Chapter 9: SUMMARY OF DOSE AND RISK ESTIMATES

9.1 Introduction

This chapter (Chapter 9) presents the summarized results of the calculated doses and risks for the facilities analyzed in Chapters 2 through 7 of Volume II. Also overall uncertainties in these estimates are discussed.

Four separate steps are involved in estimating the health impact of a specific source of radioactivity: (1) measurement of emissions of radionuclides to air from the source, (2) estimation of the radionuclide concentration and annual intake of radionuclides at various locations, (3) calculation of the estimated dose and risk resulting from a unit intake or unit concentration of radioactivity in the environment, and (4) a means of scaling the risk estimates to match the specific source. In EPA's analysis, each step is associated with a computer code that performs the necessary calculations; the relationship of these codes is illustrated in Figure A-1 (Addendum A).

EPA uses the AIRDOS-EPA code (Mo79, Ba81) to analyze radionuclide emissions into air from a specific source. The results of this analysis are estimates of air and ground surface radionclide concentrations, intake rates via inhalation of air, and ingestion of radioactivity via meat, milk, and fresh vegetables. Chapter 6 presents a description of the techniques used and their limitations. The atmospheric and terrestrial transport models used in the code, their implementation, and the applicability of the code to different types of emissions are described in detail in Mo79.

The computer code used to calculate dose and risk is RADRISK (Du84, Su81, Du80). RADRISK calculates the radiation dose and risk resulting from an annual unit, e.g., 1 pCi/y, intake of a given radionuclide or the risk resulting from external exposure to a unit, e.g., 1 pCi/m^3, 1 pCi/m^2, concentration of radionuclide in air or on ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the conditions associated with a specific source. The assessment of radiation doses is discussed in Chapter 7; Chapter 8 discusses estimating the risk of health effects.

Once the radionuclide intakes and concentrations are calculated for a specific source by means of the environmental transport code, it is necessary to scale the dose and risk values resulting from a unit intake or concentration to the intake and concentration values predicted by the

transport code. As shown in Figure A-1 (Addendum A), the DARTAB computer code (Be81) performs this step using RADRISK unit doses and risks and AIRDOS-EPA concentrations and intakes. DARTAB is independent of both the environmental transport code, e.g., AIRDOS-EPA, and the dosimetric and health effects code, e.g., RADRISK. This eliminates redundant dose/risk calculations and the need for extraneous coding to calculate doses and health impacts in each environmental transport code.

9.2 Doses and Risks for Specific Facilities

Tables 9.2-1 and 9.2-2 are summaries of the doses and risks to critical groups of individuals and populations in the vicinity of facilities that discharge radioactive emissions. Data for selected facilities from each category are presented in the order they are presented in Chapters 2 through 7 of Volume II.

These dose and risk values were estimated using the environmental transport codes of AIRDOS-EPA, the dose and risk tables of DARTAB and the risk estimates that compose the RADRISK code. More detailed information, including a description of the facility, the processes causing the emissions, estimates of rates of emission, and estimates of doses and risks that result to individuals and populations are found in the respective chapters of Volume II.

9.3 Overall Uncertainties

Although the doses and risks presented in Tables 9.2-1 and 9.2-2 seem well defined and sometimes given to more than one significant figure, there are considerable uncertainties that persist when trying to fix their exact value. The individual uncertainties in the components which lead to the results in Tables 9.2-1 and 9.2-2 have been previously discussed. Source term measurement errors were discussed in Chapter 4; possible errors introduced in evaluating movement from the source through various pathways were discussed in Chapter 6; variations which could be introduced in the calculation of doses and dose rates were evaluated in Chapter 7; finally, Chapter 8 discussed the potential errors that could be introduced in the risk calculations.

9.3.1 Emission and Pathway Uncertainties

Measurement of emissions from sources have been estimated in Chapter 4 to be valid within a factor of 1.4.

In the evaluation of pathways, the uncertainties in results predicted by the atmospheric dispersion models make the most significant contribution. As discussed in Section 6.2.3, the studies by Little (Li79) and Miller (Mi82) indicate that for average annual concentrations, an uncertainty of approximately a factor of 2 for locations within 10 km of the release could be expected. Inasmuch as nearby locations to releases are of greatest concern, this uncertainty value is the most appropriate.

Table 9.2-1. Doses and risks to nearby individuals

Facility	Tissue[a]	Dose rate (mrem/year)	Lifetime[b,c,d] Risk (deaths/10^6 persons)
DOE Facilities			
Feed Materials Production Center	lung	88	100 (100)
Oak Ridge Reservation	lung	50	100 (100)
Portsmouth Gaseous Diffusion Plant	bone surface	11	20 (20)
Savannah River Plant	thyroid	4.9	40 (20)
NRC Facilities			
Research and test reactors	average all organs	1	20 (8)
Accelerators[g]	average all organs	0.0001	0.002 (0.0008)
Radiopharmaceutical suppliers	thyroid	0.3	0.2 (0.1)
AFRRI[e]	average all organs	0.005	0.09 (0.04)
U.S. Army facility	spleen	0.03	0.4 (0.2)
U.S. Navy facilities	average all organs	0.02	0.3 (0.1)
Radiation source manufacturers	average all organs	0.2	4 (2)
Coal Fired Boilers			
Utility boilers (rural)	bone surface	5	30 (10)
Industrial boilers	bone surface	0.4	0.6 (0.5)
Uranium Mines			
Ground level release (at 2000 meters)	lung	No dose available	10,000 (5,000)[f]
Plume rise release (at 2000 meters)	lung	No dose available	1,000 (500)[f]
Phosphate Industry			
Drying and grinding	bone surface	15	10
Wet process fertilizer	bone surface	2	2
Elemental phosphorus			
Pocatello, Idaho	lung	290	500
Soda Springs, Idaho	lung	610	1000
Mineral Extraction Industry			
Aluminum reduction plant	kidney	1.2	0.8
Copper smelter	lung	0.2	0.3
Zinc smelter	bone surface	0.02	0.02
Lead smelter	lung	4.8	8

(a) Organ with highest annual dose.
(b) Risk is that due to the total exposure not just that due to highest organ. This value represents the excess cancers in a lifetime for organ dose rates shown at offsite points of highest risk.
(c) The risk estimates in parentheses include a dose rate reduction factor of 2.5 for low-LET radiation, as described in Chapter 8 (Volume I) of this report.
(d) Risks are expressed per million population; for individual risks multiply each value by 10^{-6}.
(e) Armed Forces Radiobiology Research Institute.
(f) The values in the first column are based on BEIR-3 (NAS80), NRPB (NRPB82), and EPA models (Co78, K184, Mo79); the values in parentheses are based on UNSCEAR (UN82) and ICRP (Of80) risk estimates (see Chapter 8, Volume I).
(g) 6 MEV Van de Graaf

Table 9.2-2. Doses and risks to regional population

Facility	Organ(a)	Dose rate (pers-rem/year)	Risk(b,c) (Fatal cancers/year)
DOE Facilities			
Feed Materials Production Center	lung	440	0.01 (0.01)
Oak Ridge Reservation	lung	212	0.008 (0.006)
Portsmouth Gaseous Diffusion Plant	bone surface	35	<0.001
Savannah River Plant	thyroid	120	0.03 (0.01)
NRC Facilities			
Research and test reactors	average all organs	340	0.1 (0.04)
Accelerators(f)	average all organs	0.0006	<0.001
Radiopharmaceutical suppliers	thyroid	3	<0.001
AFRRI(d)	average all organs	~~0.09~~ 0.004	<0.001
U.S. Army facility	spleen	~~0.1~~ 0.01	<0.001
U.S. Navy facilities	average all organs	0.09	<0.001
Radiation source manufacturers	average all organs	8	0.002 (<0.001)
Coal Fired Boilers			
Utility boilers (rural)	bone surface	140	0.005 (0.003)
Industrial boilers	bone surface	90	0.003
Uranium Mines	lung	~~0.04~~ No dose available	0.06 (0.02)(e)
Phosphate Industry			
Drying and grinding	bone surface	110	0.001
Wet process fertilizer	bone surface	41	0.0005
Elemental phosphorus			
Pocatello, Idaho	lung	1170	0.03
Soda Springs, Idaho	lung	750	0.02
Mineral Extraction Industry			
Aluminum reduction plant	kidney	4.1	<0.001
Copper smelter	lung	0.95	<0.001
Zinc smelter	bone surface	2.5	<0.001
Lead smelter	lung	69	0.002

(a) Organs with highest annual dose.
(b) Fatal cancers in regional population per year of operation of facility for population exposure rate shown.
(c) The risk estimates in parentheses include a dose rate reduction factor of 2.5 for low-LET radiation, as described in Chapter 8 (Volume I) of this report.
(d) Armed Forces Radiobiology Research Institute.
(e) The values in the first column are based on BEIR-3 (NAS80), NRPB (NRPB82), and EPA models (Co78, E184, Mo79); the values in parentheses are based on UNSCEAR (UN82) and ICRP (Of80) risk estimates (see Chapter 8, Volume I).
(f) 6 MEV Van de Graaf

9.3.2 Dose Uncertainties

As discussed in Chapter 7 and summarized in Section 7.7, dose uncertainties are much less than would be implied by sensitivity analyses of maximum ranges of variables. The large dose ranges possible because of variation in individual characteristics must be modified by consideration of the narrower ranges indicated by studies of real populations; the dose range resulting from age dependence appears to be small for lifetime exposures, and the range resulting from experimental error is negligible by comparison. Based on these observations, it is reasonable to estimate that EPA's doses calculated on the basis of unit intakes or unit concentrations should be accurate within a factor of three or four. Much of the "uncertainty" in the dose calculation is not caused by parameter error but reflects real differences in individual characteristics within the general population. Therefore, the uncertainty in the dose estimates cannot be dissociated from specification of the segment of the population to be protected.

9.3.3 Risk Uncertainties

The uncertainties in estimating risk have been discussed in Chapter 8. Table 8.5-1 ranks and estimates the degree of uncertainty introduced by various sources in estimating the risk of cancer. The uncertainties listed in Table 8.5-1 are largely independent of each other and therefore unlikely to be correlated in sign. Their root mean square sum is about 300 percent, indicating the expectation that calculated risks would be within a factor of three or so of the true value. (This result is likely to be somewhat low because it does not include consideration of the uncertainty introduced by the bias in the A-bomb dosimetry or by the constrained regression analysis used by the BEIR-3 Committee.)

9.3.4 Overall Uncertainty

As indicated in the previous discussion, the individual uncertainties which combine to provide a basis for the overall uncertainty in risk evaluation are the following:

- Emission estimates are valid within a factor of 1.4*
- Air concentration estimates are valid within a factor of 2
- Dose calculations should be valid within a factor of 3 or 4
- Risk calculations should be valid within a factor of 3.

If these uncertainty estimates are independent and uncorrelated and can reasonably be considered to estimate the 2σ fractile of a log normal

---

*If the nominal value is multiplied or divided by the factor to give a range, the true value is expected to be within that range.

distribution, then the overall uncertainty in EPA's risk estimation can be estimated as a factor of about 7*. That is the maximum expected variation would range from about 15 percent to 700 percent of the nominal value.

The various uncertainties, however, may not be uncorrelated or independent. In this case, the overall uncertainty is likely to be less than predicted by the above procedure.

EPA concludes that risk estimates in this Background Information Document are accurate within a factor of 10. This estimate of uncertainty is believed representative of state-of-the-art procedures for estimating risks due to airborne radionuclide emissions.

---

*$\exp\{[(\ln^2(1.4) + \ln^2(2) + \ln^2(4) + \ln^2(3)]^{1/2}\}$.

REFERENCES

Ba81 Baes C. F. III, and R. D. Sharp, A Directory of Parameters Used in a Series of Assessment Applications of the AIRDOS-EPA and DARTAB Computer Codes, ORNL-5720, Oak Ridge National Laboratory, Oak Ridge, Tenn., March 1981.

Be81 Begovich C. L., Eckerman K. F., Schlatter E. C., Ohr S. Y., and Chester R. O., DARTAB: A program to combine airborne radionuclide environmental exposure data with dosimetric and health effects data to generate tabulation of predicted impacts, ORNL/5692, Oak Ridge National Laboratory, Tenn., August 1981.

Co78 Cook J. R., Bunger B., and Barrick M. K., A Computer Code for Cohort Analysis of Increased Risks of Death (CAIRD), EPA 520/4-78-012, 1978.

Du80 Dunning D. E. Jr., Leggett R. W., and Yalcintas M. G., A Combined Methodology for Estimating Dose Rates and Health Effects from Exposure to Radioactive Pollutants, ORNL/TN-7105, 1980.

Du84 Dunning D. E. Jr., Leggett R. W., and Sullivan R. E., An Assessment of Health Risk from Radiation Exposures, Health Phys., 46(5):1035-1051, 1984.

El84 Ellett W. H., RADRISK/BEIR-3, Part I: Basis for EPA Radiation Risk Estimates, to be published.

Li79 Little C. A. and Miller C. W., The Uncertainty Associated with Selected Environmental Transport Models, ORNL-5528, Oak Ridge National Laboratory, Oak Ridge Tennessee, November 1979.

Mi82 Miller, C. W. and Little C. A., A Review of Uncertainty Estimates Associated with Models for Assessing the Impact of Breeder Radioactivity Releases, ORNL-5832, Oak Ridge National Laboratory, Oak Ridge, Tennessee, August 1982.

Mo79 Moore R. E., Baes C. F. III, McDowell-Boyer L. M., Watson A. P., Hoffman F. O., Pleasant J. C., and Miller C. W., AIRDOS-EPA: A Computerized Methodology for Estimating Environmental Concentrations and Dose to Man from Airborne Releases of Radionuclides, EPA 520/1-79-009, EPA Office of Radiation Programs, Washington, D.C., December 1979.

NAS80 National Academy of Sciences - National Research Council, The Effects on Population of Exposure to Low Levels of Ionizing Radiation, Committee on the Biological Effects of Ionizing Radiation, Washington, D.C., 1980.

NRPB82 National Radiological Protection Board, Gut Uptake Factors for Plutonium, Americium and Curium, NRPB-R129, 1982.

Of80 Oftedal P. and Searle A. G., An Overall Genetic Risk Assessment for Radiological Protection Purposes, J. Med. Genetics, 17, 15-20, 1980.

Su81 Sullivan R. E., Nelson N. S., Ellett W. H., Dunning D. E. Jr., Leggett R. W., Yalcintas M. G., and Eckerman K. F., Estimates of Health Risk from Exposure to Radioactive Pollutants, ORNL/TM-7745, 1981.

UN82 United Nations Scientific Committee on the Effects of Atomic Radiation, Ionizing Radiation: Sources and Biological Effects, 1982 Report to the General Assembly, Sales No. E.82.IX.8, United Nations, New York, 1982.

ADDENDUM A: COMPUTER CODES USED BY EPA TO ASSESS DOSES FROM RADIATION EXPOSURE

A.1 Introduction

This addendum (to Chapter 7) provides a brief overview of the computer codes used by the Environmental Protection Agency (EPA) to assess the health risk from radiation exposures. It describes how the basic dose calculations are performed. Comprehensive descriptions of the various parts of this methodology have been published in a series of reports by the Oak Ridge National Laboratory and the Environmental Protection Agency (Du84, Be81, Ba81, Mo79). The risk estimates in current use are described in Chapter 8 and reflect the change from the BEIR-1 report (NAS72) to the BEIR-3 report (NAS80).

Three separate steps are required to estimate the health impact of a specific source of radioactivity: (1) estimate at various locations the radionuclide concentration and annual intake of radionuclides resulting from specific sources of radioactivity in the environment, (2) calculate the estimated dose and risk resulting from a unit intake or unit concentration of radioactivity in the environment, and (3) use a means of scaling the risk estimates to match the specific source. In EPA's analysis, each step is associated with a computer code that performs the necessary calculations, as illustrated in Figure A-1.

A.2 Overview of the EPA Analysis

The computer code used to calculate dose and risk is RADRISK (Du84, Su81, Du80). RADRISK calculates the radiation dose and risk resulting from an annual unit intake of a given radionuclide or the risk resulting from external exposure to a unit concentration of radionuclide in air or on ground surface. Since both dose and risk models are linear, the unit dose and risk results can then be scaled to reflect the exposure associated with a specific source.

As outlined in Chapter 7, estimates of the annual dose rate to organs and tissues of interest are calculated using, primarily, models recommended by the International Commission on Radiological Protection (ICRP79, ICRP80). Because EPA usually considers lifetime exposures to a general population, these dose rates are used in conjunction with a life table analysis of the increased risk of cancer resulting from radiation (Co78). This analysis, described in Addendum B, takes account of both competing risks and the age of the population at risk.

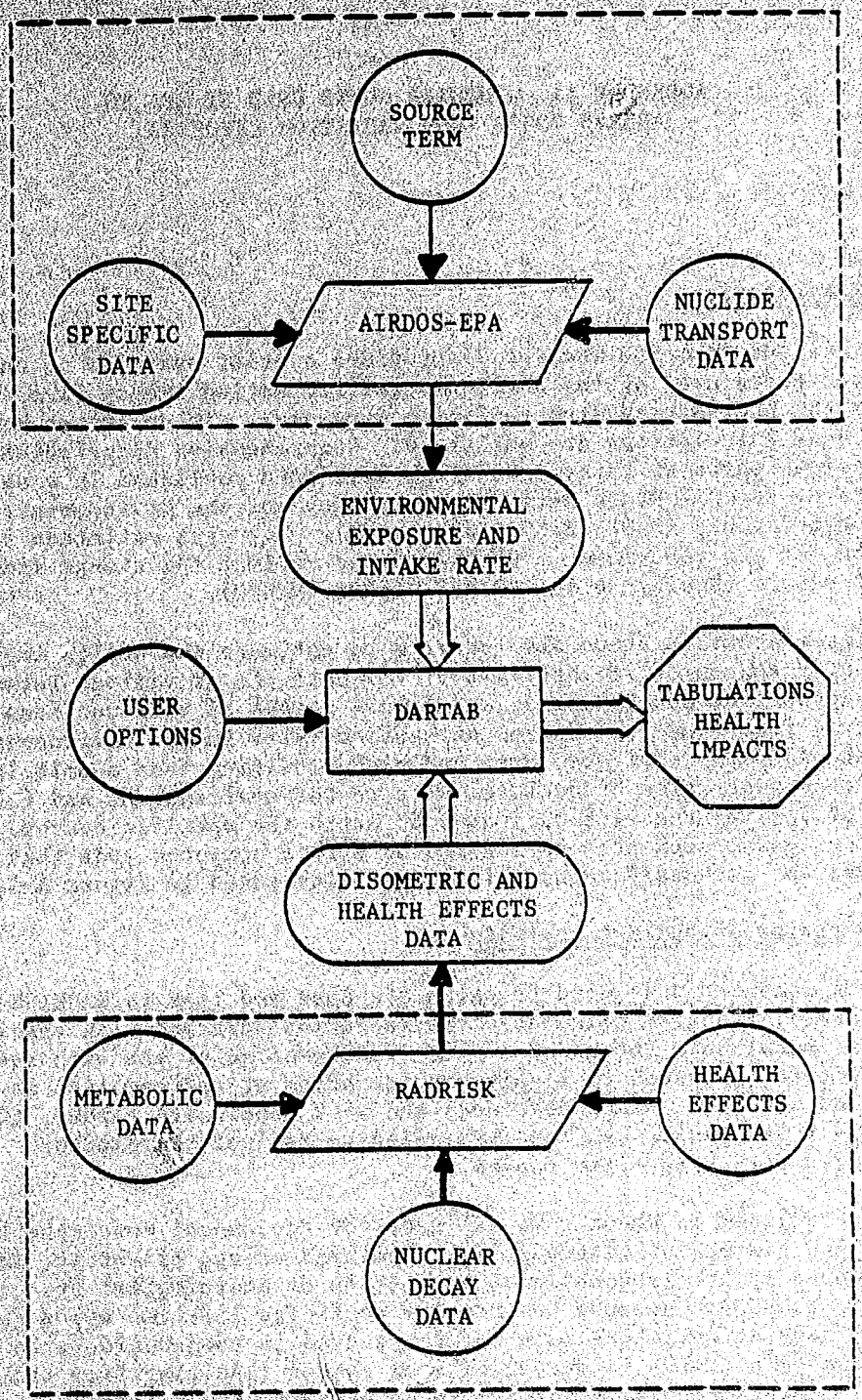

Figure A-1. Assessment of radiological health impacts.

Various computer codes are available to predict how radionuclides are transported through environmental pathways. As noted in Figure A-1, EPA uses the AIRDOS-EPA code (Mo79, Ba81) to analyze the transport of radionuclide emissions into air from a specific source. The results of this analysis are estimates at various distances from the source of air and ground surface radionuclide concentrations, intake rates via inhalation of air, and ingestion of radioactivity via meat, milk, and fresh vegetables. The atmospheric and terrestrial transport models used in the code, their implementation, and the applicability of the code to different types of emissions are described in Chapter 6.

A.3 Dose Rates from Internal Exposures

Internal exposures occur when radioactive material is inhaled or ingested. The RADRISK code implements contemporary dosimetric models to estimate the dose rates at various times to specified reference organs in the body from inhaled or ingested radionuclides. The dosimetric methods in RADRISK are adapted from those of the INREM II code (Ki78), based primarily on models recommended by the International Commission on Radiological Protection (ICRP79). The principal qualitative difference is that RADRISK computes dose rates to specified organs separately for high and low linear energy transfer (LET) radiations, whereas INREM II calculates the committed dose equivalent to specified organs. The time-dependent dose rates are used in the life table calculations of RADRISK.

In RADRISK, the direct intake of each nuclide is treated as a separate case. For chains, the ingrowth and dynamics of daughters in the body after intake of a parent radionuclide are considered explicitly in the calculation of dose rate. Consideration is also taken of different metabolic properties of the various radionuclides in a decay chain.

The dose rate $\dot{D}_i(X,t)$ to target organ X at time t due to radionuclide i ($1 \le i \le N$) residing in organs $Y_1, Y_2, \ldots, Y_m$ is a measure of the energy deposited annually in a given mass of tissue as a result of radioactive decay, and is computed as:

$$\dot{D}_i(X;t) = \sum_{k=1}^{m} \dot{D}_i(X \leftarrow Y_k;t) \qquad \text{(A-1)}$$

where

$$\dot{D}_i(X \leftarrow Y_k;t) = S_i(X \leftarrow Y_k) A_{ik}(t) \quad , \qquad \text{(A-2)}$$

$A_{ik}(t)$ = activity of radionuclide i in organ Y_k at time t measured from the initial intake of i into the body,

$S_i(X \leftarrow Y_k)$ = average dose rate to target organ X per unit activity of the radionuclide i uniformly distributed in source organ Y_k (Sn74, Du80).

The summation is taken over all source organs Y. Implicit in the definitions is the assumption of uniform distribution of activity of radionuclide i in each source organ, as is the assumption of averaging the dose rate over the mass of the target organ. Although estimates of dose to an organ include contributions from activity distributed throughout the body (for penetrating radiations), activity within that organ generally contributes the principal component of dose [i.e., $D_i(X \leftarrow X;t)$ is the principal component of $D_i(X;t)$].

The time rate of change of activity in the body is modeled by a system of ordinary differential equations, with each equation describing the rate of change of activity in a conceptual compartment of the body. For radionuclides that are part of a decay series there may be formation of radioactive daughters in a given compartment that have different chemical and physical properties from those of the parent. Unlike the models given in ICRP80, the specific metabolic properties of the daughter are evaluated when they differ from those of the parent. This refinement is under active consideration by ICRP experts. In almost all cases, doses to soft tissues calculated on this basis differ only slightly, if at all, from ICRP80 dose estimates, but the difference is large for some radionuclides when the parent is incorporated into bone, for example lead-210. For this radionuclide the ICRP80 model has been used without any modifications.

The pathways in the body by which activity is assumed to move were illustrated in Chapter 7. Except for radon daughters, which are considered separately, inhaled activity is assumed to be originally deposited in the lungs (distributed among the nasal-pharyngeal, tracheobronchial, and pulmonary regions), whereas ingested activity is originally deposited in the stomach. From the lungs, activity may be absorbed by the bloodstream or migrate to the stomach. Activity in the stomach may proceed through the small intestine, upper large intestine, and lower large intestine; activity may be absorbed by the bloodstream from any of these four segments, although only absorption from the small intestine is considered in this study.

The activity, $A_{i\ell k}(t)$, of nuclide i in organ k may be divided among several "pools" or "compartments", denoted here by the subscript ℓ. Each differential equation describing the rate of change of activity within a compartment is a special case of the equation:

$$\dot{A}_{i\ell k} = -(\lambda_i^R + \lambda_{i\ell k}^B)A_{i\ell k} + c_{i\ell k}(\lambda_i^R \sum_{j=1}^{i-1} B_{ij} \sum_{r=1}^{L_{jk}} A_{jr} + P_{ik}), \quad \ell = 1, \ldots, \ell_{ik} \qquad (A\text{-}3)$$

where

$\dot{A}_{i\ell k}$ = activity of radionuclide i in compartment ℓ of organ k,

$L_{i\ell k}$ = number of exponential terms in the retention function for nuclide i in organ k,

B_{ij} = branching ratio of nuclide j to nuclide i,

λ_i^R = rate coefficient ($time^{-1}$) for radiological decay of nuclide i,

$\lambda_{i\ell k}^B$ = rate coefficient ($time^{-1}$) for biological removal of nuclide i from compartment ℓ of organ k,

$c_{i\ell k}$ = fractional coefficient for nuclide i in the ℓ-th compartment of organ k,

P_{ik} = inflow rate of nuclide i into organ k.

If the inflow rate P_{ik} remains constant, the equations may be solved explicitly for $A_{ik}(t)$ as described by Killough, Dunning, and Pleasant (Ki78). In many cases the inflow into a compartment will not be a constant rate over a long period of time. To handle this problem, the time interval over which solution of the activity equation is desired (e.g., 110 years) is divided into 1-year subintervals. The inflow rate on each subinterval is then taken to be that constant value which would yield the total activity flowing out of the preceding compartment(s) during the same subinterval.

The model used in RADRISK for particulate deposition and retention in the respiratory tract is the ICRP task group lung model (Mo66, ICRP72). In this model, shown in Chapter 7, there are four major regions: the naso-pharyngeal, tracheobronchial, pulmonary, and lymphatic tissues. A fraction of the inhaled activity is initially deposited in each of the naso-pharyngeal, tracheobronchial, and pulmonary regions. The material clears from the lung to the blood and the gastrointestinal tract, also as shown in Chapter 7. Deposition and clearance of inspired particulates in the lung are controlled by the particle size and solubility classification.

The size distribution of the particles is specified by the activity median aerodynamic diameter (AMAD); in this document, all particulates are assumed to have an AMAD equal to 1.0 micron unless otherwise stated. The model employs three solubility classes, based on the chemical properties of the nuclide; classes D, W, and Y correspond to rapid (days), intermediate (weeks), and slow (years) clearance, respectively, of material deposited in the respiratory passages. Inhaled nonreactive, i.e., noble, gases are handled as a special case.

Movement of activity through the gastrointestinal (GI) tract is simulated with a catenary model, consisting of four segments: stomach, small intestine, upper large intestine, and lower large intestine. Exponential outflow of activity from each segment into the next or out of the system is assumed. Outflow rate constants are calculated from the transit times of Eve (Ev66). Although absorption may occur from any combination of the four segments, only activity absorbed into the blood from the small intestine is normally considered; the fractional absorption from the small intestine into the blood is traditionally denoted f_1.

Activity absorbed by the blood from the GI or respiratory tract is assumed to be distributed immediately to systemic organs. The distribution of activity to these organs is specified by fractional uptake coefficients. The list of organs in which activity is explicitly distributed (herein termed source organs) is element-dependent, and may include such organs as bone or liver where sufficient metabolic data are available. This list is complemented by an additional source region denoted as OTHER, which accounts for that systemic activity not distributed among the explicit source organs; uniform distribution of this remaining activity within OTHER is assumed.

Radioactive material that enters an organ may be removed by both radioactive decay and biological removal processes. For each source organ, the fraction of the initial activity remaining at any time after intake is described by a retention function consisting of one or more exponentially decaying terms.

The metabolic models and parameters employed in the present study have been described by Sullivan et al. (Su81). In most cases, the models are similar or identical to those recently recommended by the ICRP (ICRP79, ICRP80, ICRP81). However, some of this work was performed prior to the publication of these documents, so that differences in model parameters do exist for some radionuclides (Su81). In particular, parameter values that are thought to be more representative of metabolism following low-level environmental exposures, rather than occupational exposures, have been used in this analysis [e.g., f_1=0.2 for uranium in the environment (ICRP79, NAS83)]. For transuranic isotopes, metabolic parameters from the Proposed Guidance on Dose Limits for Persons Exposed to Transuranium Elements in the General Environment (EPA77), related comments (EPA78), and from the National Radiological Protection Board (NRPB82), have been used rather than those from ICRP80. These parameters are listed in Table A-1.

The EPA values were recommended by U.S. experts on transuranic element metabolism at Battelle Pacific Northwest Laboratory (EPA78). The recently adopted National Radiation Protection Board f_1 values for transuranics in the general environment are closer to those values proposed by EPA in 1977 than those currently advocated by ICRP for occupational exposures. Use of these larger f_1 values increases the estimated dose and risk from ingestion of transuranic materials but has little effect on doses following inhalation.

ADDENDUM B: MECHANICS OF THE LIFE TABLE IMPLEMENTATION OF THE RISK ESTIMATES

B.1 Introduction

This addendum describes the mechanics of the life table implementation of the risk estimates derived in Chapter 8. The calculation is performed as an integral part of the RADRISK code, described in Chapter 7, since time dependent organ dose rates are used.

B.2 Life Table Analysis to Estimate the Risk of Excess Cancer

Radiation effects can be classified as stochastic or nonstochastic (NAS80, ICRP77). For stochastic effects, the probability of occurrence of the effect, as opposed to the severity, is a function of dose; induction of cancer, for example, is considered a stochastic effect. Nonstochastic effects are those health effects for which the severity of the effect is a function of dose; examples of nonstochastic effects include cell killing, suppression of cell division, cataracts, and nonmalignant skin damage.

At the low levels of radiation exposure attributed to radionuclides in the environment, the principal health detriment is the induction of cancers (solid tumors and leukemia), and the expression, in later generations, of genetic effects. In order to estimate these effects, instantaneous dose rates for each organ at specified times are sent to a subroutine adaptation of CAIRD (Co78) contained in the RADRISK code. This subroutine uses annual doses derived from the transmitted dose rates to estimate the number of incremental fatalities in the cohort due to radiation-induced cancer in the reference organ. The calculation of incremental fatalities is based on estimated annual incremental risks, computed from annual doses to the organ, together with radiation risk factors such as those given in the 1980 NAS report BEIR-3 (NAS80). Derivation of the risk factors in current use is discussed in Chapter 8.

An important feature of this methodology is the use of actuarial life tables to account for the time dependence of the radiation insult and to allow for competing risks of death in the estimation of risk due to radiation exposure. A life table consists of data describing age-specific mortality rates from all causes of death for a given population. This information is derived from data obtained on actual mortality rates in a real population; mortality data for the U.S. population during the years 1969-1971 (HEW75) are used throughout this study.

The use of life tables in studies of risk due to low-level radiation exposure is important because of the time delay inherent in radiation risk. After a radiation dose is received, there is a minimum induction period of several years (latency period) before a cancer is clinically observed. Following the latency period, the probability of occurrence of a cancer during a given year is assumed to be constant for a specified period, called a plateau period. The length of both the latency and plateau periods depends upon the type of cancer.

During or after radiation exposure, a potential cancer victim may experience years of life in which he is continually exposed to risk of death from causes other than incremental radiation exposure. Hence, some individuals will be lost from the population due to competing causes of death, and are not potential victims of incremental radiation-induced cancer.

It is assumed that each member of the hypothetical cohort is exposed to a specified activity of a given radionuclide. In this analysis each member of the cohort annually inhales or ingests 1 pCi of the nuclide, or is exposed to a constant external concentration of 1 pCi/cm^3 in air or 1 pCi/cm^2 on ground surfaces. Since the models used in RADRISK are linear, these results may be scaled to evaluate other exposure conditions. The cohort consists of an initial population of 100,000 persons, all of whom are simultaneously liveborn. In the scenario employed here, the radiation exposure is assumed to begin at birth and continue throughout the entire lifetime of each individual.

No member of the cohort lives more than 110 years. The span from 0 to 110 years is divided into nine age intervals, and dose rates to specified organs at the midpoints of the age intervals are used as estimates of the annual dose during the age interval. For a given organ, the incremental probability of death due to radiation-induced cancer is estimated for each year using radiation risk factors and the calculated doses during that year and relevant preceding years. The incremental probabilities of death are used in conjunction with the actuarial life tables to estimate the incremental number of radiation-induced deaths each year.

The estimation of the number of premature deaths proceeds in the following manner. At the beginning of each year, m, there is a probability P^N of dying during that year from nonradiological causes, as calculated from the life table data, and an estimated incremental probability P^R of dying during that year due to radiation-induced cancer of the given organ. In general, for the m-th year, the calculations are:

$M(m)$ = total number of deaths in cohort during year m,
$= [P^N(m) + P^R(m)] \times N(m)$

$Q(m)$ = incremental number of deaths during year m due to radiation-induced cancer of a given organ,
$= P^R(m) \times N(m)$

N(m+1) = number of survivors at the beginning of year m + 1
= N(m) - M(m)
(N(1)=100,000).

p^R is assumed to be small relative to p^N, an assumption which is reasonable only for low-level exposures (Bu81), such as those considered here. The total number of incremental deaths for the cohort is then obtained by summing Q(m) over all organs for 110 years.

In addition to providing an estimate of the incremental number of deaths, the life table methodology can be used to estimate the total number of years of life lost to those dying of radiation-induced cancer, the average number of years of life lost per incremental mortality, and the decrease in the population's life expectancy. The total number of years of life lost to those dying of radiation-induced cancer is computed as the difference between the total number of years of life lived by the cohort assuming no incremental radiation risk, and the total number of years of life lived by the same cohort assuming the incremental risk from radiation. The decrease in the population's life expectancy can be calculated as the total years of life lost divided by the original cohort size (N(1)=100,000).

Either absolute or relative risk factors can be used. Absolute risk factors, given in terms of deaths per unit dose, are based on the assumption that there is some absolute number of deaths in a population exposed at a given age per unit of dose. Relative risk factors, the percentage increase in the ambient cancer death rate per unit dose, are based on the assumption that the annual rate of radiation-induced excess cancer deaths, due to a specific type of cancer, is proportional to the ambient rate of occurrence of fatal cancers of that type. Either the absolute or the relative risk factor is assumed to apply uniformly during a plateau period, beginning at the end of the latent period.

The estimates of incremental deaths in the cohort from chronic exposure are identically those which are obtained if a corresponding stationary population (i.e., a population in which equal numbers of persons are born and die in each year) is subjected to an acute radiation dose of the same magnitude. Since the total persons years lived by the cohort in this study is approximately 7.07 million, the estimates of incremental mortality in the cohort from chronic irradiation also apply to a one year dose of the same magnitude to a population of this size, age distribution, and age-specific mortality rates. More precise life table estimates for a specific population can be obtained by altering the structure of the cohort to reflect the age distribution of a particular population at risk.

B.3 Risk Analysis Methodology

Risk estimates in current use at EPA are based on the 1980 report (BEIR-3) of the National Academy of Sciences Advisory Committee on the Biological Effects of Ionizing Radiation (NAS80). The form of these risk estimates is, to some extent, dictated by practical considerations,

e.g., a desire to limit the number of cases which must be processed for each environmental analysis and a need to conform to limitations of the computer codes in use. For example, rather than analyze male and female populations separately, the risk estimates have been merged for use with the general population; rather than perform both an absolute and a relative risk calculation, average values have been used.

The derivation of the risk estimates from the BEIR-3 report is presented in Chapter 8. A brief outline of the general procedure is summarized below. Tables referenced from Chapter V of NAS80 are designated by a V prefix.

(1) The total number of premature cancer fatalities from lifetime exposure to 1 rad per year of low LET radiation is constrained to be equal to the arithmetic average (280 per million person rad) of the absolute and relative risk values (158 and 403) given in Table V-25 of the BEIR-3 report (NAS80) for the L-L and L-L models for leukemia and solid cancers respectively.

(2) For cancers other than leukemia and bone cancer, the age and sex specific incidence estimates given in Table V-14 were multiplied by the mortality/incidence ratios of Table V-15 and processed through the life table code at constant, lifetime dose rates of 1 rad per year. The resulting deaths are averaged, using the male/female birth ratio, and proportioned for deaths due to cancer in a specific organ as described in Chapter 8. These proportional risks are then used to allocate the organ risks among the (235.5) deaths per million person rad remaining after the 44.5 leukemia and bone cancer fatalities (Table V-17) are subtracted from the arithmetic average of 280 given in Table V-25.

(3) The RADRISK code calculates dose rates for high- and low-LET radiations independently. A quality factor of 20 has been applied to all alpha doses (ICRP77) to obtain the organ dose equivalent rates in rem per year. The derivation of the proportional organ risks and mortality coefficients for alpha particles are based on the dose in rads as described in Chapter 8, Table 8-6.

A typical environmental analysis requires that a large number of radionuclides and multiple exposure modes be considered. The RADRISK code has been used to obtain estimates of cancer risk for intakes of approximately 200 radionuclides and external exposures by approximately 500 radionuclides. For each radionuclide and exposure mode we assume that each member of a cohort of 100,000 persons is exposed to a constant radionuclide intake of 1 pCi/year, or a concentraton of 1 pCi/cc-year for air immersion, or of 1 pCi/cm^2-year from the ground surface, until they die or are 110 years old, the maximum cohort. The mean life span of the cohort population is 70.7 years, a result obtained from 1970 age specific mortality rates. The calculated dose rates and mortality coefficients described in the preceding sections are then processed through the life table subroutine of the RADRISK code to obtain lifetime risk estimates. At the low levels of contamination normally encountered in the environment, the life table population is not appreciably

perturbed by the excess radiation deaths calculated and, since both the dose and risk models are linear, these unit exposure results may be scaled to reflect excess cancers due to the radionuclide concentrations predicted in the analysis of a specific source.

As noted in the discussion of the life table analysis, risk estimates for chronic irradiation of the cohort may also be applied to a stationary population having the same age-specific mortality rates as the 1970 U.S. population. That is, since the stationary population is formed by superposition of all age groups in the cohort, each age group corresponds to a segment of the stationary population with the total population equal to the sum of all the age groups. Therefore, the number of excess fatal cancers calculated for lifetime exposure of the cohort at a constant dose rate would be numerically equal to that calculated for the stationary population exposed to an annual dose of the same magnitude. Thus, the risk estimates may be reported as a lifetime risk (the cohort interpretation) or as the risk ensuing from an annual exposure to the stationary population. This equivalence is particularly useful in analyzing acute population exposures. For example, estimates for a stationary population exposed to annual doses which vary from year to year may be obtained by summing the results of a series of cohort calculations at various annual dose rates.

REFERENCES

Co78 Cook J. R., Bunger B., and Barrick M. K, A Computer Code for Cohort Analysis of Increased Risks of Death (CAIRD), EPA 520/4-78-012, 1978.

Bu81 Bunger B. M., Cook J. R., and Barrick M. K., Life Table Methodology for Evaluating Radiation Risk: An Application Based on Occupational Exposures, Health Physics 40, 439-455.

HEW75 U.S. Department of Health, Education, and Welfare, 1975, "U.S. Decennial Life Tables for 1969-1971, Vol. 1., No. 1.," DHEW Publication No. (HRA) 75-1150, Public Health Service, Health Resources Administration, National Center for Health Statistics, Rockville, Maryland.

ICRP77 International Commission on Radiological Protection, 1977, Recommendations of the International Commission on Radiological Protection, ICRP, Vol. 1, No. 1, Pergamon Press, 1977.

NAS80 National Academy of Sciences - National Research Council, 1980, The Effects on Population of Exposure to Low Levels of Ionizing Radiation, Committee on the Biological Effects of Ionizing Radiation, Washington, D.C.

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2	3. RECIPIENT'S ACCESSION NO.
520/1-84-022-1		

4. TITLE AND SUBTITLE	5. REPORT DATE
Radionuclides. Background Information Document for Final Rules. Volume 1	October 1984
	6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
Office of Radiation Programs, PEI Associates, Batelle Columbus Laboratories & Kilkelly Associates	PN 3642-2

9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
PEI Associates, Inc. 11499 Chester Road Cincinnati, Ohio 45246-0100	11. CONTRACT/GRANT NO. 68-02-3878 Work Assignment No. 2

12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
U.S. Environmental Protection Agency Office of Radiation Programs Washington, D.C. 20460	Final 14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

Project Officer: Charles Robbins (703) 557-8977

16. ABSTRACT

On December 27, 1979, EPA published a notice in the Federal Register listing radionuclides as hazardous air pollutants under Section 112 of the Clean Air Act. On April 6, 1983, EPA published a notice in the Federal Register proposing standards for radionuclide emissions from four sources: 1) DOE facilities, 2) NRC-licensed facilities and non-DOE Federal facilities, 3) underground uranium mines and 4) elemental phosphorus plants. This Background Information Document supports the Agency's final actions on radionuclides. It presents an integrated risk assessment that provides the basis for the proposed standards. Volume I addresses historical and current regulatory programs and strategies, hazard identifications (health effects), radionuclide emissions, reduction of dose and risk, movement of radionuclides through environmental pathways, radiation dosimetry, and estimating the risk of health effects resulting from radionuclide air emissions.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Air Pollution	National Emission Standards for Hazardous Air Pollutants (NESHAPS)	13B
Radioactive isotopes	Radionuclides	18B
Radiobiology	Health Physics	06R
Uranium	Underground Mining	08G
Phosphate Industry	Elemental Phosphorus Plants	08G

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS (This Report)	21. NO. OF PAGES
Unlimited	Unclassified	273
	20. SECURITY CLASS (This page) Unclassified	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE