the data available for use with the model. *Sensitivity analyses* are studies directed toward identifying those parameters which are most influential in determining model predictions. The identification of these key parameters enables priorities to be established for performing experiments. *Validation* refers to the attempt to determine the degree to which model predictions agree with field or laboratory measurements of those same quantities. *Code comparison* is the process of comparing the strengths and weaknesses of various codes designed to perform similar tasks."

Not all of these steps were attempted in our review of the health effects model, however.

7.2.1 Model Examination

Narrowly defined, the RSS HEM gives the responses to be expected for certain kinds of doses. If the doses are incorrect, then the predicted responses are likely to be incorrect also. Thus, one examines the model for radiation types or radionuclides incorrectly ignored, pathways mistakenly estimated, doses incorrectly calculated. In general, no major examples of such errors were found. Similarly, the errors of omission likely in dose-response modeling would be overlooking a type of effect or assuming prematurely that a type of response is negligible. Several examples of this kind of shortcoming are discussed above in the sections on early effects and on late effects; they are summarized below. (In some cases, more research will be needed to determine whether or not certain response types can be neglected.)

7.2.2 Algorithm Examination

The approximation of the dose-response curves in the RSS by piece-wise linear functions in the CRAC code called for this kind of examination. In general,

Figure 7-1. Flowchart of a methodology for evaluating environmental assessment models.

the uncertainty in the dose-response functions seemed sufficiently great that the approximations used in the code should not lead to equally large or larger additional uncertainty, or bias, although this is a matter of judgment.

7.2.3 Data Evaluation

The data which are needed for the model (besides release types, magnitudes, and probabilities) include meteorological information, demographic information, description of the movement of individuals potentially exposed, details about the structures protecting them from radiation, etc. Demographic information is readily available, as is information about the kind of housing and the degree of protection such structures are likely to offer. Prediction of the behavior of people is inherently more difficult, however. The weather at the time of an accident is extremely important in determining the magnitude and distribution of doses and can vary greatly.

Meteorological "data" can be joint frequency distributions of such important variables as wind speed, wind direction, mixing height, atmospheric stability class, and precipitation. Although these distributions may be well characterized for a site, the weather information at the time of an accident may or may not be available, and such weather variables may be somewhat different in regions of interest away from the site. Further, the dispersion model in which they are used works progressively less well as one deals with less and less uniform topography.

Evaluation of these data was deemed outside our scope. Data that were reviewed were those which might reveal systematic errors or uncertainties in the dose-response relationships, with emphasis on those data gathered since the the preparation of the RSS.

7.2.4 Sensitivity Analyses

A limited number of trials were made using the CRAC Code, to get preliminary information about the degree to which changes or uncertainties in the parameters of the dose-response relationships lead to changes or uncertainties in the model's predictions. These are discussed in Appendix 2. We believe more work of this type is warranted.

7.2.5 Validation

Although the models for dose prediction could be subjected to further validation through experiment, it is unlikely that the relationships between dose and response for many of the response types would be studied that way, except for observational studies in connection with future radiological accidents, should they occur. It has been argued that few if any populations have not yet been subjected to epidemiological scrutiny who would substantially improve our estimates of the low-dose (< 5 rem or < 5 rem/yr) dose-response relationships between radiation and cancer, for example (Dreyer et al., 1980). Work on atmospheric dispersion continues actively.

7.2.6 Code Comparison

The methodologies used by various countries and agencies for assessing the

risks from nuclear power are currently the subject of a major international study, but this kind of evaluation was not within our scope.

7.3 BRIEF SUMMARY OF PREVIOUS COMMENTS ON RSS HEM

The published comments on the RSS HEM are presented in summary form as Appendix 1 of this report. Here those summaries are discussed briefly. The comments are most easily understood as they relate to each of the submodels of the HEM: early effects (non-stochastic effects), late effects - somatic, and late effects - genetic. In addition to specific substantive comments on the model form and/or parameters there were many comments on the presentation of material in Appendix VI to WASH-1400. The presentation was criticized as being unclear and inconsistent.

7.3.1 Early Effects

Most of the comments on the early effects models centered on the specification of the dose-mortality function for bone marrow, particularly the choice of the $LD_{50/60}$. The Lewis Committee (Lewis et al., 1978) agreed with the use of the $LD_{50/60}$ of 510 rad (consistent with "supportive" treatment) to produce central estimates of early fatalities, but noted that the true value was quite uncertain and noted that the use of a $LD_{50/60}$ of 340 rad (consistent with "minimal" treatment) would yield about three times as many early fatalities. EPA (1976) stated that a more complete analysis and discussion of the factors leading to the choice of $LD_{50/60}$ values appropriate for minimal, supportive and heroic treatment was necessary. A group at ITRI, lead by Hahn, felt that while available data would support an LD_{50} for external exposure to gamma rays it was inadequate for development of an LD_{10} or LD_{50} applicable to exposure from internal low-LET radiation. Filipy at Battelle suggested that synergisms due to exposure of other organs deserved more attention. The Union of Concerned Scientists (UCS, 1977) agreed with the early-effects dose-response models chosen for the RSS, but noted that the environmental pathways and dosimetric models could be expected to underestimate the early doses systematically.

The models for early morbidity did not receive much criticism. However, the UCS felt that the definition of morbidity used in the study should be expanded to include effects such as hypothyroidism, suppression of menstruation, and certain respiratory effects. In addition the UCS called the attention of the RSS groups to the possibility of "life shortening" due to severe, but not immediately fatal, damage to organs.

7.3.2 Late Effects - Cancers

The comments on the model for cancer induction reflect concerns over the resolution of each of the major uncertainties in the interpretation of data on cancer induction. These include:

- a. shape of the dose-response function;
- b. absolute risk vs. relative risk interpretation of epidemiologic studies;
- c. effect of dose rate on cancer induction;

d. length of plateau period for expression of disease.

The EPA expressed the opinion that late health effects were underpredicted (by the RSS HEM central estimates) by a factor of between two and ten. This comment was based on the position that a lower-bound model should be based upon absolute risk, a thirty-year plateau, and dose reduction factors no smaller than 0.5. In this context the EPA sought clear analysis and explanation of the reasoning leading to the dose reduction factors used in the RSS HEM. A recent report by the NCRP (1980) supported the use of such risk reduction factors. An upper-bound acceptable to the EPA reviewers would be based on relative risk with a lifetime plateau. The UCS suggested that production of certain cancers would be underestimated by factors of 3 to 4 by the RSS HEM central model, and proposed that central estimates should be produced by a linear model without dose reduction factors. The Lewis Committee questioned the use of the absolute risk concept, concurred with the use of dose reduction factors (but could not agree on specific factors to be used), noted that the use of relative risks would increase the estimates of cancer production by a factor between 1.5 (30-year plateau) and 4 (lifetime plateau), and offered that overall uncertainties in modeling cancer production (thought by the Lewis Committee to be on the order of a factor of 5, definitely less than a factor of 10) had been underestimated by the RSS group.

7.3.3 Late Effects - Genetic

The UCS expressed the opinion that the model for genetic effects was adequate. However they claimed that RSS summary figures showed less than 1% of the true genetic effects. The EPA questioned the doubling dose used in the development of genetic risk factors, and sought clarification of the reasoning leading to these risk factors.

7.3.4 Effects on the Thyroid

Logically, thyroid effects would be expected to be included in the discussions of both early and late effects. However, the RSS HEM used a separate set of dosimetric and dose-response models for the thyroid, and several comments were directed to these models.

Both the Lewis Committee and the UCS questioned the dose effectiveness reduction factor of 0.1 (relative to external gamma rays) applied by the RSS HEM to the I-131 dose in the prediction of thyroid nodules. The EPA remarked that the thresholds used in the estimation of early thyroid effects appeared to be high by factors of between 2 and 2.5, and sought more clear documentation of the process leading to the development of the thyroid risk factors used in the RSS HEM.

7.3.5 Other Remarks

These remarks were also made:

- a. Protective actions which the public can take (e.g., potassium iodide tablets, use of "filter masks") should be discussed in WASH-1400. (UCS, 1977)

b. The effects on terrestrial, aquatic and atmospheric ecosystems were not addressed. (EPA, 1978)

7.4 SUMMARY OF OUR COMMENTS

7.4.1 Dosimetry

At dose levels greater than 200 rad, uncertainty in the dose can lead to great uncertainty in the prediction of the effects, given the extremely nonlinear response functions for mortality due to the bone marrow or lung syndromes. For the late effects, the fraction of the population affected will change much more nearly proportionately to dose.

Given correct air and ground concentrations of radionuclides, the RSS relationships for calculating external dose -- though not presented very clearly -- seem correct, considering the state of the art.

Prediction of dose due to material taken internally (inhaled or ingested) could readily be improved. The application of the model to children needs to be re-checked. Predicted inhalation dose will depend strongly on the assumptions about inhalation rate and the particle size distribution of the radioactive aerosol, both of which may need revision. The chemical form of the aerosol is important and uncertain. Assumptions made in calculating the dose to the gastrointestinal tract should be checked, especially the energy distribution of the beta radiation, the residence times in the portions of the GI tract, and the depth of tissue at which the dose rates should be calculated.

Rates for particle resuspension from surfaces are still quite uncertain. They have been shown to vary over many orders of magnitude. This uncertainty affects estimation of chronic dose, as do questions about the distribution of radionuclides within the soil.

For estimates of breast cancer induction, it would be preferable to use dose estimates for the breast, rather than a surrogate, and the same is true for evaluation of effects on developing fetuses.

Of the various aspects of the uptake of radioactive material by various organs, perhaps the uptake by the thyroid of radioiodine is most worthy of continued research.

7.4.2 Early Effects

Little new information on early effects, due to large (> 100 rad) doses of radiation, has been published since the RSS itself, which presented very competent evaluations of the risks of mortality due to damage to the bone marrow, the lung, and the gastro-intestinal tract.

Early effects will be quite sensitive to details of population exposure, because the dose-response functions have thresholds. Efforts at mitigation can make substantial differences (Aldrich et al., 1978). Details of evacuation, sheltering, relocation, and interdiction can be quite important in affecting outcomes.

The current reviews of the dosimetry data from Hiroshima and Nagasaki may also change the dose estimates in this high-dose regime. Analysis of these data may affect details of the dose-response functions chosen.

More work is warranted with regard to subgroups of the population which are especially sensitive, and the model might well need to be able to take such subgroups into account explicitly, rather than as part of population-averaged dose-response function.

The question whether heroic treatment could be applied needs reconsideration, given the difficulty of finding appropriate transplant donors. The availability of even supportive treatment can be expected to change with the scale of the accident and the demands on regional medical facilities.

Research showing a dose-rate effect for external radiation received within a few days may warrant changes in the model, tending to increase the LD_{50/60} values for the bone marrow syndrome fatalities.

New work on modeling the dose-response relationship for the lung syndrome and some new data indicate the RSS HEM may need revision in this regard.

There are some new data on gastro-intestinal tract exposures, generally in the radiation medicine literature, which should be considered in any revision.

Radiation anxiety was not considered and needs to be.

Illnesses needing to be incorporated into the calculations include hypothyroidism and bone marrow morbidity, and, perhaps, burns due to beta radiation.

Regarding damage to fetuses, information published near the publication date of the RSS may not have been fully incorporated into the model and deserve review. Birth defects need to be included in the computer code.

In general, the assumption that the various effects can be treated as independent of each other needs more justification.

Finally, the functional forms of the dose-response models need review. Perhaps a "hazard function" approach should be used for combining effects or an "index of harm" or yet another method for producing a meaningful aggregation.

7.4.3 Late Effects

The relationship between low levels of radiation and health effects is a matter of very active current research interest, and, in comparison with the study of early effects, much has been done since the publication of the RSS. Especially important is the BEIR III (NAS, 1980) report and the ongoing re-evaluations of the data from Hiroshima and Nagasaki.

The risk of leukemia now appears to persist for at least 25 years and the risk of other cancers continues essentially for life, so the plateaus used in the RSS HEM need reconsideration.

Any code revision might well include the enumeration of non-fatal as well as fatal cancers.

The mortality rate assumed for thyroid cancer seems unjustifiably high. Thyroid nodules, cancers, and deaths from thyroid cancers need to be included in the computer code.

The special sensitivities of certain sectors of the population may need to be taken into account.

Current estimates put the risk of genetic damage somewhat lower than that used in the HEM.

More information on dose-rate effects in cancer induction is available and should be considered in any revision (NCRP, 1980).

All three estimates of cancer mortality (upper, central, lower) should appear in a revised code.

If the model is to be used for safety analyses of other parts of the nuclear fuel cycle than light water reactors, doses from alpha particles and dose-response relationships for such doses will need to be incorporated.

Synergisms between radiation and other environmental hazards may be significant in special circumstances and deserve study.

Recent literature on "non-specific aging" should be taken into account to help determine whether there are such effects, beyond the life-shortening due to the effects explicitly enumerated in the model.

7.4.4 Computer Code Implementation

The new documentation associated with the revised computer code (CRAC-2) is easier to understand than the original. The CRAC code contains essentially all the dose-response models considered and then retained by the RSS group. Other codes incorporating health effects models of similar types are available as well, but we have not reviewed them in detail.

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APPENDIX 1

COMMENTS RELATING TO THE RSS HEALTH EFFECTS MODEL

Douglas W. Cooper

What follows is a summary of comments we have found in the literature and which pertain directly (or nearly so) to the final report of the RSS.

Environmental Protection Agency (1976)

EPA (1976) provided extensive commentary on the RSS final report, most of which involved the health effects model, the evacuation model, accident sequences and probabilities, and the consequences of release of materials to the environment.

"It appears that if late somatic health effects were adjusted in accordance with EPA's assessment of the numerical health risks, the estimates would increase by a factor of from 2 to 10. The potential change in the estimated early fatalities and injuries could not be determined by EPA from the information provided."

It is stressed that the major disagreement is in the health effects work, where EPA believed that the RSS should have used the BEIR (1972) estimates as the central estimates for the relationship between cancer induction and radiation dose.

It was recommended that the calculational procedures be put forward with a clear and concise presentation.

"As a supplement to the Reactor Safety Study report, further estimates should be made of the health effects from accidents using different upper bound, central and lower bound risk models.

"The Agency suggests that (a) lower bound estimates should be based on absolute risk, 30 year plateau, and dose rate effectiveness factors not smaller than 0.5; (b) central estimates should utilize an average of the absolute and relative risk estimates stated in the BEIR report by the National Academy of Sciences; (c) while upper bound estimates may require more speculation about additive and synergistic health hazards than is warranted at this time, some consideration of the sensitivity of the estimates to the use of a convex relationship appears justified, or, alternatively, the relative relative risk estimates in the BEIR report with a life risk plateau could be used as a surrogate."

"At the least, the Study should have provided a better analysis of the following problems:

proper categorization of the upper, central and lower bound somatic health risk estimates,

justification of the selected dose rate effectiveness factors (DREF) for chronic and acute bioeffects,

minimal, supportive and heroic treatment for the mitigation of acute effects as hypothesized in the report,

radiation bioeffects of the thyroid gland,

risk estimates for genetic disorders,

estimates of radiation doses that result in specific clinical health effects."

The critique is followed by further discussion, with many references to the health effects literature. The case for the linear, non-threshold dose-response model is made. The lower estimate should have been made from various concave upward models. The central-estimate dose rate effectiveness factors were too small, it is stated.

"Thyroid injury is an important aspect of a reactor accident. The associated risks may have been underestimated in the Reactor Safety Study. The stated thresholds for radiation thyroiditis (25,000 rem) and hypothyroidism post-exposure (20 rem) may be high by factors of 2.5 and 2, respectively." Thyroid injury is discussed at length.

"Estimates of genetic risks due to radiation accidents are not as clear cut as indicated in the Reactor Safety Study." The differential sensitivity of oogonia and spermatogonia is cited as contested. Mutation rates are being debated. A more accurate description of post-radiation recessive mutations is offered. Some factors related to mutation were not addressed, it is claimed. The doubling dose is a source of continued controversy.

"The Study should have specified SD values for respiratory impairment and hematopoietic failure expected after a reactor accident, if only in terms of ranges."

"To the extent that data are available, the following consequences should have been discussed:

(1) The synergistic and additive effects of other environmental pollutants for specific indicators of life shortening such as cancer, and

(2) A review of the extent of bioeffects anticipated in the terrestrial, aquatic and atmospheric ecosystems."

"...we recommend that the Reactor Safety Study group publish a technical report detailing the framework of the consequence calculations. The report should give all the important analytical relationships and their interconnections as well as complete documentation of the computer program details, so that any compromises and limitations introduced in the programming are also made clear."

Union of Concerned Scientists (1977)

The Union of Concerned Scientists (UCS) reviewed the entirety of the Reactor Safety Study (RSS), and we have excerpted and paraphrased below their

comments on the health effects model.

"The underlying RSS dose/response models for correlating *early fatalities* and genetic effects with radiation exposure appear valid (although they are not correctly applied by RSS because RSS radiation dispersion calculations lead to underestimates of the doses). RSS models used in assessing long-term consequences, however, appear to understate certain cancer fatalities by a factor of 3 to 4, and the nodules and cancers of the thyroid gland by a factor of 2.

"The RSS summary figures and discussion badly misrepresent the total health consequences of a major radioactive release. If properly presented, early illnesses should be anywhere from 10 times (largest accidents) to 6000 times (smallest accidents) the numbers displayed on the RSS risk consequence curves, as the result of hypothyroid cases alone. Similarly genetic effects displayed in RSS show less than 1% of the true totals. Cancer fatalities and thyroid nodules graphs show about 3% of the true totals."

"The long-term health consequences including cancers and genetic defects typically dwarf the prompt ones. Correction of the RSS risk results for understatement of long-term consequences shows that the largest accident considered could result in over 300,000 total fatalities. In general, nuclear risks are comparable to many other man-caused risks and for accidents with more than about 2000 fatalities may exceed the sum of all of them."

"The final RSS report underestimates or minimizes certain important health effects which would result from a major radioactive release. However, we agree with the RSS dose-effect models for early fatalities and for genetic effects. We also believe that the calculations of early illnesses are based on essentially correct dose-effect models, but that the numbers of early illnesses described in the RSS summary represent only small fractions of the totals likely to occur."

"There is no consideration of delayed life shortening in heavily irradiated survivors who may also have experienced an early illness."

"No consideration is given in the RSS to possible life-shortening effects that may stem from severe damage to organs (especially to bone, lung, and thyroid) *in utero* and in early childhood."

Lack of estimates of damage to fetal thyroids or of other developmental abnormalities in fetuses is also pointed out.

"The RSS fails to discuss the possible late deaths (life shortening) that might arise from impairment of lung function early in life or, more especially, the consequences over many years of multiple impairments, such as damage to thyroid plus lungs. The matter of life shortening clearly is relevant to how the public may view the risks of nuclear power."

"In our view, respiratory damage, hypothyroidism, and suppression of menstruation ought all to be considered as radiation illnesses, and the numbers of persons considered to be ill ought to be increased by a factor of approximately 10 in severe accidents to account for thyroid damage and for the fact that syndromes will be to some extent mutually exclusive."

"While the number of radiation victims among fetuses may be numerically small because they comprise only 1% of the population, their effect on the cost of damage suits may be major."

"While there is some evidence from animal studies for applying dose-effectiveness factors in cancer risk calculations, we believe that the case for doing so is insufficiently convincing at this time to warrant abandoning the simpler and more conservative linear dose-response model."

"The effect of using the linear response model...rather than the nonlinear model (RSS) would be to increase the calculated cancer risks by about three-fold."

"A point of possible contention concerns the conclusion in RSS that there is a ten-fold lower yield of thyroid nodules from iodine-131 irradiation than from external x-rays. There is indeed some evidence for this conclusion, but we find the data ambiguous and as yet rather unconvincing."

"Because the dose of iodine-131 would constitute at least half the total dose to thyroid in a reactor accident (RSS Figure VI 13-4), the effect of discounting iodine-131 by ten fold is to lower the expected number of thyroid nodules by a factor of about 2."

"Without explanation about delayed health effects, the risk comparison curves (RSS Figures 1-1 to 1-3) in the Executive Summary are totally misleading."

Criticism was raised about the choice of levels of dose chosen to determine whether interdiction would occur.

"The presentation should include both prompt and latent effects."

"The use of potassium iodide tablets (which can block radioactive iodine uptake by the thyroid), a mild laxative, and a filter mask are simple ways to diminish doses from internal emitters in the first day of an accident. Information about these and other measures ought to be widely known by the public."

U.S. House of Representatives (1977)

The U.S. House of Representatives subcommittee (1977) under Morris Udall produced "Observations on the Reactor Safety Study," which had few direct comments on the health effects model. It did outline the history of the study and of the Price-Anderson bill, which it stated were closely related.

"The most detailed criticism of the Study's technical details is that prepared by the Environmental Protection Agency and its contractor, Inter-mountain Technologies, Inc. (Hearings, pp. 221-355.)"

"A pervasive criticism of the Reactor Safety Study is its failure to emphasize that the greatest health hazard associated with nuclear powerplant accidents would be in the long-term effects...."

"...Therefore, the same accident which caused approximately 10 'early'

deaths would also cause approximately 8,000 'latent' but fatal cancers."

"...Dr. Wolfgang Panofsky...stated...'in terms of total fatalities caused by a potential accident, the latent effects potentially can cause several hundred times or so more deaths than the prompt effects."

Questions were raised about the adequacy of NRC's responses to criticisms of the Draft Report.

It was stated that Prof. Joel Yellin of MIT had published an extensive critique of the final report.

A news item in Technology Review (vol.79, January 1977, pp. 24-25) summarized Dr. Yellin's major criticism of the RSS, that it does not compare long-term deaths with those of other technologies: "Dr. Yellin notes [that] WASH-1400's authors estimated that, on the average, every prompt fatality from a nuclear accident is expected to be accompanied by about 670 latent cancer fatalities, spread over a period of 10 to 40 years." It reported that Richard Wilson (fall 1976 issue of Bell Journal) countered that long-term effects of coal combustion to produce power are much larger than those of nuclear.

Lewis Committee (1978)

The Risk Assessment Review Group report (Lewis, 1978) noted that the goal of the RSS was to make a realistic, rather than conservative, estimate of the risks due to nuclear power production. They agreed with the LD50/60 for supportive treatment and thought it to be a reasonable choice of condition to select. Some problems remained:

"One key problem is the possible inapplicability of the data that were used. For example, if a human is exposed not only to bone marrow dose from inhalation and ingestion, but also to external radiation, then the LD-50/60 might be significantly lower. "

"However, there seems to be a very wide range of uncertainty in opinion, with values cited in the range from perhaps 400 to 800 rads." "The use of the response function for only minimal intervention (LD-50/60 = 340 rads) would result in approximately three times as many fatalities."

"...the total number of accident-induced cancers over a given period will be greater in the relative than in the absolute risk model. The RSS analyses used the absolute-risk model, and therefore, the estimate of latent cancer fatalities from a reactor accident has been criticized by some as an underestimate." "The issue of the 'plateau period'...is tied in with the relative-risk/absolute-risk issue." Leukemia and bone cancer were noted as perhaps displaying plateau effects. (Thus use absolute risk model.) Others do not have plateaus. "If all cancers obeyed a relative-risk model, the predicted increase over the RSS estimates would be about a factor of 1.5 for a 30-year plateau period and a factor of 4 for a lifetime at risk." They noted that BEIR, NCRP, and UNSCEAR have "stated the possibility that radiation may be less carcinogenic at low doses and low dose rates." (RSS used dose-effectiveness factors except for breast cancer.) The committee found agreement that some such factors should be used, but not agreement on their values.

"It is our summary viewpoint that an upper limit of the full range of uncertainty on delayed dose-effect estimates of the order of a factor of 5, but certainly no more than a factor of 10, above the RSS values would encompass most of the range of scholarly disagreement in the biological community relative to carcinogenesis at low dose rates."

"The calculations in WASH-1400 used a dose effectiveness reduction factor of 0.1 for iodine -131 dose relative to X-rays....As a result this factor has been questioned and further review is necessary."

"...although all members of the RSS team contributed their full and honest efforts to the task, the spectrum represented by that team was not broad enough to encompass the full range of scholarly opinion on the subject. This led the RSS team to make estimates with a narrower range of stated 'uncertainty' that would otherwise have been the case."

Australian Atomic Energy Commission (1979)

The Australian Atomic Energy Commission (Gilbert, 1979) published a review of WASH-1400 which was generally quite favorable. The last two paragraphs of the abstract read as follows:

This review briefly describes the methods used in RSS, and indicates how the results were obtained. It then discusses the nature of these results, and the comparisons which RSS makes between them and other accident statistics. Finally, the criticisms which have been levelled at RSS are assessed, and the conclusions of a very recent US Nuclear Regulatory Commission review are included in an appendix.

RSS concluded that the risk due to reactor accidents is smaller than that associated with many other aspects of everyday life. While there are shortcomings in RSS, the author has little doubt that the above conclusion is valid.

The published comments on the RSS are outlined as follows:

When the draft RSS was made available, in 1974, 61 organizations and 25 individuals submitted comments for consideration before the final report was written. The major documented commentaries were prepared by:

The US Environmental Protection Agency [Ref. 4]; The US Atomic Energy Commission [Ref. 5]; The Union of Concerned Scientists combined with the Sierra Club [Ref. 6]; The University of California. Los Angeles [Ref. 7]; A Euratom Working Group at Ispra [Ref. 8];

and, in addition, the American Physical Society made its own independent review of reactor safety [Ref. 9] which contains a number of references to RSS. Staff of the Australian Atomic Energy Commission also made a detailed examination of the draft RSS.

It is fair to say that while there were many criticisms of detailed aspects of RSS, most of them were constructive. Most reviewers

regarded the draft RSS as a major pioneering achievement and supported its methods and its general conclusions even though some had major reservations about certain individual numerical results. The only exception to this general statement was the contribution of the Union of Concerned Scientists, whose major objections are considered below.

The Australian AEC report agreed with UCS that the Executive Summary presentation was inadequate, possibly misleading. It agreed that sabotage and design adequacy are important elements of reactor safety not covered by the report, but that safeguards against sabotage could have hardly been studied without breaching security and that design adequacy analysis would have been a monumental task. It agreed, too, that the report is hard to follow, though substantially better than the draft report. It disagreed with the remaining major points of the UCS criticism, and it cited concluding remarks from the UCS evaluation which indicated that the risks from a nuclear plant "could be within, and under pessimistic upper bound assumptions could exceed, the range of impact of a comparable coal plant." The Australian AEC report called the RSS "a major landmark in safety analysis." The references cited in its list of major critiques were:

4. Comments by the Environmental Protection Agency on RSS. EPA, Washington, DC, November 1974.
5. Review of RSS: Comments of the AEC Regulatory Staff. USAEC, Washington, DC, November 1974.
6. Preliminary Review of the AEC Reactor Safety Study: Joint Review Committee, Sierra Club - Union of Concerned Scientists, San Francisco - Cambridge, December 1974.
7. What Did WASH-1400 Prove? UCLA Course 239D, E.N. Cranier, December 1974.
8. Comments on the RSS WASH-1400, Euratom JRC Working Group, Ispra, January 1975.
9. Report to the American Physical Society by the Study Group on Light-Water Reactor Safety. Reviews of Modern Physics, Vol. 47, Supplement No.1. Summer 1975.
10. The Risks of Nuclear Power Reactors: A Review of the NRC Reactor Safety Study WASH-1400; Union of Concerned Scientists, Massachusetts, August 1977.

ITRI Consequence Model (Hahn, 1979)

Hahn (1979) presented the ITRI version of a model for "Early Mortality Estimates for Different Nuclear Accidents," which reviewed the available information on dose-response relationships and came to the following conclusions (which we quote), which were not directed at the RSS health effects model, but which clearly relate to it:

The LD50/30 for whole-body exposure of man to gamma rays is estimated to be about 300 rad. The value of 300 rad is the same as that reported in WASH-1400 for the dose that affects 50% of the sample exposed within 60 days (LD50/60), but is somewhat less than one would estimate from the dose-response curve for man reported in the NASA Life Science Data Book (Webb, 1962).

Presently available data for man are insufficient for estimating the D10 and D50 for early mortality from injury to bone for internal low-LET radiation sources.

Major Inadequacies in Presently Available Data

Presently available data for early death from radiation-induced injury to bone are insufficient for determining the following:

1. The radiation dose to bone marrow from internal low-LET radiation sources.
2. Appropriate dose-response relationships for early death injury to the hemopoietic system following exposure to internal high-LET radiation sources.
3. An appropriate method of predicting dose-response relationships for early death from injury to the hemopoietic system following exposure to multiple low- and high-LET radiation sources.
4. The influence of age at exposure on the dose response relationship for man for early death from radiation induced injury to the hemopoietic system.
5. Shape of dose-response relationships for external radiation doses in the region of low incidence of mortality (less than 5%) from radiation injury to the hemopoietic system.

In developing the simulation model, a number of research needs have been identified which must be filled to refine estimates of early mortality for different nuclear accidents. A summary of these needs is shown in Table IX-1 in relation to the type of accidental exposure. The most essential data which are needed fall in the following areas:

1. The combined effects on the lung of inhaled beta-emitting radionuclides of various solubilities which result in a complex retention pattern in the lung.
2. The combined effects on the lung of various inhaled alpha-emitting radionuclides which have different specific activities.
3. The combined effects on the lung of alpha-emitting and beta-emitting radionuclides inhaled at the same time.
4. The combined effects of inhaled beta-emitting radionuclides and whole-body irradiation.

5. The combined effects of inhaled soluble and insoluble forms of beta-emitting radionuclides where there is damage to both lung and bone marrow.

Further analyses of existing data are needed in several areas to complete certain aspects of estimating early mortality. These are:

1. The estimates of mortality from external whole-body irradiation.
2. The determination of corrections needed for wasted radiation dose in the lung.
3. The determination of accurate bone marrow doses in rat, dog and man so that comparable doses can be used in analysis.

Battelle Consequence Model (Filipy et al., 1980)

Comments offered by Filipy et al. (1980) included:

"We agree with the Reactor Safety Study (1) that damage to bone marrow is the primary cause of death from large radiation doses to the whole body. As a consequence, mortality predictions based on whole-body irradiation represent the upper limit of estimates of death from bone-marrow irradiation. We further concur, at this stage of our data investigation, that the estimated whole-body dose-response curve for minimal treatment (curve A of Figure VI 9-1 (1)) is appropriate for estimating percent mortality in 365 days....to apply to human populations receiving minimal-to -supportive treatment following irradiation."

"...the combined early mortality model...is useful only in predicting the consequences from internal dose to bone marrow and lung. If death were partially due to internal irradiation of other organ systems, the percent mortality calculated from this model would be underestimated."

"Relatively little is known, however, about multiple-organ effects...."

REFERENCES

Filipy, R.E. et al. A Mathematical Model for Predicting the Probability of Acute Mortality in Human Population Exposed to Accidentally Released Airborne Radionuclides. NUREG/CR-1261, available from Division of Technical Information and Document Control, U.S.N.R.C., Washington, D.C., 1980.

Gilbert, C.P. Nuclear Reactor Safety - A Review of the Rasmussen Report (WASH-1400). Australian Atomic Energy Commission Report AAEC/IP14, available from NTIS, Springfield, VA, 1979.

Hahn, F.F. Early Mortality Estimates for Different Nuclear Accidents. NUREG/CR-0774, available from NTIS, Springfield, VA, 1979.

Lewis, H.W. et al. Risk Assessment Review Group Report to the U.S. Nuclear Regulatory Commission. NUREG/CR-0400, available from N.T.I.S., 1978.

Union of Concerned Scientists. The Risks of Nuclear Power Reactors: A Review of

the NRC Reactor Safety Study WASH-1400 (NUREG-75/014). Union of Concerned Scientists, Cambridge, MA, 1977.

U.S. Environmental Protection Agency. Reactor Safety Study (WASH-1400): A Review of the Final Report. EPA-520/3-76-009 Environmental Protection Agency, Washington, D.C. 1976.

U.S. House of Representatives, Subcommittee on Interior and Insular Affairs. Observations on the Reactor Safety Study. U.S. Government Printing Office, Washington, D.C., 1977.

APPENDIX 2

CRAC CODE CALCULATIONS

J.S. Evans and D.G. Smith

A2.0 INTRODUCTION

The CRAC (Calculation of Reactor Accident Consequences) code developed in conjunction with the Reactor Safety Study was entered into the computing facilities at Environmental Research and Technology, Inc. (ERT) of Concord, Massachusetts and used to enhance our understanding of the implementation of the model. Several computer runs were made with the CRAC code to obtain typical and upper-estimate values of doses in the various distance intervals for a class PWR 2 accident with a 0.5-hour duration of release. Several simplifications were involved in these runs. Wind speed, direction, stability class and precipitation were all held constant throughout the entire length of time required for the plume to pass through the region. The upper-bound (RSS) linear dose-response model was used to predict latent cancer fatalities. Finally, the model was operated in a manner which gave results for five highly aggregated rings rather than the 34 rings more typically used. The dimensions of these rings and estimates of exposed population within the rings are given in Table A2-1.

In all of the discussion which follows these simplifications must be kept in mind. For example, there in reality may be cases in which doses to individuals quite close to the reactor are enough higher than the calculated dose at the midpoint of the first ring that the doses certain individuals receive are above thresholds for response while the calculated midpoint doses are not.

The CRAC code provides for various program "start codes". The first three sets of runs (A, B & C) were run with "start code 9". In theory this should have caused the meteorology to be constant throughout the five spatial rings. However, in practice, it appeared to induce rain in the intervals close to the reactor, but not throughout the entire area of interest. The predicted consequence values from these runs are suspect, and are not considered further in this analysis. Therefore, the next five sets of runs (D, E, F, G & H) were run with "start code 0". Under "start code 0" the user may provide different meteorology in each spatial ring. However, as discussed above we input identical meteorology for all five spatial rings. Runs D through H were set up in such a way that the outer radius of the fifth ring was 2000 mi. In these five runs any material remaining in the air at 500 mi was deposited uniformly over the interval from 500 mi to 2000 mi by setting the parameter LIRAIN=34. The population density in this outermost zone was set at 78 persons/sq. mi. Finally, runs F, G & H were repeated with the outer radius of the fifth interval set at 500 miles, by specifying LIRAIN=0.

The conditions used in each of the runs are summarized in Table A2-2. (Blank entries indicate that the values are the same as those in rows above.) The first (series) and second (run) columns are arbitrary codes assigned to give the runs unique identifiers. An asterisk following this code indicates that "start code" 9 was used in running the program. Double asterisks indicate that both start code 0 and 9 were used in different versions of this run. All other results were obtained using start code 0. There were rather large differences between

Table A2-1. Spatial Intervals for CRAC Runs

<u>Ring No.</u>	<u>Outer Radius (mi)</u>	<u>Area of Plume Coverage (sq. mi.)</u>		<u>Population Exposed Within Ring</u>	
		<u>Class 6 Stability</u>	<u>Class 4 Stability</u>	<u>Class 6 Stability</u>	<u>Class 4 Stability</u>
1	2.5	0.44	0.76	44	76
2	5.0	0.99	1.90	99	190
3	10.0	3.47	6.83	347	683
4	50.0	91.0	184.	9100	18400
5(a)	2000.0	102000.	209000.	7990000	16300000
(b)	500.0	7355.	14969.	735500	1496900

Note: Population estimates based upon 100 persons per sq. mi. within 500 mi. of the site and 78 persons per sq. mi. in the interval from 500 mi. to 2000 mi.

1 mi. = 1.6 km.

1 sq. mi. = 2.56 sq. km.

Table A2-2. Summary of CRAC Code Runs - Conditions (PWR 2, 0.5-hr release)

Index		Conditions					
Series	Run	Evacuation Velocity (m/s)	Delay (hr)	Wind (m/s)	Meteorology Wake	Stability	Rain
A	3*	2	0	1	0	6	0
	4*			5		4	
B	3*	0		1		6	
	4*			5		4	
C	3*	2	1	1		6	
	4**			5		4	
D	1			1	1	6	1
	2	0	0				
	3	2	1				0
	4	0	0				
E	1				0		
	2	2					
	3		1			4	2
	4						1
F	1	1.2	0	2			
	2						0
	3					6	
	4						1
G	1	0				4	
	2						0
	3					6	
	4						1
H	1	1.2	1			4	
	2						0
	3					6	
	4						1

Key:

* start code 9

** start code 9 and start code 0

wake effects: 0 = not incorporated, 1 = incorporated in calculations

stability class: standard Pasquill-Gifford classifications 1=A,
2=B, ...6=F

rain: 0 = no rain, 1 = rain, 2 = intense rain

Notes:

(1) Run E2 is identical to run A3 with the exception of start code.

(2) Run E1 is identical to run B3 with the exception of start code.

the C4 results depending upon which start code was used. The next column indicates the velocity, in m/s, of the evacuation (1 m/s is approximately 2 miles per hour). If evacuation occurred immediately, the delay (hours) is zero. The wind speed assumed is given (m/s) and whether the wake effect of the structure was (1) or was not (0) incorporated is indicated. Pasquill-Gifford stability classifications A through F are given as the numbers 1 through 6; C (3) and D(4) are most common, with F(6) being a very stable (little plume dispersion) condition that occurs at night and in the early morning, occasionally with rain. The presence of no rain (0), moderate rain (1), or intense rain (2) is indicated in the final column.

Typical daytime meteorological conditions would be class C or class D (3 or 4 numerically in our table) stability and winds of about 5 m/s. Rain would not occur more than a few percent of the time in most locales. Atypical evacuation would be no movement whatsoever. The atypical conditions have been used to identify some "upper-estimate" situations, but these are not truly "upper-bound," since one could always imagine a particularly unlikely and unfavorable set of conditions which would produce even more severe predicted consequences.

A2.1 RESULTS

The health consequences estimated by CRAC for each of these trial runs are summarized in Table A2-3. In all but one (G1) of these twenty-six runs, total latent fatalities exceed early fatalities. G1 involves no evacuation, a low (2 m/s) wind speed, stability 4 (D) and rain. In fact, only in seven of the runs are there any early fatalities. All seven of these scenarios involve rain and/or no evacuation. (It is possible that if finer spatial resolution had been used close to the reactor early fatalities would have been predicted to occur in other scenarios.) Typically, most of the latent cancer deaths are due to early exposure. However, there are three cases (F1, G1, and H1) in which this is not true. In these cases, which all involve rain and class 4 (D) stability, 75% or more of the latent deaths are due to chronic exposure. This simple description of example cases of predicted consequences reflects the possible variability in the importance of the various exposure modes.

From the twenty-six PWR 2 runs, eight were initially selected for more careful analysis. These eight were considered to be representative of the various combinations of early and late deaths obtained in the complete set of twenty-six runs. Due to the problems encountered with start code 9, results for run C4 have been deleted from all the tables which follow.

As noted earlier in this report, for each model run there are three basic sets of doses and predicted consequences. These are early deaths and injuries, latent deaths from early exposure, and latent deaths from chronic exposure. For these eight exemplary cases, the various doses (i.e., early, latent-early, latent-chronic) occurring at the midpoints of the sectors downwind from the releases are shown in Tables A2-4, A2-5 and A2-6, respectively. As the error and sensitivity analysis discussion in Section 6 of this report indicates, the dose received by typical individuals is a major determinant of not only the response but also the degree to which changes in or uncertainties about the dose-response functions would affect predictions and uncertainties about these predictions.

Table A2-3. Health Consequences Estimated by CRAC for 26 Scenarios Described in Table A2-2. (Numbers in parentheses are for distances out to 500 miles, all others are for distances to 2000 miles.) [Numbers in brackets for runs A3-C4 were obtained with start code 9 and are of questionable validity.]

Scenario	Deaths			Non-Fatal Injuries
	Early (n_e)	Latent-Early Exposure (n_{le})	Latent-Chronic Exposure (n_{lc})	
A3	[0]	[158]	[47]	[976]
A4	[0]	[156]	[98]	[1]
B3	[0]	[162]	[48]	[998]
B4	[0]	[183]	[100]	[66]
C3	[0]	[158]	[47]	[976]
C4	[0]	[162] 202	[98] 1338	[20] 140
D1	0	162	54	2040
D2	143	182	54	2240
D3	0	121	29	907
D4	0	125	30	925
E1	143(143)	197(198)	54(55)	2260(2260)
E2	0	178	54	2050
E3	75	82	91	45
E4	0	0	5	0
F1	0(0)	11(11)	95(96)	1(1)
F2	0(0)	172(173)	75(74)	343(374)
F3	0(0)	170(168)	51(51)	1830(1820)
F4	0(0)	258(222)	60(61)	3390(3340)
G1	173(171)	31(31)	96(97)	104(105)
G2	0(0)	233(176)	75(75)	425(375)
G3	0(0)	171(169)	52(52)	1830(1820)
G4	54(54)	243(342)	61(61)	3540(3560)
H1	33(32)	16(15)	94(96)	33(34)
H2	0(0)	228(174)	75(74)	425(374)
H3	0(0)	170(168)	54(51)	1830(1820)
H4	3	228(227)	61(61)	3380(3390)

Table A2-4. Early Doses (rad) to Individuals by Organ and Ring

		E1	G4	F4	G1	G2	H1	F1
	<u>Ring</u>							
Marrow	1	1920.00	1024.00	209.000	3494.00	21.260	514.000	71.4000
	2	641.00	417.00	27.900	514.00	24.680	70.700	1.2980
	3	261.00	207.00	27.900	70.70	41.160	1.244	1.2980
	4	149.00	196.00	199.000	3.38	74.170	3.382	3.4370
	5	.07	.06	.065	.01	.011	.011	.0118
GI Tract	1	1221.00	652.00	138.000	2218.00	13.820	326.000	47.3000
	2	406.00	265.00	27.300	326.00	20.180	45.100	.8070
	3	165.00	132.00	27.300	45.10	42.670	.665	.8070
	4	95.70	121.00	131.000	1.99	53.210	1.994	2.1360
	5	.02	.02	.017	.00	.003	.003	.0030
Lung	1	1571.00	838.00	242.000	2856.00	17.670	425.000	85.7000
	2	523.00	344.00	148.000	425.00	74.390	65.000	2.6280
	3	221.00	189.00	148.000	65.00	240.000	1.352	2.6280
	4	179.00	191.00	284.000	3.09	158.000	3.087	4.3630
	5	.03	.03	.029	.01	.005	.005	.0052

Table A2-5-A. Latent Individual Doses (rem) in First Time Interval (0-1 yr)
Due to Early Exposure

		E1	G4	F4	G1	G2	H1	F1
	<u>Ring</u>							
Marrow	1	568.00	1928.00	385.000	716.000	21.300	110.000	138.000
	2	1230.00	777.00	51.200	110.000	32.200	136.000	1.620
	3	491.00	380.00	51.200	136.000	69.400	.485	1.620
	4	151.00	198.00	207.000	3.410	80.600	3.410	3.550
	5	.07	.07	.067	.011	.011	.011	.011
Lung	1	2952.00	1578.00	382.000	585.000	17.700	838.000	139.000
	2	1003.00	638.00	160.000	838.000	77.800	118.000	2.800
	3	407.00	328.00	160.000	118.000	253.000	1.530	2.800
	4	179.00	192.00	169.000	3.090	158.000	3.090	4.360
	5	.03	.03	.029	.005	.005	.005	.005
Breast	1	3795.00	2028.00	402.000	751.000	22.700	116.000	144.000
	2	1289.00	817.00	49.800	116.000	32.600	142.000	1.520
	3	513.00	397.00	49.800	142.000	67.300	1.410	1.520
	4	157.00	203.00	211.000	3.430	80.800	3.430	3.540
	5	.05	.05	.053	.009	.009	.009	.009
Skeleton	1	3623.00	1935.00	388.000	718.000	21.400	111.000	140.000
	2	1235.00	781.00	55.200	111.000	34.000	137.000	1.670
	3	493.00	382.00	55.200	137.000	75.800	1.530	1.670
	4	156.00	201.00	212.000	3.470	83.900	3.470	3.610
	5	.06	.07	.066	.011	.011	.011	.012
Intestine	1	2293.00	1225.00	249.000	455.000	13.800	647.000	90.000
	2	778.00	494.00	41.900	647.000	24.900	86.900	1.010
	3	309.00	242.00	41.900	86.900	60.500	.837	1.010
	4	98.20	128.00	136.000	204.000	58.200	2.040	2.210
	5	.02	.02	.019	.003	.003	.003	.003
Other	1	3795.00	2028.00	402.000	752.000	22.700	116.000	144.000
	2	1289.00	817.00	49.800	116.000	32.600	142.000	1.520
	3	513.00	397.00	49.800	142.000	67.300	1.410	1.520
	4	157.00	203.00	211.000	3.430	80.800	3.430	3.540
	5	.05	.05	.053	.009	.009	.009	.009

Table A2-5-B. Latent Individual Doses (rem) in Second Time Interval
(1-10 yr) Due to Early Exposure

A2-3

		E1	G4	F4	G1	G2	H1	F1
	<u>Ring</u>							
Marrow	1	.0000	.00000	1.8300	.00000	.0002	.01030	.70300
	2	.0078	.00472	3.3200	.01030	1.3700	.01920	.04560
	3	.2320	.03290	3.3200	.01920	5.2600	.00528	.04560
	4	1.6600	.06200	3.3200	.00528	2.6300	.00528	.04560
	5	.0000	.00183	.0018	.00027	.0003	.00027	.00000
Lung	1	.0000	.00032	15.9000	.00055	.0016	1.51000	6.13000
	2	.0684	.79400	28.9000	1.51000	11.9000	2.12000	.39800
	3	2.0300	5.50000	28.9000	2.12000	45.9000	.14200	.39800
	4	14.4000	9.99000	28.9000	.14200	22.9000	.14200	.39800
	5	.0000	.00184	.0018	.00027	.0003	.00027	.00000
Breast	1	.0000	.00000	.5410	.00001	.0001	.01600	.20800
	2	.0023	.00783	.9830	.01600	.4050	.02730	.01350
	3	.0689	.05440	.9830	.02730	1.5600	.00580	.01350
	4	.4910	.10000	.9830	.00580	.7780	.00582	.01350
	5	.0000	.00183	.0018	.00027	.0003	.00027	.00000
Skeleton	1	.0000	.00007	5.7900	.00012	.0006	.32000	2.23000
	2	.0249	.16800	10.5000	.32000	4.3300	.45500	.14500
	3	.7380	1.16000	10.5000	.45500	16.7000	.03360	.14500
	4	5.2600	2.12000	10.5000	.03360	8.3400	.03360	.14500
	5	.0000	.00183	.0018	.00027	.0003	.00027	.00000
Intestine	1	.0000	.00000	.4620	.00000	.0000	.01200	.17800
	2	.0020	.00558	.8400	.01200	.3460	.02160	.01160
	3	.0589	.03880	.8400	.02160	1.3300	.00544	.01160
	4	.4200	.07230	.8400	.00544	.6650	.00544	.01160
	5	.0000	.00183	.0018	.00027	.0003	.00027	.00000
Other	1	.0000	.00000	.5410	.00001	.0001	.01600	.20800
	2	.0023	.00783	.9830	.01600	.4050	.02730	.01350
	3	.0689	.05440	.9830	.02730	1.5600	.00580	.01350
	4	.4910	.10000	.9830	.00580	.7780	.00582	.01350
	5	.0000	.00183	.0018	.00027	.0003	.00027	.00000

Table A2-6. Population Doses and Fraction of Cancer Deaths Attributable to Each Organ - in 1st Time Interval (0 to 1 yr) after Accident Due to Early Exposure

Population Doses from Early Exposure in First Time Interval (10 ⁶ person rem)						
Organ						
Run	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract*</u>	<u>Other**</u>
E1	1.7	2.0	1.9	1.9	1.2	1.9
G4	2.1	2.0	2.2	2.1	1.3	2.2
F4	2.0	1.6	2.0	2.0	1.3	2.0
G1	0.25	0.35	0.25	0.25	4.0	0.25
G2	1.6	3.1	1.6	1.6	1.1	1.6
H1	0.11	0.15	0.11	0.12	0.11	0.11
F1	0.094	0.10	0.091	0.096	0.053	0.091

Fraction of Deaths Due to Organ						
Organ						
Run	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract*</u>	<u>Other**</u>
E1	0.22	0.20	0.23	0.06	0.08	0.22
G4	0.24	0.18	0.23	0.06	0.07	0.22
F4	0.25	0.16	0.23	0.06	0.08	0.22
G1	0.08	0.09	0.08	0.02	0.66	0.07
G2	0.20	0.31	0.19	0.05	0.07	0.18
H1	0.22	0.23	0.20	0.06	0.11	0.19
F1	0.24	0.20	0.22	0.06	0.07	0.21

* including stomach and rest of alimentary canal.

** including pancreas but excluding thyroid.

Throughout the discussion which follows comments are made concerning parameter sensitivity and propagation of error. The comments must be interpreted with caution. They apply strictly to the consequences predicted by CRAC for seven specific sets of conditions for a PWR 2 release with 0.5-hour duration under the simplifying assumptions described earlier (i.e., proportional dose-mortality for cancer, use of highly aggregated spatial resolution (5 rings vs 34), etc.). In addition during the course of this work we were informed by our project officer and others at Sandia that there were many potential problems with using CRAC which might influence the exact numerical results. Most of the difficulties encountered in using the code stem from the inadequacy of documentation accompanying the code. For example, the most complete users manual available to us was a Draft Users Manual which bore no date or indication of authorship.

Early Effects (Deaths and "Injuries")

For the seven cases finally selected for detailed analysis the early doses to each of the three organs considered in modeling early effects are given in Table A2-4. As discussed in Section 2 of this report, these doses result from cloudshine, groundshine, and inhalation of materials from the passing cloud. The bone marrow dose includes cloudshine, groundshine, the first 7-day dose due to inhalation of materials from the cloud, and one-half of the dose received between the 8th and 30th-day due to inhalation of materials from the cloud. The lung dose includes contributions from cloudshine, groundshine, and the full 365-day dose due to inhalation of materials from the cloud. The GI tract dose includes contributions from cloudshine, groundshine and the 7-day dose due to inhalation of materials from the cloud. For all three organs early groundshine doses are truncated after 4 hours, 24 hours, or 7 days depending upon distance from the site and details of evacuation.

The number of early deaths for each of these cases is calculated by combining information on doses with information on dose-response. Figures 6-1 through 6-3 are the dose-response curves used for early effects in the RSS health effects model, as implemented in the CRAC code.

A careful review of Table A2-4, in light of these dose-response curves, indicates that for the eight exemplary cases, estimated lung doses at the midpoints of the spatial intervals are never above the 5000 rad threshold for early death. In fact, the highest lung dose of 2586 rad (case G1, ring 1) is only approximately 60% of the threshold dose. Therefore, it would seem that, for the conditions studied, moderate errors in the postulated lung dose-response function would have little effect on the predicted number of early fatalities.

Similarly, estimated doses to the lower large intestine at the midpoints of the spatial intervals are below the 2000 rad threshold for early death in all but one of these eight cases. In this case (G1, ring 1) the estimated dose of 2218 rad at the midpoint of ring 1 results in only a 0.07 probability of early death to each of the 76 people in the area of exposure within ring 1, or approximately 5 early deaths. (Note, however, that in this case the total marrow doses are so high in ring 1 that the entire population at risk would die anyway.) The second highest dose of 1221 rad (case E1, ring 1) is about 60% of the threshold. Thus, the calculated early fatalities would seem to be insensitive to small errors in the dose-response function for the gastrointestinal tract.

In contrast, for half of the exemplary cases, the estimated total marrow doses at the midpoints of the spatial intervals are substantial enough to produce appreciable probabilities of early death for individuals in ring 1, and in three of the cases, relatively large probabilities of death for those in rings 2 and 3 as well. For example, in case G1 the entire population at risk (76 persons) in ring 1 is modelled to receive bone marrow doses of approximately 3500 rems, nearly six times the 615 rem upper threshold dose thought to result in certain death. In addition, the 190 people at risk in ring 2 receive doses of about 510 rem, associated with a 0.5 probability of death. Adding the bone marrow deaths in ring 1 and ring 2 produces a total early death toll of 171 deaths, approximately equivalent to the 173 early deaths estimated by the code for case G1.

The results of this analysis of early fatalities are consistent with the conclusions of others that in the vast majority of cases bone marrow dose is responsible for most early fatalities. In order to ensure unbiased and precise estimates of early fatalities in any revision of the model, attention should be focused on the dose-mortality function for the bone marrow.

Until the computer code and the model are revised, efforts to estimate the uncertainty in early fatality estimates due to uncertainty in the health effects model given by CRAC should concentrate on parameter uncertainty in the bone marrow dose mortality function. To explore possible ways of examining model sensitivity and propagation of error, two mini-analyses were conducted using the bone marrow doses from four cases (G1, G4, H1 and E1). Of the seven cases under detailed consideration, these four were selected because they were predicted to yield appreciable numbers of early fatalities due to bone marrow dose.

Although the numerical results of the analyses are of limited value (both because of the small number of cases (4) examined and because of the crude spatial resolution (5 rings) used in our modeling), they indicated that: (a) the sensitivity coefficient, $\Delta n_e / \Delta LD_{50}$, (deaths/rad) was less than 1 in absolute value for small (10%) changes in the LD_{50} ; and (b) the estimated number of early deaths seems to be more sensitive to changes in the dose-mortality function which reduce its threshold dose than they are to changes which increase the threshold dose. This seems to result from the interaction between the rates at which doses decrease and population at risk increases as one moves away from the reactor site.

Latent Deaths from Early Exposure

The latent doses due to early exposure are calculated for ten time intervals following the accident: 0-1 year, 1-10 year, and ten year intervals thereafter. The total dose to any organ in the first time interval after the accident includes contributions from cloudshine, groundshine and inhalation of materials from the passing cloud. For all later time intervals the dose to an organ due to early exposure is simply the result of materials inhaled from the passing cloud. In Tables A2-5-A and A2-5-B the total latent doses due to early exposure to each organ in each ring are presented for the first (0-1 year) and second (1-10 year) time intervals following the accident.

Comparison of the values given in both tables indicates that in most of the seven cases studied the vast majority of the total latent dose to any organ from

early exposures occurs within the first time interval after the accident. For most all organs the doses received in all rings in the second time interval are less than 1% of the doses received in the first time interval. However, in three cases (F4, G2, F1) the second period doses to the lung and skeleton in some rings are as high as 10 to 25% of the first period doses. The reasons for this relationship between first and second interval doses are clear when the contributions of the various exposure pathways to the total doses are examined. In most all of cases studied, for all organs, the first interval dose is dominated by the contribution of groundshine, which does not contribute to the doses due to early exposure in later time intervals. However for some cases (e.g., F4, G2, F1) there are significant contributions to total first interval dose of the lungs and skeleton from materials inhaled from the passing cloud. This seems a bit puzzling for those cases (F4, F1) involving rain.

Since in these seven runs the first time interval doses seem to dominate the total dose due to early exposure, our more detailed investigations were limited to these first time interval doses. In Table A2-6 the total population doses to each organ and the fraction of total deaths attributable to each organ due to dose received in the first time interval following the accident from early exposure are presented. The entries in the table were found by summing the products of organ-specific dose and the exposed population of the ring for all five rings and converting these to deaths using the "upper bound" BEIR organ-specific constants of proportionality. The interesting feature(s) of this table are: (a) the twenty- to forty-fold variation in these eight runs in the organ-specific population doses, and (b) the constancy of the fractional contributions of various organs to total latent deaths due to early exposure. With the exception of run G1 we see a remarkable invariance in the relative importance of the various organs in the face of considerable variations in meteorology and evacuation. As noted previously the G1 conditions (no evacuation, a low wind (2 m/s), rain and class 4 stability) would be expected to be unlikely. Throughout the other seven runs we note that three organs (marrow, lung, and breast) are of about equal importance in determining the latent deaths from early exposure, and that they together account for approximately 65% of these deaths. These results are consistent with the results of Wall et al., which indicated that for PWR 2 accidents, breast cancer, leukemia and lung cancer were the leading causes of death, accounting together for 73% of cancer deaths.

As mentioned previously, these results must be interpreted quite cautiously. Our runs were all for a PWR 2 release with 0.5-hour duration, and many simplifications were made in running the code. In addition only eight runs have been discussed in detail. Nonetheless, the potential value of a thorough sensitivity and error analysis of health effects dose-response modelling should be clear, as should the connection between population dose and parameter sensitivity.

Latent Deaths from Chronic Exposure

The latent doses due to chronic exposure are calculated for ten time intervals following the accident: 0-1 year, 1-10 year, and ten-year time intervals thereafter. The exposure pathways which contribute to these doses are groundshine, inhalation of resuspended materials, and the four chronic ingestion pathways described in Section 2 of this report. In Tables A2-7-A and A2-7-B the total latent doses due to chronic exposure to each organ in each ring are

Table A2-7-A. Latent Individual Doses (rem) in Second Time Interval (1-10 yr)*
Due to Chronic Exposure

	Ring	E1	G4	F4	G1	G2	H1	F1
Marrow	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	12.4000	.000	.000
	3	.0000	.0000	.0000	70.600	21.2000	70.600	70.600
	4	21.3000	24.9000	24.9000	16.900	11.1000	16.900	16.900
	5	.0000	.0000	.0000	.000	.0000	.000	.000
Lung	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	11.7000	.000	.000
	3	.0000	.0000	.0000	40.200	13.0000	40.200	40.200
	4	13.1000	15.1000	15.1000	15.100	8.2000	15.100	15.100
	5	.0000	.0000	.0000	.000	.0000	.000	.000
Breast	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	10.2000	.000	.000
	3	.0000	.0000	.0000	44.100	15.0000	44.100	44.100
	4	15.1000	17.1000	17.1000	14.300	8.6000	14.300	14.300
	5	.0000	.0000	.0000	.000	.0000	.000	.000
Skeleton	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	18.0000	.000	.000
	3	.0000	.0000	.0000	122.000	33.2000	122.000	122.000
	4	26.4000	39.7000	39.7000	22.000	16.3000	22.000	22.000
	5	.0000	.0000	.0000	.000	.0000	.000	.000
Intestine	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	8.1900	.000	.000
	3	.0000	.0000	.0000	52.300	15.0000	52.300	52.300
	4	15.1000	17.8000	17.8000	15.100	7.4400	15.100	15.100
	5	.0000	.0000	.0000	.000	.0000	.000	.000
Other	1	.0000	.0000	.0000	.000	.0000	.000	.000
	2	.0000	.0000	.0000	.000	10.2000	.000	.000
	3	.0000	.0000	.0000	44.100	15.0000	44.100	44.100
	4	15.1000	17.1000	17.1000	14.300	8.6000	14.300	14.300
	5	.0000	.0000	.0000	.000	.0000	.000	.000

Table A2-7-B. Latent Individual Doses (rem) in Eighth Time Interval (61-70 yrs) Due to Chronic Exposure

	E1	G4	F4	G1	G2	H1	F1
	Ring						
Marrow	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	1.28000	.00000	.00000
	3	.00000	.00000	.00000	5.59000	5.59000	5.59000
	4	2.52000	2.82000	2.82000	1.09000	1.09000	1.09000
	5	.00000	.00000	.00000	.00000	.00000	.00000
Lung	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	1.05000	.00000	.00000
	3	.00000	.00000	.00000	4.93000	4.93000	4.93000
	4	2.09000	2.36000	2.36000	.90200	.90200	.90200
	5	.00000	.00000	.00000	.00000	.00000	.00000
Breast	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	1.28000	.00000	.00000
	3	.00000	.00000	.00000	5.53000	5.53000	5.53000
	4	2.51000	2.81000	2.81000	1.10000	1.10000	1.10000
	5	.00000	.00000	.00000	.00000	.00000	.00000
Skeleton	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	1.56000	.00000	.00000
	3	.00000	.00000	.00000	7.32000	7.32000	7.32000
	4	4.08000	3.32000	3.32000	1.35000	1.35000	1.35000
	5	.00000	.00000	.00000	.00000	.00000	.00000
Intestine	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	.75100	.00000	.00000
	3	.00000	.00000	.00000	3.24000	3.24000	3.24000
	4	1.47000	1.65000	1.65000	.72700	.64500	.64500
	5	.00000	.00000	.00000	.00000	.00000	.00000
Other	1	.00000	.00000	.00000	.00000	.00000	.00000
	2	.00000	.00000	.00000	1.28000	.00000	.00000
	3	.00000	.00000	.00000	5.53000	5.53000	5.53000
	4	2.51000	2.81000	2.81000	1.10000	1.10000	1.10000
	5	.00000	.00000	.00000	.00000	.00000	.00000

presented for the second (1-10 year) and eighth (61-70 year) time intervals. These time periods were chosen for review since the second time interval tended to be characterized by the highest doses of any of the ten time intervals and the doses in the eighth time interval were as low as those in any of the following intervals.

Several features of these tables are noteworthy. The impact of relocation of the population and interdiction of land and crops is dramatically evident, resulting in 0 rem doses in the first, second, and third rings in many cases. We also note that the $t=8$ doses are typically 10% to 15% of the $t=2$ doses, reflecting a much slower decrease in dose than was evident in the latent doses from early exposure. This would appear to be due to the domination of chronic doses by external exposures, and the domination of external exposures by Cs-137, with a half life of about 30 yr. Third, the equivalency of the organ- and ring-specific dose among several runs is notable. For example, the doses from runs G1, H1 and F1 are identical (as would be expected for three runs which differ only in evacuation). Finally, by comparing Table A2-6-A with Table A2-5-B, we see that for this second time period, with the exception of the areas of interdiction, the latent doses due to chronic exposure are larger than the latent doses due to early exposure. It would be expected that this relationship would hold for all of the later time intervals.

In Tables A2-8 and A2-9 the population doses and fraction of deaths attributable to each organ due to dose in the second and eighth time intervals respectively from chronic exposures are shown. Once again we see that of the six cancers considered, leukemia is the leading cause of death (roughly $1/4$ to $1/3$ of the cancer deaths), with breast cancer and cancer of "other" organs next, followed by lung cancer. Together these four organs are shown to account for over 80% of the estimated cancer deaths due to chronic exposure.

A2.3 OVERVIEW OF CRAC CALCULATIONS AND ANALYSES

In summary, the CRAC code was run to simulate the health impacts of a class PWR2 accident, with an 0.5-hour release duration, under twenty-six different sets of meteorological conditions and/or evacuation scenarios. For each run predicted early deaths and illnesses as well as chronic deaths were estimated and tabulated. Early and chronic radiation doses expected to be received at the midpoint of each of five spatial intervals surrounding the reactor were calculated and tabulated. The relevance of these doses for error and sensitivity analyses was highlighted. The contribution of each organ (or type of cancer) to the overall death toll, for eight of the twenty-six trial cases, was examined.

These sample CRAC calculations were a valuable part of our overall analysis. They gave us some insight into the relative contributions of various pathways and organs to the deaths estimated by CRAC. They allowed us to verify by comparison with hand calculations that the dose-response functions used in the code are the same dose-response which are described in the CRAC Manual. As we have stated many times in this appendix, the numerical results of our analyses are of limited value. They are not based upon a large enough set of circumstances to support broad generalization as to the importance of various organs or pathways. However, these initial analyses have convinced us of the complexity and variability of the matrix of doses given by the model. At the

Table A2-8

Population Doses from Chronic Exposure in Second Time
Interval (10^6 person-rem)

<u>Run</u>	<u>Organ</u>					
	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract</u>	<u>Other</u>
E1	0.19	0.12	0.14	0.24	0.14	0.14
G4	0.23	0.14	0.16	0.36	0.16	0.16
F4	0.23	0.14	0.16	0.36	0.16	0.16
G1	0.36	0.31	0.29	0.49	0.31	0.29
G2	0.22	0.16	0.17	0.33	0.15	0.17
H1	0.36	0.31	0.29	0.49	0.31	0.29
F1	0.36	0.31	0.29	0.49	0.31	0.29

Fraction of Deaths from Chronic Exposure in Second Time
Interval Attributable to Each Organ

<u>Run</u>	<u>Organ</u>					
	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract</u>	<u>Other</u>
E1	0.28	0.14	0.19	0.09	0.11	0.19
G4	0.29	0.14	0.19	0.11	0.10	0.18
F4	0.29	0.14	0.19	0.11	0.10	0.18
G1	0.26	0.17	0.19	0.09	0.11	0.19
G2	0.27	0.15	0.19	0.10	0.09	0.19
H1	0.26	0.17	0.19	0.09	0.11	0.19
F1	0.26	0.17	0.19	0.09	0.11	0.19

Table A2-9

Population Doses from Chronic Exposure in Eighth Time
Interval (10^6 person-rem)

<u>Run</u>	<u>Organ</u>					
	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract</u>	<u>Other</u>
E1	0.023	0.019	0.023	0.037	0.013	0.023
G4	0.026	0.021	0.026	0.030	0.015	0.026
F4	0.026	0.021	0.026	0.030	0.015	0.026
G1	0.024	0.020	0.024	0.030	0.014	0.024
G2	0.025	0.020	0.025	0.029	0.015	0.025
H1	0.024	0.020	0.024	0.030	0.014	0.024
F1	0.024	0.020	0.024	0.030	0.014	0.024

Fraction of Deaths from Chronic Exposure in Eighth Time
Interval Attributable to Each Organ

<u>Run</u>	<u>Organ</u>					
	<u>Marrow</u>	<u>Lung</u>	<u>Breast</u>	<u>Skeleton</u>	<u>GI Tract</u>	<u>Other</u>
E1	0.24	0.16	0.22	0.10	0.07	0.22
G4	0.25	0.16	0.23	0.07	0.07	0.22
F4	0.25	0.16	0.23	0.07	0.07	0.22
G1	0.24	0.16	0.23	0.08	0.07	0.22
G2	0.25	0.16	0.23	0.07	0.07	0.22
H1	0.24	0.16	0.23	0.08	0.07	0.22
F1	0.24	0.16	0.23	0.08	0.07	0.22

same time it seems clear that a thorough analysis of model sensitivity and error propagation should be conducted that would be of great value to those who are concerned with revision of the health effects dose response functions but who are not familiar with the intricacies of the code and the details of the methods for summarization of calculated consequences. This level of effort would seem consistent with the extensive sensitivity analyses which have already been conducted in the areas of evacuation and meteorological modelling.

APPENDIX 3

INTRODUCTION TO SENSITIVITY ANALYSIS

Given a function of several variables, such as $f(x,y)$, one wants to know the effect on f of changes in x or y or perhaps the contribution to errors (variance) in f due to errors (variance) in x or y . The answers to these questions can be determined by sensitivity analysis and error analysis, respectively. In some cases, sensitivity and propagation of error can be determined by manipulations performed on the relationship $f(x,y)$, but we will assume that we are generally interested in models of sufficient complexity that this is not feasible.

The strengths and weaknesses of error and sensitivity analysis have been presented by Gardner et al. (1981), who argued for error analysis as being more generally applicable and more relevant, especially when the question is the allocation of scarce resources to the improvement of parameters in models with the purpose of decreasing the uncertainty in their predictions.

A3.1 Sensitivity (Effect on Mean)

The sensitivity of $f(x,y)$ to small changes in x and y is just the partial derivative of f with respect to x or y . Evaluating such a derivative means choosing some value of x and of y around which to vary x and y . The choice is not trivial and a different choice may produce substantially different partial derivatives. To obtain more representative values of the partial derivatives, one can choose many x,y pairs, evaluate $f(x,y)$ for those pairs and then regress f on x and y by multiple regression. The plane thus formed has as its slopes the "best fit" values of the rate of change of $f(x,y)$ with respect to x and y over the range studied. If $f(x,y)$ has substantial curvature, this estimate may be quite misleading, however, obscuring the difference in the relationships of f and x and of f and y in various domains. The sensitivity analysis does show whether f typically increases with x or with y (through the sign of the coefficients).

A3.2 Error (Effect on Variance)

For small variances and no correlation between the variances the variance in f caused by variances of x and y can be obtained by multiplying the variance of x by the square of the partial derivative of f with respect to x and multiplying the variance of y by the square of the partial derivative of f with respect to y :

$$\text{var}(f) = f_x^2 \text{var}(x) + f_y^2 \text{var}(y) + \text{H.O.T.} \quad (\text{A3-1})$$

in which H.O.T. stands for higher-order terms. The standard deviation is just the square root of the variance. When one term becomes three or four times smaller than the other, reducing it further makes very little difference in the standard deviation of f . Again, rather than choosing a single pair of x and y values around which to calculate the partial derivatives, one can calculate many f,x,y triplets, then regress f on x and y . The coefficients are the sensitivity factors, but the regressions also give the fractional contributions of each of the variables to the variance, which is the error analysis.

The sensitivity analysis is informative in indicating how the mean value may

be expected to change with changes in the independent variables; the error analysis indicates how much each term contributes to the variance, a measure of the uncertainty.

A3.3 Elasticity

For correlating values which vary over orders of magnitude, engineers and economists, among others, have found it useful to use power function approximations, such as

$$f(x,y) = ax^b y^c \quad (\text{A3-2})$$

instead of linear approximations. To perform the correlation (regression), one then transforms to the logarithms of the variables:

$$\log(f) = \log(a) + b\log(x) + c\log(y) \quad (\text{A3-3})$$

and regresses $\log(f)$ on $\log(x)$ and $\log(y)$. The slopes of the regression, b and c , are the elasticities, the percentage change in f due to a one percent change in x or y :

$$b = d\log(f) / d\log(x) = (df/f) / (dx/x) \quad (\text{A3-4})$$

$$c = d\log(f) / d\log(y) = (df/f) / (dy/y) \quad (\text{A3-5})$$

and the regression program also indicates the contributions to the variance of $\log(f)$ due to the variances of $\log(x)$ and $\log(y)$.

A3.4 Discussion

In principle these methods seem quite straightforward. In practice there are several complications which must be addressed. These include:

1. Selection of ranges of input variables over which parametric sensitivity studies and error analyses should be conducted.
2. Determination of the "variance" due to uncertainty of each of the parameters of interest.
3. Methods of selection of points, within the ranges of interest, at which the partials will be evaluated. (For example, are purely random methods or stratified random methods, such as Latin hypercube, to be preferred?)
4. Choice of methods for summarizing and presenting sensitivities and uncertainties when substantial curvature of the partial derivative response surface is present.

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