WASH—1400 App-6
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Reactor Safety Study

An Assessment of Accident Risks in U.S. Commercial Nuclear Power Plants

Appendix VI

United States Nuclear Regulatory Commission

October 1975

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CALCULATION OF REACTOR ACCIDENT CONSEQUENCES

APPENDIX VI
to
REACTOR SAFETY STUDY

U.S. NUCLEAR REGULATORY COMMISSION
OCTOBER 1975

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Section I

Introduction

This appendix of the Reactor Safety Study describes the input data and mathematical models employed to calculate the consequences of a potential release of radioactive material in the event of a nuclear power plant accident. Emphasis has been placed on documenting the rationale and referencing the source material underlying these inputs and models. In the course of this work, it has become increasingly clear that the consequence model is complex, with dependencies between many different scientific and medical disciplines, and that, in many areas, the available base of data is limited. The model described herein represents a reasonable assessment of consequences considering the state of knowledge in each subject. Future refinements should reduce the uncertainties, but it is believed at this time that the best-estimate values of the probabilities and magnitudes of the consequences should not greatly change from the values reported herein.

To assist in the development of the consequence model, the Reactor Safety Study has solicited the advice of many nationally recognized consultants in the many disciplines involved. In particular, an advisory group on health effects was formed; its membership is listed in section 14 of this appendix. The advisory group was unanimous that the selected model and input data were reasonable given the current state of knowledge. Of course, as would be expected in such a complex area, there were some conflicting opinions within the group over some details; however, these differences did not detract from the unanimity of opinion on the adequacy of the overall health model. The judgments and opinions expressed in this appendix are nonetheless the responsibility of the Reactor Safety Study.

It is the objective or the study to assess the risk from commercial nuclear power plants in as realistic a way as can reasonably be attained and to bound this assessment with upper and lower values. It is important that the estimate be as realistic as is reasonably attainable, in order to provide a proper perspective on potential risks. This realism is especially needed where risk comparisons are made as in chapter 6 of the Main Report.

A schematic outline of the model is shown in Fig. VI 1-1. The starting point for the calculation is the quantity of the radioactive material that could be released from the containment to the environment in the event of a nuclear power plant accident. The spectrum of releases to the environment are discretized into the nine PWR[1] and five BWR release categories as stated in Table VI 2-1, each with its associated probability of occurrence and release magnitude. Though the probability values that were developed in preceding appendices included estimated confidence bounds, these bounds are not propagated in the consequence model. However, they are used to estimate the confidence bounds on the results reported herein. The release magnitudes are used as best-estimate values, although, as discussed in the Main Report, they are believed to be conservative. The meteorological model computes the dispersion of radioactive material in terms of concentration in the air and on the ground as a function of time after the accident and distance from the reactor. The model used to compute dispersion is described in section 4, and the data that support its selection are presented in Appendix A. The model includes the following factors:

1. The decay of radioactivity as a function of time after the accident.

2. A standard Gaussian dispersion model that has been modified to include the effects of thermal stability, wind speed, and precipitation as a function of time after the accident. The model includes neither the temporal variation of wind direction nor the effect of wind shear.

[1]One PWR release category was subdivided into two releases to more properly represent the range of heat rates included within the category.

3. Dry deposition by contact between the cloud and the ground and wet deposition by washout due to the temporal variation in the occurrence of precipitation, as described in section 5 and Appendix B.

4. The temporal variation of weather parameters (stability, wind speed, and precipitation) are obtained by using 90 stratified samples from 1 year's weather data from applicable reactor sites. The diurnal and seasonal variations of the mixing layer are included. The details of the sampling scheme are described in section 13.

5. The effects of the plume lifting off the ground due to the release of sensible heat. Latent heat and radioactive heating are not included. The plume is not permitted to penetrate the mixing layer.

Having computed the concentrations of radioactivity in the air and on the ground, the model then computes the potential doses that could accrue from the following potential modes of exposure:

1. External irradiation from the passing cloud. This exposure would occur over a period of about one-half to a few hours.

2. Internal irradiation from inhaled radionuclides. While the inhalation would take place over the same time period as external irradiation from the passing cloud, the dose accumulated would be controlled by the residence time of the various radionuclides in the various parts of the body.

3. External irradiation from radionuclides deposited on the ground.

4. Internal irradiation from the inhalation of resuspended radionuclides that had been deposited on the ground. This exposure mode would not contribute significantly to predicted doses.

5. Ingestion of radionuclides from contaminated crops, water, and milk. Since this type of exposure could be controlled by constraints placed on consumption until levels of radioactivity are below maximum permissible concentrations, it would not contribute significantly to predicted doses.

All these different modes of exposure and the corresponding dosimetric models are discussed in section 8, with supporting data supplied in Appendices C through E.

The risk for the first 100 commercial nuclear power plants is calculated by using the following considerations. Meteorological data were obtained from six representative reactor sites, and each of the 68 sites was assigned to one of the six meteorological data sets to form a composite site representative of those reactors that are subject to similar weather. The meteorology for these six sites is described in section 5.

The distribution of people as a function of azimuth and distance from the reactor was obtained from 1970 census data. The populations in 22.5° sectors associated with the reactors assigned to a particular meteorological data set were combined to form a composite population distribution and its associated probability for that weather set. The details of this combination are described in section 10. It was assumed that people located within 25 miles downwind of the reactor would be evacuated in the event of an accident. By statistically analysing evacuation data (Appendix J), an evacuation model was developed as described in section 11.

The health effects models are described in section 9 with supporting data in Appendices F through I. The costs of decontaminating land or relocating the resident population are calculated with models described in sections 11 and 12 with supporting data in Appendix K.

The overall accident set is computed by convoluting the dispersion of radioactive material associated with the 10 PWR and 5 BWR release categories by using the 90 weather samples from each of the six sets of meteorological data over each of 16 population sectors for each of the six combined population distributions. These 130,000 hypothetical accidents are then ranked to generate complementary cumulative distribution functions for each of the potential consequences.

The results of the calculations are presented in section 13. Some additional studies are presented to show the sensitivity of specific consequences to important input parameters. In general, interdependencies between two parameters have not been assessed, and the study recommends more work in this area.

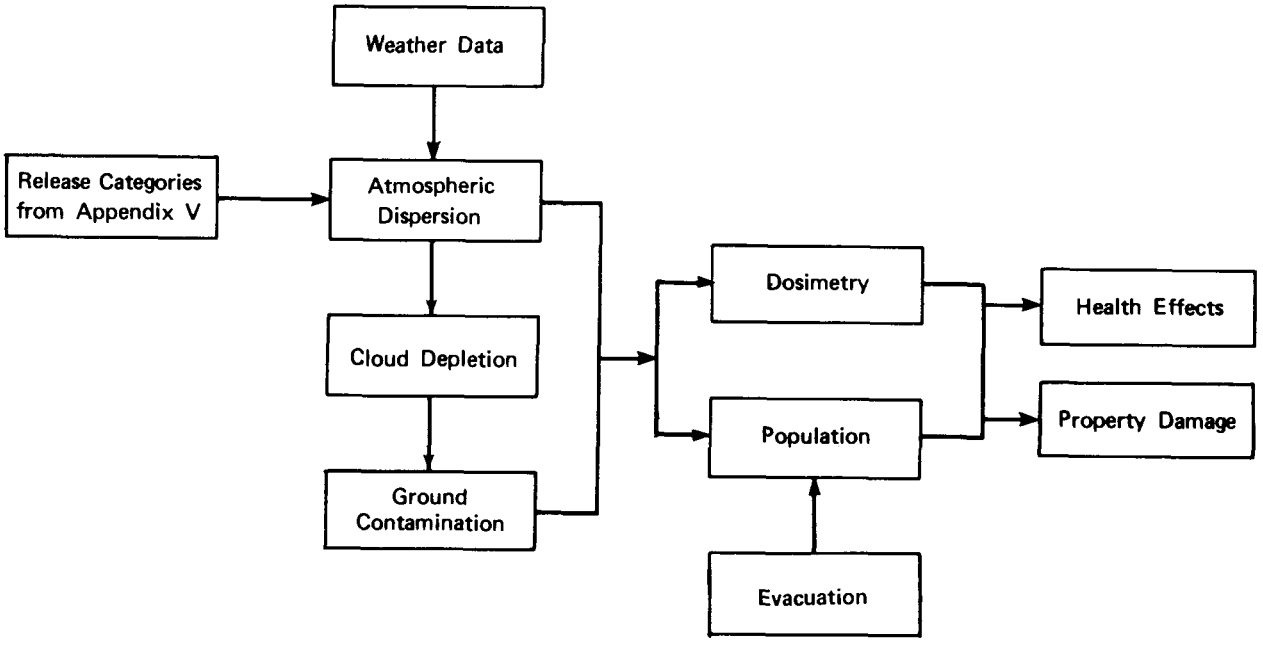

Fig VI 1-1 Schematic Outline of Consequence Model

Section 2

Releases from Containment

2.1 GENERAL REMARKS

A large portion of the work of the Reactor Safety Study was expended in determining the probability and magnitude of various radioactive releases. This work is described in detail in the preceding appendices as well as Appendices VII, and VIII. In order to define the various releases that might occur, a series of release categories were identified for the postulated types of containment failure in both BWRs and PWRs. The probability of each release category and the associated magnitude of radioactive releases (as fractions of the initial core radioactivity that might leak from the containment structure) are used as input data to the consequence model.

In addition to probability and release magnitude, the parameters that characterize the various hypothetical accident sequences are time of release, duration of release, warning time for evacuation, height of release, and energy content of the released plume.

The time of release refers to the time interval between the start of the hypothetical accident and the release of radioactive material from the containment building to the atmosphere; it is used to calculate the initial decay of radioactivity. The duration of release is the total time during which radioactive material is emitted into the atmosphere; it is used to account for continuous releases by adjusting for horizontal dispersion due to wind meander. These parameters, time and duration of release, represent the temporal behavior of the release in the dispersion model. They are used to model a "puff" release from the calculations of release versus time presented in Appendix V.

The warning time for evacuation (see section 11.1.1) is the interval between awareness of impending core melt and the release of radioactive material from the containment building. Finally, the height of release and the energy content of the released plume gas affect the manner in which the plume would be dispersed in the atmosphere.

Table VI 2-1 lists the leakage parameters that characterize the PWR and BWR release categories. It should be understood that these categories are composites of numerous event tree sequences with similar characteristics, as discussed in Appendix V.

2.2 ACCIDENT DESCRIPTIONS

To help the reader understand the postulated containment releases, this section presents brief descriptions of the various physical processes that define each release category. For more detailed information on the release categories and the techniques employed to compute the radioactive releases to the atmosphere, the reader is referred to Appendices V, VII, and VIII. The dominant event tree sequences in each release category are discussed in detail in section 4.6 of Appendix V.

PWR 1

This release category can be characterized by a core meltdown followed by a steam explosion on contact of molten fuel with the residual water in the reactor vessel. The containment spray and heat removal systems are also assumed to have failed and, therefore, the containment could be at a pressure above ambient at the time of the steam explosion. It is assumed that the steam explosion would rupture the upper portion of the reactor vessel and breach the containment barrier, with the result that a substantial amount of radioactivity might be released from the containment in a puff over a period of about 10 minutes. Due to the sweeping action of gases generated during containment-vessel meltthrough, the release of radioactive materials would continue at a relatively low rate thereafter. The total release would contain

approximately 70% of the iodines and 40% of the alkali metals present in the core at the time of release.[1] Because the containment would contain hot pressurized gases at the time of failure, a relatively high release rate of sensible energy from the containment could be associated with this category. This category also includes certain potential accident sequences that would involve the occurrence of core melting and a steam explosion after containment rupture due to overpressure. In these sequences, the rate of energy release would be lower, although still relatively high.

PWR 2

This category is associated with the failure of core-cooling systems and core melting concurrent with the failure of containment spray and heat-removal systems. Failure of the containment barrier would occur through overpressure, causing a substantial fraction of the containment atmosphere to be released in a puff over a period of about 30 minutes. Due to the sweeping action of gases generated during containment vessel meltthrough, the release of radioactive material would continue at a relatively low rate thereafter. The total release would contain approximately 70% of the iodines and 50% of the alkali metals present in the core at the time of release. As in PWR release category 1, the high temperature and pressure within containment at the time of containment failure would result in a relatively high release rate of sensible energy from the containment.

PWR 3

This category involves an overpressure failure of the containment due to failure of containment heat removal. Containment failure would occur prior to the commencement of core melting. Core melting then would cause radioactive materials to be released through a ruptured containment barrier. Approximately 20% of the iodines and 20% of the alkali metals present in the core at the time of release would be released to the atmosphere. Most of the release would occur over a period of about 1.5 hours. The release of radioactive material from containment would be caused by the sweeping action of gases generated by the reaction of the molten fuel with concrete. Since these gases would be initially heated by contact with the melt, the rate of sensible energy release to the atmosphere would be moderately high.

PWR 4

This category involves failure of the core-cooling system and the containment spray injection system after a loss-of-coolant accident, together with a concurrent failure of the containment system to properly isolate. This would result in the release of 9% of the iodines and 4% of the alkali metals present in the core at the time of release. Most of the release would occur continuously over a period of 2 to 3 hours. Because the containment recirculation spray and heat-removal systems would operate to remove heat from the containment atmosphere during core melting, a relatively low rate of release of sensible energy would be associated with this category.

PWR 5

This category involves failure of the core cooling systems and is similar to PWR release category 4, except that the containment spray injection system would operate to further reduce the quantity of airborne radioactive material and to initially suppress containment temperature and pressure. The containment barrier would have a large leakage rate due to a concurrent failure of the containment system to properly isolate, and most of the radioactive material would be released continuously over a period of several hours. Approximately 3% of the iodines and 0.9% of the alkali metals present in the core would be released. Because of the operation of the containment heat-removal systems, the energy release rate would be low.

[1]The release fractions of all the chemical species are listed in Table VI 2-1. The release fractions of iodine and alkali metals are indicated here to illustrate the variations in release with release category.

PWR 6

This category involves a core meltdown due to failure in the core cooling systems. The containment sprays would not operate, but the containment barrier would retain its integrity until the molten core proceeded to melt through the concrete containment base mat. The radioactive materials would be released into the ground, with some leakage to the atmosphere occurring upward through the ground. Direct leakage to the atmosphere would also occur at a low rate prior to containment-vessel meltthrough. Most of the release would occur continuously over a period of about 10 hours. The release would include approximately 0.08% of the iodines and alkali metals present in the core at the time of release. Because leakage from containment to the atmosphere would be low and gases escaping through the ground would be cooled by contact with the soil, the energy release rate would be very low.

PWR 7

This category is similar to PWR release category 6, except that containment sprays would operate to reduce the containment temperature and pressure as well as the amount of airborne radioactivity. The release would involve 0.002% of the iodines and 0.001% of the alkali metals present in the core at the time of release. Most of the release would occur over a period of 10 hours. As in PWR release category 6, the energy release rate would be very low.

PWR 8

This category approximates a PWR design basis accident (large pipe break), except that the containment would fail to isolate properly on demand. The other engineered safeguards are assumed to function properly. The core would not melt. The release would involve approximately 0.01% of the iodines and 0.05% of the alkali metals. Most of the release would occur in the 0.5-hour period during which containment pressure would be above ambient. Because containment sprays would operate and core melting would not occur, the energy release rate would also be low.

PWR 9

This category approximates a PWR design basis accident (large pipe break), in which only the activity initially contained within the gap between the fuel pellet and cladding would be released into the containment. The core would not melt. It is assumed that the minimum required engineered safeguards would function satisfactorily to remove heat from the core and containment. The release would occur over the 0.5-hour period during which the containment pressure would be above ambient. Approximately 0.00001% of the iodines and 0.00006% of the alkali metals would be released. As in PWR release category 8, the energy release rate would be very low.

BWR 1

This release category is representative of a core meltdown followed by a steam explosion in the reactor vessel. The latter would cause the release of a substantial quantity of radioactive material to the atmosphere. The total release would contain approximately 40% of the iodines and alkali metals present in the core at the time of containment failure. Most of the release would occur over a 1/2 hour period. Because of the energy generated in the steam explosion, this category would be characterized by a relatively high rate of energy release to the atmosphere. This category also includes certain sequences that involve overpressure failure of the containment prior to the occurrence of core melting and a steam explosion. In these sequences, the rate of energy release would be somewhat smaller than for those discussed above, although it would still be relatively high.

BWR 2

This release category is representative of a core meltdown resulting from a transient event in which decay-heat-removal systems are assumed to fail. Containment overpressure failure would result, and core melting would follow. Most of the release would occur over a period of about 3 hours. The containment failure would be such that radioactivity would be released directly to the atmosphere without significant retention of fission products. This category involves a relatively high rate of energy release due to the sweeping action of the gases generated by the molten mass. Approximately 90% of the iodines and 50% of the alkali metals present in the core would be released to the atmosphere.

BWR 3

This release category represents a core meltdown caused by a transient event accompanied by a failure to scram or failure to remove decay heat. Containment failure would occur either before core melt or as a result of gases generated during the interaction of the molten fuel with concrete after reactor-vessel meltthrough. Some fission-product retention would occur either in the suppression pool or the reactor building prior to release to the atmosphere. Most of the release would occur over a period of about 3 hours and would involve 10% of the iodines and 10% of the alkali metals. For those sequences in which the containment would fail due to overpressure after core melt, the rate of energy release to the atmosphere would be relatively high. For those sequences in which overpressure failure would occur before core melt, the energy release rate would be somewhat smaller, although still moderately high.

BWR 4

This release category is representative of a core meltdown with enough containment leakage to the reactor building to prevent containment failure by overpressure. The quantity of radioactivity released to the atmosphere would be significantly reduced by normal ventilation paths in the reactor building and potential mitigation by the secondary containment filter systems. Condensation in the containment and the action of the standby gas treatment system on the releases would also lead to a low rate of energy release. The radioactive material would be released from the reactor building or the stack at an elevated level. Most of the release would occur over a 2-hour period and would involve approximately 0.08% of the iodines and 0.5% of the alkali metals.

BWR 5

This category approximates a BWR design basis accident (large pipe break) in which only the activity initially contained within the gap between the fuel pellet and cladding would be released into containment. The core would not melt, and containment leakage would be small. It is assumed that the minimum required engineered safeguards would function satisfactorily. The release would be filtered and pass through the elevated stack. It would occur over a period of about 5 hours while the containment is pressurized above ambient and would involve approximately 6×10^{-9} % of the iodines and 4×10^{-7} % of the alkali metals. Since core melt would not occur and containment heat-removal systems would operate, the release to the atmosphere would involve a negligibly small amount of thermal energy.

TABLE VI 2-1 SUMMARY OF RELEASE CATEGORIES REPRESENTING HYPOTHETICAL ACCIDENTS

Release Category	Probability (reactor-yr^{-1})	Time of Release (hr)	Duration of Release (hr)	Warning Time for Evacuation (hr)	Elevation of Release[g] (meters)	Energy Release (10^6 Btu/hr)	Fraction of Core Inventory Released[a]							
							Xe-Kr	Organic I[b]	I[b]	Cs-Rb	Te-Sb	Ba-Sr	Ru[c]	La[d]
PWR 1	9×10^{-7}[e]	2.5	0.5	1.0	25	20 and 520[e]	0.9	6×10^{-3}	0.7	0.4	0.4	0.05	0.4	3×10^{-3}
PWR 2	8×10^{-6}	2.5	0.5	1.0	0	170	0.9	7×10^{-3}	0.7	0.5	0.3	0.06	0.02	4×10^{-3}
PWR 3	4×10^{-6}	5.0	1.5	2.0	0	6	0.8	6×10^{-3}	0.2	0.2	0.3	0.02	0.03	3×10^{-3}
PWR 4	5×10^{-7}	2.0	3.0	2.0	0	1	0.6	2×10^{-3}	0.09	0.04	0.03	5×10^{-3}	3×10^{-3}	4×10^{-4}
PWR 5	7×10^{-7}	2.0	4.0	1.0	0	0.3	0.3	2×10^{-3}	0.03	9×10^{-3}	5×10^{-3}	1×10^{-3}	6×10^{-4}	7×10^{-5}
PWR 6	6×10^{-6}	12.0	10.0	1.0	0	N/A	0.3	2×10^{-3}	8×10^{-4}	8×10^{-4}	1×10^{-3}	9×10^{-5}	7×10^{-5}	1×10^{-5}
PWR 7	4×10^{-5}	10.0	10.0	1.0	0	N/A	6×10^{-3}	2×10^{-5}	2×10^{-5}	1×10^{-5}	2×10^{-5}	1×10^{-6}	1×10^{-6}	2×10^{-7}
PWR 8	4×10^{-5}	0.5	0.5	N/A[f]	0	N/A	2×10^{-3}	5×10^{-6}	1×10^{-4}	5×10^{-4}	1×10^{-6}	1×10^{-8}	0	0
PWR 9	4×10^{-4}	0.5	0.5	N/A	0	N/A	3×10^{-6}	7×10^{-9}	1×10^{-7}	6×10^{-7}	1×10^{-9}	1×10^{-11}	0	0
BWR 1	1×10^{-6}	2.0	0.5	1.5	25	130	1.0	7×10^{-3}	0.40	0.40	0.70	0.05	0.5	5×10^{-3}
BWR 2	6×10^{-6}	30.0	3.0	2.0	0	30	1.0	7×10^{-3}	0.90	0.50	0.30	0.10	0.03	4×10^{-3}
BWR 3	2×10^{-5}	30.0	3.0	2.0	25	20	1.0	7×10^{-3}	0.10	0.10	0.30	0.01	0.02	4×10^{-3}
BWR 4	2×10^{-6}	5.0	2.0	2.0	25	N/A	0.6	7×10^{-4}	8×10^{-4}	5×10^{-3}	4×10^{-3}	6×10^{-4}	6×10^{-4}	1×10^{-4}
BWR 5	1×10^{-4}	3.5	5.0	N/A	150	N/A	5×10^{-4}	2×10^{-9}	6×10^{-11}	4×10^{-9}	8×10^{-12}	8×10^{-14}	0	0

(a) Background on the isotope groups and release mechanisms is presented in Appendix VII.
(b) Organic iodine is combined with elemental iodines in the calculations. Any error is negligible since its release fraction is relatively small for all large release categories.
(c) Includes Ru, Rh, Co, Mo, Tc.
(d) Includes Y, La, Zr, Nb, Ce, Pr, Nd, Np, Pu, Am, Cm.
(e) Accident sequences within PWR 1 category have two distinct energy releases that affect consequences. PWR 1 category is subdivided into PWR 1A with a probability of 4×10^{-7} per reactor-year and 20×10^6 Btu/hr and PWR 1B with a probability of 5×10^{-7} per reactor-year and 520×10^6 Btu/hr.
(f) Not applicable.
(g) A 10 meter elevation is used in place of zero representing the mid-point of a potential containment break. Any impact on the results would be slight and conservative.

Section 3

Radioactive Inventory of Reactor Core

3.1 METHOD OF CALCULATING BURNUP

The potential radioactive source (fission products, transuranics, and activation products) in the reactor immediately preceding the initiation of an an accident was obtained from analyses performed with the ORIGEN computer program (Bell, 1973). The set of equations describing the formation, transmutation, and decay of nuclides within an operating nuclear reactor is approximated by ORIGEN as a homogeneous set of simultaneous first-order ordinary differential equations with constant coefficients. Rigorously, the set of equations is nonlinear since the neutron flux varies as the composition of the fuel changes at constant power. However, this variation with time is small, and the neutron flux can be considered constant over short time intervals, thus permitting the linear approximation.

ORIGEN solves this set of equations by the matrix-exponential method. Most computer programs solve this set of equations by the method of Bateman (1910), which involves direct solution of the governing differential equations in a general form. In general, programs utilizing this technique have not been able to treat the full range of transmutations that might occur. They also have experienced difficulties in computing nuclide concentrations for decay chains in which (1) a nuclide decays to produce one of its precursors (e.g., neutron capture followed by alpha decay) or (2) a nuclide decays to produce a daughter that is present in another decay chain. The matrix-exponential method eliminates these difficulties.

3.2 REACTOR COMPOSITION, DESIGN, AND OPERATING HISTORY

Radionuclide inventories were calculated by means of the ORIGEN program for a 3200-MWt three-region PWR core with a composition that is representative of PWRs (Bell, 1973). (This composition represents a typical four-loop Westinghouse PWR.) It was assumed that the three regions of the core operate at a constant specific power density of 40 kW/kg of uranium charged. Inventories were calculated for an equilibrium core initially charged with 3.3% enriched uranium at a time when the three regions have average burnups of 880, 17,600, and 26,400 megawatt-days per metric ton of uranium charged.

Pressurized water reactors generally operate with power densities in the range of 30 to 35 kW/kg. Calculations based on a power density of 40 kW/kg will overestimate the inventory of short-lived radionuclides by 14 to 33% since the inventory of isotopes that reach equilibrium during irradiation is directly proportional to the power density (neutron flux). The inventory of the long-lived radionuclides, however, is proportional to burnup (i.e., neutron flux times time) and is not sensitive to power density at any given exposure.

Boiling water reactors typically operate at a lower specific power density (approximately 23 kW/kg of uranium charged) and with less enriched fuel (approximately 2.2%). However, because of the lower enrichment of BWR, the average thermal neutron flux for both PWRs and BWRs is approximately the same. Thus fission-product generation and transmutation by neutron absorption are equivalent in both types of reactors when operating at the same power densities. There may be some differences in the inventory of activation products between PWRs and BWRs because the atom densities of the various constituents of core structural and cladding material differ. However, activation products are not significant in comparison to the fission products and transuranics, and, in general, the variations in the quantity of activation products between the two types of reactor can be ignored.

3.3 SELECTION OF RADIONUCLIDES

The ORIGEN program calculates the time-dependent concentration of a very large number of nuclides: 246 activation products, 461 fission products, and 82 transuranics. Although many of these nuclides are not radioactive, the total number of radionuclides is quite large and significant amounts of computer storage and computational time would be required to handle all of them in the consequence model. At a very small sacrifice in the precision of the radiation dose calculations, the number of radionuclides considered can be reduced to a manageable size.

The elimination of radionuclides from consideration in radiation dose calculations was based on a number of parameters, such as quantity (curies), release fraction, radioactive half-life, emitted radiation type and energy, and chemical characteristics. The precise method of elimination is explained in section 8.2.1. In addition, it is possible to eliminate radionuclides with half-lives shorter than 25.7 minutes (decay constants greater than 4.5×10^{-4} sec^{-1}) because, as explained in Appendix V, the minimum delay time between termination of the chain-reaction (start of the accident) and the release of radioactive material to the atmosphere would be at least 0.5 hour and could be as long as 30 hours.

These eliminations resulted in the list of 54 radionuclides presented in Table VI 3-1, which also gives their activity at the time the accident is assumed to be initiated.

REFERENCES

Bell, M. J., 1973, ORIGEN, the ORNL Isotope Generation and Depletion Code, ORNL-4628, Oak Ridge National Laboratory.

Bateman, H., 1910, Proc. Cambridge Phil. Soc., 15, p. 423.

TABLE VI 3-1 INITIAL ACTIVITY OF RADIONUCLIDES IN THE NUCLEAR REACTOR CORE AT THE TIME OF THE HYPOTHETICAL ACCIDENT

No.	Radionuclide	Radioactive Inventory Source (curies x 10^{-8})	Half-Life (days)
1	Cobalt-58	0.0078	71.0
2	Cobalt-60	0.0029	1,920
3	Krypton-85	0.0056	3,950
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
9	Strontium-90	0.037	11,030
10	Strontium-91	1.1	0.403
11	Yttrium-90	0.039	2.67
12	Yttrium-91	1.2	59.0
13	Zirconium-95	1.5	65.2
14	Zirconium-97	1.5	0.71
15	Niobium-95	1.5	35.0
16	Molybdenum-99	1.6	2.8
17	Technetium-99m	1.4	0.25
18	Ruthenium-103	1.1	39.5
19	Ruthenium-105	0.72	0.185
20	Ruthenium-106	0.25	366
21	Rhodium-105	0.49	1.50
22	Tellurium-127	0.059	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	3.88
29	Antimony-129	0.33	0.179
30	Iodine-131	0.85	8.05
31	Iodine-132	1.2	0.0958
32	Iodine-133	1.7	0.875
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
38	Cesium-136	0.030	13.0
39	Cesium-137	0.047	11,000
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	32,500
49	Plutonium-239	0.00021	8.9×10^6
50	Plutonium-240	0.00021	2.4×10^6
51	Plutonium-241	0.034	5,350
52	Americium-241	0.000017	1.5×10^5
53	Curium-242	0.0050	163
54	Curium-244	0.00023	6,630

Section 4

Atmospheric Dispersion

4.1 INTRODUCTION

As the plume from a reactor accident is carried away from the site by the wind, atmospheric diffusion would be continually acting to disperse the contaminants at a rate depending on the wind speed, thermal stability, and the underlying characteristics of the terrain. The average wind from ground level to a height that encompasses the plume top is the single most important parameter, as it determines the directions of transport and the initial volume of air into which the contaminant is diluted.

The wind variability in three dimensions and time is defined by a broad scale of eddy sizes, which, when integrated from the molecular scale to plume dimensions, makes up the turbulent diffusion. Estimating the intensity of turbulence by various accepted techniques based on theoretical and experimental experience makes it possible to make quantitative estimates of the ever-expanding volume into which the effluent is dispersed. For a description of these techniques and their relation to the consequence model, the reader is referred to Appendix A.

Important parameters used to estimate the dilution capability of the atmosphere besides the wind are (1) the temperature structure with height above the ground and (2) the occurrence of rain and its probability after any specific time. The former is important for its strong effect on the intensity of turbulence and the latter because of its efficiency in removing particulate and some gaseous materials from the air. The removal processes that are important both in reducing the source strength of the plume and in the deposition effects are discussed in section 6 and Appendix B.

4.2 *THE ATMOSPHERIC DISPERSION MODEL*

The general features of atmospheric dispersion modeling are described in Appendix A. This section describes the actual calculational techniques used in the consequence model. Given a specific reactor site and an accident starting time, which in turn specify the sequence of precipitation occurrence, stability category, and wind speed necessary to calculate plume transport, computations of plume dimension (vertical and lateral) are made for each downwind spatial interval. These spatial intervals are given in Table VI 4-1. In the calculations, the air concentrations and ground concentrations of the released radioactivity are computed at the midpoint of the interval and assumed to be uniform within the interval.

Initially the plume is treated as a simple ground-level continuous release of 0.5-hour duration. It is then corrected for building-wake effects, buoyant rise, differences in release duration, and depletion by dry and wet deposition as well as radioactive decay. At the midpoint of each downwind spatial interval, the ratio of ground-level contaminant concentration in the air to the source strength is given by

$$\chi/Q = 2(3\sigma_y)^{-1}(\sqrt{2\pi}\sigma_z)^{-1}\,U^{-1}, \qquad \text{(VI 4-1)}$$

where χ is the contaminant concentration in air (curies per cubic meter), Q is the source strength (curies per second), $3\sigma_y$ is the lateral width (meters) of the assumed rectangular (uniform) distribution, σ_z is the vertical standard deviation of the contaminant (meters), and U is the mean transport wind speed (meters per second).

The distribution parameters σ_y and σ_z are each a function of downwind distance and thermal stability category. The stability categories are specified by temperature lapse rate data (decrease with altitude) from each reactor site according to the scheme of Regulatory Guide 1.23, as given in Table VI 4-2.

Use of σ_θ values, while more satisfying theoretically, presents difficulties in low-wind-speed situations, when vanes become nonresponsive to wind fluctuations. Therefore only the temperature structure between about 10 and 40 meters above ground was used. The derived distributions of stability by reactor site are shown in section 5.

The coefficients and exponents of Martin and Tikvart (1968) are used to determine the vertical and lateral standard deviations in the formulation $\sigma = AX^B + C$ (see Table VI A-1 in Appendix A). Values for σ_z are modified for the presence of a finite mixing depth by using a scheme suggested by Turner (1969), which increments σ_z until it equals or exceeds 0.8L, where L is the mixing height. Thereafter σ_z is maintained equal to 0.8L as the concentration becomes vertically uniform. Values of L by season and atmospheric stability category have been compiled by Holzworth (1972). The tabulated values used for the mixing height are given in Table VI 5-3 of section 5.

Transport speed, stability, and precipitation occurrence are updated by each successive hourly weather observation as indicated. Thus, the plume expands continuously by vertical and horizontal increments according to spatial increment length and location, hourly value of wind speed, stability class, and mixing depth.

The plume dimension is initialized to typical reactor building cluster size by setting $3\sigma_y = 100$ meters and $2.15\sigma_z = 25$ meters; that is, the concentration of the plume at the building top is assumed to be one-tenth of the centerline value. If the effect of the building wake is to be treated in a calculation, the plume cross-sectional area is allowed to expand by only a factor of 5 to a specified downwind distance not to exceed about 2.3 kilometers. Beyond this distance, ambient atmospheric turbulent expansion is allowed to begin. With no building-wake effects included, atmospheric turbulent expansion is assumed to start immediately at the point of release.

The χ/Q ratios are modified first for the effect of bouyant plume rise. The recommendations of Briggs (1969), as discussed in section A3.3 of Appendix A, are used for estimating rise versus distance, wind speed, source heat flux, and stability. The plume centerline height is not allowed to rise above the maximum encountered mixing-layer depth L; that is, no penetration of the mixing layer is allowed.

Allowance for release durations in excess of 0.5 hour is made by using the factor $(\Delta t \text{ (hours)}/0.5)^p$, where p is taken ad 1/3. Actually p varies between 1/5 and 1/2, depending on stability and height.

The final modification to Equation (VI 4-1) is for plume depletion by fallout and precipitation removal, as discussed in section 6 and Appendix B. At each spatial interval, the amount of material deposited is subtracted from the plume source.

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Turner, D. B., 1969, Workbook of Atmospheric Dispersion Estimates, U.S. Department of Health, Education and Welfare, Public Health Service, Publ. No. 999-AP-26.

U.S. Nuclear Regulatory Commission, Regulatory Guide 1.23, "Onsite Meteorological Programs."

TABLE VI 4-1 MESH SPACING FOR 22.5° SECTOR[a]

Spatial Interval	Outer Radius (miles)	Midpoint miles	Spatial Interval	Outer Radius miles	Midpoint miles
1	0.5	0.25	18	20	18.75
2	1.0	0.75	19	25	22.5
3	1.5	1.25	20	30	27.5
4	2.0	1.75	21	35	32.5
5	2.5	2.25	22	40	37.5
6	3.0	2.75	23	45	42.5
7	3.5	3.25	24	50	47.5
8	4.0	3.75	25	55	52.5
9	4.5	4.25	26	60	57.5
10	5.0	4.75	27	65	62.5
11	6.0	5.50	28	70	67.5
12	7.0	6.50	29	85	77.5
13	8.5	7.75	30	100	92.5
14	10.0	9.25	31	150	125.0
15	12.5	11.25	32	200	175.0
16	15.0	13.75	33	350	275.0
17	17.5	16.25	34	500	425.0

(a) Some special calculations used a modified mesh spacing.

TABLE VI 4-2 ATMOSPHERIC STABILITY CLASSIFICATION

Description	Category	Change with Height (ΔK/100 m)		σ_θ[a] (Radian)
		Temperature	Potential Temperature	
Extremely unstable	A	< -1.9	< -0.9	0.436
Moderately unstable	B	-1.9 to -1.7	-0.9 to -0.7	0.349
Slightly unstable	C	-1.7 to -1.5	-0.7 to -0.5	0.262
Neutral	D	-1.5 to -0.5	-0.5 to +0.5	0.175
Slightly stable	E	-0.5 to +1.5	0.5 to +2.5	0.0873
Very stable	F	1.5 to +4.0	> 2.5	0.0436

(a) Standard deviation of horizontal wind direction fluctuation over a period of 15 to 60 minutes. The values shown are averages for each category.

Section 5

Reactor Sites and Meteorological Data

From the many reactor sites in the United States, a total of seven[1] broad types were selected as being representative of variability of climatic or topographic features. These types are listed in Table VI 5-1.

TABLE VI 5-1 SITE CHARACTERISTICS

Site Type	Characteristics
A	Large river valley in northeast
B	Great Lakes shore
C	Dry western desert
D	Central Midwest plain
E	Pacific coastal site
F	Atlantic coastal site
G	Southeast river valley influenced by Bermuda High

Each of these sites had at least 1 year of complete recorded data of the specified type suitable for incorporating into a computer file for ready retrieval. For direct comparisons among sites, exactly 1 year's worth of data was processed. The individual elements are specified in Regulatory Guide 1.23 and are sufficient to specify hourly values of wind speed and direction, temperature and temperature lapse rate, and rainfall occurrence.

The data file for each of the seven sites consists of the following:

1. Seasonal data on wind direction frequency, 16 sectors (actually wind transport vector).
2. Stability category (see Table VI 4-2) by lapse rate and wind speed in meters per second.
3. Seasonal data on mixing height for stable and unstable categories.
4. Hourly data on rain occurrence ($\geq$ 0.25 mm collection).

Figures VI 5-1a through g show wind transport vector (direction towards which wind blows) roses for each site for the hourly data averaged over the year. Comparing the different wind-transport roses points out the influence of the individual site topography on the average wind flow. Site A shows the influence of the river valley,

[1] Seven composite sites were originally constructed. However, because the seventh site was a dry western desert and only one reactor was located therein, it was deemed appropriate to include it in the midwestern plain category. This introduces a small degree of conservatism in the final results.

with a predominant flow toward the south-southwest and a secondary maximum up-valley toward the north-northwest. The valley curves at the site location, which explains the nonalighment of the up- and down-valley maxima. Site D on the flat plain has the most uniform (quasi-isotropic) direction distributions. Site E has a remarkable maximum direction frequency at southwest, due to the presence of a small hill to the northwest and a gully cutting through the elevated coastal plain adjacent to the hill, which effectively channels the nighttime land breeze. The site G valley is quite level near the site but rises to several thousand feet on the southeast side at the Blue Ridge-Smokey Mountains complex. Thus the up- and down-valley flow is quite dominant. Actually, for the six composite sites representing the 68 actual reactor sites (described in section 10), it was assumed that the wind direction distribution for such summations would approach uniformity. This assumption is based on the fact that local topographic features which may strongly influence surface winds are generally randomly oriented when taken over such a large number of individual locations.

Neither thermal stability and wind speed nor thermal stability and the occurrence of precipitation[1] are independent variables. The incidence of rain in a given hour is defined as at least 0.25 mm of rain equivalent within that hour. Tables VI 5-2a through g show the joint frequency distributions for thermal stability, wind speed and rain for each site. The frequency distributions for wind speed have a relatively similar shape; but the mean speeds are different at each site. This characteristic has been pointed out by Luna and Church (1974). The stability distributions indicate that the median value for all sites lies between stability categories D and E. Sites B and C show an interesting contrast in the combined occurrences of stability categories E and F (approximately 35 and 59% for sites B and C, respectively). This effect is caused by the presence of a modifying body of water at site B compared to the dry continental situation at site C. The generation of a stable layer at site B depends on the temperature difference between the existing air mass and the lake water.

Table VI 5-2A JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE A

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0183	0.0293	0.0269	0.0252	0.0217	0.0142	0.0114	0.0154	
	RAIN	0.0009	0.0006	0.0003	0.0005	0.0	0.0001	0.0	0.0001	0.1650
B	NO RAIN	0.0049	0.0034	0.0026	0.0021	0.0025	0.0015	0.0006	0.0015	
	RAIN	0.0001	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0192
C	NO RAIN	0.0051	0.0040	0.0040	0.0031	0.0034	0.0027	0.0023	0.0046	
	RAIN	0.0001	0.0005	0.0002	0.0	0.0002	0.0	0.0	0.0001	0.0313
D	NO RAIN	0.0606	0.0452	0.0404	0.0354	0.0268	0.0233	0.0122	0.0257	
	RAIN	0.0037	0.0051	0.0041	0.0019	0.0019	0.0010	0.0019	0.0023	0.2987
E	NO RAIN	0.0761	0.0564	0.0477	0.0345	0.0245	0.0118	0.0008	0.0179	
	RAIN	0.0053	0.0054	0.0057	0.0039	0.0016	0.0006	0.0006	0.0017	0.3015
F	NO RAIN	0.0811	0.0486	0.0251	0.0121	0.0050	0.0031	0.0015	0.0017	
	RAIN	0.0025	0.0010	0.0009	0.0008	0.0007	0.0001	0.0001	0.0	0.1344
SUMMATION		0.2587	0.2005	0.1661	0.1135	0.0885	0.0583	0.0335	0.0710	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =5.5%

[1] Precipitation includes rain, snow, etc. The study treated all precipitation as rain.

Table VI 5-2B JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE B

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0047	0.0162	0.0171	0.0092	0.0066	0.0022	0.0010	0.0008	
	RAIN	0.0001	0.0	0.0	0.0	0.0001	0.0001	0.0001	0.0	0.0583
B	NO RAIN	0.0022	0.0065	0.0103	0.0068	0.0078	0.0039	0.0010	0.0008	
	RAIN	0.0003	0.0	0.0001	0.0001	0.0002	0.0	0.0005	0.0001	0.0406
C	NO RAIN	0.0021	0.0001	0.0137	0.0087	0.0078	0.0051	0.0025	0.0013	
	RAIN	0.0	0.0	0.0001	0.0	0.0	0.0003	0.0003	0.0002	0.0409
D	NO RAIN	0.0365	0.0765	0.0838	0.0725	0.0702	0.0411	0.0220	0.0158	
	RAIN	0.0034	0.0094	0.0119	0.0118	0.0134	0.0089	0.0051	0.0057	0.4976
E	NO RAIN	0.0573	0.0648	0.0455	0.0259	0.0158	0.0039	0.0018	0.0022	
	RAIN	0.0056	0.0065	0.0046	0.0034	0.0025	0.0002	0.0005	0.0003	0.2385
F	NO RAIN	0.0648	0.0276	0.0092	0.0048	0.0026	0.0007	0.0002	0.0021	
	RAIN	0.0011	0.0005	0.0003	0.0003	0.0006	0.0001	0.0	0.0	0.1151
SUMMATION		0.1782	0.2169	0.1963	0.1426	0.1364	0.0645	0.0358	0.0292	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =9.9%

Table VI 5-2C JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE C

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0024	0.0054	0.0058	0.0078	0.0042	0.0065	0.0022	0.0037	
	RAIN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0379
B	NO RAIN	0.0089	0.0121	0.0083	0.0092	0.0057	0.0033	0.0014	0.0017	
	RAIN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0507
C	NO RAIN	0.0159	0.0210	0.0104	0.0071	0.0042	0.0030	0.0010	0.0021	
	RAIN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0646
D	NO RAIN	0.1025	0.0644	0.0283	0.0212	0.0100	0.0087	0.0046	0.0145	
	RAIN	0.0009	0.0006	0.0003	0.0006	0.0	0.0002	0.0	0.0002	0.2568
E	NO RAIN	0.0965	0.0462	0.0215	0.0234	0.0153	0.0106	0.0041	0.0074	
	RAIN	0.0015	0.0005	0.0001	0.0007	0.0002	0.0002	0.0	0.0001	0.2283
F	NO RAIN	0.2207	0.0846	0.0299	0.0189	0.0024	0.0010	0.0	0.0049	
	RAIN	0.0	0.0	0.0001	0.0	0.0	0.0	0.0	0.0	0.3616
SUMMATION		0.4492	0.2347	0.1048	0.0889	0.0421	0.0336	0.0132	0.0334	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =0.6%

Table VI 5-2D JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE D

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0041	0.0143	0.0192	0.0205	0.0208	0.0124	0.0075	0.0098	
	RAIN	0.0001	0.0003	0.0006	0.0001	0.0003	0.0005	0.0003	0.0003	0.1113
B	NO RAIN	0.0035	0.0078	0.0065	0.0076	0.0063	0.0029	0.0018	0.0026	
	RAIN	0.0002	0.0	0.0001	0.0	0.0	0.0003	0.0002	0.0	0.0400
C	NO RAIN	0.0059	0.0113	0.0100	0.0082	0.0094	0.0042	0.0025	0.0035	
	RAIN	0.0002	0.0002	0.0006	0.0007	0.0005	0.0002	0.0	0.0001	0.0576
D	NO RAIN	0.0387	0.0595	0.0622	0.0557	0.0522	0.0295	0.0178	0.0216	
	RAIN	0.0018	0.0050	0.0056	0.0071	0.0051	0.0031	0.0032	0.0033	0.3713
E	NO RAIN	0.0548	0.0575	0.0481	0.0318	0.0213	0.0113	0.0094	0.0111	
	RAIN	0.0024	0.0029	0.0040	0.0037	0.0018	0.0006	0.0002	0.0009	0.2618
F	NO RAIN	0.0946	0.0377	0.0177	0.0038	0.0009	0.0001	0.0	0.0003	
	RAIN	0.0015	0.0007	0.0002	0.0003	0.0001	0.0	0.0	0.0	0.1580
SUMMATION		0.2080	0.1971	0.1748	0.1396	0.1187	0.0651	0.0430	0.0537	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =6.0%

Table VI 5-2E JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE E

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0037	0.0380	0.0677	0.0404	0.0164	0.0061	0.0016	0.0021	
	RAIN	0.0001	0.0001	0.0	0.0	0.0	0.0	0.0001	0.0	0.1822
B	NO RAIN	0.0017	0.0087	0.0065	0.0030	0.0011	0.0011	0.0003	0.0002	
	RAIN	0.0	0.0001	0.0001	0.0	0.0	0.0	0.0	0.0	0.0229
C	NO RAIN	0.0023	0.0076	0.0039	0.0048	0.0016	0.0007	0.0001	0.0005	
	RAIN	0.0	0.0	0.0	0.0001	0.0	0.0001	0.0	0.0	0.0217
D	NO RAIN	0.0454	0.0495	0.0385	0.0250	0.0128	0.0086	0.0051	0.0081	
	RAIN	0.0001	0.0001	0.0002	0.0	0.0	0.0005	0.0008	0.0011	0.1959
E	NO RAIN	0.1097	0.0821	0.0583	0.0379	0.0265	0.0156	0.0061	0.0091	
	RAIN	0.0013	0.0007	0.0015	0.0018	0.0011	0.0011	0.0007	0.0029	0.3563
F	NO RAIN	0.0627	0.0486	0.0401	0.0320	0.0186	0.0102	0.0042	0.0006	
	RAIN	0.0005	0.0008	0.0008	0.0008	0.0006	0.0001	0.0003	0.0002	0.2210
SUMMATION		0.2324	0.2373	0.2176	0.1458	0.0788	0.0441	0.0194	0.0247	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =1.9%

Table VI 5-2F JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE F

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0017	0.0058	0.0075	0.0061	0.0066	0.0047	0.0031	0.0040	
	RAIN	0.0001	0.0003	0.0	0.0	0.0	0.0	0.0	0.0	0.0397
B	NO RAIN	0.0024	0.0050	0.0096	0.0106	0.0081	0.0063	0.0040	0.0078	
	RAIN	0.0	0.0001	0.0	0.0	0.0	0.0	0.0	0.0	0.0539
C	NO RAIN	0.0014	0.0043	0.0026	0.0024	0.0030	0.0025	0.0013	0.0047	
	RAIN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0221
D	NO RAIN	0.0311	0.0494	0.0623	0.0613	0.0497	0.0393	0.0259	0.0428	
	RAIN	0.0014	0.0042	0.0035	0.0043	0.0029	0.0030	0.0018	0.0037	0.3863
E	NO RAIN	0.0943	0.0708	0.0515	0.0364	0.0203	0.0118	0.0081	0.0121	
	RAIN	0.0026	0.0024	0.0041	0.0018	0.0009	0.0007	0.0010	0.0015	0.3203
F	NO RAIN	0.0676	0.0340	0.0199	0.0155	0.0135	0.0067	0.0040	0.0073	
	RAIN	0.0014	0.0005	0.0014	0.0005	0.0010	0.0009	0.0005	0.0026	0.1776
SUMMATION		0.2039	0.1767	0.1622	0.1389	0.1059	0.0758	0.0497	0.0869	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =4.0%

Table VI 5-2G JOINT FREQUENCY DISTRIBUTION FOR THERMAL STABILITY, WINDSPEED, AND RAIN FOR SITE G

THERMAL STABILITY		WINDSPEED (M/SEC) 0-1	1-2	2-3	3-4	4-5	5-6	6-7	>7	SUMMATION
A	NO RAIN	0.0008	0.0006	0.0001	0.0006	0.0016	0.0002	0.0002	0.0005	
	RAIN	0.0	0.0	0.0	0.0001	0.0001	0.0	0.0	0.0	0.0048
B	NO RAIN	0.0008	0.0009	0.0003	0.0015	0.0011	0.0007	0.0001	0.0007	
	RAIN	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0062
C	NO RAIN	0.0025	0.0015	0.0024	0.0037	0.0025	0.0025	0.0005	0.0017	
	RAIN	0.0	0.0	0.0	0.0001	0.0001	0.0002	0.0	0.0	0.0177
D	NO RAIN	0.0626	0.0444	0.0374	0.0295	0.0211	0.0253	0.0217	0.0330	
	RAIN	0.0027	0.0017	0.0016	0.0009	0.0013	0.0009	0.0006	0.0014	0.3060
E	NO RAIN	0.0804	0.0627	0.0619	0.0546	0.0587	0.0395	0.0252	0.0316	
	RAIN	0.0043	0.0030	0.0026	0.0025	0.0073	0.0061	0.0046	0.0090	0.4739
F	NO RAIN	0.0562	0.0394	0.0387	0.0271	0.0148	0.0078	0.0024	0.0011	
	RAIN	0.0006	0.0007	0.0007	0.0019	0.0005	0.0002	0.0001	0.0002	0.1914
SUMMATION		0.2108	0.1608	0.1527	0.1385	0.1191	0.0834	0.0554	0.0792	1.0000

PERCENTAGE OF HOURS HAVING PRECIPITATION =7.5%

The mixing heights stated in Table VI 5-3 are taken from Holzworth (1972) who examined 5 years of data (see Figures VI A-4 and VI A-5 for average mixing heights over a year).

TABLE VI 5-3 SEASONAL MIXING HEIGHTS BY SITE

Season	Seasonal Mixing Height (meter x 10^{-2}) (Stable/Unstable) Site A	Site B	Site C	Site D	Site E	Site F	Site G
Winter	8/9	8.5/9	2.5/13	5/6	5/10	8/9	5/10
Spring	8/15	6.5/14	3/26	5/15	8/10	8/13	5/18
Summer	6.5/16	4.5/16	2.5/32	3/16	5/6	6/13	5/18
Fall	7/11	6/12	2.5/20	4/12	5/8	7/10	3.5/14

For all meteorological data for a particular site, the consequence model assumes that the condition that occurs at the site at a given hour also occurs simultaneously at all downwind locations to whatever distance the plume has traveled. Although it is recognized that such is frequently not the case, the assumption is realistic: since many hours or cases are included, the usual horizontal translation of weather systems has the effect of averaging conditions out into a quasi-homogeneous horizontal set.

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Section 6

Plume Depletion: Radioactive Decay and Deposition

6.1 INTRODUCTION

As the plume of radioactive material travels away from its point of origin, turbulent effects act on the plume to reduce the concentration of the material by dispersing it in a greater volume of air. The processes of radioactive decay and deposition combine with turbulent effects to reduce the airborne concentrations. These processes must be accounted for in order to adequately calculate air concentrations at large distances from the plume origin and to assess the impact of deposited radioactivity on the environment over which the plume travels. These processes, and the manner in which they are handled in the consequence model, are discussed in this section.

6.2 RADIOACTIVE DECAY

The process of radioactive decay is well understood and can be accurately calculated. The time period over which the radioactive material may remain airborne after release from the containment is relatively short, and so only radionuclides with half-lives of less than a few days will decay appreciably in this interval. For each spatial interval outward from the plume origin, the airborne concentration is adjusted in the model for the radioactive decay that occurred during the time of travel from the preceding spatial interval. In addition, since some of the radionuclides have daughters that are also radioactive (e.g., iodine-133 decays to xenon-133), the growth of radioactive daughters into the plume is also calculated for the time interval of travel from one spatial interval to the next.

6.3 DEPOSITION

The formulation and description of the deposition process are more complex and less well determined from experiment. Aerosols are removed from the atmosphere by sedimentation under the influence of gravity, by impaction on obstacles on or near the earth's surface, and by precipitation scavenging. These processes are collectively called deposition. Precipitation scavenging is generally referred to as wet deposition. Sedimentation is an important removal process for aerosols with particles of 15 microns in diameter or larger. For smaller particle sizes, the processes of impaction or precipitation scavenging are dominant. The reason for this phenomenon is the magnitude of the fall velocity; for sufficiently low fall velocities, the vertical movement of the aerosol is largely controlled by the vertical turbulence and mean air motions.

In general, wet and dry removal of particles and gases cannot yet be specified precisely. It is known that there are significant dependencies of removal rates on precipitation type, rate, and hydrometeor size distributions; on particle density, wettability, and size distributions; on gaseous chemical composition, water solubility, and reaction rates; on vegetative type, biomass, and physiological state; and on atmospheric stability, wind field, and humidity. However, experimental tests and theoretical developments have not yet been sufficiently extensive to quantify the influence of all these and other variables. Because of these complexities, it is only possible to bound and approximate the removal processes using state-of-the-art knowledge. For a discussion of the deposition processes, the reader is referred to Appendix B.

6.3.1 DRY DEPOSITION

As mentioned above, the dominant mechanism for dry removal of 1-micron-diameter aerosols (particles and vapors) is impaction on obstacles. Removal by impaction has generally been stated in terms of a deposition velocity, which is defined as the ratio of the deposition flux to the air concentration at some particular distance from the surface. Since in diffusion-controlled flow the flux is proportional to the concentration gradient, and not a single point concentration, it is apparent that in using the deposition velocity to compute the flux it is necessary to specify the parameters that control the concentration profile and diffusion coefficient. In general, however, it is not possible to account for such complexities in the atmosphere and environs that would influence dry deposition. Therefore, in the consequence model

a constant deposition velocity is utilized. This should be adequate if sufficiently broad ranges of the parameter are accommodated. The dry deposition flux of aerosols to the surface is taken to be $v_d\chi_0$, where v_d is the deposition velocity and χ_0 is the air concentration at essentially ground level. Dry deposition is assumed to proceed at all times, and variations in deposition velocity because of precipitation, surface wetting, vegetative cover, desorption, etc., are ignored. The consequence model uses a deposition velocity of 10^{-2} m/sec, with a possible range of 10^{-3} to 10^{-1} m/sec, for both particles and gases (except the noble gases, for which $v_d = 0$).

6.3.2 Wet Deposition

Precipitation scavenging of airborne material (wet deposition) can occur by the process of in-cloud scavenging or by below-cloud scavenging. These processes are discussed in Appendix B and only a brief outline is presented here.

In-cloud scavenging occurs when the airborne material stimulates or even initiates precipitation by condensation participation. This process has been identified as an important mechanism for the deposition of airborne debris from atmospheric nuclear weapons testing. If a convective storm is present, the released aerosols are swept up into the cloud and very effectively removed from the atmosphere. In below-cloud scavenging, the precipitation falling through the plume impacts upon and collects the airborne material.

To treat the wet deposition process in the consequence model, a simple exponential formulation is used. The plume concentration is assumed to decrease because of precipitation scavenging according to $\exp[-\Lambda(t - t_0)]$, where $(t - t_0)$ is the time since the onset of precipitation, at time t_0, and Λ is the wet removal rate. The removal rate Λ is taken to be 10^{-4} sec^{-1} under stable conditions (warm frontal storms) and 10^{-3} sec^{-1} under unstable conditions (convective storms). Particles and gases are treated identically. The noble gases are assumed to be insoluble and not removed by precipitation scavenging.

Since only information on precipitation occurrence within a given clock hour is available in the meteorological data file, an average rain duration of half the time within any specified hour of precipitation was assigned. This hourly fraction was deduced from a perusal of various studies of storm character (Marshall and Holtz, 1970; Austin and Houze, 1972; Zawadski, 1973).

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Section 7

Finite Distance of Plume Travel

When the atmospheric dispersion of airborne releases of gases and aerosols from a nuclear reactor accident is considered, it is important to realistically estimate the final history of the radioactive material. In the approach taken in this study, single-station meteorological data have been used in the Gaussian plume model to characterize travel over large distances. This approach introduces substantial uncertainties in the exact location and concentration at distances greater than a few tens of kilometers and at travel times greater than tens of hours. (As discussed in section 5, the accuracy that is needed to make reasonable calculations of the effects justifies this method.) By the time the cloud is a few hundred miles from the reactor, it would be spread over a large area and possibly over a considerable height. To accurately predict the health consequences, this very dilute concentration at far distances must be included since a linear extrapolation to low doses is used to calculate the incidences of latent cancer and genetic effects.

When a contaminant is released into the atmosphere, it will normally have a finite residence time before it approaches the natural background level. Since the primary concern in the lower atmospheric layers is with tropospheric aerosols, the study of Junge and Gustafson (1957) offers a guideline for estimating aerosol residence time. Another useful study has been performed by Machta (1970). The basic assumption of these works is that tropospheric aerosols wash out from the lower layers of the atmosphere with mean residence time of 2 to 4 days (48 to 96 hours). Residence time (Bolin and Rodhe, 1973; Eriksson, 1961; Eriksson, 1971) is defined as the expected lifetime in the atmosphere for particles and should be distinguished from the length of time required to reduce a concentration to an arbitrary safe level. The residence time for dry deposition is given in terms of the variables of the atmosphere surface boundary layer. Bolin et al. (1974) have investigated this problem in terms of the height of emission, roughness of the surface, deposition velocity, and wind speed. The times that are calculated are for the time period that an emission will remain over a particular point. No allowance was made for the three-dimensional dispersion patterns within the atmosphere. Bolin and Rodhe (1973) conclude that, compared to wet deposition processes, the effect of dry deposition on residence time is not important.

Clearly, to be realistic, one must allow for the distribution of rainy and dry periods. One way of doing this has been developed by Rodhe and Grandill (1972). By knowing the precipitation intensity (mm/hr) as a function of the percentage of time during which precipitation occurred and solving the appropriate Markhov process, they were able to estimate the average removal times for aerosols. They only considered one year at one Swedish location for their precipitation distribution function. It is interesting to note that for an arbitrary time of release and over a rather large range of conditions, they obtained expected removal times (turnover time) of 35 to 80 hours in winter and 100 to 300 hours in summer. With scavenging coefficients (see section 6.3) of 0.4 hr^{-1} in summer and 0.25 hr^{-1} in winter, the removal times are 150 and 50 hours, respectively.

The complementary cumulative distribution function for travel time to 500 miles is shown in Fig. VI 7-1. The average wind speed distributions from the six meteorological data sets, as given in section 5, indicate that the radioactive plume would require 100 hours or more to reach a distance of 500 miles from the reactor for 50% of the release times. Since most of the samples equal or exceed the measured turnover times, it appears reasonable to deposit the remaining airborne radioactive material (except noble gases) at 500 miles and thereby calculate the remaining population dose.

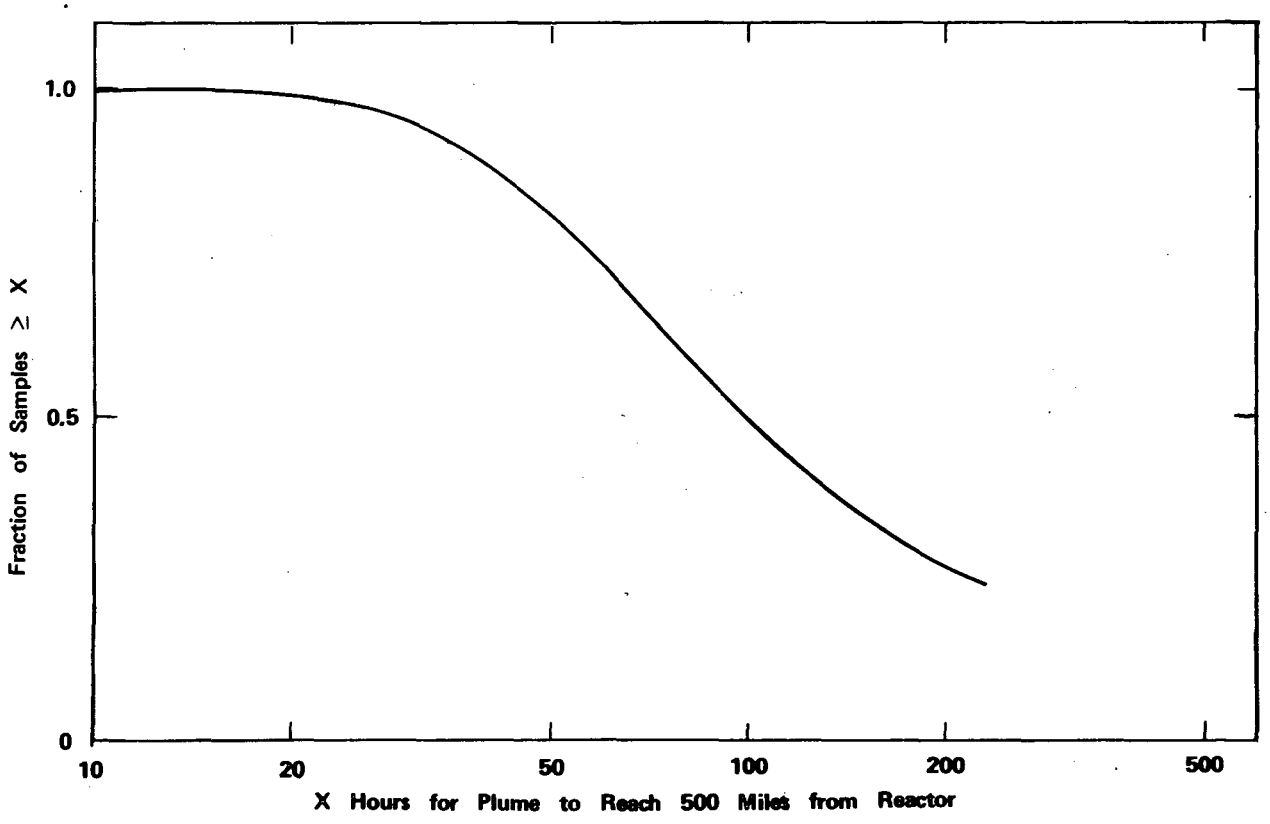

FIGURE VI 7-1 Complementary cumulative distribution function for plume travel time to 500 miles.

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Section 8

Dosimetric Models

8.1 INTRODUCTION

The release of radioactive materials to the environment constitutes a potential hazard to man. The word "potential" is used to stress the point that, for all practical purposes, man's concern about radioactive materials in the environment exists primarily when the material is in sufficient concentrations to impose a radiological burden higher than some value which the particular individual finds acceptable in light of the exposure he receives from natural sources. There are, therefore, two basic elements to the problem of assessing the hazard to man of radioactive materials in the environment. First, the concentration of the radioactive material in the environment (air, soil, water, on and in vegetation, animals, etc.) must be established. Second, mechanisms of exposure to be considered must be selected, since for the radiation to act upon the human there must be mechanisms by which the individual is exposed to the radioactive materials. The exposure may be a one-time interaction, or it may be on an essentially continuous basis.

Much of the past research to assess the hazards of radioactive materials in the environment is not directly applicable to the problem considered here: the instantaneous, or short-term, point-source release. Natural processes in the environment are constantly at work on most forms of released contaminants to disperse and dilute them. If the releases of the contaminants are essentially continuous, the concentrations of the contaminants at points along the various dispersion paths generally reach equilibrium values; the time constants to establish these equilibrium values may, in some cases, be quite large. The population is exposed to this equilibrium value or some value directly proportional to it. This type of problem is addressed in assessing the hazards due to global fallout from atmospheric testing of nuclear weapons or due to low-level continuous releases from nuclear facilities.

In the event of an instantaneous, or short-term, release, the concentration of the contaminants along the various dispersal paths generally builds up to a maximum value shortly after the release and decreases steadily thereafter. Therefore, it is paramount in this type of problem to describe accurately the time-dependent behavior of the contaminants in the environment in order to make adequate predictions of the hazards to which the population is exposed. A small change in a time constant can significantly change the magnitude of the exposure.

To illustrate these problems, let us consider the sequence of events that would follow an airborne release of relatively short duration. While the material was airborne the "cloud" would be dispersed and become diluted by turbulent diffusion. When the cloud passed over an area occupied by people, they would be exposed to the radiation emanating from the cloud and they might inhale some of the material. The latter exposure mode would exist only for as long as the people were immersed in the cloud. The cloud of material would be continuously depleted by radioactive decay and by deposition of the material onto exposed surfaces. The exposure of people from the passage of the cloud is generally called early exposure because it would be of relatively short duration and the radioactive material would be relatively concentrated. This phase of the radiation exposure of the population is discussed in section 8.2.

In contrast, the exposure of people to the material deposited out of the cloud is generally called chronic exposure. Here the length of exposure would be long (measured in years) because the material would contaminate the environment

in which the people reside. In addition, since the quantity of material deposited in a given area would generally represent only a small fraction of the material present in the passing cloud at that point, the dose rates received from chronic exposure would be significantly lower than those from early exposure. The presence of the deposited radioactive material in the environment would expose the population to radiation through several modes: irradiation by the material in a person's immediate vicinity, inhalation of material resuspended into the air, or ingestion of material contaminating the water or food supply. These modes of exposure are discussed in section 8.3.

Once the exposure (both early and chronic) is known the dose must be evaluated. Different radionuclides will have different chemical behavior, and so will be transported and relocated to different organs and tissues of the body. The dosimetric models utilized to relate curies of radioactive material to radiation doses are described in section 8.4.

8.2 SHORT-TERM EXPOSURE

In this section the methodology employed for the calculation of the radiation dose received within days after the accident is discussed. These doses are a result of the exposure that people would receive from external radiation from the radioactive cloud as it passed over the population areas, the dose to the body organs from internally deposited radioactive material from inhalation, and external radiation from radionuclides that would be deposited on the ground. Each of these exposure modes requires elaborate dosimetric models for the accurate calculation of radiation doses received.

8.2.1 Identification of Significant Radionuclides

A nuclear reactor that has operated for many hundreds of hours contains several hundred different radionuclides - almost the full range of the chart of the nuclides. Not all these radionuclides need be considered in the calculation of exposure from accidental release. With very little sacrifice in the accuracy of the calculated consequences, the number of radionuclides considered can be reduced to a manageable size. The selection of the radionuclides for dose calculations is discussed here.

The first criterion for the selection of radionuclides is their radioactive half-life as described in section 3.3. The second criterion applied was based on total activity in the reactor. If the activity of the radionuclide was several orders of magnitude lower than the activity of some of the most prevalent radionuclides, it was eliminated from consideration. The remaining radionuclides were grouped by chemical behavior (e.g., noble gases, actinides). Within the same chemical group, radionuclides having similar decay schemes (radiation type, energy, and daughter products) were grouped, some were eliminated because they contributed only a few percent of the total dose from the group. In other words, the nuclides were compared from approximate relative dose calculations.

The number of radionuclides considered was thus reduced from several hundred to 54 with little loss of accuracy. Table VI 3-1 lists these 54 radionuclides.

8.2.2 EXTERNAL EXPOSURE

In the calculation of external doses, two modes of exposure are considered:

1. Immersion in contaminated air

2. Irradiation by exposure to a contaminated ground surface.

Clearly the dose from external radiation to an individual exposed to the radioactive cloud would occur only while the cloud remained nearby. The details of this calculation are given in section 8.2.2.1. The material deposited from the passing cloud could also provide an external exposure. The factors affecting this exposure and the method of calculation are discussed in section 8.2.2.2.

8.2.2.1 External Exposure from the Radioactive Cloud

External radiation from a radioactive cloud would last only as long as the cloud was sufficiently close to the receptor. Since the mean free path of energetic gamma rays in air is quite long, the individual need not be immersed directly in the radioactive cloud to receive external exposure.

The gamma-ray dose can be calculated as the radiation received by an individual from a differential volume regarded as a point source. The radiation is then integrated over the entire cloud, taking into account the geometry of the cloud, variations in attenuation between the source and individual, and scattering of gamma rays from material outside this direct path. This type of calculation is possible but time consuming if cloud changes with distance due to atmospheric dispersion are considered. However, atmospheric dispersion acts rapidly and the cloud becomes large enough within a short travel distance for approximations to the external dose to be made with little sacrifice in accuracy. This approximation rests on the assumptions that the cloud is semi-infinite and the concentration of radioactive material is uniform. For the short travel distance that the cloud is not considered "large", a correction factor can be applied to the approximate method. The advantage of this procedure is that the gamma dose calculation is decoupled from the cloud dispersion calculation. The gamma dose calculations thus need be performed only once for a unit cloud concentration and supplied as input to the consequence model.

The dose received from the radioactive cloud is calculated in the consequence model as the product of the cloud exposure (in curie-seconds per cubic meter) and the semi-infinite cloud gamma dose-conversion factor D_{∞}^{C} (in rem per curie-second per cubic meter). The cloud exposure is the cloud concentration multiplied by the time that an individual would be exposed to the cloud. It is calculated, as discussed in section 4, at each particular distance of concern. The calculation of the semi-infinite cloud-dose conversion factors is discussed in section 8.4.1 and tables of the values used in the consequences model are given in appendix C. If the dimensions of the cloud were small compared to the range of the gamma rays in air, as would be the case near the source of the cloud, the doses would be smaller than those estimated by the above method. This can be corrected by applying correction factors to the dose calculation to determine the dose from a cloud of finite size. For the consequence model a simple, yet conservative, approach has been adopted. To evaluate the approximations and limitations of these cloud dose calculations it is important to understand how the consequence model treats the radioactive cloud.

At some point removed from its physical origin, the cloud is viewed as a rectangular slab of radioactive materials. The width of the cloud is specified by the weather stability class and the expected wind shifts, the thickness in the direction of travel is assumed to be small compared to the length of a spatial interval, and the height is given by the standard deviation of vertical diffusion. Only in the vertical direction is the concentration of radioactivity allowed to vary. The calculations for the finite-cloud dose corrections are based on a sphere with a uniform concentration of radioactivity. This sphere is assumed to have a radius equal to one standard deviation (σ_z) in the vertical height of the cloud in the consequence model. The concentration of radioactivity in the sphere is the same as that at the centerline of the cloud, and the sphere's center is positioned on the axis of the cloud. This is shown in Fig. VI 8-1 where the centerline of the cloud is a distance of z' above the ground. For this spherical model, dose calculations can be performed for a finite sphere and a cloud of infinite extent to find the ratio of the two doses (D^{C}/D_{∞}^{C}). Slade (1968) has performed such calculations and has tabulated the results as a function of σ_z and z'. His results, given in Table VI 8-1, are utilized in the consequence model for the finite cloud corrections.

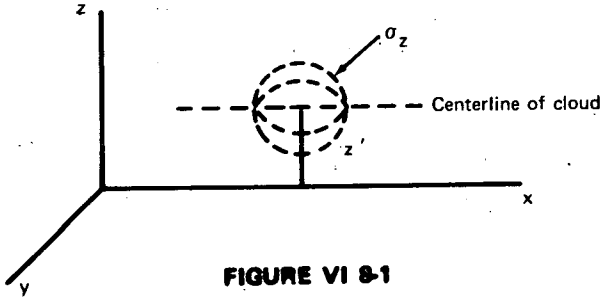

FIGURE VI 8-1

Geometry for finite-cloud correction

The procedure for calculating the finite cloud dose in the consequence model is as follows: the dose is first calculated as if the receptor were located in a semi-infinite medium with a uniform concentration equal to that at the centerline of the actual cloud. A correction factor to this dose is then applied to account for the fact that the cloud is finite and that the receptor need not be on the centerline of the cloud. This correction factor is read from the values given in Table VI 8-1. To get a continuous spectrum of factors, logarithmic interpolation between the values is performed as necessary. When the range of the table is exceeded, no extrapolation is done--rather interpolation at the edge of the table is performed. The external exposure is the product of χ, D_∞^C and (D^C/D_∞^C), where χ is in curie-seconds per cubic meter and D_∞^C is in rem per curie-second per cubic meter.

8.2.2.2 External Exposure from Ground-Deposited Radionuclides

The passing cloud of radioactive material would deposit material on the ground by the processes of wet and dry deposition, as discussed in section 6. This deposited material would provide a source of gamma radiation for a long time after cloud passage. The amount of material on the ground would depend on the degree of dispersion of the cloud and the deposition process.

In this section, the procedure used to calculate the initial radiation exposure of individuals by the deposited radionuclides is discussed. If it became necessary to relocate people to avoid long-term radiation doses from the contaminated ground in excess of the allowable exposure criteria (see section 11.2.1), there would be some delay before relocation could take place. Even if people started being evacuated at or before the time of cloud passage, there would be some short period of unavoidable exposure to the contaminated ground.

Because of the short time period of interest here it is not necessary to consider effects of weathering on the deposited material. Therefore, the calculation is not as complex as that for long-term external exposure (section 8.3).

TABLE VI 8-1 FINITE-CLOUD DOSE CORRECTION FACTORS (D^C/D_∞^C) [a,b]

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] $D^C/D_\infty^C = \frac{\text{dose from spherical cloud}}{\text{dose from semi-infinite cloud}}$

The short-period dose received from the ground is calculated in the consequence model as the product of ground contamination level (in curies per square meter) and a time integral dose-conversion factor D_{∞}^{g} (in rem per curie per square meter). The time integral dose-conversion factor is derived for a point one meter above an infinite smooth plane surface source and includes radioactive decay and daughter product buildup in the calculation of the radiation dose, as explained in section 8.4.1. Therefore, it is necessary to apply a shielding factor (also known as the dose reduction factor) to account for shielding provided by ground roughness, structures, vehicles, etc. The shielding factors are discussed in section 11.3.2.

A reference dose is calculated for a hypothetical infinite smooth plane source. Shielding for ground roughness, structures, etc., is accounted for by introducing a shielding factor (SF). Typical shielding factors are stated in Table VI 11-8. The external exposure dose from radioactive material deposited on the ground is calculated from

$$GC(D_{\infty}^{g})(SF),$$

where GC is in curies per square meter and D_{∞}^{g} is in rem per curie per square meter.

8.2.3 EXPOSURE FROM INHALED RADIONUCLIDES

During the period when individuals were immersed in the radiatioactive cloud, the air they breathed would contain radionuclides. Some of this activity would be breathed out again immediately, but some fraction would remain on the surface area of the respiratory tract. The amount of radioactivity retained would depend upon the particle size distribution, its chemical state, and the age of the individual.

The breathing rate for standard man is 2.3×10^{-4} m^3/sec (2×10^4 liters per day) averaged over 24 hours. Normally, about one half of the air is inhaled during the nominal 8-hour working day. Since people could be in an active state during passage of the cloud, the breathing rate is assumed to be 2.66×10^{-4} m^3/sec (2.3×10^4 liters per day) for inhaled radioactive material from the cloud. Different breathing rates are used for non-adults as stated in Table VI 8-6.

The dose is calculated as the product of the time-integrated air exposure (curie-seconds per cubic meter), the breathing rate (cubic meters per second), and the dose-conversion factor (rem per curie inhaled). The calculation of the dose conversion factors is described in Section 8.4.2.

8.3 CHRONIC EXPOSURE FROM DEPOSITED RADIONUCLIDES

This section discusses the methods used in the consequence model to calculate the long-term radiation exposure of an individual residing in a radionuclide contaminated environment. The radiation exposure modes are irradiation from surface deposits, inhalation of resuspended radionuclides, and ingestion of contaminated food.

The radioactive material deposited on surfaces in the environment would result in a continuous exposure of the population. Because of their long mean free paths, the gamma rays would be of particular importance. For a given level of contamination, the radiation dose received by an individual is determined by the amount of shielding that would exist between him and the deposited material. This form of radiation exposure would require no additional environmental transport and would result in the most restrictive limits on the utilization of the contaminated environment. We shall call this mode of radiation exposure "external exposure". The time-dependent behavior of the material on the surfaces would determine the total dose received by an individual over a given period of time. Depending on the forces binding the material to surfaces, some material could be washed away by rain and other water sources, to drainage systems. On porous surfaces the material could penetrate inward, and in soil the material could migrate appreciable distances downward. These factors reduce the dose that would be received by an individual either because the distance between individual and material is increased, or because of the additional shielding effect that results.

The other radiation exposure modes mentioned above require some additional transport mechanism before a radiation dose can be received by an individual. For this reason the calculation of the possible radiation exposure in these other modes is more difficult and uncertain but less important.

One of these modes is the resuspension into the air of the deposited material, and its subsequent inhalation. Resuspension would be a result of mechanical disturbance of the deposited material and entrainment in moving air adjacent to the surface. Small aerosol particles adhere well to surfaces and are therefore not easily resuspended. Hence only a small amount of the total deposited material would be resuspended. This is an important means of radiation exposure for transuranic nuclides since, in general, they emit no penetrating radiation nor are they generally available for ingestion. With time, the deposited material would become trapped under root mats, penetrate into the ground, or become unavailable to man by other mechanical means. Therefore, the fraction of deposited material resuspended generally decreases with time.

The remaining mode of radiation exposure is ingestion, and there would be two distinct periods of ingestion hazard. Immediately after deposition a significant portion of the radioactive material would be deposited on vegetation that is consumed by man, or by animals furnishing food products for man. This is called here "direct contamination". Only a single crop would be affected by direct deposition, so that the potential exposure would exist for less than 1 year. (This is the only significant mechanism for ingesting the short-half-life radionuclides such as iodine-131.) The level of contamination on the vegetation would decrease with time because of the influence of weather; for example, wind and rain are very effective in removing deposited material from vegetation.

The radioactive material deposited on the soil would be available for incorporation into the vegetation either by absorption at the base of the plant or uptake through the plant roots. We shall call this radiation exposure mechanism "incorporation into vegetation". It is a long-term radiation exposure mechanism and is relatively small compared to the others discussed above. The radioactive material contaminating the soil would be available for plant uptake over a period of several years, but generally only a few percent, at most, would be taken up by plants in one growing season. With time the material may move to depths below the root zone, or it may become unavailable for uptake by plants.

The models utilized to calculate acceptable contamination limits for these various radiation exposure modes are discussed in the following sections. The bases for the models and the associated data are given in Appendix E.

8.3.1 SELECTION OF SIGNIFICANT RADIONUCLIDES FOR CHRONIC EXPOSURE

The radionuclides deposited on the terrain after the passage of a radioactive plume would all contribute some fraction to the total chronic dose commitment of the exposed population. However, for each particular exposure mode, only a few of the radionuclides released from the reactor would contribute significant amounts of dose commitments. This is a result of many distinct factors including the radioactive half-life, the release fraction, the type and energy of radiation emitted, the chemical characteristics, and the metabolic behavior in man and animals of the radionuclides. Therefore, for the chronic exposure calculations, the 54 nuclides listed in Table VI 3-1 were screened to reduce the total number of radionuclides considered. This reduction significantly diminished the computation effort involved at a sacrifice of less than 1% in accuracy, which is substantially less than the overall uncertainty.

8.3.1.1 External Exposure

The selection of the radionuclides important in external exposure was based on their radioactive half-life and the type and energy of the emitted radiation. For a few weeks after deposition, the radioiodines would be the most important contributors to dose. After this decay of iodine, the isotopes of ruthenium would dominate the dose up to about 1 year. Thereafter cesium would clearly dominate the calculation of dose commitments. Therefore, of the 54 radionuclides, only those listed below were considered in the calculation of external exposure.

Radionuclide	Half-life (Days)
Cobalt-58	71.0
Cobalt-60	1920.0
Niobium-95	35.0
Zirconium-95	65.2
Ruthenium-103	39.5
Ruthenium-106	366.0
Iodine-131	8.05
Cesium-134	750.0
Cesium-136	13.0
Cesium-137	1.10×10^4

8.3.1.2 Inhalation of Resuspended Radionuclides

For this particular exposure mode, only radionuclides with appreciable half-lives would contribute significant doses over long time periods since only a small fraction of total deposited radioactive material would be resuspended. Therefore, the radionuclides considered are the transuranics and a few of the fission products, as listed below.

Radionuclide	Half-life (Days)
Strontium-90	1.03×10^4
Ruthenium-106	366.0
Cesium-137	1.10×10^4
Plutonium-238	3.25×10^4
Plutonium-239	8.91×10^6
Plutonium-240	2.47×10^6
Plutonium-241	5.35×10^3
Americium-241	1.58×10^5
Curium-242	1.63×10^2
Curium-244	6.63×10^3

8.3.1.3 Ingestion of Radionuclides

The metabolic characteristics of the radionuclides in man and animals determine which of them would contribute significantly to "internal" dose. These radionuclides have been identified as a result of extensive experimental studies on radionuclides from nuclear weapons fallout in man's diet. The radionuclides selected from the complete list of 54 were the following:

Radionuclide	Half-life (Days)
Iodine-131	8.05
Iodine-133	0.875
Strontium-89	52.1
Strontium-90	1.03×10^4
Cesium-134	750.0
Cesium-136	13.0
Cesium-137	1.10×10^4

The radioiodines are considered only for the ingestion of milk because of their short half-lives. A fuller discussion is provided in Appendix E.

8.3.2 EXTERNAL EXPOSURE FROM GROUND CONTAMINATION

Radionuclides deposited from the air onto the ground or other surfaces would present a large, nearly uniform thin layer of contamination. The population occupying the space above this thin layer would receive an exposure to radiation. This contrasts sharply with the other exposure modes, in which additional environmental transport would necessarily be involved. External exposure would mainly be gamma rays, except for close body contact, in which case some beta rays would also have an effect. The amount of radiation received from a contaminated surface would depend upon the amount of shielding between the contaminant and the receptor (human body) and on the length of time involved.

There are a number of very difficult problems that must be addressed to properly calculate the total external irradiation of the exposed population. Primary among the problems is an adequate description of the time-dependent behavior of the radionuclides on the surfaces. This behavior is, of course, a function of the surface properties, the chemical and physical form of the radionuclides, and the external forces acting on the surface. For example, the largest fraction of surface type exposed to depositing radionuclides in soil are the rate at which they would penetrate downward into the soil and the rate at which they would be removed by soil erosion. A clear understanding of the deposition and adherence of aerosol particles on hard surfaces (paved areas, structures, etc.) is essential to adequate calculations of long-term dose commitments to a city population. Unfortunately, however, few of the factors that influence particle adherence can be quantitatively defined. To calculate the protection afforded by structures it is very important to be able to describe the deposition of radioactive aerosols on vertical surfaces and the long-term behavior of the particles on the surfaces.

The best understood aspect of this problem is the effect of weathering on radionuclides deposited on the surface of soils. It has been well demonstrated that all radionuclides penetrate into the soil with time. The actual rate of movement depends on the chemical form of the radionuclide, the properties of the soil, and the external environment (e.g., rainfall, temperature fluctuations). The method used in the consequence model to account for the effects of weathering is based on extensive experimental data on the behavior of radionuclides deposited on soils. The actual model and supporting data base are described in Appendix E.

In the model the effect of weathering on the external radiation levels is represented by a function with two exponentials, and the effective fraction of deposited radioactive material after t years is

$$f_w(t) = 0.63 \exp(-0.693t/0.612) + 0.37 \exp(-0.693t/92.6). \qquad \text{(VI 8-1)}$$

Radioactive decay will decrease the external radiation levels further. Assuming for simplicity here only one radionuclide with a half-life of $T_{\frac{1}{2}}$ years, the external radiation decreases as

$$f(t) = f_w(t) \exp(-0.693t/T_{\frac{1}{2}}). \qquad \text{(VI 8-2)}$$

These relationships are shown in Fig. VI 8-2 for a radionuclide with a half-life of 30 years.

A simplified conceptual model of the total external radiation dosimetry model is shown in Fig. VI 8-3. With a specified external radiation exposure criterion, an acceptable level of contamination can be calculated for the particular mixture of radionuclides deposited. This is indicated in Fig. VI 8-3 as the horizontal line L_1. This acceptable contamination level is used to define the contaminated area within which the exposure exceeds the criterion. In Fig. VI 8-3, this contaminated area is indicated by the distance between the reactor and the point R_1. The size of the area considered between these points depends on the spread of the plume as it travels outward from the reactor.

The time-dependent behavior of the ground contamination level is calculated for radioactive decay and weathering. This behavior of the contamination level in Fig VI 8-3 is shown with the time axis. Without physical intervention, in a 10 year period the point R_1 has moved to R_1'. Therefore, the area between the reactor and R_1' will still exceed the acceptable contamination level L_1 after 10 years of radioactive decay and weathering. Strictly speaking, however, the level of acceptable ground contamination in Fig. VI 8-3 will also change because of the effect of radioactive decay on the fractional composition of the mixture of radionuclides. The effect will always result in raising the level L_1.

The major assumptions involved in the model for external exposure are the following:

1. The penetration of radionuclides into the ground is not disturbed by man (e.g., by soil cultivation for agricultural purposes).

2. There is negligible runoff of the radionuclides.

Concerning the first point, the partial burial of radionuclides by mechanical disturbance of the ground would provide more shielding. However, land that is normally tilled is mostly far from residential areas and from areas having appreciable occupancy (except by farmers). Since most of the external dose received by an individual would be from gamma radiation originating from contaminated surfaces less than 100 feet from the individual, the total reduction in dose by tillage would not be large. Therefore, the first assumption, although conservative, is not significant.

In areas of high population density, where a large fraction of the surface area is paved, one would expect that some of the deposited radionuclides would be washed into the drainage system within a short time. This expectation is supported by some studies on weapons fallout. However, the data are not adequate to provide any general guidelines for constructing a dosimetric model. Furthermore, the differences between weapons fallout and reactor releases, if they could be quantitatively stated, might show data to be unsuitable for the present study.

If one were to assume that there was little weathering of the contamination in areas of high population density because of the strong adhesion of the deposited material to the extensive paved surfaces, the use of Equation (VI 8-1) would result in an underestimation of the dose. This is compensated for by the two factors that city buildings shield each other and typically have more massive structures than are used in the calculations (see section 11.3).

Considering the above arguments, the use of a single external irradiation dose model appears to be justifiable and reasonable.

8.3.3 INHALATION OF RESUSPENDED RADIONUCLIDES

The inhalation of resuspended radioactive particles would not, by itself, impose restrictions on the use of a contaminated environment, because only a very small fraction of the deposited material would be resuspended. For example, the resuspension factor recommended most frequently for quiescent conditions outdoors is 10^{-6} per meter.[1] There

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[1]The resuspension factor K is defined as follows:

$$K = \frac{\text{air concentration } (\mu Ci/m^3)}{\text{surface deposit} (\mu Ci/m^2)} .$$

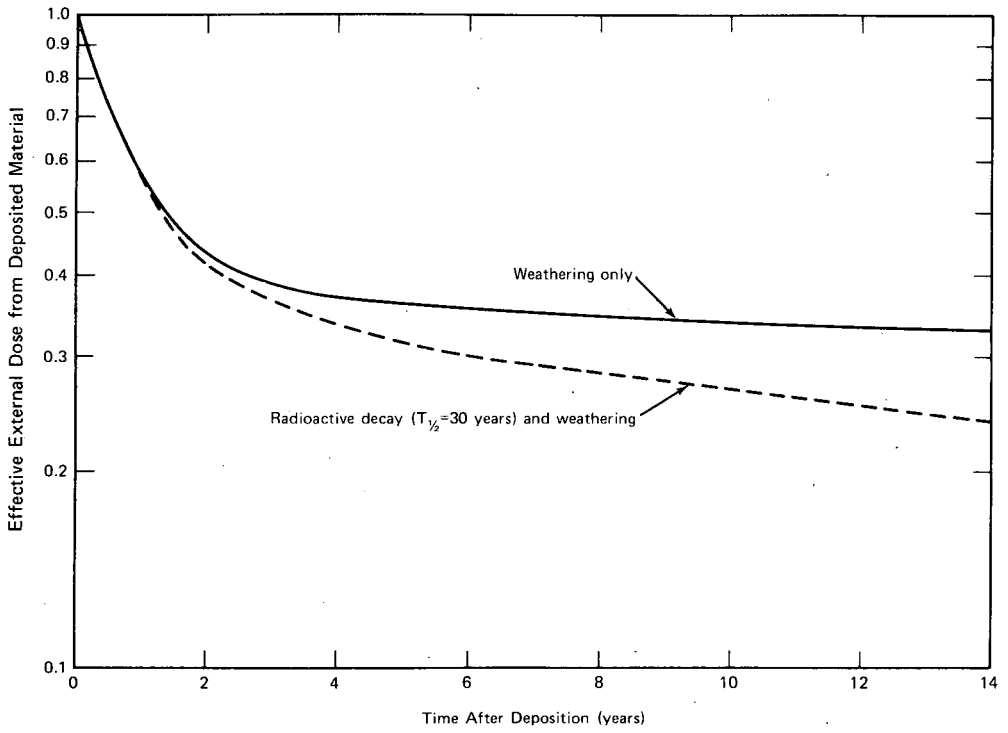


FIGURE VI 8-2 Time-dependent behavior of external dose from deposited radionuclides.

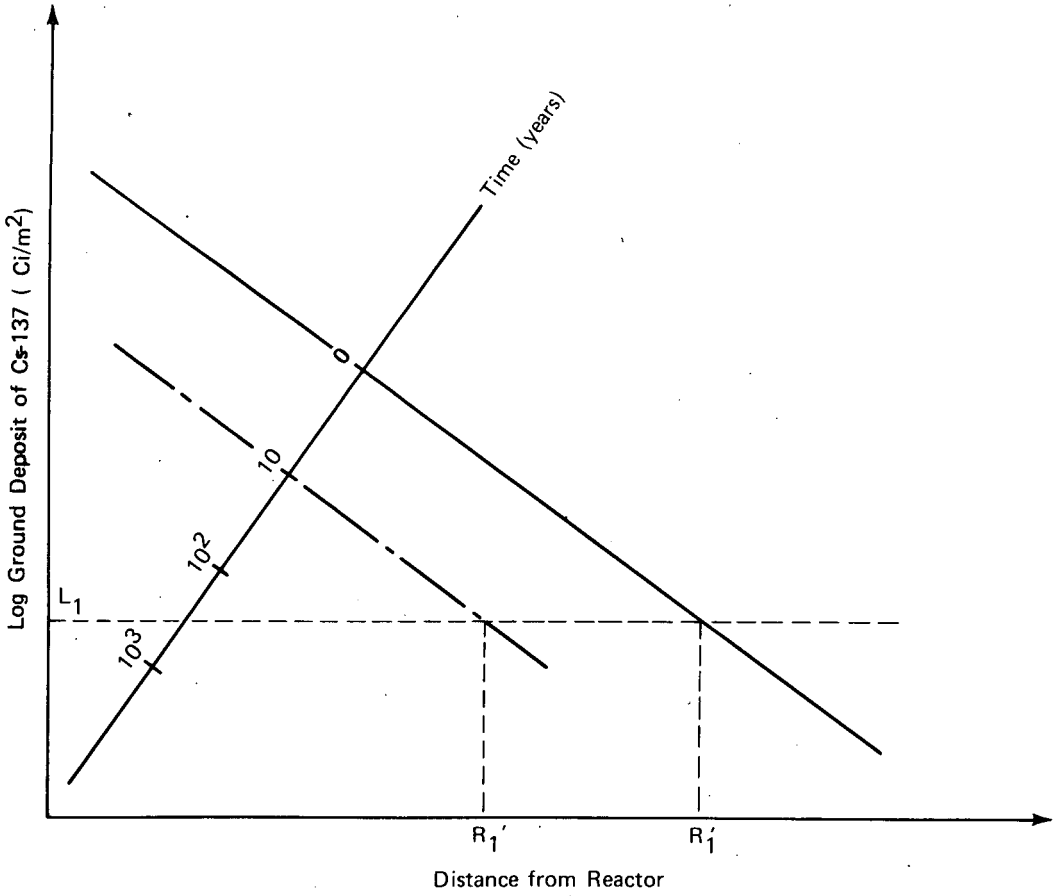


FIGURE VI 8-3 Chronic external exposure model (ground-level release).

are conceivable conditions under which the resuspension factor would be higher (e.g., driving on a dusty road), but the corresponding time period of exposure would be relatively small. Nevertheless, the resuspension and inhalation mode does contribute to the total exposure of a population living on contaminated land and is therefore included in the calculation.

In Appendix E the available experimental data from resuspension measurements are reviewed and a general model is developed. In this model it is assumed that the initial resuspension factor is 10^{-5} per meter. This value is considered adequate for the average population in relatively well vegetated areas. The initial resuspension factor is assumed to decrease with a constant half-life until it reaches a value of 10^{-9} per meter in 17 years, after which time it remains constant for the life of the radionuclide. The data reviewed in Appendix E indicate that the resuspension factor decreases much more rapidly immediately after deposition.

Furthermore, one could reasonably expect the resuspension factor to continue to decrease even after a period of 17 years, probably with increasing half-life. However, in view of the large uncertainties associated with the application of the limited data to the problem, these conservative assumptions are made. In summary, the time-dependent resuspension factor is assumed to be represented by the following equaiton:

$$K(t) = K_0 \exp(-\lambda t) + K_e,$$

where $K_0 = 10^{-5}\ m^{-1}$, $K_e = 10^{-9}\ m^{-1}$, and $\lambda = 0.677\ yr^{-1}$. This model for the time-dependent resuspension factor is shown graphically in Fig. VI 8-4. The reader is referred to Appendix E for a fuller discussion of the model for the inhalation of resuspended radioactive materials.

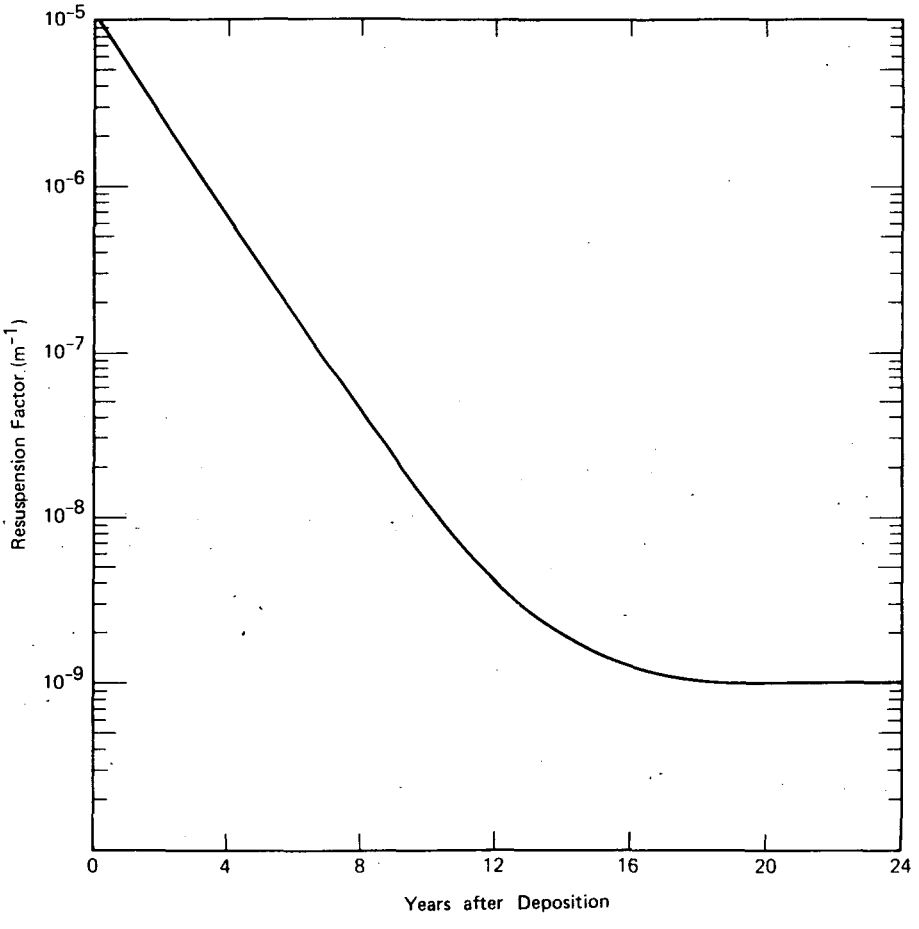
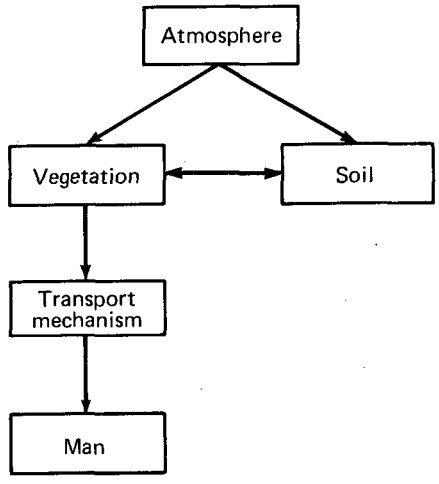


FIGURE VI 8-4 Time-dependence of resuspension factor.

8.3.4 INGESTION OF CONTAMINATED FOODS

Radioactive materials deposited in the environment may present a hazard when ingested as contaminated food and water. Consumption of contaminated plants is the most efficient mode for turning contamination into dose received. An area that is acceptable for other modes may be unacceptable for direct consumption of plants.

The contamination of food could result either by direct deposition on vegetation or by incorporation into the vegetation. This is best illustrated by a simple block diagram. The radioactive material (vapor, and aerosol) from the atmosphere is



deposited on the vegetation and on the soil. If the vegetation were not removed the material on it would eventually enter the soil by such means as being washed off by rain, blown off by wind, or by the death of the vegetation. Material deposited on the soil could be incorporated into the vegetation either by plant base absorption or by uptake from the plant roots. The contaminated vegetation may be eaten directly by man or by animals furnishing food products for man.

The direct contamination of vegetation would be a transitory problem since it would affect only a single crop. Disposal of the contaminated vegetation, or not using the affected vegetation, would eliminate the exposure by direct contamination. Incorporation of the radioactive material into the vegetation would affect many crops over many growing seasons, but presents a much reduced hazard in comparison with the ingestion of plant material bearing contamination on its surface since only a small amount of the deposited material (a few percent at most) would be taken up in one growing season.

8.3.4.1 Direct contamination of vegetation

The calculation of acceptable contamination levels involves a large number of parameters, many of which are poorly known and/or extremely variable. There can be a large variation in local conditions from the national averages which directly affect the level of contamination ingested, but since the areas affected are large, this variability is expected to average out. This implies that the effects of local "hotspots" would be offset by an individual consuming food from a wide area.

For each particular reactor site and date of accident considered in the model a test is made to determine whether the accident occurs during the growing season for crops or forage. If not, then direct contamination of vegetation is not considered to be an operative radiation exposure mode.

The major factors considered in the calculation of ingestion of radionuclides deposited on vegetation are (a) the fraction of deposited material initially retained on the vegetation, (b) its behavior on the vegetation as a function of time, and (c) the possible mechanisms that would lead to eventual ingestion by man. The explicit models and data are described in Appendix E and only a brief discussion is given here.

The fraction of deposited material initially retained on the vegetation is taken to be 0.5. Weathering effects would reduce the amount of material remaining on the vegetation. The fraction remaining t days after deposition is described by the function

$$f_w(t) = 0.85 \exp(-0.693t/14.0) + 0.15.$$

In addition to weathering, radioactive decay would also reduce the amount of radioactive material remaining on the vegetation. This time-dependent behavior is illustrated in Figure VI 8-5 for a radionuclide with half-life of 8 days.

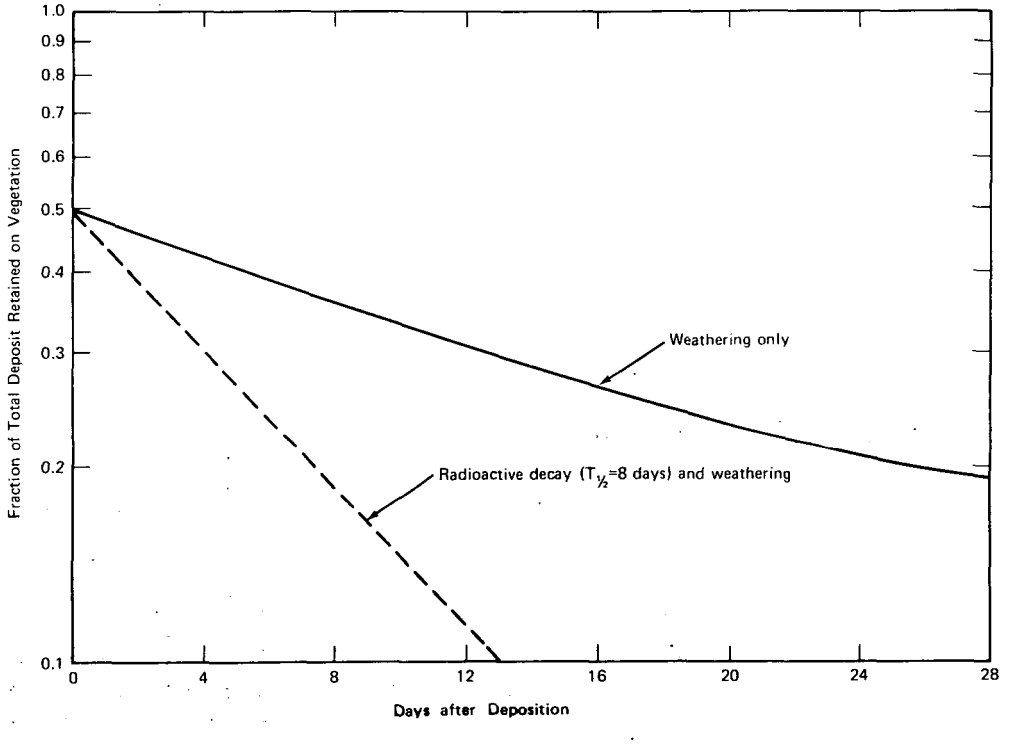


FIGURE VI 8-5 Retention of radioactive material on vegetation.

These relationships are used to determine the time required for vegetation contamination levels exceeding the established criteria to fall to an acceptable level. The criteria are based on the dose limits given in section 11.2.1 and models relating vegetation contamination and total quantity of radioactive material ingested by an individual, as given in Appendix F. These models consider vegetation contamination and total radionuclide ingestion from milk, milk products, meat, vegetables, and other foodstuffs.

8.3.4.2 Incorporation of contaminants from soil into vegetation

It is not necessary to calculate acceptable soil contamination levels for crop growing. The uptake of radionuclides by the plant roots would be an inefficient mechanism of radiation exposure. At most a few percent of the deposited radionuclides would be taken up by plants in one growing season. Furthermore, the fraction of material taken up declines rather rapidly in subsequent growing seasons. An area that had enough soil contamination to produce unacceptable doses by plant uptake and ingestion of food plants would be forbidden already by other restrictions (e.g., external irradiation). This can be demonstrated with a simple calculation. Assuming, conservatively, that an individual were to receive all of his food requirements from the contaminated land and that one-half of the total strontium-90 transfered from soil to man over all time, as calculated in Appendix E, were ingested in the first day after deposition, an individual would ingest 1.005 μCi of strontium-90 per $\mu Ci/m^2$ of surface soil contamination. With a dose criterion of 0.5 rem to the bone marrow in the first year and a dose conversion factor of 0.0425 rem in the first year per μCi intake, the acceptable contamination level of the soil is

$$SD = \frac{0.5 \text{ rem}}{(0.0425 \text{ rem}/\mu Ci)\ (1.005\ \mu Ci/(\mu Ci/m^2))}$$

$$= 11.7\ \mu Ci/m^2 \text{ for strontium-90.}$$

The corresponding cesium-137 contamination of the soil can be found from the ratio of cesium-137 to strontium-90 in the reactor core before the accident and the ratio of their respective release fractions.[1] The ratio of the reactor core activity is 1.27 and the release fraction ratio varies from 5 to 5×10^4. Therefore, the cesium-137 contamination of the soil would be between 74.3 and 74.3×10^4 $\mu Ci/m^2$. The lower value would result in an external whole body radiation exposure of 13.2 rem in 30 years (using the methods described in section 8.3.2) which exceeds the criterion of 10 rem in 30 years (see Table VI 11-6).

Uptake by plant roots should not be neglected as a mode of radiation exposure because it does contribute to the total dose commitment received when the contaminated land is used for agricultural purposes. The dose commitment for this mode is calculated as for direct contamination of vegetation. Rather than initial retention by vegetation and subsequent weathering, the important factors are rate of uptake by plants and rate of decrease of availability to plants (e.g., by leaching to below root zones). Other than these two differences, the methods are conceptually the same. The model and supporting data base are explained in Appendix E.

8.4 DOSIMETRIC MODELS

As discussed in sections 8.2 and 8.3, the calculation of the radiation doses requires appropriate dose conversion factors. These dose conversion factors, for the incorporation of radioactive material in the body, give the dose received by individual organs over a time interval per curie intake by inhalation or ingestion. For external exposure, the dose conversion factors give the dose received by each organ per curie of radioactive material in a cubic meter of air or per curie of radioactive material deposited uniformly on a square meter of horizontal surface. The calculation of these dose conversion factors requires elaborate computer models with appropriate physiological parameters for a human body. Fortunately these calculations need only be performed once for each radionuclide, organ, exposure mode, and time interval. From these calculations, a table can be prepared for use in the consequence model. The manner in which these dose conversion factors are calculated for a "standard" adult is discussed in the following sections and appendices C and D. Section 8.4.3 describes appropriate corrections to be applied to these dose conversion factors to calculate doses to the organs of children.

8.4.1 EXTERNAL EXPOSURE

All external dose conversion factors were calculated with the EXREM III computer program developed at Oak Ridge National Laboratory. Photon doses to individual organs were calculated for immersion in a semi-infinite radioactive cloud and exposure to an infinite smooth plane of contamination. The dose from the infinite smooth plane was calculated at a distance of 1 meter above the plane. The calculations with EXREM III were set up so that all daughter contributions to dose were added to the parent radionuclide of the decay chain. The details of these calculations are described in Appendix C. Tables of dose conversion factors are also given in Appendix C for a few organs for immersion in contaminated air and exposure to contaminated ground.

8.4.2 INTERNAL EXPOSURE

Inhalation or ingestion of radionuclides will result in their incorporation in the body and a subsequent dose to individual organs. The model used to determine the dose conversion factors for inhaled radionuclides is discussed in Section 8.4.2.1 and that for ingested radionuclides is discussed in Section 8.4.2.2.

8.4.2.1 Inhalation Model

Among the factors that must be considered in an inhalation model for the calculation of dose conversion factors are: (1) the chemical form in which the radionuclides exist; (2) the characteristics of the aerosol in which they occur, including relative abundance; (3) the aerodynamic behavior of the aerosol as they are inhaled and deposited in various sections of the respiratory system; (4) the movement of the aerosol within the respiratory tract and out of it into the lymphatic system and the gastrointestinal tract; (5) the absorption of the radionuclide into the bloodstream; (6) the distribution of the radionuclides among organs and tissues; and (7) excretion from the body.

The chemical forms in which each radionuclide might exist is important since it determines its solubility, transfer through membranes, distribution among the various organs of the body, and excretion from the body. The most likely chemical state of the released radionuclides was determined and a suitable class was assigned. Such a class is denoted by either D, W, or Y which represents the longest clearance half-times from the pulmonary region on the order of days (D), weeks (W), and years (Y) respectively. The classification of radionuclides in this manner for the inhalation model is given in Table VI 8-3. In general, because of the highly oxidizing environment that would accompany a reactor accident, oxide formation is expected. For several of the transition elements, the formation of oxygenated anions is also possible. Molybdenum oxides are assigned to class Y, whereas the molybdates are assigned to class D. Since molybdenum-99 has a short half-life of 218 days, a small overestimate of the lung dose will result. The iodines can exist as elemental iodine, hydroiodic acid, or organic halides. The possible formation of hydroiodous acid may also lead to the presence of iodides and iodates. The categorizing of the aerosol clearance from the pulmonary region does not include the noble gases.

The inhalation model is the ICRP Task Group model, with some changes in the parameters to reflect newer data (Morrow, 1975a and 1975b; Lindenbaum et al., 1972).[1] The percent of deposition (or deposition fraction) of radionuclides in the regions of the respiratory tract as a function of particle characteristics is shown in Fig. VI 8-6 (Morrow, 1966). This deposition model estimates the fraction of inhaled activity deposited in the naso-pharyngeal (N-P) region, in the tracheobronchial (T-B) region, and in the pulmonary (P) region as functions of the activity median aerodynamic diameter (AMAD). The most important factors that influence the distribution of a radionuclide are the size of the particles and the chemical form. If the chemical compound is readily soluble in physiological fluids, it will easily pass into the blood.

The deposition model provides estimates of the amount of contaminated material deposited in the regions of the respiratory tract. A schematic representation of the deposition sites and clearance processes is shown in Fig. VI 8-7. This retention model gives the different absorption and translocation processes for the clearance of the compartments. There is a small, but finite, possibility for uptake of matter deposited in the nasal-pharynx to pass directly into the systemic blood (pathway a in Fig. VI 8-7). Dusts in the nasal-pharynx are cleared by mucus transport (pathway b). Aerosols deposited in the tracheobronchial compartment are absorbed into the blood (pathway c). Clearing the throat removes material directly into the gastrointestinal tract (pathway d). The translocation of dust from the pulmonary region to the blood is shown as path e. The aerosol cleared by pathways f and g goes to the gastrointestinal tract via the tracheobronchial tree. There is a slow removal of radionuclides from the pulmonary compartment through the lymphatic system (pathway h). The material cleared by the lymphatic system is introduced into the blood (pathway i). The radioactive material in the gastrointestinal tract can be directly transported into the blood (pathway j). After the material is in the blood system, it is deposited in various organs and tissues. For the noble gases (Xe and Kr) the ICRP Task Group Model is not applicable, and a separate retention model developed by Bernard and Snyder (1975) is utilized. Any exposure by inhalation also leads to some of the activity entering the gastrointestinal tract, and thus the model of the gastrointestinal tract is involved in all the dose estimates.

[1]References are given in Appendix D.

The dosimetric model for the gastrointestinal tract is essentially that due to Eve (1966), insofar as the subdivisions of the tract and the transit times through the sections are concerned. Four subdivisions of the tract are defined: the stomach, S, the small intestine, SI, the upper large intestine, ULI, and the lower large intestine, LLI. The estimates of dose are considered to be averaged over these sections.

Dose is computed by the method described by Snyder et al. (1974). In calculating the dose from photons, the specific absorbed fractions presented in that report are used.

To account for the attenuation of the beta radiation between the surface of the wall and the regenerative cells of the lower large intestine (LLI), the ratio of the dose at 500-micron depth to that of the surface of the wall was computed. This correction was applied only to the beta dose at the wall. The calculation of this correction factor was approximate but conservative; equilibrium was assumed and the highest effective beta energy of the radioactive chain was used. It was assumed that the fractional makeup of the radionuclides contributing to the dose would not change appreciably over time. Therefore, the fraction of beta dose to total dose at the wall was computed at 7 days after the inhalation of radioactive material. The results of these calculations are given in Table VI 8-3.

The internal dosimetric models are described in greater detail in Appendix D. In addition, selected tables of the dose conversion factors are given in Appendix D for various time intervals.

8.4.2.2 Ingestion Model

The inhalation model is modified to calculate dose conversion factors for ingestion of radionuclides. The calculations were performed by assuming that the radionuclides enter directly into the gastrointestinal tract from the respiratory system (i.e., the residence time in the respiratory system is zero). Ingestion is considered as an exposure mode for long-term , low-level activity present in the diet. The only radionuclides for which estimates of dose following ingestion are calculated are strontium-89, strontium-90, iodine-131, cesium-134, cesium-136, and cesium-137. The dose conversion factors for ingestion are given in Table VI 8-4.

8.4.3 CORRECTION FACTORS FOR CHILDREN

Because of differences in organ masses, ingestion rates, breathing rates and metabolism between children and adults, there is a variation of dose absorbed with age. For photon emitters, the dose per disintegration is higher for adults than for infants and children, and it can vary greatly with photon energy. For beta and alpha particles the dose per disintegration is inversely proportional to organ mass and will show considerable variation with age. The ratio of doses per particle to organs of individuals of different ages are found to be just inversely proportional to their organ masses. The ingestion and breathing rates of children are lower than for adults, but the child's metabolism is more rapid than the adult's and this can lead to more rapid elimination of radioactive material. Although the geometric and metabolic factors will tend to cancel each other, one cannot ignore the effects of age.

To account for the dose received by children, a correction factor is calculated by which the estimated adult dose should be multiplied. In considering age effects, only iodine-131, cesium-137, strontium-89 and strontium-90 are included. The details of the calculation are given in Appendix D with a summary shown in Table VI 8-5. The factors D_{ch}/D_{ad} in Table VI 8-5 are the ratio of the organ dose per unit of radioactivity inhaled and it accounts for the difference in organ masses. If this factor is multiplied by the ratio of the breathing rates, child to adult, the correction factor for children's doses is determined. In this study, no credit was taken for a child's dose being lower than an adult's; therefore a minimum factor of 1 was used.

For internal exposure from ingestion, the age dose factors are obtained by replacing the ratio of the breathing rate by the ratio of the ingestion rates.

TABLE VI 8-2 LUNG CLEARANCE CLASSES FOR THE RADIONUCLIDES INCLUDED IN THE DOSIMETRY MODEL

Group[a]	Radionuclides in Group	Expected Chemical Species Released from Containment	Assigned Lung-Clearance Class[b]
Halogens	I-131, I-132, I-133, I-134, I-135	I_2, CH_3I, iodides, iodates	D
Alkali metals	Rb-86, Cs-134, Cs-136, Cs-137	Oxides, hydroxides	D
Tellurium, antimony	Te-127, Te-127m, Te-129, Te-129m, Te-131m, Te-132, Sb-127, Sb-129	Oxides	W
Alkaline earths	Sr-89, Sr-90, Sr-91, Ba-140	Oxides	D
"Transition" group	Ru-103, Ru-105, Ru-106, Rh-105	Oxides, elemental	Y
	Co-58, Co-60	Oxides, hydroxides	Y
	Mo-99	Molybdates (possibly oxides)	
	Tc-99m	Oxide, pertechnetate	D
"Lanthanide" group	Y-90, Y-91, La-140	Oxides	W
	Zr-95, Zr-97, Nb-95	Oxides	Y
	Ce-141, Ce-143, Ce-144, Pr-143, Nd-147	Oxides	Y
	Np-239, Pu-238, Pu-240, Pu-241, Am-241, Cm-242, Cm-244	Oxides	Y

(a) The names "transition" and lanthanide" are in quotation marks because they are not employed precisely, merely as convenient labels

(b) The letters D, W, and Y represent respiratory clearance half-times on the order of days, weeks, and years, respectively

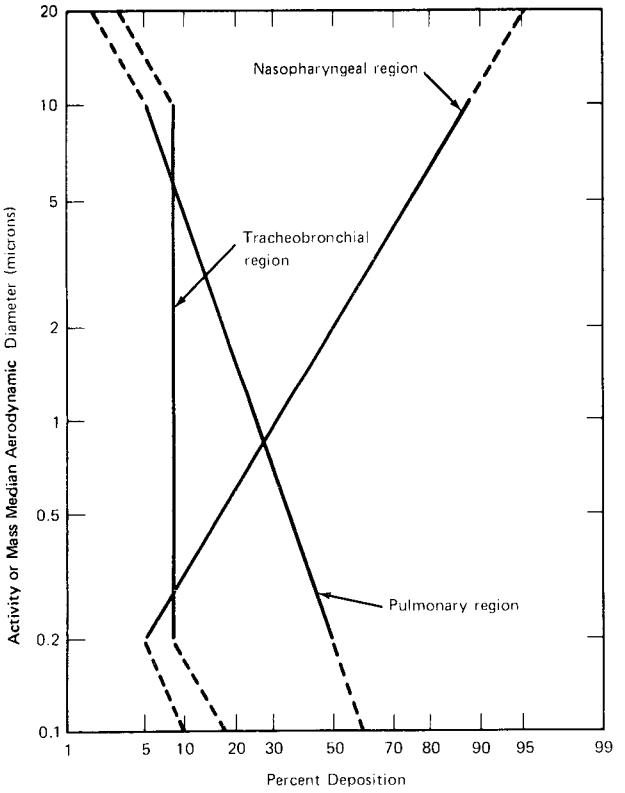


FIGURE VI 8-6 Deposition model. The radioactive or mass fraction of an aerosol that is deposited in the nasopharyngeal, tracheobronchial, and pulmonary regions is given in relation to the activity of mass median aerodynamic diameter (AMAD or MMAD) of the aerosol distribution. The model is intended for use with aerosol distributions that have an AMAD or MMAD between 0.2 and 10 microns with geometric standard devisions 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.

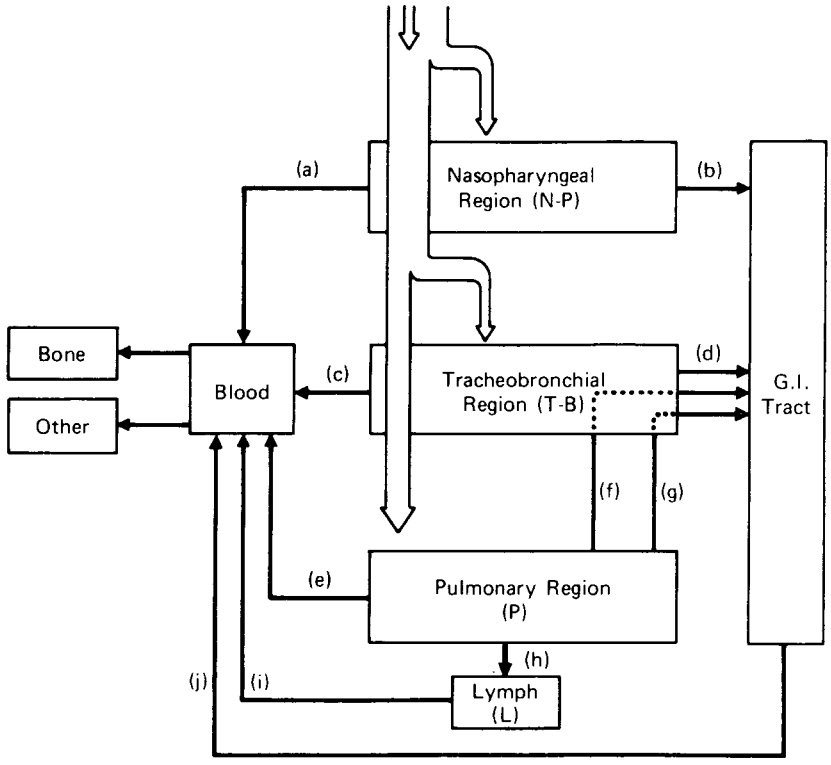


FIGURE VI 8-7 Retention model. See text for description of pathways (a) through (j).

TABLE VI 8-3 DEPTH DOSE CORRECTION FACTORS - LOWER LARGE INTESTINE (LLl), AND FRACTIONAL TRANSFER FROM GASTRO-INTESTINAL TRACT TC BLOOD (f_1)

Nuclide	Selected Average Energy (MeV)	Ratio A[a]	Ratio B[b]	Dose Correction Factor (1 - B + AB)	f_1
Co-58	.2	.09	.42	.62	0.05
Co-60	.1	.05	.46	.56	0.05
Kr-85	.37	.33	.99	.34	NA
Kr-85m	.28	.23	.74	.43	NA
Kr-87	1.33	.73	.72	.81	NA
Kr-88	2.1	.8	.65	.90	NA
Rb-86	.62	.50	.96	.52	0.95
Sr-89	.58	.49	1.00	.49	0.2
Sr-90	.93	.65	1.00	.65	0.2
Sr-91	.62	.50	.91	.55	0.2
Y-90	.93	.65	1.00	.65	10^{-4}
Y-91	.62	.50	1.00	.50	10^{-4}
Zr-95	.12	.05	.76	.28	2×10^{-3}
Zr-97	.71	.55	.94	.58	2×10^{-3}
Nb-95	.05	0	.55	.45	0.01
Mo-99	.40	.35	.97	.37	0.95
Tc-99m	low energy e^-	0	.37	.63	0.95
Ru-103	.06	0	.80	.20	0.04
Ru-105	.42	.37	.94	.41	0.04
Ru-106	1.42	.74	.99	.74	0.04
Rh-105	.17	.05	.97	.08	0.04
Te-127	.22	.23	1.00	.23	0.25
Te-127m	.26	.20	.99	.21	0.25
Te-129	.50	.43	.96	.45	0.25
Te-129m	.50	.43	.99	.44	0.25
Te-131m	.72	.55	.83	.63	0.25
Te-132	.51	.44	.80	.55	0.25
Sb-127	.34	.30	.93	.35	0.05
Sb-129	.73	.56	.90	.60	0.05
I-131	.18	.06	.83	.22	0.95
I-132	.51	.44	.47	.74	0.95
I-133	.42	.37	.85	.46	0.95
I-134	.66	.53	.30	.86	0.95
I-135	.32	.28	.71	.49	0.95
Xe-133	.1	.05	.73	.31	NA
Xe-135	.31	.27	.70	.49	NA
Cs-134	.15	.05	.26	.75	0.95
Cs-136	.11	.05	.18	.83	0.95
Cs-137	.20	.09	.57	.48	0.95
Ba-140	.49	.42	.88	.49	0.1
La-140	.49	.42	.84	.51	10^{-4}
Ce-141	.14	.05	.97	.08	10^{-4}
Ce-143	.37	.33	.96	.36	10^{-4}
Ce-144	1.21	.71	1.00	.71	10^{-4}
Pr-143	.31	.27	1.00	.27	10^{-4}
Nd-147	.23	.16	.97	.19	10^{-4}
Np-239	.14	.05	.97	.08	10^{-3}
Pu-238	-	-	.16	.1	3×10^{-5}
Pu-239	-	-	.13	.1	3×10^{-5}
Pu-240	-	-	.16	.1	3×10^{-5}
Pu-241	.005	0	1.00	0	3×10^{-5}
Am-241	-	-	.39	.1	10^{-3}
Cm-242	-	-	.14	.1	10^{-3}
Cm-244	-	-	.13	.1	10^{-3}

(a) A = (β dose at 500-μ depth)/β dose at 0 depth.

(b) B = "β" dose/total dose at 0 depth for LLI at 7 days.

TABLE VI 8-4 DOSE CONVERSION FACTORS FOR INGESTION OF RADIONUCLIDES (REM PER CURIE INGESTED).

	0-10 Yr	0-20 Yr	0-30 Yr	0-40 Yr	0-50 Yr
			Whole Body		
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}
			Total Marrow		
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}
			Bone (Mineral)		
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}
			Thyroid		
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}
			Lung		
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}

TABLE VI 8-4 DOSE CONVERSION FACTORS FOR INGESTION OF RADIONUCLIDES (REM PER CURIE INGESTED) (CONTINUED)

	0-10 Yr	0-20 Yr	0-30 Yr	0-40 Yr	0-50 Yr
		Lower Large Intestine Wall			
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.

TABLE VI 8-5 DOSE CORRECTION RATIOS D_{ch}/D_{ad} PER UNIT ACTIVITY INTAKE AND BREATHING RATES FOR VARIOUS AGES AND RADIONUCLIDES

Age (years)	Breathing Rate (10^{-4} 1/day)	Ratio (child/adult) Breathing Rates	Radionuclide	Organ of Reference	D_{ch}/D_{ad}	Age Dose Factor For Inhalation[a]
1	0.38	0.17	I-131	Thyroid	5.4	0.9
			Cs-137	Total body	1.1	0.2
			Sr-89 or Sr-90	Bone or red marrow	5	0.9
5	0.96	0.42	I-131	Thyroid	4.6	1.9
			Cs-137	Total body	1.2	0.5
			Sr-89 or Sr-90	Bone or red marrow	1	0.4
10	1.5	0.65	I-131	Thyroid	2.4	1.6
			Cs-137	Total body	1.1	0.7
			Sr-89 or Sr-90	Bone or red marrow	1	0.7
20 (adult)	2.3 (active)	1	I-131	Thyroid	1	1
			Cs-137	Total body	1	1
			Sr-89 or Sr-90	Bone or red marrow	1	1

[a] Age Dose Factor = D_{ch}/D_{ad} X Ratio of Breathing Rates (child/adult). When calculated Age Dose Factor is less than unity, a factor of 1 was used.

9. HEALTH EFFECTS

9.1 INTRODUCTION

This section describes the health effects that might be associated with a hypothetical release of radioactive material from a reactor. The clinical and experimental data on which the calculations are based are described in Appendices F, G, H, and I on the early and continuing somatic effects, late somatic effects, thyroid effects, and genetic effects.

The health effects that could be associated with a reactor accident are divided into three categories. Early and continuing somatic effects include the early mortalities and morbidities that are usually observed after large, acute doses of radiation and can occur within days to weeks after exposure; they also include illnesses and deaths that can become manifest within a year or so. In general, these early and continuing somatic effects are primarily associated with individual total-body doses of 100 rads or more and thus would be limited to persons in the immediate vicinity of the reactor.

The late somatic effects include latent cancer fatalities and morbidities as well as benign thyroid nodules. In radiation therapy experience, such effects are typically observed 2 to 30 years after irradiation. Finally, there are genetic effects, which do not manifest themselves in the irradiated individuals, but rather in their descendants. In contrast to the early somatic effects, both latent cancer and genetic diseases are random phenomena whose probability of occurrence is some function of the dose magnitude. Consider, for example, a large number of individuals who all receive an equal dose: one may develop leukemia 25 years later, another may have a grandchild with a genetic disease (e.g., diabetes). For this reason, both late somatic and genetic effects are calculated on the basis of population dose (cases per million man-rem) rather than individual doses. Late somatic and genetic effects may result from even very low doses but with a very low incidence.[1] Consequently, these effects may occur at long distances (e.g., 200 miles) from the reactor, at which a small dose might still be received.

As stated in the preceding sections, the underlying objective of the Reactor Safety Study is to make as realistic an assessment of risk as is possible and to indicate the uncertainties in the estimate. In conformance with this objective, three estimates have been made of the major health effects. The "central estimate" represents a judgment of the most likely health consequences, and the upper and lower bounds represent the range of uncertainty. For the less common health effects, the available clinical or experimental data are usually inadequate for more than one estimate.

Three units, the roentgen, the rad, and the rem, are commonly used in radiobiology. The roentgen refers only to X or gamma radiation and is a measure of the total charge produced by the photons per unit mass of air. The rad is the unit of absorbed dose and is a measure of the energy deposited per unit mass of irradiated material. The rem is applicable to all types of radiation (i.e., alpha, beta, gamma, and neutrons) and is the product of the absorbed dose in rads and several other factors that relate the deposited energy in rads to the resulting damage. For the predominantly low-LET[2] radiation that would result from a reactor accident, the rad and rem are numerically equal. Since most literature dealing with early and continuing health effects uses rads, this unit is used in section 9.2. Observed late somatic and genetic effects

[1] Scientific evidence is inconclusive with respect to the incidence of latent cancer from very low doses. For more discussion, the reader is referred to section 9.3 and Appendix G.

[2] Linear energy transfer (LET) is a measure of the rate of energy loss along the track of an ioinzing particle, expressed in units of energy per unit track length (thousands of electron volts per micron). Low-LET radiation includes beta particles and gamma rays; high-LET radiation includes alpha particles.

have often resulted from exposure to high-LET radiation, so these effects are usually related to doses in rem (i.e., dose equivalent); this unit is used in sections 9.3 and 9.4

9.2 EARLY AND CONTINUING SOMATIC EFFECTS

9.2.1 INTRODUCTION

As stated in the preceding section, the early and continuing somatic effects are directly related to the radiation dose received by an individual. The dose to a specified organ (e.g., lung) is calculated as a function of distance from the reactor, and it is assumed that all persons located at that distance would receive the same dose. The mortality or morbidity rates in this group are determined by comparing this dose to the dose-mortality (or dose-morbidity) criterion corresponding to that organ.

As described in section 8, there would be four exposure modes: external radiation from the passing cloud, external radiation from ground contamination, internal radiation from inhaled radionuclides, and internal radiation from ingested contaminated crops. The last-named mode would not contribute to early and continuing somatic effects. The external dose from the passing cloud would be received over a period of 30 minutes to a few hours, depending on the duration of the release.[1] If a radioactive nuclide were inhaled, its radioactive decay would continue to irradiate various organs internally over a period of days to years, depending on the effective half-life of that particular radionuclide. The external dose from contaminated ground would continue over a period of years unless the individual were evacuated or relocated, or the ground were decontaminated. The temporal behaviors of these different doses are shown in Figs. VI 13-1 through VI 13-17.

It is important that the dose computation and the dose-mortality (or dose-morbidity) criterion be compatible. For example, if some criterion is based on the dose received within 7 days, only the internal dose accumulated up to that time should be included in the comparison, even though the dose after 30 days may be higher. The correct dose factors must be used for each effect.

9.2.2 MORTALITIES

Some perspective on the important contributors to the mortality risk will be helpful to the reader. As shown in Fig. VI 13-1, the three exposure modes from cloudshine, 24 hours of groundshine, and internally deposited radionuclides would contribute approximately equal doses to the bone marrow. On the other hand, the lung dose would be dominated by that from the internally deposited radionuclides (Fig. VI 13-2). With respect to the regenerative cells of the lower large intestine, Fig. VI 13-3 shows that the internal dose would be the largest contributor but less overwhelmingly so than for the lung.

There are essentially three competing risks for early and continuing mortality resulting from damage to the bone marrow, lung, and gastrointestinal tract. Figure VI 13-7 shows that, for the specific radionuclide mixture that could be released in a reactor accident, damage to the bone marrow would always lead to the dominant risk of mortality.

9.2.2.1 Bone Marrow

As described in Appendix F, large doses of radiation have a damaging effect on the bone marrow and other blood-forming organs and on their ability to produce new blood cells. It is generally believed that damage to the bone marrow is the most important contributor to early death from large doses to the whole body. That is, radiation damage to the lung or to the gastrointestinal tract is not likely to be lethal unless accompanied by bone marrow damage.

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[1] Expansion of the cloud in the direction of the passage would also affect the duration of the cloud dose but the consequence model does not account for this phenomena.

Clinical evidence cited in Appendix F suggests that radiation protracted over 2 to 4 weeks is only half as effective as the same dose delivered within hours. On this basis, the study has assumed that the dose received within the period of 7 to 30 days after initial exposure would be only half as effective as that received within the first 7 days. Therefore, the critical dose to the bone marrow is the sum of:

external dose from passing cloud

+ external dose from contaminated ground

+ internal dose received during the first 7 days from inhaled radionuclides

+ 1/2 of the internal dose received from day 8 through day 30

As described in section 11.3.4, within 25 miles of the reactor, the calculated dose from ground contamination is truncated after 4 hours since people are being evacuated during the release. Beyond a distance of 25 miles from the reactor, the ground dose is truncated at 7 days if the people are relocated and at 24 hours if people are immediately relocated from small areas due to locally high dose rates.[1]

Appendix F proposes three dose-mortality criteria, depending on the degree of medical treatment. The curves are reproduced in Figure VI 9-1 and are denoted by A, B, and C for minimal, supportive, and heroic treatments, respectively. Mortality criteria are often stated in terms of the dose that would be lethal to 50% of the exposed population within 60 days (denoted by $LD_{50/60}$). In Fig. VI 9-1, the $LD_{50/60}$ may be read on the abscissa opposite the 50% value on the ordinate.

As explained in Appendix F, an early and commonly accepted value for $LD_{50/60}$ is 450 roentgens (exposure in air), which translates to about 300 rads (dose to tissue) to the whole body. What is less recognized is that the data on which this value is based were derived from the atomic bomb victims, who received only limited medical treatment at a time when radiation medicine was less advanced than today. As a result of the additional data accumulated in the past 30 years, it was estimated by the advisory group on health effects that the $LD_{50/60}$ would be 340 rads if only minimal medical treatment were available.

In the study's opinion, it is inconceivable that, in the event of a serious reactor accident in the United States, the Federal and state governments and the utility involved would not mobilize medical resources throughout the nation to aid the exposed population; a major constraint would be the availability of specialized resources. For this reason, the study's medical advisors evaluated two levels of medical treatment, supportive and heroic. Supportive treatment would include barrier nursing, copious antibiotics, and transfusions of whole blood, packed cells, or platelets. How many people could receive this treatment? One need is adequate medical personnel and good laboratory support. There are 433 approved programs for residencies in internal medicine of which 90% are in teaching hospitals. At least these hospitals are capable of treating severely irradiated people. Since they are generally large hospitals, each hospital should be capable of handling 5 to 10 patients. On this basis, it was estimated that 2500 to 5000 people could receive supportive treatment. The advisory group on health effects judged that for such people the $LD_{50/60}$ would be 510 rads. It should be remembered that the supportive treatment is not needed immediately following irradiation but can be started about 20 days later.

"Heroic" treatment includes, in addition to the therapy outlined above, extraordinary procedures such as bone marrow transplantation. There are currently eight medical centers performing transplantations on a regular basis, and an additional 12 hospitals have started programs. If each center were assigned five patients, a total of 50 to 150 people could receive heroic treatment. For these more serious cases, transplantation should be initiated within 10 days after exposure. For these people, the advisory

[1] In this report, 'evacuation' denotes an expeditious movement to avoid exposure to the passing cloud. 'Relocation' denotes a post-accident response to reduce exposure from ground contamination.

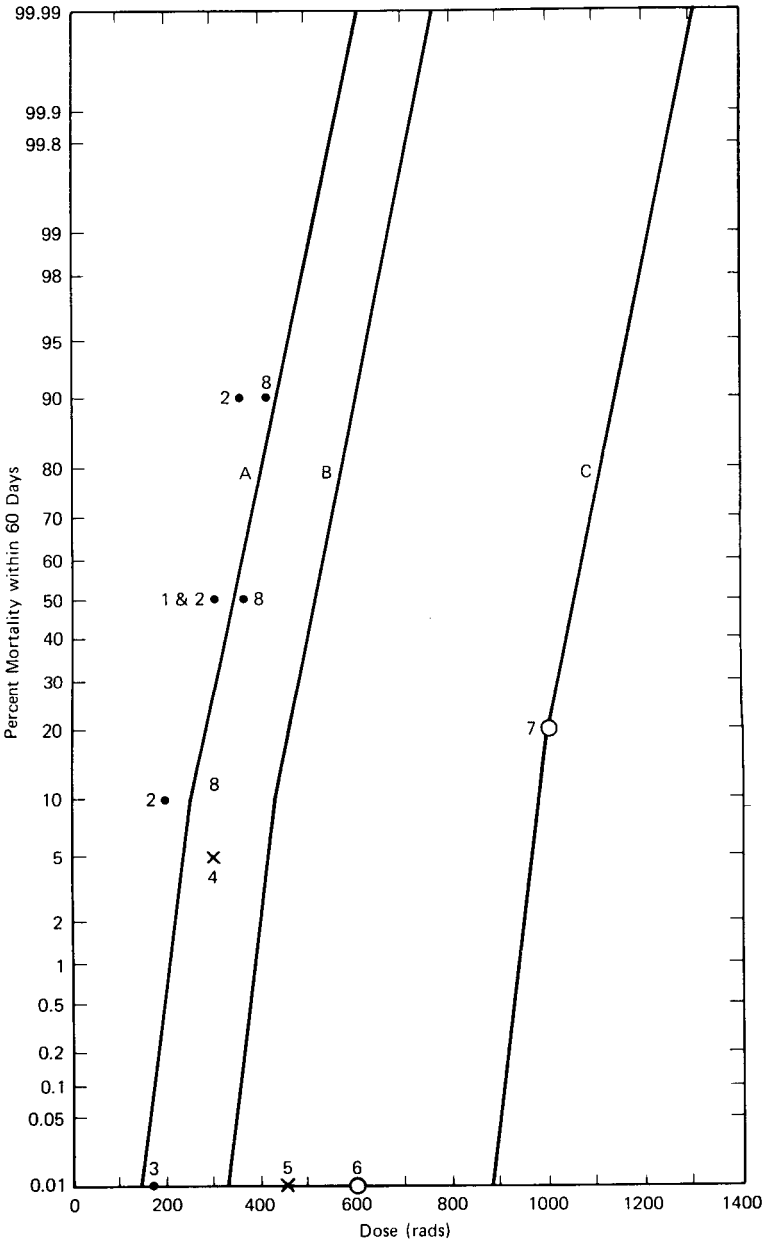

Figure VI 9-1 Estimated dose-response curves for 50% mortality in 60 days with minimal treatment (curve A), supportive treatment (curve B), and heroic treatment (curve C). Origin if data points: 1, NCRP Report 42 (converted to rads using factor given in NCRP Report 42); 2, Langhorn (1957, Table 12, estimate for "normal man?"); 3, Marshall Islanders (protracted exposure); 4, radiation therapy series, 22 patients (Rider and Hasselback, 1968); 5, clinical group III accident patients (Thoma and Wald, 1959, with newer cases added); 6, Pittsburgh accelerator accident patient (E.D. Thomas, 1971; Wald, 1975); 7, 37 leukemia patients (E.D. Thomas, 1975); 8, "best estimate" of the Biomedical and Environmental Assessment Group at the Brookhaven National Laboratory.

group judged that the $LD_{50/60}$ would be 1050 rads. It should be noted that data point 7 on curve C of Fig. VI 9-1 is based on 37 leukemic patients who were treated with 1000-rad whole-body irradiation followed by bone marrow transplantation from matched donors. The deaths in this group were caused largely by recurrent leukemia, and not depression of the bone marrow. This fact and the severe illness that justified this therapy suggest that this data point is probably conservative for normal healthy individuals.

The critical dose range for supportive treatment is 350 to 550 rads to the bone marrow. Below 350 rads, most people would not need specilized facilities (e.g., fresh platelets). In order to survive a dose in excess of 550 rads, most people would need heroic treatment. In the event of the worst calculated accident (corresponding to a probability of about 10^{-9} per reactor-year), the number of people receiving a dose in the range of 350 to 550 rads would be about 5000; none would receive a dose above 550 rads. For less severe accidents, these numbers would be smaller, being approximately proportional to the total number of fatalities. Since the above numbers are consistent with the approximate constraints estimated in the preceding paragraphs, the number of early fatalities stated in section 13 is estimated on the basis of curve B in Fig. VI 9-1.

9.2.2.2 Lung

Appendix F describes in detail the clinical and experimental evidence of the effect of radiation on the lung. The basic animal experiments on which most of the data are based were performed with aerosols of either yttrium-90 or yttrium-91, whose radioactive half-lives are 64 hours and 59 days, respectively. As shown in Fig. VI 9-2, the rate of pulmonary dose accumulation in the event of an accident is estimated to be approximately the same as that due to yttrium-90 for the first few days and to approximate that due to yttrium-91 after 60 days. Under these circumstances, the advisory group on health effects judged that the dose-mortality curves estimated for yttrium-90 and yttrium-91 should bound an appropriate dose-mortality curve for the lung dose from a reactor accident. The dose-mortality curves given in Appendix F for yttrium-90 and yttrium-91 are reproduced in Fig. VI 9-3 together with the criterion utilized in the program. As shown in Fig. VI 13-7 for two hypothetical weathers, at any given distance from the reactor, the probability of death from lung dose would always be substantially lower than that from the associated bone-marrow dose. This relationship usually holds for the time-dependent weather used in the consequence model.

As shown in Fig. VI 9-2, the yttrium-90 dose would be delivered within a matter of days, whereas the yttrium-91 dose would be accumulated over a considerably longer period. These different time periods account for the considerably higher doses needed to induce death by pulmonary injury from yttrium-91. The rate of dose accumulation is a critical parameter, and protracted doses delivered at a low rate would not apparently contribute to mortality. In the event of an accident about 50% of the final dose (after infinite time) would be delivered within less than 60 days, and over 80% of the final dose would be delivered within 1 year. These data suggest that the appropriate period for the lung dose calculation should lie between 60 and 365 days. The study conservatively selected a period of 365 days for the lung dose, which is therefore calculated as follows:

external dose from passing cloud

+ external dose from contaminated ground

+ internal dose within 365 days from inhaled radionuclides

As stated in section 9.2.2.1 for the bone-marrow dose, the ground dose is truncated after 4, 24, or 168 hours, depending on the distance from the reactor. Since the ground dose to the lung would be small in comparison to the dose from inhaled radionuclides (Fig. VI 13-2), the precision of this truncation is unimportant to the lung-dose calculation.

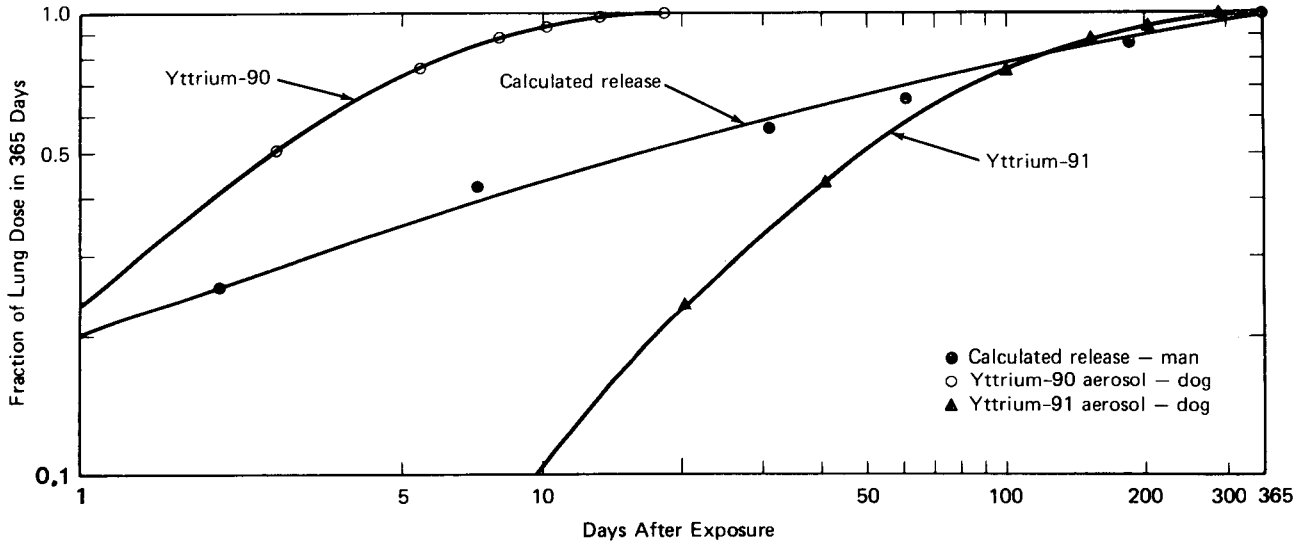

FIGURE VI 9-2 Fractions of a 365-day dose to the lung accumulated at various times after exposure.

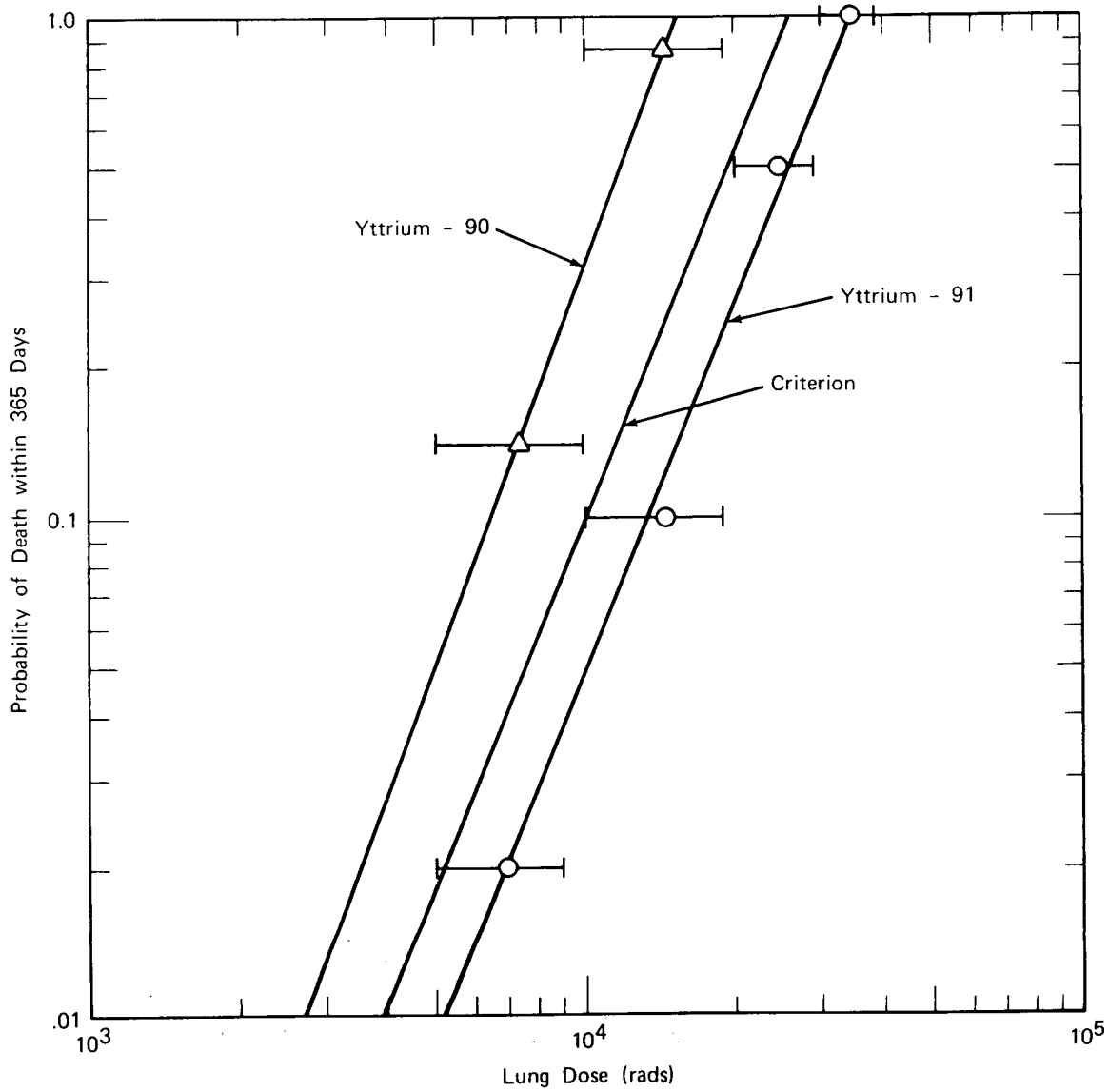

FIGURE VI 9-3 Dose-response curves for yttrium-90 and yttrium-91 and criterion used in consequence model.

9.2.2.3 Gastrointestinal Tract

Appendix F describes in detail the clinical and experimental evidence of the effect of radiation on the gastrointestinal tract. Fatalities due to local irradiation of the gastrointestinal tract would be caused by the killing of the intestinal cell population, which would lead to denudation of the gut lining, manifestations such as diarrhea and hemorrhage, and finally death. This effect should be distinguished from the classical gastrointestinal syndrome, which requires the impairment of the functional efficiency of both the gastrointestinal tract and the blood-forming organs. The classical gastrointestinal syndrome is the result of very large acute whole-body irradiation.

For the purposes of the present discussion, the gastrointestinal tract is divided into five segments: esophagus, stomach, small intestine, upper large intestine and lower large intestine. Of these segments, the esophagus would receive relatively little irradiation from inhaled radionuclides because of the short transit time and hence is not usually discussed from this standpoint. As described in Appendix F, the radiosensitive cells in the gastrointestinal tract are the regenerative cells. They are shielded from the radionuclides in the contents of the tract by a layer of mucus and/or villi. For the same intake of radionuclides, the lower large intestine would receive a larger dose than the other segments since the residence time of the contents is the longest. Accordingly, early death from irradiation of the gastrointestinal tract would be determined by the dose to the regenerative cells of the lower large intestine. The shielding (dose-reduction) factor for these regenerative cells from beta radiation depends on the energy of the beta particles, and its calculation is described in section 8.4.2.1.

By virtue of the normal clearance processes of the gastrointestinal tract, the major dosage from internally deposited radionuclides would occur during the first 7 days after inhalation (see Fig. VI 13-3). Accordingly, the dose to the gastrointestinal is calculated as follows:

external dose from passing cloud

+ external dose from contaminated ground

+ internal dose within 7 days from inhaled radionuclides

As stated in the discussion of bone marrow and lung, the calculated ground dose is truncated after 4, 24, or 168 hours, depending on the distance from the reactor.

There are no data on which to base a dose-mortality criterion for local irradiation of the gastrointestinal tract in humans. On the basis of numerous experiments with dogs and by assuming that the intestinal responses of dog and human are comparable, the advisory group on health effects recommended the dose-mortality criterion shown in Fig. VI F-6 which indicates a threshold for early death of 3500 rads and no survivors for doses above 7000 rads. In order to account for any possible delayed deaths, a more conservative criterion is used as shown in Fig. VI 9-4, with an LD_{50} of 3500 rads. Although there is considerable uncertainty in this criterion, it should be noted in Fig. VI 13-7 that the probability of death resulting from irradiation of the gastrointestinal tract is negligible compared to that from irradiation of the bone marrow. In order for the two risks to be equal, the LD_{50} for the gastrointestinal tract would have to be 600 rads which is clearly not supported by either clinical or experimental evidence.

The internal dose to the gastrointestinal tract from inhaled or ingested radionuclides can be reduced by a factor of 2 to 4 by the administration of a mild laxative. The 7-day period over which the dose builds up provides ample time for such supportive treatment. The study did not account for such action which would not, of course, have reduced the calculated early fatalities but would have reduced the estimated late fatalities due to gastrointestinal cancer.

9.2.2.4 Radiation Thyroiditis With Thyroid Storm

Very large doses of iodine-131 to the thyroid can cause an accelerated release of thyroid hormone. In extreme cases, severe thyrotoxicosis characterized by disorientation, fever, heart failure, and adrenal exhaustion can develop. This condition is called thyroid

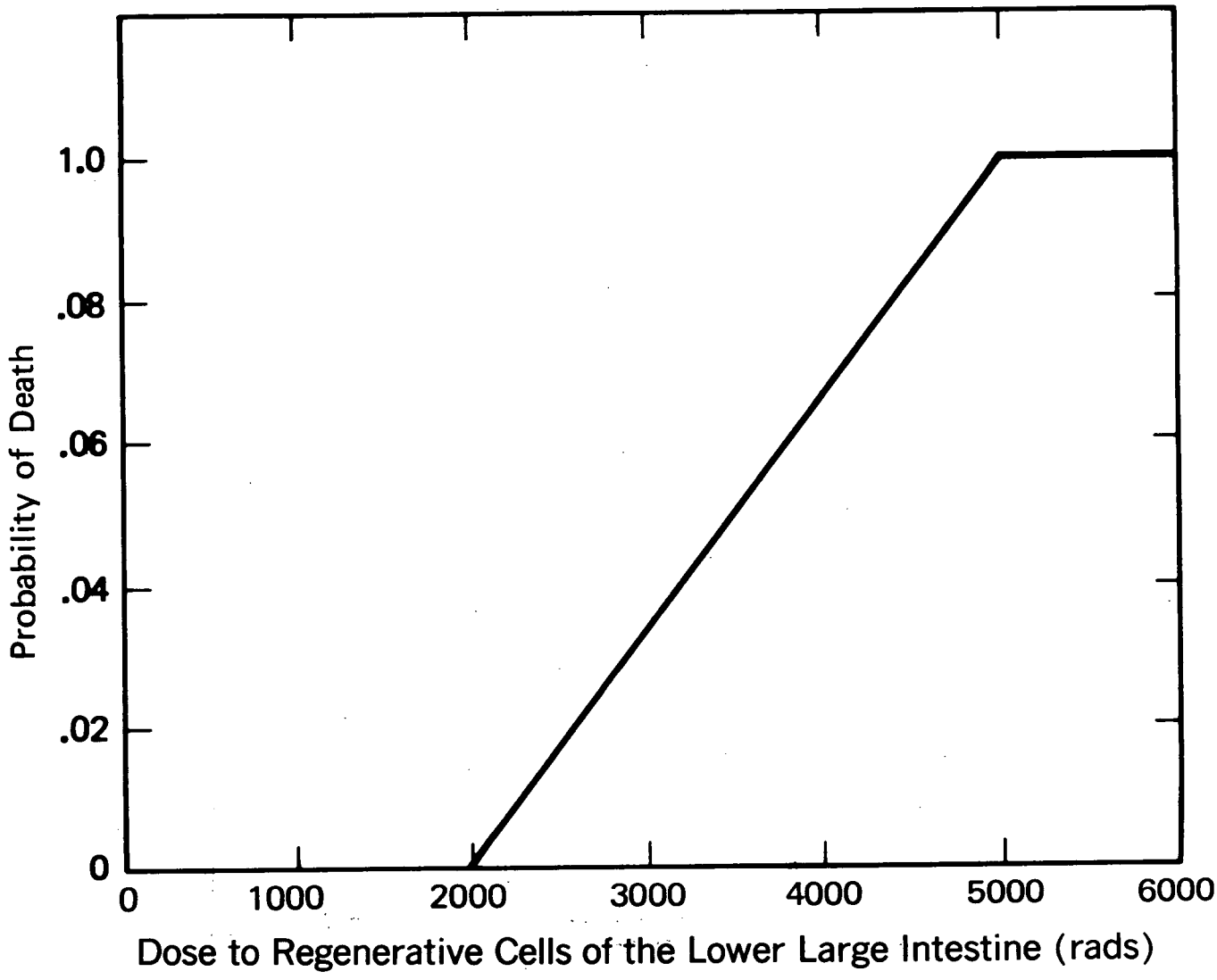

FIGURE VI 9-4 Dose-mortality criterion for irradiation of the lower large intestine.

storm. For people with preexisting thyrotoxicosis or severe ischemic (blood deficiency) heart disease, radiation thyroiditis with even mild thyroid storm could lead to death. As described in Appendix H, the prevalence of death due to acute thyroiditis would not be expected to exceed 21 per 100,000 people receiving thyroid doses of more than 25,000 rads.

As shown in Fig. VI 13-4, inhaled iodine-131 contributes about two-thirds of the dose to the thyroid. Since iodine-131 has an 8-day radioactive half-life (other radioiodines in the release have a shorter half-life), essentially the full thyroid dose is received within 30 days of inhalation. Therefore, the dose to the thyroid is calculated as follows

external dose from passing cloud

+ external dose from contaminated ground

+ internal dose within the first 30 days from inhaled radionuclides

Figure VI 13-19 shows that anyone who receives less than a lethal dose to the bone marrow would receive less than 25,000 rads to the thyroid. Therefore, radiation thyroiditis would not be expected to contribute to early fatalities.

9.2.2.5 Prenatal and Neonatal Deaths

The mammalian organism is particularly sensitivie to radiation damage during the embryonic and fetal stages.[1] In its detailed description of the effects of prenatal exposure, Appendix F distinguishes between several effects. For this section, it suffices to observe that the embryo is more radiosensitive than the fetus, so these two stages should be treated separately.

The dosimetry for the embryo and fetus is somewhat uncertain but may be estimated from their location within the mother. The advisory group on health effects judged that the fetus would be shielded by the mother's pelvis from external radiation and therefore recommended that the external dose to the mother's lower large intestine be used as an approximation to the fetal dose. For the dose from internally deposited radionuclides, the advisory group recommended the use of the dose to "other tissues" rather than "whole body" (see section 8.4.2 for definition). With these considerations, the dose to the embryo is conservatively calculated as follows:

external dose to ovaries from passing cloud

+ external dose to ovaries from contaminated ground

+ internal dose to ovaries within 60 days from inhaled radionuclides

For the dose to the fetus, the calculation is as follows:

external dose to lower large intestine from passing cloud

+ external dose to lower large intestine from contaminated ground

+ internal dose to "other tissues" within 180 days from inhaled radionuclides

As stated previously for other organs, the calculated ground dose is truncated after 4, 24, or 168 hours, depending on distance from the reactor site.

[1] For humans, the embryonic period is from fertilization through the eighth week of pregnancy and the fetal period is from that time until birth.

Appendix F describes the available clinical evidence for dose-mortality criteria for in utero exposure. On the basis of these data, the advisory group on health effects recommended the use of the dose-mortality criteria in Fig. VI 9-5 for the first trimester and curve A in Fig. VI 9-1 for the second and third trimesters.

Approximate calculations show that the numbers of embryonic and fetal deaths would be fewer than 10 and 5%, respectively, of the early fatalities stated in Fig. VI 13-30. It should be noted that, of the embryonic deaths, over 90% of them would be less than 3 weeks since conception, and it is likely that the woman concerned would know neither that she was pregnant nor that the embryo had died. The number of prenatal deaths is expected to be about 5% of the early fatalities, and this lies within the uncertainty in overall numbers.

9.2.2.6 Calculation of Number of Mortalities

The number of early deaths due to radiation thyroiditis and prenatal exposure were estimated in section 9.2.2.4 and 9.2.2.5, respectively, and are expected to be relatively small in comparison to the overall total; they are not calculated by the consequence model for each accident scenario. The overall number of early fatalities is calculated by comparing the doses to bone marrow, to lung, and to gastrointestinal tract with the corresponding dose-mortality criteria. As clearly shown in Figs. VI 13-5 and VI 13-6, individuals receiving a high dose to the bone marrow would also receive high doses to the lungs and to the gastrointestinal tract. Consideration of each effect as independent would grossly overestimate the number of mortalities since a person can only die once. Therefore, if the fractions of the population at a given distance from the reactor site who would die from radiation to the bone marrow, lung, and gastrointestinal tract are f_1, f_2, and f_3, respectively, where f_i is calculated by comparing the dose to the criterion for organ i, then the overall fraction of that population that would die from early and continuing somatic effects is

$$f_1 + (1 - f_1)\ f_2 + (1 - f_1)\ (1 - f_2)\ f_3.$$

As suggested in Figs. VI 13-5 and VI 13-6, at all distances from the reactor, the probability of death from irradiation of bone marrow usually dominates the corresponding probabilities for lung and gastrointestinal tract. Therefore, the overall number of early mortalities is effectively determined by f_1.

The above calculation presumes no synergistic effects between different organs, (e.g., sublethal doses to both the bone marrow and the lung might be fatal). The advisory group on health effects discussed possible synergisms and concluded that there might be one between irradiation of the bone marrow and of the lower large intestine but that no quantitative evaluation was possible. In the absence of any positive data and in consideration of overall uncertainties, the advisory group recommended that synergism be omitted from the calculations.

9.2.3 EARLY MORBIDITY

In this section, the result of exposure to sublethal doses is considered. The SD is used to indicate the criterion for the symptom, and the SD_{10}, SD_{50}, and SD_{90} are the doses that would be expected to cause a specific clinical response in 10, 50, and 90% of the exposed population, respectively. The SD_{50} doses are more difficult to estimate than LD_{50} doses since the endpoint is less definite and somewhat subjective.

9.2.3.1 Respiratory Impairment

The early changes induced by radiation from external sources and from internally deposited radionuclides in the lungs may either regress or may progress to cause a loss of functional lung volume. Extensive exposure would result in respiratory impairment, which can affect heart function. The severity of these reactions and their time course would be determined by the total radiation dose, dose rate, fraction of lung irradiated, and the condition of the lung before exposure. As the volume of damaged lung increased, shortness of breath and coughing, particularly after exertion, would become apparent. Initially, the effects may be accompanied by increased pulmonary infections. The results of exposure are strongly dependent on time and dose. For acute external exposures, the SD_{50} is probably above 1000 rads, but, since such a whole-body dose plus the

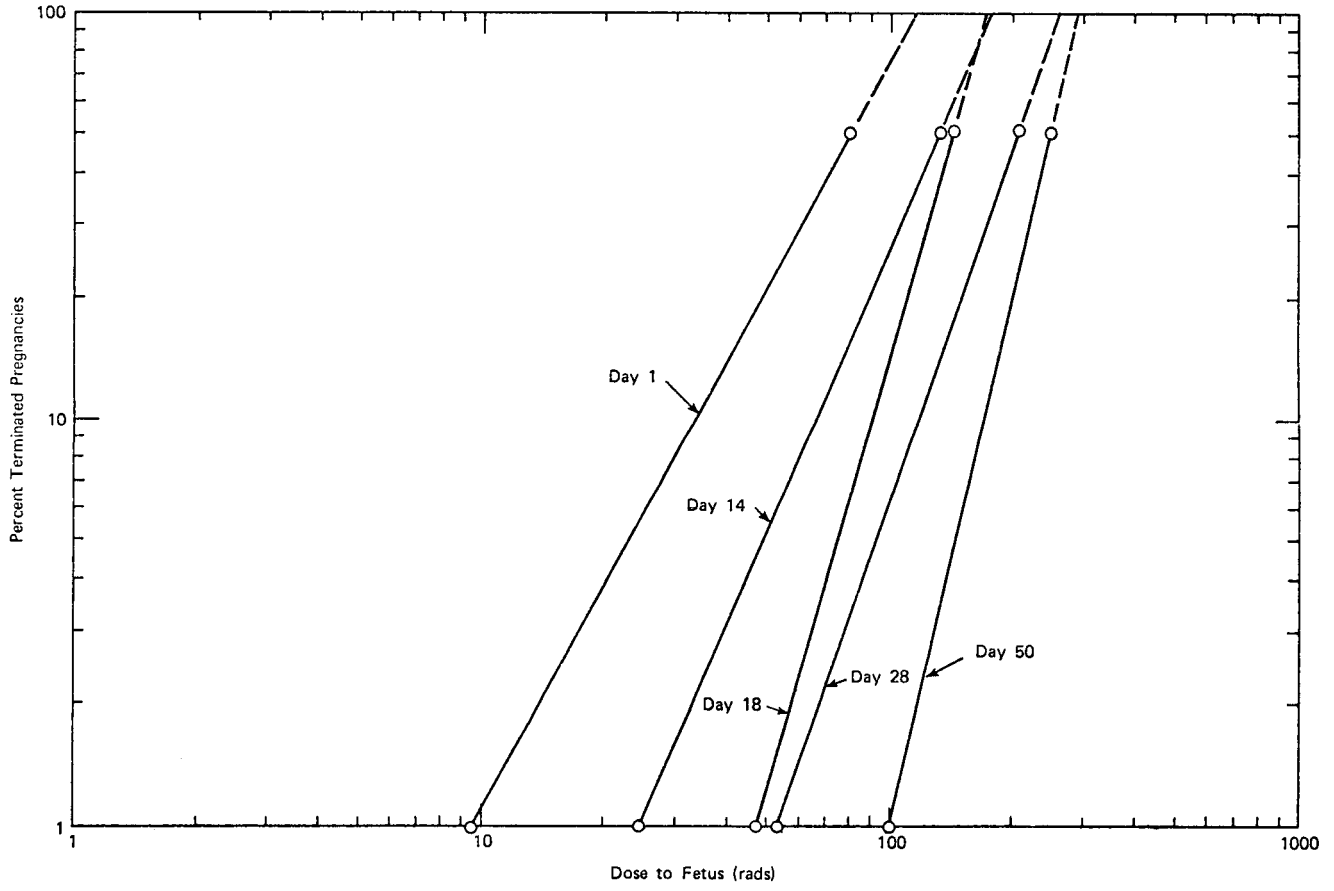

FIGURE VI 9-5 Percent termination of pregnancy following brief external exposure during the first trimester of gestation.

probable concurrent internal exposure would be fatal, pulmonary complications would not be of major concern in such a case. The probability of pulmonary morbidity from combined internal and external exposures that can be expected from an hypothetical reactor accident is shown in Fig. VI 9-6, which indicates an SD_{50} of about 4500 rads. The dose is calculated by the method stated in section 9.2.2.2.

9.2.3.2 Gastrointestinal Morbidity

Sublethal doses to the intestine, even relatively severe ones, would produce only temporary changes, which are repaired by cell regeneration. The critical factor in survival is whether or not adequate numbers of cells would survive to resume cell production and thus repair the damage in time. The effects are dependent on the dose rate, and for low dose rates, larger total doses are required to produce a given degree of intestinal damage than are required in a single brief exposure. The radiation response of the stomach and large intestine is similar to that of the small intestine, but damage develops more slowly because of their lower rate of cell renewal and associated lower radiosensitivity. However, in the case of internal exposure, the effects in animals were observed in the large intestine since that organ would receive considerably greater dose due to the longer residence time of the content. Thus, the gastrointestinal effect is primarily on the large intestine. The threshold is at about 1000 rads and reaches the 100% level at 2500 rads. Figures VI 13-5 and VI 13-6 show that, at all distances from the site, the dose to the gastrointestinal tract would be approximately equal to that to the bone marrow. Since 1000 rads to the bone marrow would be lethal, gastrointestinal morbidity would never be a major concern.

9.2.3.3 Thyroid Morbidities

9.2.3.3.1 Hypothyroidism

Hypothyroidism is a deficiency of thyroid activity that occurs spontaneously and may be induced by irradiation of the thyroid. Hyperthyroidism (overactivity of the thyroid gland, clinically termed Graves' disease) and thyroid cancer are often treated by administering to the patient a dose of iodine-131, which, taken up by the thyroid, diminishes thyroid function and may destroy functioning thyroid cancer cells. A hypothyroid person is normally prescribed replacement thyroid hormones, which are taken orally and are inexpensive, effective, and safe.

An early and continuing effect in the event of a reactor accident would be some cases of hypothyroidism. Appendix H discusses the available clinical data relating the incidence of hypothyroidism with radiation exposure. It should be emphasized that the available clinical data are largely limited to patients being treated with iodine-131 for heart disease or for hyperthyroidism and to patients undergoing thyroid examinations for suspected thyroid problems. Most of the patients received high doses of I-131 (>2500 rem) and there are relatively few data for patients exposed to lower doses. The application of these data to estimate the incidence of hyperthyroidism in exposed people who have normal thyroids has been necessary because of the paucity of data from normal populations. An approximate calculation indicates that the number of cases of hypothyroidism might be of the same order of magnitude as the number of thyroid nodules. The study recommends more work in this subject in order to generate a stronger basis for risk estimates.

9.2.3.3.2 Radiation Thyroiditis

Section 9.2.2.4 described the effects of iodine-131 in excess of 25,000 rem to the thyroid. Although such doses would only be expected to result in about 21 deaths per 100,000 people, there would be some morbidity from radiation thyroiditis. This effect is an inflamation of thyroid gland, and the symptoms include pain and tenderness in the gland, mild fever, and occasional thyrotoxicosis. Most of the cases would be expected to be very mild and could be treated with aspirin. Other drugs might be required in the most severe instances.

From an analysis of clinical data, it is estimated in Appendix H that, at doses slightly above 25,000 rems, 4.5% of individuals might develop thyroiditis and that, for each 10,000-rem increment, an additional 5% incidence might be expected. The dose to the thyroid is calculated by the method set forth in section 9.2.2.4. As shown in Fig. VI 13-19, no person would receive in excess of 25,000 rem to the thyroid unless he also received a lethal dose to the bone marrow. Therefore, no cases of radiation thyroiditis would be expected.

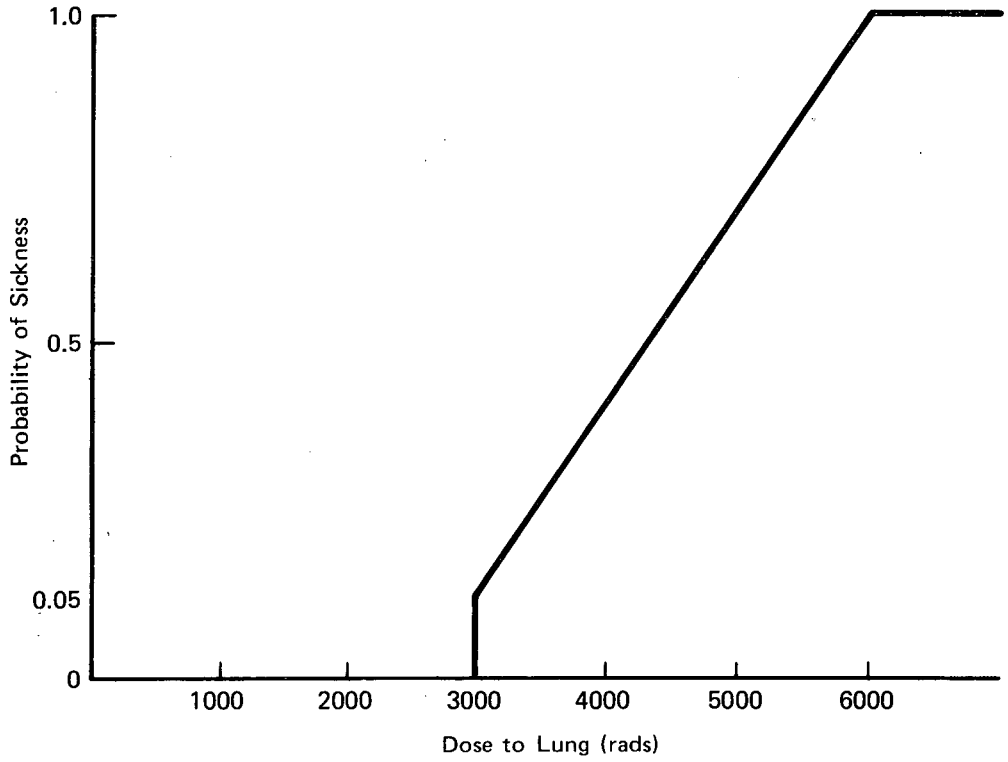

FIGURE VI 9-6 Dose-morbidity criterion for lung.

9.2.3.4 Sterility

9.2.3.4.1 Males

In the human male, radiation doses beginning above 10 rads and extending to 600 rads produce a decrease in, or absence of, sperm beginning at least 6 to 7 weeks after exposure and continuing for a few months to several years. Within this dose range recovery follows. The extent of sperm count decrease and the rate of return are related to the magnitude of the exposure. It should be noted, however, that even the dose at the high end of the range, which would probably be lethal if administered to the whole body, is not sufficient to produce permanent sterility. It should also be noted that libido and potency are not affected in this dose range, although psychological factors may affect sexual capacity. The dose-response relationship for transient sterility in males within 210 days of exposure is shown in Fig. VI 9-7. The dose to the testes is calculated as follows:

external dose to testes from passing cloud

+ external dose to testes from contaminated ground

+ internal dose to testes within 60 days from inhaled radionuclides

9.2.3.4.2 Females

Radiation effects on the human ovary differ from those in the testes because, unlike the testes, the ovaries contain their entire supply of germ cells or oocytes early in life and lack regenerative cells capable of replacing any that are lost thereafter. Since the oocytes are relatively radiosensitive, loss of such cells due to radiation damage irreversibly reduces the reproductive potential of the woman exposed. The age of the ova and their stage of development have an effect on their radiosensitivity.

Although human experimental data are lacking, there is information from studies of the effects of localized radiation therapy as well as followup studies of the Japanese atomic bomb survivors and Marshall Island women exposed to radioactive fallout. Neither of the population studies have shown any apparent effect on fertility, although adequately controlled quantitative evaluation was not feasible. On the basis of radiation therapy data, it appears that single doses of 125 to 250 rads to the ovaries may produce prolonged or permanent suppression of menstruation in about 50% of women, whereas a dose of about 600 rads is required to produce permanent suppression in virtually all women so exposed. A dose of 1000 to 2000 rads may be needed to reach this endpoint if the exposure is fractionated over about 2 weeks and the subjects are young women, who are more radioresistant. If the exposure is delivered over 6 weeks at 100 rads daily 5 days a week, the 50% probability level for permanent sterility would be about 2000 rads in young women. These data are presented in Fig. VI 9-8. In the consequence model, the dose is calculated as follows:

external dose to the ovaries from passing cloud

+ external dose to the ovaries from contaminated ground

+ internal dose to ovaries within 2 days from inhaled radionuclides

and the single-dose curve is used as criterion.

9.2.3.4.3 Estimated Cases of Temporary Sterility

Temporary sterility is critical only for persons in the age cohort 15 to 50 years, which represents 49% of the population. By using the data in the preceding sections, an approximate calculation indicates that the number of cases of temporary sterility in males might be a factor of 8 higher than the number of early illnesses stated in Fig. VI 13-31 and in females 50% higher. As shown in Fig. VI 13-18, no one would receive in excess of 550 rads to the bone marrow, so no cases of permanent sterility in women would be expected. Since most individuals only conceive children intermittently and over a period of less than 35 years, the vast majority of these cases would not be aware of their temporary sterility. For this reason, temporary sterility should be

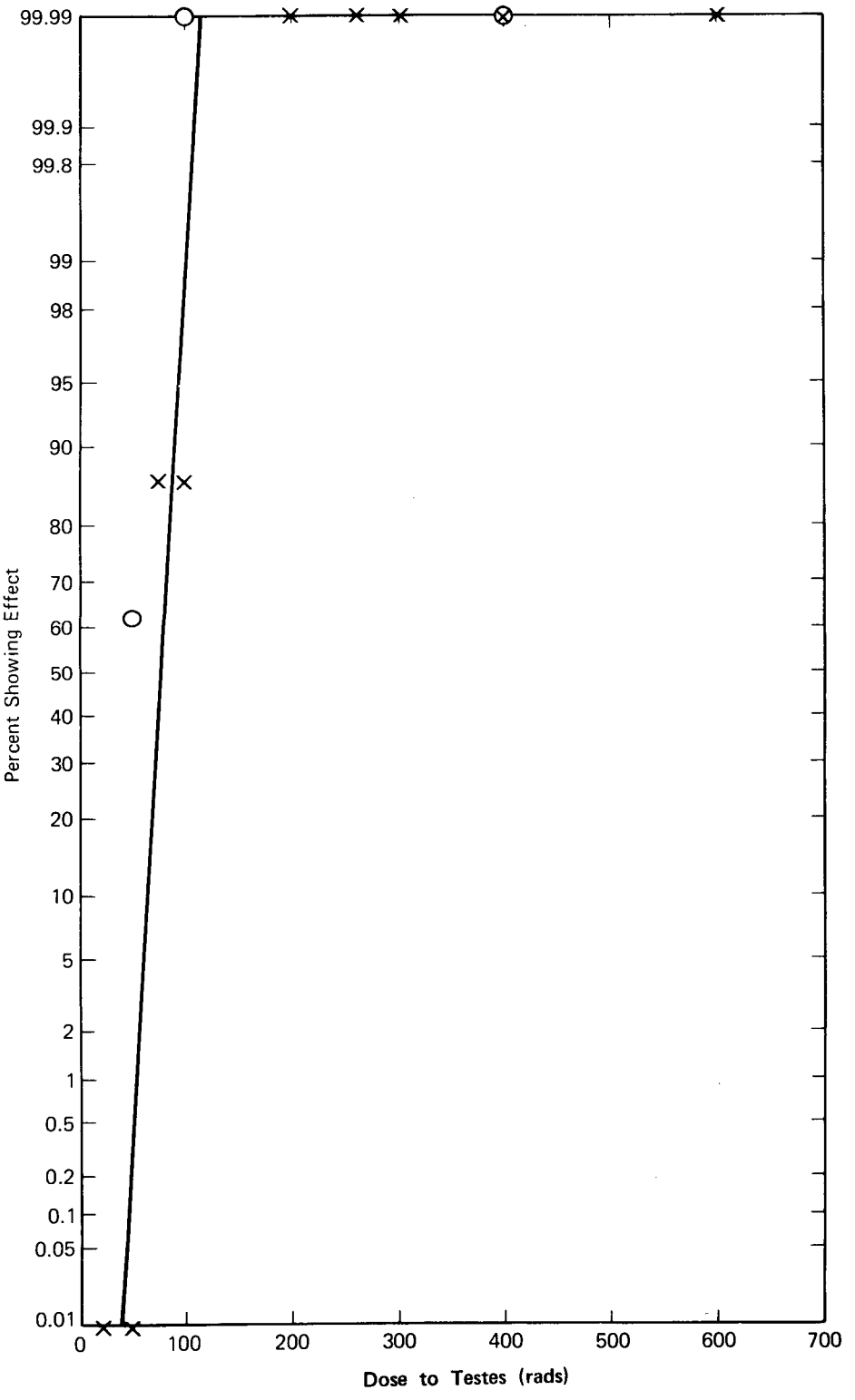

FIGURE VI 9-7 Transient sterility (azoospermia) in males within 210 days after exposure. The circles are data from Thorslund and Paulsen (1972); the crosses are data from Rowley et al. (1974, 1975). The midorgan x-ray doses in roentgens were converted to rads using an f factor of 0.95.

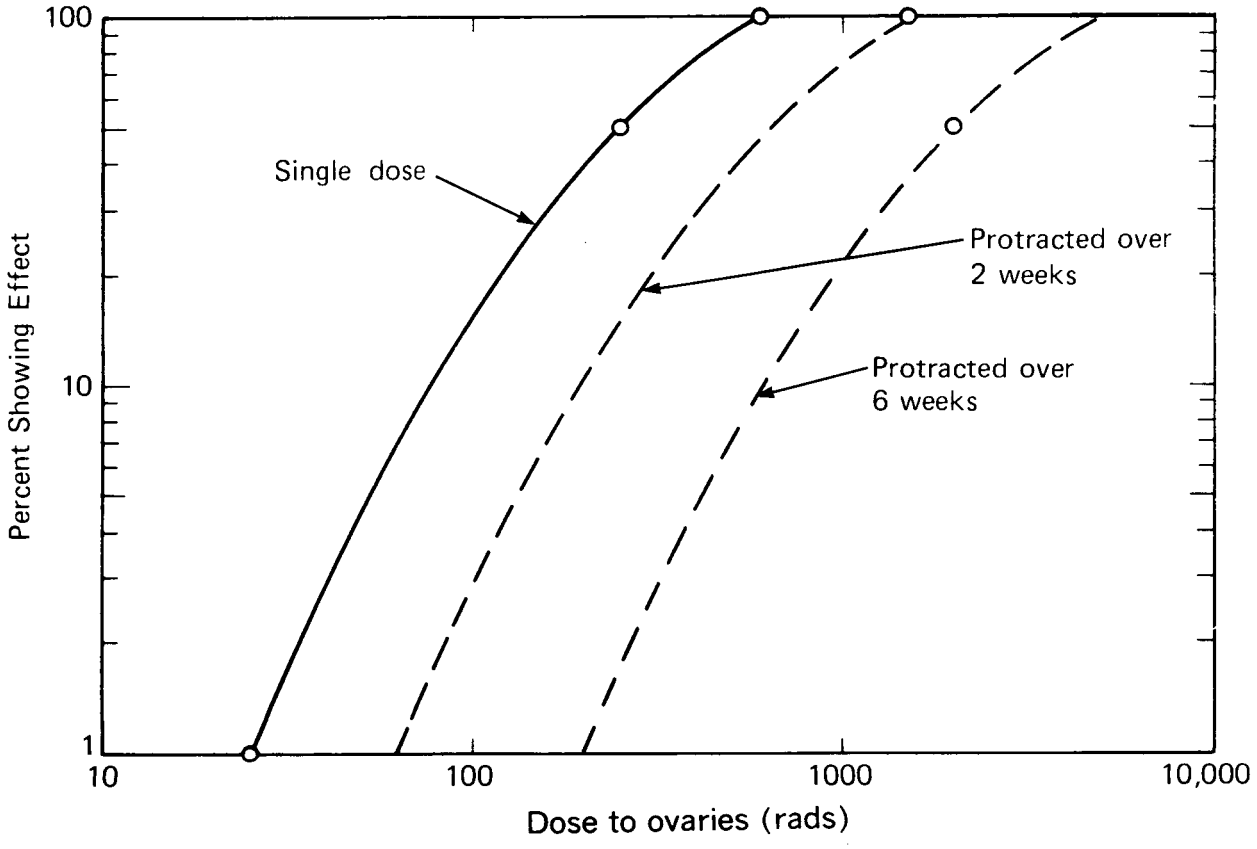

FIGURE VI 9-8 Dose-response curves for sterility in females.

regarded as less serious than other early radiation illnesses (e.g., respiratory impairment).

9.2.3.5 Congenital Malformations and Growth Retardations

Studies on the atomic bomb survivors and medically exposed patients indicate that the major developmental effects of prenatal irradiation in humans are impairment of growth, microcephaly (small head circumference), and mental retardation. The peak incidence of such developmental abnormalities is caused by irradiation during the period of organogenesis (first trimester); they are manifest at birth. Irradiation during the fetal stage can alter the structure and function of adult organs and tissues, causing defects that may become apparent only later in life and result in reduced growth.

Radioiodine is taken up by the fetal thyroid (second and third trimesters only) and may induce hypothyroidism in the fetus, which can lead to the impairment of mental and physical development. Prompt diagnosis and therapy in the immediate neonatal period and continued throughout life would appear to minimize the manifestations of in utero hypothyroidism.[1] Limited data suggest that the radiosensitivity of the fetal thyroid may be from 1 to 18 times greater than that of the adult thyroid. It is estimated in Appendix D that the dose to the fetal thyroid is five times that to the maternal thyroid. On these bases, it is estimated that the number of cases of fetal hypothyroidism might be about 1% of the total number of cases of hypothyroidism. The caveats noted in section 9.2.3.3.1 apply to this estimate.

Figure VI 9-9 states the incidence of microcephaly as a function of dose received during the first 4 months of pregnancy. For this estimate, the dose to the ovaries is conservatively calculated from external sources plus internally deposited radionuclides during the first 6 months. An approximate calculation indicates that the number of cases of microcephaly might be about 50% of the early fatalities. There are insufficient data to calculate the number of other congenital malformations. As a very approximate estimate, the number of other effects might be equal to the number of cases of microcephaly. Some women who are in the first 4 months of pregnancy at the time of exposure and who receive a significant dose, may wish to consider a therapeutic abortion although this remains the subject of controversy and each case would be reviewed individually.

9.2.3.6 Cataracts

The threshold radiation dose for the induction of cataracts severe enough to impair vision varies from 200 to 500 rads for a single brief exposure of low-LET radiation. Progressive cataracts are formed after doses of at least 500 rads, the probability of progression increasing with dose. The latent period for cataract formation varies in relation to the dose and dose rate, ranging from 6 months to 35 years, with an approximate average of 2 to 3 years. Since only external exposure applies, the dose to the lens of the eye would be equal to the external whole-body dose from the passing cloud. In view of the high dose required, cataract formation would be a consideration only for individuals who received preferential exposure to the head without a corresponding whole-body exposure. Such nonuniform exposure is unlikely in the event of a reactor accident. Therefore, eye cataracts in any survivors are very unlikely.

9.2.3.7 Prodromal Vomiting

Prodromal vomiting would be the cause of temporary discomfort, which would clear up quickly and would not recur unless there is radiation damage to the gastrointestinal tract. It would unlikely be a source of permanent injury to the affected person.

The incidence of vomiting within 2 days (without pretreatment) increases quickly with absorbed dose. The effect is also dependent on the exposure rate. As explained in Appendix F, the SD_{50} for vomiting is estimated to be 182 rads when delivered in a single dose, 500 rads when delivered over 1 to 8 days, or 600 rads when delivered at a rate of 20 rads per day for at least 30 days. Thus, the protracted dose over a week or more appears to be about one-third as effective as the same dose delivered acutely. Since the dose-morbidity curve shown in Fig. VI F-19 is based on effects after 48 hours, the dose for this effect can be conservatively calculated as follows:

[1]The American Thyroid Association has a pilot program for testing newborns for hypothyroidism.

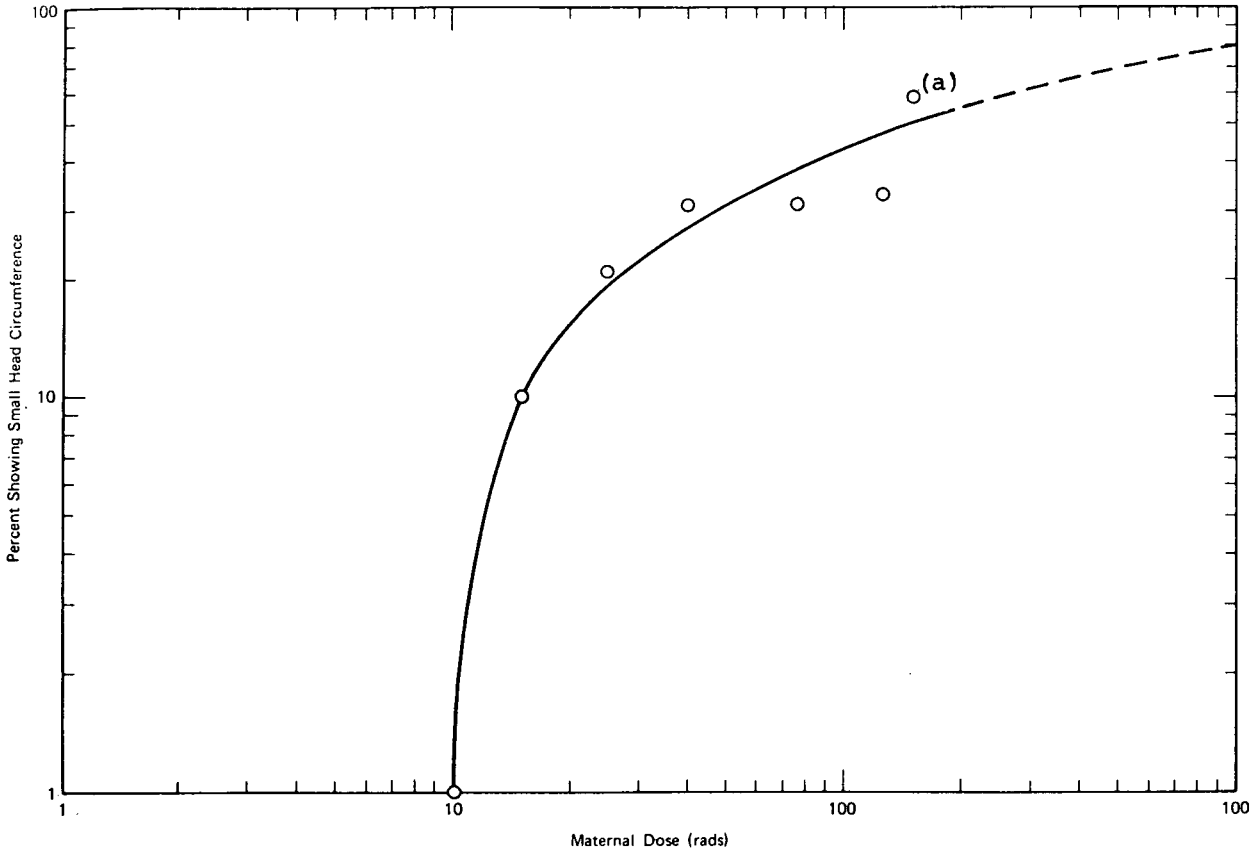

FIGURE VI 9-9 Incidence of small head circumference versus dose for individuals exposed between the 0 and 17 weeks of gestation in Hiroshima and Nagasaki.

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

external dose from passing cloud

+ external dose from contaminated ground

+ internal dose to whole body within 2 days from inhaled radionuclides

+ 1/3 of any dose delivered at a rate greater than 20 rads per day.

Since dose rates in excess of 20 rads per day could only be experienced within a mile or so of the reactor in the event of the largest release and the exposed people would receive lethal doses to the bone marrow, the last-named dose contribution is negligible. As stated previously for other organs, the calculated dose from ground contamination is truncated after 4, 24, or 168 hours, depending on distance from the reactor.

9.2.3.8 Calculation of Early Morbidities

The study defines early morbidities as those requiring medical attention and possibly hospital treatment. Respiratory impairment and hypothyroidism clearly fall into this category, but prodromal vomiting, lasting only a short time and having no lasting effect on the individual, would be excluded under this definition. The number of early morbidities stated in section 13 are based on only the cases of respiratory impairment. A small segment, (e.g., 5%) of the population might have a more serious reaction to prodromal vomiting. The number of such cases would be about 25% of the respiratory impairments and thus are included within the stated uncertainties.

Other morbidities are either less serious by numbers or effect (e.g., radiation thyroiditis, cataracts, or temporary sterility) or are very approximate estimates by virtue of the limited data. The approximate numbers of these morbidities are stated in the preceding sections.

9.3 LATE SOMATIC EFFECTS

9.3.1 INTRODUCTION

As stated in section 9.1, late somatic effects would be limited to latent cancer fatalities and morbidities plus benign thyroid nodules. These are random phenomena whose probability of occurrence for an individual is some function of the dose received; there is no direct relationship between being irradiated and incurring cancer 25 years later. For this reason, late somatic effects are calculated on the basis of population dose (cases per million man-rem). Since no clinical distinction can be made between a cancer that was induced by radiation and one that occurs spontaneously, the late somatic effects stemming from a major release of radioactive material would manifest themselves as an increase in the normal incidence of cancer for the exposed population.

The basic model for latent cancer is sketched in Fig. VI 9-10. Following the irradiation of a large number of people, there is a latent period during which no increase in cancer incidence is detectable.[1] After this period, the radiation-induced cancers appear at an approximately uniform rate for a period of years, which is termed the plateau. The model depicted in Fig. VI 9-10 is clearly idealized. In reality, neither the latent nor plateau periods would be so clearly defined, and undoubtedly the cancer incidence during the plateau would be nonuniform. The risk of latent cancers is normally stated either in terms of the incidence rate during the plateau period (cases per million exposed population per year per rem) or in terms of the expected number of cases (cases per million man-rem). The latter value is merely the integral under the curve, or the incidence rate times the plateau period.

The risk of radiation-induced latent cancer has been extensively summarized in several recent reports including those issued by the United Nations (1972), the National Academy of Sciences (1972), and the National Council on Radiation Protection and Measurements (1971, 1975). As a starting point, the study uses the estimates stated

[1] As stated in section 13.4, the highest incidence of latent cancer fatalities attributable to a reactor accident would almost certainly not be statistically detectable.

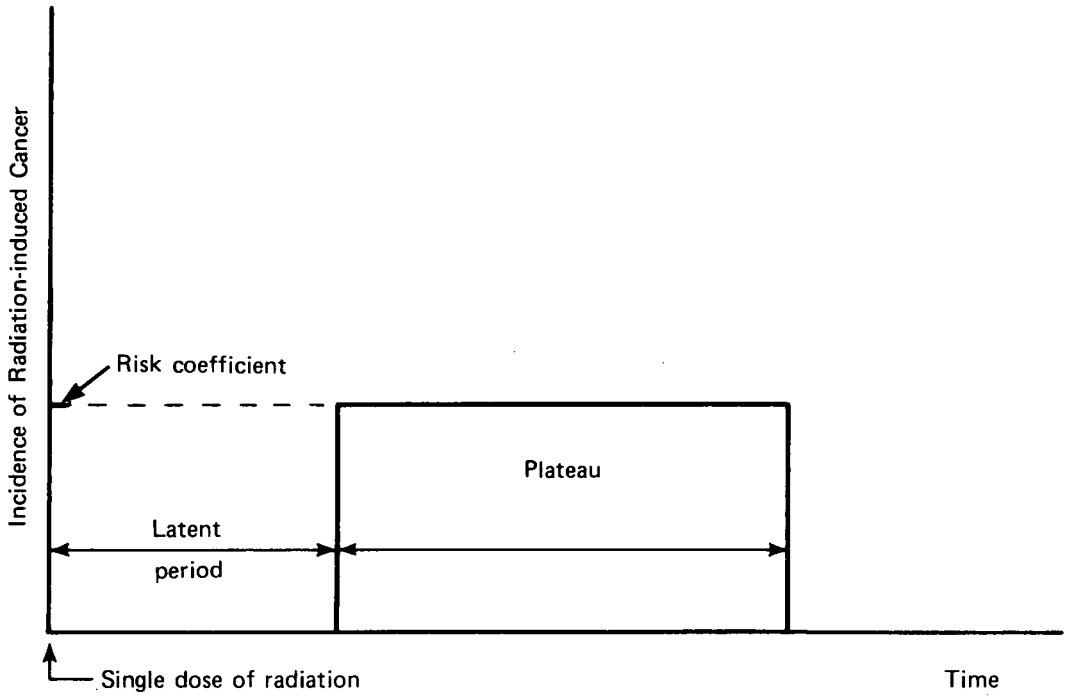

FIGURE VI 9-10 Basic model for latent cancer fatalities.

in a report issued by the National Academy of Sciences on the biological effects of ionizing radiations (the BEIR Report). The BEIR Report estimates risks on both an absolute and relative basis. The distinction between these bases is described in Appendix G. For the reasons stated there, the study accepts the absolute basis as being the more appropriate for the evaluation of reactor risks.

The BEIR Report relied heavily on the ongoing study of the Japanese atomic bomb survivors, who received very high dose rate exposure of gamma, beta, and neutron (high-LET) irradiation. Furthermore, the dose magnitudes were estimated to range from 10 to over 300 rem. Those survivors receiving less than 10 rem were used as a control population group for the BEIR estimates. The doses from a reactor accident would be almost exclusively due to low-LET radiation (i.e., no neutrons and less than 1% due to alpha radiation). Except for a few individuals who might be irradiated by the passing cloud very close to the reactor, the dose rates to the whole body would be less than 1 rem per day, which, with respect to latent cancer, is a low dose rate. Finally, a reactor accident would expose a few individuals to large doses and many people to small doses. Figure VI 13-18 shows the number of people versus bone marrow dose. Over 95% of the exposed population would receive a bone marrow dose of less than 10 rem. This curve omits those people born after the accident who would be exposed to ground contamination. The inclusion of such people or the evaluation of smaller releases under less adverse weather conditions would result in a distribution that was even more skewed towards low doses. For all these reasons, the exposures resulting from a reactor accident would be different from the exposures on which the BEIR Report bases its estimates with respect to quality of radiation, dose rate and dose magnitude.

The risk estimates generated in the BEIR Report are based on a linear extrapolation from the aforementioned data to zero doses and exclusion of any threshold dose, that is, a dose magnitude below which there would be zero induction of cancer. Both the BEIR and United Nations reports caution that this linear hypothesis is likely to overestimate the risks for low doses and/or low dose rates of low-LET radiation and that, in cases of low exposure, it cannot be ruled out that the risk may actually be zero. Following the publication of these reports, the National Council on Radiation Protection and Measurements (1975) issued a report in which it cautioned governmental policy-making agencies that use of the BEIR estimates, derived as they are from large doses at high dose rates, have such a high probability of overestimating the actual risks from low doses of low-LET radiation delivered at low dose rates as to be of marginal value, if any, for purposes of realistic risk-benefit evaluation. These important caveats are developed in more detail in Appendix G.

Since the objective of the Reactor Safety Study is to make as realistic assessment of risks as is possible and to place bounds on the uncertainty, the study makes three estimates of the number of latent cancers from a reactor accident. The upper bound is based on the BEIR estimates with some small changes reflecting recent data. For the central estimate, the upper bound is modified by dose-effectiveness factors. These factors, which are based on recent experimental data for animals, reduce the expected incidence of latent cancers for small doses and/or low dose rates. In the opinion of the study, these central estimates represent a more realistic assessment of latent cancers in the event of a reactor accident, although the advisory group on health effects were of the unaminous opinion that the dose effectiveness factors they recommended probably overestimate the central estimate. As discussed in Appendix G, the overall pattern of data shows no observable difference from an unirradiated control population for persons receiving either an acute dose of less than 25 rem or a chronic dose of less than 1 rem per day to the whole body. As an approximate indication of a possible nonzero lower bound, the study estimates the population dose received by individuals in excess of a threshold and applies the incidence rate used for the upper bound.

The BEIR Report estimates the incidence of radiation-induced latent cancer fatalities for individual organs and summarizes the overall effect in terms of whole-body radiation. The latter approach was appropriate since the BEIR Report was primarily concerned with external radiation to the whole body. In the event of a reactor accident, inhalation of radioactive material from the passing cloud will result in a nonuniform dose distribution in the body; certain organs (e.g. the lung) will receive much higher doses than others. External irradiation by gamma rays, on the other hand, results in an almost uniform dose distribution throughout the body. In order to accommodate this nonuniform dose distribution, the doses and the expected radiogenic latent cancer deaths are calculated for individual organs. For reference purposes, the whole-body values are also calculated. As shown in Table VI 13-3, inhalation of radionuclides

from the passing cloud contributes only about 15% of the whole-body man-rem (both short term and chronic), but results in about 71% of the latent cancer fatalities. For different accident scenarios, the sum of the cancer deaths calculated based on doses and risk factors for individual organs exceeds those based upon the whole-body dose by 30 to 100%.

The thyroid is treated separately from other organs since it concentrates radioiodines, which are released in large quantities in the dominant reactor accidents. The thyroid gland can be ablated by large doses, thus markedly altering subsequent cancer and nodule probabilities.

9.3.2 UPPER BOUND FOR LATENT CANCER FATALITIES

9.3.2.1 The BEIR Risk Estimates

As stated in the preceding section, the BEIR risk estimates are based on a linear, no threshold model. It is assumed that all risks of somatic effects are proportional to dose, that is, that each increment in absorbed dose carries an equal increment in risk. This linear hypothesis implies that the number of cancer deaths is proportional to the population dose (man-rem), which is determined by the product of the number of exposed individuals and their dose, independent of the dose magnitude. For example, the same number of radiation-induced cancer deaths would be expected from 10,000 people each receiving 100 rem as from 10,000,000 people each receiving 0.1 rem.

The BEIR Report adjusts the numerical risk estimates to account for possible differences in the radiosensitivity of the fetus, child, or adult. For each age cohort, the report estimates the latent period after radiation during which the cancer risk is unchanged and the following plateau period during which the cancer risk is higher. For risk estimates on an absolute basis, the actual table from the BEIR Report is reproduced as Table VI G-1 in Appendix G. To assist the reader in the following discussion, this table is expanded as Table VI 9-1.

9.3.2.2 Changes to BEIR Risk Factors

For the upper bound, the advisory group on health effects recommended four small changes to the BEIR risk coefficients (Table VI 9-1), based on data accumulated since the BEIR Report was published. The bases for these changes are discussed in Appendix G and are merely recapitulated below.

First, the BEIR risk coefficient of 25 leukemia deaths per year per rem per million children irradiated in utero was primarily derived from the data of Stewart and Kneale.[1] Since publication of the BEIR Report, these authors have revised the dosimetry so that the risk coefficient is now reduced to 15 deaths per million per rem per year.

Second, the gastrointestinal tract is treated slightly differently. The BEIR risk coefficient for radiation-induced cancer of the gastrointestinal tract including the stomach is 1 death/per million per rem per year, which is further subdivided into a value of 0.6 for stomach and 0.4 for the rest of gastrointestinal tract. Examination of the data base for the latter value shows that 60% of the deaths from gastrointestinal cancer were really from cancer of pancreas and none from cancer of the large intestine. With these considerations, the advisory group on health effects recommended that the "gastrointestinal tract" be subdivided into the stomach, the rest of the alimentary tract, and the pancreas and that risk coefficients of 0.6, 0.2, and 0.2 death/per million per rem per year, respectively, be assigned. The dose to the lower large intestine is used in these calculations for both the stomach and the rest of the alimentary canal. This dosimetry is very conservative since the dose to the lower large intestine is much larger than the dose to the rest of the gastrointestinal tract. However, since the stomach and alimentary canal would contribute less than 10% of the latent cancer deaths, the error is small. The pancreatic dose is assumed equal to the dose to "other tissues."

Third, the BEIR Report assigns a value of 0.2 death per million per rem per year to bone cancer for the 10+ age cohort and lumps bone cancer deaths for children into the "all other cancer" category. For the reasons stated in Appendix G, the advisory group on health effects recommended that (a) the age cohort 0 to 20 be treated separately from adults, (b) the risk coefficient be doubled to 0.4 for this cohort and (c) for both

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[1]References are in Appendix G.

age cohorts, the latent period be reduced from 15 to 10 years. Since the incidence of bone cancer given in the BEIR Report is calculated in terms of the dose to mineral bone, the average dose to skeleton mass (mineral bone) is used.

Fourth, the above change in the risk coefficient for bone cancer fatalities in the 0 to 10 age cohort requires a corresponding reduction for this cohort in the "all other cancer" category. It should be noted that the "all other cancer" category is conservatively high since, unlike the BEIR Report, the thyroid cancer is being calculated separately here, but the "all other" category has not been reduced.

The BEIR Report estimated latent cancer fatalities for two plateau durations, 30 year and duration of life. For the reasons stated in Appendix G, the study uses the 30-year duration as being the more realistic. The effects of the above changes to the BEIR risk coefficients are summarized in Table VI 9-2. These are the values utilized for the upper bound estimates of latent cancer fatalities.

9.3.2.3 Expected Latent Cancer Fatalities

In this section, the risk coefficients stated in Table VI 9-2 are translated into the expected numbers of latent cancer fatalities per million man-rem. As an example, the calculation for leukemia is displayed in Table VI 9-3. The fractions of the population by age and the life expectancy are based upon 1970 census data; the former is shown graphically in Fig. VI I-1 of Appendix I. The years at risk are equal to either the plateau period or the remaining life expectancy, whichever is the shorter. For each age cohort, the expected leukemia deaths are the product of the population fraction, the years at risk, and the risk coefficient. A similar calculation is made for each organ and the results are summarized in Table VI 9-4.

The incidence of fatalities from latent cancer stated in Table VI 9-4 is calculated assuming either a single radiation exposure of relatively short duration or a stable exposed population. That is, a population whose age distribution is invariant. The first assumption is satisfied for the external exposure delivered by the passing cloud, and the second is assumed to be met for the chronic external exposure from contaminated ground. However, neither is satisfied for the internal exposure from internally deposited radionuclides inhaled from the passing cloud. Only people alive at the time of the accident would receive this exposure, which would continue through the remainder of their lives. Since the size of this population decreases by natural causes, the internal dose received within the period 40 to 50 years, for example, would cause fewer cancer deaths per unit dose than that received within the first year after the accident. A conservative estimate is made that all of the internal dose received during the first year would be delivered at the time of the accident, and the expected cancer fatalities stated in Table VI 9-4 are taken for this increment of internal dose. It is also conservatively assumed that the dose actually delivered within any subsequent time period is delivered at the beginning of that time period. For later time periods, for example, 11 to 20 years after the accident, there would be no irradiated age cohort less than 11 years so the expected leukemia deaths stated in Table VI 9-3 for the in utero, 0 to 0.99, and 1 to 10 cohorts are deducted from the overall total.[1] The results of such computations for each time period and each organ are stated in Table VI 9-5.[2] Since doses from internally deposited radionuclides were not computed beyond 50 years, the dose received within the 41 to 50-year time period is used for later time periods. Although this approach is conservative, the numbers are very small.

9.3.2.4 Reconcilation With the BEIR Report

The study thought it would be helpful to the reader to be able to compare the expected number of latent cancer deaths calculated in the preceding section with the corresponding estimates in the BEIR Report. There are important differences in the two calculations. As stated in the preceding section, the study considers a single release of radionuclides. The BEIR Report considers a continuous low-level irradiation.

[1]For example, for internal exposure delivered within 11 to 20 years after the accident, the expected leukemia deaths are 28.36 - 1.65 - 0.70 - 7.30 = 18.71 per million per man-rem.

[2]For the 1 - to 10-year time period, 75% of expected cancer deaths for the 0 to 0.99 cohort is included to account for children who were in utero at time of accident being alive in this time period.

Furthermore, the BEIR Report quotes several absolute numbers of expected cancer deaths (e.g., for whole U.S. population and for 1 million people) for radiation doses of 0.1, 0.17, and 5 rem per year.

Let us consider Table 3-4 of the BEIR Report; the portion of it that uses a 30-year plateau is reproduced as Table VI-9-6. This table is calculated from the risk coefficients stated in Table VI 9-1. The reader should note that the population base assumed is 198 million. Although the exposure is stated as 0.1 rem per year, the annual deaths are calculated on the basis that an individual has received 0.1 rem/per year since conception (i.e., a 40-year-old man received 1 rem by age 10 plus an additional 3 rem by age 40). The number of deaths listed for each age cohort is a summation of the deaths resulting from each annual increment of exposure accounting for the latent and plateau periods, which varies with age at irradiation. For example, the 179 other cancer deaths quoted opposite the 35-44 age cohort for irradiation received since age 10 years is the product of 23.838 million people times five other cancer deaths per million per rem per year[1] times 15-year exposure to 0.1 rem per year. The 15-year exposure accounts for the 15-year latent period and consideration of exposure only after age 10. The other values in the table may be calculated in a similar manner. Thus, the 516 + 1210 = 1726 total excess deaths are deaths per year based on a stable population of 197.9 million receiving 0.1 rem per year since conception.

The above 1726 deaths from 0.1 rem per year translates to about 3000 deaths from 0.17 rem per year, which number is stated in the summary on page 91 of the BEIR Report. The summary states a range of 3000 to 15,000 annual deaths from 0.17 rem per year. The low end of the range is based on the absolute risk model and a 30-year plateau, and the upper end on the relative model and a lifetime risk. For the reasons stated in Appendix G, the relative risk model and the lifetime plateau are not used by the study.

By using the values of 3000 deaths per year, 197.9 million population, and 0.17 rem per year, one can calculate 89 cancer deaths per year per million man-rem per year. This value reflects an equilibrium situation that is clearly different from the one-shot external exposure that is the basis for Table VI 9-4. For this reason, the numbers stated on page 91 of the BEIR Report are an inappropriate basis for risk calculations for reactor accidents.

9.3.3 CENTRAL ESTIMATE FOR LATENT CANCER FATALITIES

The central estimate for latent cancer fatalities is calculated by modifying the values stated in Tables VI 9-4 and VI 9-5 by the dose-effectiveness factors stated in Table VI 9-7. For example, if 100,000 people each receive 10 rem to their bone marrow at a rate of less than 1 rem per day, the expected leukemia deaths would be 0.2 times 28.4. The bases for the ranges on dose and dose rate and the factors themselves are discussed in Appendix G. The dose-effectiveness factors are applied to each organ except the breast for which evidence shows no reduced cancer incidence for fractionated doses delivered at high dose rates.

Since a reactor accident would be a one-time event, the dose rates would be at a maximum immediately after the accident and then decrease exponentially. With such time dependence, an individual might receive the first half of his total dose at a higher dose rate than the second half. For ease of calculation, the study examines only the initial dose rate and assumes that the whole dose is received at this rate. To offset this conservatism, the initial dose rate is determined by the dose received within the first month after the accident; that is, <1 rem per day is translated into <30 rem within the first month. Since most of the total man-rem would be accumulated from external exposure to the contaminated ground of the population that is not relocated (see section 11.2) and such doses are typically <10 rem at a dose rate of less than 1 rem per year, the above approximations will have a negligible effect on the calculations of total latent cancer fatalities.

Table VI 9-7 does not appear to envisage total doses in excess of 300 rem. Only individuals close to the reactor would receive such large doses to whole body or bone marrow and the associated dose rates would be >10 rem per day; therefore, no dose

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[1]Includes lung, gastrointestinal tract, breast, bone, and all other.

effectiveness factor is applied. Similarly the large local doses to the lung and regenerative cells of the gastrointestinal tract would all be received at dose rates in excess of 10 rem per day. In practice, only the factors on the diagonal of Table VI 9-7 are ever used. For example, it is impossible to receive less than 10 rem if the initial dose rate is greater than 10 rem per day.

9.3.4 LOWER BOUND FOR LATENT CANCER FATALITIES

It was emphasized in section 9.3.1 that, for low doses and low dose rates of low-LET radiation, the risk of cancer induction might be expected to be appreciably smaller per unit dose than for high doses and high dose rates. The BEIR Report (page 88) notes that the possibility of zero is not excluded by the data.

For the hypothetical reactor accident, a percentage of the exposed population would receive fairly large doses; thus, even if the incidence rate were zero for low doses, one would still expect a small number of expected latent cancer fatalities. In order to estimate this lower bound, the study estimates the number of latent cancer fatalities by assuming threshold doses of 10 or 25 rem.

9.3.5 THYROID NODULES AND CANCERS

A thyroid nodule is an abnormal growth that can be benign or malignant. If a nodule is thought to be malignant, it is usually surgically removed. The patient may also be given a therapeutic dose of iodine-131. Since the majority of thyroid cancers are well-differentiated, relatively slow growing, and relatively amenable to therapy, their mortality rate is much lower than that of other cancers (American Cancer Society, 1974). The study uses a 10% mortality rate for thyroid cancer. This rate would appear to be somewhat higher than the data presented in Tables VI H-6 and VI 9-9 which imply a 5% rate.

Appendix H reviews the available clinical data on thyroid nodules, both benign and malignant. There is strong evidence that there is a lower incidence of nodules from iodine-131 irradiation than from external x-rays; the clinical data for humans suggest that the factors are 1/53 and 1/67 for nodules and cancers respectively. Data from animal experiments suggest that these factors are somewhat larger, 1/10 to 1/20. Since the data are limited, the study chooses to use the most conservative factor of one-tenth. Iodine-131 doses in excess of 50,000 rem to the thyroid appear to cause ablation with no subsequent risk of nodules either benign or malignant.

In calculating the incidence of nodules, it is assumed that all thyroid doses from sources other than iodine-131 are equivalent to external x-ray irradiation. With these two assumptions, the dose to the adult thyroid is calculated as follows:

external dose to thyroid from passing cloud

+ external dose to thyroid from contaminated ground

+ internal dose during the first 30 days from all inhaled radionuclides except iodine-131

+ 1/10th of internal dose during the first 30 days from iodine-131

As shown below, dose factors for children (<20 years) are incorporated into the calculation of expected cases; their basis is explained in section 8.4.3.

For external x-ray irradiation, the incidence of nodules, both benign and malignant, appears to be linearly proportional to doses below 1500 rem. Appendix H recommends the following risk factors for external doses below 1500 rem:

	Nodules per 10^6 persons per rem per year		
	Benign	Cancerous	Total
Children (<20)	8.1	4.3	12.4
Adults	4.0	4.3	8.3

Table VI H-11 of Appendix H compares the above estimate for cancer induction to other estimates (BEIR, 1972; UNSCEAR, 1972); the above estimate is at the high end of their ranges. For higher doses, limited data suggest that the induction of nodules falls off rapidly with increasing dose, presumably because there is more extensive damage to the thyroid. Appendix H recommends the use of risk factors that are one-half of the above values for external doses in the range 1500 to 2500 rem. There is no evidence for the induction of nodules, either benign or malignant, at external doses above 2500 rem. Since there is no apparent risk of nodules for iodine-131 doses above 50,000 rem and it is assumed that iodine-131 is one-tenth as effective as external x-rays (i.e., 5000 rem of x-rays is equivalent to 50,000 rem of iodine-131), the above range is extended from 1500 to 5000 rem as a further conservatism.

Appendix H reviews the clincial data on latent periods and concludes that an average period is 10 years. The longest lapse of time reported for thyroid cancer is 40 years. On this basis, the study assumes a latent period of 10 years and a plateau period of 30 years; these values are consistent with the BEIR Report.

With the above considerations, the expected cases per million man-rem of thyroid nodules both benign and cancerous is calculated in Table VI 9-8. The fraction of the population by age and the life expectancy are based on 1970 census data. The expected cases are summarized below using the above calculation of dose:

	Expected nodules per 10^6 man rem	
Dose range (rem)	Benign	Cancerous
<1500	200	134
1500 - 5000	100	67
>5000	0	0

It should be emphasized that the available clinical data are from x-ray irradiation of small children and that the data for iodine-131 are very limited. The study recommends additional investigation in this subject in order to generate a stronger basis for risk estimates.

9.3.6 SPONTANEOUS INCIDENCE OF CANCER

As stated in section 9.3.1, radiation-induced cancers manifest themselves as an addition to the spontaneous incidence of cancer for the exposed population. As a basis for estimating such an increase, the current incidence (American Cancer Society, 1974) of cancer mortalities and morbidities are stated in Table VI 9-9.

9.4 GENETIC EFFECTS

9.4.1 INTRODUCTION

As discussed in Appendix I, the genetic material of the human consists of several thousand genes arranged in 46 bodies called chromosomes, 23 of which are inherited from each parent. There are thus 23 pairs of chromosomes, with each pair carrying a unique portion of the total genetic information. With the exception of a single pair, the sex chromosomes (XX in the female, XY in the male), the two members of each chromosome pair are approximately alike in genetic content; these 22 pairs of chromosomes are called autosomes to distinguish them from the sex chromosome pair.

Changes in the genetic material are called mutations. Mutations can occur spontaneously, from unknown causes, or can be induced by a variety of physical or chemical agents, one of which is ionizing radiation. The effects of mutations can be very obvious (e.g., albinism) or they can be so slight as to be detectable only by laboratory tests (e.g., protein variants). The health consequences of mutation can range from those of severe functional and structural abnormalities, generally with appreciable life shortening, to small and trivial effects that are neither disfiguring nor incapacitating. The effects considered here are those that produce significant disorders. Table VI 9-10 lists the major categories of genetic disease and their current incidences.

Mutations are said to be recessive or dominant. If a mutation is recessive, its effect will be apparent only if the offspring has inherited the same defective gene from both parents. If a mutation is dominant, its effects will be apparent when either the maternal or the paternal gene is defective.

The effect of ionizing radiation is to increase the frequency of mutation. Radiation does not, however, induce mutations that produce new kinds of effects: genetic disorders that would arise from radiation-induced mutation would not differ from those that have been occurring naturally for as long as man has existed. Living things have been exposed to background radiation from the very beginning, and this radiation may account for some fraction of the naturally occurring mutations in man. Thus, exposure to man-made radiation would not lead to the appearance of new and unexpected kinds of genetic disorders.

Radiation can also bring about chromosomal aberrations, either causing major shifts of material between chromosomes or altering the number of chromosomes. As a result, the new individual does not have a complete and proper set of hereditary information. The abnormal development caused by chromosomal aberrations may result in early death of the developing embryo (spontaneous abortion), which may be so early as to be undetectable (i.e., it may occur before the fertilized egg is implanted in the uterus).

The genetic effects of radiation are measured in terms of the frequencies of certain types of changes in the genetic material, and not in terms of human disorders. In order to express the estimates of genetic damage in terms of human health effects, it is necessary to use certain indirect methods, which are explained in Appendix I. The term "genetic damage" means damage to the reproductive cells. Hence, radiation-induced genetic damage affects the descendants of an exposed generation rather than the exposed generation itself.

The estimates made by the study are based on the recommendations contained in a report issued by the National Academy of Sciences-National Research Council (1972) on the biological effects of ionizing radiations, commonly known as the BEIR Report. The BEIR Report gives the base figures for the amount of human damage expected from exposure to low-level ionizing radiations, and these figures can be applied to virtually all of the exposures anticipated from a reactor accident. To apply the BEIR values to the accident situation, it is necessary only to take into account (1) the nature of the population exposed and (2) the amounts and distributions of the exposures.

9.4.2 POPULATION CHARACTERISTICS AND EXPOSURES

Reactor accidents could result in two types of exposure to radiation: external and internal (from inhaled or ingested radionuclides). The study therefore estimated human exposures for both external and internal irradiation, taking into account doses accumulated over various periods of time after the accident.

The dose of radiation from external sources would depend on the time elapsed since the accident and the radiological half-life of the radionuclides, which determines the rate at which they would be eliminated from the environment. All of the population that is exposed to the radioactive environment would be affected, including persons born after the accident, but the dose rate would decrease with time.

An internal burden of radionuclides would be acquired only by the population born prior to the accident. Exposure levels would depend on the time elapsed since the accident, the rate of radionuclide elimination from the body, and the radiological half-life of the radionuclides. The radiation dose from incorporated radionuclides would accumulate with time, and the genetic damage would depend on the time elapsed between radionuclide incorporation and conception. The total population effect would depend on the fraction of all newborns whose fathers are of such an age as to have incorporated radionuclides.[1]

These fractions are estimated from census data on the distribution of live births by paternal age (1973 data). It is assumed that the exposed population would in all respects, resemble the current (1974) domestic population of the United States. All effects are estimated per rem per million persons in the general population. Thus the calculations tabulated in this report can be applied to specific accident scenarios.

9.4.3 ESTIMATES OF HUMAN GENETIC DISORDERS

The BEIR Report estimated the increases in human genetic disorders in the first generation and at equilibrium (i.e., the steady condition in which the rate of arrival of new mutations equals the rate of elimination of old mutations) after an assumed permanent increase in background radiation. Since a reactor accident would be a one-time event, there would be an initial increase in mutations which will be slowly eliminated from the population; a modified calculation is therefore necessary. The study has chosen to estimate the increased incidence expected in each of two 30-year time periods after the accident and to estimate the total consequences of genetic damage induced by radioactive material released by the accident. For this calculation, it is necessary to take into account the overlapping of the generations produced by the exposed population.

The results of these calculations are shown in Tables VI 9-11 and VI 9-12 for external and internal exposure, respectively. The methods used are described in Appendix I.

[1] As explained in Appendix I, the genetic damage results almost entirely from the irradiation of the fathers.

9.4.3.1 Single-Gene Disorders

The BEIR Report used the current incidence of genetic disorders in human populations as the basis for estimating the increase in disorders that would follow an increase in the mutation rate. The method is to determine two factors: (1) the increase in mutation rate that would be expected from a given radiation exposure and (2) the extent to which the incidence of any given kind of genetic disorder is dependent on recurrent mutation. These factors permit estimating the fractional increase in human genetic disorders to be expected from any set of radiation exposures. Given the current incidence of human genetic disorders, this increase can be expressed in terms of the probable absolute increase in the incidence of genetic disorders.

The effectiveness of radiation in causing genetic change is sometimes expressed as a "doubling dose"; that is, the radiation dose that produces as many additional mutations as already occur spontaneously. The BEIR Report estimated that the doubling dose for humans probably lies between 20 and 200 rem; a more realistic estimate would probably place this value near 100 rem, which is the value used by the Reactor Safety Study. It is important to note that a high doubling dose means that a large amount of radiation is needed to produce a given effect. The lower the estimate of doubling dose, therefore, the more conservative the estimate.

If mutation rates were to remain at a higher level for a number of generations, as a result of a permanent increase in background radiation, a new equilibrium would be reached between new occurrences of mutation and the elimination of old mutations from the population. At this point, the incidence of genetic diseases maintained by recurrent mutation would be proportionate to the mutation rate, and hence the increase in the incidence of genetic disorders would be proportionate to the increase in mutation rate. However, it requires many generations to reach this equilibrium, and the estimation for earlier generations would depend on the rate at which mutations are eliminated from the population.

The genetic disorders that would most clearly be dependent on the recurrence of mutation would be those caused by a dominant mutation in one of the autosomes. For autosomal dominant disorders, the equilibrium incidence is directly related to the mutation rate. A single radiation exposure would produce an increase in the incidence of autosomal dominant disorders in the offspring of the exposed generation, with many of these genes being transmitted to the second and subsequent generations. It is assumed that there is a 20% elimination of autosomal dominants in each generation, so that over all time, about one-fifth of the total number of genetic disorders attributable to radiation-induced autosomal dominant mutations would be seen in the first-generation offspring of the exposed persons. Sex-linked mutations (i.e., mutations in genes contained in the X sex chromosome) are similar in behavior to autosomal dominant mutations, although they do differ in some details.

Human genetic disorders due to autosomal recessive mutations would show only very slow increases, which the BEIR Report regarded as being negligible in comparison with the increases expected for other disorders.

9.4.3.2 Multifactorial Disorders

Multifactorial disorders are those that depend on more than a single gene pair. These represent a large and important class of human disorders. The dependence of these disorders on recurrent mutation is more complex and more difficult to assess. The BEIR Report estimated that 5 to 50% of the incidence of these may depend on the mutation rate, and this range has been adopted here. The rate of elimination of mutant genes in this category has been taken to be 10% per generation, as in the BEIR Report. This rate of elimination would result in about one-tenth of the total amount of multifactorial disorders, ascribable to mutations resulting from the accident, would be seen in the immediate offspring of the exposed persons. For an expected transmission of 90% from generation to generation, the increase in incidence would slowly disappear as the damage is eliminated from the population.

The BEIR Report used a survey of the population of the Northern Ireland as the best available source of information on the current incidence of genetic disorders. It appears likely that the values of incidence that were derived may be too high, in which case the estimates of genetic damage should be correspondingly lowered.

9.4.3.3 Chromosomal Disorders

The estimates of incidences of chromosomal disorders are also based on the BEIR Report, where they were estimated by direct methods, and not through the application of a doubling dose to current incidences. Chromosomal damage often results in early spontaneous abortions (loss of the fetus during the first trimester of pregnancy). Of the affected individuals that survive and show adverse effects, most are sterile. Deleterious effects after the first generation would be limited to the offspring of carriers of balanced rearrangements; it can be expected that about one-half of the offspring of such carriers would be abnormal and that most of the abnormal individuals would be lost very early in development, during the first trimester of pregnancy.

The study has defined genetic effects in terms of live births with a genetically caused disorder that could be transmitted to their children. This definition excludes spontaneous abortions.

TABLE VI 9-1 RISK COEFFICIENTS FOR LATENT CANCER FATALITIES FROM BEIR REPORT

Type of Cancer	Age at Time of Irradiation	Latent Period (years)	Plateau Period (years)	Risk Coefficient (deaths/10^6/ yr/rem)
Leukemia	In utero	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)	In utero	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.

TABLE VI 9-2 UPPER BOUND RISK COEFFICIENTS FOR LATENT CANCER FATALITIES

Type of Cancer	Age at Time of Irradiation	Latent Period (years)	Plateau Period (years)	Risk Coefficient (deaths/10^6/ yr/rem)
Leukemia	In utero	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 alimentary 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	In utero	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.

TABLE VI 9-3 CALCULATION OF EXPECTED LEUKEMIA DEATHS FOR EXTERNAL EXPOSURE

Age Cohort (years)	Fraction of Population	Life Expectancy (years)	Latent Period (years)	Years at Risk	Risk Factor (10^6/rem/year)	Expected Cases
In utero	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	0.09
						28.36

TABLE VI 9-4 EXPECTED LATENT CANCER (EXCLUDING THYROID) DEATHS PER MILLION MAN-REM OF EXTERNAL EXPOSURE

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

TABLE VI 9-5 EXPECTED TOTAL LATENT CANCER (EXCLUDING THYROID) DEATHS PER 10^6 MAN-REM FROM INTERNAL RADIONUCLIDES DELIVERED DURING SPECIFIED PERIODS

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.

TABLE VI 9-6 CALCULATION OF ANNUAL NUMBER OF EXCESS CANCER DEATHS IN THE U.S. POPULATION FROM CONTINUOUS EXPOSURE TO 0.1 REM/YEAR, USING ABSOLUTE RISK MODEL (TABLE 3-4 OF BEIR REPORT)

Age	1967 U.S. Pop'n (millions)	Leukemia: Excess Deaths Due to Irradiation in Period			Total Excess Deaths	All Other Malignancies: Excess Deaths Due to Irradiation During			Total Excess Deaths
		In utero	0-9 yr	10+ yr		In utero	0-9 yr	10+ yr	
0-4	19,191	36	3	-	39	36	-	-	36
5-9	20,910	39	23	-	62	39	-	-	39
10-14	19,885	-	38	2	40	-	-	-	-
15-19	17,693	-	35	10	45	-	4	-	4
20-24	14,572	-	29	15	44	-	11	-	11
25-29	11,958	-	24	19	43	-	12	15	27
30-34	10,860	-	11	22	33	-	11	41	52
35-44	23,838	-	1	60	61	-	24	179	203
45-54	22,588	-	-	56	56	-	11	282	293
55-64	17,571	-	-	46	46	-	-	263	263
65-74	11,678	-	-	29	29	-	-	175	175
75-84	5,945	-	-	15	15	-	-	89	89
85+	1,174	-	-	3	3	-	-	18	18
Total	197,863	75	164	277	516	75	73	1062	1210

TABLE VI 9-7 DOSE-EFFECTIVENESS FACTORS

Total Dose (rem)	Dose Rate (rem per 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

TABLE VI 9-8 CALCULATION OF EXPECTED CASES PER MILLION MAN-REM OF BENIGN AND CANCEROUS THYROID NODULES

Age Cohort (years)	Fraction of Population	Life Expectancy (years)	Latent Period (years)	Years at Risk	Age Dose (a) Factor	Benign Nodules		Cancers	
						Risk Coefficient(b)	Expected Cases	Risk Coefficient(b)	Expected Cases
0 - 0.99	0.014	71.3	10	30	1.0	8	3.36	4.3	1.81
1 - 10	0.146	69.4	10	30	1.9	8	66.58	4.3	35.78
11 - 20	0.196	60.6	10	30	1.6	8	75.26	4.3	40.45
21 - 30	0.164	51.3	10	30	1	4	19.68	4.3	21.1
31 - 40	0.118	42.0	10	30	1	4	14.16	4.3	15.22
41 - 50	0.109	32.6	10	22.6	1	4	9.85	4.3	10.59
51 - 60	0.104	24.5	10	14.5	1	4	6.03	4.3	6.48
61 - 70	0.080	17.1	10	7.1	1	4	2.27	4.3	2.44
71 - 80	0.044	11.1	10	1.1	1	4	0.14	4.3	0.21
80+	0.020	6.5	10	0	1	4	0	4.3	0
Total							197.4		134.1

(a) From Table VI 8-5

(b) Number of cases per million population per rem per year.

TABLE VI 9-9 CANCER MORTALITY RATES (PER 10^6 PER YEAR) WITHIN UNITED STATES

Cancer Type	Mortality
Leukemia	71
Lung, trachea, bronchi	379
Stomach	67
Rest of alimentary tract	264
Pancreas	91
Breast	152
Bone	9
Thyroid	5
All other	666
Total	1704

TABLE VI 9-10 CURRENT INCIDENCE OF SPONTANEOUSLY OCCURRING GENETIC DISORDERS

Disorder	Disease incidence among newborns and spontaneous abortions per million population per 30 years
Autosomal dominant disorders	4,200
Multifactorial disorders[a]	17,000
Chromosomal and recessive disorders	2,700
Spontaneous abortions	23,500

[a] Denoted by congenital anomalies, anomalies expressed later, and constitutional and degenerative diseases in the BEIR Report.

TABLE VI 9-11 DISORDERS AND SPONTANEOUS ABORTIONS ATTRIBUTABLE TO RADIATION FROM EXTERNAL SOURCES DERIVED FROM RELEASES AT THE TIME OF THE HYPOTHETICAL ACCIDENT

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)
		Autosomal Dominant Disorders		
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
		Multifactorial Disorders		
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
		Disorders Due to Chromosomal Aberrations		
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
		Spontaneous Abortions		
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 VI 9-12 DISORDERS AND SPONTANEOUS ABORTIONS DUE TO RADIATION FROM INTERNAL SOURCES INCORPORATED AT THE TIME OF THE HYPOTHETICAL ACCIDENT

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)
		Autosomal Dominant Disorders		
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)
		Multifactorial Disorders		
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)
		Disorders Due to Chromosomal Aberrations		
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)
		Spontaneous Abortions		
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.

Section 10

Demographic Data

10.1 SUMMARY

Calculation of exposure of the population in the path of a plume traveling over a region requires that the specific geographic population distribution around the release site be known. Such information for the present study was obtained from a data base established by the Office of Telecommunications, U.S. Department of Commerce, in programs supplied by the Office of Radiation Programs, U.S. Environmental Protection Agency (Athey et al., 1973). The details of this program are given in section 10.3.

Evaluation of the economic impact requires knowledge of not only the distribution of population but also the general nature of the land use by sector. The real property loss due to damage, the agricultural damage and costs, and decontamination costs all involve knowledge of the percent of the area used for agricultural and for urban activities. For example, economic damage to agricultural land is calculated from the equation

$$V_f = V_a ALF,$$

where V_f is the value of farmland in the sector, V_a is the value of farmland per acre, A is the area of the sector, L is the land fraction in the sector, and F is the fraction of land being farmed.

However, in estimating costs related to nonagricultural assets, it is assumed that the value of these assets is proportional to the population,

$$V = CP,$$

where V is the value of nonagricultural assets in the sector, C is the value per capita, and P is the population in the sector. This calculation is discussed in detail in section 10.5 under land use character.

10.2 DATA COLLECTION AND REACTOR SITES

It was considered that the use of actual demographic and meteorological data for individual reactor sites would produce more realistic results than would the use of average distributions. To this end, a list was compiled of the first 100 commercial light-water reactors, which is about the number expected to be operational before 1980 (see Table VI 10-1). Only reactors with electrical outputs greater than 400 MWe (1250 MWt) were considered.

The study group did not have the time or resources to generate all the detailed data needed to evaluate each of the 68 sites (on which the first 100 reactors are located). To reduce this task to a manageable size, six composite sites were constructed: (1) eastern seashore, (2) eastern river, (3) southern inland, (4) midwestern plain, (5) lake shore, (6) western seashore. Each of the 100 reactors was assigned to one of these six composite sites.

Land-use fractions were derived from the Statistical Abstracts of the United States, which gives the fraction in agricultural use for each state. Typical land-usage fractions were then generated for all geographical sectors for each of the six composite sites, by methods discussed below. Table VI 10-2 shows the numbers of reactors assigned to each of the six sites.

TABLE VI 10-1 THE FIRST 100 COMMERCIAL LIGHT-WATER REACTORS IN THE UNITED STATES

No.	Reactor	Reactor Capacity (MWt)
1	Haddam Neck	1725
2	Millstone, Units 1 and 2	1956 and 2484
3	Calvert Cliffs, Units 1 and 2	2535
4	Maine Yankee	2370
5	Pilgrim, Unit 1	1992
6	Seabrook, Unit 1	3600
7	Forked River, Unit 1	3210
8	Oyster Creek	1920
9	Salem, Units 1 and 2	3270 and 3345
10	Fitzpatrick	2463
11	R. E. Ginna	1470
12	Indian Point, Units 2 and 3	2619 and 2895
13	Nine Mile Point, Units 1 and 2	1875 and 3240
14	Shoreham	2457
15	Sterling	3450
16	Beaver Valley, Units 1 and 2	2556
17	Limerick, Unit 1	3195
18	Peach Bottom, Units 2 and 3	3195
19	Susquehanna, Unit 1	3150
20	Three Mile Island, Units 1 and 2	2457 and 2715
21	Vermont Yankee, Unit 1	1542
22	Arkansas, Units 1 and 2	2550 and 2736
23	Farley, Units 1 and 2	2487
24	Browns Ferry, Units 1, 2, and 3	3195
25	Bellefonte, Unit 1	3639
26	Crystal River	2475
27	St. Lucie, Unit 1	2430
28	Turkey Point, Units 3 and 4	2079
29	E. I. Hatch, Units 1 and 2	2358 and 2385
30	Riverbend, Unit 1	2802
31	Waterford, Unit 3	3339
32	Grand Gulf, Unit 1	3750
33	Brunswick, Units 1 and 2	2463
34	McGuire, Units 1 and 2	3540
35	North Anna, Units 1, 2, and 3	2694, 2694, and 2721
36	Surry, Units 1 and 2	2364
37	Catawba, Unit 1	3459
38	Oconee, Units 1, 2, and 3	2658
39	H. B. Robinson, Unit 2	2100
40	Virgil C. Summer	2700
41	Sequoyah, Units 1 and 2	3420
42	Watts Bar, Units 1 and 2	3507
43	Braidwood, Unit 1	3360
44	Byron, Unit 1	3360
45	Dresden, Units 2 and 3	2427
46	LaSalle, Units 1 and 2	3234
47	Quad-Cities, Units 1 and 2	2400
48	Zion, Units 1 and 2	3150
49	Bailly	1943
50	Duane Arnold	1707
51	D. C. Cook, Units 1 and 2	3180
52	Fermi, Unit 2	3279
53	Midland, Unit 2	2454
54	Palisades	2100
55	Monticello	1635
56	Prairie, Units 1 and 2	1590
57	Fort Calhoun, Unit 1	1372
58	Cooper	2334
59	Davis-Besse, Unit 1	2718
60	Perry, Unit 1	3615
61	Zimmer, Unit 1	2430
62	Kewaunee	1623
63	Point Beach, Units 1 and 2	1491
64	Palo Verde, Unit 1	3713
65	Trojan	3390
66	Diablo Canyon, Units 1 and 2	3252 and 3318
67	San Onofre, Units 1 and 2	1290 and 3420
68	Rancho Seco	2739

TABLE VI 10-2 NUMBER OF REACTORS ASSIGNED TO THE COMPOSITE SITES

Characteristics	Number of Sites	Number of Reactors BWR	PWR
Atlantic coastal site	10	5	9
Large river valley in northeast	10	6	8
Great Lakes shore	4	3	2
Southeast river valley influenced by Bermuda High	17	7	23
Central midwest plain	23	13	18
Pacific coastal site	4	0	6
	68	34	66

The following illustrates the use of the population density distributions in the calculations, with the first site as an example. Fourteen reactors are assigned to this site. The actual population around each of these 14 reactors was calculated for 16 sectors of 22.5° angle. These 224 sectors were then ranked from highest to lowest population density based on the cumulative population within 50 miles. The population distributions of the 224 sectors were used to generate 16 representative sectors in the manner indicated in Table VI 10-3. For instance, the highest ranked sector (of the 224) was assigned to sector 1 of the composite site. The third sector of the composite site was assigned a population distribution that, mesh point by mesh point, is the average of the population distributions in the third and fourth most populous sectors of the 224.

TABLE VI 10-3 CONSTRUCTION OF COMPOSITE REACTOR SITES

Sector	Sectors from Ranked Listing	Conditional Probability of Sector Being Exposed
1	1	1/224
2	2	1/224
3	3, 4[a]	2/224
4	5, 6[a]	2/224
5	Average of next 6	6/224
6	Average of next 6	6/224
7	Average of next 12	12/224
8	Average of next 22	22/224
9	Average of next 22	22/224
10	Average of next 23	23/224
11	Average of next 22	22/224
12	Average of next 22	22/224
13	Average of next 20	20/224
14	Average of next 20	20/224
15	Average of next 21	21/224
16	Average of next 22	22/224
		224/224

[a] Two reactors in one site.

10.3 POPULATION DENSITY

The population data bases used are those developed by the U.S. Census Bureau in its 1970 Master Enumeration District List with Coordinates. Athey et al. (1973) have put this information into a usable package containing the housing and population counts for each census enumeration district and the geographic coordinates of the population centroid for the district. In densely populated areas, the size of a census enumeration district is usually a physical city block ("block group"). In rural areas, the district may cover several square miles. Over the United States, 250,000 enumeration districts have been defined, with an average population of 800 per district in 1970.

The population distribution around each reactor site is determined from its latitude and longitude, and the radial meshing spacing for each 22.5° sector. The population within each annular sector is the summation of the contained districts.

10.4 POPULATION PROJECTIONS TO 1980

The population data base was the 1970 census. These census data include extrapolation factors for each enumeration district, and these factors can be used to estimate the population in 1980 or other future times. The population has grown by 4.6% since 1970 and is expected to grow an additional 4.2% by 1980. These percentage increases could be applied to all calculated consequences but are less than the associated uncertainties.

10.5 LAND-USE CHARACTER

Land-use characterization is required for estimating the cost of decontamination and agricultural costs. The percentage of each annular sector that is land or water were determined and is then identified by the state in which it falls. From City and County Data Book 1972, the percentage of the land in use for agriculture purposes is found as a function of state. This information is stated in Table VI 10-4.

REFERENCES

Athey, T. W., R. A. Tell, and O. E. Janes, 1973, The Use of an Automated Population Data Base in Population Exposure Calculations, U.S. Environmental Protection Agency.

U.S. Department of Commerce, County and City Data Book (published annually).

U.S. Department of Commerce, 1970, Master Enumeration District List with Coordinates.

U.S. Department of Commerce, Statistical Abstract of the United States (published annually).

TABLE VI 10-4 AGRICULTURAL LAND USE CHARACTER

State	Fraction of State Used as Farm Land	Average Annual Sale of Farm Products[a] ($/acre-year)	Average Share of Dairy Products	Average Value of Farm [a] ($/acre)	Major Farming Season
Maine	0.089	170	0.177	340	May-September
New Hampshire	0.106	120	0.453	500	May-September
Vermont	0.323	110	0.811	470	May-September
Massachusetts	0.140	290	0.291	1180	May-September
Rhode Island	0.102	340	0.258	1520	May-September
Connecticut	0.174	400	0.298	1930	May-September
New York	0.332	140	0.552	520	May-September
New Jersey	0.215	310	0.202	2260	May-September
Pennsylvania	0.309	160	0.420	790	May-September
Ohio	0.652	110	0.181	620	May-September
Indiana	0.761	120	0.078	560	May-September
Illinois	0.838	130	0.052	640	May-September
Michigan	0.327	100	0.275	520	May-September
Wisconsin	0.520	120	0.548	460	May-September
Minnesota	0.568	90	0.215	330	May-September
Iowa	0.938	160	0.051	560	May-September
Missouri	0.734	67	0.082	350	May-September
North Dakota	0.973	26	0.048	130	May-September
South Dakota	0.937	31	0.060	110	May-September
Nebraska	0.936	70	0.025	220	May-September
Kansas	0.944	55	0.041	210	May-September
Delaware	0.531	280	0.052	790	April-October
Maryland	0.439	180	0.245	1220	April-October
Virginia	0.418	79	0.176	560	April-October
West Virginia	0.282	36	0.220	300	April-October
North Carolina	0.408	140	0.069	570	April-October
South Carolina	0.361	77	0.091	450	April-October
Georgia	0.425	97	0.067	470	April-October
Florida	0.405	120	0.096	550	April-October
Kentucky	0.629	71	0.141	410	April-October
Tennessee	0.569	61	0.176	460	April-October
Alabama	0.421	73	0.074	340	April-October
Mississippi	0.530	63	0.089	320	April-October
Arkansas	0.472	92	0.036	380	April-October
Louisiana	0.340	75	0.131	500	April-October
Oklahoma	0.818	40	0.063	280	April-October
Texas	0.850	34	0.054	240	April-October
Montana	0.675	14	0.023	100	May-September
Idaho	0.272	67	0.082	280	May-September
Wyoming	0.570	10	0.022	75	May-September
Colorado	0.553	44	0.038	170	April-October
New Mexico	0.602	11	0.049	66	April-October
Arizona	0.526	24	0.048	130	April-October
Utah	0.215	28	0.166	170	April-October
Nevada	0.152	11	0.082	140	April-October
Washington	0.412	65	0.123	300	May-September
Oregon	0.293	44	0.085	290	May-September
California	0.357	160	0.104	510	April-October

(a) The values are adjusted from the year of survey (1969) to 1975 values.

Section 11

Mitigation of Radiation Exposure

The preceding sections have described the deposition of radioactive material released by a reactor accident into man and onto the ground, the methods used to estimate the radiation dose, and the resultant health effects and property damage. This section primarily discusses the actions that could be taken to mitigate the radiation exposure and hence the health effects. In addition to describing the effects of societal actions, it is convenient, since the technical bases are similar, to also cover some mitigating factors that do not depend on human agents (e.g., the normal self-shielding of terrain).

It is helpful to distinguish between two time periods: (1) immediate actions to reduce early exposure during the passage of the radioactive cloud and (2) long-term actions to reduce chronic exposure from radioactive material deposited on the ground or vegetation. Since the radioactive material is transported by the atmosphere at wind speeds of 1 to 22 mph and an individual's exposure to the cloud would be terminated within an hour or so, immediate actions, to be of any value, must be taken within hours of the accident. Possible actions are evacuation, sheltering (i.e., ordering the public to remain indoors), and issuance of potassium iodide pills to block the absorption of inhaled radioiodines by the thyroid. (In Great Britain, potassium iodate pills, which are similar in action to potassium iodide pills, are stockpiled at reactors for use in an emergency.) The first two actions are mutually exclusive, but the third could be taken in conjunction with either evacuation or sheltering. Section 11.1 discusses evacuation. Sheltering might reduce the dose incurred from both inhalation and external cloudshine.

There are several modes of chronic exposure, the more important being direct irradiation from contaminated ground and ingestion of contaminated milk or crops. Under the scenarios evaluated in this study, the former would contribute about 67% and the latter about 33% of the chronic population dose.[1] Chronic exposure would generally involve lower dose rates than early exposure, but the time scales would run from several weeks for milk ingestion and one season for crops to 50 years or more for ground contamination. For these two reasons, long-term mitigating actions could be delayed for days or weeks while the situation is fully evaluated. Only a marginal increase would occur in the population dose, but treatment would have to continue for a long period. There are basically two long-term mitigating actions: interdiction and decontamination of land. Interdiction means denial of the use of land for a period of time either by relocating people or by impounding milk and crops. (Relocation should be distinguished from evacuation. Relocation could be initiated within days or weeks after a release and might continue for months or years, whereas evacuation would be initiated immediately and would last only for a day or two.)

In order to facilitate an understanding of the long-term mitigating actions described in section 11.2, a simplified interdiction model is shown in Fig. VI 11-1. For a ground-level release, the degree of ground and vegetation contamination would decrease monotonically with distance from the reactor. For self-consistent health criteria, the most restrictive contamination criterion would be applied to milk, and hence the largest interdicted area would be associated with milk impoundment. A less restrictive criterion would be applied to the direct contamination of foliage, and therefore the interdicted crop-growing area would be smaller. The least restrictive criterion would be applied to the continuing occupancy by people. Hence the critical exposure mode would be direct external irradiation from contaminated ground. Decontamination of land can be used to reduce the period of land interdiction. The choice between interdiction and decontamination is an economic one, and some analyses are described in section 11.2.2.

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[1] The percentages stated are based on the assumption that an individual would receive all nutrients from the contaminated area. In a more realistic case, the percentages would be 90 and 10%, respectively.

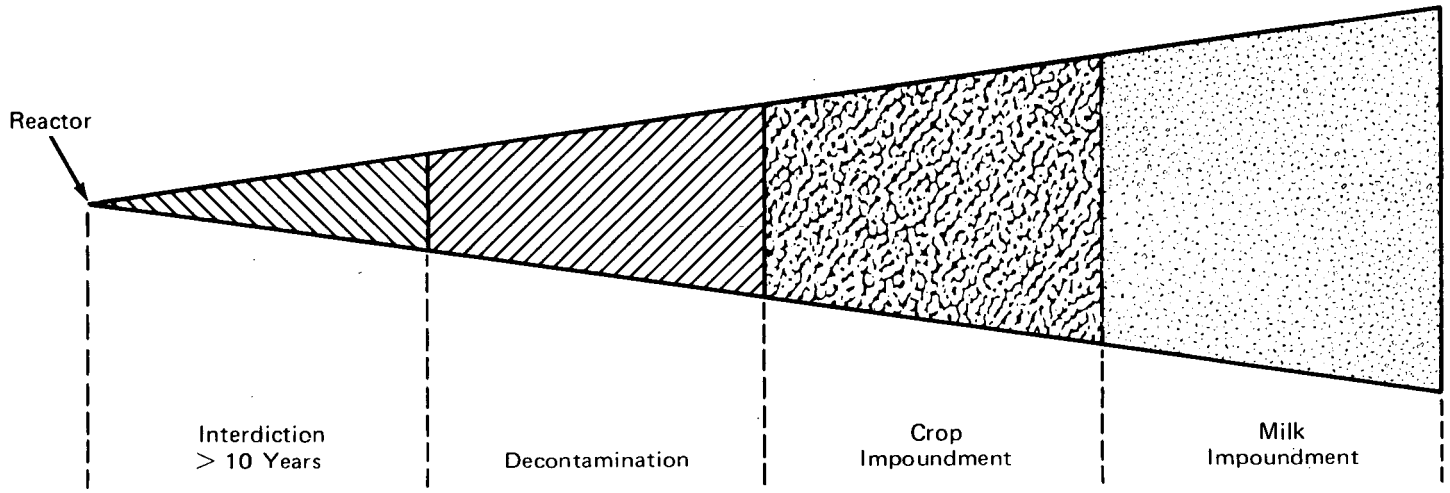

FIGURE VI 11-1 Simplified interdiction model.

Shielding or shelter enters into both short- and long-term actions to mitigate exposure and also into the normal dosimetry for chronic exposure. Section 11.3 describes all shielding factors for the consequence model. For early exposure, it describes the shielding of ground contamination by an automobile and the shielding of the effects of the passing cloud by buildings. For chronic exposure, the shielding of ground contamination by buildings is described. For all shielding by buildings, it is necessary to consider how and where the public spends its time, a topic covered in section 11.3.3. The effective shielding factors are summarized in section 11.3.2.

11.1 ACTIONS TO REDUCE EARLY EXPOSURE DURING CLOUD PASSAGE

11.1.1 EVACUATION

11.1.1.1 Introduction

As stated in Table VI 2-1, there would be a few hours' warning of a significant release of radioactive material, and, depending on the wind speed, several more hours could pass before the radioactive cloud reached a particular population group. This time period could be used for evacuation. Evacuation experience in the period of 1959 to 1973 has been summarized by Hans and Sell (1974) for the U.S. Environmental Protection Agency (EPA). Statistical analysis of the EPA data shows an underlying behavior pattern for mass evacuations that can be modeled for use in risk assessments. This section outlines the principal findings of this statistical analysis and describes the model; the reader is referred to Appendix J for the complete report.

11.1.1.2 Analysis of EPA Data

The EPA report provides data on 64 evacuations caused by transportation accidents (usually involving noxious gases), floods, and hurricanes. For 33 such events there are sufficient data to permit the type of statistical analysis described in Appendix J. The parameters that might be expected to influence an evacuation include (1) area evacuated, (2) distance moved, (3) number of people moved, and (4) population density. The range of values for these parameters in the 33 evacuations is stated in Table VI 11-1. On comparison, the corresponding values for the hypothetical reactor accident are seen to be of the same order of magnitude as the range of experience. Furthermore, the evacuations described by EPA were carried out predominantly by private vehicles, which are the expected mode of transportation in the event of a reactor accident. Thus, the EPA data appear to be a reasonable basis for an evacuation model for reactor accidents.

TABLE VI 11-1 COMPARISON OF REACTOR EVACUATION PARAMETERS TO EXPERIENCE PARAMETERS

Parameters	EPA Data		Potential Values for Reactor Accidents
	Minimum	Maximum	
Area evacuated, square miles	0.08	1,200	400
Distance moved, miles	0.5	150	20
Number of evacuees	20	150,000[a]	0 to 733,000
Population density (number per square mile)	6.7	19,000	0 to 2986

[a] The EPA data contained one evacuation of 501,000 persons, but this was not analyzed due to insufficient data.

From the viewpoint of the evacuation model, the key conclusions of the statistical analyses are as follows: (1) a log-normal distribution can be used to describe the effective evacuation speed, (2) the likely speeds are slow, (3) the range of potential speeds is very large and (4) the number of persons evacuated had no significant effect on the speed of evacuation. The effective evacuation speed is defined as the distance moved in the time period after the warning; it includes any initial confusion and lost motion.

The data on evacuations caused by transportation accidents, floods, and hurricanes are analyzed both separately and together. The effective evacuation speeds for all three categories are describable by a log-normal distribution; the log-normal fits to the data points are not rejected at significance levels ranging from about 25 to 50%. However, the individual log-normal parameters (i.e. effective speeds) for the three evacuation categories are apparently different. For each evacuation category, the modal, mean, and 90% probability interval (5th to 95th percentiles) for the effective speed are stated in Table VI 11-2.

TABLE VI 11-2 EFFECTIVE EVACUATION SPEED PARAMETERS FOR THE LOG-NORMAL DISTRIBUTION

	Effective Speed (mph)		
Evacuation Category	Modal	Mean	90% Probability Interval
Transportation	0.08	4.7	0.1 to 20
Hurricanes	0.63	13.8	0.45 to 55
Floods	0.08	2.3	0.06 to 9
All 33 evacuations	0.10	6.7	0.1 to 30

A secondary finding, which is not explicitly used in the evacuation model, is that the effective evacuation speed is almost linearly proportional to the distance traveled. This correlation is shown in Fig. VI J-5 of Appendix J and is not rejected at a 0.1% significance level, which indicates a very strong correlation.

Of equal importance to the above positive correlations are the null hypotheses that were tested and not rejected. As reported in detail in Appendix J, the effective evacuation speed is found to be apparently independent of the area evacuated, the number of evacuees, the time period, weather, and time of day. However, these conclusions may be partly due to the character of the available data; the recorded evacuation periods varied over only a small range, so that recording errors could mask some correlations. A more subtle finding is that the variance in the effective evacuation speed appears to be independent of the number of evacuees. This result suggests that populations move as a group since otherwise a smaller variance in the average group speed would be expected for large groups than for small ones. Civil Defense personnel have observed a minority of approximately 5% who stay behind and never evacuate, but the concept of such a nonparticipating minority is not resolvable from the analyses performed.

11.1.1.3 Evacuation Model for Reactor Accidents

In the evacuation model incorporated into the consequence calculations, the evacuation area is postulated to be shaped like a keyhole centered on the prevailing wind direction at the time of the release. The dimensions of the area are chosen to be 5 and 25 miles and 45° (see Fig. VI 11-2) for the following reasons. The evacuation would be carried out to mitigate the early exposure to individuals; the early exposure from the passing cloud would contribute little to the population dose. Since the resources of the local authorities -- all that would be available immediately after the accident -- are limited, it would be desirable to minimize the evacuation area and the number of evacuees. On the other hand, the goal would be to evacuate anyone who might receive a significant dose. The values 25 miles and 45° represent a compromise. In addition to this sector, it was judged prudent to evacuate all people within a 5-mile radius of the reactor. The evacuation costs are calculated on the basis of the number of people living in this evacuation area.

In order to calculate doses to individuals within the evacuation area, people are postulated to move radially away from the reactor at a specified effective evacuation speed until the cloud reaches them and then to move in a circumferential direction. For example, if an effective evacuation speed of 1 mph is assumed, people located between 2 to 3 miles from the reactor are assumed to be 7 to 8 miles away from the reactor 5 hours after the warning.

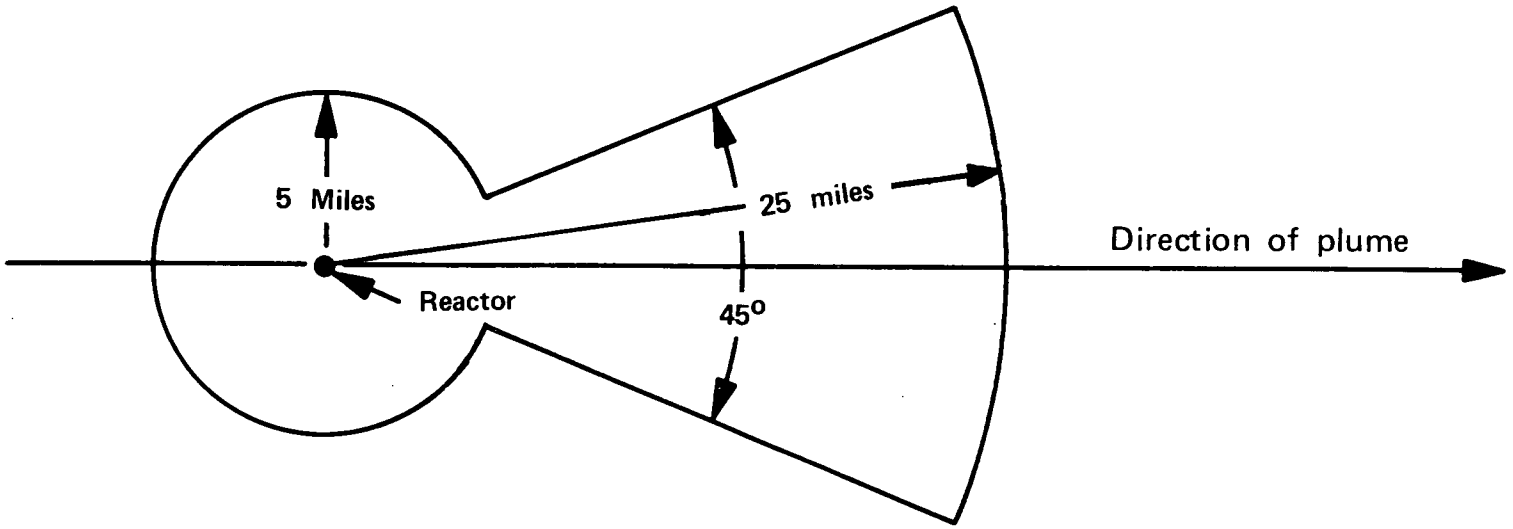

FIGURE VI 11-2 Evacuation area used for cost calculations.

Evacuations due to transportation accidents are used as the descriptive model for reactor accidents since they often involve airborne releases of noxious gases and the warning times and evacuation movements are comparable. Since there is a large variation in evacuation speed, the use of one "representative" speed might not be appropriate. The log-normal distribution is therefore represented by three discrete evacuation speeds of 0, 1.2, and 7.0 mph, with probabilities of 30, 40, and 30%, respectively. As shown in Fig. VI 11-3, the 1.2- and 7.0-mph values are the probability midpoints of the associated intervals (the 1.2-mph value is the 50th percentile, and the 7.0-mph value is the 85th percentile). Although the probability midpoint of the first interval is 0.2 mph, zero mph (ineffective evacuation) is assigned. On the other hand, although the presence of a 5% nonparticipating minority is considered to be a realistic phenomenon, it was not incorporated into the model because its effect did not seem to justify an increase in the complexity of the consequence model. The net effect is thought to be conservative since a 30% probability of ineffective evacuation has higher consequences than a 100% probability of 5% of the population remaining. Future work will study the effect of the nonparticipating minority.

With respect to the relation between effective speed and distance relation shown in Fig. VI J-5 of Appendix J, the 1.2- and 7-mph values correspond to evacuation distances of 5 and 35 miles, respectively. If the detailed distance relation were incorporated into the evacuation model, it might show the present, discreticized model to be conservative since the evacuation speed would increase with the distance traveled and the variability of speed for a given distance would be smaller than that in the present model. The treatment of this distance relation is somewhat complex and will be deferred for future study.

None of the evacuations covered in the EPA report involved a major population center (e.g., New York City). It is not to be expected that either the results of the statistical analyses or the evacuation model would be applicable to such centers. However, this restriction does not invalidate the use of the model for reactor risk assessments. Current and past siting practices by the U.S. Nuclear Regulatory Commission have precluded reactors being sited within 20 miles of a major metropolitan area. A review of the 68 sites at which the first 100 commercial LWRs are located (Table VI 10-1) shows that the largest city within 25 miles of a reactor site is Cincinnati, Ohio, with a population of 427,000. New York City, Boston, Philadelphia, Chicago, and Los Angeles are all beyond 25 miles from a commercial power reactor. For the accident scenarios evaluated in this report, there is no presumption that the population in any of these major cities could be moved in less than 1 week.

11.1.2 VENTILATION

One potential benefit from remaining indoors during the passage of the radioactive cloud would be reduction in the quantity of radionuclides inhaled. The important parameter in this respect is the ventilation or turnover rate of the air within the building, which is a function of meterological conditions and the construction of the building.

The ventilation rate is affected by the inside-outside temperature differential, wind speed and direction, quality of construction, and topographical setting. Building ventilation is measured by the fraction of building volume turned over per hour. A survey of the literature of home ventilation rates found this to vary from 0.07 to 3.0 per hour (Handley and Barton, 1973). Although one would expect a considerable variation of this parameter from one region of the country to another, none was indicated by this survey. This invariance is probably a reflection of the rather limited data and the use of standard construction materials and practices. With the building at ambient temperature, the ventilation rate should approach zero as the wind speed approaches zero. Megaw (1962) found that, for a wooden hut with tight-fitting windows and snug doors, there is a linear relationship between the mean wind speed, $\bar{u}$, and the ventilation rate; that is, for speeds of up to 6 m/sec, $\eta = 0.9\bar{u}$, where $\bar{u}$ is in meters per second and η is in reciprocal units of 1 hour. For a cloud of constant air concentration, which would give a dosage external to the building of $\bar{\Psi}_o$, in a time Δt, the dosage inside a shelter, $\bar{\Psi}_i$, is given by (Slade, 1968)

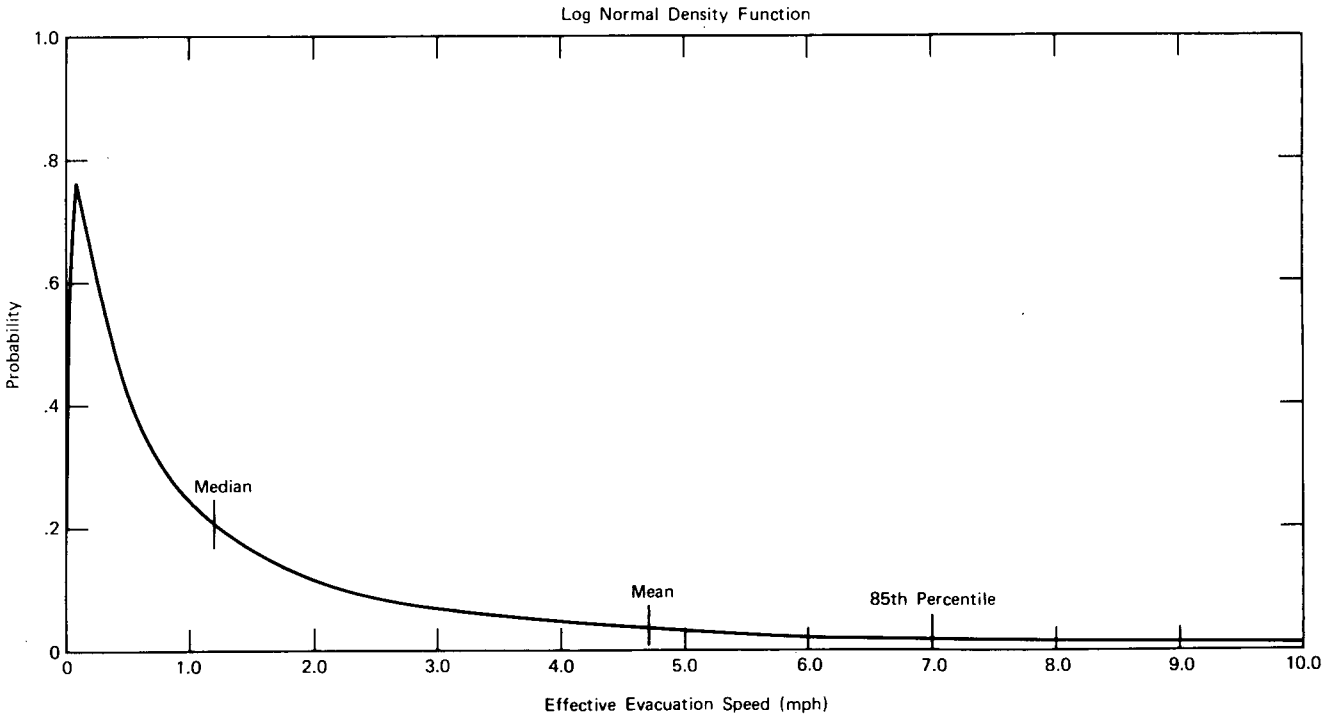

FIGURE VI 11-3 Log-normal distribution of evacuation speeds.

$$\bar{\Psi}_i = \bar{\Psi}_0 \left[1 - \frac{1}{\eta \Delta t}(1 - e^{-\eta \Delta t})\right]. \qquad \text{(VI 11-1)}$$

The time of exposure, Δt, would depend on the particular accident and would normally range between 0.5 to 5 hours. The ratio of the dosage inside a shelter to that outside a shelter can be calculated as a function of ventilation rate. The results are shown in Fig. VI 11-4.

Clearly, short transit times and residency within a well-sealed building could considerably reduce the quantity of radionuclides inhaled. The above analysis assumes a constant outdoor concentration during the time of cloud passage. Actually, the outdoor concentrations would be represented by curve A of Fig. VI 11-5. Because of the restricted turnover of the air within a shelter after passage of the radioactive cloud, the indoor concentration of radioactive material during and after cloud passage would follow curve B. The total inhaled radioactive material for people inside would be the integral under curve B, which may be smaller or greater than the integral under curve A for people outside. If a person were instructed to open his windows at time T (Fig. VI 11-5) to clear the contaminated air, he would minimize his inhalation of radionuclides and sheltering would have been beneficial in this regard.

Protection against inhaled radioactivity can also be enhanced by breathing filtered air Unfortunately, the general public will not have ready access to suitable respirators or gas masks. Guyton, Decker, and Auton (1959) have shown that eight layers of a man's cotton hankerchief or two layers of a bath towel have removal efficiencies of 89 and 85%, respectively, for Bacillus globigii spores with a mass mean aerodynamic diameter of 2.1 microns. However, infants cannot tolerate such a filter over the nose and mouth.

The study concluded that, averaged over a large population, little reduction in inhaled radionuclides would be expected for the following reasons:

a. Since a reactor accident is expected to be a once-in-a-lifetime experience, the public would be unprepared to take sophisticated protective measures.

b. In many geographical locations and for several months of the year, people live and sleep with the windows open, and no reduction in inhaled dose is possible without positive action.

c. It would be difficult for authorities to persuade the public to close windows and, once they had done so, even more difficult to persuade them to reopen them at the right time.

Accordingly, no reduction in inhaled radionuclides is included in the calculation of consequences.

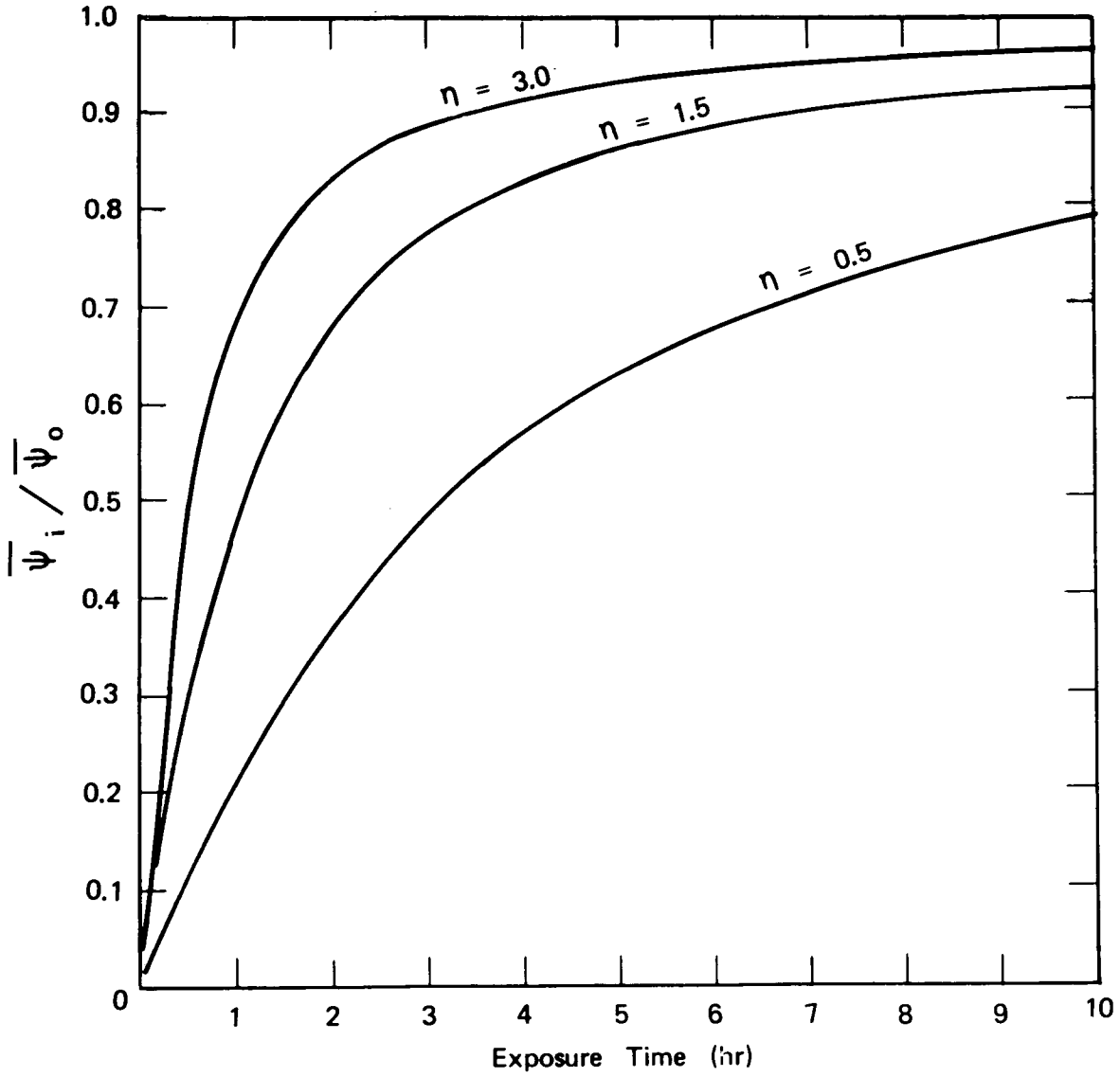

FIGURE VI 11-4 Ratio of the inhaled dose inside a shelter to that outside the shelter as a function of ventilation rate η.

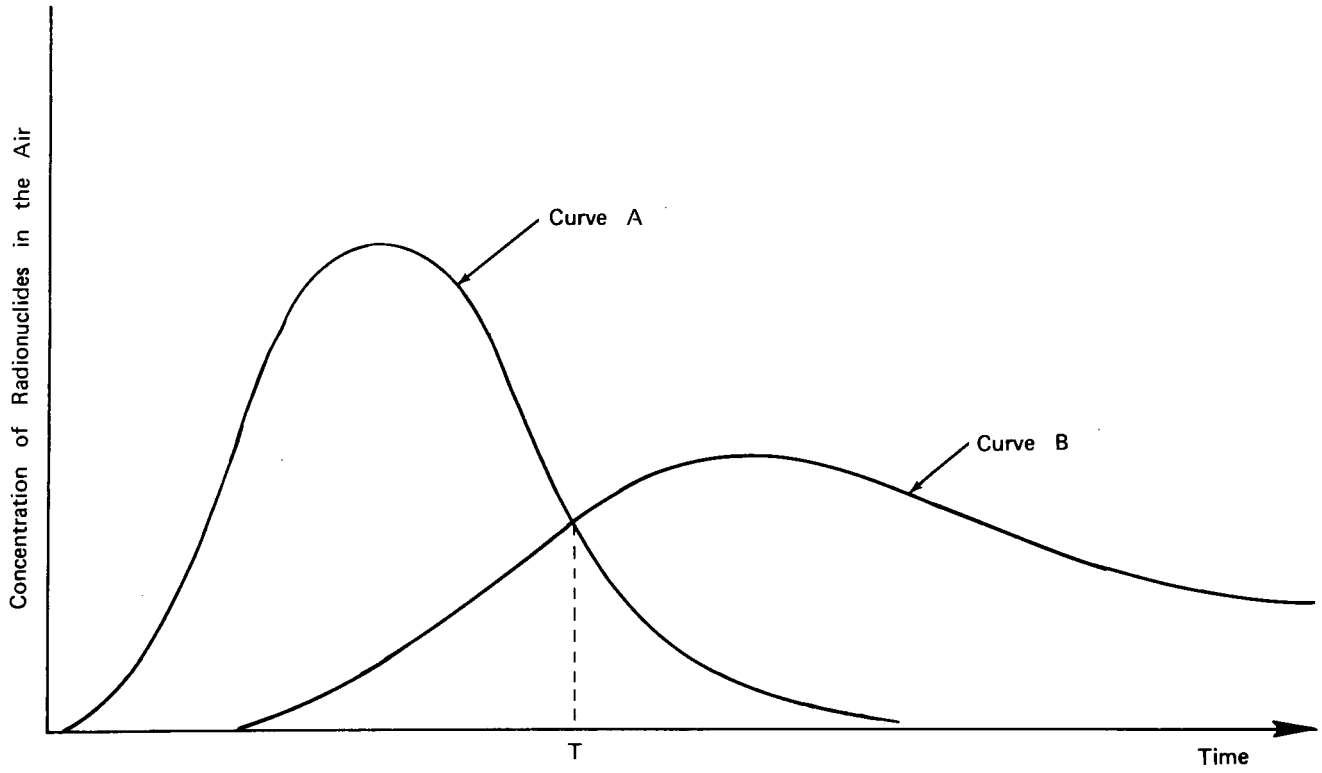

FIGURE VI 11-5 Concentration of radioactive material outdoors (curve A) and indoors (curve B) as a function of time during the cloud passage.

11.2 ACTIONS TO REDUCE LONG-TERM EXPOSURE FROM DEPOSITED RADIOACTIVITY

11.2.1 RADIATION DOSE CRITERIA

11.2.1.1 Introduction

It must be realized that the Reactor Safety Study is not recommending acceptable exposure criteria for the public or acceptable contamination levels in food. Such recommendations should be based on benefit/risk evaluations, which are the province of other organizations. In order to assess the potential consequences from a hypothetical reactor accident, the study has calculated consequences for a range of possible criteria and, for the nominal statement of consequences, used values consistent with those recommended by the Federal Radiation Council and the Medical Research Council of Great Britain.

Recommended limits on the radiation doses received by members of the public have been published by the International Commission on Radiological Protection (ICRP), the National Council on Radiation Protection and Measurement (NCRP), and the Federal Radiation Council (FRC). The NRC dose limits for licenses given in Part 20, Title 10, of the Code of Federal Regulations are derived from those of the FRC. The published criteria differ somewhat in detail, but many of the concepts are common to all. The ICRP (1966) recommendations are given in Table VI 11-3.

TABLE VI 11-3 ICRP (1966) ANNUAL DOSE LIMITS FOR MEMBERS OF THE PUBLIC

Organ or Tissue	Dose Limit (rem/yr)
Gonads and red bone marrow (and, in the case of uniform irradiation, the whole body)	0.5
Skin, bone, thyroid	3.0[(a)]
Hands and forearms; feet and ankles	7.5
Other single organs	1.5

[(a)] 1.5 rem/yr to the thyroid of children to 16 years of age.

With respect to the exposure of the whole population, ICRP has principally considered genetic effects. For planning purposes, ICRP recommends that over the first 30 years of life (the mean age of paternity) the average genetically significant dose should not exceed 5 rem from man-made sources other than medical, with the dose delivered at a fairly uniform rate. The ICRP notes that this dose should not be used up by a single type of exposure. No firm recommendations on the apportionment of the genetic dose are made, but having regard to occupational exposure and the desirability of maintaining a reserve against unforeseen contingencies, the ICRP recommends that the average genetic exposure of the population at large should be limited to 2 rem per individual in 30 years. To this end, it is recommended that genetic exposure from internal sources should on the average be kept to below 0.05 rem/yr.

With respect to somatic doses, the ICRP proposes no definite limits of tissue dose. Adherence to the ICRP's recommendations for the protection of the individual members of the population should keep the exposure of the population as a whole within acceptable limits. As a guide to industrial planning, the ICRP suggests that the average intake of radionuclides throughout the population should be kept to one-third of the limit set for individuals.

A clear distinction is drawn by the ICRP between controllable exposure, "in which the occurrence of the exposure is foreseen and can be limited in amount by control of the source and by the development of proper operating procedures," and uncontrolled exposure, "in which the particular exposure is accidental and which can be limited in amount only, if at all, by remedial actions." The basic standards for controllable exposure take the form of annual dose limits for body organs or tissues (see Table VI 11-3). Exposure limits for uncontrolled exposure are discussed in section 11.2.1.2.

The standards (maximum permissible concentration of radionuclides in air or water) for acceptable exposure to ingested or inhaled radionuclides are based on the assumption that the radionuclides in the body or in the critical organ should not deliver more than the annual dose limit. These standards are derived by using a set of physiological parameters that describe the movement of each element in and out of the critical organ, the mass of the organ, and the rate at which the radionuclides are inhaled or ingested. The ICRP and the NCRP have prepared tabulations of such maximum permissible concentrations of radionuclides in water or air, which if ingested or inhaled continuously would, in a lifetime exposure of 50 years, result in a body burden delivering the maximum dose limit to one or more organs of the body. The physiological parameters and the critical organ masses are based on a "standard man," as defined by the ICRP. Obviously, there are many reasons why these may not be valid for children, infants, fetuses, or members of the population who have certain diseases. In addition, the use of maximum permissible concentrations of radionuclides in water or air does not consider indirect exposure pathways to man (e.g., the buildup or reconcentration of radionuclides in certain parts of man's food chain).

11.2.1.2 Recommendations for Exposure Limits to Accidental Releases

For a widespread contamination resulting from unplanned occurrences involving uncontrolled sources, such as a nuclear reactor accident, the possibility of limiting radiation exposure will depend to a great extent on actions taken after the event. The view of the ICRP is that a decision to institute actions for the mitigation of exposure must take into account the particular prevailing circumstances and, in general, the actions should be undertaken only when the social cost and risk will be smaller than that resulting from the exposure. For all practical purposes, this is essentially the same position as that taken by the Federal Radiation Council, as explicitly stated in its reports (FRC, 1964, 1965).

The Federal Radiation Council has concerned itself with setting guidelines for actions relating to the accidental contamination of crops or other dietary components. In establishing the guidelines, it made the basic assumption that a condition requiring protective action is unusual and should not be expected to occur frequently--in fact, to be so infrequent that it is unlikely that the same individual will be exposed to more than one event. It has defined a term, "protective action guide" (PAG), as the projected absorbed dose to individuals in the general population that warrants protective action after a contaminating event. The projected dose is the dose that individuals would receive from the contaminating event if no protective actions were taken.

The PAGs are defined for three separate categories. Categories I and II relate to intake in the first year after early deposition, and category III considers intake after the first year. These categories cover explicitly the following areas:

a. Category I is concerned with the immediate transmission of radionuclides through the pasture-cow-milk-man pathway.

b. Category II is concerned with the transmission of radionuclides to man through dietary pathways other than that specified as category I during the first year after an acute contaminating event.

c. Category III is primarily concerned with the long-term transmission of strontium-90 through the soil into plants in the years following a contaminating event.

The FRC position regarding the application of the PAGs is as follows:

> "In considering the desirability of initiating protective actions following a contaminating event, it is necessary to consider the three categories separately. The benefits of a protective action taken in one category are largely independent of whether action is taken in another. Individuals may be exposed to radioactivity from all three categories; however, the guides for individual categories recommended are sufficiently conservative (i.e., low) that it is unnecessary to provide an additional limitation on combined doses."

The explicit FRC recommendations for protective action in each of the three categories are as follows:

Category I

The guidance applicable to strontium and cesium is given in terms of the projected dose to the whole body or bone marrow. The PAG is a mean dose of 10 rads in the first year to the bone marrow or whole body of individuals in the general population and a total dose not exceeding 15 rads. For the purpose of applying this guide, the total dose from strontium-89 and cesium is assumed to be the same as the dose in the first year, whereas the total dose from strontium-90 is assumed to be five times the dose from strontium-90 in the first year. As an operational technique, it is assumed that the guide will be met effectively if the average projected dose to a suitable sample of the population (children approximately 1 year of age) does not exceed one-third of the numerical value prescribed for the individual.

For iodine-131, a projected dose of 30 rads to the thyroid of individuals in the general population has been recommended as the PAG. As an operational technique, it is assumed that this condition will be met effectively if the average projected dose to a suitable sample of the population (children) does not exceed 10 rads.

Category II

The PAG for the transmission of strontium and cesium through food crops or animal feed crops is 5 rads in the first year to the bone marrow or whole body of the individual in the general population. As an operational technique, it is assumed that the guide will be met effectively if the average projected dose to a suitable sample of the population is no larger than 2 rads in the first year to the whole body or bone marrow.

Category III

If it appears that the annual doses to the bone marrow after the first year may exceed 0.5 rad to individuals or 0.2 rad to a suitable sample of the population, such situations shall be appropriately evaluated.

These recommended guidelines are summarized in Table VI 11-4. The Bureau of Radiological Health of the Food and Drug Administration (Anderson, 1974) has proposed that these PAGs be utilized in the event of a major contaminating event.

The Medical Research Council of Great Britain has also derived proposed guidelines for decisions following a major contaminating event. Their guidelines are similar to the FRC's protective action guides. The Medical Research Council expresses its guides as emergency reference levels (ERL) and defines them as a value, either of dose or an environmental measurement, that divides situations in which countermeasures are unlikely to be justified unless they have a very small impact on the community from those in which countermeasures are desirable if they can be carried out safely and effectively (Medical Research Council, 1975). The recommended ERLs are given in Table VI 11-5.

TABLE VI 11-4 PROTECTIVE ACTION GUIDES OF FRC

Category	Dose to Individual	Dose to Segment of Population	
Category I (milk):			
Strontium	10	3.3	rads to bone marrow in first year
Cesium	10	3.3	rads to whole body in first year
Iodine	30	10	rads to thyroid in first year
Category II (other ingestion routes):			
Strontium	5	2	rads to bone marrow in first year
Cesium	5	2	rads to whole body
Category III:			
Strontium	0.5	0.2	rad/yr to bone marrow

TABLE VI 11-5 EMERGENCY REFERENCE LEVELS RECOMMENDED BY THE MEDICAL RESEARCH COUNCIL OF GREAT BRITAIN

Tissue	ERL (rem)
Whole body	10
Thyroid	30
Lung	30
Bone:	
Endosteal cells	30
Marrow	10
Gonads	10
Superficial tissues irradiated by beta particles	60
Any other organ or tissue	30

An ERL of dose is to be regarded as a dose commitment that is defined as the total radiation dose received by a tissue from external and internal sources as a result of an accident, regardless of the period over which the dose is accumulated.

In the Reactor Safety Study, the 10 rem in 30 years criterion was used in cases where the population density was low. However, in cases where an urban area is involved, a somewhat higher criterion of 25 rem in 30 years was used. This differentiation is made since the problems of relocating people in urban areas involve expenses and risks that seem unjustified for the relatively small reduction in total dose. Such a position is consistent with the FRC guidelines. The FRC, on page 28, states, "if only high impact action would be effective, initiation of such action may be justifiable only at projected doses higher than the PAG." This policy is also consistent with that of the British MRC, which states that "if doses are only moderately in excess of the ERL's the countermeasures should be such that they do not involve appreciable risk to the community. Countermeasures involving greater hazard should be applied only if radiation exposures would otherwise be considerable."

The dose criteria used by the Reactor Safety Study, shown in Table VI 11-6, were adapted from the recommendations of the FRC and MRC.

TABLE VI 11-6 DOSE CRITERIA USED BY REACTOR SAFETY STUDY FOR NOMINAL STATEMENT OF CONSEQUENCES

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

11.2.2 INTERDICTION AND DECONTAMINATION

11.2.2.1 Introduction

After widespread contamination of an area, the simplest means available for mitigating long-term radiation exposure to the population would be the interdiction of the contaminated land. If the land contains improvements and is important economically, the costs of interdiction could be quite high. On the other hand, the interdiction of limited-use land (e.g., marshes) would involve small costs. However, since the land received limited use in the first place, its interdiction could not greatly mitigate any radiation exposure to the population. Generally, the interdiction of land for the purpose of avoiding radiation exposure to the population is simple to carry out but may be economically expensive.

The alternative to interdiction is decontamination. Land can be decontaminated either by burying the radioactive material in place (plowing) or by physically removing the material. The costs and effectiveness of decontamination depend strongly on the characteristics of the contaminant material and the properties of the contaminated surface. In general, however, it is less expensive to decontaminate than to interdict land over long periods.

This section discusses in greater detail interdiction and decontamination as means of mitigating long-term radiation exposure to the population from contaminated land.

11.2.2.2 Interdiction

The process of interdiction would involve the denial of land and its improvements for normal intended use. For example, if the land were contaminated to such an extent that a specified radiation dose would be exceeded over a period of time, use of the land could be prohibited until such time as the radiation dose that an individual would receive over the succeeding period of time has decreased (due to radioactive decay and weathering forces) below the specified criterion. In a decreasing order of impact, interdiction could fall into any of the following categories:

a. Total land and asset interdiction for long periods (more than 10 years)

b. Limited land interdiction (restrictions imposed for a few years)

c. Crops

d. Milk

The criteria for establishing any of these categories of interdiction are based on projected doses to the population, as stated in Table VI 11-6. The first two categories are based on external radiation doses to people residing or working on the land. The last two, crop and milk interdiction, are based on radiation doses resulting from the ingestion of contaminated foodstuffs.

Crop and milk interdiction would be necessitated by the external contamination of vegetation. It would, therefore, be only a transitory problem affecting a maximum of 1 year's vegetation. The crops and milk from potentially contaminated areas would be carefully controlled and, if they exceeded specified contamination limits, would be destroyed. Therefore, if the accident were to occur during the growing season, it would be possible to lose (1) a year's crops and (2) the use of milk for periods of up to several weeks if the milk comes from cows grazing on pastures. If it is unnecessary to interdict the land because of external radiation doses to people, it may still be necessary to impound crops and milk from the second and subsequent growing seasons. This conclusion is based on the mixture of radionuclides that could possibly be released in a large accident, the radiation dose criteria discussed in section 11.2.1, and the fact that the uptake of radionuclides by plant roots is not an efficient means of transferring radioactive material to man.

In order to facilitate an understanding of the concept of interdiction, a simplified interdiction model is sketched in Figs. VI 11-6 and VI 11-7 for a ground-level release and an elevated release of radioactive material, respectively. For a ground-level release, the degree of ground and vegetation contamination would decrease monotonically with distance from the reactor.[1] For self-consistent health criteria, the most restrictive contamination criterion would be on milk, and hence the largest interdicted area would be associated with milk impoundment. The level of ground contamination above which milk must be impounded is shown in L_1 in Fig. VI 11-6 and involves the land area covered by the plume traveling from the reactor out to point R_1. A lower contamination criterion applies to directly contaminated foliage, and hence a smaller crop-growing area would be interdicted. The acceptable ground contamination for crops is shown in Fig. VI 11-6 as L_2 and it requires the impoundment of crops grown in an area extending from the reactor out to a distance R_2. The least restrictive criterion would be applied to the continuing occupancy by people, the critical exposure mode being direct external radiation from contaminated ground. This criterion is illustrated in Fig. VI 11-6 by the level L_3 and involves the area between the reactor and the radial point R_3.

[1] Contamination levels may not decrease monotonically with distance when wind speeds and rain occurrence are time-dependent.

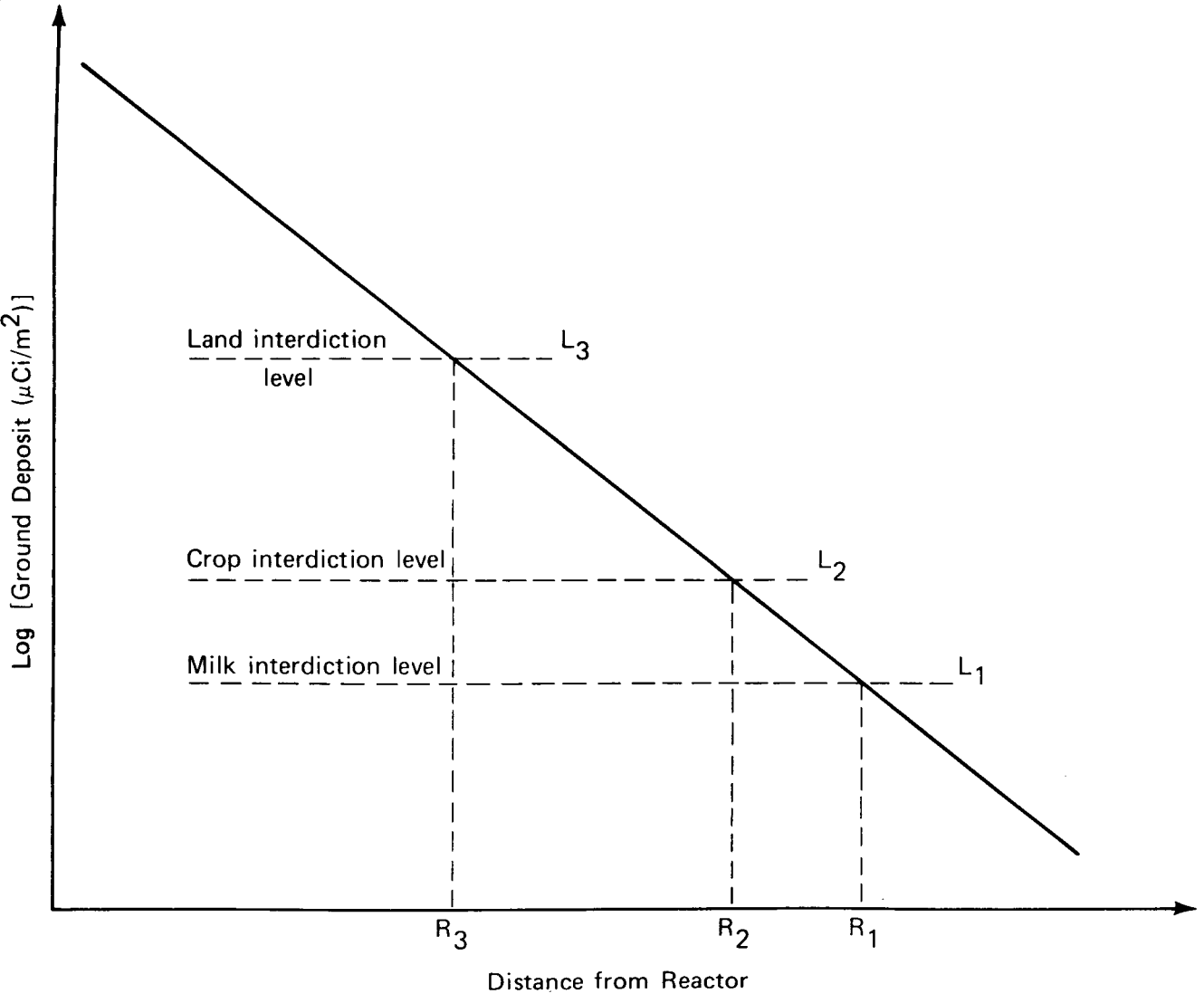

FIGURE VI 11-6 Illustrative interdiction model for ground level release.

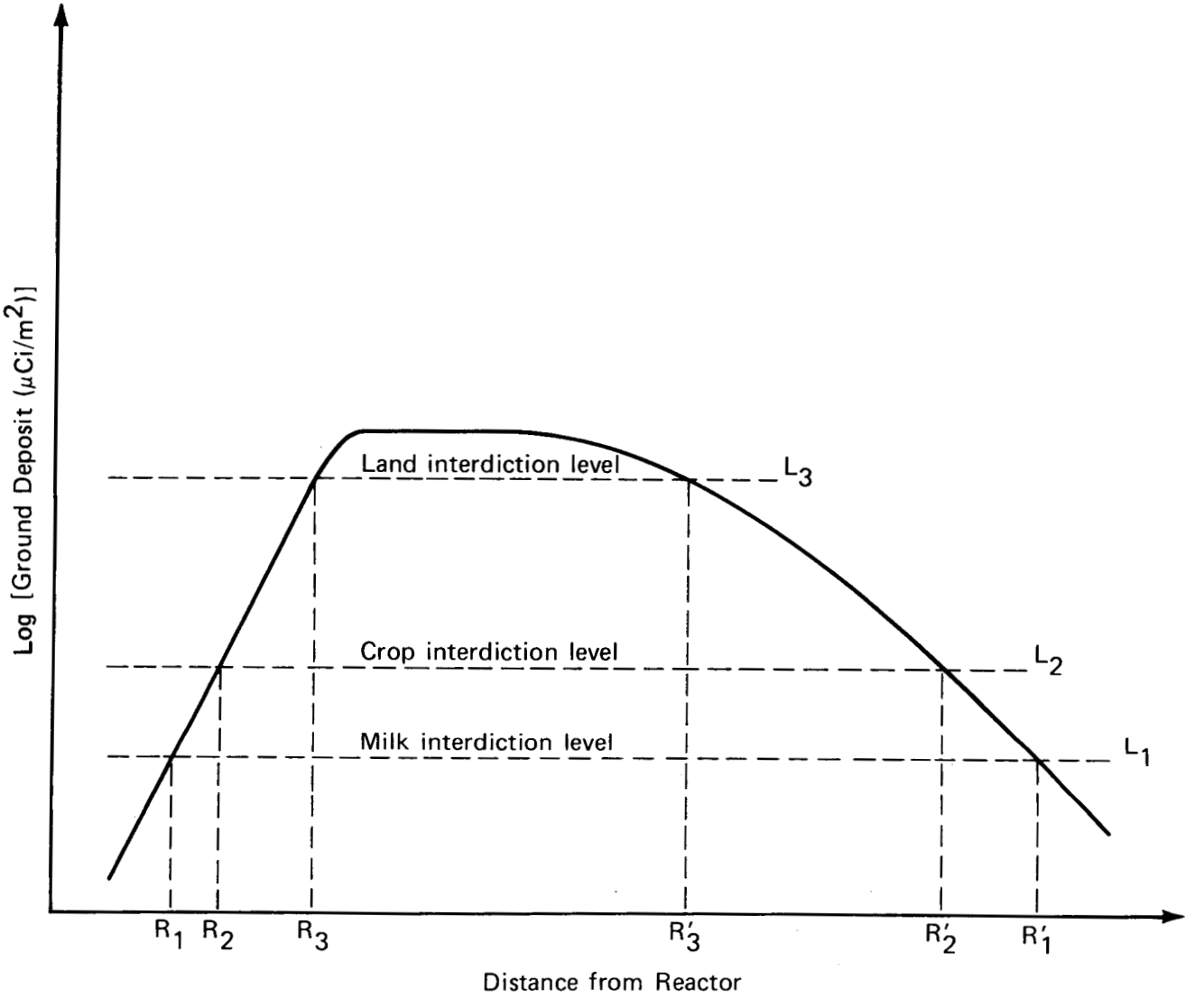

FIGURE VI 11-7 Illustrative interdiction model for elevated release.

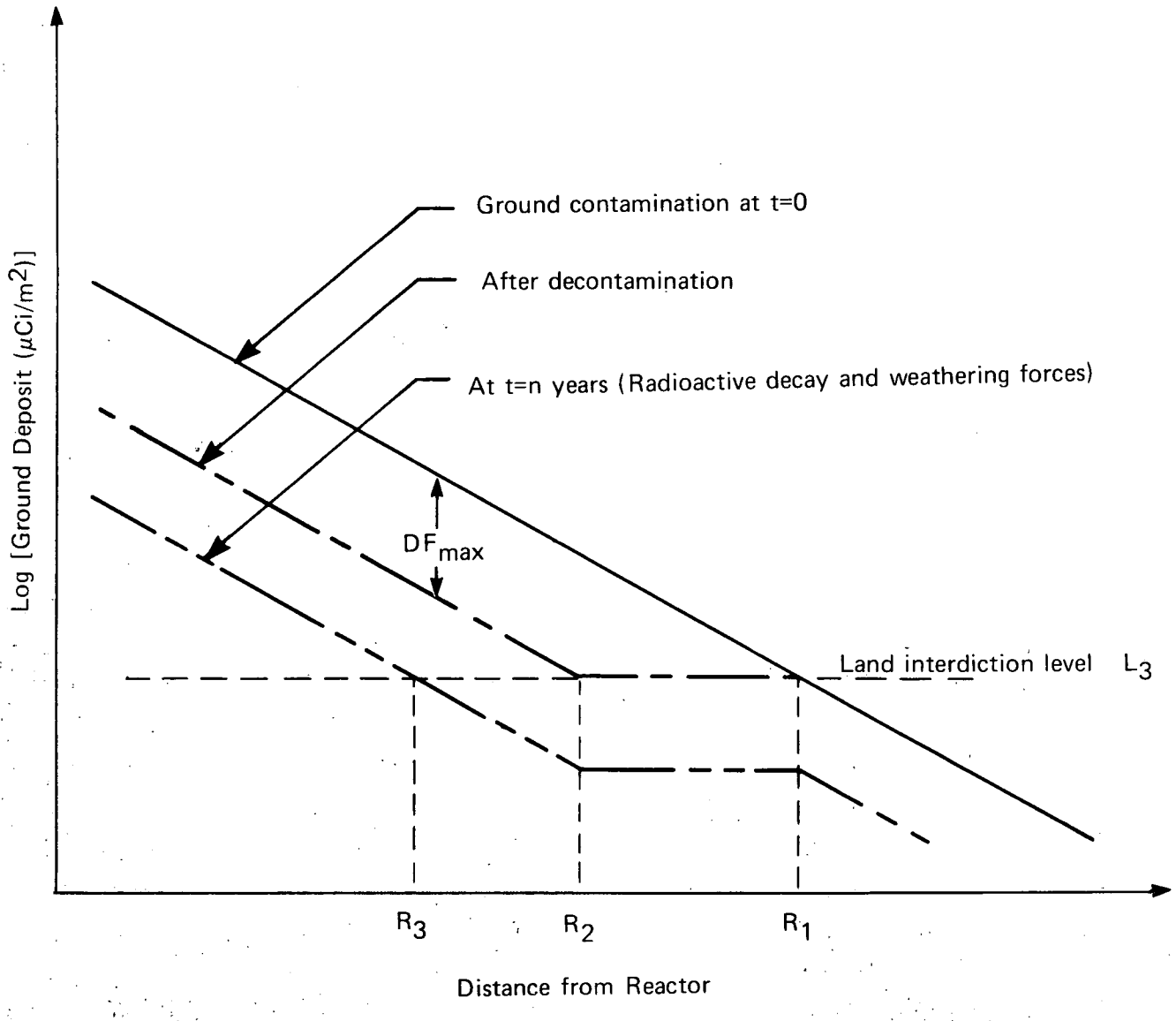

FIGURE VI 11-8 Illustrative decontamination model for ground level release.

Because of radioactive decay and weathering forces, the level of ground contamination will decrease with time. Therefore, the point R_3 associated with criterion L_3 in Fig. VI 11-6 would move toward the reactor with time. If R_3 has moved to R_3' in a matter of a few years, then the area between R_3 and R_3' will be interdicted only for those years. This is referred to as limited land interdiction.

For an elevated release of radioactive material (see Fig. VI 11-7), the degree of ground contamination would increase initially as the plume diffuses toward the ground. A maximum level of ground contamination would be achieved at some distance from the reactor and thereafter would decrease monotonically. Conceptually the areas of interdictions are the same as those explained in Fig. VI 11-6 for ground-level release.

As explained above, the area of interdicted land would decrease with time as the level of contamination decreases due to radioactive decay and weathering forces. However, decontamination would make it possible to recover some of this land immediately. Decontamination is discussed in the following section.

11.2.2.3 Decontamination

Decontamination, in the broad sense of the word, is the cleanup and removal of radionuclides. The possible decontamination modes include physical removal of the radionuclides, stabilization of the radionuclides in place, and environment management. The particular procedure utilized in a given case would depend on many factors, including (1) the type of surface contaminated, (2) the external environment to which the surface is exposed, (3) the possible hazards to man, (4) the costs involved, (5) the degree of decontamination required, and (6) the consequences of the decontamination operation.

There is a large body of experimental data on the decontamination of structures, pavements, and land. These data were generated, for the most part, for the planning of reclamation in the event of a nuclear war. Because of differences in the contaminant particle size and decontamination criteria, some of these experimental data are not directly applicable to the particular case considered here. These problems are discussed more fully in Appendix K, and only a summary is provided in this section.

A measure of effectiveness of decontamination operations is the decontamination factor DF, which is defined as the contaminant density (in microcuries per square meter) before decontamination divided by the contaminant density after decontamination. Therefore, the larger the DF, the better the decontamination method. For example, a 90% removal of contaminants from a surface gives a DF of 10 and a 99% removal gives a DF of 100.

As discussed in Appendix K, present experimental evidence is not adequate to support any assumptions on the effectiveness of wet decontamination (i.e., firehosing) for the small aerosol particles released during the reactor accident. Therefore, the removal of contaminated surfaces is the only decontamination procedure postulated by the study for hard surfaces. The various procedures for surface removal are the following:

a. Hard surfaces (roofs, walls, pavements, etc.)
   - Replacement of roofing material
   - Sandblasting of walls and pavements
   - Resurfacing of pavements

b. Land areas (soil, vegetation, etc.)
   - Vegetation removal and disposal
   - Surface soil removal and burial
   - Deep plowing

The maximum decontamination factor that is considered practical, averaged over large areas, is 20. This limitation is based on the practicality of large-scale decontamination operations, the costs involved, and the consequences of decontamination operations.

The decontamination model utilized in the consequence model is conceptually illustrated in Fig. VI 11-8 for a ground-level release of radioactive material. The acceptable level of ground contamination for occupancy by people is shown in the figure by the level L_3. The land area between the reactor and the point R_1 would have to be interdicted or decontaminated. If the maximum decontamination factor attainable over large areas were DF_{max}, then the land area between R_1 and R_2 would be recoverable by decontamination. The consequence model assumes that the actual decontamination factor attained at any given point is only sufficient to bring the ground contamination level down to the acceptable level of L_3.

In conjunction with decontamination, the consequence model also assumes limited land interdiction. In this case the maximum decontamination factor DF_{max} is assumed to be attained for the land area to the right of the point where L_3 times DF_{max} is exceeded. Radioactive decay and weathering forces will bring the ground contamination level down the additional amount required to attain the acceptable level L_3. As shown in Fig. VI 11-8, the land between R_2 and R_3 would be recovered in this manner in n years.

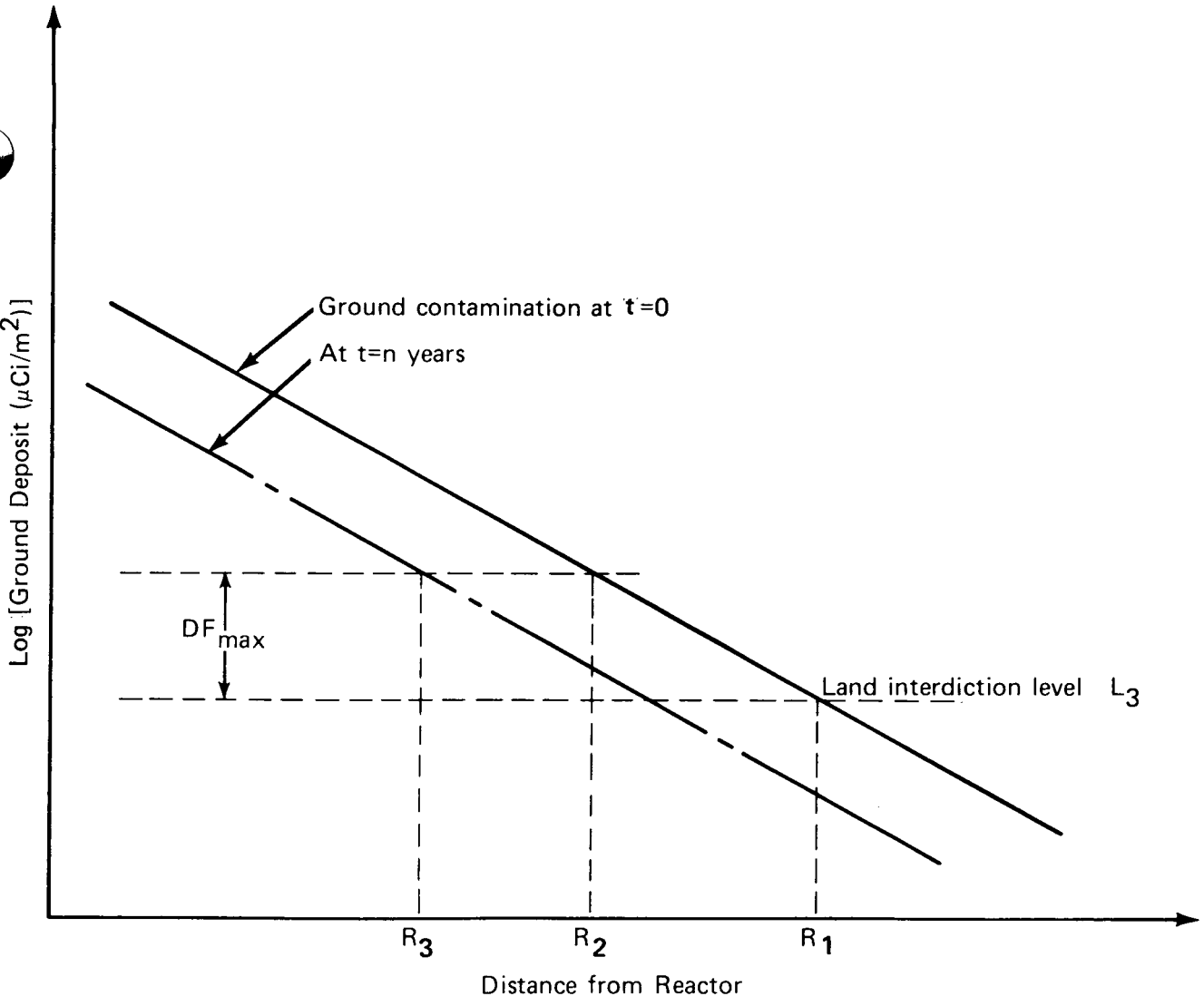

FIGURE VI 11-8 Illustrative decontamination model for ground level release.

11.3 SHIELDING

11.3.1 SHIELDING FROM AIRBORNE RADIOACTIVE MATERIAL

As discussed in section 8.2.2, people caught within or under the moving cloud of radioactive material would receive an external dose to the whole body from gamma radiation.[1] Since the walls of a building will absorb and scatter gamma rays, anyone inside a building would receive an attenuated (i.e., lower) dose. The shielding effectiveness of a structure is measured by its shielding factor (SF), which is the ratio of the interior dose to the exterior dose.[2]

Dose attenuation depends on two factors: distance and attenuation by passage of radiation through material. The dose from a point source is inversely proportional to the square of the distance. For this reason, the dose in the center of a large building is lower than that near an exterior wall. Thus, with the same walls, a large building can provide greater shielding than a small one. The attenuation of gamma radiation through material depends on the properties of the material (e.g., number of protons per atom) and on the energy of the gamma rays. Linear attenuation coefficients have been established for most common materials and for various gamma-ray energies. The shielding factor for a structure can be readily estimated from the spectrum of gamma energy, the linear attenuation coefficient of the wall material, and the geometry of the structure.

Using currently available shielding technology, Burson and Profio (1975) have made estimates of structure shielding. They have shown that the gamma energy spectrum in the cloud from a reactor accident would be comparable to that measured in nuclear weapons tests. By using the general approach set forth by Slade (1968) and by assuming a semiinfinite cloud surrounding the structure, they have estimated the shielding factors for simple and complex structures. Their results are summarized in Table VI 11-7.

TABLE VI 11-7 REPRESENTATIVE SHIELDING FACTORS FROM GAMMA CLOUD SOURCE

Structure or Location	Shielding Factor[(a)]	Representative Range
Outside	1.0	--
Vehicles	1.0	--
Wood-frame house[(b)] (no basement)	0.9	--
Basement of wood house	0.6	0.1 to 0.7[(c)]
Masonry house (no basement)	0.6	0.4 to 0.7[(c)]
Basement of masonry house	0.4	0.1 to 0.5[(c)]
Large office or industrial building	0.2	0.1 to 0.3[(c,d)]

(a) The ratio of the interior dose to the exterior dose
(b) A wood frame house with brick or stone veneer is approximately equivalent to a masonry house for shielding purposes.
(c) This range is mainly due to different wall materials and different geometries.
(d) The reduction factor depends on where the personnel are located within the building (e.g., the basement or an inside room).

[1] In this section, consideration is limited to gamma radiation since beta and alpha particles cannot penetrate the walls of structures.

[2] The shielding factor is usually referred to in the literature as the reduction factor.

11.3.2 SHIELDING FROM SURFACE-DEPOSITED RADIOACTIVE MATERIAL

The dose conversion factors given in Appendix C relate the tissue dose (in rem per hour) at 1 meter above ground to contamination (in microcuries per square meter) spread uniformly in a thin layer over an infinite smooth surface. The height of 1 meter is used because it is approximately the distance to the vital organs of a standing person. The hypothetical contaminated surface is a reference point for shielding calculations and experiments. The shielding factors (SF) stated in this section modify the aforementioned dose-conversion factors.

Obviously, the hypothetical infinite smooth plane does not exist in nature. The contaminant particle sizes are small enough to allow the contaminant to distribute itself over the real surface of the terrain. The irregularities in the surface are referred to as ground roughness and have long been recognized as a mechanism of natural shielding from a fallout source (Ksanda et al., 1956; Huddleston et al., 1965). Therefore, even for a person standing in an open, relatively flat field, the shielding factor is on the order of 0.7. In an urban environment, the presence of nearby buildings results in mutual self-shielding and may give a shielding factor of 0.4 to 0.6 (Defense Civil Preparedness Agency, 1973).

The protective shielding afforded by single- and two-story houses from external penetrating radiation will primarily be a function of the mass of material in the wall and roof. The size and shape have only a relatively small influence on the overall shielding factor. Because of the long mean free path of high-energy (>0.2 MeV) gamma radiation in air, a large contribution to the dose within a structure will come from radioactivity deposited on the surrounding ground. However, the deposited activity on the roof and walls of the structure can also give substantial exposure. For one- and two-story single-family dwellings with a uniform contamination of the roof and surrounding ground, and one-fifth as much contamination per surface area on the walls as on the roof, the shielding factors range from 0.04 to 0.5.

Burson and Profio (1975) have shown, by using the point-kernel integration method (including buildup from scattering), that the extensive fallout shielding technology developed from (1) calculations for radionuclides with 1.12-hour half-lives and (2) experiments with cobalt-60 can be directly applied to the case of radioactivity deposited after a reactor accident. A summary of the shielding factors suggested by Burson and Profio (1975) for gamma radiation from uniformly deposited radionuclides from a reactor accident is given in Table VI 11-8. For use in the consequence model, these results are summarized in Table VI 11-9.

Numerous shielding experiments have been conducted as part of nuclear weapons tests and in laboratory mockups with monoenergetic gamma-ray sources (e.g., cobalt-60 or cesium-137). These experiments have been used to verify calculational techniques (Spencer, 1962) for multienergy gamma spectra and complex structures (Auxier, et al., 1959; Borella, et al., 1961; Burson, et al., 1962; Burson, 1963a,b, 1966, 1970; Burson and Borella, 1962; Spencer, 1962; Strickler and Auxier, 1960).

TABLE VI 11-8 REPRESENTATIVE SHIELDING FACTORS FOR SURFACE DEPOSITION

Structure or Location	Representative Shielding Factor(a)	Representative Range
1 m above an infinite smooth surface	1.00	--
1 m above ordinary ground	0.70	0.47-0.85
1 m above center of 50-ft roadways, half contaminated	0.55	0.4-0.6
Cars on 50-ft road:		
Road fully contaminated	0.5	0.4-0.7
Road 50% decontaminated	0.5	0.4-0.6
Road fully decontaminated	0.25	0.2-0.5
Trains	0.40	0.3-0.5
One- and two-story wood-frame house (no basement)	0.4(b)	0.2-0.5
One- and two-story block and brick house (no basement)	0.2(b)	0.04-0.40
House basement, one or two walls fully exposed:	0.1(b)	0.03-0.15
One story, less than 2 ft of basement, walls exposed	0.05(b)	0.03-0.07
Two stories, less than 2 ft of basement, walls exposed	0.03(b)	0.02-0.05
Three- or four-story structures, 5000 to 10,000^2 ft^2 per floor:		
First and second floors	0.05(b)	0.01-0.08
Basement	0.01(b)	0.001-0.07
Multistory structures, >10,000 ft^2 per floor:		
Upper floors	0.01(b)	0.001-0.02
Basement	0.005(b)	0.001-0.015

(a) The ratio of the interior dose to the exterior dose

(b) Away from doors and windows.

TABLE VI 11-9 SELECTED SHIELDING FACTORS FROM SURFACE CONTAMINATION USED IN THE CONSEQUENCE MODEL

Structure or Location	Representative Shielding Factor[a]	Representative Range
1 m above an infinite smooth surface	1.0	--
1 m above ordinary ground	0.7	0.5-0.8
One- and two-story frame house	0.4	0.2-0.5
One- and two-story block or brick house	0.2	0.04-0.4
Office or large apartment building	0.02	0.001-0.08
Cars on roadways	0.5	0.2-0.7

(a) The ratio of the interior dose to the exterior dose.

11.3.3 OCCUPANCY FREQUENCY FOR BUILDINGS

The preceding sections discussed the shielding provided by different types of buildings and vehicles. In order to assess the shielding of people, these data must be complemented by estimates of the relative occupancies of various buildings.

Several factors will influence the shielding obtained by the public. First, different segments of the population have different lifestyles. For example, housewives, infants, and retired people spend large periods of time in their homes, whereas students and workers commute to school or work, where they spend 6 to 8 hours each weekday. Second, the shielding factors for single-family residences differ from those for large commercial or office buildings. Third, there is a geographic variation in the type of housing across the United States.

Data from the Robinson and Converse time-use study (1966) were used to estimate the fraction of time the population spends in various locations or activities. The Robinson and Converse time-use study sampled the adult population below 65 years of age. Because of this selective sampling, the retired and student populations are not fully represented. However, the time-use study is used because (1) it gives actual measured data and (2) the student population (28% of the total population), though it might be expected to have more outdoor activity than the adult population, should be somewhat balanced by infants and retired persons (about 18% of the population), who should have somewhat less outside activity.

The Robinson and Converse study was intended to establish activities, not the locations of these activities. Consequently, to determine the effect of building shielding, it was necessary to categorize each activity into a location or type of activity. The categories used were (1) home, (2) school or work, (3) commuting, and (4) outdoors. For example, sleeping, reading, and watching television are home activities. The hours per day for each location or activity averaged over a 7-day week are shown in Table VI 11-10.

TABLE VI 11-10 DAILY HOURS AT PRINCIPAL LOCATIONS OR ACTIVITIES, AVERAGED OVER A 7-DAY WEEK

Location or Activity	Hours per day	Fraction of Total Time (%)
Home	16.6	69.2
School or work	4.7	19.6
Commuting	1.2	5.0
Outdoors	1.5	6.2

In order to generate a probability density function for shielding available to the public, the frequencies stated in Table VI 11-10 must be combined with the shielding factors provided by the houses or other buildings occupied by the various population segments. The shielding available from a brick house is significantly greater than that from a wood house. Figure VI 11-9 shows graphically the percentages of brick family units for different parts of the country; the wide variation is conveniently categorized within five regions. Data for this figure were derived from the 1970 Census of Housing (U.S. Department of Commerce) and the 1971 FHA Homes, Data for States and Selected Areas data book published by the Department of Housing and Urban Development (HUD). The HUD book gives statistics by state for existing single-family homes sold under the Federal Housing Administration (FHA) Section 203 program. These data show percentages of those existing (used) houses sold that have brick, stone, or concrete-block exteriors. These percentages have been assumed to be typical of all single-family houses within the state. The data were then adjusted to account for multifamily structures, which were assumed to be of heavy construction (i.e., brick). By using the housing census data on multifamily structures, the percentage of brick or equivalent housing units was estimated as follows:

(% multifamily units) + (% single-family homes)(fraction, brick units)

The frequency distribution for structures in each of five regions is related to the corresponding shielding factors for the passing cloud and ground contamination in Tables VI 11-11 and VI 11-12, respectively.

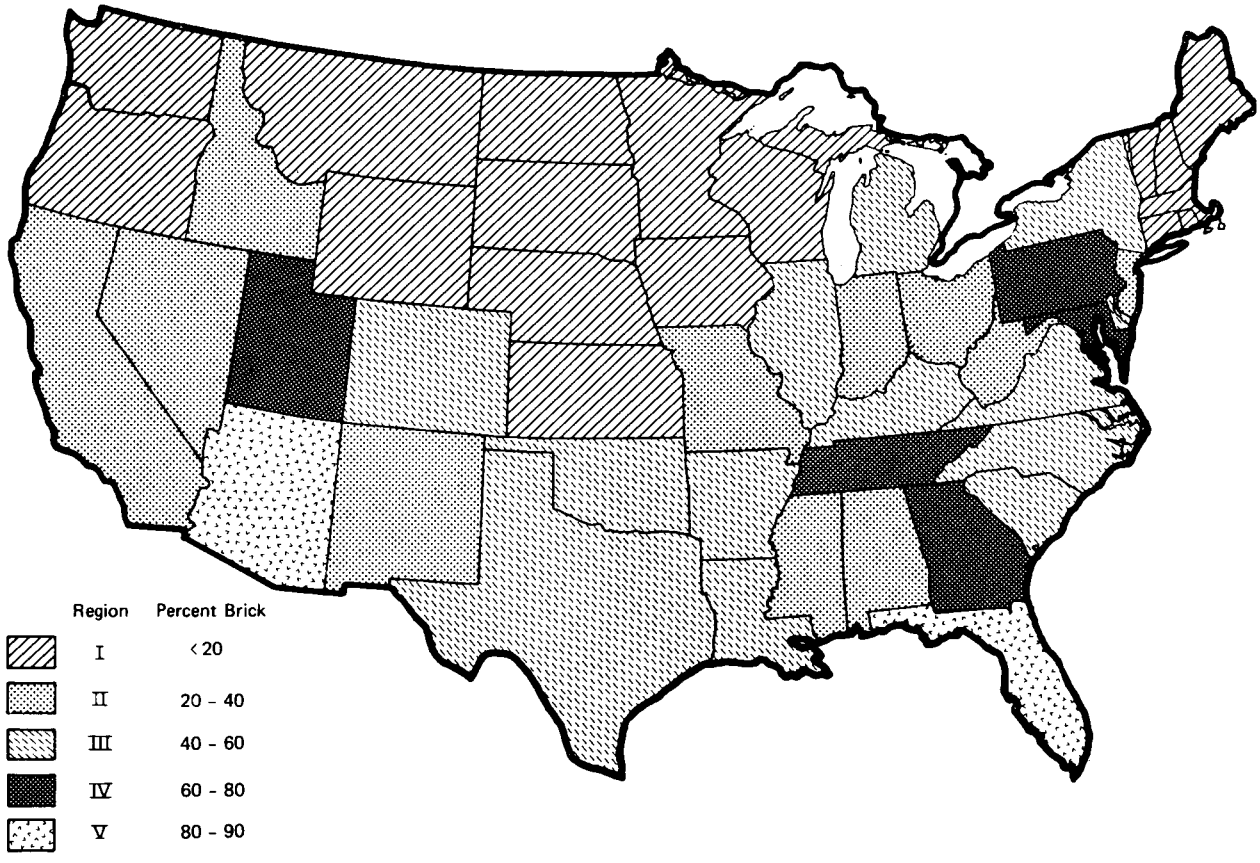

FIGURE VI 11-9 Percentage of brick housing units by region.

TABLE VI 11-11 FREQUENCY DISTRIBUTION FOR SHIELDING FACTORS FROM PASSING CLOUD BY GEOGRAPHICAL REGION

Region[a]	Frequency Distribution (%)						Average SF[b]
	Home		School or Work			Commuting and Outdoors	
	Brick, SF = 0.6	Wood, SF = 0.9	Large Building SF = 0.2	Brick SF = 0.6	Wood, SF = 0.9	SF = 1.0	
I	9.8	59.4	6.5	1.9	11.2	11.2	0.83
II	19.7	49.5	6.5	3.7	9.4	11.2	0.80
III	35.6	33.6	6.5	6.7	6.4	11.2	0.74
IV	44.1	25.1	6.5	8.4	4.7	11.2	0.71
V	57.9	11.3	6.5	11.0	2.1	11.2	0.66

(a) The regions are shown in Figure VI 11-9.
(b) The shielding factor (SF) is the ratio of the interior dose to the exterior dose.

TABLE VI 11-12 FREQUENCY DISTRIBUTION FOR SHIELDING FACTORS FROM GROUND CONTAMINATION, BY GEOGRAPHICAL REGION

Region[a]	Frequency Distribution (%)							Average SF[b]
	Home		School or Work			Commuting, SF = 0.5	Outdoors, SF = 0.7	
	Brick, SF = 0.2	Wood, SF = 0.4	Large Building SF = 0.02	Brick, SF = 0.2	Wood SF = 0.4			
I	9.8	59.4	6.5	1.9	11.2	5.0	6.2	0.38
II	19.7	49.5	6.5	3.7	9.4	5.0	6.2	0.35
III	35.6	33.6	6.5	6.7	6.4	5.0	6.2	0.31
IV	44.1	25.1	6.5	8.4	4.7	5.0	6.2	0.29
V	57.9	11.3	6.5	11.0	2.1	5.0	6.2	0.26

(a) The regions are shown in Fig. VI 11-9.
(b) The shielding factor (SF) is the ratio of the interior dose to the exterior dose.

With respect to schools and workplaces, it was assumed that one-third of the people are in large offices or similar structures, and the remaining buildings have a distribution of construction types similar to that of local single-family dwelling--that is, the same percentage of brick buildings. This assumption is seen to be conservative when it is remembered that government (federal, state and municipal) employs about 30% of the work force, and public buildings are usually substantial structures. In Table VI 11-11 for the passing cloud, no account is taken of the additional shielding available in basements, although over 50% of U.S. homes have a basement. For a sheltering scenario in which it is assumed that the public is advised to take shelter (as opposed to evacuate), it would be reasonable to assume that some percentage (e.g., 30 to 60%) of the population at risk would take advantage of their basements for the few hours of cloud passage. Since this percentage is uncertain and no correlation is available between basements and type of house construction, this additional shielding has been neglected.

11.3.4 SUMMARY

The shielding factors used in the calculations for shielding are summarized in Table VI 11-13. Different shielding factors are used for locations within 25 miles of the reactor and beyond and, of course, for the passing cloud and the contaminated ground.

TABLE VI 11-13 SUMMARY OF SHIELDING FACTORS UTILIZED IN CALCULATIONS

Location	Shielding Factor	
	Passing Cloud	Ground Contamination
$\leq$ 25 miles from reactor	1.0	0.5[(a)]
> 25 miles from reactor	0.75	0.33[(b)]

(a) Ground dose is limited to 4 hours.
(b) If relocation is required, the ground dose is limited to 7 days.
If evacuation is required, the ground dose is limited to 24 hours.

Within 25 miles of the reactor, the doses could be sufficiently large to cause early mortalities or morbidities, so that individual doses must be considered. As stated in Table VI 11-7, an automobile provides essentially no shielding from airborne radioactive material; thus a shielding factor of 1.0 is assumed for evacuation. In addition, evacuees are assumed to spend 4 hours in their automobiles, which have a shielding factor of 0.5 (Table VI 11-8) from ground contamination. As shown in Fig. VI J-5 of Appendix J, the median speed to travel 25 miles is estimated to be 5 mph, which translates into about 5 hours of travel. It should be recognized that until the cloud catches them, the evacuees are travelling over uncontaminated ground. Since the evacuees are assumed to move in a circumferential direction after passage of the cloud, the assumption of 4 hours exposure to ground contamination is probably reasonable. Since a stubborn minority (see section 11.1.1) would be expected to refuse to evacuate (i.e., would remain at home, where there is greater shielding), these shielding assumptions are probably slightly conservative.

ground contamination varies from 0.02 to 0.7, an average value of 0.33 is used since very few people would remain either outside for this time period or remain in basements.

Beyond 25 miles of the reactor, where doses would be usually relatively low, individual doses would become unimportant, and latent somatic and genetic effects would depend on the population dose (man-rem). For this reason, average shielding factors are used to calculate shielding both from the passing cloud and from the contaminated ground. The regional variation is omitted since it is smaller than the overall uncertainties in the problem and would unnecessarily complicate the consequence model. If the ground contamination were sufficiently large to warrant relocation of people, it is assumed that such relocation will be accomplished within an average period of 7 days. If rain were to result in an unusually high ground contamination within a small area, the population within such an area is assumed to be evacuated within an average of 24 hours.

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Section 12

Economic Model

12.1 INTRODUCTION

The adverse health effects that could result from a major reactor accident would originate from the airborne radioactive material and from the material which would be deposited in the environment. The principal action taken to minimize the harmful effects due to the airborne material would be to evacuate the people situated in the path of the radioactive cloud.

Measures to mitigate the effects of radioactive material that had been deposited on the ground could include impoundment of contaminated crops, interdiction of land (prohibition or restriction of its use) and decontamination of land and structures. This chapter describes the costs of these mitigating measures and the economic consequence model for estimating these costs.

The scope of the economic consequence model is defined in section 12.1.1. Section 12.2 contains a conceptual outline of the model. Sections 12.3 and 12.4 describe in detail how the costs are estimated in the model. The final section summarizes the values assigned to important parameters of the model.

It is important to the understanding of the economic effects of a reactor accident to appreciate that these effects are partly determined by the standards used to define the boundaries of the interdiction and contamination zones. Ideally, these standards would be chosen so that the total cost of interdiction (including the "cost" of adverse health effects accompanying the permitted uses) would be minimized. Although this study does not assess the dollar cost of human exposure to radiation, these costs exist nevertheless and will be perceived by the people affected. If an interdiction plan is designed on the basis of excessively tolerant radiation standards, excessive biological costs could be incurred. On the other hand, if the standards are overstringent, the cost of mitigating measures could be excessive.

One of the principal parameters used in the consequence model for estimating the costs associated with a hypothetical release of radioactive material is the population as a function of distance and direction from the reactor For each release analyzed, the consequence model calculates the number of affected people and the extent to which they would be affected. It is assumed that the contaminated area is large enough for population-averaged economic values to be reasonable. Therefore, the input of the consequence model is given as per capita costs for the various economic categories. The total reactor accident costs are calculated as the product of the number of affected people and the various per capita costs.

12.1.1 SCOPE OF THE MODEL

The model is intended to estimate the direct costs of measures to mitigate the effects of a reactor accident. These costs would include the cost of managing a possible evacuation, the cost of temporary accommodation for the evacuees, the value of any goods that might be condemned, the decrease in value of interdicted property, and the cost of decontaminating property.

A distinction should be made between this direct cost and the national cost of mitigating measures. The direct cost is necessarily larger than the national aggregate or "resource" cost because it includes only losses and is not offset by any of the gains that may result. While the nation as a whole would be assumed to obtain no economic gains from the mitigating measures, certain individuals might do so. For example, if a community were dispersed as a mitigating measure, its children would go to schools in other areas. As a result, some unemployed teachers might become employed, offsetting the lost earnings of the children's former teachers over the period during which they relocate and seek new jobs. The relocated teachers' lost earnings would be included in the assessment of direct costs, but an assessment of national cost would reduce this amount by the added earnings of the previously unemployed teachers.

12.2 CONCEPTUAL OUTLINE OF THE ECONOMIC CONSEQUENCE MODEL

The cost of mitigating measures would depend on the specific measures employed and the extent of the areas to which they were applied. The measures employed would depend on the nature of the radioactive contamination, the human exposure associated with normal activity (land use) in the area and the standards for acceptable exposure. The nature of the contamination would depend on the mode of the reactor accident, meteorological conditions during release and passage of the radioactive cloud, and local geography.

The model treats mitigating measures in relation to two separate exposure phases, early and chronic. Measures for mitigating--or actually, for preventing--early exposure are assumed to be initiated on the basis of a forecast of the path of the radioactive cloud. Measures for mitigating chronic exposure would be instituted following a survey to determine the pattern of contamination that had actually occurred.

12.2.1 EARLY EXPOSURE PHASE

The model "forecasts" the early exposure area by reference to an assumed emergency plan. According to the plan, the evacuation area is shaped like a key-hole as shown in Fig. VI 11-2.

It is assumed that after an accident during the local growing season, crops and milk produced from animals feeding on pasture within the contaminated area may be condemned. For an accident during the local dormant season, crops would not be exposed. Since milk is presumed to be produced from uncontaminated feed, it, too, would be uncontaminated.

The cost of acute phase mitigation measures is computed as the sum of the following:

- Evacuation cost
- Value of crops condemned
- Value of milk condemned.

If the reactor accident were less severe than a core melt, evacuation of people would not take place. Depending on the magnitude of the radioactivity release and meteorological conditions, some milk and crops could be condemned.

12.2.2 CHRONIC EXPOSURE PHASE

The consequence model provides a calculation of the area of chronic exposure hazard, as explained in sections 8.3 and 11.2. Where calculated radiation levels are high (relative to an assumed standard) the mitigation countermeasure is taken to be interdiction: continued human activity in the area is forbidden. Where calculated radiation levels are above the standard, but low enough for decontamination to become feasible, there is a choice between decontamination and interdiction. The costs of chronic exposure mitigation are computed for all regions by using either the cost of decontamination (where feasible) or the cost of interdiction (where decontamination is not feasible) (see section 11.2.2 for details on decontamination and interdiction). The cost of interdiction is computed as the sum of the following costs:

- Loss in value of public and private property
- Loss of income during period of relocation and temporary unemployment.

12.3 COSTS OF ACUTE EXPOSURE MITIGATING MEASURES

The costs resulting from acute exposure mitigating measures would include:

- Costs of evacuation and temporary food and shelter
- Value of condemned crops and other farm products

Table VI 12-1 shows unit costs for evacuation as estimated in an EPA study (Hans and Sell, 1974) of 64 evacuations following disasters in the United States. The study reports that cost records of these disasters are fragmentary and inconsistent. However, using some of the records, a knowledge of how evacuation costs are incurred, and general data on prices and labor rates, the EPA study constructed the estimates shown.

The EPA study states that the number of personnel required to supervise an evacuation ranges from 0.4 to 5% of the number of evacuees and averages 2%. The evacuation costs appear to include the cost of securing property, although the incidence of looting in disasters is reported to be low. The EPA estimates do not include any costs for unpaid assistance. Nor do they appear to include costs of special equipment and supplies, although it is recognized that these costs are incurred.

On the assumption that 80% of evacuees are transported by private vehicles and obtain commercial accommodation, 20% are transported in buses and accommodated in mass care facilities, and prices have increased about 15% since the EPA study, the unit daily cost would be $13.50 per evacuee for food, shelter, evacuation personnel, and the cost of transportation. The total of these costs for an evacuation lasting about a week would therefore be $54 per day for a family of four.

12.3.2 CONDEMNATION OF AGRICULTURAL PRODUCTS

Farm losses would include the value of condemned milk and crops. Current price data are readily available and need not be quoted here.

To compute the crop losses following an accident the model accounts for deposition of radioactive material on the crops as a function of distance from the reactor and of weather conditions. The diminishing strength of this deposited radioactivity from the time of contamination to the time when the crops would normally be harvested is then calculated. If the contamination level calculated for the time of harvest is within the acceptable standard, the crops are assumed to be harvested and therefore not lost.

12.4 CHRONIC EXPOSURE - COSTS OF DECONTAMINATION AND INTERDICTION

In the chronic exposure phase, mitigating measures are assumed to consist of either land interdiction (including relocation of residents), decontamination, or both. The following section discusses the considerations involved in the calculation of the economic impact of each type of measure.

12.4.1 COSTS OF DECONTAMINATION

12.4.1.1 Farmland

Whether or not farmland should be decontaminated, and the best method to use, would depend on the intensity and decay rate of the contamination, on climate, on physical characteristics of the land, and on how the land is utilized. Table VI 12-2 summarizes the estimated unit costs and effectiveness of three decontamination techniques. The cost estimates are derived from the updated costs of construction (Mohon, 1974; Robert Snow Means Co., 1974).

Some technical limitations on the choice among decontamination methods are suggested by Table VI 12-2. Deep plowing would not be suitable for orchards. Obviously, removal of the trees would increase the cost of the decontamination operations; but it would also increase the loss, because several years are required to develop a fully productive tree.

In the past a farm commonly served as both a residence and a productive enterprise, and this is still often the case. However, in recent decades specialization, mechanization, and the development of a dense network of rural roads has made it feasible, and not unusual, to live in a town and to work a farm several miles away, or even to work a

farm consisting of several parcels separated by considerable distances. Therefore, decontaminating a farm could be wholly for the purpose of protection of farm workers, or also partly for the protection of residents. If contamination were below a certain level, it might be satisfactory to decontaminate only the area surrounding the residence, or to use a technique with a high decontamination factor for the residential area and a technique having a lower factor (and cost) for the remaining land.

12.4.1.2 Unit Costs for Decontaminating Developed Property

The costs of decontaminating developed property are estimated on the assumption that two alternative methods would be used, depending on the degree of decontamination required to meet the radiation exposure standards. If a decontamination factor of 2 would suffice (50% reduction in contamination), the method would consist of replacing lawns and firehosing roofs and paving. If a decontamination factor of 20 were required (95% reduction in contamination), lawns, paving, and roofing would be replaced. The unit costs of these operations are estimated to be:

	Cost per square foot
Replace lawns	$0.11 - 0.14
Replace paving	$0.15 - 0.30
Replace roofing	$0.50 - 2.0
Firehose paving	$0.05
Firehose roofing	$0.05

These costs include the costs associated with the preparation of a disposal site and restoration of the decontaminated properties.

12.4.1.3 Housing

The cost of decontaminating a residence depends on the degree of decontamination sought and such additional factors as the type and size of structure, as well as the areas of surrounding lawns, driveways and streets. As a general rule, the closer a residence is to the "center" of the city (i.e., metropolitan area) and the larger the population of that city, the higher will be the residential density. Thus, in the central areas of large cities, residences tend to be apartments or houses occupying very small sites. Suburban residential development consists predominantly of single family units and both the site and the surface area occupied by the structure tend to be larger than in central areas. Similarly, suburban apartments tend to use more land per household than central city apartments. Ex-urban and rural development is even less dense than suburban. Thus, the costs of residential decontamination would depend partly on distance from a city center and the size of the city. It will be useful, therefore, to consider the costs of residential decontamination for a range of development densities.

Table VI 12-3 shows estimates for decontaminating two single-family residences where the development densities are one residence per acre and five residences per acre, respectively. A density of one unit per acre is typical of rural areas and usually reflects a public health standard for the minimum area for septic field drainage. The cost of decontamination is estimated to be in the range $1370 to $1710 per capita to obtain a decontamination factor (DF) of 2, and in the range $1860 to $3590 to obtain a DF of 20.

A typical urban lot size for single family dwellings is one-sixth of an acre and corresponds to a development density of about five units per acre (allowing for streets). Table VI 12-3 estimates the decontamination cost of a structure occupying 2000 square feet to be in the range $320 to $370 per capita for a decontamination factor of 2 and in the range $560 to $1630 per capita for a decontamination factor of 20.

It is assumed that in the typical apartment development, 30% of the area is occupied by structures and the remainder (which includes streets) is paved. It is assumed that each apartment occupies 1200 square feet (including corridors, etc.); that 3.2 persons live in each apartment; the number of apartments per floor is 10.9; and the number of people per floor is 34.8, or 31 if 90% occupancy is assumed. If three floors of apartments are assumed, the decontamination cost becomes about $30 per person for a DF of 2, and in the range $140 to $420 per person to achieve a DF of 20. For a six-floor structure, these per capita costs would be halved. These results are summarized in Table VI 12-4.

12.4.1.4 Commercial, Industrial, and Public Property

The costs for decontaminating commercial, industrial, and public property may be constructed in the same manner as for residences, on the basis of cost estimates for decontamination of roofs, paving and lawns. If it is assumed that an industrial or commercial lot is 50% occupied by a structure and the remainder is paved, the decontamination cost becomes about $2200 per acre for DF=2, and in the range $14,000 to $56,000 per acre for DF=20. The proportion of the lot occupied by structures depends primarily on its location and the industrial or commercial activity for which it is used. Activities requiring large areas for shipping and receiving or for parking cannot usually support the high price of land in the central areas of large cities and are located in rural or suburban areas. Activities that are carried out in densely developed areas usually are those that can obtain a high level of land utilization, usually through the use of high-rise structures. Table VI 12-5 shows the land use per 100 population for commercial, industrial and public purposes in a sample of central cities and satellite cities of various sizes. In general, land is more intensively used in small central cities than in large. That such tendencies are not observed in the data for satellite cities probably is a reflection of their lower land values, which do not provide as strong incentives for sparing use of land.

A weighting of the data in Table VI 12-5 by the distribution of U.S. population among the urbanized areas leads to the averages shown in Table VI 12-6. By assuming commercial and industrial land is 50% occupied by structures and 50% paved for streets, parking lots and driveways, the cost of decontamination of these areas would be about $21 per capita for DF=2 and in the range $140 to $490 for DF=20. By assuming parks to be mainly lawn with surrounding streets, the per capita cost would be in the range $26 to $33 for DF=2 and $31 to $46 for DF=20. Public areas include a variety of buildings, such as schools, government buildings, and sewage plants. In general, the land use in these areas is less intensive than in commercial and industrial areas. On the assumption that public land is 30% occupied by structures and the remainder is paved for streets and parking lots, the decontamination cost would be about $2200 per acre for DF=2 and in the range $11,000 to $35,000 per acre for DF=20. The per capita cost would be about $40 fo DF=2 and in the range $200 to $640 for DF=20.

Table VI 12-7 is a summary of the cost estimates for decontamination of commercial, industrial, and public property.

12.4.1.5 Summary of Decontamination Costs

Farmland

A reliable estimate would consider the level of contamination for each area and select between decontamination and relocation of individuals to limit their exposure. The costs are likely to be in the range zero to $5000 per acre. When higher costs would be incurred, abandoning the land is likely to be the preferred measure. The model assumes that surface soil burial by deep plowing would be used for tilled land and grazing land, and scraping surface soil would be used for orchards. The costs are weighted by the area's share of farmland use in the United States. The weighted cost is $230 per acre. The overall decontamination factor is about 20.

Developed Land

For land used for residential, commercial, industrial, and public purposes, the costs would depend very much on how intensively the land is used, and this in turn would depend on the size of the urban area and where the affected area is located within it. The cost estimates for decontamination of residential property in Table VI 12-4 are weighted by the total U.S. housing statistics of location and housing type. The weighted cost is in the range $530 to $640 per capita for DF=2 and in the range $780 to 1830 per capita for DF=20. The cost estimates for decontamination of commercial, industrial and public properties are shown in Table VI 12-7. The overall cost estimates for developed land are derived by combining residential land costs and the values of Table VI 12-7 and are in the range $620 to $730 for DF=2 and in the range of $1150 to $3000 for DF=20. The model uses $680 for DF=2 and $1700 for DF=20.

12.4.2 INTERDICTION AND RELOCATION

If land were to be interdicted, the occupants and owners would bear two kinds of costs--loss of productive use of the land and its improvements (structures and other fixtures), and the costs of relocation. The general principles for calculating the cost of interdiction are the same for most types of land. The costs of relocation are not so easily calculated because of a scarcity of data.

12.4.2.1 General Principles for Calculating Cost of Interdiction

The property is assumed to have a market value, and this value may be considered to be the sum of the value of the land, plus the value of the improvements. The value of the property to the owner is the value of the uses to which he can put it or the amount that it could be sold for, whichever is higher. However, in this discussion the latter will be assumed, i.e., its value is the market value.

If the property is interdicted for T years, it is assumed that no use can be made of it for that time. This does not mean that the land has lost all value. The property would be valueless only if either it were permanently interdicted, with no possibility of the interdiction order being canceled; or the fixed cost of owning it were more than any possible future benefit to the owner. The likely situation is that it will be potentially useful at the end of T years.

Let V_L be the value of the land before interdiction and let V_I be the value of the improvements. Assume that the property could be as valuable in real terms after T years as before interdiction if it were in the same condition.

Although the condition of the land is assumed to be essentially unchanged, the improvements will have depreciated because of functional obsolescence and lack of maintenance. Let d be the annual rate of depreciation. Then T years later the value of the property will be

$$V_T = V_L + \exp(-Td)\ V_I.$$

There is a cost associated with holding the property for T years. If it were sold at any earlier time, the proceeds could be invested at interest or existing loans could be reduced with a consequent saving in interest costs. In addition, it is assumed that the property would continue to be subject to real estate taxes in proportion to its value. Let r be the interest rate on money plus the property tax rate. Then the value of the property immediately after interdiction (PV) is its value at time T reduced by the cost of holding it until then:

$$PV = \exp(-rT)\ V_T$$

$$= \exp(-rT)[V_L + \exp(-Td)\ V_I].$$

Let the value before interdiction be

$$V_0 = V_L + V_I$$

and let the value of improvements as fraction of total value be

$$a = V_I/V_0.$$

Then

$$PV = \exp(-rT)[(1 - a)\ V_0 + a \exp(-Td)\ V_0]$$

$$= \exp(-rT)[(1 - a) + a \exp(-Td)]\ V_0.$$

To see what this means in practical terms, assume that the interest rate is 9% and the property tax is 3%. Then r = 0.12.

Let improvements depreciate at 20% per year to reflect cost of maintenance. Then

$d = 0.20$ and $PV = \exp(-0.12T)[(1 - a) + a \exp(-0.120T]\ V_0$.

For residential, business and public property, the improvements are usually valued at about 70% of the total. For farm property, improvements may be valued at about 25% of the total. Table VI 12-8 uses this equation for PV to show the effect of interdiction periods of 1, 5, 10, and 20 years on properties whose values before interdiction were 100 units. The only parameter in the equation whose value could be seriously in error is the depreciation rate on improvements. The value of 20% is judged to be appropriate in view of the lack of maintenance during interdiction. Where property is maintained, depreciation is usually judged to be in the range 3 to 5%.

The crucial assumption in the calculation is that land will regain its previous value (adjusted for inflation) when interdiction ends. However, if a community were interdicted it would become a ghost town and it might or might not be restored. Because of the deteorioration of structures, the former is certainly a probable outcome. On the other hand, the infrastructure of utilities, sewers, streets and roads could be attractive to a developer who might find that by purchasing the entire stock of real estate in the community he could reduce the deterioration or redevelop the area to advantage, exploiting the infrastructure and any locational advantages. The valuation of farms in these calculations inspires more confidence. Unlike residences and commercial or industrial establishments, a farm's value is not dependent on its close proximity and ease of access to other establishments which might not be restored after interdiction. The important locational requirement of a farm is access to markets for its supplies and its products, which would probably not be changed by a period of interdiction.

The valuation of loss by the calculation above could be refined considerably to reflect differences in the nature of holding costs for various periods of interdiction. For example, if interdiction were for no more than 5 years the depreciation rate could be judged too high, but additional carrying costs would be incurred. If a farm were interdicted for a short period, say a few months, the owner would not relocate and would continue to hold stocks and movable equipment, although they could not be used. The additional carrying costs would include interest, insurance and possibly personal property taxes. For a longer period, say 5 years, stocks and movable equipment would probably be relocated or sold, but insurance on the structures would probably be kept in force. Thus, while the depreciation rate of 20% may be high for shorter interdiction periods, the resulting bias is offset by the absence of other holding costs. Whether the net result is a high or low estimate of loss for shorter interdiction periods has not been ascertained.

12.4.2.2 Relocation Costs

In the event that land and structures come to be interdicted, the people must be relocated in some permissible area. The cost of such a relocation is made up of two factors — loss of income and moving costs.

Loss of Income

Loss of income is subdivided into the parts associated with the residential sector and the corporate business sector. The residential or household sector is made up of wages and salaries, proprietor's income, and rental income. Excluded from this category are types of income which would not be affected by interdiction and relocation, such as interest from personal savings accounts, dividends, unemployment insurance, etc. The U.S. average for this type of income is $4400 per capita per year[1].

[1]This number is an estimate for 1975 (Statistical Abstracts of the United States) using 1972 data and an 8% increase per year.

This income loss would only be applicable during the period of resettlement. This study assumes that this period lasts 90 days, allowing the person time to resettle and to find a job, if unemployed. This number is based on information that the average actual duration of unemployment benefits given from 1960 to 1972 was 11.4 to 14.3 weeks (80 to 100 days). The household loss of income therefore would be about $1100 per capita.

Loss of income for corporations would partly be the result of loss of profits and partly the result of continued interest on debts, and depreciation of equipment. In 1974 these categories amounted to 385 billion dollars, with the profits being taken before tax. This value amounts to $1850 per capita per year. In this study it was assumed that corporate relocation took on the average six months to complete. This was chosen with the knowledge that although some businesses require much longer than 6 months to relocate, others take significantly less than this. Thus the cost for relocation due to loss of income is $940 per capita.

Moving Costs

The costs incurred in moving people to a new area are made up of household costs and business costs. The shipping of 10,000 pounds of family belongings by commercial movers costs $1100 to $1400 for a distance of 50 to 100 miles. Since the average family in the U.S. has 3.2 members, this cost would average $340 to $440 per capita. A value of $400 per capita was used in this study.

Estimates of the cost of moving a business are not so readily available. In this study the cost was assumed to be 10% of the value of the equipment and inventory. The value of such equipment and inventories has been placed at 850 billion dollars in 1975, or $4200/per capita. The moving cost is therefore estimated to be about $420 per capita.

The cost of moving the public sector (i.e., governmental agencies, etc.) must also be accounted for. Once again, it is assumed that the moving cost is 10% of the value of equipment and inventory. The value of such items was placed at 111 billion dollars in 1975, or about $500 per capita. The moving cost is therefore about $50 per capita.

The total per capita moving cost is the sum of the cost from each sector, or about $870 per capita. The total relocation cost is this figure plus that for loss of income, or about $2900 per capita.

12.5 VALUES ASSIGNED TO IMPORTANT PARAMETERS

The values of parameters used in the model for cost calculations are shown in Table VI 12-9.

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TABLE VI 12-1 EPA ESTIMATES OF FOOD, SHELTER, AND TRANSPORTATION COSTS FOR EVACUEES AND EVACUATION PERSONNEL[a]

Type of Expenditure	Cost per Person
Evacuee cost:	
Food and shelter, daily cost:	
Commercial[b]	$11.00
Mass care	5.00
Transportation:	
Private[c]	1.00
Commercial[d]	0.55
Evacuator personnel cost:	
Compensation	35.00
Food, shelter, transportation	Same as for evacuees

[a] From Hans and Sell (1974).

[b] Assumes two or more persons to a room.

[c] Privately owned vehicle, three or four passengers per vehicle, round-trip distance 30 miles, 12¢/mile operating cost.

[d] Assuming 45 to 50 persons per vehicle, round-trip distance 30 miles, 65¢ to 80¢ per vehicle-mile.

TABLE VI 12-2 COST AND EFFECTIVENESS OF FARMLAND DECONTAMINATION

Condition of Land	Technique	Reduction in Contamination[a] R (%)	Decontamination Factor[b] DF	Unit Cost[c] ($/acre)
Tilled soil	Scrape surface and dispose of it	99	100	520-810
	Bury surface soil in place by grading	94	17	47-120
	Bury surface soil in place by deep plowing	95.5	22	75
Grazing land	Bury surface soil in place by deep plowing[d]	95.5	22	320
Orchards	Scrape surface soil and dispose of it[e]	99	100	3000-5000

[a] Percentage reduction in amount of contaminant per unit of surface area. See section 11.2.2.3 and Appendix K.

[b] DF = 100/(100-R). See Appendix K for discussion.

[c] Estimates based on data presented by the Robert Snow Means Co. (1974), Mohon (1974), and the U.S. Department of Agriculture (1974).

[d] Includes restoring land by reseeding grass.

[e] Includes (1) removing and replacing the plantings and (2) loss of harvest for 5 years.

TABLE VI 12-3 DECONTAMINATION COST FOR A SINGLE-FAMILY DWELLING[a]

Parameter	Low-Density Dwelling	Medium-Density Dwelling
Development density (units per acre)	1	5
Average lot size (ft^2)	40,000	7260
Street area per residence (ft^2)	3,560	1450
Area of driveway (ft^2)	1,000	300
Area occupied by structure (ft^2)	2,500	2000
Area occupied by lawn (ft^2)	36,500	4960
Decontamination factor = 2:		
Cost per dwelling	$4370-5460	$1020-1170
Cost per capita[b]	$1366-1706	$ 319-366
Decontamination factor = 20:		
Cost per dwelling	$5950-11,500	$1800-5220
Cost per capita[b]	$1860- 3,590	$ 560-1630

[a] Rough estimates constructed on the basis of the structure parameters listed in this table.

[b] Assuming 3.2 residents per dwelling.

TABLE VI 12-4 SUMMARY OF CONSTRUCTED COST ESTIMATES FOR DECONTAMINATION OF RESIDENTIAL PROPERTY

Type of Structure	Development Density	Per Capita Decontamination Cost DF = 2	Per Capita Decontamination Cost DF = 20
Single-family dwelling unit	1 per acre	$1370-1710	$1860-3590
Single-family dwelling unit	5 per acre	$320-370	$560-1630
Three-story suburban apartment building (90% occupancy)	Structure occupies 30% of land	$30	$140-420
Six-story urban apartment building (90% occupancy)	Same	$15	$70-210

TABLE VI 12-5 COMMERCIAL, INDUSTRIAL, AND PUBLIC USE OF URBAN LAND[a]

	Acres per 100 Persons		
	Commercial and Industrial Use	Parks	Public Use
Central cities	0.76	0.50	0.93
Satellite cities	1.14	0.69	2.50

[a]From Bartholomew and Wood (1955).

TABLE VI 12-6 POPULATION-WEIGHTED LAND USE

Type of Use	Acres per 100 Persons
Commercial and Industrial	0.98
Parks	0.61
Public Areas	1.83

TABLE VI 12-7 COST ESTIMATES FOR DECONTAMINATION OF COMMERCIAL, INDUSTRIAL, AND PUBLIC PROPERTY

Type of Structure	Per Capita Decontamination Cost	
	DF = 2	DF = 20
Industrial or commercial building	$21	$140-490
Parks	$26-$33	$31-46
Public Areas	$40	$200-640

TABLE VI 12-8 EFFECT OF INTERDICTION ON PROPERTY VALUES

Interdiction Period T (years)	Residential, Business, and Public (a = 0.70)	Farm (a = 0.25)
None	100	100
1	77	85
5	31	46
10	12	24
20	3	7

TABLE VI 12-9 INPUT PARAMETERS FOR THE MODEL

Parameter	Value	Remarks
Distance of evacuation in the downwind direction (m)	3.2×10^4 (25 miles)	
Distance of evacuation in the upwind direction (m)	8.0×10^3 (5 miles)	
Angle of evacuated area (degrees)	45	
Cost of evacuation per evacuee	95	$13.5/day x 7 days
Loss of milk and crops	See Table VI 10-4	
Loss of property:		
Depreciation rate of improvements (yr^{-1})	0.20	
Value of farm property	See Table VI 10-4	
Value of developed property[a] (per capita)	$17,000	
Relocation cost per capita	$2,900	
Decontamination:		
Decontamination cost of farmland ($/acre)	$230	
Decontamination cost of developed land for DF = 2	$680	
Decontamination cost of developed land for DF = 20	$1,700	

[a] Data from National Bureau of Economic Research (1971).

Section 13

Calculated Results with Consequence Model

This section is directed towards the calculation of the probability versus magnitude of the consequences of an accident at one of the first 100 commercial light water reactors. Before discussing these calculations, it is necessary to establish the framework for the calculations, with regard to the health effects described in section 9 and also the sampling scheme for the meterological data. In order to understand the model, it is also helpful to do some parametric studies with a uniform population, thereby eliminating one variable. With this groundwork laid, the basic risk calculations are described followed by some additional calculations giving greater perspective on the results. Finally, there is a discussion of the overall uncertainties in the results.

13.1 MEDICAL FRAMEWORK

As was frequently noted in section 9.2, the selection of dose-mortality criteria depend, to some extent, on the temporal pattern of dose to different organs and the relative magnitudes of the doses to different organs. This section shows a series of hypothetical calculations to provide this basis and to contribute to the understanding of the health effects model.

Figures VI 13-1 through VI 13-4 show (1) the external dose from the passing cloud, including a shielding factor of 0.75, (2) the external dose from ground contamination within a 24-hour time period[1] after deposition, including a shielding factor of 0.33, and (3) the dose, as a function of time, from internally deposited radionuclides to the bone marrow, the lung, the regenerative cells of the lower large intestine[2] and the thyroid. The relative magnitudes of the doses from the three exposure modes and the temporal distributions of the internal doses are self-evident; they are discussed in section 9.2.

For the critical time periods (specified in the appropriate sections of section 9.2), the contributions of the major radionuclides are shown in Fig. VI 13-1 et seq. The dose contributions are for radionuclides released from the containment and include any contribution from their radioactive daughters. The reader should also note that the graphs are logarithmic with respect to dose. The radionuclide contributors are ranked in descending order and in general "others" contribute less than about 5% of the total dose. The relative importance of certain radionuclides will be discussed later after presentation of similar calculations for the late health effects.

At any distance from the reactor, the external doses from the passing cloud, the external dose from ground contamination, and the dose from internally deposited radionuclides are all approximately proportional to the concentration of radionuclides in the air near the ground at that distance. This airborne concentration as a function of distance from the reactor is strongly influenced by the prevailing stability and wind speed, especially within 20 miles of the reactor. However, for a given release category, the relative magnitudes of the three doses and the time-dependence of the internal dose are basically independent of distance and weather within this region since (1) they are all approximately proportional to the same variable, (2) the time periods after release are short, so that differences in radioactive half-life do not become significant, and (3) the deposition velocities (and washout coefficients) of 1-micron particles and iodine are equal so that all radionuclides except the noble gases are uniformly depleted from the plume. Furthermore, the noble gases are negligible contributors to dose within this region. Therefore, the temporal behavior of doses is shown in Fig. VI 13-1 et. seq. for only one distance (0.5 mile) from the reactor and only one weather type (stability category A, 0.5 m/sec, or 1.1 mph) for a large, cold, ground-level release.

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[1]For shorter time periods, the dose from ground contamination is approximately proportional to the time period.

[2]For brevity in section 13.1 the regenerative cells in the lower large intestine will be referred to as the gastrointestinal tract.

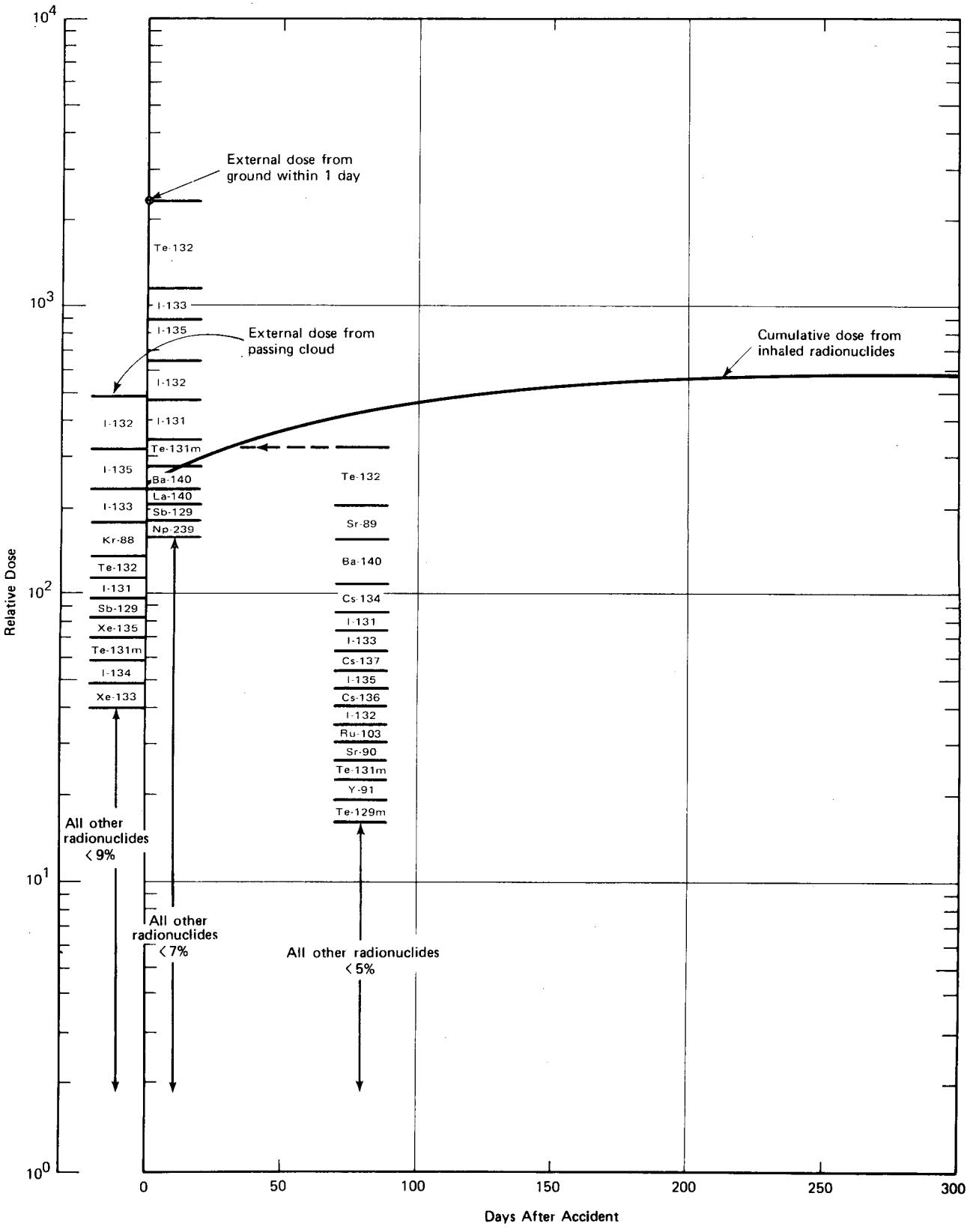


FIGURE VI 13-1 Relative doses to bone marrow at 0.5 miles from reactor.

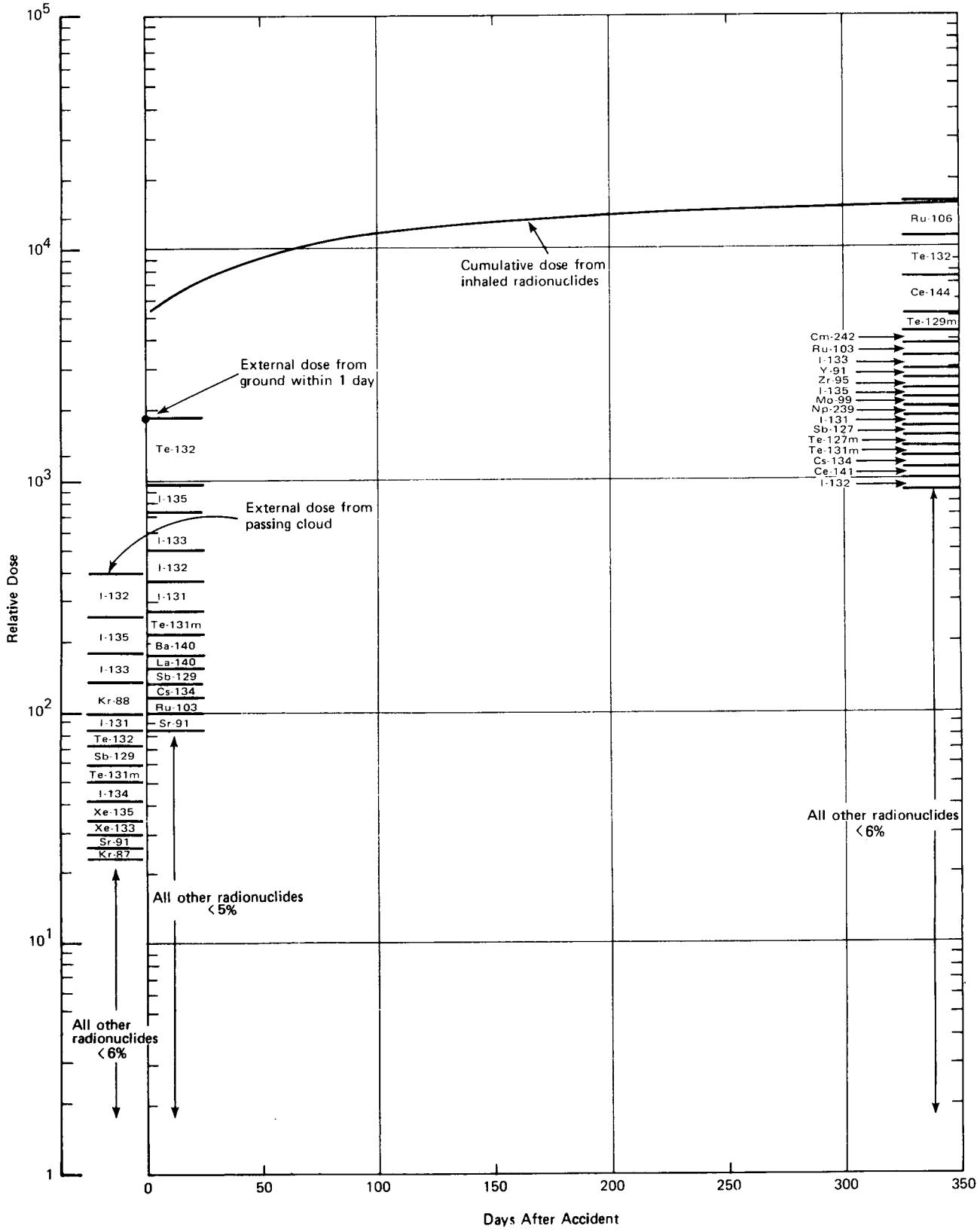


FIGURE VI 13-2 Relative doses to lung at 0.5 miles from reactor.

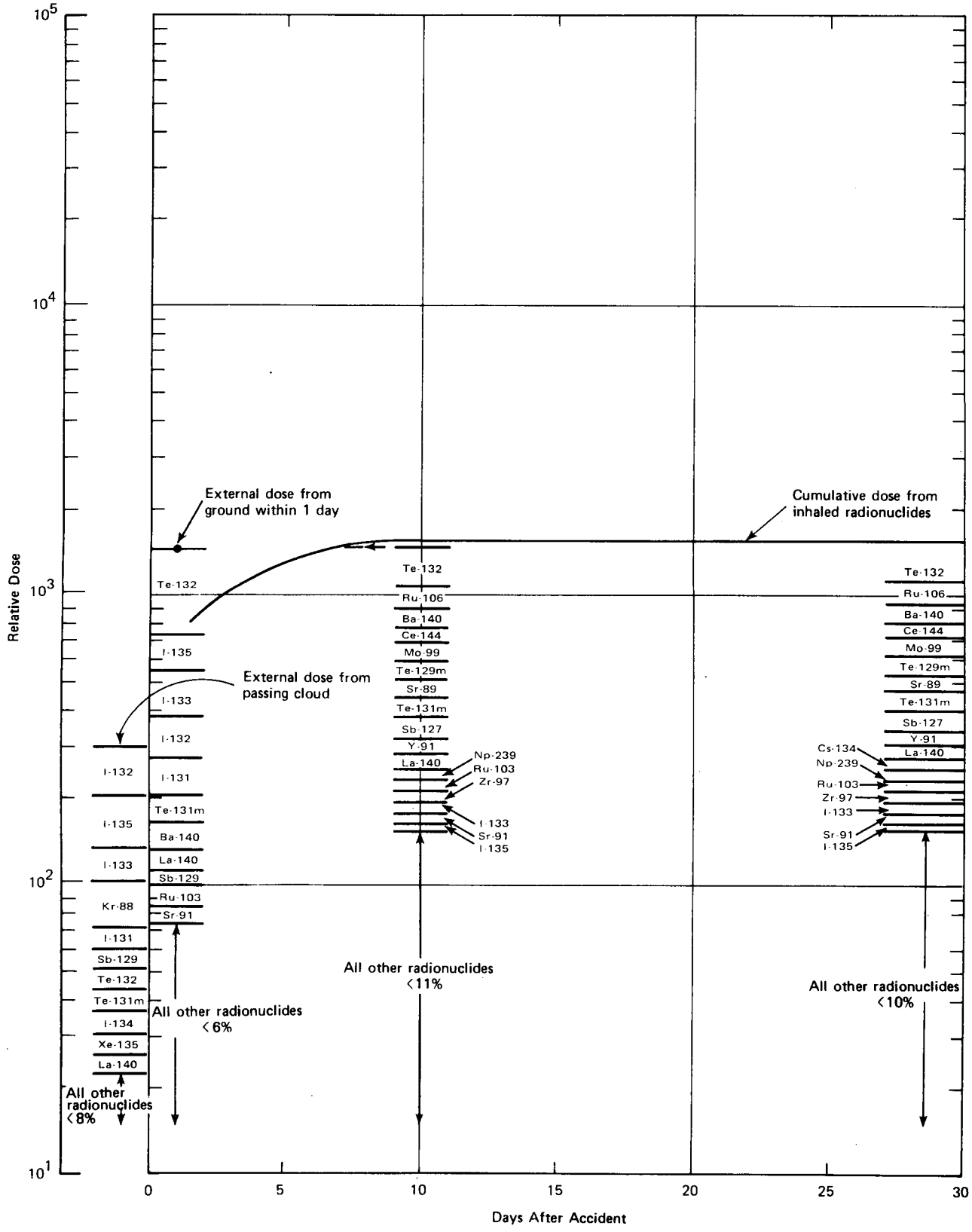


FIGURE VI 13-3 Relative doses to regenerative cells of lower large intestine at 0.5 miles from reactor.

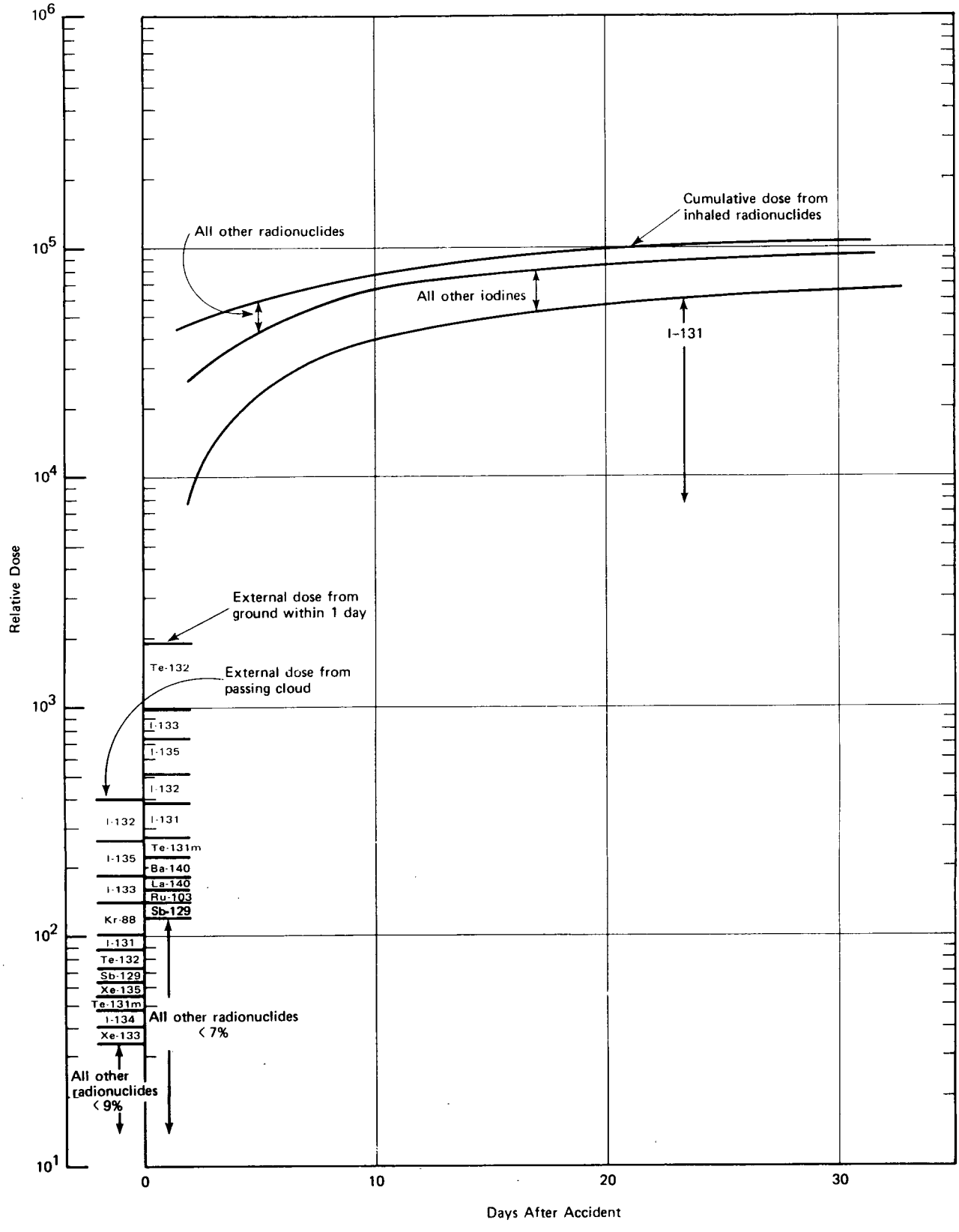


FIGURE VI 13-4 Relative doses to thyroid at 0.5 miles from reactor.

To establish the effect of weather, graphs have been drawn of the magnitude of the doses to the bone marrow, the lung, the gastrointestinal tract and the thyroid within the appropriate time periods as a function of distance from the reactor. Figs. VI 13-5 and VI 13-6 are such graphs for two hypothetical cases in which a large, cold, ground-level release is assumed and the weather is assumed to be time-invariant. These weather conditions are (1) stability category A with 0.5 m/sec (1.1 mph) wind speed, and (2) stability category F with 2.0 m/sec (4.5 mph). The earlier dose-time graphs may be scaled for different distances and weather types by using the relationships depicted in these graphs for these hypothetical cases. Out of all possible combinations of stability and wind speed, the above two weather types were selected to represent extremes with respect to doses within 20 miles but still having relatively high probabilities of occurrence. It should be noted in Figs. VI 13-5 and VI 13-6 that, for each weather type, the doses to the four organs as a function of distance are approximately parallel. This feature reflects the proportionality argument given above. By comparing the doses to the bone marrow, the lung and the gastrointestinal tract at a given distance with the corresponding dose-mortality criteria for these organs in Fig. VI 9-1, VI 9-3, and VI 9-4, respectively, one can compute the percentage of the population at that distance that will die within 60 or 365 days. Fig. VI 13-7 shows that, for these hypothetical weathers, early and continuing mortalities are limited to 10 miles from the reactor and that damage to the bone marrow is the dominant mechanism.[1]

Latent cancer fatalities and genetic effects may result from smaller doses and lower dose rates than those that cause early health effects, accumulated over time periods as long as 50 years or more after the accident. These long-term doses depend on the interdiction policies which are discussed in section 11.2.2. It is helpful to consider separately two population groups. The first group would be relatively close, e.g., 10 to 30 miles, to the reactor at the time of the hypothetical accident so that they inhaled a substantial quantity of radioactive material but insufficient to result in early death. The land on which this population lived would probably be sufficiently contaminated to require relocation of these people so that they would receive no long-term external dose. The inhaled radioactive material is largely retained in the body where it either decays radioactivity or is eliminated from the body throughout the remaining life-span. The long-term doses to the bone marrow, the lung, the mineral bone, the breast, and the testes from these internally deposited radionuclides are plotted in Figs. VI 13-8 through VI 13-12 for consecutive time periods from 1 to 50 years after a large, cold release. The dose magnitudes are calculated for a distance of 10 miles from the reactor by assuming time-invariant weather of stability category A and 0.5 m/sec. Only people born prior to the accident can receive this internal dose and, as explained in sections 9.3.2.3 and 9.4.2, the aging of this population should be taken into account in calculations of latent cancer fatalities and genetic effects. In order to account for aging, the dose received in each time period is used, so Figs. VI 13-8 et seq. are not plotted on a cumulative basis. The main radionuclides contributing to dose in each period are shown and will be discussed below.

The second population group would be located relatively far from the reactor (e.g., 30 to 100 miles) at the time of the hypothetical accident. Their doses both external from the passing cloud and internal from the inhaled radionuclides would be relatively small. However, the land on which this population lived would probably not be contaminated above the level requiring their relocation. As long as these people remained on this land, they would receive small external doses from the contaminated ground, and small internal doses from ingested foodstuffs and inhalation of resuspended radioactive material. The long-term doses to the bone marrow, the lung, the mineral bone, the breast, and the testes from each of these exposure modes (a 0.33 shielding factor is applied to the external ground dose) are shown in Figs. VI 13-13 through 13-17.[2] The dose magnitudes shown in these graphs are representative of 100 miles from the reactor in the event of a large, cold, ground-level release. It should be understood that these calculated doses are for individual who lives continuously at the same location and ingests locally produced foodstuffs exclusively, i.e., no produce, meat,

[1] When time-dependent meteorology is considered, there are infrequent weather sequences in which early fatalities can occur beyond 10 miles and the risk of death from lung damage competes with that from bone marrow damage. For example, several hours of brisk winds with stability category A followed by a near calm or rain can result in high ground dose rates at 15 or more miles from reactor. See section 13.3.1 and Fig. VI 13-23.

[2] The dose from a specified radionuclide includes any contribution from its daughters.

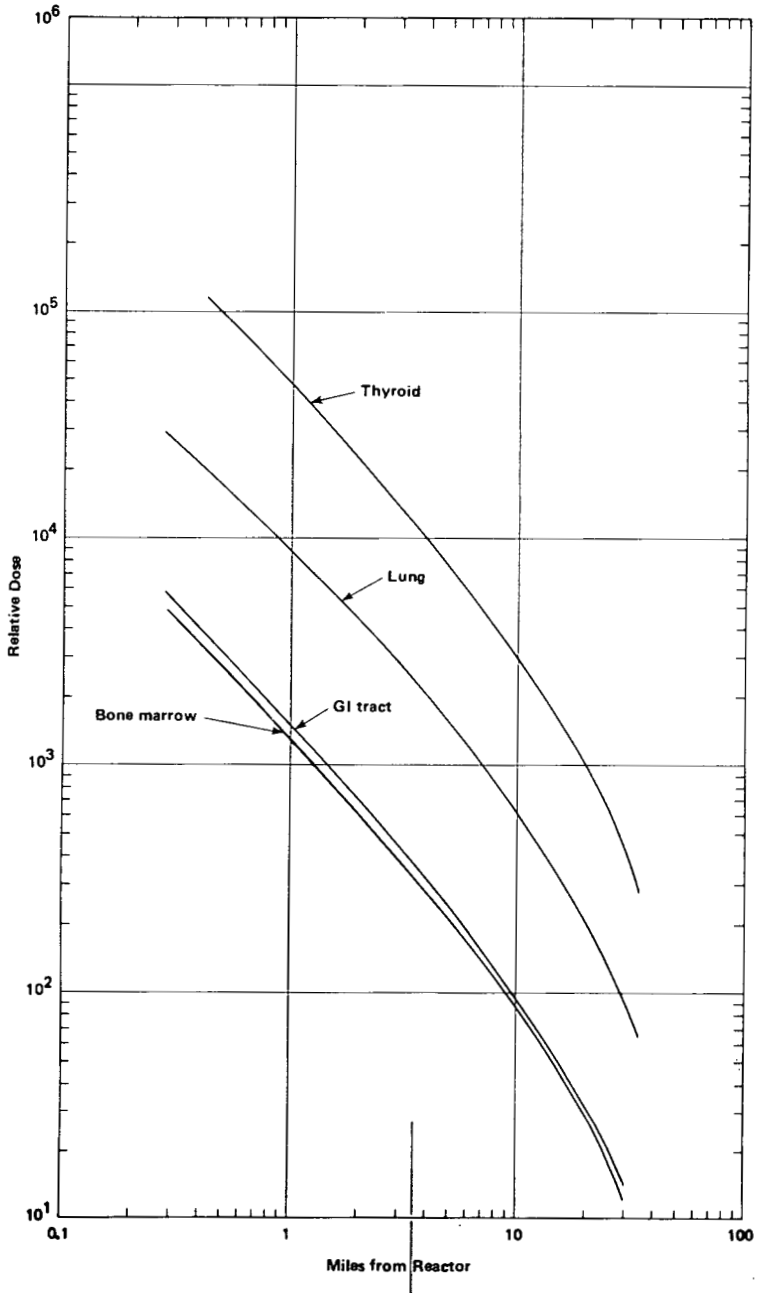


FIGURE VI 13-5 Total organ doses versus distance from reactor for hypothetical weather; stability A, wind speed of 0.5 m/sec. Thyroid dose = 1-day ground + external cloud dose + 30-day inhalation dose; Lung dose = 1-day ground + external cloud dose + 1-year inhalation dose; GI tract dose = 1-day ground + external cloud dose + 7-day inhalation dose (the GI tract dose is the dose to the regenerative cells of the lower large intestine); bone marrow dose = 1-day ground + external cloud dose + ½(7-day inhalation + 30-day inhalation dose)

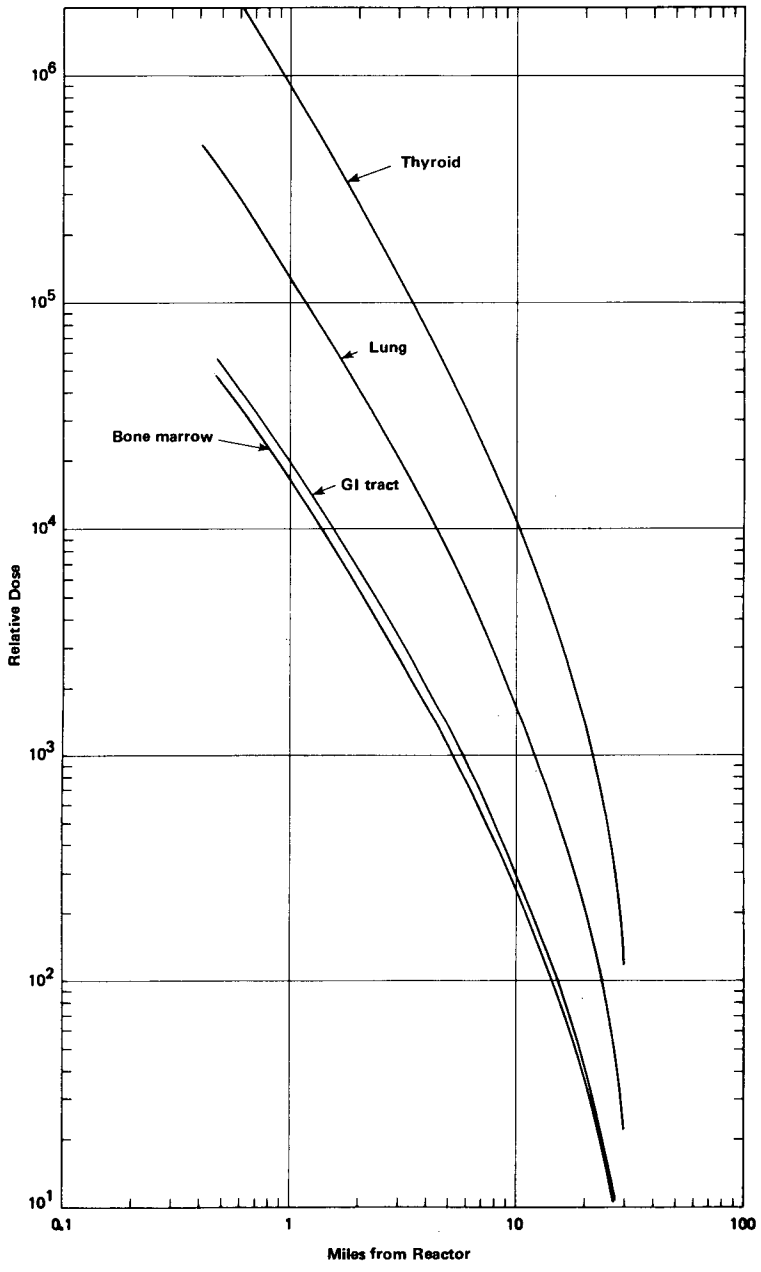


FIGURE VI 13-6 Total organ doses versus distance from reactor for hypothetical weather; stability F, wind speed = 2.0 m/sec. Thyroid dose = 1-day ground + external cloud dose + 30-day inhalation dose; Lung dose = 1-day ground + external cloud dose + 1-year inhalation dose; GI tract dose = 1-day ground + external cloud dose + 7-day inhalation dose (the GI tract dose is the dose to the regenerative cells of the lower large intestine); bone marrow dose = 1-day ground + external cloud dose + ½(7-day inhalation + 30-day inhalation dose)

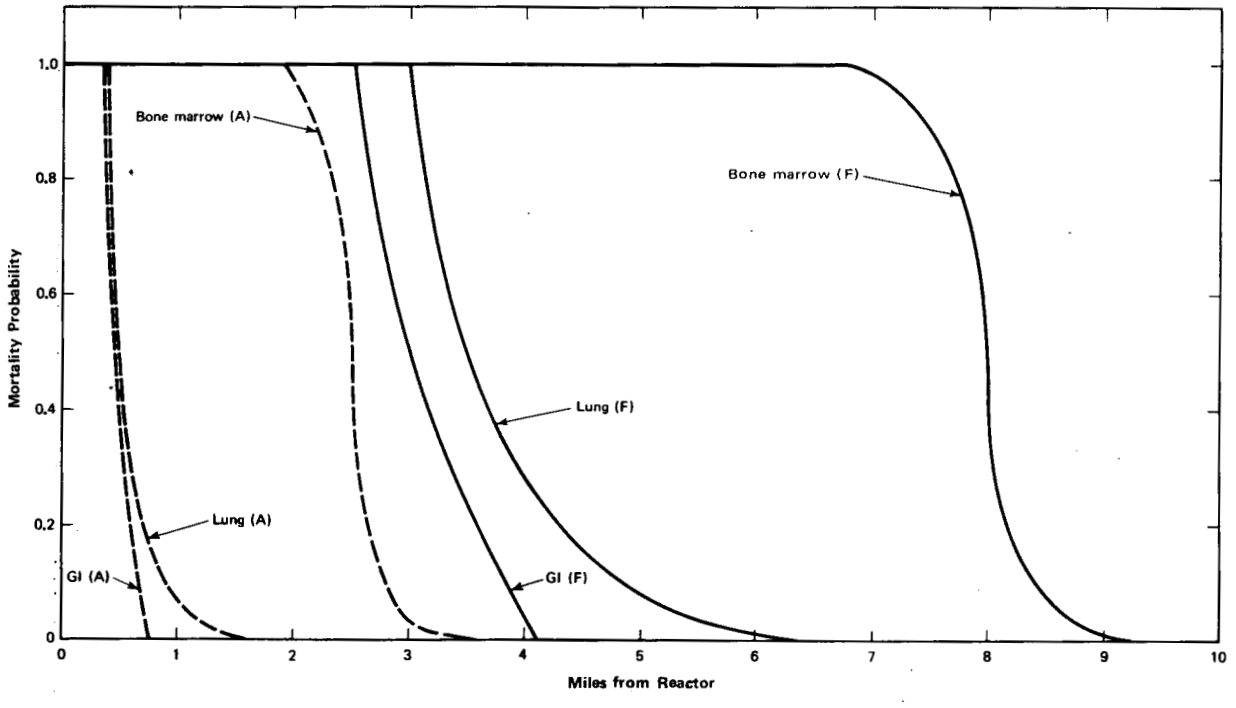


FIGURE VI 13-7 Mortality probability for an affected population versus distance from reactor for two hypothetical weathers: stability category A, wind speed = 0.5 m/sec; stability category F, wind speed = 2.0 m/sec.

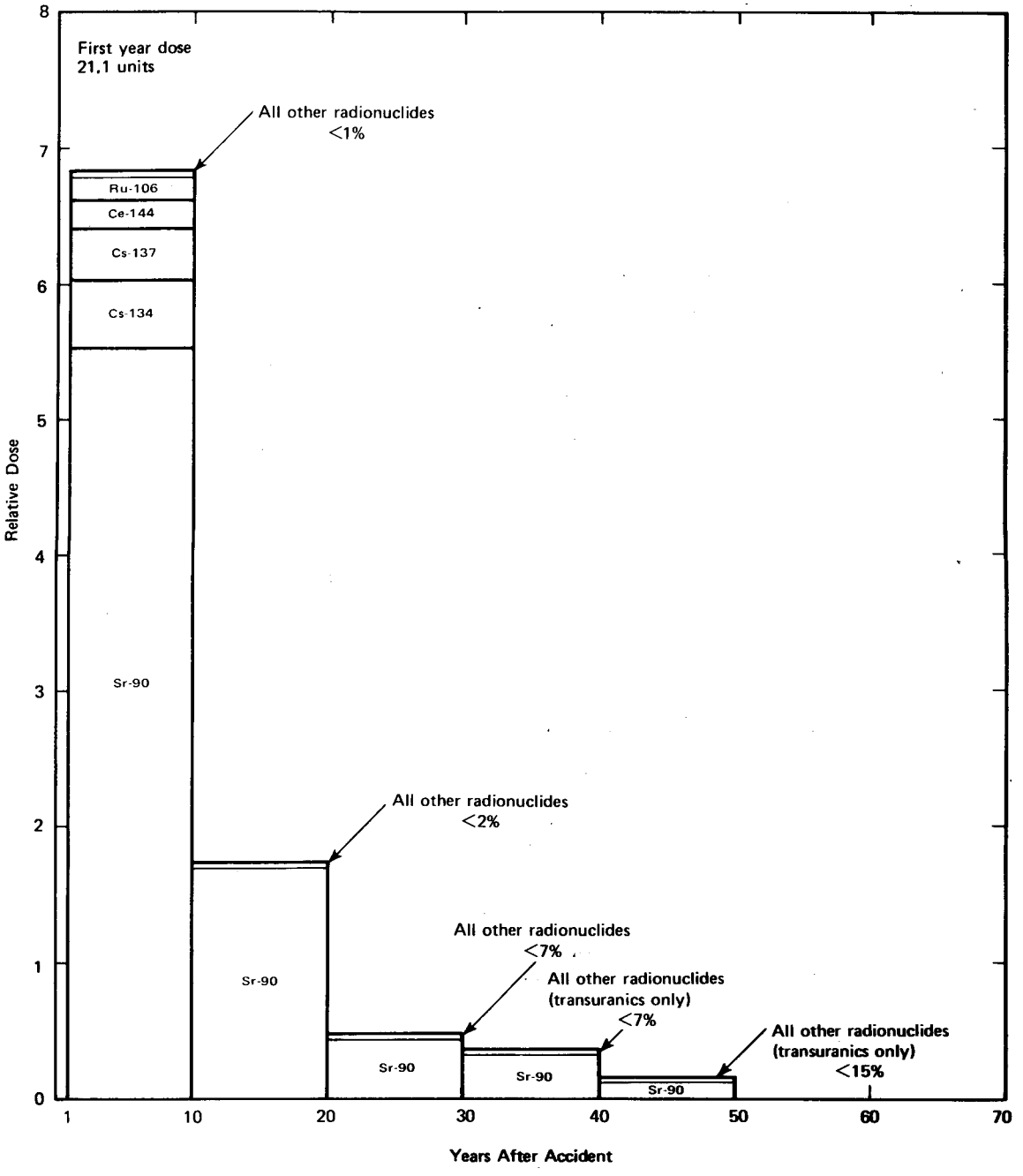


FIGURE VI 13-8 Relative incremental dose to bone marrow from inhaled radionuclides at 10 miles from reactor.

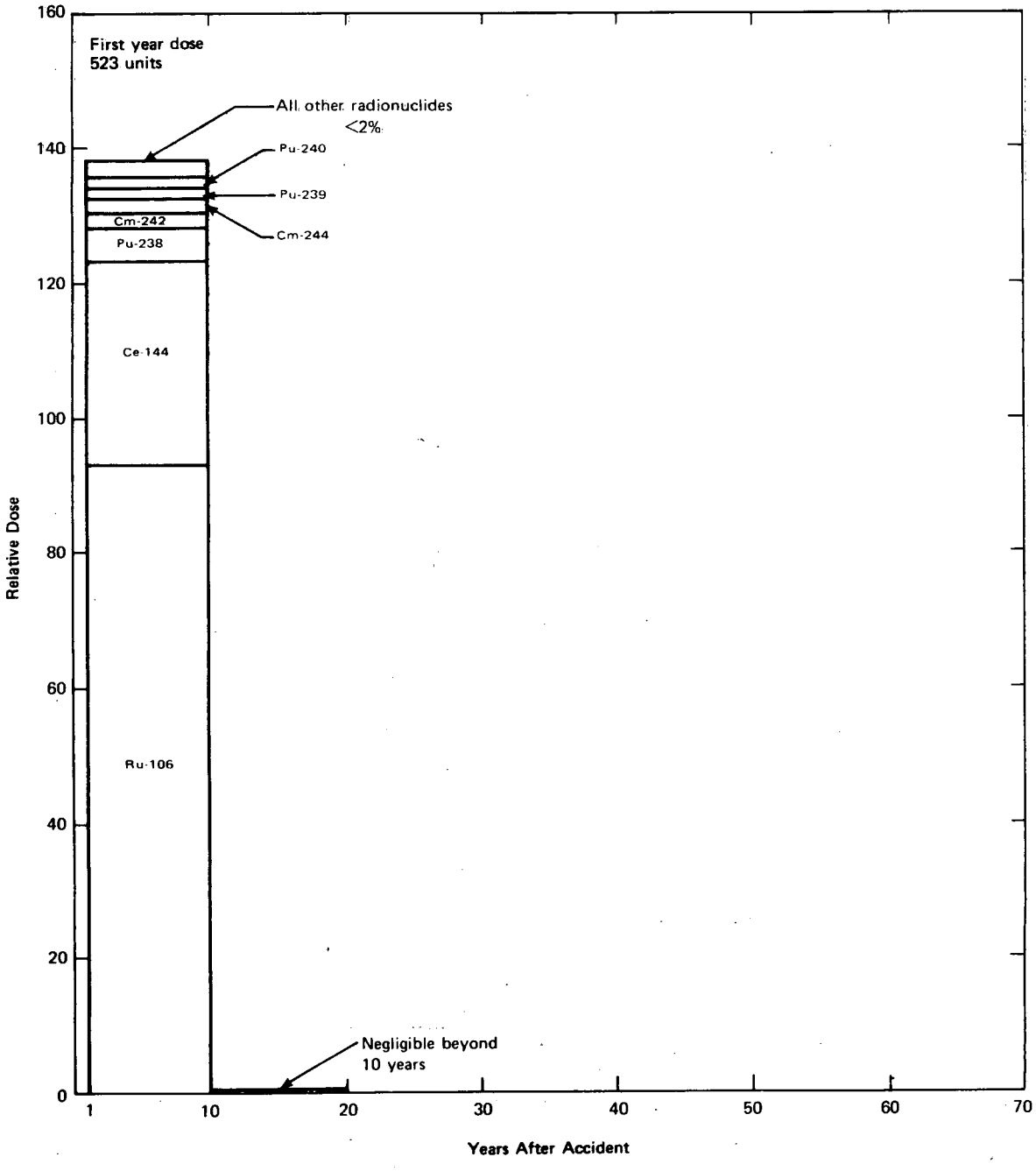


FIGURE VI 13-9 Relative incremental dose to lung from inhaled radionuclides at 10 miles from reactor.

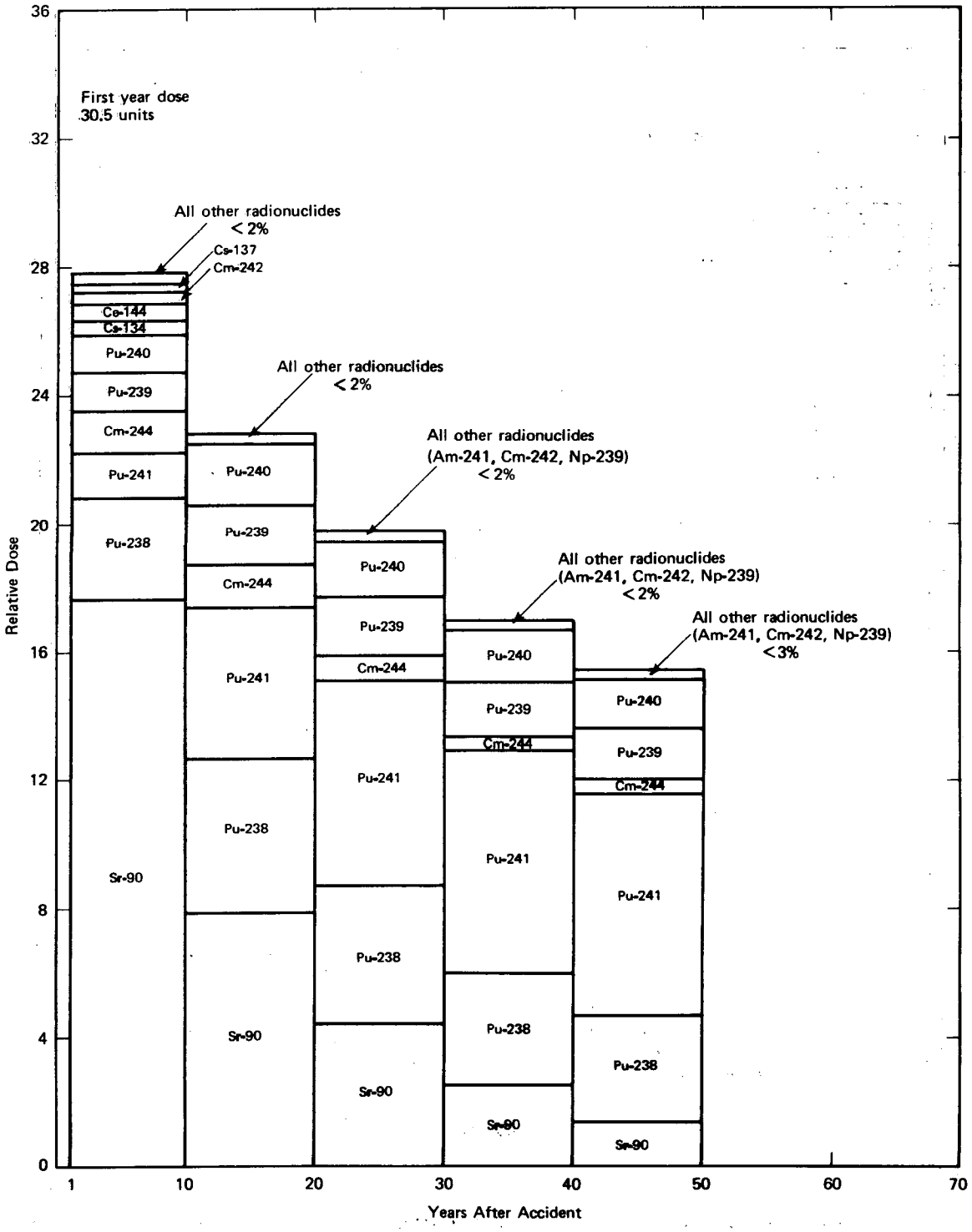


FIGURE VI 13-10 Relative incremental dose to mineral bone from inhaled radionuclides at 10 miles from reactor.

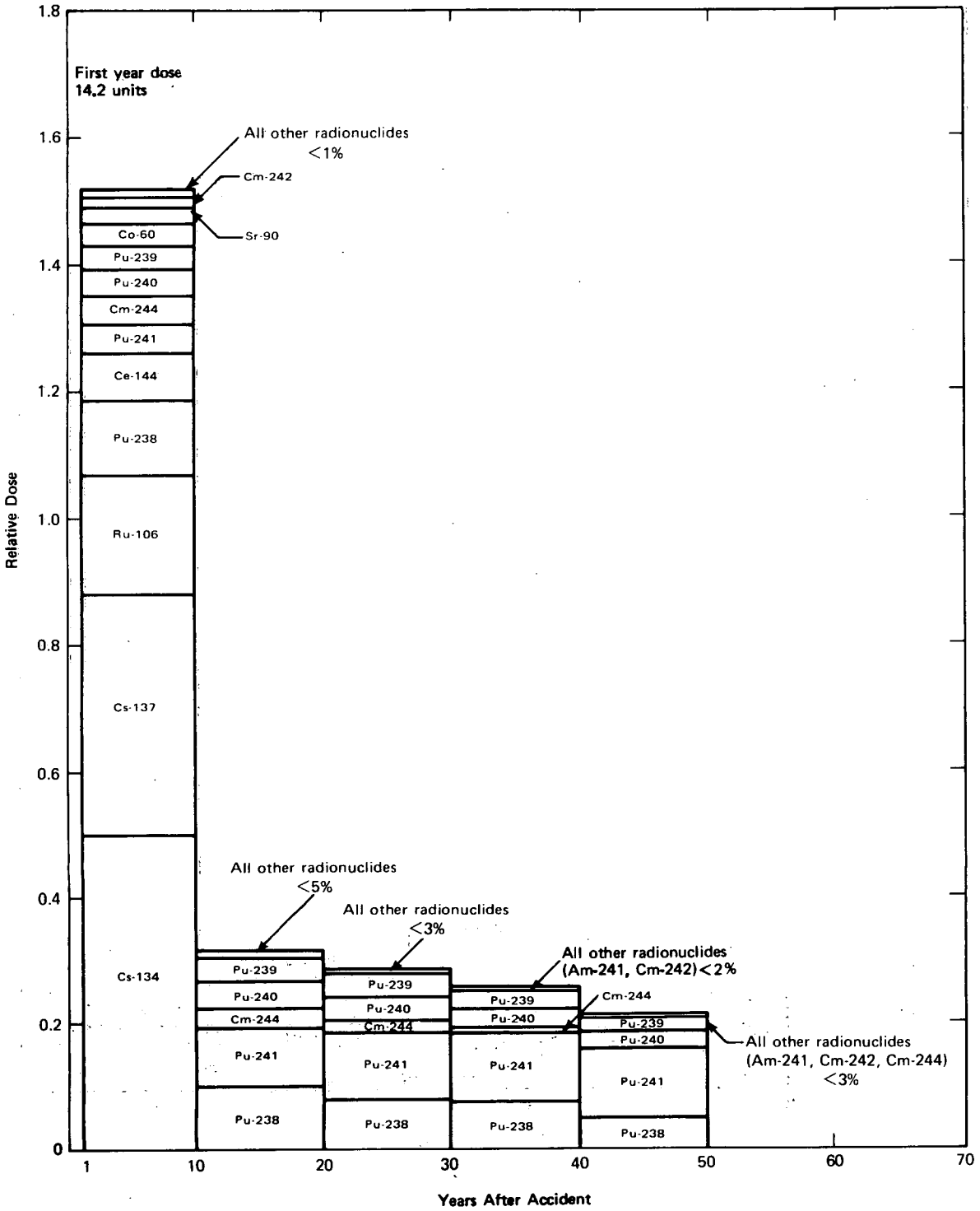


FIGURE VI 13-11 Relative incremental dose to breast from inhaled radionuclides at 10 miles from reactor.

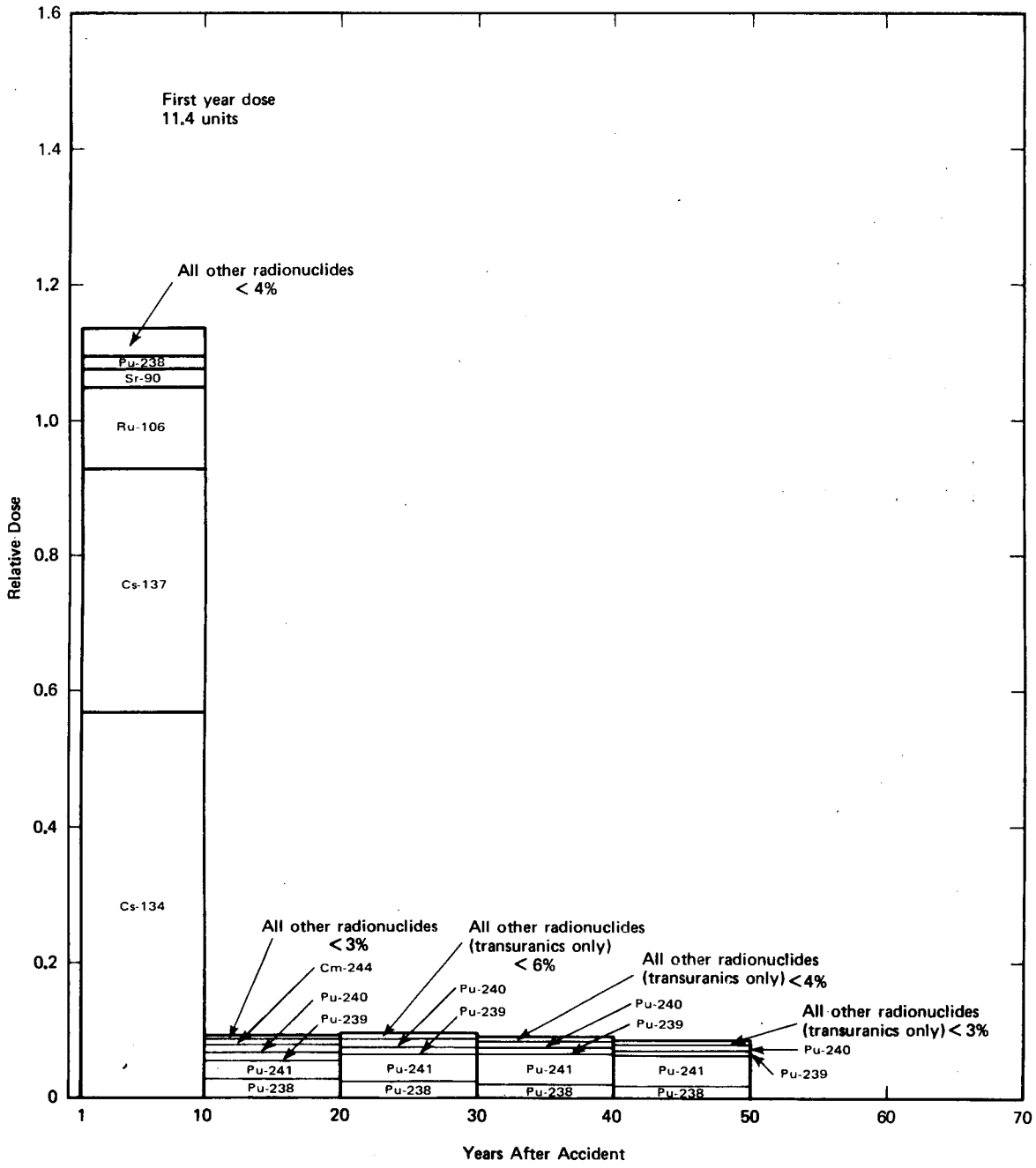


FIGURE VI 13-12 Relative incremental dose to testes from inhaled radionuclides at 10 miles from reactor.

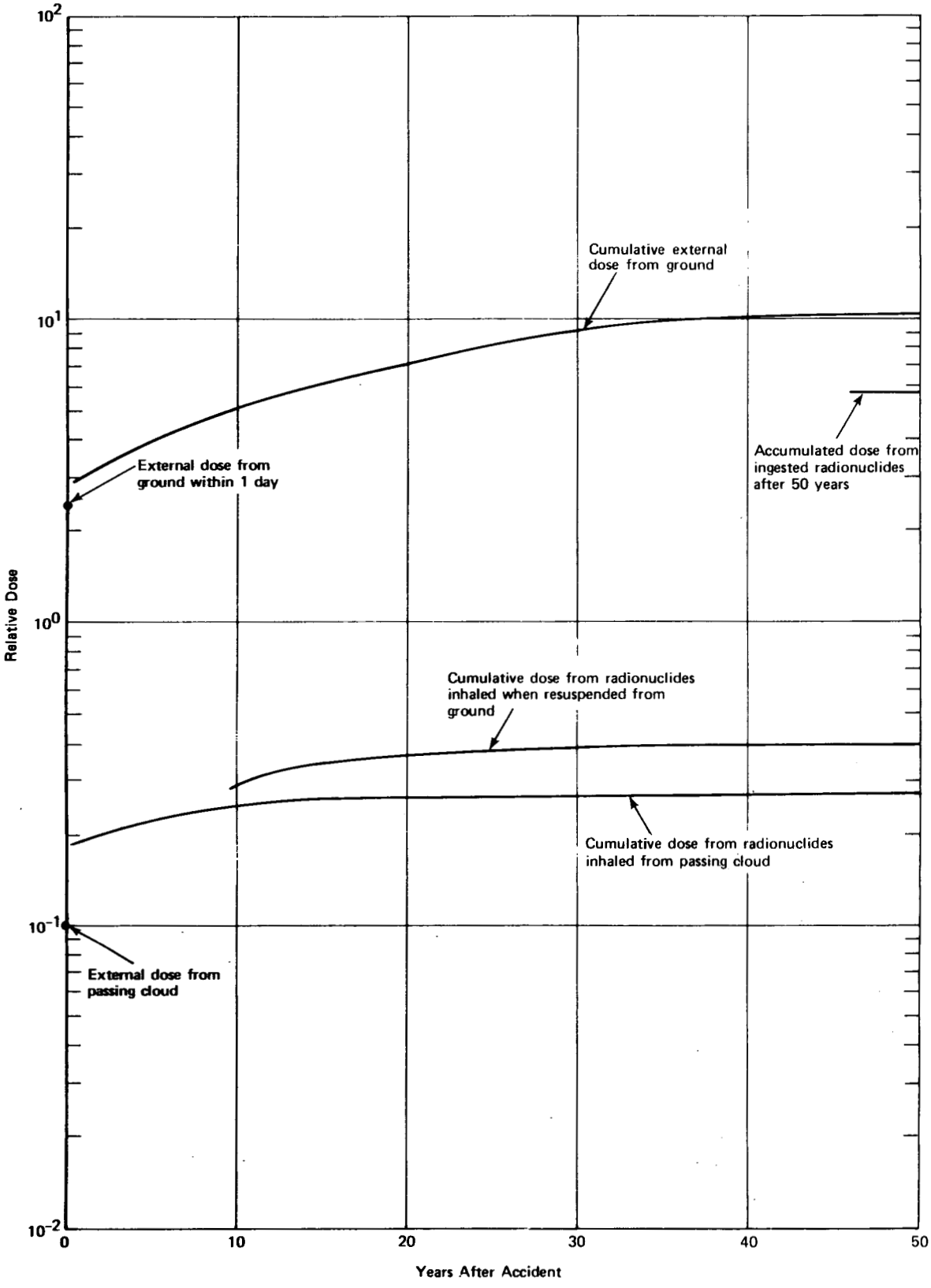


FIGURE VI 13-13 Relative doses to bone marrow at 100 miles from reactor.

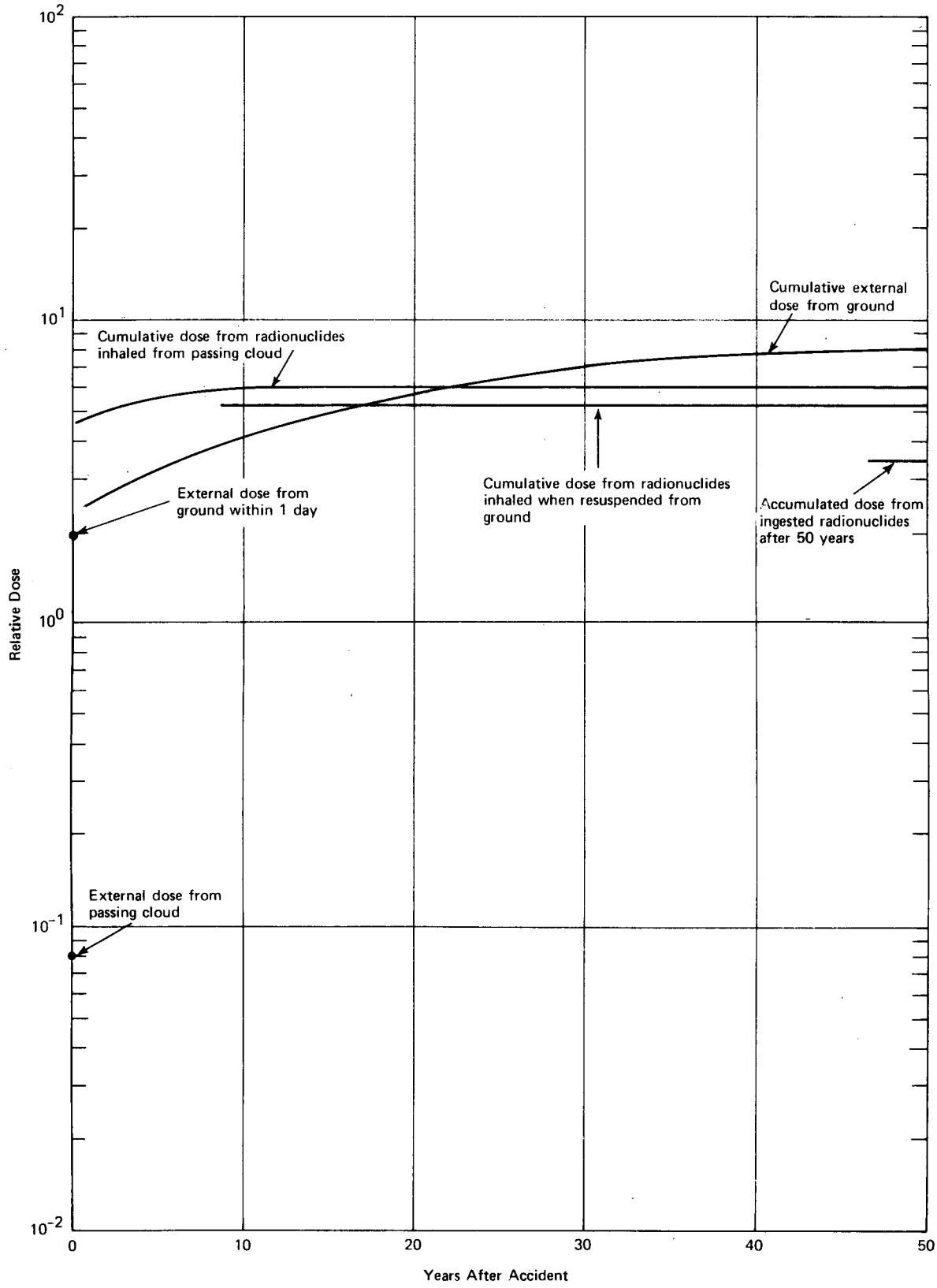


FIGURE VI 13-14 Relative doses to lung at 100 miles from reactor.

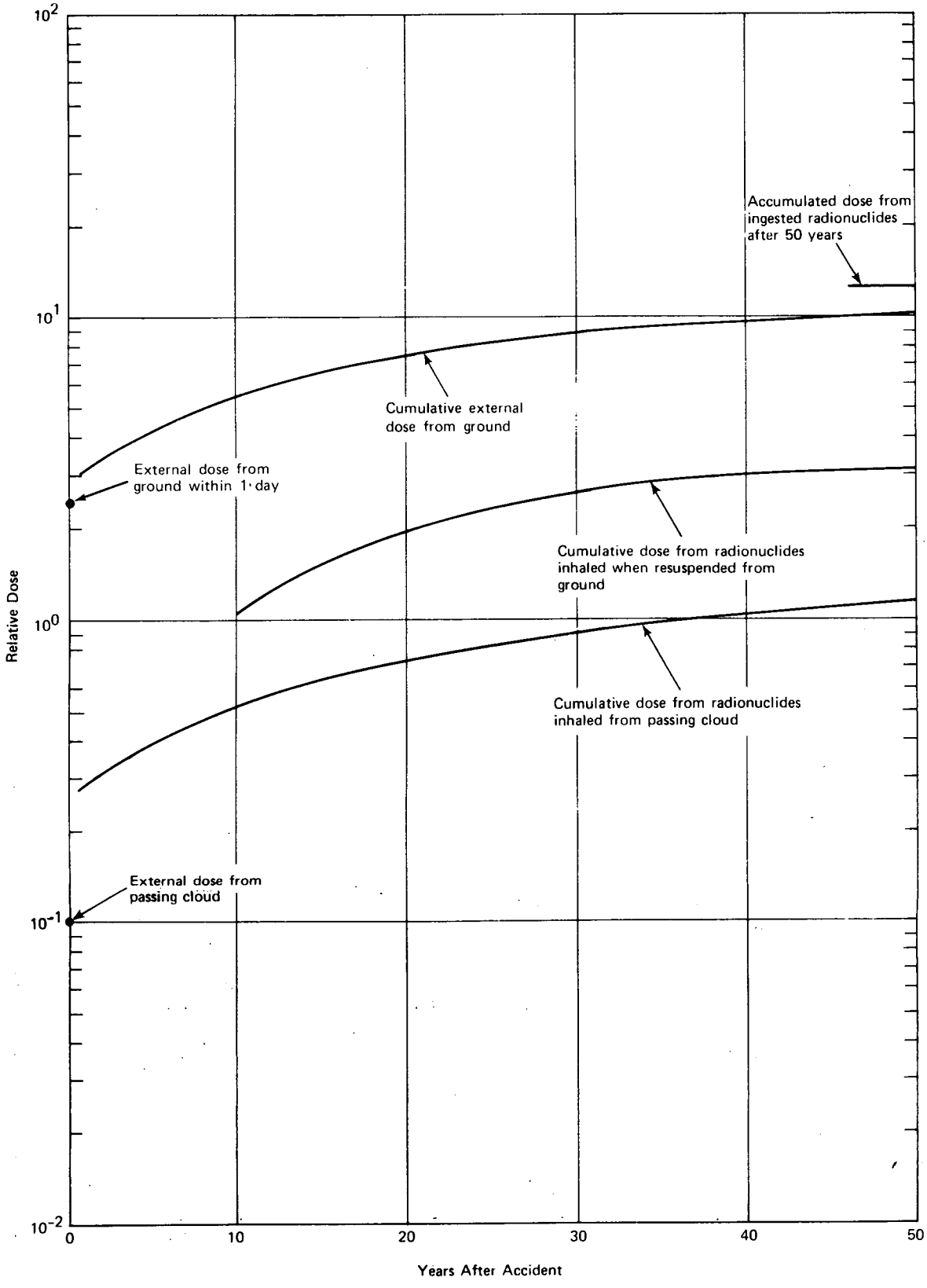


FIGURE VI 13-15 Relative doses to mineral bone at 100 miles from reactor.

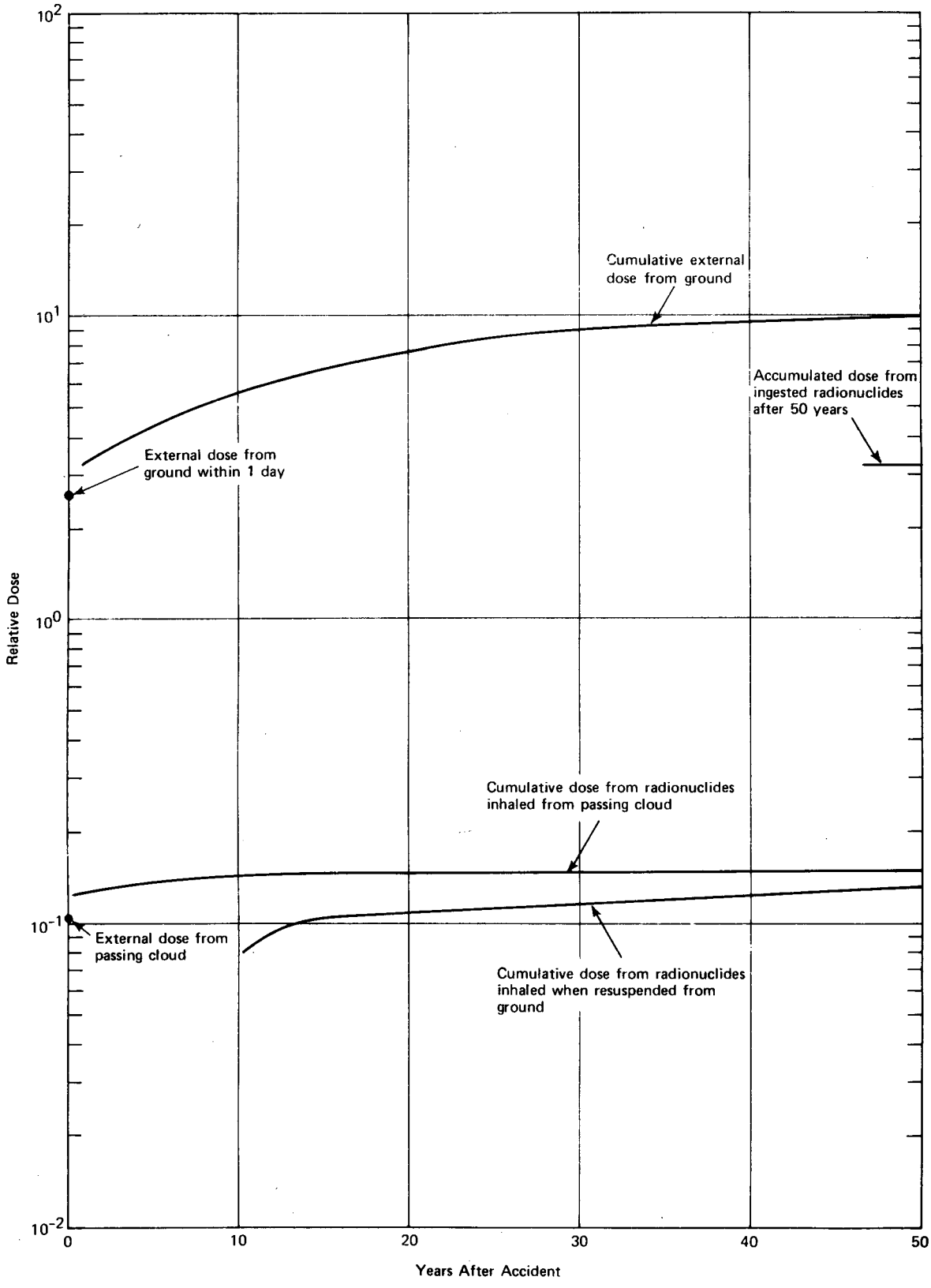


FIGURE VI 13-16 Relative doses to breast at 100 miles from reactor.

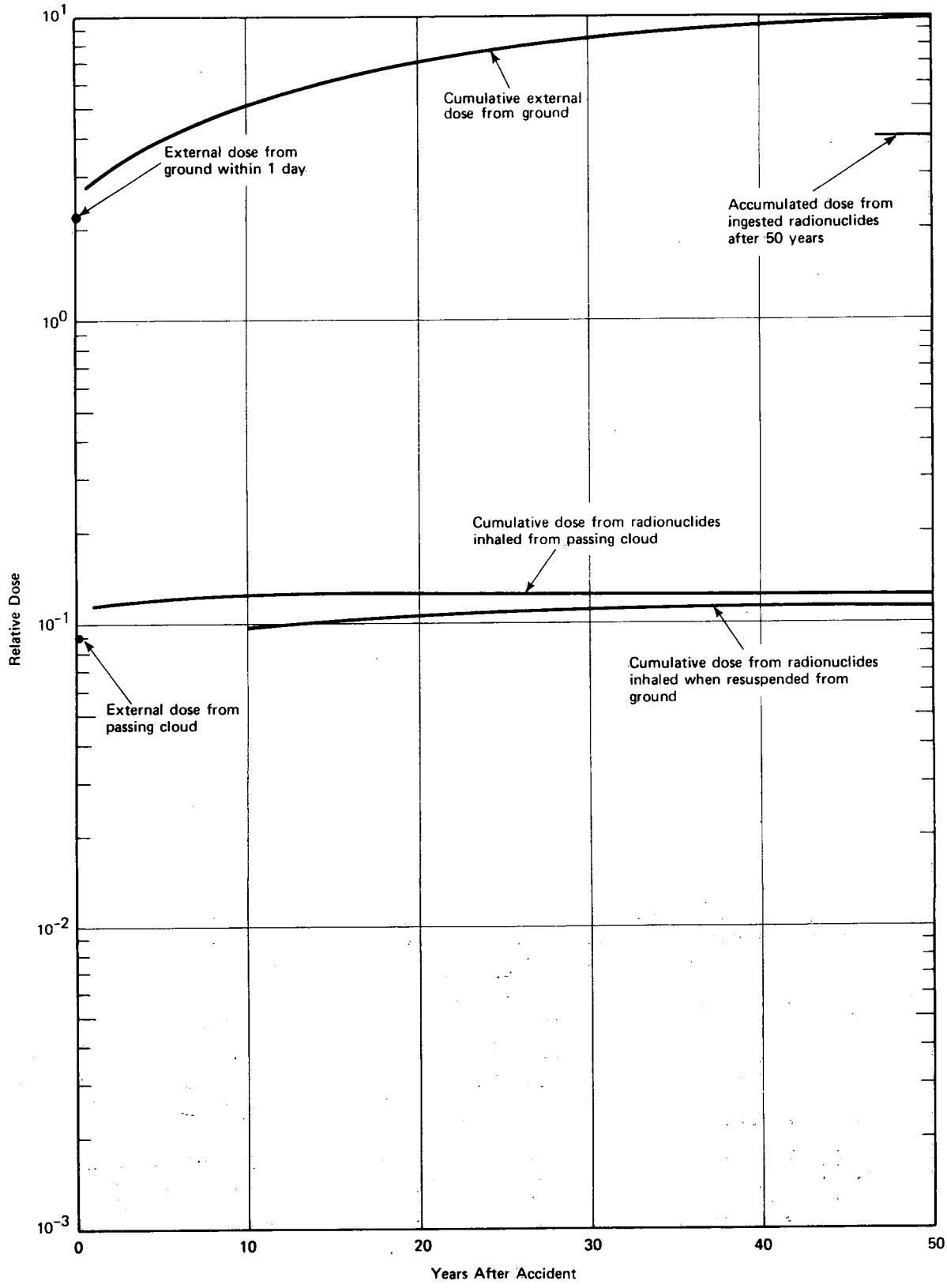


FIGURE VI 13-17 Relative doses to testes at 100 miles from reactor.

grains, or milk imported from other geographical locations. In today's mobile society in which foodstuffs are shipped thousands of miles, these assumptions are clearly conservative. As discussed in section 11.2.2, interdiction criteria are based on doses received to the whole body (bone-marrow) or gonads over a 30-year period. It is evident in Figs. VI 13-13 et seq. that external exposure from contaminated ground is the controlling exposure mode. Furthermore, at distances at which interdiction occurs, the doses received either external from the passing cloud or internal from inhaled radionuclides are small compared to that received from the contaminated ground over 30 years so that only the latter dose need be considered in establishing the interdicted area.

Each of the 54 radionuclides used in this study can be characterized by its contribution to the doses to various organs in various time periods. Table VI 13-1 shows such a breakdown. The doses considered are the doses from inhaled radionuclides to the bone marrow, lung, gastrointestinal tract, mineral bone, thyroid, "other tissues," and testes as well as those due to external exposure from the ground and from the passing cloud. Both early and late contributions to health effects are noted. A scale from zero to two is established; on which 2 indicates that the radionuclide contributed significantly to the specified dose and 1 indicates a small contribution. By summing these values for each radionuclide, a very crude ranking of the radionuclides is obtained. From this ranking, the radionuclides in the release that make little contribution can be identified.[1] They are listed in Table VI 13-2. From this table, it is seen that one-third of the released radionuclides used in this study could be neglected with small loss of accuracy, but with substantial reduction in the computation time of the consequence model.

TABLE VI 13-2 RELEASED RADIONUCLIDES CONTRIBUTING LITTLE TO HEALTH EFFECTS

Y-90	Kr-85m	Co-58	Rh-105	Te-129	Nd-147
Nb-95	Kr-87	Co-60	Ru-105	Ce-143	Am-241
Tc-99m	Rb-86	K-85	Te-127	Pr-143	

In the event of a reactor accident, the spectrum of doses received by the exposed population is very broad. As noted in section 9, the models for several health effects are influenced by the relative numbers of people receiving large or small doses. Figs. VI 13-18 and VI 13-19 give the frequency distribution of the number of people versus dose received to the bone marrow within 50 years and to the thyroid within 30 days respectively. The numbers of people are the mean values of 90 trials from one meteorological data set, assuming a large release, 100 people/mile^2 and an interdiction criterion of 25 rem in 30 years. The reader should note that the histograms are plotted with nonuniform increments on the abscissa. The use of equal increments would more readily illustrate the skewness of the distributions towards low doses. The population dose versus the 50-year bone marrow dose is also plotted in Fig. VI 13-18. From this histogram, the percentages of the total whole-body man-rem associated with individuals receiving in excess of 10, 30 and 50 rem are 75, 60 and 13% respectively. From similar calculations, the approximate percentages of latent cancer fatalities attributable to each exposure mode are stated in Table VI-13-3, both on a whole-body and organ-by-organ basis.[2] It is evident that lung cancer due to inhalation of radioactive material in the passing cloud is the dominant contribution to the total latent cancer fatalities. Furthermore, this domination underscores the importance of calculating latent cancer fatalities due to an accident on an organ-by-organ basis.

[1] A released radionuclide is important if it contributes significantly to a health effect and/or its radioactive daughters contribute significantly. For example, the released quantity of Am-241 is unimportant but that quantity which results from the decay of Pu-241 is important. The dose from the decay of Pu-241 is unimportant.

[2] The contribution of each exposure mode is estimated by setting its conversion factors to zero and subtracting corresponding latent fatalities from the total. This method is only approximate since the central estimate is based upon a nonlinear model.

TABLE VI 13-1 IMPORTANCE OF VARIOUS RADIONUCLIDES FOR HEALTH EFFECTS CALCULATIONS[a]

Released radionuclide	Contributions to Early and Continuing Health Effects							Contribution to Late Health Effects (0-50Y)						Σ
			Inhalation						Inhalation					
	Cloud Dose	Short-Term Ground Dose	Bone Marrow (30 days)	Lung (365 days)	GI (7 days)	Thyroid (30 days)	Testes (60 days)	Ground	Bone Marrow	Lung	Mineral Bone	Other	Testes	
Co-58														
Co-60														
Kr-85														
Kr-85m														
Kr-87														
Kr-88	2													2
Rb-86														
Sr-89			2		1		1		2		2	1	1	10
Sr-90			1						1		2			4
Sr-91		1			1									2
Y-90														
Y-91			1	1	1				1	1	1			6
Zr-95				1						1				2
Zr-97					1									1
Nb-95														
Mo-99				1	1					1				3
Tc-99m														
Ru-103		1	1	1	1			1	1	1		1		8
Ru-105														
Ru-106				2	2			1		2		1		8
Rh-105														
Te-127														
Te-127m				1						1				2
Te-129														
Te-129m			1	1	1				1	1	1			6
Te-131m	1	1	1	1	1				1	1				7
Te-132	1	2	2	2	2	1	2		2	2	2	2	2	22
Sb-127				1	1					1				3
Sb-129	1	1												2
I-131	1	2	1	1		2	1		1	1	1	1	1	13
I-132	2	2	1	1			1		1	1		1	1	11
I-133	2	2	1	1	1	1	1		1	1		1	1	13
I-134	1													1
I-135	2	2	1	1	1	1	1		1	1		1	1	13
Xe-133	1													1
Xe-135	1													1
Cs-134			2	1			2	2	2	1	2	2	2	16
Cs-136			1				1		1			1	1	5
Cs-137			1				1	2	1		1	2	2	10

TABLE VI 13-1 (Continued)

Released radionuclide	Contributions to Early and Continuing Health Effects							Contribution to Late Health Effects (0-50Y)						Σ
			Inhalation						Inhalation					
	Cloud Dose	Short-Term Ground Dose	Bone Marrow (30 days)	Lung (365 days)	GI (7 days)	Thyroid (30 days)	Testes (60 days)	Ground	Bone Marrow	Lung	Mineral Bone	Other	Testes	
Ba-140		1	2		2		1		2		1	1	1	11
La-140	1	1			1									3
Ce-141				1						1				2
Ce-143														
Ce-144				2	1					2				5
Pr-143														
Nd-147														
Np-239				1	1					1				3
Pu-238											1			1
Pu-239											1			1
Pu-240											1			1
Pu-241											1			1
Am-241														
Cm-242				1						1	1 (b)			3
Cm-244											1			1

(a) Key: 1 = small but important contribution to total dose; 2 = substantial contribution to total dose.

(b) Cm-242 contributes significantly within the first year to the mineral bone dose.

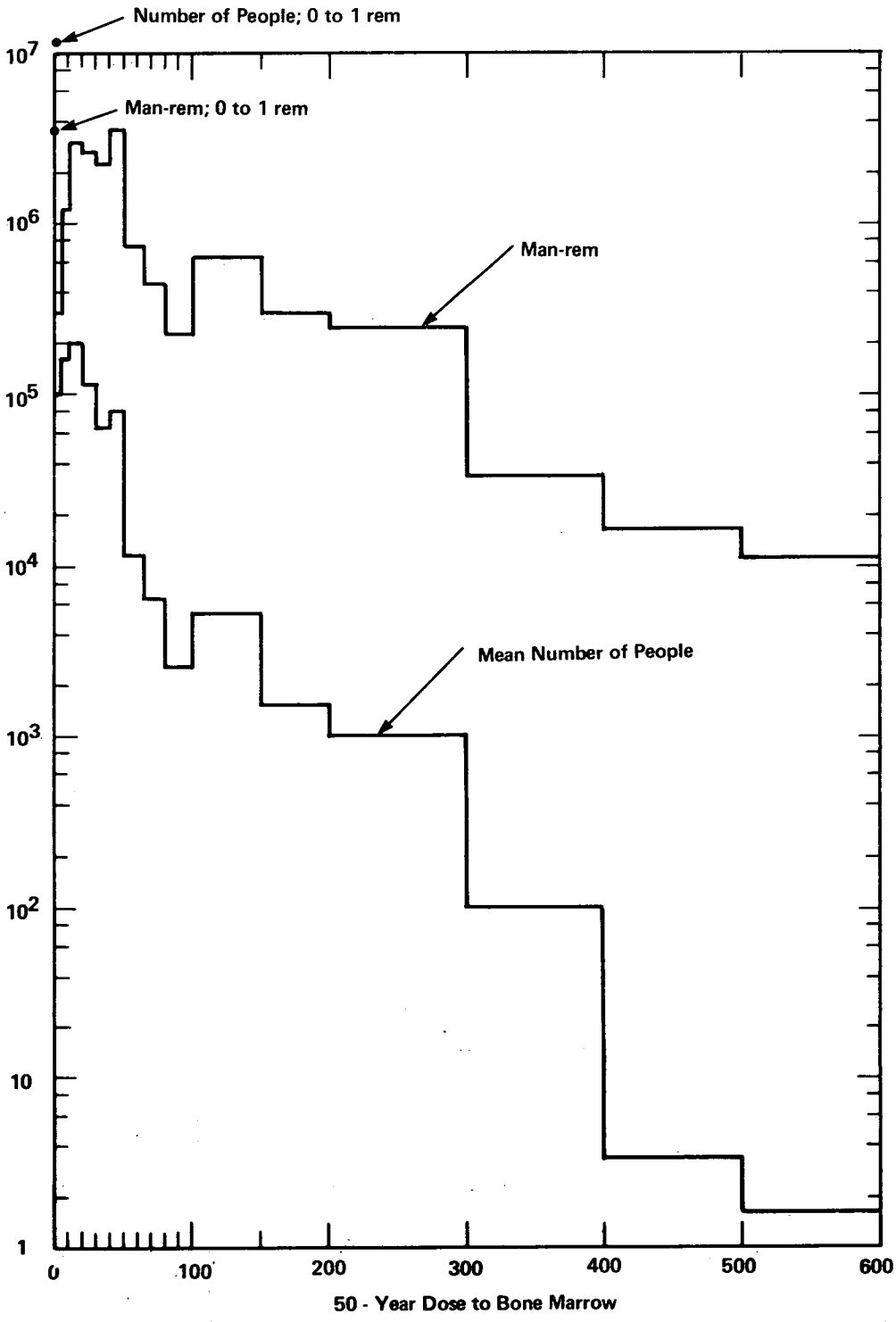


FIGURE VI 13-18 Frequency distribution of number of people and population dose versus bone marrow dose within 50 years (PWR-1B release, 100 people/mile^2)

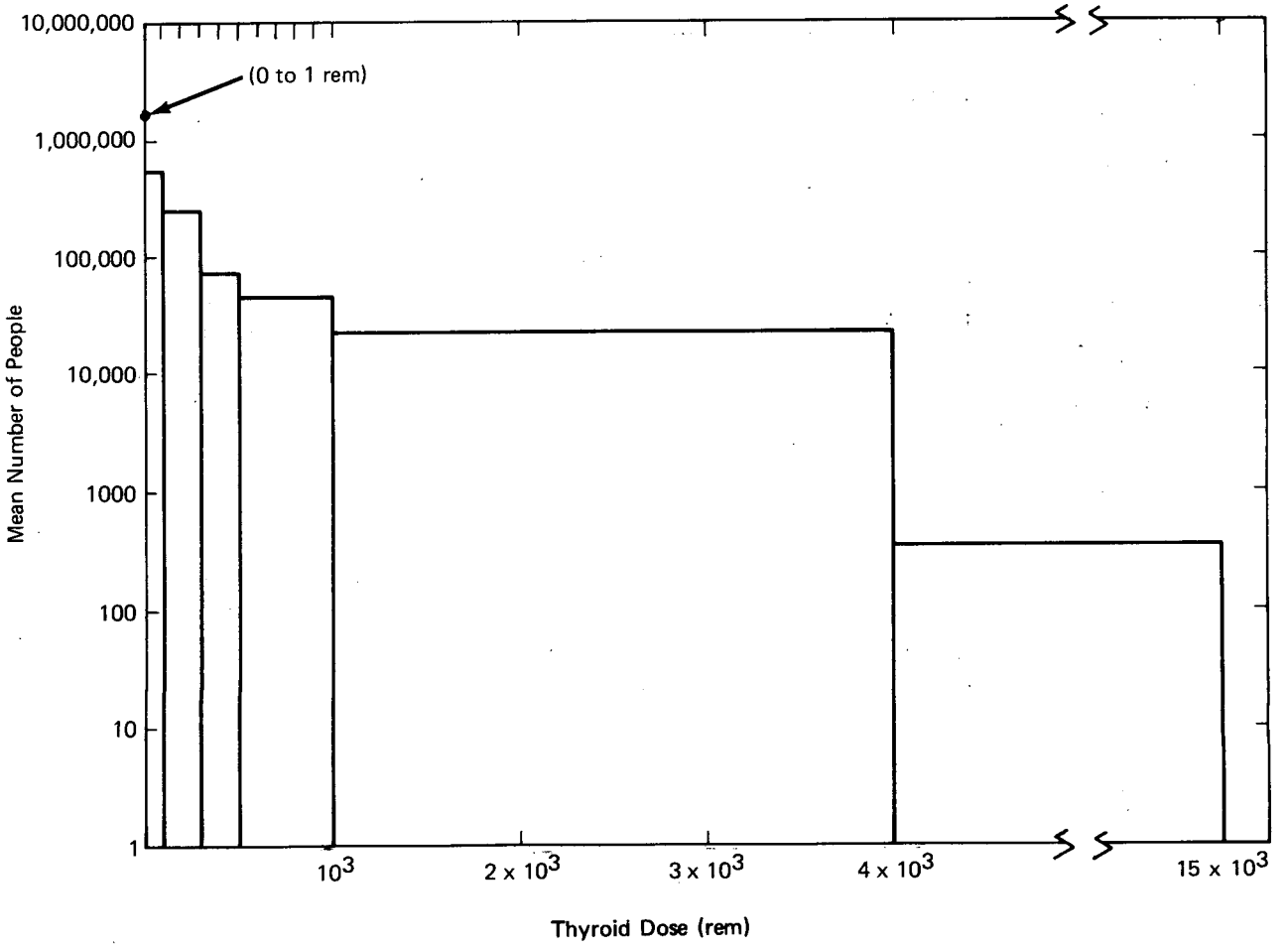


FIGURE VI 13-19 Frequency distribution of number of people versus thyroid dose within 30 days (PWR-1B release).

TABLE VI 13-3 CONTRIBUTION OF DIFFERENT EXPOSURE MODES TO LATENT CANCER FATALITIES

	Percentages							
	Leukemia	Lung	Breast	Bone	GI Tract	All Other	Total	Whole Body (a)
External cloud	0.2	0.5	0.5	0.1	0.1	0.3	1	3
Inhalation from cloud	0.5	59.0	10.0	0.2	1.0	0.2	71	15
External ground (<7 days)	4.0	8.0	8.0	1.0	1.0	3.0	25	47
External ground (>7 days)	2.0	2.0	6.0	1.0	1.0	2.0	13	30
Inhalation of resuspended contamination	0.1	3.0	0.1	0.1	0.1	0	3	2
Injestion of contaminated foods	0.2	0.2	0.5	0.1	0.1	0.2	1	4
Subtotals	7	66	16	2	3	6	100	100

(a) Whole body values are proportional to 50-year whole-body man-rem.

Section 9.3 discusses different calculation methods for the latent cancer fatalities; whole body versus organ-by-organ and upper bound versus central estimate. Table VI 13-4 shows the effective incidence by the four methods. When using the whole-body dose and BEIR (upper bound) values, the incidence is 121.6 per 10^6 man-rem as stated in Table VI 9-4. On an organ-by-organ basis, this incidence is increased to 190 due to the preferential dose to the lung. Introduction of the dose-effectiveness factors reduces both these incidences but has less effect on the organ-by-organ value than on the whole body value since lung cancers are the dominant contribution and the lung dose is usually greater than 25 rem. Mean values are given in Table VI 13-4. For the largest calculated accidents, values on an organ-by-organ basis are somewhat higher and the dose-effectiveness factors have less influence.

TABLE VI 13-4 EXPECTED CASES OF LATENT CANCER FATALITIES PER MILLION MAN-REM

Method	Upper Bound (BEIR)	Central Estimate
Whole body	122	48
Sum of individual organs	190	100

13.2 SAMPLING

The atmospheric dispersion of the radioactive material depends on the weather over a period of 10 to 30 hours. The magnitudes of specified consequences are determined by the interaction of this atmospheric dispersion with the distribution of population which varies with distance and direction from the reactor. Clearly, there is an almost infinite number of combinations of weather and population, each leading to an unique set of consequences. Such a problem is not amenable to closed-form solution. A common approach to such a problem is to sample the underlying distributions a finite number of times in such a way that the true distribution of consequences is closely approximated. This latter approach is incorporated into the consequence model. This section describes the sampling method and presents some analyses that establish its adequacy.

13.2.1 SAMPLING METHODS

Sampling methods are particularly suitable to computer techniques and there is a large literature on choosing a sampling method and determining its adequacy (Hammersley and Handscomb, 1964; Kahn, 1957; Kempthorne, 1952). The available meteorological data are hourly readings of stability, wind speed and direction and precipitation at a reactor site over a period of one or more years. Three sampling methods were considered for the consequence model. The most obvious method is a random sampling from these 8760 readings (one year's worth). That is, a starting hour, when the radioactive material is assumed to be released, is randomly selected and, by using this and the succeeding 20 or so hours of weather readings, the resultant atmospheric dispersion is calculated. By repeating this process for 100 or more random trials, a population (in a statistical sense) of radioactive plumes is generated which may be used in conjunction with the population (people) distributions to calculate a frequency distribution for each consequence.

The above sampling technique is simple but rather inefficient for the following reason. Meteorology is a cyclical process. A 24-hour day has a distinct cycle in which the heat of the sun during the day causes greater instability and a higher mixing layer than during the night. This diurnal cycle is superimposed on a seasonal cycle. An adequate sampling of weather must include an appropriate frequency of samples from each diurnal and seasonal period which requires a large number when the samples are completely random. A more sophisticated approach is called stratified random sampling. In this method, the random starting times are stratified by day and night and by month. For example, the weather data for each month is randomly sampled eight times, four samples from night periods and four from day periods. The 96 stratified samples will give a better representation of the underlying population than 96 completely random samples. It was found that the statistical noise from even this method was unacceptable.

A third sampling method, which was adopted for the consequence model, uses completely stratified samples, i.e., no random selection. It has been observed (Van der Hoven, 1957) that meteorological phenomena have a broad spectrum of fluctuations in the variance of the wind-speed which appear to have a four-day cycle. In order to ensure complete coverage of diurnal, seasonal and four-day cycles, starting times are selected every four days plus one hour. In this manner, each hour of the day is represented in 24 samples. Ninety samples are available from one year's worth of data. To obtain 45 or 180 samples, the samples would be selected each 8 days plus 13 hours and each 2 days plus 13 hours, respectively. This sampling method provides as complete coverage as the second method without the statistical noise from the random numbers.

Let us consider the probabilities associated with this last method. From each regional meteorological data set described in section 5, 90 weather samples are selected as described above. For each release category stated in Table VI 2-1, each of these 90 weather samples is used to calculate isopleths for airborne radioactive material and for ground contamination. For corresponding pairs of meterological data sets and composite demographic sites (see section 10), the interaction of each of these 90 isopleths with each of the 16 sectors is calculated. Thus, for the 10 PWR release categories, 10 x 16 x 90 or 14,400 trials are calculated for each composite site. One of these trials will result in the largest consequence.[1] What is the probability associated with this trial? The largest consequence always results (a) from the largest releases, i.e., either PWR-1A or PWR-1B, whose probabilities are about 5×10^{-7} per reactor-year (see Table VI 2-1) and (b) from the sector with the largest population whose probability at site 1 is 1/224 (see Table VI 10-3). Therefore, the probability associated with the largest consequence is:

$$(5 \times 10^{-7})(1/224)(1/90) = 2.5 \times 10^{-11} \text{ per reactor-year.}$$

Since this site 1 is a composite for 14 reactors, it would be more reasonable to state that the probability of the largest calculated consequence is 3.5×10^{-10} per 14 reactor-years. Clearly, if 45 or 180 samples are used the above probabilities are doubled or halved. For other composite sites, representing a different number of reactors, a correspondingly different probability would be assigned to the sector with the largest population, and a corresponding change would be made in the above two statements of probability. As discussed in section 13.2.3, the uncertainty in probability due to sampling error increases with magnitude of consequence, and would be sufficiently large for the largest calculated consequence as to render its actual value almost meaningless. In order to reduce this sampling error, the complementary cumulative distribution functions are truncated at 10^{-7}/year for 100 reactors.

13.2.2 EVALUATION OF SAMPLING METHOD

How many samples are needed to adequately calculate the overall consequences? The number of samples is a compromise between computation time and accuracy, although there is a maximum number of samples that may be drawn without overworking the available data.

To investigate the sampling error, samples of different sizes were taken from the meteorological data and the statistics of the computed consequences were compared. For a given sample size, the mean, variance, and third and fourth moments were plotted for the probability density functions for each consequence parameter. If the number of

[1] The reader is cautioned that a given trial is unlikely to maximine each consequence. That is, one trial may result in maximum early fatalities but another may result in maximum property damage.

samples is sufficient to incorporate all pertinent variation in weather and its interaction with population, then a larger sample size should not give significantly different results. For example, if the sample size is large and representative enough to give an accurate estimate of the mean number of early fatalities, then a larger sample size would not alter the estimate of this mean. Any differences between means would indicate the presence of a sampling error due to different sample sizes and selections. The variance and the third and fourth moments characterize the shape of the probability density function (the third moment is a measure of its skewness and the fourth moment its kurtosis). The change in the variance and in the third and fourth moments with change in sample size will indicate the sufficiency of the sample in determining the tail behavior of the probability density function, i.e., the "peak" behavior.

As the sample size is increased, the sampling error should decrease so that a plot of any of the above statistics versus sample size should resemble the smooth curve in Fig. VI 13-20. In practice, the third sampling method described in section 13.2.1 results in disjoint samples (i.e., the 90-sample case does not include the starting times in the 45-sample case). The curve is therefore noisy and becomes asymptotic to the smooth curve only for large numbers of samples as illustrated in Fig. VI 13-20. The region in which the curve "stabilizes" (to right of line AA in Fig. VI 13-20) indicates the number of samples that is large enough for determination of the calculated statistic. Alternatively, in this stabilized region, the difference between the calculated statistic for different sample sizes is small compared to the accuracy required for the consequence model. The other types of errors inherent in the calculation will dominate the sampling errors in this stable (acceptable) region and hence the sample size denoted by AA is sufficiently large.

In order to assess the required number of samples, 45, 90 and 180 stratified samples were drawn from one set of meteorological data and the numbers for early fatalities and for property damage were calculated for a peak population section at one composite site.[1] The mean, variance and third and fourth moments for the early fatalities and property damage are shown in Figs. VI 13-21 and VI 13-22. The results show that the statistics for the early fatalities are more sensitive to the number of samples than are those for property damage. It is apparent that 45 is a sufficient number of stratified samples to calculate property damage but that 90 samples would be preferable for the calculation of early fatalities. Additional samples beyond these numbers are not warranted since the stated differences in statistics are within the accuracy of the consequence model. The basic calculations for 100 reactors, presented in section 13.4, are made by using 90 stratified samples. Many of the parametric studies were made with 45 stratified samples since, in general, these calculations are comparative and the same 45 samples are utilized. The statistics for 100 random samples are also shown in Figs. VI 13-21 and VI 13-22. Comparison of these statistics to the corresponding ones for stratified samples shows that this sampling method is less efficient.

13.2.3 UNCERTAINTY FOR SAMPLING

The complementary cumulative distribution functions for each consequence, presented in section 13.4, are calculated by sampling the meteorological data and by combining the resultant isopleths with a frequency distribution for population sectors. Since the curves are statistical estimates, standard confidence bounds can be calculated for them. For each curve, the magnitude of the confidence bounds will grow as the consequences increase, reflecting increasing uncertainty. The confidence bounds are calculated as factors which, for a specified consequence magnitude, multiply or divide the estimated probability for the upper and lower bounds respectively.

The complementary cumulative distribution functions are generated by ranking the consequences by their magnitude and assigning a probability to each magnitude based upon the probability of the release and the fraction of trials producing this magnitude. In this ranking, the r^{th} consequence magnitude is the r^{th} largest, i.e., there are (r-1) larger ones. By treating the calculations as a Poisson process, the upper 95% confidence factor for the r^{th} consequence magnitude is given by $(\chi^2_{0.95,(2r+2)})/2r$ where $\chi^2_{0.95,(2r+2)}$ is the 95th percentile of the chi-squared distribution with (2r+2) degrees of freedom. Similarly, the lower 5% confidence factor is $2r/(\chi^2_{0.05,(2r)})$. These factors are obtained from standard Poisson statistical treatments (Johnson and Katz, 1969).

[1] A similar calculation using a uniform population density shows less sensitivity to number of samples as would be expected.

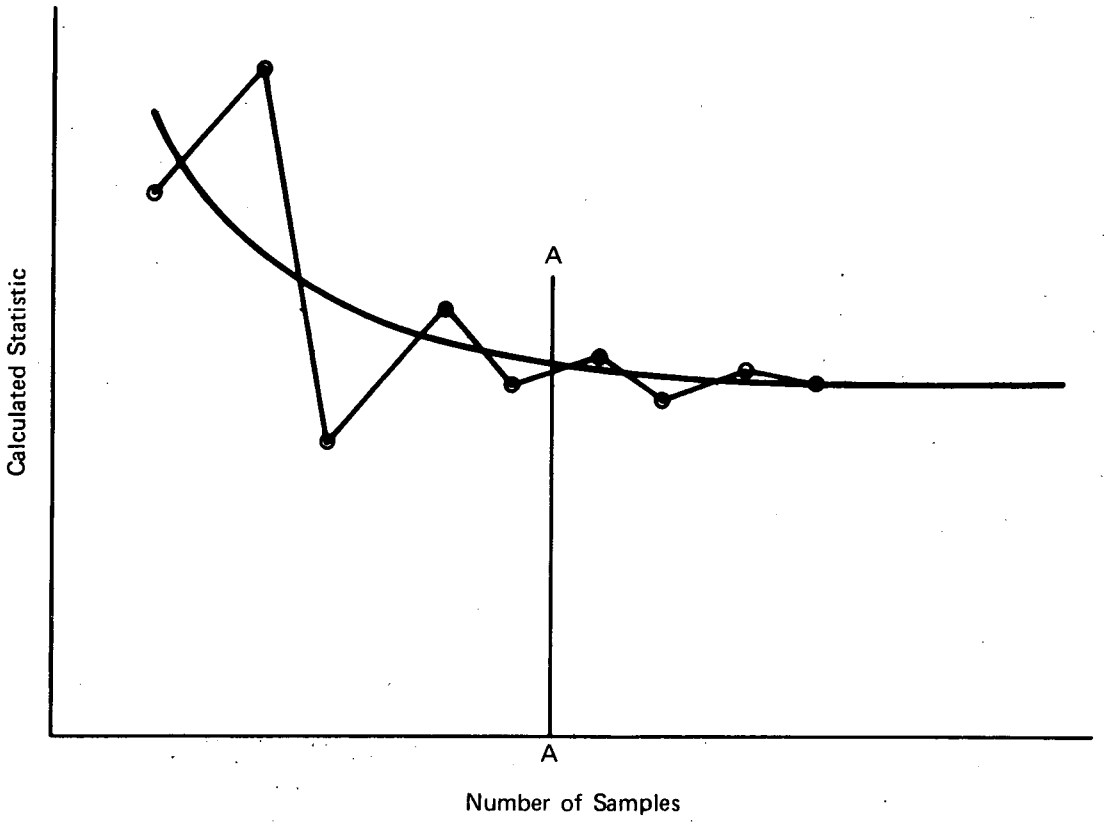


FIGURE VI 13-20 Typical calculated statistic versus number of samples.

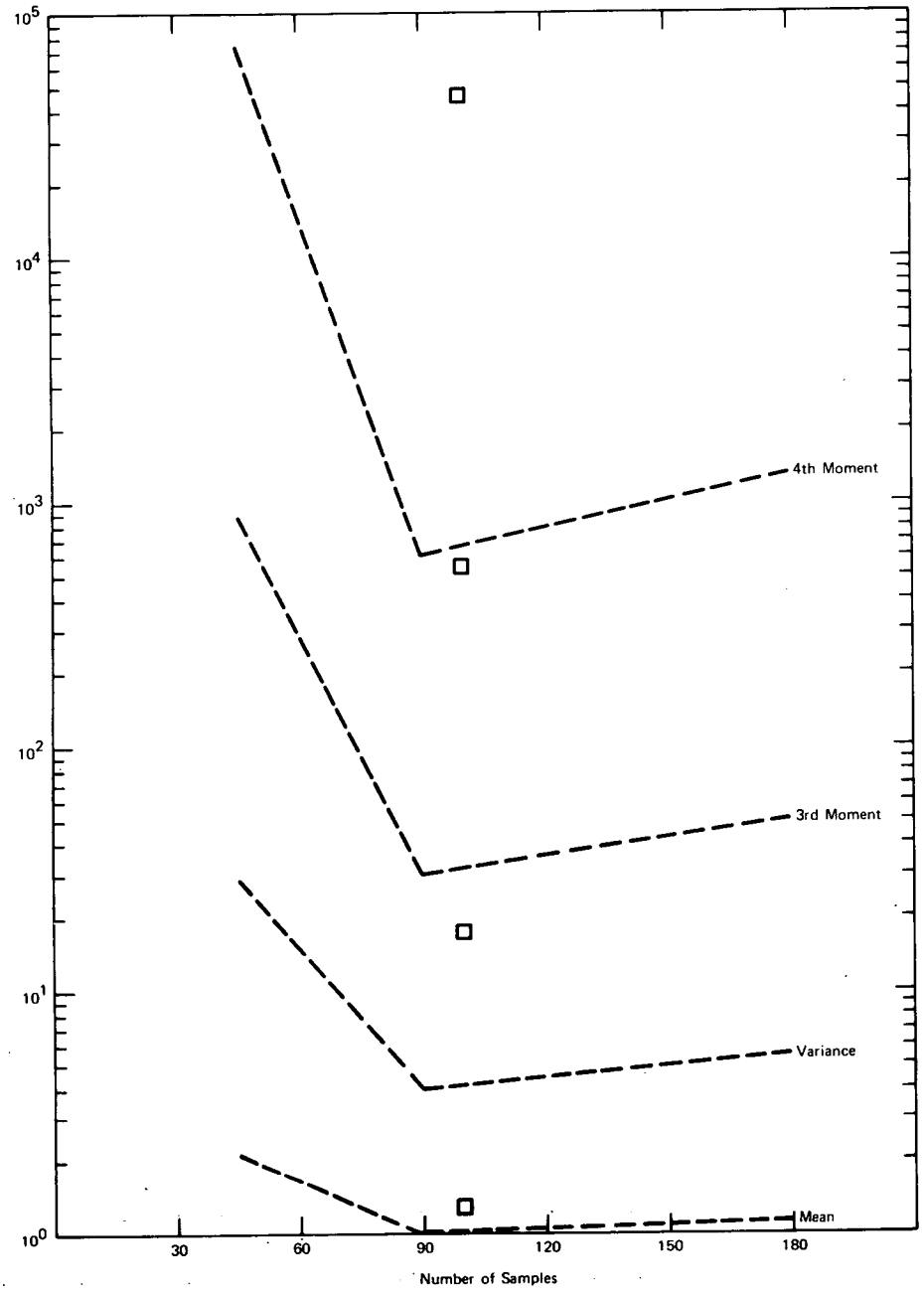


FIGURE VI 13-21 Relative statistics for early fatalities at a peak population sector. The squares indicate values for 100 random trials.

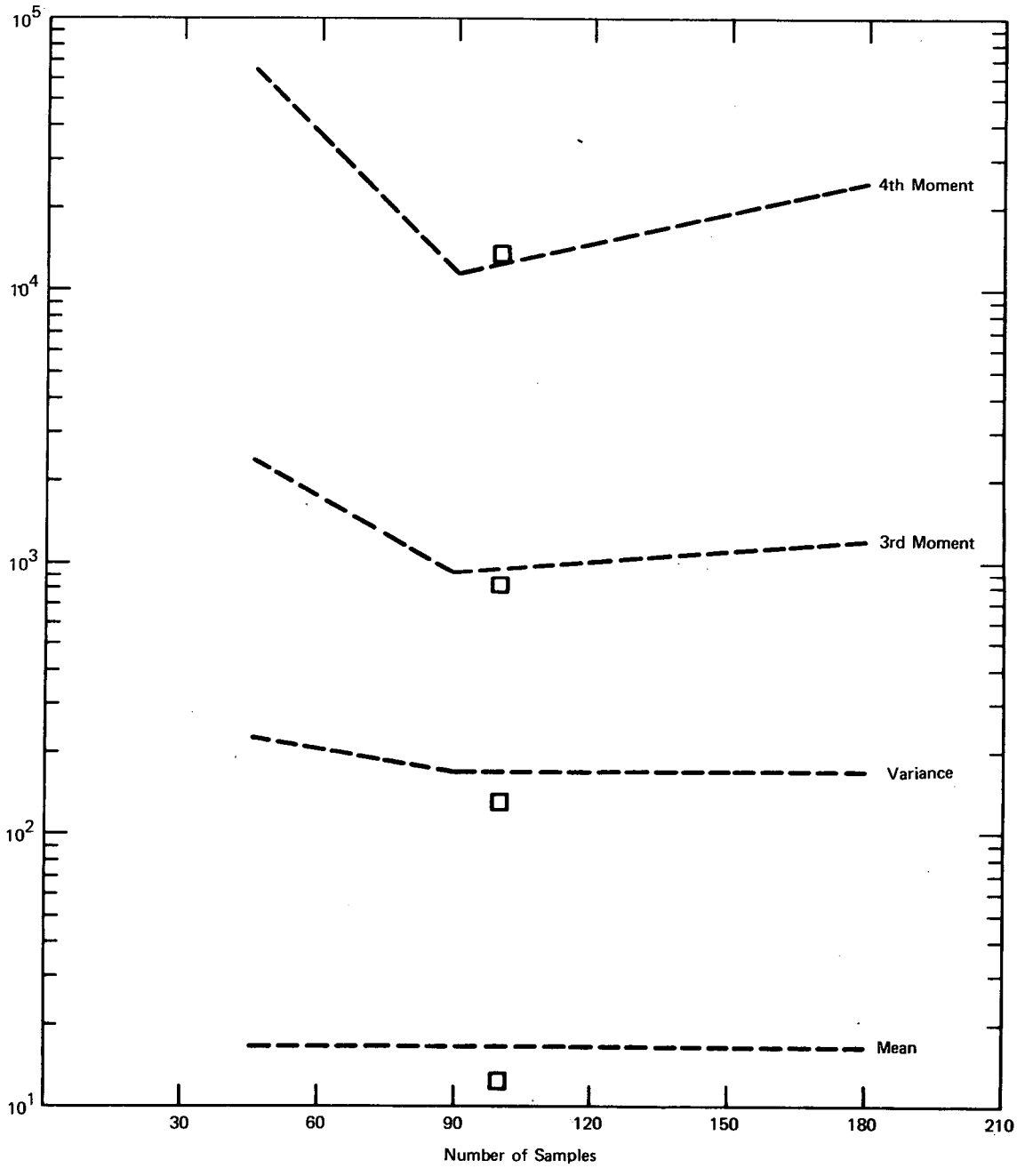


FIGURE VI 13-22 Relative statistics for property damage at a peak population sector. The squares indicate values for 100 random trials.

Table VI 13-5 presents representative values of the confidence factors as a function of the rank of the consequence magnitude. It is evident from Table VI 13-5 that the confidence bounds increase dramatically for the larger calculated values for which r is small. For the largest calculated values of a consequence (r=1), the upper bound on probability is a factor of 5 above the estimated value while the lower bound is a factor of 20 below. This large uncertainty in probability renders this calculated value almost meaningless. Accordingly, the complementary cumulative distribution functions are truncated at 10^{-9} per reactor-year which results in upper and lower bound confidence factors of about 2.5 on the largest stated value of a consequence. For the smallest value, having the lowest rank and the largest value of r, the confidence bound is effectively zero. It should be remembered that these confidence bounds only reflect uncertainty from sampling and not other uncertainties in the physical model and input parameters. These subjects are discussed in section 13.5.

TABLE VI 13-5 CONFIDENCE FACTORS FOR SAMPLING

r	95% Upper Bound[a]	5% Lower Bound[b]
1000	1.05	1.05
100	1.2	1.2
50	1.3	1.3
20	1.4	1.5
10	1.7	1.8
5	2.1	2.5
1	4.7	19.4

(a) Estimated probability should be multiplied by this value to obtain upper confidence bound.

(b) Estimated probability should be divided by this value to obtain lower confidence bound.

13.3 PARAMETRIC STUDIES

This section describes a series of parametric studies that show the sensitivity of the major consequences to certain input parameters. These studies are based on a uniform population density of 100 people per square mile. Eliminating the variation in population with radius and direction from the reactor permits a clearer understanding of the impact of other parameters on results.

13.3.1 EARLY FATALITIES

The differences between the meteorological data sets would be expected to affect all consequences. Since latent cancer fatalities and property damage are consequences that are integrated over large areas and over long distances from the reactor, they would be expected to be less sensitive to weather than would be the early fatalities (which would occur mostly within 10 miles of the reactor). The most sensitive yardstick of weather variation therefore would be the early fatalities.

Figs. VI 13-23 and VI 13-24 show the conditional probability of early death for an individual as a function of distance from a reactor given the PWR-1A and PWR-1B releases, respectively. Since PWR-1A is a relatively cool release, the cloud of radioactive material

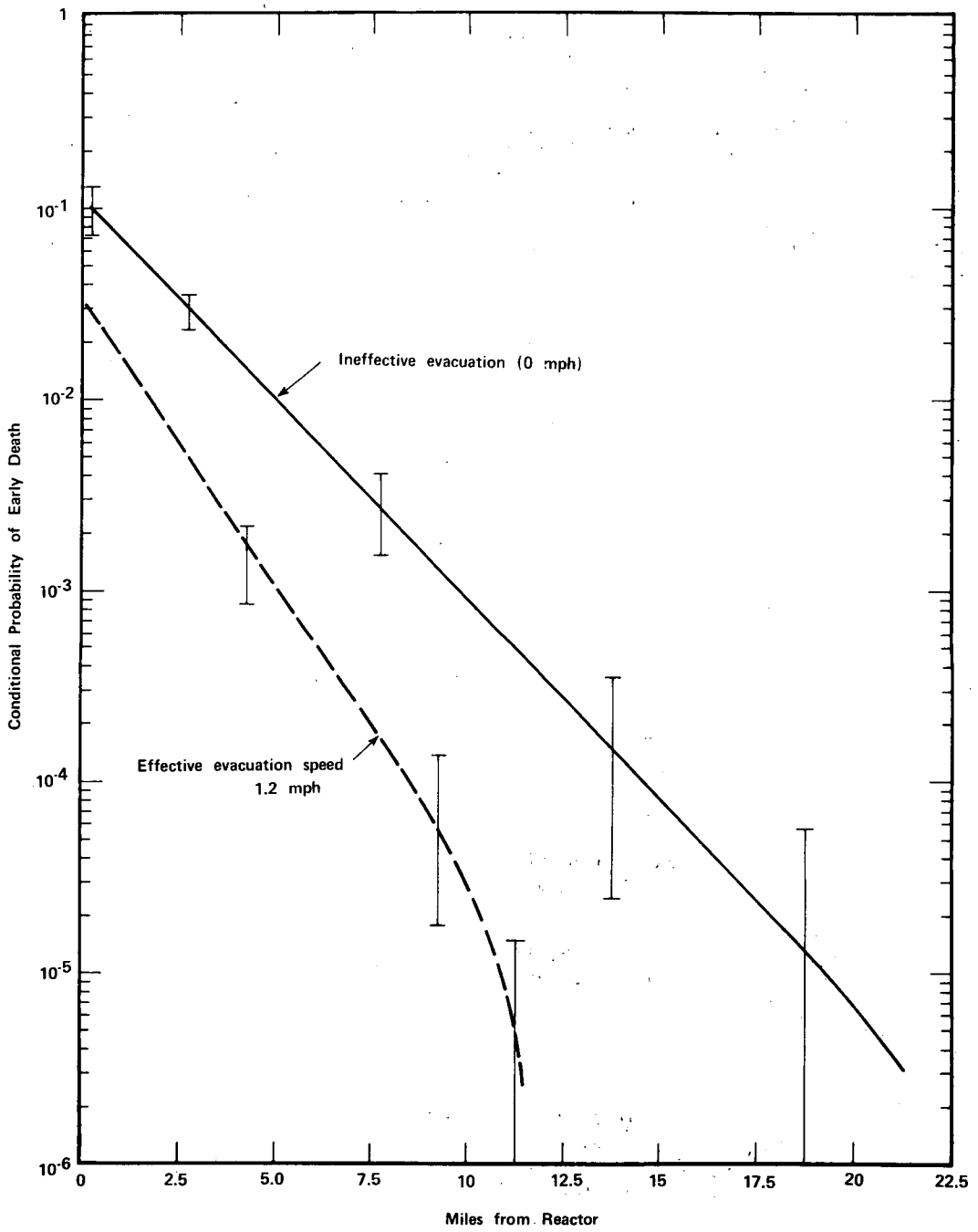


FIGURE VI 13-23 Conditional probability of early death as a function of distance from reactor for three effective evacuation speeds given a PWR-1A release.

(a) Approximately, absolute mortality probabilities are 10^{-6} per reactor year times quoted values.
(b) The error bars denote the variation in the mean values for the six meteorological data sets.
(c) For effective evacuation speeds of 4.7 and 7 mph, the conditional probability of early death is effectively zero within 25 miles.

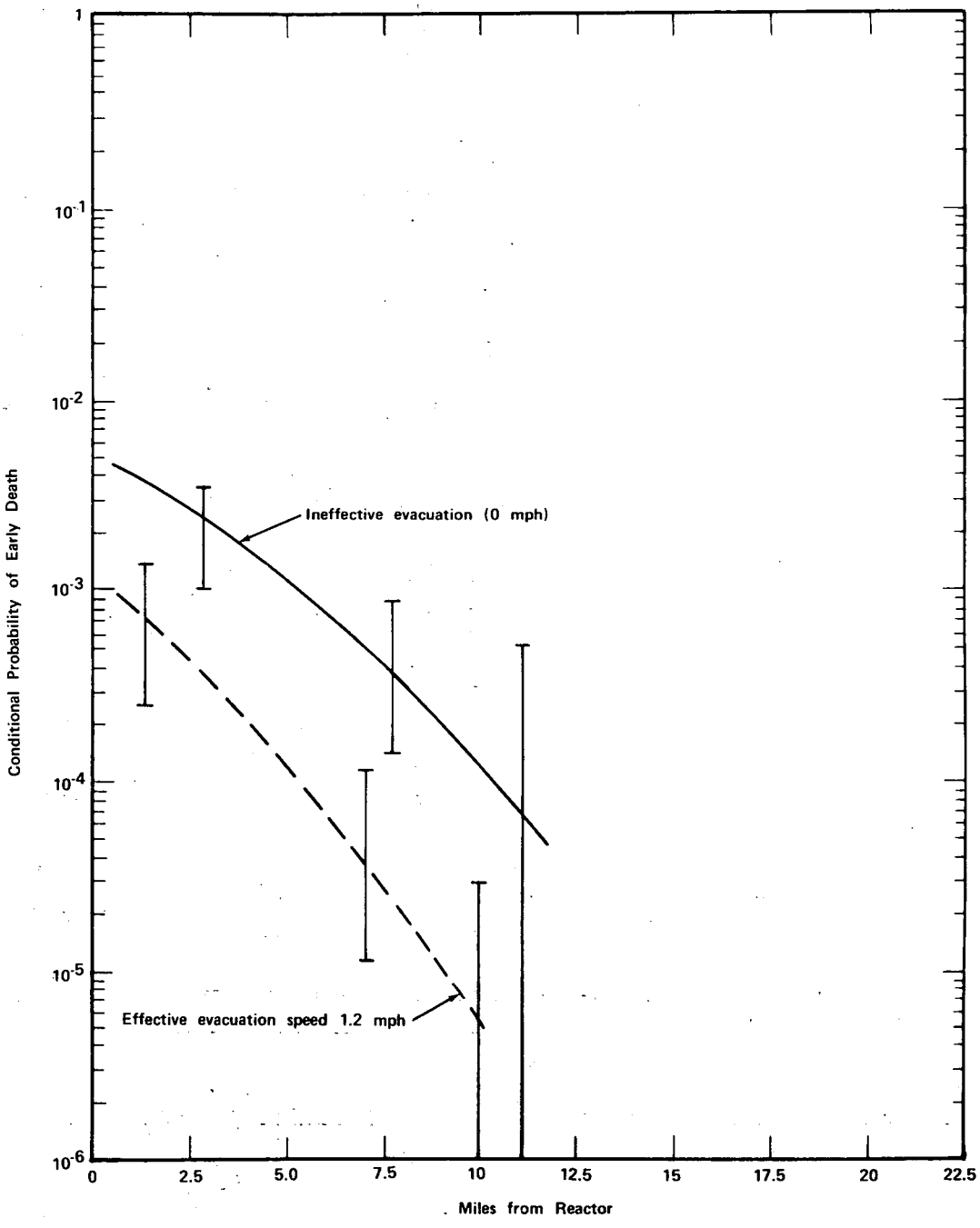


FIGURE VI 13-24 Conditional probability of early death as a function of distance from reactor for three effective evacuation speeds given a PWR-1B release.

(a) Approximately, absolute mortality probabilities are 10^{-6} per reactor year times quoted values.
(b) The error bars denote the variation in the mean values for the six meteorological data sets.
(c) For effective evacuation speeds of 4.7 and 7 mph, the conditional probability of early death is effectively zero with 25 miles.

reaches ground level almost immediately. As it depletes by deposition and disperses, the ground level concentration decreases and the probability of death therefore decreases with distance. In contrast, PWR-1B is a hot release, and the cloud does not reach the ground until 5 to 10 miles from the reactor, by which time the cloud concentration is relatively low. The net effect is that for a hot release the probability of death is about an order of magnitude lower than that for a cool release.

Figs. VI 13-23 and VI 13-24 show the mean probability, obtained with 45 trials from each of the six meteorological data sets. The variation of the six means from the six weathers is indicated by the error bars. For the cool release (PWR 1A) and, within 15 miles, for the hot release (PWR 1B), the early fatalities seem to be quite insensitive to weather. This result might be expected from the climatological analyses in section 5. For the hot release there is substantial scatter beyond 15 miles since the cloud is initially high and early fatalities would be expected only if there were heavy local ground contamination due to rain. For the hot release there is substantial scatter beyond 15 miles since the cloud is initially high, and early fatalities would be expected only if there were heavy local ground contamination due to rain. For a specific distance, rain has a relatively low probability of occurrence which cannot be accurately represented by only 45 trials so the scatter beyond 15 miles stems primarily from sampling variations rather than specific differences in weather types. It should be remembered that the probabilities of the highest calculated values are less than 10^{-9} per reactor-year and thus the scatter is not important since it would not be reflected in the complementary cumulative distribution functions which are truncated at this probability for the reasons stated in section 13.2.3.

The effect of evacuation on the probability of early death is also shown in Figs. VI 13-23 and VI 13-24. The upper curves in each figure are for the ineffective evacuation case. [*Ineffective evacuation (zero effective speed) should be distinguished from no evacuation. The latter scenario would imply more shielding than is assumed for evacuation.*] An effective evacuation speed of 1.2 mph (modal value) reduces the individual early mortality probability by a factor of 10. For effective speeds of 4.7 mph (mean value) and 7.0 mph, the probability of early death is reduced to essentially zero within 25 miles. For these higher speeds, the only early fatalities are caused by high localized ground contamination due to rain and, as stated earlier, the occurrence probabilities are very low. As stated in section 11.1.1, evacuation is modeled by using three effective evacuation speeds, 0, 1.2 and 7 mph with probabilities of 30, 40 and 30%, respectively. The sensitivity of the maximum values (10^{-9} per reactor-year) for early fatalities, early illness and latent cancer fatalities shown in Figs. VI 13-30, VI 13-31, and VI 13-33, respectively are stated in Table VI 13-6. In the event of a very ineffective evacuation in which the evacuees were exposed to ground contamination for longer than 4 hours, the number of early fatalities and illnesses might be increased by a factor of 3 or 4. The study recommends more work in modelling of evacuations.

TABLE VI 13-6 SENSITIVITY OF MAXIMUM HEALTH CONSEQUENCES TO EFFECTIVE EVACUATION SPEED

Effective Evacuation Speed (mph)	Early Fatalities	Early Illness	Latent Cancer Fatalities per Year
0	6200	80,000	1400
1.2	2300	30,000	1400
7	350	4,500	1400
Average	3300	45,000	1400

Figs. VI 13-23 and VI 13-24 show that the probability of early death is strongly influenced by the heat rate in the release of radioactive release. To evaluate this sensitivity from the viewpoint of total early fatalities, the mean and peak fatalities in a uniform population sector of 100 people/mile^2 are calculated as a function of the heat rate in a PWR-1 release with one meteorological data set. Fig. VI 13-25 shows that, though the mean fatalities decrease for the hotter releases, the peak fatalities remain about constant (the slight rise in both the mean and peak values for heat rates exceeding 50 million Btu/hour is probably not statistically significant). The reasons for these

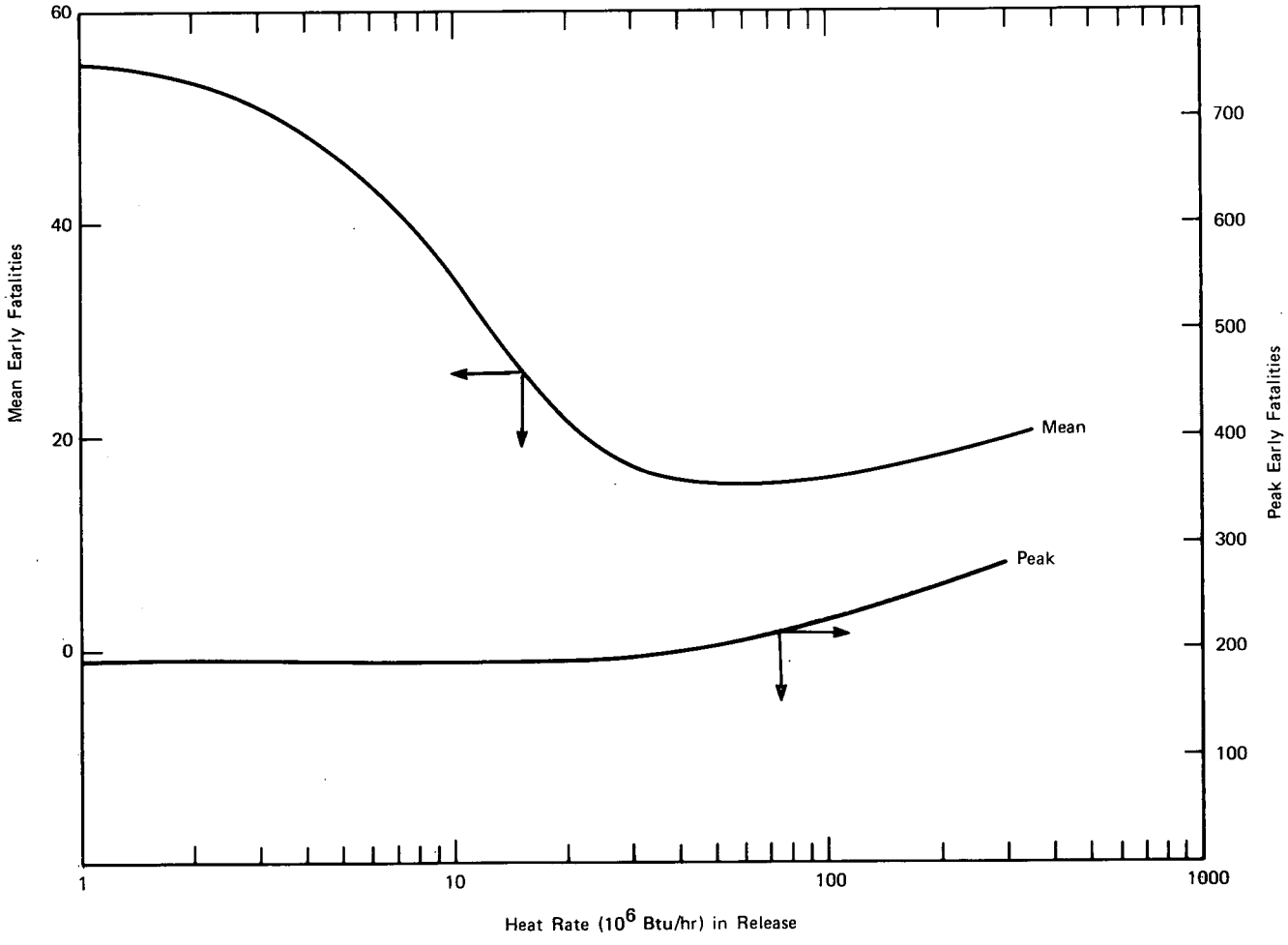


FIGURE VI 13-25 Effect of heat rate in release on early fatalities.

phenomena are that the peak early fatalities are caused by the occurrence of rain fairly soon after the release and the washout rate is independent of the height of the radioactive cloud. The mean early fatalities are dominated by the more frequent events of stable conditions and low wind speeds which cause fewer lethalities when the cloud is high.

13.3.2 LATENT CANCER FATALITIES

As mentioned in the preceding section, one would not expect the calculation of latent cancer fatalities to be sensitive to the various meteorological data sets. In fact, the averaging of latent cancer fatalities from the many different combinations of weathers should produce a distribution that is directly proportional to the population distribution as a function of distance from the reactor. This is a result of the latent cancer fatality calculation being an integral of dose multiplied by number of exposed persons over very large areas.

Figure VI 13-26 shows the conditional probability for an individual of dying from latent cancer as a function of distance from a reactor given the PWR-1A and PWR-1B releases. The probability of latent cancer fatalities is relatively constant out to about 100 miles from the reactor beyond which it decreases rapidly. The small difference between the two curves is due to the different heat rates in the PWR-1A and PWR-1B releases. With the large heat rate in the PWR-1B release, it is less likely for an individual close to the reactor to be subject to significant doses. Therefore, the probability for a latent cancer death would be lower close to the reactor for the hot release case (PWR-1B) than for the cool release case (PWR-1A). In contrast, in the hot release it is more likely for greater quantities of the released radioactive material to reach greater distances. Therefore, under unusual weather conditions, or rain, there is a greater quantity of radioactive material available for deposition. This will result in a higher probability for latent cancer fatalities at 20 to 50 miles from a reactor for a hot release. Beyond about 50 miles, there is essentially no difference in the probabilities for the two releases.

13.3.3 PROPERTY DAMAGE

As discussed in section 11.2.2, a measure of the degree of environmental contamination is the decontamination factor that would be required to bring the area down to an acceptable level of radioactivity.

The decontamination factor DF is defined as the ratio of the amount of contaminant per unit surface area to the amount after decontamination, as specified in section 11.2.1.

Figure VI 13-27 shows the distribution of the mean required decontamination factor as a function of distance from the reactor for a composite of the major releases (BWR-1, BWR-2, and BWR-3). The radiation dose criteria utilized in these results was 10 rem to the whole body in 30 years. The curve labeled with DF>1 shows the distance to which some decontamination would be required. The curve on the left-hand side (DF>20) illustrates the maximum distance to which land interdiction would be required. All the curves in Figure VI 13-27 display a similar behavior; that is a relatively flat portion followed by a sharp drop. This behavior reflects the high probability of large quantities of radioactive material being deposited within 50 miles but not beyond. Raising the acceptable limit on radiation exposure to some value greater than 10 rems in 30 years would shift the curves to the left.

13.3.4 INTERDICTION CRITERION

The model for interdiction and decontamination of land is described in section 11.2.2. The criteria for requiring either interdiction or decontamination assumed by the study are stated in Table VI 11-6 and are 10 and 25 rem to whole body within 30 years for rural and urban areas respectively. In the event of an accident, establishment of these criteria would be a compromise between additional latent-cancer fatalities and genetic effects and increased costs. This section presents some calculations that illustrate the magnitudes of these tradeoffs.

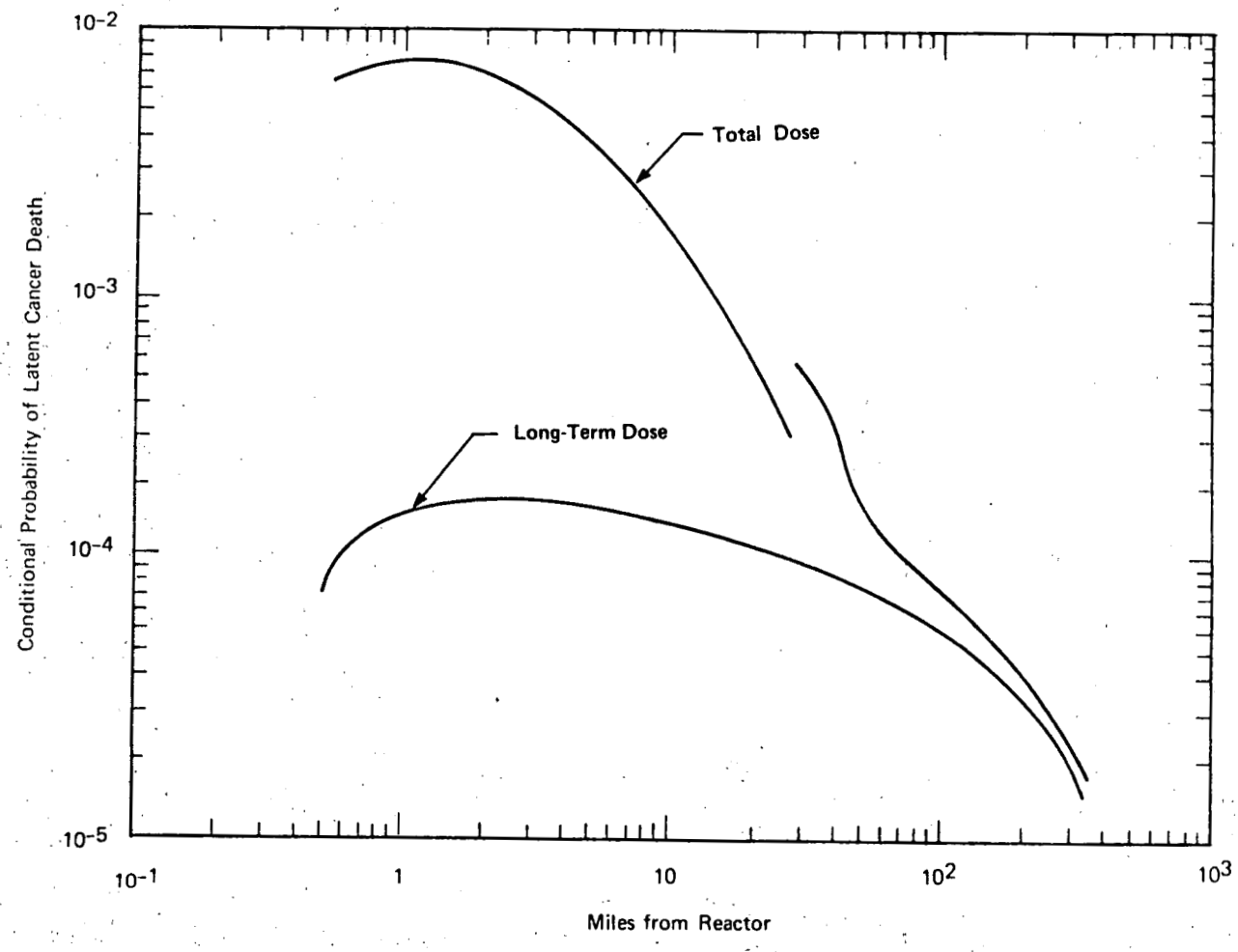


FIGURE VI 13-26 Conditional probability of latent cancer death given a PWR-1a release. Long-term dose is from external radiation from ground, inhalation of resuspended contaminants and ingestion of contaminated foods. An interdiction criterion of 10 rem in 30 years is assumed. Total dose is long-term dose plus dose from passing cloud due to external radiation and inhalation and from short-term ground exposure. An effective evacuation speed of 1.2 mph is assumed. (Approximately, absolute mortality probabilities are 10^{-6} per reactor year times stated ones).

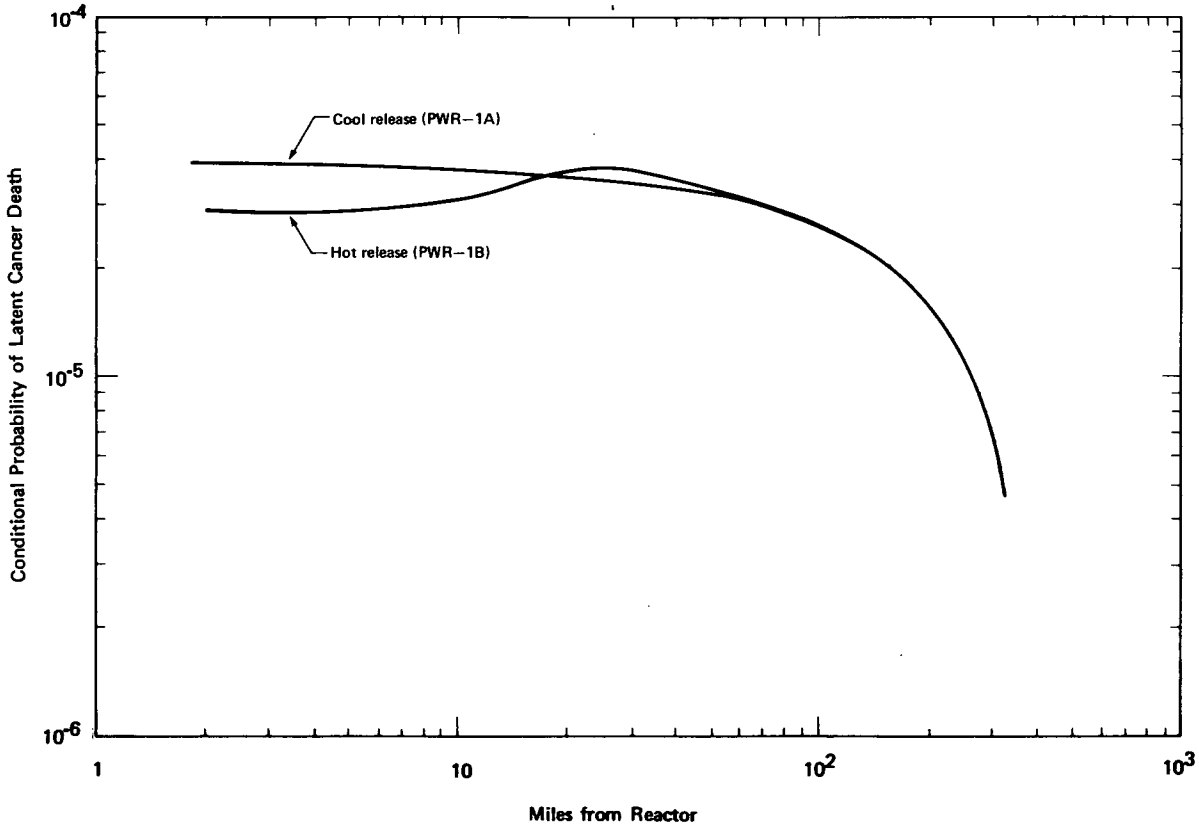


FIGURE VI 13-26 Conditional probability of latent cancer death given a PWR-1A or PWR-1B release. (Approximately, absolute mortality probabilities are 10^{-6} per reactor year times stated ones).

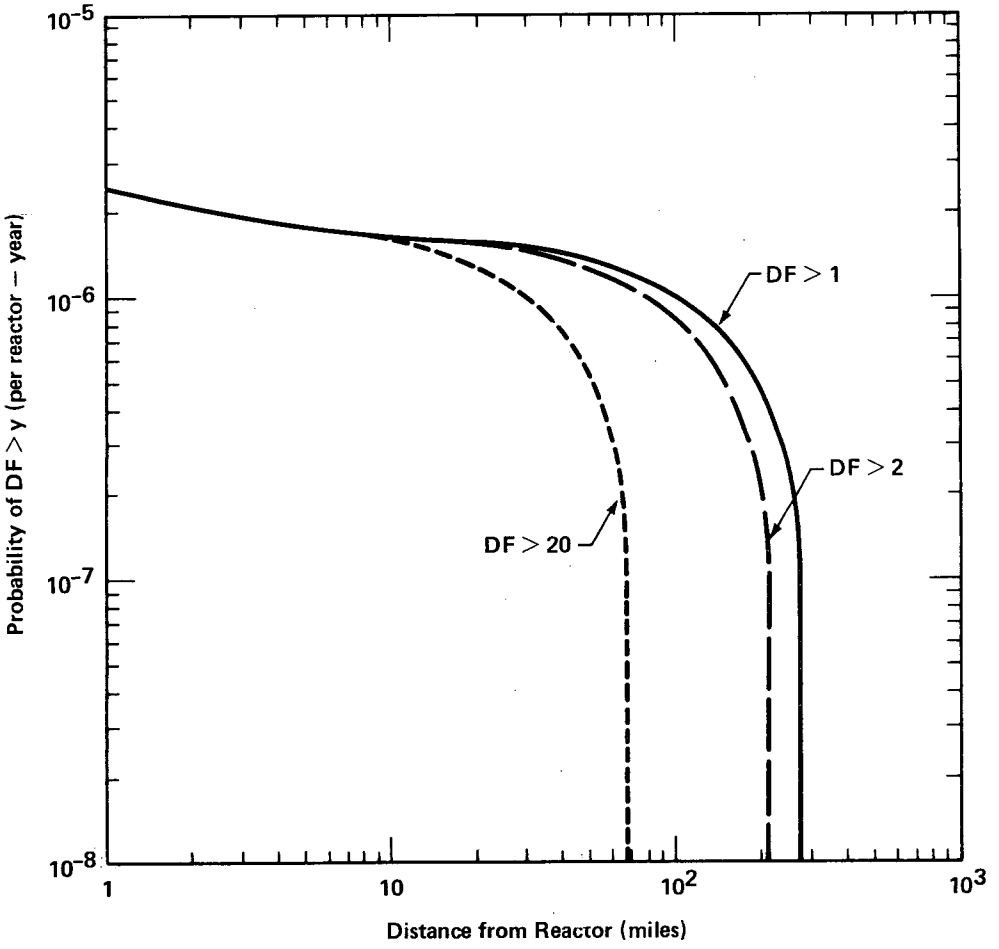


FIGURE VI 13-27 Probability of exceeding selected decontamination factors as a function of distance for composite of BWR-1, BWR-2 and BWR-3 releases.

Figures VI 13-28 and VI 13-29 show the changes in the mean latent cancer fatalities, genetic effects and land areas requiring decontamination and interdiction of longer than 10 years as a function of the interdiction criterion in the event of the largest release, PWR 1A and PWR 1B. In order to avoid the discontinuties associated with a nonuniform population, the calculations are for a uniform population density of 100 people/mile^2 and 45 trials of one meteorological data set. For other population densities or distributions, the areas would not change but the number of health effects and property damage costs would be roughly proportional to the number of people involved. Reviewing Figs. VI 13-28 and VI 13-29, there appears to be only a small difference between the results for the cool release (PWR 1A) and the hot release (PWR 1B).

The figures show that increasing the criterion from 10 to 25 rem in 30 years would increase the mean health effects by about 10% and would decrease the areas (and property damage) by about 60%. Similarly, increasing the criterion from 25 to 50 rem in 30 years would further increase health effects by about 15% and would further decrease the areas by about 55%. The corresponding changes in the maximum values (10^{-9} per reactor-year) for increasing the criterion from 10 to 25 rem are 10% for health effects and 33% for areas and property damage.

13.4 RISK CALCULATION FOR 100 REACTORS

The discussions in the preceding sections have provided insight into expected individual risk from characteristic types of releases under average meteorological conditions. In this section the calculated societal risk from 100 commercial light water reactors at 68 different sites is discussed. The risk is given by the probability and magnitude of seven different consequences. These consequences are early fatalities (death within approximately one year after a potential accident), early illnesses, thyroid nodules, latent cancer fatalities, genetic effects, land contamination, and property damage costs. These societal risks are stated as complementary cumulative distribution functions per reactor-year in Figures VI 13-30 through VI 13-36. The complementary cumulative distribution function shows the probability that a consequence will exceed a given magnitude. This probability is obtained by integrating the consequence magnitude over the entire accident spectrum. The early health effects were calculated with three effective evacuation speeds weighted by their probabilities of occurrence.

The probability-consequence relationships shown in these curves are based on population and meteorological distributions applicable to the 68 sites at which the first 100 reactors will be located. They thus represent the average risk obtained from all sites and are not necessarily representative of a given reactor at a particular site.

As mentioned in previous sections, the calculation of early fatalities is sensitive to the methodology and input data utilized. This sensitivity appears also in the complementary cumulative distribution function for early fatalities, Fig. VI 13-30. The differences between the curves for the PWR and BWR are less than the uncertainties inherent in the calculational methods and input data. In all the other consequence results the differences between the average PWR and BWR curves are quite small. Fig. VI 13-31 shows a similar distribution for early illnesses. 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 Fig. VI 13-31.

Radiation exposure of the thyroid gland increases the likelihood of thyroid nodules. Figure VI 13-32 shows the distribution for the expected number of cases of nodules per year in the event of a reactor accident. In practice, the total number of cases of thyroid nodules is calculated by the method set forth in section 9.3.5. These nodules would primarily manifest themselves in the time period 10 to 40 years after the release. The annual rate shown in Fig. VI 13-32 is the total number divided by 30. The largest calculated accident, corresponding to a probability of 10^{-9} per reactor-year, affects 10 to 15 million people. As stated in Table VI H-6, the normal incidence of thyroid nodules is 0.08% which translates into 8000 cases of nodules per year for 10 million people. By comparison to the maximum value shown in Fig. VI 13-32, the largest calculated accident might double the incidence of cases of nodules over a period of 30 years, which would be a statistically detectable effect. As stated in section 9.3.5, about one-third of the cases of nodules would be expected to be cancerous, of which 10% might be lethal. The fatalities from thyroid cancer are added to the other latent cancer fatalities.

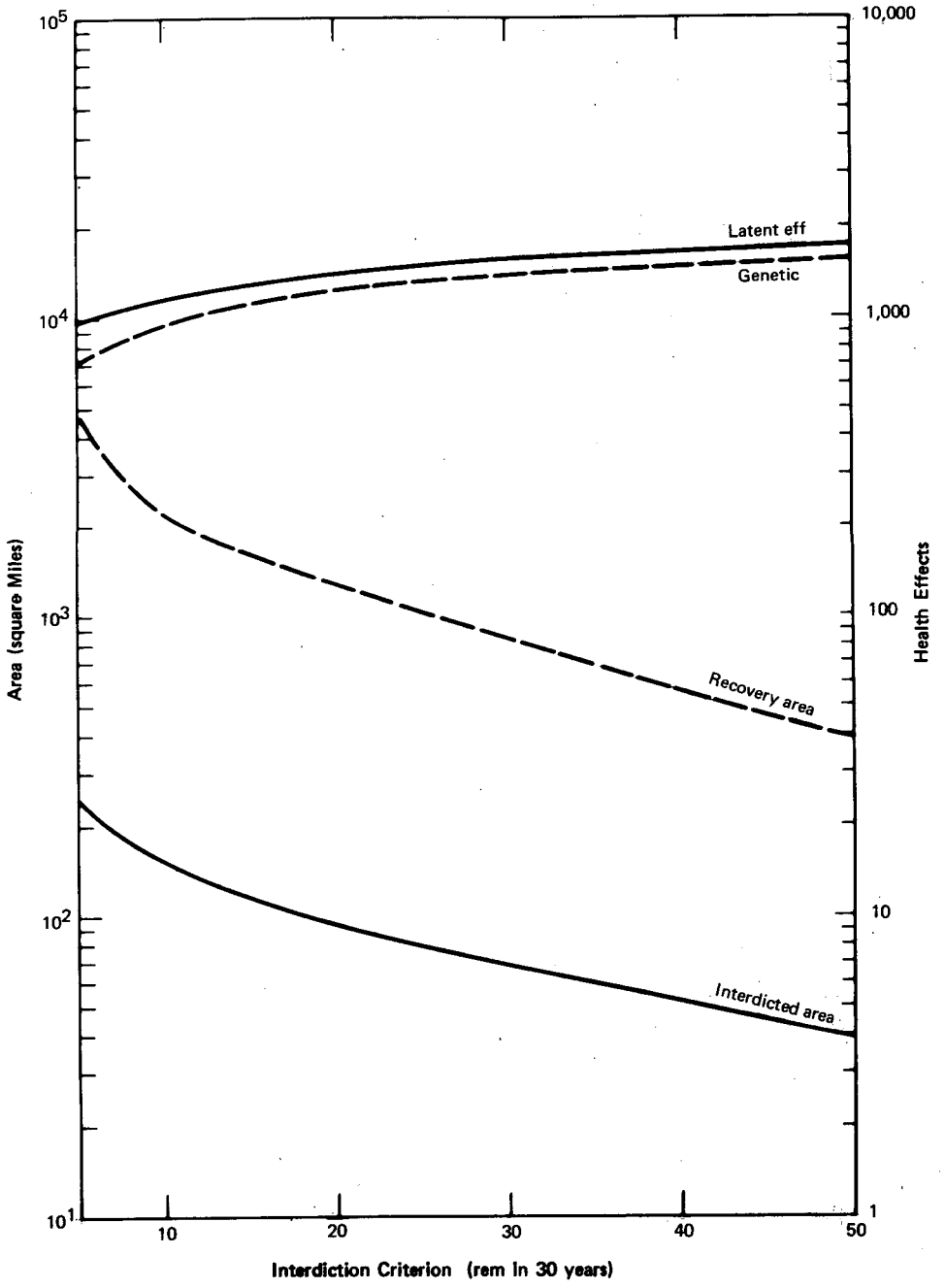


FIGURE VI 13-28 Effect of interdiction criterion upon long term health effects and property damage areas for PWR-1A release.

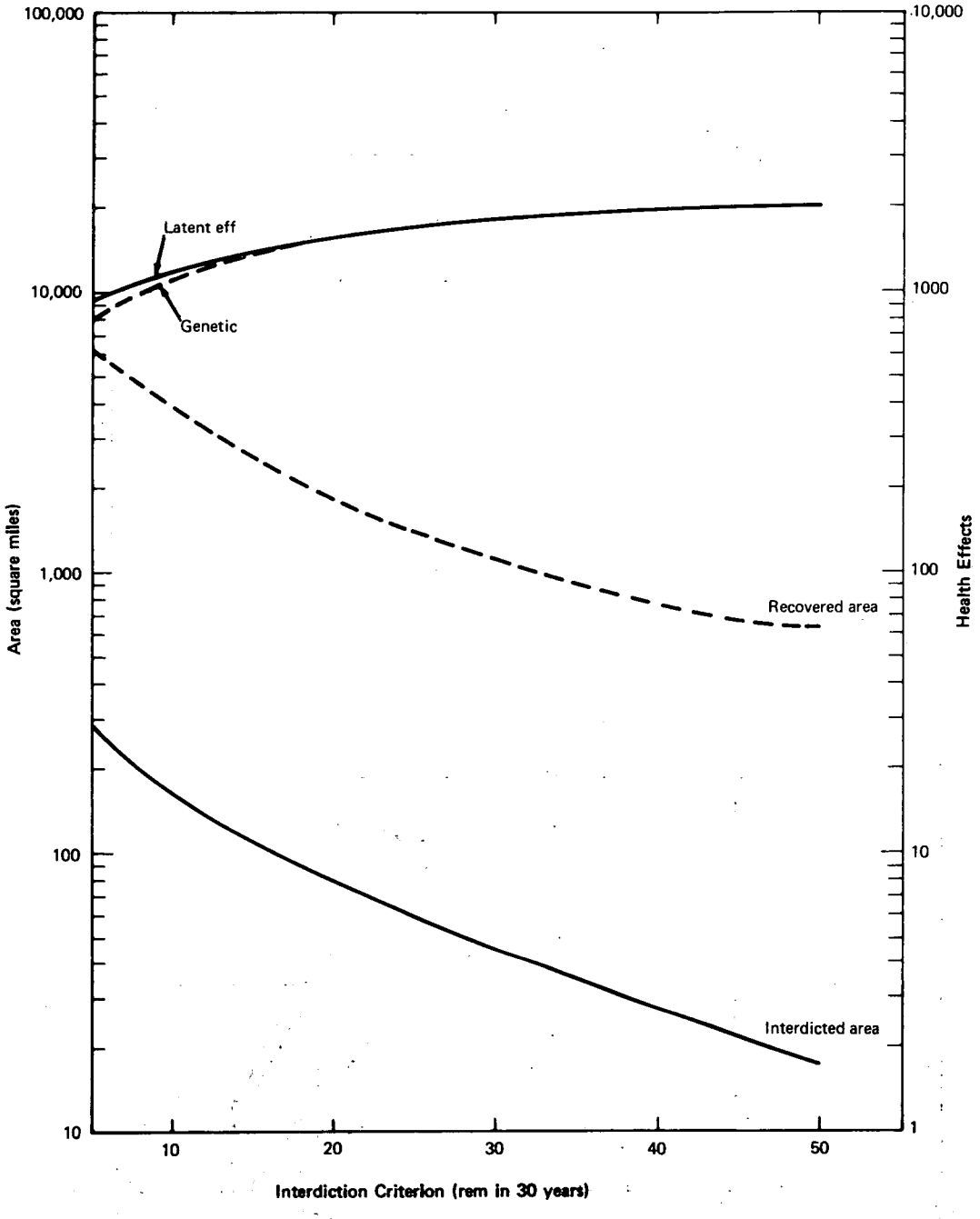
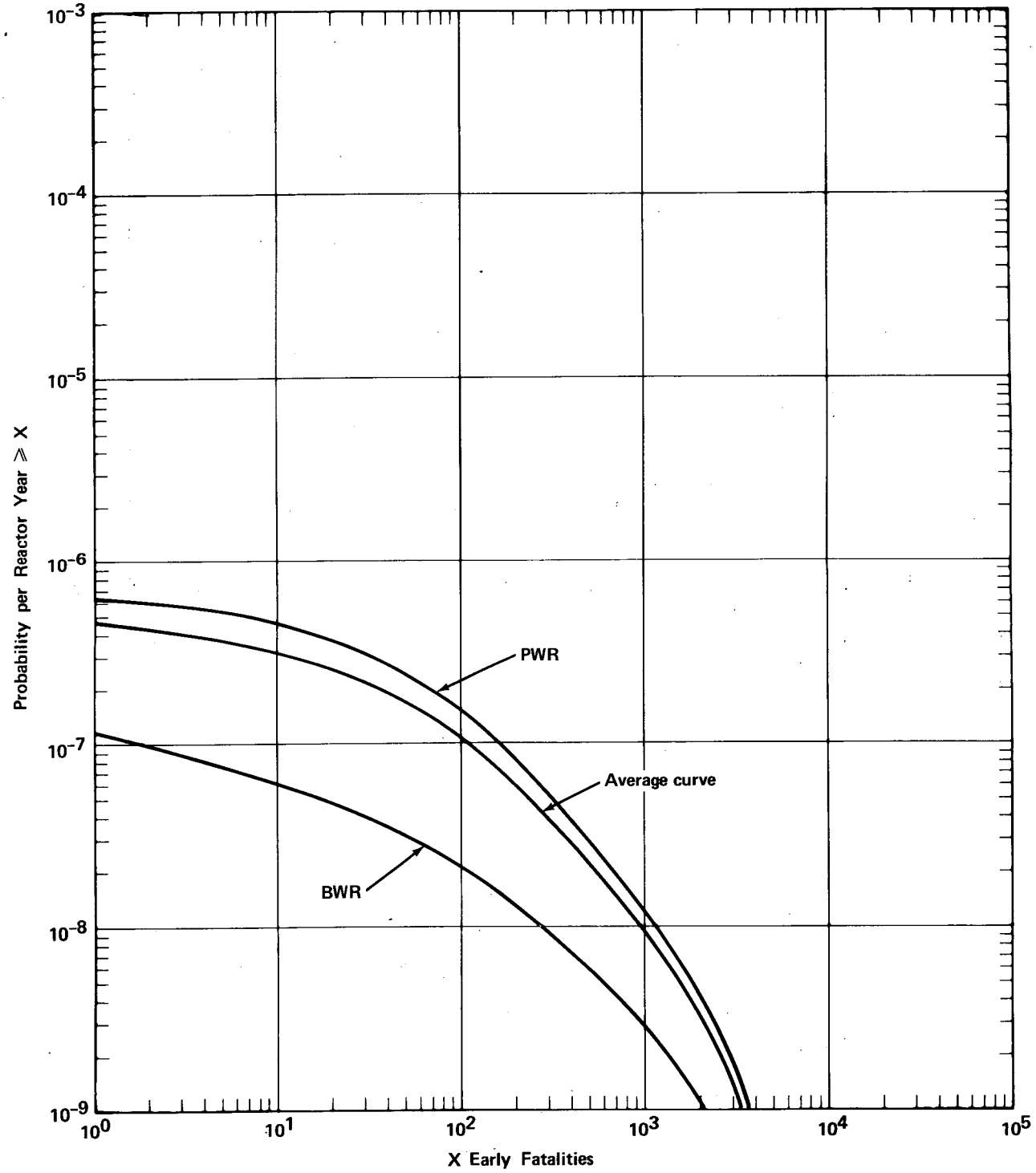
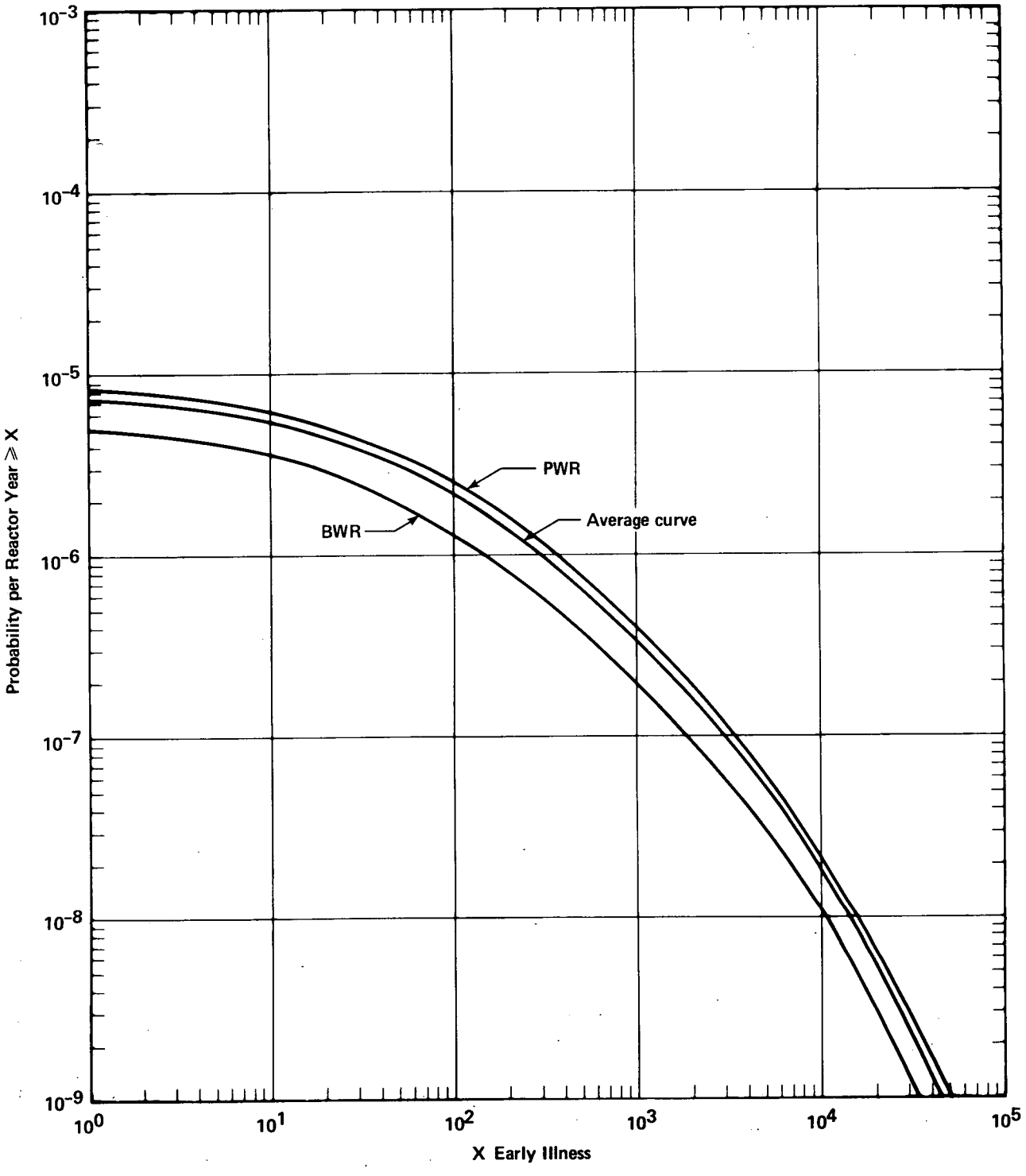


FIGURE VI 13-29 Effect of interdiction criterion upon long term health effects and property damage area for PWR-1B release.



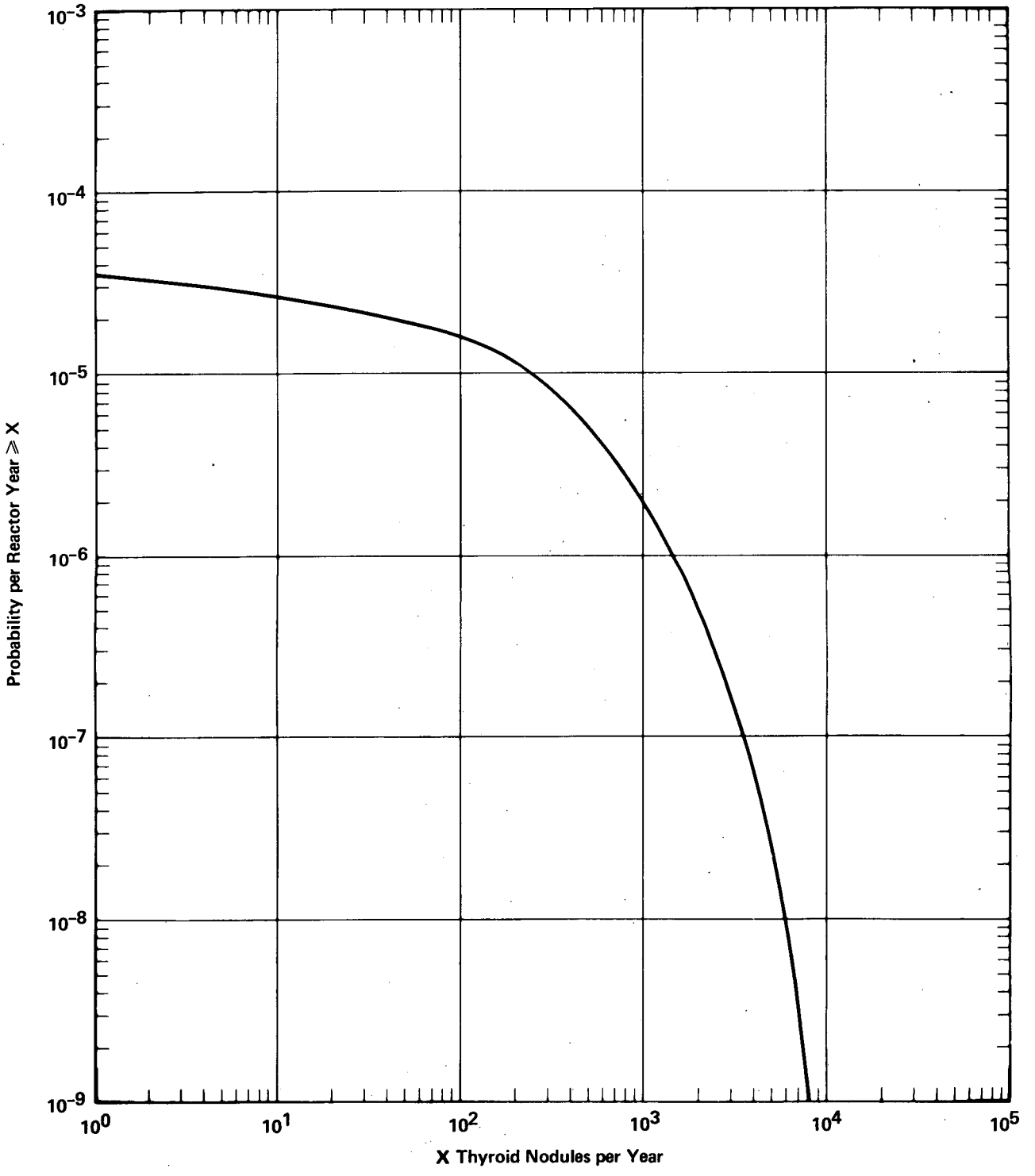
Note: Approximate uncertainties are estimated to be represented by factors of 1/4 and 4 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-30 Complementary cumulative distribution function for early fatalities.



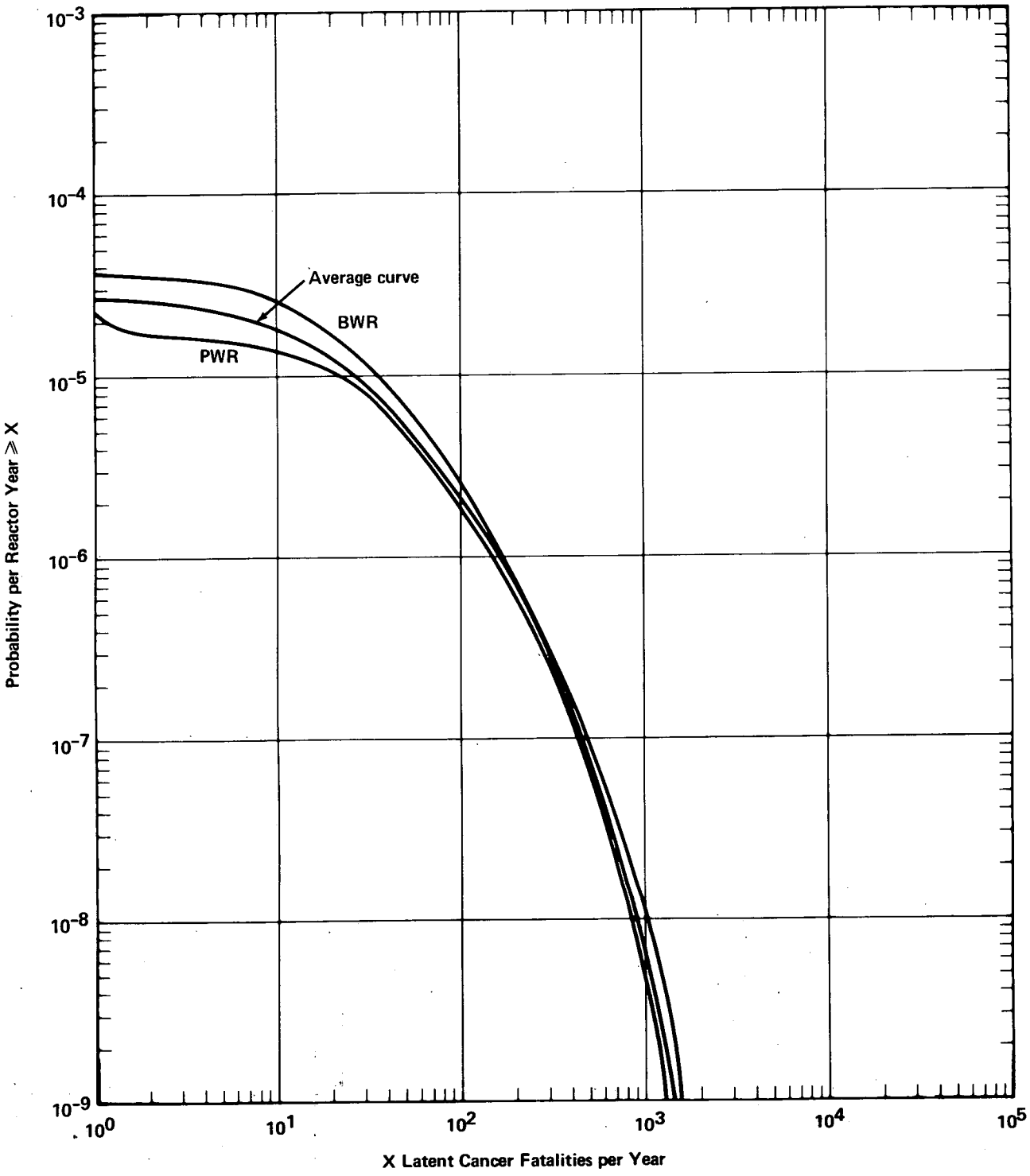
Note: Approximate uncertainties are estimated to be represented by factors of 1/4 and 4 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-31 Complementary cumulative distribution function for early illnesses.



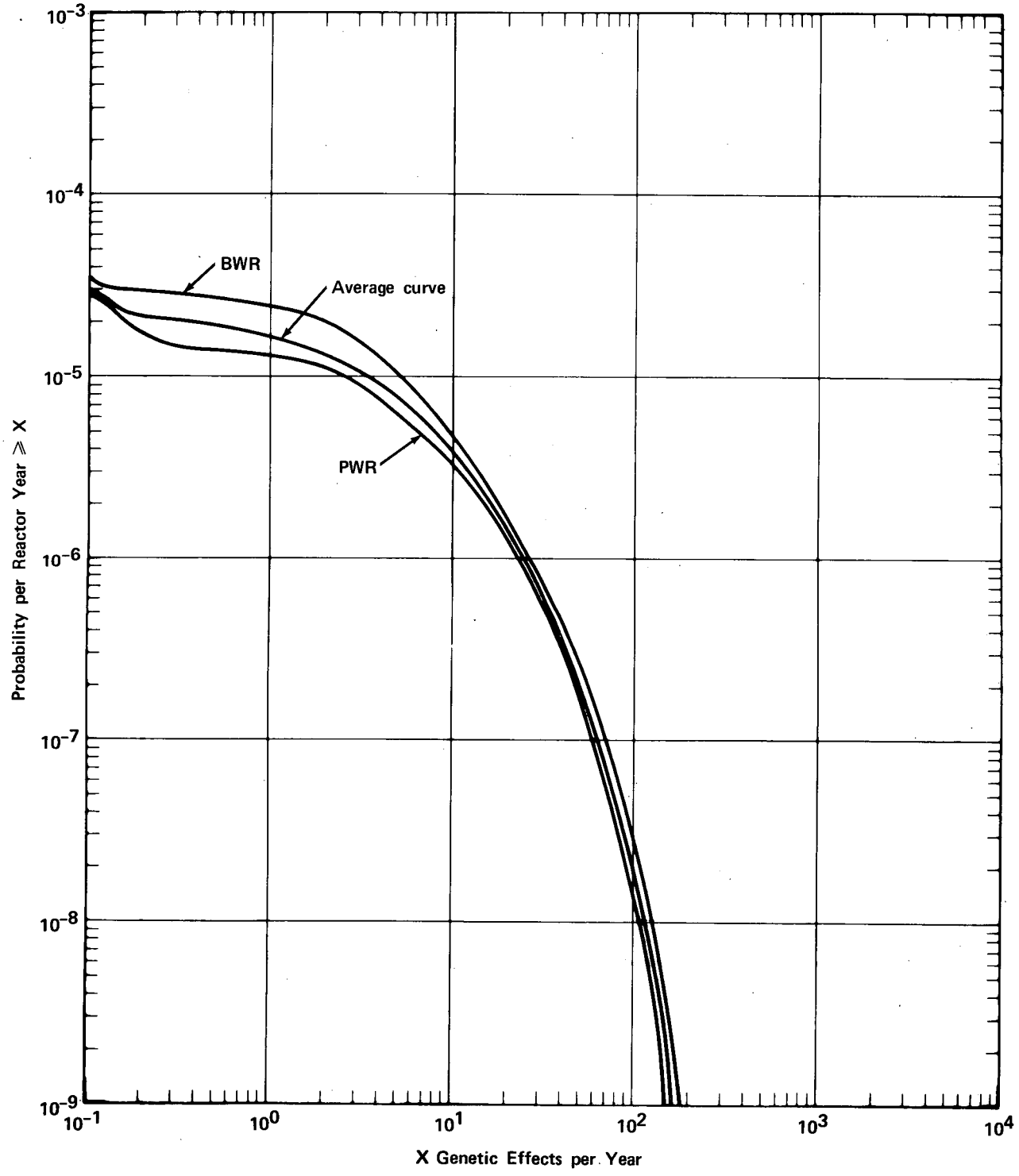
Note: Approximate uncertainties are estimated to be represented by factors of 1/3 and 3 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-32 Complementary cumulative distribution function for thyroid nodules per year.



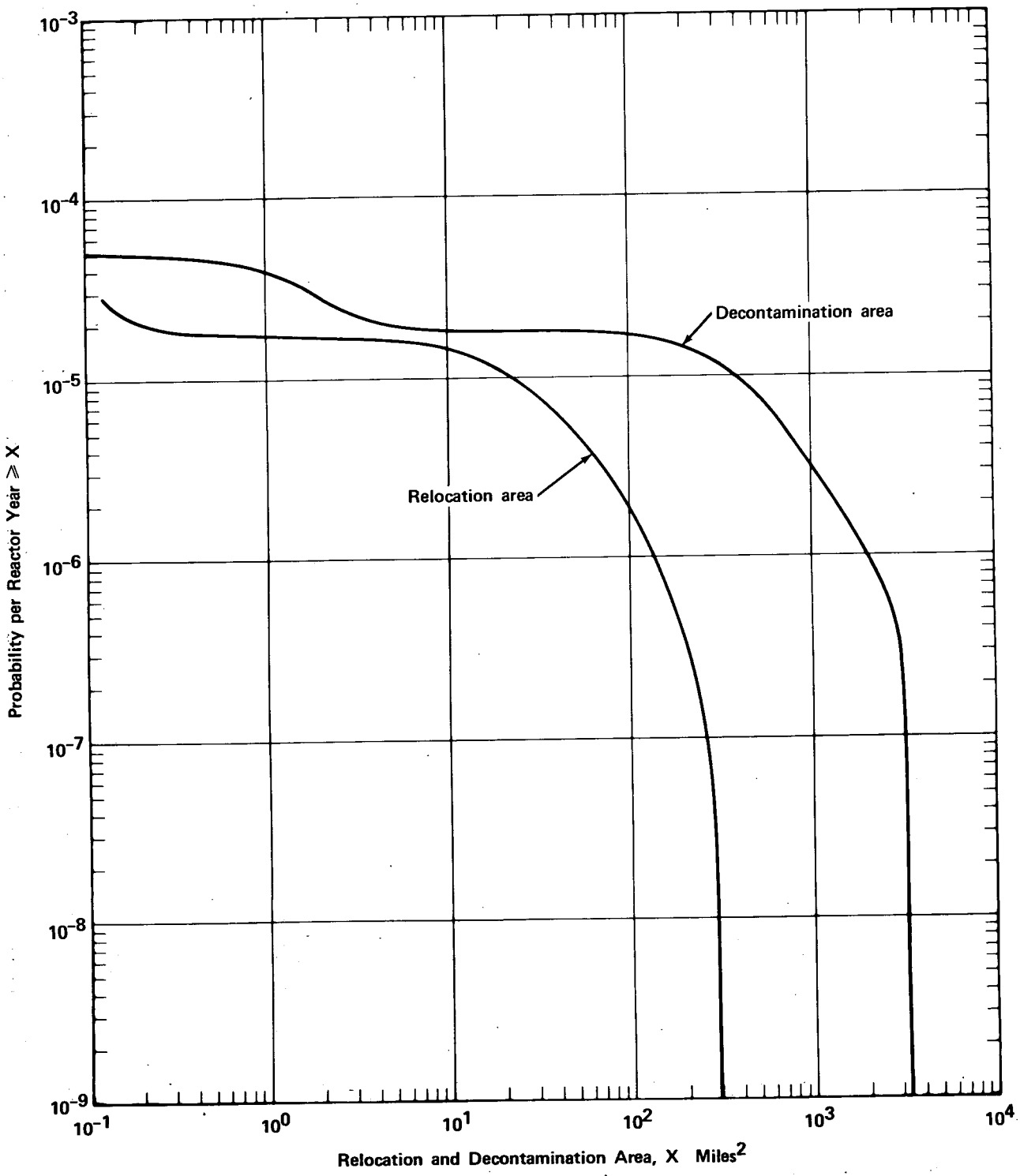
Note: Approximate uncertainties are estimated to be represented by factors of 1/6 and 3 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-33 Complementary cumulative distribution function for latent cancer fatalities per year.



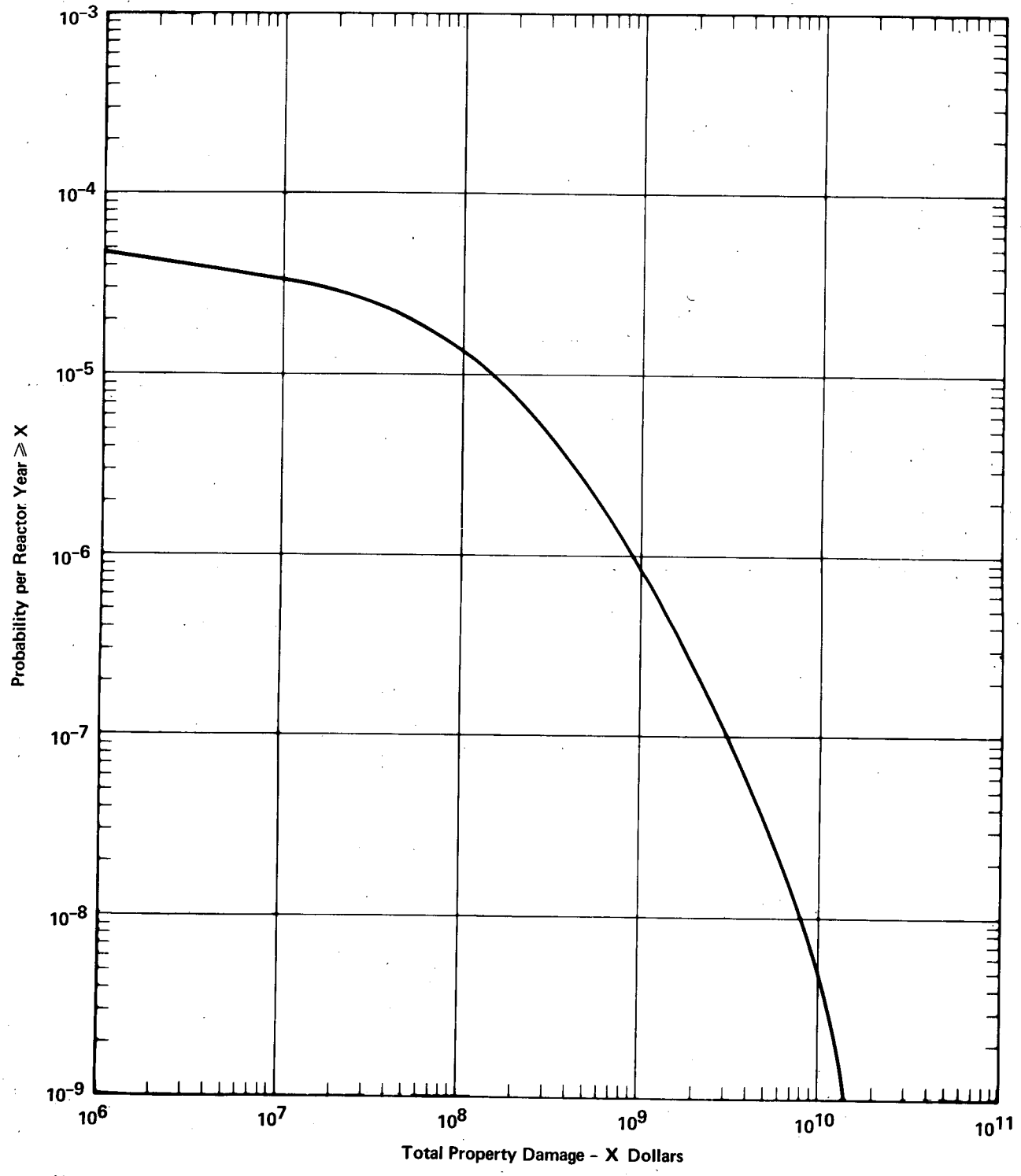
Note: Approximate uncertainties are estimated to be represented by factors of 1/3 and 6 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-34 Complementary cumulative distribution function for genetic effects per year.



Note: Approximate uncertainties are estimated to be represented by factors of 1/5 and 2 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-35 Complementary cumulative distribution function for relocation and decontamination area.



Note: Approximate uncertainties are estimated to be represented by factors of 1/5 and 2 on consequence magnitudes and by factors of 1/5 and 5 on probabilities.

FIGURE VI 13-38 Complementary cumulative distribution function for total property damage.

The number of cancer fatalities that might result from a reactor accident is estimated by the methods described in section 9.3.2.3. They are calculated on an organ-by-organ basis including the dose effectiveness factors stated in Table VI 9-7. Over what time period would these fatalities be seen? In accordance with the latent periods stated in Table VI 9-2, leukemias caused by irradiation in utero would manifest themselves shortly after the accident. In theory, at least, there might be latent cancers attributable to the accident as long as ground contamination persists, that is, 100 or more years. However, the majority of latent cancer fatalities would occur within the time period 10 to 40 years after the accident. The annual rate of latent cancer fatilities shown in Fig. VI 9-33 is based on the total number of late fatalities divided by 30 which is somewhat conservative. As stated in Table VI 9-9, the spontaneous incidence of cancer fatalities is 1700 per million population. For a 10-million population at risk, this incidence translates into 17,000 per year. By comparison to the maximum value shown in Fig. VI 9-33, the largest calculated accident ($\sim 10^{-9}$/reactor year) might increase the incidence of fatal cancers by 1500/17,000 or by about 9% in the population at risk. Such an increase would probably not be statistically detectable because of the normally large variation in the rate.

The number of genetic effects in live births that might be attributable to a reactor accident is calculated by the methods set forth in section 9.4. It is helpful to consider the numbers appearing in the first generation separately from those in succeeding generations. A generation length is about 30 years, so the annual incidence shown in Fig. VI 13-34 equals the number of genetic effects in the first generation divided by 30. As stated in Table VI 9-10, the number of spontaneous genetic effects excluding spontaneous abortions is 24,000 per million per 30 years which translates to an incidence of 8000 per year for a population of 10 million which might be at risk. Comparison of this incidence to the maximum rate (190/year) shown in Fig. VI 13-34, the largest calculated accident might increase the spontaneous rate by about 2% which is unlikely to be statistically detectable. As shown in Tables VI 9-11 and VI 9-12, the total number of genetic effects is about five times the number in the first generation. Since the incidence declines exponentially with consecutive generations, several hundred years must elapse before the last mutant gene attributable to the accident is eliminated. Clearly, the annual incidence declines with generation so that the rate shown in Fig. VI 13-34 is the maximum.

Interdiction criteria are discussed in section 11.2.2 and their effect on long-term health effects and property damage is illustrated in section 13.3.4. For reasons stated in section 11.2.2, the study assumed that a criterion of 10 rem in 30 years would be applied for rural areas and 25 rem in 30 years for highly populated areas. Thus, the preceding distributions for latent cancer fatalities and genetic effects and the following distributions for area and property damage were calculated for both criteria. The final distributions discussed in this section are interpolations between these results; the 10-rem curve is used for small consequences and 25-rem curve for large consequences. As shown in section 13.3.4 and Fig. VI 13-28 and VI 13-29, there is a small difference for the long-term health effects but the peak area and property damage ($\sim 10^{-9}$ per reactor-year probability) are substantially reduced. The study judged that this approach is a reasonable interpretation of the radiation guides. Figures VI 13-35 and VI 13-36 show probability distributions for the decontaminated and interdicted areas and for the property damage respectively.

13.5 UNCERTAINTY ESTIMATES ON CALCULATED RESULTS

With the use of limited parametric studies, the impact of various assumptions, input data, and methodologies on the calculated consequences has been considered. Because of the nature of the problem, these results indicate only the general degree to which the results are influenced and they should not be taken as representing absolute limits. The quoted uncertainties represent judgments of the study based upon an understanding of the problem gained in the course of the work.

It is helpful to consider uncertainties within two categories, the dispersion-dosimetric model and the dose-response criteria or cost parameters. The first category includes uncertainties in (a) the release fractions, probabilities and physical characteristics, (b) atmospheric dispersion model and input data including depletion of cloud, and (c) dosimetry. These factors influence all calculated consequences. The second category includes the individual dose-responses, e.g., $LD_{50/60}$, number of latent cancer fatalities per 10^6 man-rem, and cost parameters, e.g., capital assets per capita. These factors affect only their corresponding consequences.

In general, the calculation of early fatalities and illnesses is quite sensitive to the first category especially the physical characteristics of the release. Fortunately, the early health effects are limited to the proximate area of the reactor within which distance the plume model is probably a reasonable approximation since it is supported by experimental data. As shown in section 13.3.1, the effective evacuation speed has a strong influence on the number of early fatalities. On the other hand, the dose-mortality or -morbidity criteria for the principal health effects are relatively well known. A major perturbation such as reducing the $LD_{50/60}$ from 510 to 340 rads increases the early fatalities by a factor of 3 or 4 depending upon circumstances. It is judged by the study that the overall uncertainties in the calculation of early fatalities and illnesses are factors of 4 higher and lower on the quoted magnitudes.

The other consequences, latent cancer fatalities, genetic effects and property damage appear to be less sensitive to the first category of uncertainties since they are integral effects over a large area. On the other hand, each of these consequences has a larger uncertainty in its individual effect. As shown in section 13.1 the upper bound for latent cancer fatalities is about a factor of two higher than the quoted central estimate. The lower bound, assuming a threshold dose, is about a factor of 5 or more lower. Thus, the study assigns uncertainties to this calculation of factors of 3 higher and 6 lower to the quoted magnitudes. As stated in Appendix I, the genetic effects are based upon a doubling dose of 100 rem. The BEIR Report quotes a range of 20 to 200 rem which would translate into factors of 5 higher and 2 lower for the number of genetic effects. Thus, the study assigns uncertainties of factors of 6 higher and 3 lower to genetic effects.

The property damage calculated for the major releases of radioactive material is considered to be conservative because of the conservative rain model and the lack of credit taken for possible washoff and runoff of the deposited radionuclides. In addition, it is also felt that the economic model and economic parameters may be conservative. The study estimates that the overall uncertainty on the property damage calculations are factors of 2 higher and 5 lower.

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Section 14

Acknowledgments

14.1 HEALTH EFFECTS

The incidence of radiation-induced health effects in humans is generally very low, and available data are often limited to a few cases whose circumstances are usually different from a hypothetical reactor accident. In this situation, the interpretation of available data to make risk estimates is a highly specialized subject for which expert judgment is paramount. The Reactor Safety Study was aided in making these judgments by an advisory group on health effects, whose membership is listed below. The advisory group recommended dose-response relationships, which were in all cases used by the study; however, the judgments and opinions expressed within this appendix are the responsibility of the Reactor Safety Study.

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Metabolism, and Digestive Diseases; R. B. Shafer, M.D., Veterans Administration Hospital, Minneapolis; R. N. Smith, M.D., The Royal Infirmary, Sheffield, Great Britain; J. D. Stanbury, M.D., Massachusetts Institute of Technology; O. P. Schumacher, M.D., Cleveland Clinic; and W. M. Tunbridge, M.D., Royal Victoria Infirmary, Newcastle, Great Britain.

14.2 OTHER SUBJECTS

In the subjects of atmospheric dispersion and wake effects, the study received valuable assistance from Drs. Frank A. Gifford, G. Eugene Start and Isaac Van der Hoven, National Oceanic and Atmospheric Administration; Dr. David P. Hoult, Massachusetts Institute of Technology; Dr. James Halitsky, private consultant, New York City; and Mr. Hugh W. Church, Sandia Laboratories, Albuquerque, New Mexico. The study appreciates the assistance of Dr. Peter E. McGrath, Sandia Laboratories, Albuquerque, New Mexico, for preparing the appendices on the chronic exposure model and decontamination, with input data from Dr. P. E. James, U.S. Department of Agriculture, Beltsville, Maryland; J. R. Horan, Energy Research and Development Administration, Idaho Falls, Idaho.

Appendix A

Review of Atmospheric Dispersion

A1 INTRODUCTION

In assessing the consequences of a hypothetical reactor accident, a phenomenon needing analytical description is that of atmospheric transport and diffusion of a contaminating aerosol released from the containment. The term "aerosol" is here used in its general sense as the system of gases and suspended particles, solid and liquid, natural and artificial, that would become dispersed into the atmosphere. Unfortunately, however, sufficient knowledge required to make an exact analysis is lacking, so a somewhat compromised description will be given based on state-of-the-art methods developed over the years.

In general, the atmosphere appears to operate on a given aerosol parcel in a similar manner no matter what method of parcel identification is chosen. Any one of a number of identification properties depending on the particular problem being considered may be chosen. Such parcel properties include momentum, heat energy, vorticity, chemical composition, light scattering, and particle concentration (size and number).

Of primary interest to this report is the chemical composition and particle concentration as a function of distance from the reactor. For a release of radioactive materials, this composition or concentration is normally expressed as radioactivity per unit volume of air (curies per cubic meter) for each radionuclide being tracked. For those radionuclides whose daughter products are important, their radioactive decay schemes are followed.

This appendix briefly describes the important characteristics of transport and diffusion in the atmosphere and their relation to measurable or identifiable characteristics of the atmosphere; it also discusses the relative importance and typical values of some important parameters as well as their measurement and use in the dispersion section of the computer model. The removal of gases and particles by dry deposition and wet removal by precipitation are covered in section 6 and Appendix B.

A2 ATMOSPHERIC TRANSPORT

For a continuous source (plume), the description of effluent concentration basically involves estimating how much effluent (particles, curies, etc.) per unit volume is at a particular point in space and time in relation to a specified amount of effluent release per unit of time; for an instantaneous source (puff), it involves establishing the time-integrated concentration at a particular point. The points of interest selected, or region of points, will vary with the particular problem considered but will generally be near the ground and at a location downwind from the point of release.

The most important meteorological parameter for this analysis is the atmospheric transport vector, or wind. For the continuous-source case, the wind will determine the direction of effluent travel and the quantity of air flowing past the point of release into which the effluent is initially diluted; for the instantaneous-source case, it will determine how quickly the effluent puff is carried past the downwind receptor. The time variability of the wind, in general, is a direct result of atmospheric turbulence, which will be discussed later. Wind that has little long-term variability is said to be highly persistent.

In addition to the time variability of the wind vector, space variability, both in the vertical direction above the release point and horizontally along the transport path between source and receptor are also important in determining the transport trajectory and the dilution ability of the atmosphere. Spatial and temporal wind variability (turbulence) near the ground is highly dependent on the geographic location, the vertical thermal stability, and the prevailing synoptic weather situation at the time of interest. A typical example of the type of surface concentration pattern to be expected downwind from an elevated source is shown in Fig. VI A-1a. The averaging effects on crosswind profiles are shown in Figs. VI A-1b and c.

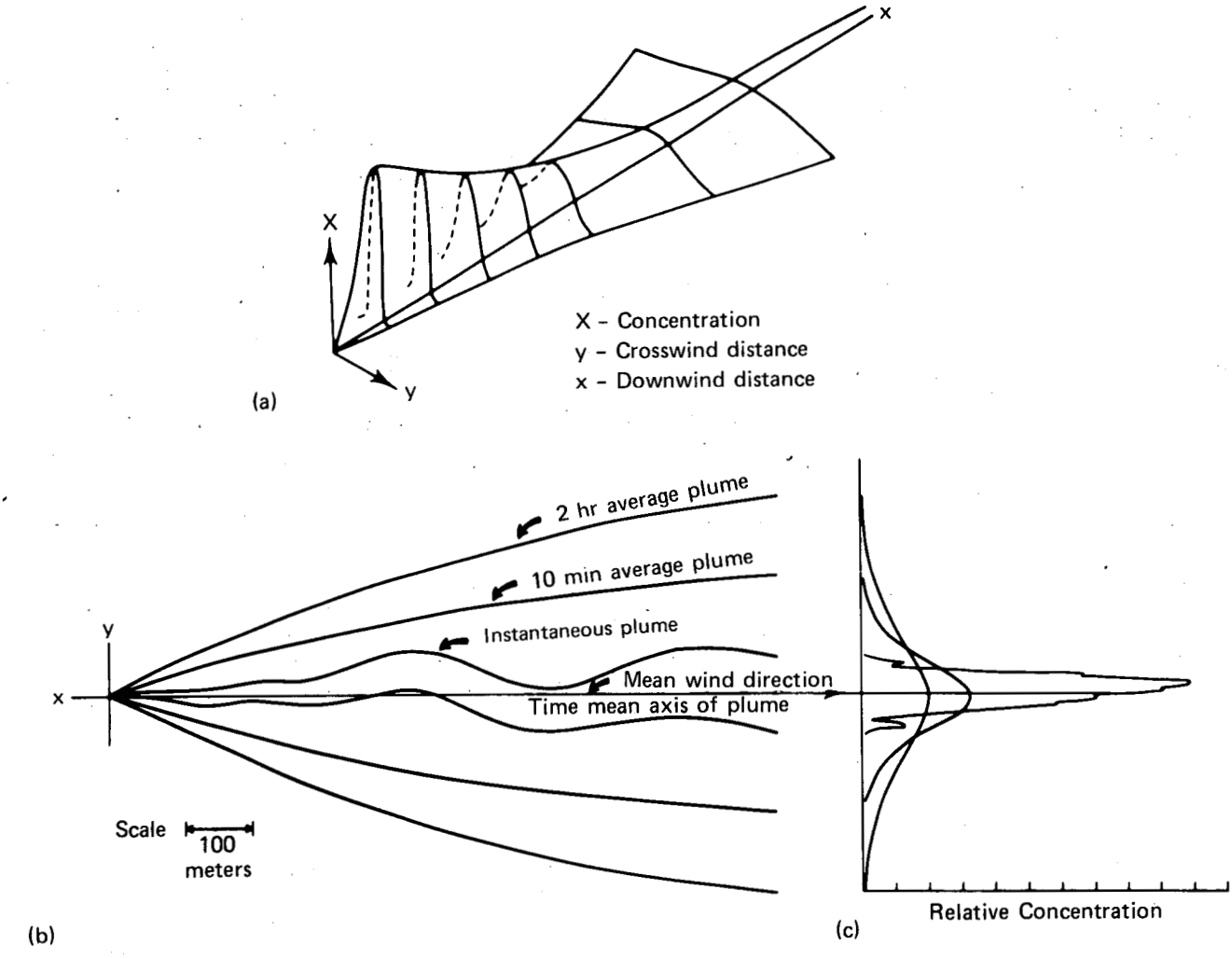

FIGURE VI A-1 (a) Surface concentration pattern downwind from an elevated source; (b) approximate outlines of a smoke plume observed instantaneously and of plumes averaged over 10 min and 2 hr; (c) corresponding cross-plume distribution pattern. From Slade (1968).

A3 TURBULENT DIFFUSION

The phenomenon by which adjacent parcels of air separate from each other under the influence of turbulent eddies is called atmospheric diffusion. The process is similar to the diffusion of a solute in a liquid solvent, heat in a thermally conducting body, or electrons in a conductor. All of these problems start with some form of the classical diffusion equation, which for simplicity can be written in one-dimensional form as

$$\frac{dq}{dt} = K\frac{\partial^2 q}{\partial x^2}, \qquad \text{(VI A-1)}$$

where q is some identifiable parcel property that is conserved, t is time, K is a diffusivity coefficient (here assumed to be constant), and x is a space coordinate. When this equation is appropriately generalized to the scale of the atmosphere and proper boundary conditions are applied, the solution can be shown to take on the form of the classic Gaussian function. If one generalizes to three dimensions and adds the additional restrictions that x is along the mean downwind direction, y is horizontally across wind, z is vertically upward, the receptor is at ground level (z = 0), and the source is at z = h, assumes total reflection at the ground (no deposition) and neglects diffusion downwind compared to gross transport by the mean wind of speed u, the specific solution of interest becomes

$$\chi(x,y,0;h) = \frac{Q}{\pi\sigma_y\sigma_z u}\exp\left[-\frac{y^2}{2\sigma_y{}^2} - \frac{h^2}{2\sigma_z{}^2}\right] \qquad \text{(VI A-2)}$$

where the distribution parameters σ_y and σ_z are the standard deviations of the crosswind and vertical distribution of contaminant, respectively, each a separate function of x. The derivation of this solution is not simple and straightforward, requiring several assumptions such as constant diffusivity to allow the integration of Equation (VI A-1) and subsequent empirical description of diffusivity behavior to fit experimental observations. Some of this behavior is discussed next. A detailed summary of the historical and technical developments of theories of diffusion in the lower layers of the atmosphere along with supporting data has been presented by Gifford (1968).

This expression of the so-called Gaussian diffusion model is of identical form for continuous and instantaneous sources, except that the source Q and the dosage χ have slightly different meanings and dimensions in the two cases and the values of σ_z and σ_y are somewhat different because of the truncated spectrum of turbulence intensity versus scale size operating in the instantaneous-source case. The spectrum of eddy sizes that is present in the atmosphere and contributes to the intensity of turbulence is continuous from molecular scale up through tropospheric depth dimension (~10 kilometers) for the vertical scale and up through global quadrant lengths (~10^4 kilometers) for the horizontal scale. These spectral functions are neither monotonic nor steady-state ones, but the fact that significant turbulent energy contributions exist in the large-scale portion has a powerful effect on atmospheric turbulent diffusion phenomena. This fact also has important consequences on the attempt to scale-model atmospheric flow problems within wind tunnels, which inherently restrict turbulent fluctuation to scales less than about one-tenth of the characteristic dimension of the tunnel (Cermak, 1971).

The distribution parameters σ_y and σ_z can be written in the form

$$\sigma_j = a_j x^{b_j} \qquad (j = y,z) \qquad \text{(VI A-3)}$$

Both a and b may be dependent on atmospheric turbulence intensity or such related parameters as thermal stability, wind speed, local surface roughness, and height. Smith and Hay (1961) suggested the form

$$\sigma_j = \frac{2}{3}\beta i_j^2 x$$

as representative of cluster growth over a large size range; here, i_j is the intensity of turbulence, $i_j = (\overline{u'^2})^{\frac{1}{2}}/u$ and β is the ratio of Lagrangian to Eulerian time scales of turbulence. Later Wandel and Kofoed-Hansen (1962) showed that β could itself be expressed as a function of i, such that

$$\sigma_j \simeq \frac{2}{9} i_j x. \qquad \text{(VI A-4)}$$

This relation, adequate to distances of x < 10 to 20 kilometers, is used in the DIFOUT computer program developed by Luna and Church (1969). Values of turbulence intensity that correspond to the Pasquill atmospheric stability categories are given in a paper by Luna and Church (1972).

The method set out by Pasquill (1961) established a series of atmospheric diffusive ability, or stability, categories that could be defined given only a minimum of data (location, time of day, cloud cover, wind speed). Because it is sufficiently simple and accurate, this technique has become widely known and used in many handbooks for atmospheric dispersal calculations (Slade, 1968; Turner, 1969; Beals, 1971). Studies have shown (Golder, 1972; Luna and Church, 1972) that such categories correspond generally to direct measurements of turbulence intensity but that there is considerable variability in observations made within each class.

Recently G. A. Briggs (summarized in Gifford, 1975) proposed a series of interpolation formulas for $\sigma_j(x)$ that agree with the so-called Pasquill-Gifford curves for 100 m < x < 10 km, except that the σ_z relations for A and B stability categories more closely approximate the "very unstable" and "unstable" curves recommended by Smith (1968).[1] The values recommended by Briggs are shown in Table VI A-1 and in Fig. VI A-2. Table VI A-1 also shows the coefficient and exponent fit to the Pasquill-Gifford curves as made by Martin and Tikvart (1968) and reported by Eimutis and Koricek (1972). The following subsections will give more detail on (1) the vertical component of diffusion, (2) mixing depth, (3) plume rise, (4) the lateral or horizontal component of diffusion, (5) building-wake effects, and (6) treatment of variable release duration.

A3.1 VERTICAL DIFFUSION

The vertical component of turbulence intensity is a strong function of thermal stability, which in turn may be quite variable with height above ground. This variation is because the atmosphere is basically a stratified fluid in hydrostatic equilibrium between gravitational and pressure-gradient forces. The temperature decrease with altitude (lapse rate) is usually smaller than the dry adiabatic rate (9.8 C/km), which is the equilibrium value. However, the ambient lapse rate, the primary determinant of stability, is frequently quite complex, with the effect that the whole atmosphere may be considered to be a superposition of many layers of air, each with its own characteristic stabilities. For layers near the ground, stability is mainly influenced by the heating characteristic of the surface such as type (dirt, water, snow, vegetation, rocks, or concrete) and its ability to absorb, retain, conduct, or reemit solar thermal energy.

If solar heating at the surface is sufficient, then the surface layer of air warms until the adiabatic lapse rate is reached, at which point the layer becomes unstable, and buoyant overturning (convection) occurs. This is thermally generated turbulence in its most basic form and results in efficient vertical mixing up to the height at which the ambient lapse rate becomes smaller than the adiabatic lapse rate. For a graphic depiction of the effect of varying stability and turbulence intensity profiles on plume behavior, see Fig. VI A-3.

The other mechanism for turbulence generation is the mechanical interaction of the wind, either with itself (shear) or with roughness elements on the ground (grass, rocks, trees, buildings, mountains). General rules have been derived for estimating turbulence intensity based on readily observed properties of the atmosphere and the surrounding terrain of interest. Such rules take into account varying roughness with flow (wind) direction, or such downwind changes as shorelines, urban-rural boundaries, and diurnal heat flux changes (sunshine, cloudiness, urban heat island).

In the present model, the stabilities at each site have been categorized by temperature structure (as specified in Regulatory Safety Guide No. 1.23) as measured at two levels on a mast, separated vertically by at least 30 meters, and more than 10 meters above the ground. Many studies have shown (e.g., Brown et al., 1975) that the values of σ_θ, the standard deviation of wind direction, are highly correlated with downwind plume width. However, problems arise during very light wind situations, when most wind vanes do not respond, in which case only temperature differences remain as a useful indicator.

[1] For a fuller description of these interpolations, see Gifford (1975).

TABLE VI A-1 RECOMMENDED FORMULAS FOR $\sigma_y(x)$ AND $\sigma_z(x)$ FOR OPEN-COUNTRY CONDITIONS $10^2 < x < 10^4$ METERS

Pasquill Stability Category	Martin and Tikvart (1968)		Briggs[a] (1973)	
	σ_y	σ_z	σ_y	σ_z
A	$0.3658x^{0.9031}$	$0.00024x^{2.094} - 9.6$	$0.22x(1 + 0.0001x)^{-1/2}$	$0.20x$
B	$0.2751x^{0.9031}$	$0.055x^{1.098} + 2.0$	$0.16x(1 + 0.0001x)^{-1/2}$	$0.12x$
C	$0.2089x^{0.9031}$	$0.113x^{0.911}$	$0.11x(1 + 0.0001x)^{-1/2}$	$0.08x(1 + 0.0002x)^{-1/2}$
D	$0.1471x^{0.9031}$	$1.26x^{0.516} - 13.0$	$0.08x(1 + 0.0001x)^{-1/2}$	$0.06x(1 + 0.0015x)^{-1/2}$
E	$0.1046x^{0.9031}$	$6.73x^{0.305} - 34.0$	$0.06x(1 + 0.0001x)^{-1/2}$	$0.03x(1 + 0.0003x)^{-1}$
F	$0.0722x^{0.9031}$	$18.05x^{0.18} - 48.6$	$0.04x(1 + 0.0001x)^{-1/2}$	$0.016x(1 + 0.0003x)^{-1}$

[a] Cited in Gifford (1975).

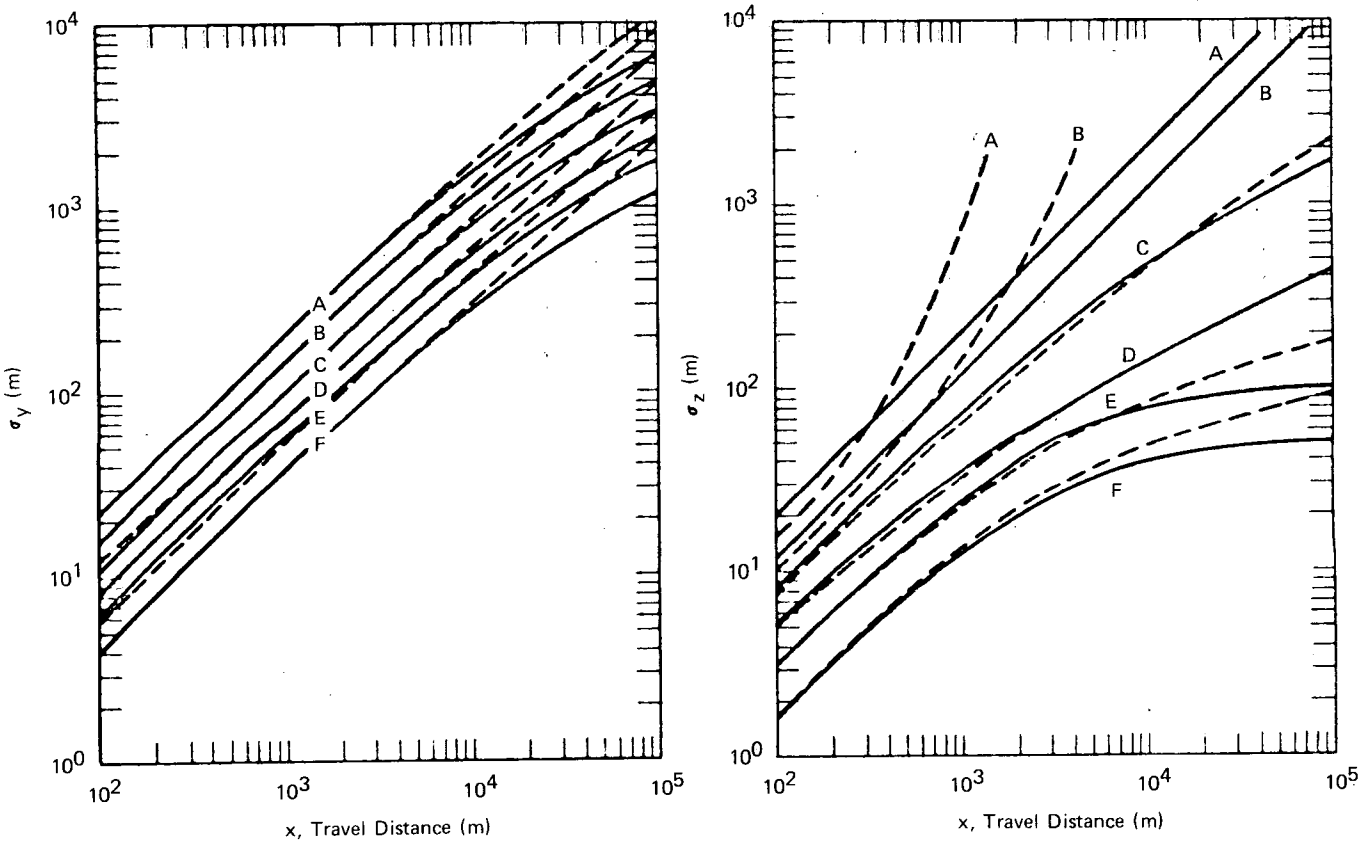

FIGURE VI A-2 Briggs rural-data-based dispersion coefficients (solid curves) compared with the Pasquill-Gifford coefficients (broken curves): (a) lateral coefficients; (b) vertical coefficients. From Hosker (1974).

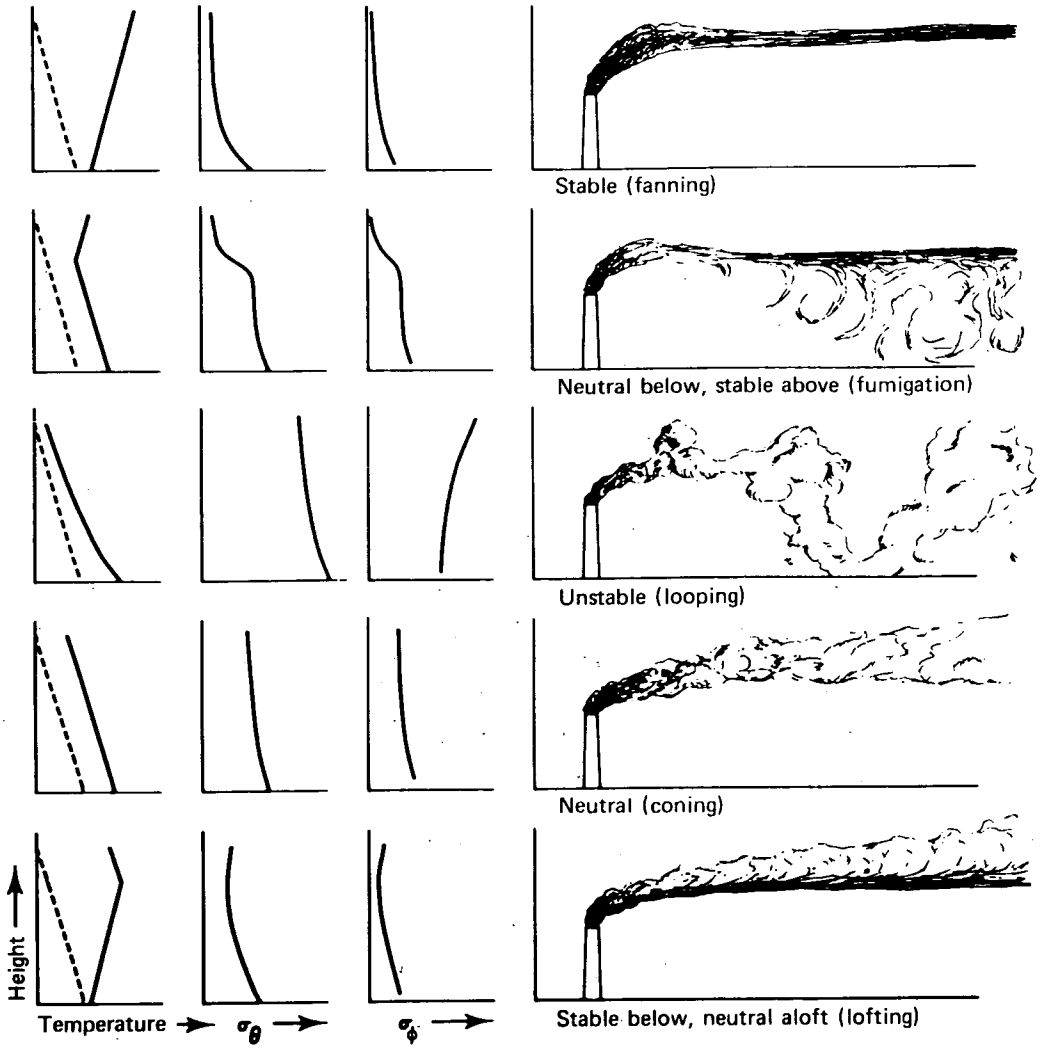

FIGURE VI A-3 Various types of smoke-plume patterns observed in the atmosphere. The broken curves in the left-hand column of diagrams show the adiabatic lapse rate, and the solid curves are the observed profiles. The abscissae of the columns for the horizontal and vertical wind direction standard deviations (σ_θ and σ_ϕ) represent a range of about 0° to 25°. From Slade (1968).

A3.2 MIXING HEIGHT

In the preceding section, allusion was made to an elevated height above ground at which thermal stability becomes greater in comparison to that below. This phenomenon can have a powerful effect on vertical diffusivity, such that mixing is essentially terminated above this level. After the mixing of material from below has filled the mixing depth L, only horizontal spreading remains as a dispersal mechanism. Holzworth (1972) derived an average pattern of mixing depth from climatological studies of twice daily measured vertical temperature profiles and hourly surface air temperature records for various weather stations. Figures VI A-4 and VI A-5 show annual average mixing heights for stable and unstable times of the day for the lower 48 states. The influence of an elevated stable layer is usually felt at relatively long distances from a ground-level release, depending on the ratio of the plume depth σ_z to the mixing height L.

A3.3 PLUME RISE

The value for plume centerline height h in Equation (VI A-2) can be determined from a combination of theory and experimental data summarized by Briggs (1969). After collecting more than 30 different formulas of different investigators, comparing them against a large body of data, and weighing them against basic theoretical considerations, Briggs concluded that the following two relations are representative. For unstable or neutral lapse rate conditions,

$$\Delta h = 1.6F^{1/3}u^{-1}x^{2/3} \qquad \text{(VI A-5)}$$

out to the distance $x = 5x^*$, where $x^* = 0.25\ Q_H^{2/5}$. For stable stratifications,

$$\Delta h = 2.9\ (F/us)^{1/3} \qquad \text{(VI A-6)}$$

out to $x = 2.4u(s)^{-1/2}$. In these equations, Δh is the plume centerline height (meters) above an assumed initial emission height (here assumed to be 25 meters); F is the buoyancy flux ($F = 3.7 \times 10^{-5}\ Q_H$); Q_H is the thermal energy release rate (calories per second), radioactive heating being neglected; u is the average wind at 25-meter height (meters per second); x is the downwind distance (meters); and $s = (g/T)(\partial\theta/\partial z)$ is a stability parameter, in reciprocal units of square seconds (sec^{-2}), where $g = 9.81$ m/sec^2, T is the temperature in degrees Kelvin, θ is the potential temperature in degrees Kelvin, and z is the height in meters. Penetration of an elevated inversion (mixing height) will occur if its height L is

$$L \leq 4F^{0.4}b^{-0.6} \text{ with no wind}$$

or

$$L \leq 2(F/ub)^{1/2} \text{ with wind,}$$

where $b = g\Delta T/T$ and ΔT is the temperature difference between the top and the bottom of the elevated inversion.

A3.4 LATERAL DIFFUSION

Much of the discussion in section A3.1 applies to the lateral component except that the spectrum of eddy sizes can extend out to about 10^4 kilometers (one global quadrant). Figure VI A-2 and Table VI A-1 showed typical functions of σ_y versus distance out to a distance of 100 kilometers for the six stability classes, although confirming data for distances beyond about 20 kilometers are scarce.

For the case of changing stability types with downwind distance (or time), a scheme such as used by Start and Wendell (1974) can be used. In this case the lateral growth is incremented through each time step as a function of the accumulated size and the prevailing characteristic stability for the particular time and distance from release being considered.

Another simplification used in Equation (VI A-2) for the width description is to replace the Gaussian crosswind shape with a rectangular, or uniform, function, sometimes called a "top-hat" distribution:

$$\{(2\pi)^{\frac{1}{2}}\sigma_y\}^{-1} \exp\ (-y^2/2\sigma_y^2) = (3\sigma_y)^{-1}, \quad (-1.5\sigma_y \leq y \leq 1.5\sigma_y)$$

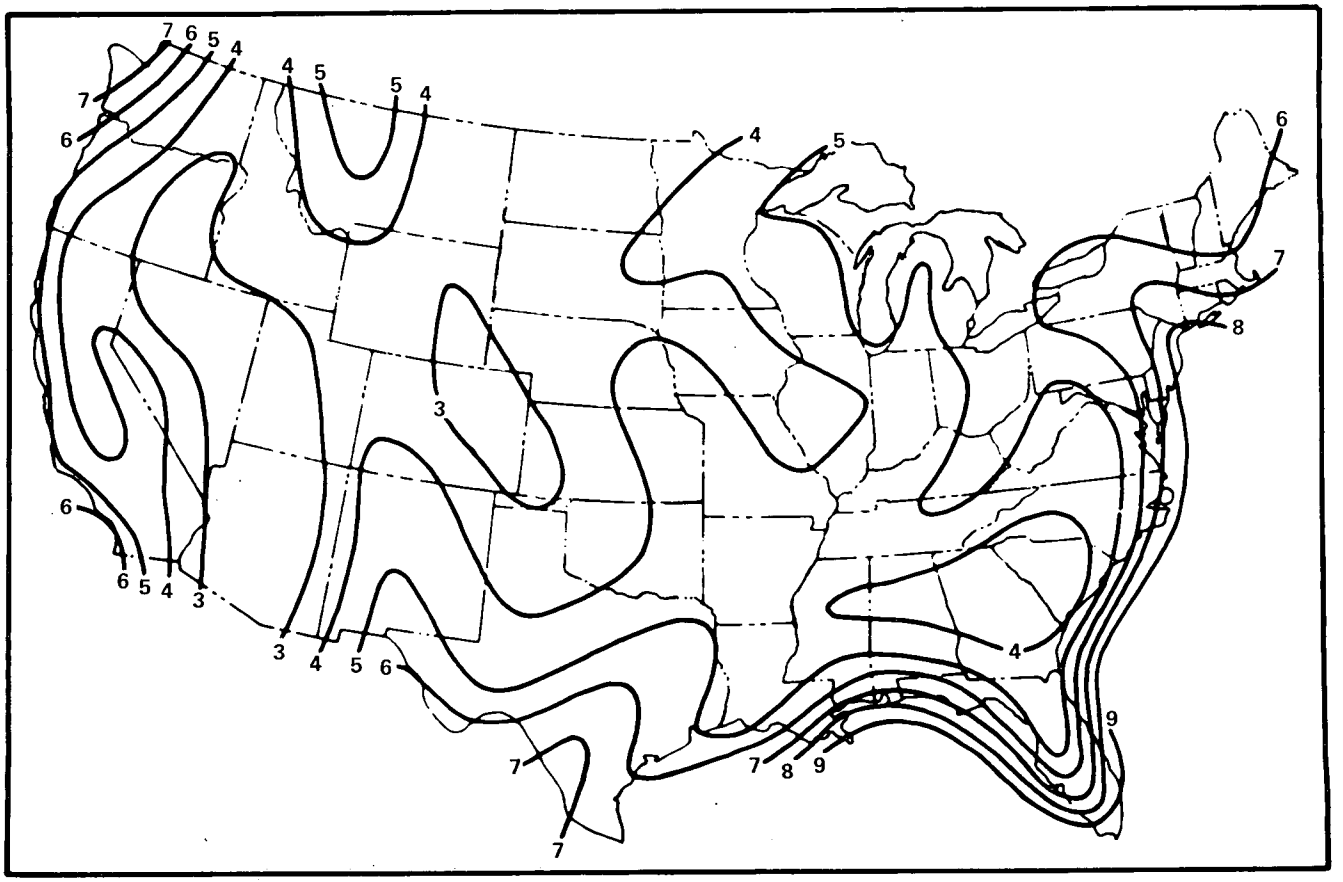

FIGURE VI A-4 Isopleths (m x 10^{-2}) of mean annual morning mixing heights. From Holzworth (1972).

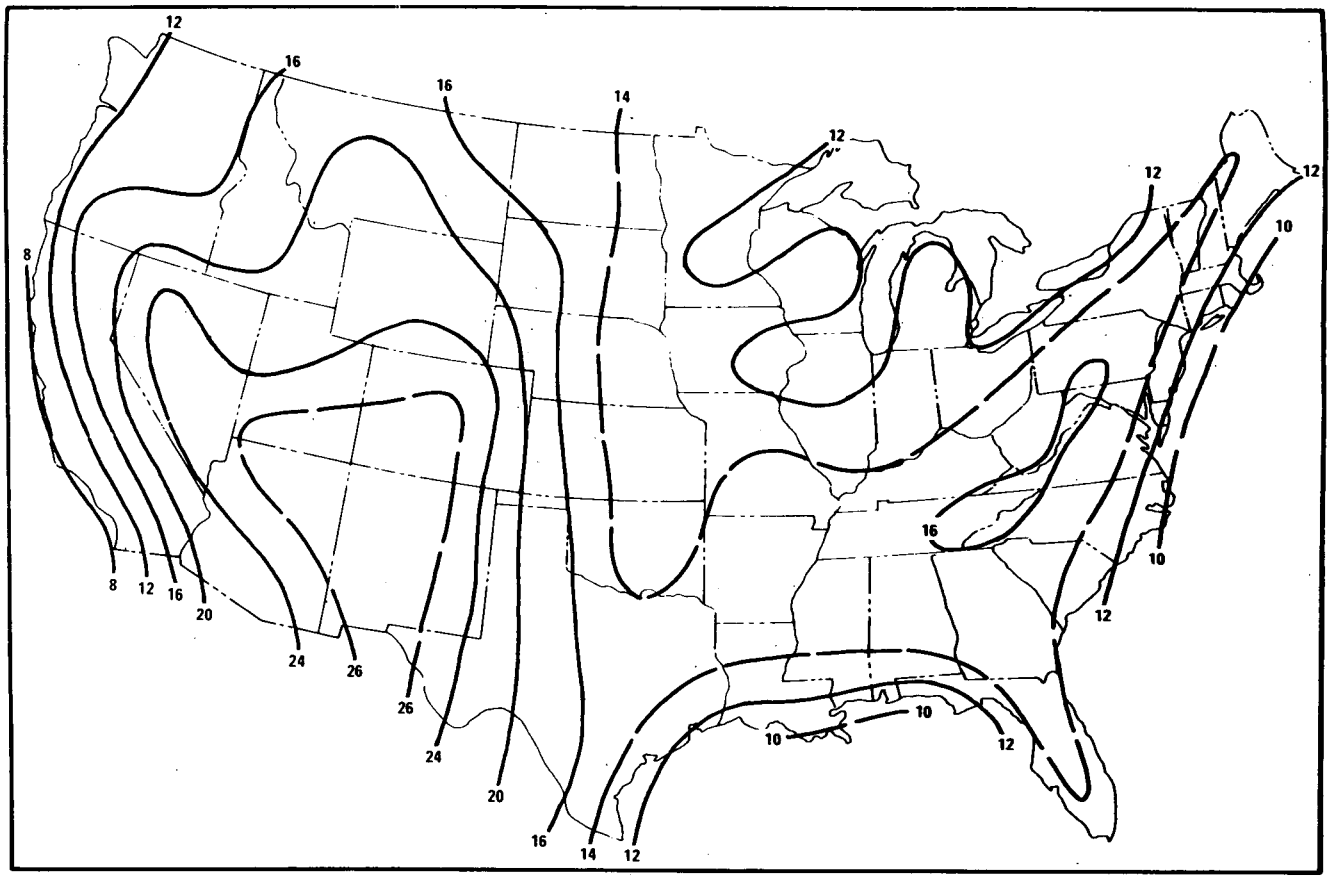

FIGURE VI A-5 Isopleths (m x 10^{-2}) of mean annual afternoon mixing heights. From Holzworth (1972).

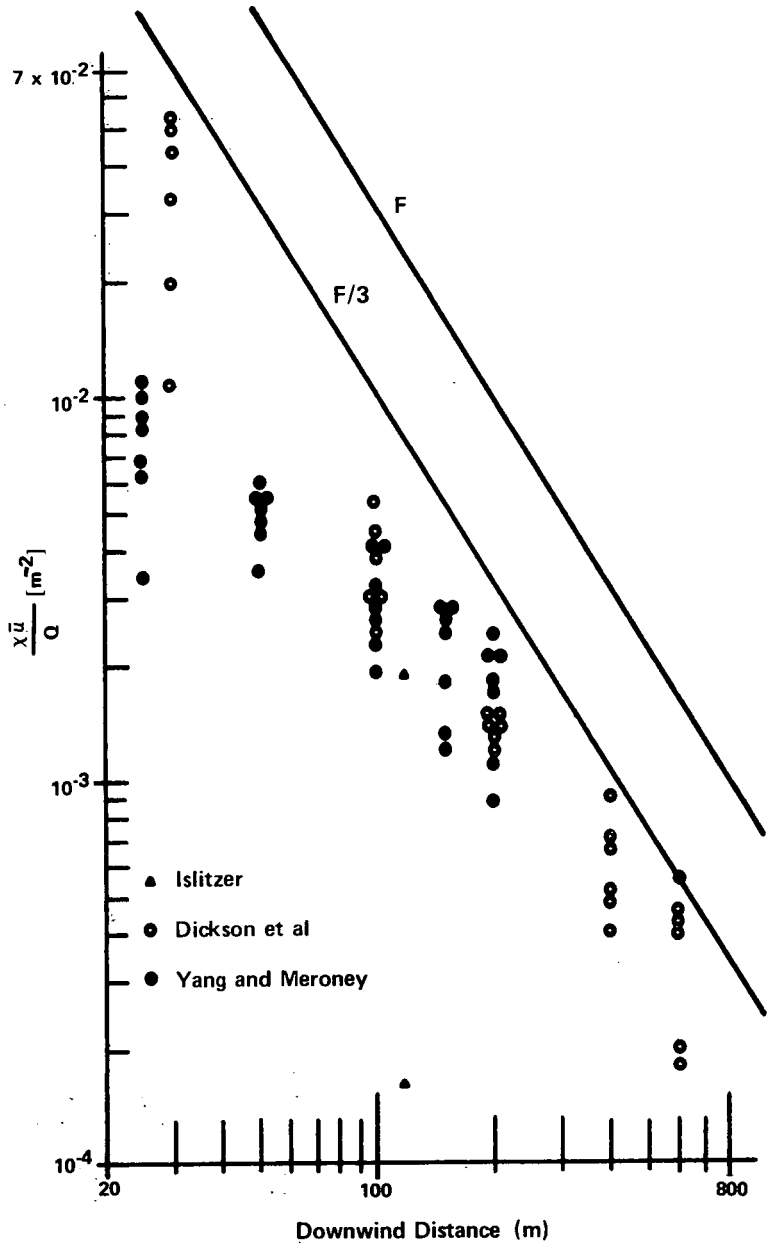

FIGURE VI A-6 Relative ground level concentrations as a function of distance and test series. The Pasquill F and F/3 curves correspond to concentrations predicted over open terrain under stable atmospheric conditions. From Abbey (1975).

This replaces the exponential shape with a square wave, which has an amplitude that is within 20% of the Gaussian peak.

A3.5 BUILDING WAKES

It has been known for some time that the presence of buildings perturbs the flow in the lowest layers of the atmosphere. Many studies have been done both in the laboratory and in the field (Dickson et al., 1967; Slade, 1968, pp. 221-255; Frost et al., 1975; Hansen et al., 1975).

Obstacle-generated wakes have been characterized by increased turbulence, a mean velocity defect, and possibly in some geometry situations, discrete standing vortices. These effects have been noted to a distance downstream of 30 or 50 building heights. The classic way of handling the turbulence-enhancement effect in the centerline diffusion equation has been to add to the plume size parameters a term that accounts for the enhanced mixing in the lee of the building:

$$\chi u/Q = (\pi\sigma_y\sigma_z + CA)^{-1}, \qquad \text{(VI A-7)}$$

where A is the building cross-sectional area downwind and C is the fraction of that area over which the plume is dispersed (on the order of 0.5).

If the building-generated turbulence is randomly spread over a broad spectrum of sizes, as is the ambient atmospheric turbulence, then clearly the plume dispersion would be more rapid than it would be if no building were present. However, if highly structured vorticity (caused by vortex shedding from the upwind roof corner) persists through the near wake of the building, the entire wake character at large distances could be significantly altered. For this to occur would require the proper conditions of flow geometry, stability, and ambient turbulence character.

Organized vortex-pair wakes have been measured in wind tunnels out to 80 heights (Hansen et al., 1975), but the extrapolation of these results to the atmosphere presents two significant problems. The tunnel's finite width and height reduce the lateral and vertical components of spread, and it is known that the turbulent line-vortex decay rate is dependent on the Reynolds number, which may be three orders of magnitude smaller in the tunnel than in the atmosphere. Hunt (1971) indicates that line vortices are persistent only if turbulent scales are smaller than the vortex length.

Some tunnel results have suggested (see Fig. VI A-6) that the plume peak concentration decreases with downwind distance to the -0.8 power, whereas Equation (VI A-7) suggests that, for the atmosphere, the power should be at least -1.5 when reasonable values of σ_y and σ_z are inserted (see Table VI A-1), each of which has exponents on x ranging from 0.7 to 1. For -0.8 to be realized would require essentially that σ_y or σ_z be held constant throughout the wake as the contaminant is carried within it. The wind-tunnel results suggest that contaminants would mix more rapidly throughout the wake than outside it, at least for as long as the wake is distinguishable from the atmospheric boundary layer. Whether or not this effect would result in higher or lower ground-level concentrations at long distances in comparison to the no-wake case is subject to considerable uncertainty. However, this effect can be important for only a few kilometers downwind of the source. It is clear that field experiments along with matched wind-tunnel tests are needed to clarify these points.

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Appendix B

Review of Deposition and Scavenging Data

B1 INTRODUCTION

To assess the consequences of the dispersal of pollutants by atmospheric turbulence, the rate of processes removing contaminants from the atmosphere must be known. These removal processes are classified as wet and dry deposition.

Dry deposition may occur by sedimentation (influence of gravity) and by impaction on obstacles on or near the earth's surface. Sedimentation is not an important removal process for aerosols with particles smaller than 15 microns, since their fall velocity is small in comparison to their vertical displacement resulting from turbulence and mean air motion. Removal by impaction on obstacles has generally been stated in terms of a deposition velocity, which is the ratio of the deposition flux to the concentration at some particular distance from the surface. Since in diffusion-controlled flow the flux is proportional to the concentration gradient, and not to a single point concentration, it is apparent that in using the deposition velocity to compute the flux it is necessary to specify the parameters that control the concentration profile and diffusion coefficient. Similarly, in flows controlled by surface resistance, the material- and temperature-dependent equilibrium constants and loading history (for vapor deposition) or the particulate resuspension rate must be known.

Wet deposition of aerosols is also referred to as precipitation scavenging. Scavenging can occur within the clouds and below the clouds. In-cloud scavenging of airborne aerosols is a consequence of (1) consumption of the aerosol as condensation nuclei during the formation of cloud droplets, (2) attachment of the aerosol to cloud elements as a result of Brownian motion, and (3) attachment of the aerosol to cloud droplets by diffusiophoresis. Below-cloud scavenging of aerosols is a process by which falling hydrometeors impact on and collect the aerosols. Wet deposition is generally described in models by a simple removal rate.

In general, wet and dry deposition of particles and gases cannot yet be specified precisely. It is known that there are significant dependencies on precipitation type, rate, and hydrometeor size distributions; on particle density, wettability, and size distributions; on gaseous chemical composition, water solubility, and reaction rates; on vegetation type, biomass, and physiological state; and on atmospheric stability, wind field, and humidity. However, experimental tests and theoretical developments have not yet been sufficiently extensive to quantify the influence of all these and other variables. Indeed, even if they were quantified, it must be acknowledged that the details of an actual release cannot yet be precisely specified, and therefore there still would remain uncertainties in describing the removal processes.

The purpose of this appendix is to discuss these removal processes and to indicate the values they might have in some commonly encountered cases of atmospheric deposition of radioactive contaminants. The last section of this appendix gives a compilation of the methods and data used in the consequence model.

B2 DEPOSITION OF IODINE VAPOR

B2.1 Dry Deposition

The dry deposition of iodine vapor takes place by turbulent diffusion to the vicinity of the deposition surfaces, molecular diffusion across a laminar sublayer, and sorption on the surface. The sorption process is influenced not only by the substrate material, but also by the concentration of iodine above the surface. The total deposition velocity v_d can be considered to be a combination of an aerodynamic part v_a which is the deposition velocity when the surface is a perfect sink, and a sorption part v_s which is the deposition velocity

when surface effects are rate limiting. The relation between the three quantities is

$$v_d = \left(\frac{1}{v_a} + \frac{1}{v_s}\right)^{-1} \qquad \text{(VI B-1)}$$

A number of attempts have been made to isolate the sorption velocity by laboratory experiments, but the results have been erratic. Silverman et al. (1973) have tabulated iodine deposition data from six sources, which indicate sorption velocities ranging from 0.001 to 0.3 cm/sec on concrete, paint, and metal surfaces. The large range of values may depend more on the variability of the boundary layer diffusion process than on the surface chemistry. Chamberlain and Chadwick (1966) measured the surface resistance of grass and clover leaves in short range field experiments and found velocities at the leaf surface of 0.5 to 2.0 cm/sec and velocities at the surface of grass of 3.4 to 16 cm/sec. Chamberlain and Chadwick's results suggest that atmospheric deposition is usually limited by aerodynamic transport processes, whereas much of the laboratory data indicate that surface resistance should be the rate-limiting process.

Similarly, Gifford and Pack (1962) cite data showing that more iodine is deposited on vegetation than on bare soil, whereas a comparison between vegetation and soil deposition (Hawley et al., 1964) indicates that they are the same.

Chamberlain (1960) has suggested the use of an equation from Sheppard (1958) to compute the deposition of iodine vapor on a perfect adsorber. This equation is

$$v_a(z) = \frac{ku_*}{\ell n(ku_* z D^{-1})} \qquad \text{(VI B-2)}$$

where k is Von Kármán's constant (= 0.4), u_* is the friction velocity, z is a reference height, and D is the diffusion coefficient for iodine in air.

Equation (VI B-2) was evaluated for a reference height of 1 meter and an assumed logarithmic wind profile. Figure VI B-1 shows the calculated deposition velocity as a function of wind speed at 10 meters for three different roughness heights. Also plotted on Fig. VI B-1 are experimental data from four sources. The large scatter in the data reflects not only uncertainties in the wind turbulence but also the surface-resistance anomalies discussed above and lack of knowledge of what fraction of the iodine is vapor or is adsorbed onto particles.

B2.2 WET DEPOSITION

The removal of iodine vapor by raindrops takes place by vapor diffusion to the drop surface, rapid equilibration with the surface liquid and subsequent diffusion into, and possible chemical reaction with, the drop. If diffusion is the controlling process, the washout coefficient Λ can be calculated from the equation

$$\Lambda = \int_0^\infty \pi D \, Sh \, sN(s) \, ds, \qquad \text{(VI B-3)}$$

where D is the gaseous diffusion coefficient in air; Sh is the Sherwood number, which is used to account for the increased diffusion of a falling drop; and N is the number of drops of diameter s in a unit volume of air. The value of Sh has been obtained for spherical drops by Ranz and Marshall (1952) and is given by

$$Sh = 2 + 0.6Re^{0.5} \, Sc^{0.33}, \qquad \text{(VI B-4)}$$

where the Reynolds number Re is dv_t/ν and the Schmidt number Sc is ν/D (v_t is the drop terminal velocity and ν is the kinematic viscosity of the air).

The washout coefficient Λ was evaluated by Engelmann et al. (1966) for five different raindrop size distributions. The value of Λ for two of these, the Best spectra (Best, 1956) and the Kelkar-Hanford spectra (Kelkar, 1959) are shown in Figure VI B-2 as a function of rainfall rate. Also shown in Fig. VI B-2 are data points taken from Engelmann et al. (1966) and Engelmann and Perkins (1966). As can be seen, the data are spread over three orders of magnitude. The points below the theoretical curves probably indicate surface resistance and saturation effects, which

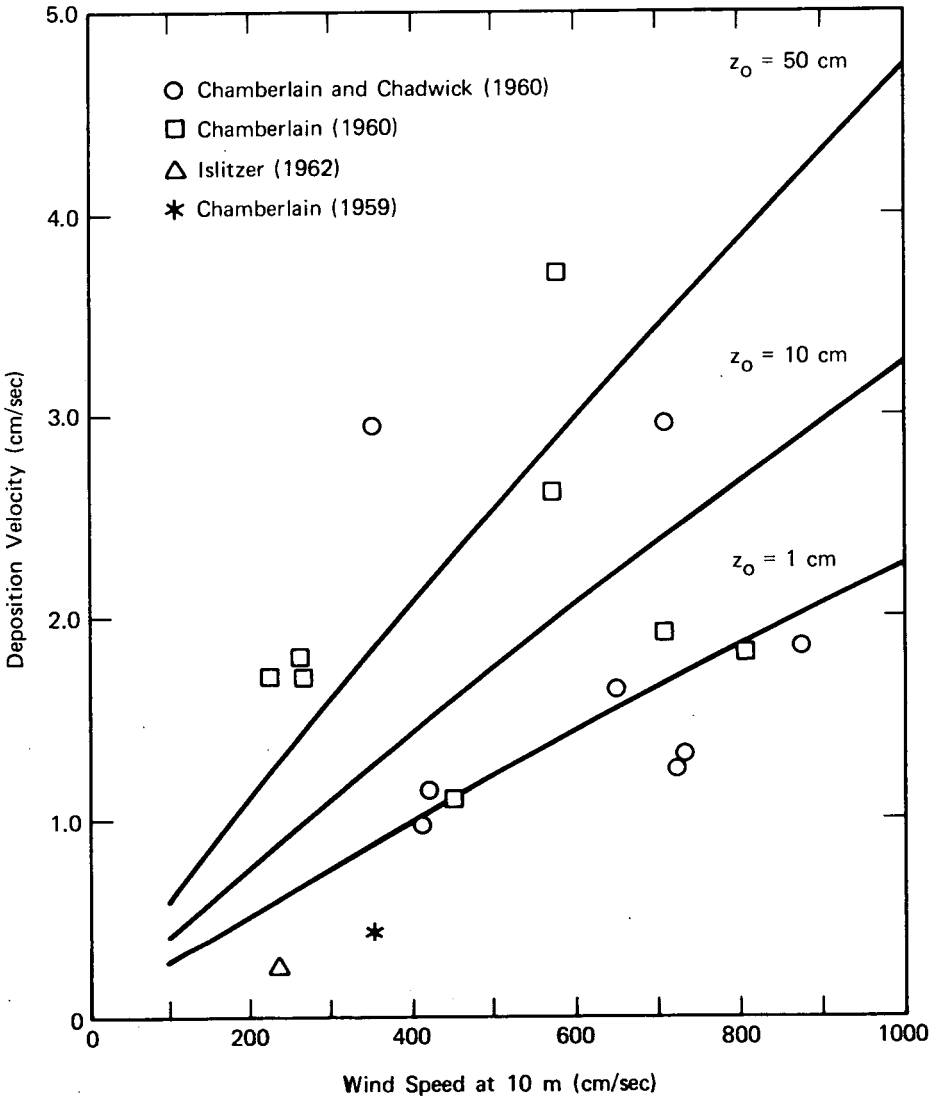

FIGURE VI B-1 Dry deposition velocity of iodine vapor.

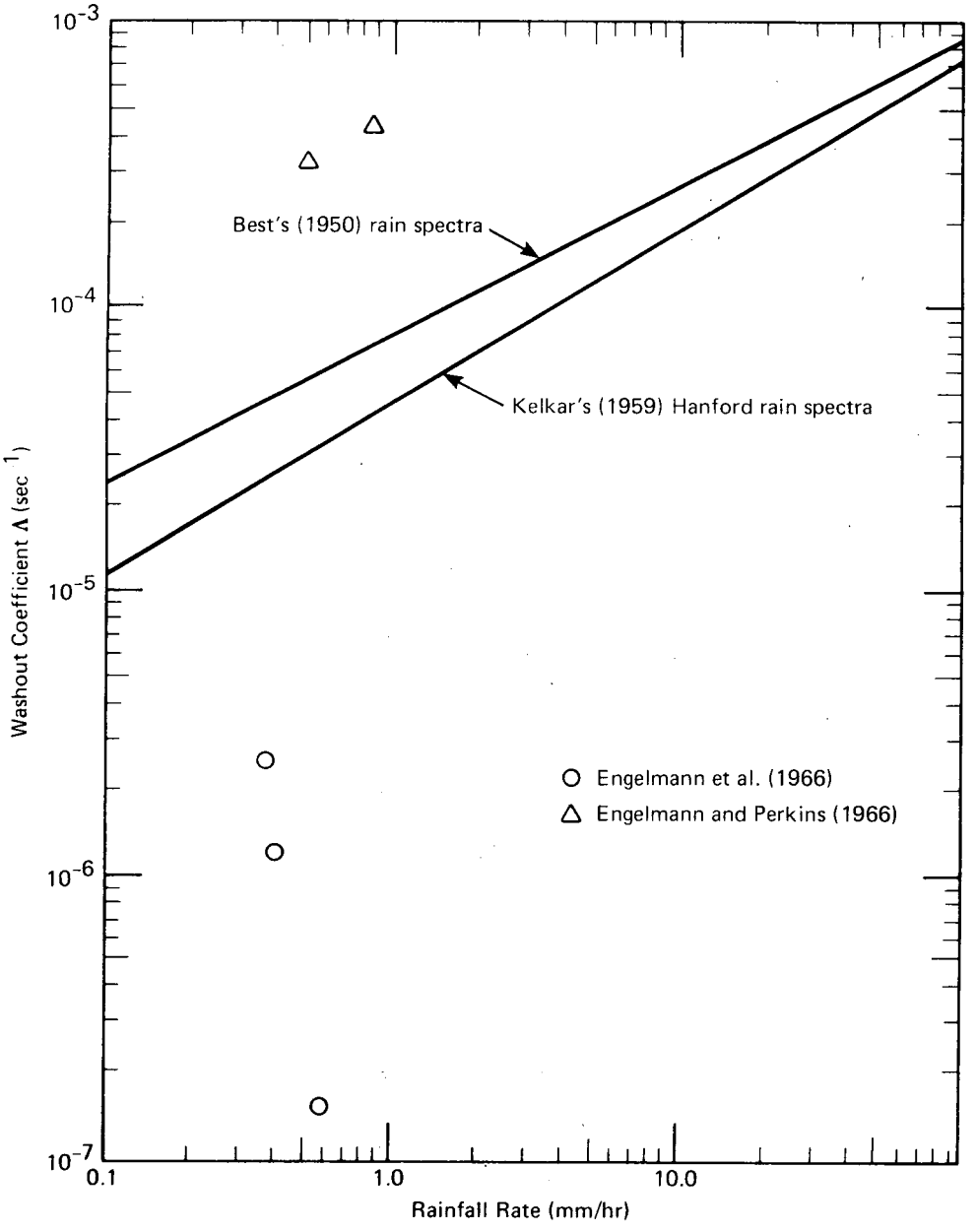

FIGURE VI B-2 Washout coefficient for iodine vapor.

were not included in the calculation. It has been postulated that the points above the curves resulted from the vapor being absorbed into a mist of condensation droplets and then scavenged out by the rain. Some snowout data, also given by Engelmann and Perkins (1966), indicate that the same value of washout coefficient can also be used to estimate iodine removal by snow. Experiments performed in connection with reactor accident studies (Hilliard et al., 1971; Nebeker et al., 1971) have yielded considerable data on iodine removal by sprays in containers. These data, however, indicate that the washout rate is a strong function of iodine concentration, which is much higher than that usually found in atmospheric deposition studies; moreover, the experiments usually used sprays containing chemical additives. For these reasons field data are preferred for making washout rate estimates.

B3 DEPOSITION OF NOBLE GASES

Because of their inert nature and low condensation temperatures, no net surface deposition of noble gases is expected. Tests on xenon (Nebeker et al., 1971) verified that, even in the prolonged (8 hours) presence of condensing steam, very little (0.008%) of the xenon dissolved in the condensate. It would appear, then, that the appropriate deposition velocity for either wet or dry deposition could be taken to be zero. One of the xenon isotopes, xenon-135, has a short half-life (9.2 hours) and decays to cesium-135, which can deposit out in particulate matter. However, since its half-life is very long, cesium-135 provides a negligible contribution to the deposited ground activity. Therefore, it may be neglected in assigning a zero deposition velocity to the noble gases.

B4 DEPOSITION OF PARTICLES

B4.1 DRY DEPOSITION

Dry removal of particles from the atmosphere is possible by sedimentation and by impaction on obstacles on or near the earth's surface. The sedimentation rate of particles depends on a balance between the aerodynamic drag force and the gravitational force exerted by the earth. The effect of sedimentation on particle deposition rates becomes negligible when the fall (or settling) velocity of the particle is much lower than the particle velocity controlled by vertical turbulence and mean air motions. This occurs when the fall velocity is lower than about 1 cm/sec (for particles smaller than 15 microns). The efficiency of impaction as a removal mechanism is demonstrated by the fact that sea-salt particles have been observed at much lower concentration near the ground than at an altitude of 300 meters and that the sea-salt content in river water is about four times that in collected rainwater. Sea-salt particles are generally in the 0.1- to 3.0-micron size range (Mason, 1971).

Methods by which particles reach surfaces have been the subject of considerable laboratory and field experimentation. Most measurements of "deposition velocity" have been made by comparing the deposition flux on a surface to the airborne particle concentration above the surface. Values have varied widely and exhibit strong dependencies on surface type, particle size, wind speed, and turbulence characteristics. Because atmospheric turbulence is generally much broader in scale size and more intense than turbulence in an enclosure (including wind tunnels), it is generally believed that deposition rates will be larger outside structures than inside them. Thus, except for a building containing an active source emission, the deposition on exterior surfaces will generally be larger than that on interior surfaces.

Essentially no experiments on deposition on vertical surfaces have been performed with real buildings. However, measurements have been made under laboratory conditions by Wells and Chamberlain (1967) and by Sehmel (1973). The results of Sehmel (1973), given in Fig. VI B-3, show the deposition velocity as a function of particle size for the floor, wall, and ceiling. As one might intuitively expect, the deposition velocity was highest on the floor except for particles smaller than 0.1 micron, in which case the orientation of the surface becomes less important. In the particle size range of 0.1 to 10 microns the wall deposition velocity was 20 to 40% of the floor deposition velocity; in this size range the ceiling deposition velocity was negligible in comparison to the other two. With curves such as these and with some idea of the particle size distribution of an aerosol, it is possible,

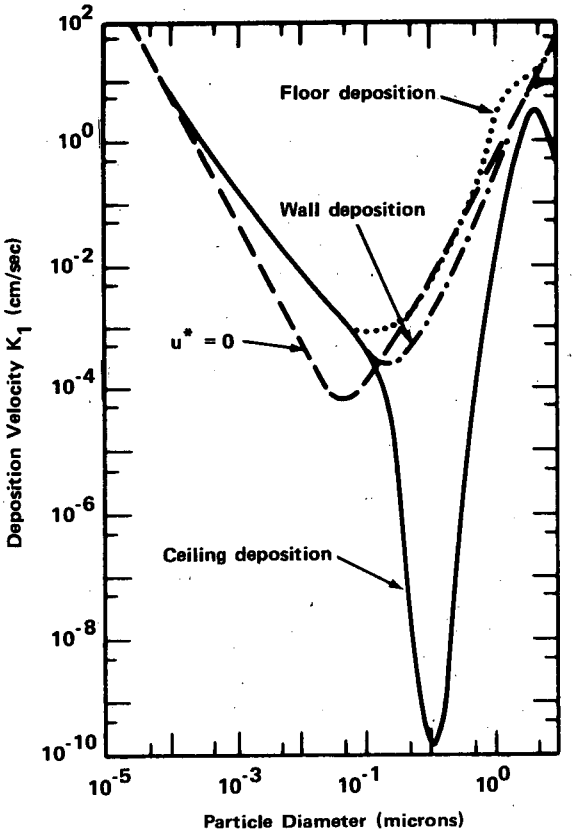

FIGURE VI B-3 Comparison of deposition velocities to smoother floor , wall, and ceiling surfaces for u* = 34.1 cm/sec and y_o = 0.004 cm; nominal velocity of 670 cm/sec. From Sehmel (1973). Reprinted by permission from Pergamon Press.

at least in theory, to estimate total deposition in a complex geometrical surface situation (e.g., an urban building cluster).

The size distribution of an aerosol may be quite uncertain because of changes that can occur. Measurements and calculations of atmospheric aerosols made by Junge (1963) lead to the conclusion that within several hours of release into the atmosphere a quasi-steady-state distribution is approached. The changing distribution is a result of particle-size-dependent coagulation and deposition mechanisms that operate continuously.

B4.2 WET DEPOSITION

Precipitation scavenging processes are classified as wet deposition. As mentioned in the introduction, the mechanisms responsible for the wet deposition of particles are (1) consumption as condensation nuclei during the formation of cloud droplets, (2) attachment to cloud droplets and other particles as a result of Brownian motion, (3) attachment to cloud droplets by diffusiophoresis, and (4) collection by falling hydrometeors (raindrops, hail, snowflakes). These four mechanisms are discussed separately below.

Measurements indicate that the concentrations of salt nuclei and maritime cloud droplets over midocean (removed from land contaminating effects) are similar, having typical values of about 10 per cubic centimeter. Cloud-level measurements over continental regions indicate that there are about 300 droplets per cubic centimeter, formed on all aerosol particles larger than 0.1 micron in radius. Thus, it is concluded that the consumption of particles that are larger than 0.1 micron in radius and are thus involved in water cloud formation is quite efficient (Mason, 1971).

The effects of Brownian motion are important for particles smaller than 0.1 micron in two significant ways. They can be captured directly on cloud droplets (whose sizes are on the order of 10 microns), or they will coagulate, such that the population of very small particles is reduced in favor of larger particles, which can then participate in more efficient removal mechanisms. The average picture for the natural aerosol is a dynamic one in which small particles (<0.01 micron) are continually formed from such processes as combustion, chemical reactions, erosion, and emission from vegetation, followed by coagulation effects shifting the size distribution in such a way that removal phenomena operate.

Diffusiophoresis operates in the vicinity of an evaporating or condensing surface and causes nearby particles to experience a force in the direction of the diffusive flux of vapor. However, calculations of this effect have shown it to be very slow in comparison to Brownian-motion capture effects for submicron particles.

Of the four wet deposition processes, the last is the removal of particles below the cloud by the impaction of falling hydrometeors. Consideration of the size spectra of particles and hydrometeors and their collection cross sections and path lengths suggests that the collection efficiency for particles smaller than 1 micron is practically zero, whereas those larger than 5 to 10 microns are very efficiently captured and removed.

The fraction of material removed from a contaminant plume in unit time is called the *washout coefficient* Λ and is best defined by

$$\Lambda = k/\chi, \qquad \text{(VI B-5)}$$

where k is the rate of material removal from the atmosphere (particles removed per unit volume of atmosphere per unit time) and χ is the local particle concentration. Predicted values of the washout coefficient cover a broad range (from approximately 10^{-7} to 10^{-2} per second). It is known that the washout coefficient is dependent on precipitation type and rate as well as the size distribution of hydrometeors; on particle density, wettability, and size distribution; on gaseous chemical composition, water solubility, and reaction rates; on vegetation type, biomass, and physiological state; and on atmospheric stability, wind field, and humidity.

An experimental determination of the washout coefficient is difficult, and the results are hard to interpret. To determine Λ theoretically, some idealization of natural conditions is mandatory. Most attempts at controlled tracer release experiments have utilized the scheme of May (1958), which requires a steady-state plume (not meandering or varying in source strength), vertical rainfall, and no competing removal processes (such as dry deposition). Often the washout coefficient determined in this manner represents a very limited situation and is difficult to compare with theoretical calculations.

B5 SUMMARY AND MODEL DEVELOPMENT

Based on a consideration of the state-of-the-art knowledge briefly discussed in the preceding sections, it is obvious that wet and dry deposition of particles and gases cannot yet be specified precisely. What is possible is to bound and to approximate the removal processes. This can be performed with the following concepts:

a. A simple exponential wet-removal process and a constant deposition velocity should be adequate if sufficiently broad ranges of the parameters are accommodated.

b. The net removal rates cannot be less than zero, and therefore the air concentrations will not be greater than those derived ignoring removal.

c. The removal rates cannot be larger than the values dictated by the removal capacity of the atmosphere, and therefore maximum possible deposition amounts can be estimated.

d. Field experimental data can be used to provide expected average removal rates and expected upper and lower bounds.

B5.1 DRY DEPOSITION OF PARTICLES AND GASES

With consideration of the above, the dry deposition flux of material to the surface is taken as $v_d\chi_0$, where v_d is the deposition velocity and χ_0 is the air concentration at essentially ground level. Within a given distance of plume travel, the total quantity of material deposited is subtracted from the source (plume). The fraction of the source deposited is calculated as

$$f = 1 - \exp\,(-v_d t_i \,/\, \bar{z}), \qquad \text{(VI B-6)}$$

where t_i is the time required for the plume to traverse a spatial interval (determined by wind velocity) and $\bar{z}$ is the effective "height" of the plume, defined by

$$z = \frac{1}{X(0)} \int_0^\infty X(z)\, dz = (\pi/2)^{\frac{1}{2}}\sigma_z \exp(h^2/2\sigma_z^2). \qquad \text{(VI B-7)}$$

Since $\bar{z}$ is large and/or t_i is small, f can be approximated by

$$f \simeq 1 - v_d t_i \,/\, \bar{z}$$

with little error.

B5.2 WET DEPOSITION OF PARTICLES AND GASES

Wet deposition is described by a simple removal rate. The rate of reduction of the air concentration, χ, is taken to be $-\Lambda\chi$, where Λ is the wet removal rate. The plume concentration decreases because of precipitation scavenging according to

$$f = \exp\left[-\Lambda(t - t_0)\right], \qquad \text{(VI B-8)}$$

where $t - t_0$ is the time since the onset of precipitation at time t_0.

B5.3 VALUES USED FOR DEPOSITION VELOCITY AND WET REMOVAL RATE

Expected values for Λ and v_d are obtained from field experimental data. The range about the average expected value (i.e., the minimum and maximum expected values)

generally covers the spread in the field data. The average values are deduced from the following sources:

a. Process plant inorganic iodine scavenged by rain and snow from beneath clouds (Engelmann, 1968).

b. Micron-size polydisperse aerosols scavenged by rain from beneath clouds (Dana, 1970).

c. Submicron-size polydisperse aerosols removed from within stable (Davis and Young, 1975) and unstable storms (Burtsev et al., 1970; Dingle, 1975; Gatz, 1975; Young et al., 1975).

d. Sulfur dioxide (Garland, 1975; Unsworth and Fowler, 1975) and iodine (Van Der Hoven, 1968; Heinemann and Vogt, 1975) dry deposited on vegetation.

e. Fallout and tracer particle deposition on a variety of surfaces and for a variety of meteorological conditions (Small, 1969; Cawse and Peirson, 1972; Krey, 1975; Sehmel and Hodgson, 1975).

The bounds and approximations for the wet and dry removal of particles and gases are given in Table VI B-1.

TABLE VI B-1 BOUNDS AND APPROXIMATIONS FOR WET AND DRY REMOVAL OF PARTICLES AND GASES

	Minimum Expected[a]	Average Expected[b]	Maximum Expected
Wet removal[c] rate (sec^{-1})	10^{-5}	Stable[d]: 10^{-4} Not stable[e]: 10^{-3}	10^{-2}
Dry deposition velocity (m/sec)	10^{-3}	10^{-2}	10^{-1}

(a) Except for gases barely soluble in water (e.g., the noble gases); then use zero.
(b) Based on field experimental data for micron-size particles and soluble gases.
(c) Multiply tabulated numerical values by ($p/1$ mm hr^{-1}), where p is the precipitation rate in rainwater equivalent.
(d) Warm frontal storm.
(e) Convective storm.

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Appendix C

External Dosimetry

All external dose factors were computed by means of the EXREM III computer program developed at the Oak Ridge National Laboratory. The operation of the program, derivation of the equations, and applications of the program have been reviewed in detail by Trubey and Kaye (1973). This appendix describes certain aspects of EXREM III that were used to compute external radiation dose factors for the present application.

Only photon doses resulting from immersion in contaminated air and exposure to a contaminated land surface were considered. The dose rate of the ith radionuclide, D_{is}, from exposure to a contaminated land surface was calculated from the equation

$$D_{is} = 827BC_i \sum_{n=1}^{N_i} [\sigma_{in}E_1(\sigma_{in}x)f_{in}E_{in}] \quad \text{(rads/hr)}, \qquad \text{(VI C-1)}$$

where

827 = constant to convert a uniformly distributed concentration (source of plane infinite extent) of a radionuclide to a dose rate,

B = backscatter correction (dimensionless) for a body immersed in air,

C_i = concentration (microcuries per square centimeter) of the ith radionuclide on the land surface,

n = photon index,

N_i = number of photons emitted by the ith radionuclide,

σ_{in} = linear energy absorption coefficient (per centimeter), $\sigma_{in} = \sigma(E_{in})$,

$E_1(\sigma_{in}x)$ = exponential integral (E-function) of the first order,

x = distance (centimeters) from the exposed individual to the contaminated surface,

f_{in} = abundance (dimensionless) of the nth photon emitted by the ith radionuclide,

E_{in} = energy (MeV) of the nth photon emitted by the ith radionuclide.

The dose rate of the ith radionuclide, D_{ia}, from immersion in contaminated air was calculated from the equation

$$D_{ia} = C_i \sum_{n=1}^{N_i} (f_{in}E_{in}) \quad \text{(rads/hr)}, \qquad \text{(VI C-2)}$$

where

C_i = the concentration (in microcuries per cubic centimeter) of the ith radionuclide in air and the other terms are as defined for Equation (VI C-1).

The calculations with Equation (VI C-1) were done with x = 100 cm and no corrections for ground roughness or radiation buildup. The backscatter correction factor B was set equal to 1.14. Immersion calculations with Equation (VI C-2) assumed that a man was immersed in a hemispherical (2π geometry) cloud with an infinite radius (i.e., at least five mean free paths). The density of air used in the immersion calculations was 1.2×10^{-3} g/cm^3.

In general, the external dose to man associated with a radionuclide depends on the distribution of the radionuclide in the environmental medium to which the reference individual is exposed, on its decay and the buildup of radioactive progeny, and on the dynamics of environmental removal and replacement of the nuclide and each daughter product. Dosimetric quantities discussed for the following equations generally assume a homogeneous distribution of radioactivity throughout a large region of the medium. The calculation of the external dose to an exposed individual requires that the concentration of each nuclide in the medium be determined as a function of time. When radioactive progeny are produced, the concentrations of the various species in the chain are interrelated. Their kinetics in an environmental compartment are generally governed by a system of ordinary linear differential equations of the form

$$dC_1/dt = -(\lambda_1^R + \lambda_1^E)C_1 + I_1(t),$$

$$dC_i/dt = -(\lambda_i^R + \lambda_i^E)C_i + \lambda_i^R \sum_{j=1}^{i-1} f_{ij}C_j + I_i(t) \qquad (i=2,\ldots,n), \qquad \text{(VI C-3)}$$

where

n = number of species in the chain;

i = index whose value indicates the position of a species in the chain;

$C_i = C_i(t)$ = activity concentration of the ith species in the medium at time t (microcuries per square centimeter or cubic centimeter for land surface and air, respectively);

λ_i^R = radioactive decay constant (per hour) of the ith species = $\ln 2/T_i^R$, where T_i^R is the radioactive half-life (hours) of the ith species;

λ_i^E = environmental removal rate constant (per hour) for all the first-order environmental removal processes that apply to the ith species;

f_{ij} = branching ratio of the jth species to the ith, where j is less than i;

$I_i(t)$ = rate of production at time t of the ith species per unit quantity (microcuries per square centimeter or cubic centimeter);

t = time (hours).

The calculations with EXREM III were set up so that all daughter contributions to dose were added to the parent radionuclide of the decay chain.

Gamma-ray dose rates were computed with EXREM III according to the general rule

$$D_{ip}(t) = \sum_j H_{ij}(t)F_{jp}, \qquad \text{(VI C-4)}$$

where

$D_{ip}(t)$ = the dose rate (rem per hour) for the pth mode of exposure (air immersion or surface source) from the ith radionuclide at time t,

$H_{ij}(t)$ = the expected rate of energy emission in the jth group by the ith radionuclide present at time t,

F_{jp} = the dose in the pth mode of exposure that would be delivered from one unit of energy in the jth energy group.

The terms F_{jp} and $H_{ij}(t)$ are not simple quantities, but are computed intermediate results in EXREM III.

Dose rates to organs are defined by

$$DORG_{ipr}(t) = \sum_j F_{jp} H_{ij}(t) G_{jpr}, \qquad \text{(VI C-5)}$$

where

$DORG_{ipr}(t)$ = the dose rate (rem per hour) from the ith radionuclide in the pth exposure mode to the rth organ at time t,

G_{jpr} = the ratio of the dose rate to organ r to the dose rate to skin for a unit of energy in the jth group and exposure in the pth mode.

A new quantity $ORGMUL_{ipr}(t)$ is defined such that

$$ORGMUL_{ipr}(t) = \frac{DORG_{ipr}(t)}{D_{ip}(t)} = \frac{\sum_j F_{jp} H_{ij}(t) G_{jpr}}{\sum_j F_{jp} H_{ij}(t)}. \qquad \text{(VI C-6)}$$

As a practical matter, data for G are available only for air immersion, that is, only for G^{ai}_{jr}. Therefore, ORGMUL is computed only for the air-immersion mode and the same factor is used to determine organ dose rates for other exposure modes. The organ dose is finally calculated by

$$DORG_{ipr}(t) = D_{ip}(t)\ ORGMUL^{ai}_{ir}(t).$$

Poston and Snyder (1974), using Monte Carlo calculations and additional extrapolation procedures, have evaluated various organ dose rates for a phantom immersed in air containing monoenergetic gamma-ray emitters. The calculation was performed for a total of 12 different energies. An adult phantom with 85 geometrical subregions was used. Schematic drawings provided the location of each organ according to subregion in the phantom. For each photon energy, the average dose in each subregion was calculated and depth-dose profiles were constructed. By combining the depth-dose profiles and the schematic drawings that give organ location by subregion, the absorbed dose to each organ or region was estimated by Poston and Snyder. In this fashion they computed the matrix $[Q_{jr}]$, where Q_{jr} is defined as the dose rate to the rth organ from immersion in air containing a unit activity of gamma-ray energy in the jth group. The dose calculated for skin was the average dose over a skin depth of 0.2 cm. If skin is the organ for which r=1, the quantity G is defined by

$$G^{ai}_{jr} = Q_{jr}/Q_{j1}$$

for other organs.

The dose-conversion factors for immersion in contaminated air are presented in Table VI C-1, and the dose-conversion factors for exposure to contaminated ground are in Table VI C-2.

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TABLE VI C-1 PHOTON DOSE-CONVERSION FACTORS FOR IMMERSION IN CONTAMINATED AIR (rem per Ci-sec/m^3)

Radionuclide	Whole Body[a]	Total Marrow	Lung	Testes
CO-58	2.16E-01	2.40E-01	2.01E-01	2.01E-01
CO-60	6.00E-01	6.31E-01	5.67E-01	4.99E-01
KR-85	4.75E-04	5.78E-04	4.47E-04	5.28E-04
KR-85M	3.64E-02	5.50E-02	3.22E-02	4.78E-02
KR-87	1.01E-01	1.02E-01	1.72E-01	1.32E-01
KR-88	4.07E-01	4.83E-01	4.47E-01	2.33E-01
RB-86	2.07E-02	2.27E-02	1.84E-02	1.97E-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	1.93E-01	1.60E-01	1.64E-01
Y-90	0.0	0.0	0.0	0.0
Y-91	6.25E-04	6.39E-04	5.94E-04	4.67E-04
ZR-95	1.62E-01	1.87E-01	1.52E-01	1.51E-01
ZR-97	4.22E-02	4.72E-02	4.00E-02	3.36E-02
NB-95	1.66E-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	3.06E-02	5.42E-02	2.54E-02	2.84E-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	4.69E-02
RH-105	1.82E-02	2.74E-02	1.61E-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	1.81E-02	1.35E-02	1.61E-02
TE-129M	7.83E-03	9.92E-03	6.97E-03	8.36E-03
TE-131M	3.14E-01	3.56E-01	2.94E-01	2.35E-01
TE-132	4.75E-02	7.31E-02	4.19E-02	6.14E-02
SB-127	1.51E-01	1.84E-01	1.43E-01	1.63E-01
SB-129	2.68E-01	2.97E-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	5.89E-01	4.83E-01	5.03E-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	8.97E-03	9.28E-03
XE-135	5.67E-02	9.47E-02	5.06E-02	7.36E-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	1.49E-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
NP-239	3.08E-02	4.07E-02	2.65E-02	3.58E-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	8.53E-10	2.94E-10	3.65E-10
AM-241	4.56E-03	9.33E-03	3.22E-03	3.98E-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 VI C-2 PHOTON DOSE-CONVERSION FACTORS FOR EXPOSURE TO CONTAMINATED GROUND (rem per Ci/m^2) (TIME-INTEGRAL DOSE TO N DAYS) (a)

	Whole Body (b)		Total Marrow		Lung		Testes	
Radionuclide	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	8.48E+02	5.88E+03	8.89E+02	6.22E+03	7.99E+02	5.58E+03	6.91E+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
RB-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	1.68E+02	2.05E+02	1.96E+02	2.39E+02	1.59E+02	1.93E+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	8.98E-01	6.06E+00	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	3.50E+02	5.89E+02
NB-95	2.49E+02	1.64E+03	2.73E+02	1.80E+03	2.33E+02	1.54E+03	2.25E+02	1.49E+03
MO-99	7.73E+01	3.25E+02	1.07E+02	4.65E+02	6.95E+01	2.91E+02	7.95E+01	3.32E+02
TC-99M	1.52E+01	1.62E+01	2.70E+01	2.88E+01	1.26E+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	1.95E+02	1.30E+03
RU-105	7.23E+01	7.94E+01	9.04E+01	9.99E+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
RH-105	2.20E+01	5.67E+01	3.32E+01	8.55E+01	1.95E+01	5.01E+01	2.87E+01	7.37E+01
TE-127	6.77E-01	8.13E-01	8.43E-01	1.01E+00	6.38E-01	7.67E-01	7.58E-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	1.98E+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	3.28E+01	2.22E+02	4.04E+01	2.74E+02
TE-131M	3.31E+02	9.60E+02	4.36E+02	1.19E+03	3.56E+02	8.90E+02	3.47E+02	8.86E+02
TE-132	6.77E+02	3.98E+03	8.04E+02	3.63E+03	6.36E+02	2.88E+03	6.87E+02	3.08E+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	7.08E+02	1.59E+02	8.73E+02	1.21E+02	6.53E+02	1.43E+02	7.85E+02
I-132	1.07E+02	1.07E+02	1.23E+02	1.23E+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	2.85E+02	2.77E+02	3.19E+02	2.38E+02	2.69E+02	2.18E+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	6.84E+02	4.10E+03	7.79E+02	4.68E+03	6.37E+02	3.82E+03	6.33E+02	3.82E+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	3.98E+03	2.02E+02	3.46E+03	1.98E+02	3.11E+03
LA-140	6.40E+02	1.80E+03	6.88E+02	1.92E+03	6.12E+02	1.71E+03	5.28E+02	1.46E+03
CE-141	2.77E+01	1.82E+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	3.08E+02	8.06E+01	2.00E+02	1.05E+02	2.59E+02
CE-144	1.72E+01	1.20E+02	2.39E+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	5.89E+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	1.02E-01	1.14E+00	7.19E-01	4.97E+00
PU-239	3.76E-01	2.63E+00	3.56E-01	2.49E+00	8.88E-02	6.22E-01	3.18E-01	2.23E+00
PU-240	7.84E-01	5.47E+00	6.53E-01	4.57E+00	1.55E-01	1.08E+00	6.25E-01	4.38E+00
PU-241	4.52E-05	2.23E-03	9.23E-05	4.52E-03	3.18E-05	1.56E-03	3.95E-05	1.94E-03
AM-241	2.06E+01	1.43E+02	4.21E+01	2.95E+02	1.45E+01	1.02E+02	1.80E+01	1.26E+02
CM-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
CM-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.

Appendix D

Internal Dosimetry

D1 INTRODUCTION

This appendix considers the problem of evaluating the dose to a varied population from exposure to 54 radionuclides in various chemical forms and in various forms of particulate dispersion. Each chemical form of each element has different specific properties of solubility, transfer across membranes, distribution among the various tissues in the body, in some cases deposition in certain tissues, and finally excretion from the body.

Each radionuclide emits different radiation. Furthermore, though an organ would be affected by any radiation from the nuclides deposited in it, gamma rays, the most numerous kind, irradiate adjacent organs -- a circumstance that presents formidable geometric problems in evaluation.

The principal factors considered include the following:

1. The chemical form in which the various nuclides occur.
2. The relative abundance of the nuclide.
3. The characteristics of the aerosol or the fine powder in which the nuclide occurs.
4. The aerodynamic behavior of the aerosol particles as they are inhaled and deposited in various sections of the respiratory system.
5. The movement of particles within the respiratory tract and out of it into the lymphatic system and the gastrointestinal tract.
6. The absorption of the nuclide into the bloodstream.
7. The distribution of the nuclide among organs and tissues.
8. The retention of the nuclide by the body.

In the present work, each nuclide is considered separately as a reference input of 1 microcurie. The chemical and physiological behavior of the chemical forms in which each element might be expected are considered in assigning to each nuclide one of more of the respiratory clearance half-times, or solubility classes D, W, or Y, according to whether the longest of these half-times are of the magnitude of days, weeks, or years. For example, plutonium might be expected as the insoluble dioxide or as a soluble form such as the nitrate. These compounds belong to the Y and the W classes, respectively. The dose that would result from the inhalation of 1 microcurie of oxide is calculated, and also the dose that would result from the inhalation of 1 microcurie of the nitrate. These calculations cover the entire scope of distribution among the various tissues, selective deposition, decrease in amount by radioactive decay, and excretion. The results are presented as the cumulative dose at a series of times.

To investigate the effects of some specific accident, the proportion of the two chemical species that would result from the specific accident is taken, and the dose from the two inputs is apportioned appropriately. If the accident considered would yield 90% of the plutonium as dioxide and 10% as nitrate (or some equivalent form), then 90% of the calculated dose from 1 microcurie of the dioxide and 10% of the calculated dose from 1 microcurie of the nitrate are added to obtain the total dose expected from the nuclide. Certain organic forms of plutonium are known that are soluble and stable and therefore have a respiratory clearance half-time that is measured in days or fractions of a day. Such compounds are not included in the model because they are not expected to occur as the result of an accident.

The lung clearance classes for which doses were calculated are indicated in Table VI D-1 for each nuclide.

D2 ORGANS STUDIED

The selection of organs for which dose or dose equivalent is estimated is governed largely by the BEIR Report (1972), which gives estimates of risk for certain organs and a separate value for risk to "other tissues." The organs specified are the following: lungs; bone marrow; bone and endosteal cells of bone; the gastrointestinal tract, comprising the stomach, small intestine, upper large intestine, and lower large intestine; thyroid; breast; and "other tissues" (i.e., the rest of the body excluding the tissues named). In addition, ovaries and testes are included here because of the interest in genetic dose. The dose equivalent is estimated for all these organs and tissues except the breast. This exception is made because none of the radionuclides included in this study is known to concentrate to any appreciable degree in the breast, and thus the breast may be considered as a portion of the "other tissues" for the purposes of dose evaluation.

D3 DOSE EQUIVALENTS

The values of the quality factor Q, of the distribution factor N, and of any other modifying factors used in calculating the dose equivalent in rem are those used by the International Commission on Radiological Protection (ICRP) and the National Council on Radiation Protection and Measurements (NCRP) in calculations of internal dose. These include Q = 1 for all photons and for electrons or positrons and Q = 10 for neutrons, which here are limited to those arising from spontaneous fission and are principally above 10 keV in energy (NCRP, 1971, p. 83). For alpha particles, Q = 10 is used, which is the value used for internal emitters in all ICRP and NCRP publications. More precise methods might indicate a factor more nearly 20 than 10. For recoil nuclei and for fission fragments, Q = 20 is used, as has been the practice in calculating the dose from internal emitters. Finally, a distribution factor N = 5 is used for all high-LET[1] radiations acting on bone (neutrons, alpha particles, recoil nuclei, and fission fragments), as has been customary for internal emitters (ICRP, 1962). For the radionuclides considered in this report, spontaneous fission is of minimal importance, and thus the quality factors used for neutrons and for fission fragments are mentioned only for the sake of completeness.

A special factor (0.01) is used for alpha particles and fission fragments irradiating the wall of the gastrointestinal tract from within the contents of the tract because of the demonstrated inefficiency of alpha particles in causing damage (Sullivan and Thompson, 1957). For recoil nuclei produced in the contents of the tract, the factor is taken as zero because of their shorter range while for neutrons the factor is taken as unity because of their much greater penetrating ability. Actually, perhaps the factor of 0.01 should be regarded as a special instance of the source being at some distance from the target, but with the distance rather poorly specified. It is thus not due to the quality of the radiation but only to its range.

D4 INHALATION MODEL

The inhalation model is that defined by an ICRP Task Group, but with some changes in the parameters to reflect newer data (Morrow, 1966; Lindenbaum et al., 1972). The schematic diagram shown in Fig. VI D-1 (Morrow, 1975a) provides an indication of the fractions of inhaled activity deposited in the nasopharyngeal (NP) region, in the tracheobronchial (TB), region and in the pulmonary (P) region as functions of the activity median aerodynamic diameter (AMAD) or mass median aerodynamic diameter (MMAD).

The specific retention statements that follow this general discussion will usually contain some information on what the clearance class would be for certain chemical compounds.[2]

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[1]Linear energy transfer (LET) is a measure of the rate of energy loss along the track of an ionizing particle, expressed in units of energy per unit track length (e.g., keV per micron).

[2]In many cases this choice has been based on a tabulation by Dr. Paul Morrow (Morrow et al., 1975b), which replaces Table 3 of his paper "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract" (Morrow, 1966), and his advice is hereby acknowledged.

TABLE VI D-1 LUNG CLEARANCE CLASSES FOR THE RADIONUCLIDES INCLUDED IN THE DOSIMETRY MODEL

Group[a]	Radionuclides in Group	Expected Chemical Species Released from Containment	Assigned Lung-Clearance Class[b]
Halogens	I-131, I-132, I-133, I-134, I-135	I_2, CH_3I, iodides, iodates	D
Alkali metals	Rb-86, Cs-134, Cs-136, Cs-137	Oxides, hydroxides	D
Tellurium, antimony	Te-127, Te-127m, Te-129, Te-129m, Te-131m, Te-132, Sb-127, Sb-129	Oxides	W
Alkaline earths	Sr-89, Sr-90, Sr-91, Ba-140	Oxides	D
"Transition" group	Ru-103, Ru-105, Ru-106, Rh-105	Oxides, elemental	Y
	Co-58, Co-60	Oxides, hydroxides	Y
	Mo-99	Molybdates (possibly oxides)	Y
	Tc-99m	Oxide, pertechnetate	D
"Lanthanide" group	Y-90, Y-91, La-140	Oxides	W
	Zr-95, Zr-97, Nb-95	Oxides	Y
	Ce-141, Ce-143, Ce-144, Pr-143, Nd-147	Oxides	Y
	Np-239, Pu-238, Pu-239, Pu-240, Pu-241, Am-241, Cm-242, Cm-244	Oxides	Y

(a) The names "transition" and "lanthanide" are in quotation marks because they are not employed precisely, merely as convenient labels

(b) The letters D, W, and Y represent respiratory clearance half-times on the order of days, weeks, and years, respectively

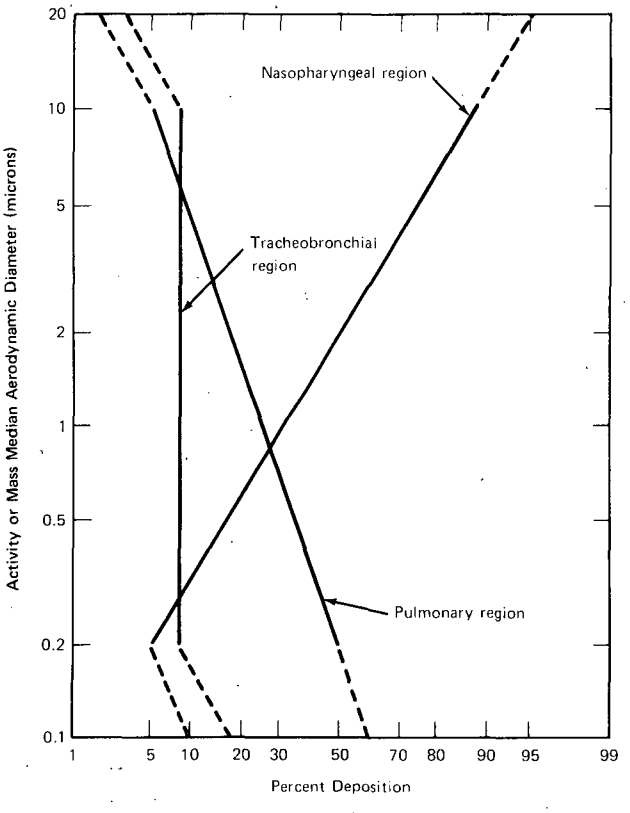
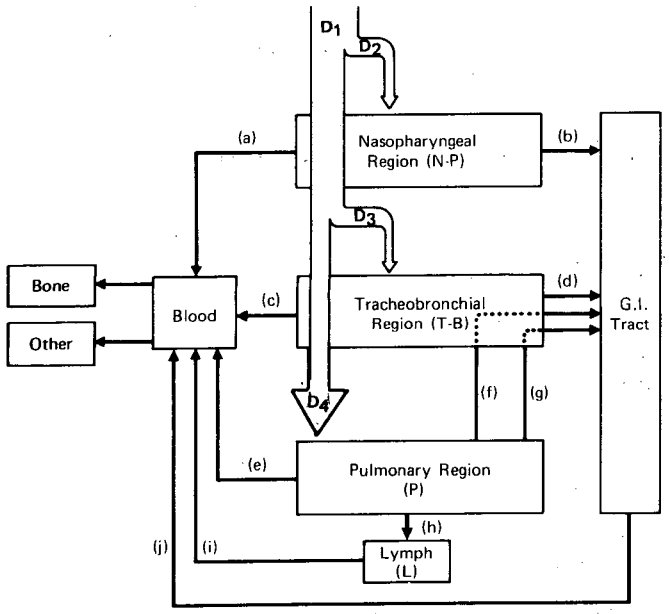


FIGURE VI D-1 Deposition model. The radioactive or mass fraction of an aerosol that is deposited in the nasopharyngeal, tracheobronchial, and pulmonary regions is given in relation to the activity of mass median aerodynamic diameter (AMAD or MMAD) of the aerosol distribution. The model is intended for use with aerosol distributions that have an AMAD or MMAD between 0.2 and 10 microns with geometric standard devisions 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.

Figure VI D-2 is a schematic portrayal of all dust deposition sites and clearance processes. In this figure, D_1 through D_4 are the amounts or concentrations of dust in various respiratory volumes or areas: D_1 is the total dust inhaled (global air concentration), D_2 is the amount of dust deposited in the nasopharyngeal region, D_3 is the dust deposited in the tracheobronchial zone, and D_4 is the pulmonary dust deposition. Ordinarily, D_2, D_3, and D_4 are expressed as percentages of D_1 and are determinable from the deposition model. In addition to the major respiratory regions, three other closely allied compartments are listed: the gastrointestinal tract, systemic blood, and pulmonary lymph,

The letters "a" through "j" indicate the various absorption and translocation processes associated with the clearance of various compartments (Morrow, 1966):

Pathway	Description
(a)	Rapid uptake of material deposited in the nasal-pharynx directly into the systemic blood
(b)	Rapid clearance of all dusts from the nasal-pharynx by ciliary-mucus transport
(c)	Rapid absorption of dust deposited in the tracheobronchial compartment into the systemic circulation
(d)	Analogous to (b) and represents the rapid ciliary clearance of the tracheobronchial region; the dust cleared by (d) goes quantitatively to the gastrointestinal tract
(e)	Direct translocation of dust from the pulmonary region to the blood
(f)	Relatively rapid clearance phase of the pulmonary region which presumably depends on recruitable macrophages and this in turn is coupled to the ciliary-mucus transport process; therefore, the dust cleared by (f) goes to the gastrointestinal tract via the tracheobronchial tree
(g)	Second pulmonary clearance process that is typically much slower than (f) but still depends on endocytosis and ciliary-mucus transport; the cleared dust goes via the tracheobronchial region to the gastrointestinal tract (the important distinction is that the clearance is apparently rate-limited in the pulmonary region by the nature of the deposited dust per se)
(h)	Process describing the slow removal of dust from the pulmonary compartment via the lymphatic system; this process can be regarded as qualitatively similar to (g) with the exception that lymph transport replaces the ciliary-mucus transport
(i)	Secondary pathway in which dust cleared by the lymphatic system (h) is introduced into the systemic blood; this pathway obviously depends on the ability of the cleared material to penetrate the lymph tissue, especially the lymph nodes (this implies partial or complete dissolution of the dust particles, but the turnover of lymphocytes may contribute)



Region	Pathway	Compound class (D)	Compound class (W)	Compound class (Y)
N-P	(a)	0.01 d/0.5 [a]	0.01 d/0.1	0.01 d/0.01
	(b)	0.01 d/0.5	0.4 d/0.9	0.4 d/0.99
T-B	(c)	0.01 d/0.95	0.01 d/0.5	0.01 d/0.01
	(d)	0.2 d/0.05	0.2 d/0.5	0.2 d/0.99
P	(e)	0.5 d/0.8	50 d/0.15	500 d/0.05
	(f)	—	1 d/0.4	1 d/0.4
	(g)	—	50 d/0.4	500 d/0.4
	(h)	0.5 d/0.2	50 d/0.05	500 d/0.15
L	(i)	0.5 d/1.0	50 d/1.0	1000 d/0.9

(a) First value is the biological half-life and second value is the regional fraction.

FIGURE VI D-2 Retention model. See text for description of pathways (a) through (j).

In Table VI D-2 are presented the conversion factors used to calculate the dose from each curie of inhaled radioactive material.

D5 INGESTION MODEL

The dosimetric model for the gastrointestinal tract is essentially that due to Eve (1966) so far as the subdivisions of the tract and the transit times through the sections are concerned. Four subdivisions of the tract are defined: the stomach (S), the small intestine (SI), the upper large intestine (ULI), and the lower large intestine (LLI), and the estimates of dose are considered to be averaged over these sections. Table VI D-3 provides data on the average mass of food in each of these sections and the average length of time it remains there. This information is quoted from Eve (1966) or from ICRP Publication 23 (1975).

The time that food remains in the various portions of the tract can be extremely variable both for different foods as well as for the same individual at different times (Hayes et al., 1963). There is also evidence that the emptying of the stomach and of other sections of the gastrointestinal tract is exponential in character (Nold et al., 1960). This model is adopted here since it allows for some activity to reach other sections and organs of the body before a fixed time period has elapsed which is in general accord with experience for animals and man.

Dose is computed by the methods described by Snyder et al. (1974), and only a very brief summary is given here. In computing the dose from photons, the specific absorbed fractions presented by Snyder et al. are used. For electrons, however, only a "surface" dose is computed. The overconservatism of this dose has been noted (Unnikrishnan et al., 1973), but it is adopted here because biological data on the distribution of the sensitive cells are lacking, as are data on penetration of the mucus blanket by the radionuclide. The ineffectiveness of alpha particles in reaching the sensitive cells has been clearly demonstrated (Sullivan and Thompson, 1957), and this is the origin of the factor of 0.01, referred to in section D3, that is applied for all alpha emission for irradiation of the gastrointestinal tract.

Direct ingestion would be a less important avenue than inhalation. The only radionuclides for which doses based on direct ingestion are calculated are the following: strontium-89, strontium-90, iodine-131, iodine-133, cesium-134, cesium-136, and cesium-137. These are considered as long-term low-level dietary constituents. The f_1 values[1] used are 0.95 for cesium and iodine, and 0.2 for strontium following Marshall (ICRP Publication 20, 1973). It is assumed that after an accident exposure by ingestion would be avoided.

Part of the inhaled material, however, would be translocated by bodily processes to the gastrointestinal tract. The model for the gastrointestinal tract must therefore include all the nuclides considered in the inhalation model.

D6 VARIATION OF DOSE WITH AGE

D6.1 GEOMETRICAL CONSIDERATIONS

There is evidence that for infants and children the dose per disintegration from photon emitters is considerably higher for some configurations of source-organs and target organs than is the case in the adult. Likewise, doses per disintegration due to beta particles, electrons, and alpha particles are inversely proportional to organ mass and thus show considerable variation with age. For example, Fig. VI D-3 shows the specific absorbed fraction Φ[2] (lungs ← lungs) as a function of age for photons of various energies. The dose per photon emitted in the lungs is directly proportional to Φ (lungs ← lungs), which increases by approximately an order of magnitude as age decreases. For energies of 100 keV or more, only a single curve is shown because the data are relatively independent of energy; however, for photon energies below 100 keV the curves seem to be

[1] The f_1 value is the fraction of a quantity ingested that is absorbed from the gastrointestinal tract to the blood.

[2] Absorbed fraction = fraction of the energy emitted from the source organ that is absorbed in the target organ.

TABLE VI D-2 DOSE-CONVERSION FACTORS FOR INHALED RADIONUCLIDES (rem/Ci INHALED)
BODY ORGAN: TOTAL MARROW

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	4.9E+02	1.1E+03	3.0E+03	3.1E+03	3.1E+03	3.1E+03
CO-60	5.5E+02	1.2E+03	2.8E+03	2.1E+04	5.6E+04	5.8E+04	5.8E+04
KR-85	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
KR-88	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-89	5.2E+02	1.7E+03	5.0E+03	1.3E+04	1.3E+04	1.3E+04	1.3E+04
SR-90	3.6E+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	1.8E+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	1.8E+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
NP-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	5.8E+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 VI D-2 (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-58	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	3.9E+06	3.9E+06	3.9E+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	1.9E+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 VI D-2 (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	8.0E+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
RB-86	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	4.0E+04	4.0E+04	4.0E+04
Y-91	2.0E+04	4.6E+04	4.9E+04	5.0E+04
ZR-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
NB-95	3.2E+03	6.9E+03	7.0E+03	7.1E+03
MO-99	1.2E+04	2.0E+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	7.8E+02	7.8E+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	1.5E+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
SB-127	1.3E+04	2.6E+04	2.6E+04	2.6E+04
SB-129	7.9E+02	8.1E+02	8.1E+02	8.1E+02
I-131	2.6E+02	3.3E+02	3.6E+02	3.6E+02
I-132	6.0E+01	6.0E+01	6.0E+01	6.0E+01
I-133	3.0E+02	3.3E+02	3.3E+02	3.3E+02
I-134	2.0E+01	2.0E+01	2.0E+01	2.0E+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	1.0E+04	1.8E+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
NP-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	4.9E+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 VI D-2 (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	4.8E+04	5.0E+04	5.0E+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	1.9E-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-89	7.2E+02	3.0E+03	1.8E+04	3.0E+04	3.0E+04	3.0E+04	3.0E+04
SR-90	5.3E+02	3.8E+03	2.1E+04	2.6E+05	1.6E+06	2.4E+06	2.8E+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	1.9E+04	1.9E+04	1.9E+04	1.9E+04
ZR-95	1.2E+02	2.8E+02	7.9E+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
NB-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	1.8E+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	6.0E+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	2.0E+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	1.9E+04	1.9E+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	1.6E+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	1.8E+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 VI D-2 (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
RB-86	5.9E+02	1.7E+03	4.8E+03	6.1E+03
SR-89	2.0E+02	5.0E+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	1.8E+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
NB-95	8.1E+01	2.1E+02	6.4E+02	9.5E+02
MO-99	9.4E+01	1.4E+02	1.5E+02	1.5E+02
TC-99M	4.6E+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	1.0E+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	1.0E+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	1.8E+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	5.0E+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	1.0E+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	6.0E+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	2.9E-01	7.9E-01	9.9E-01
ND-147	1.2E+01	2.8E+01	6.0E+01	6.8E+01
NP-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

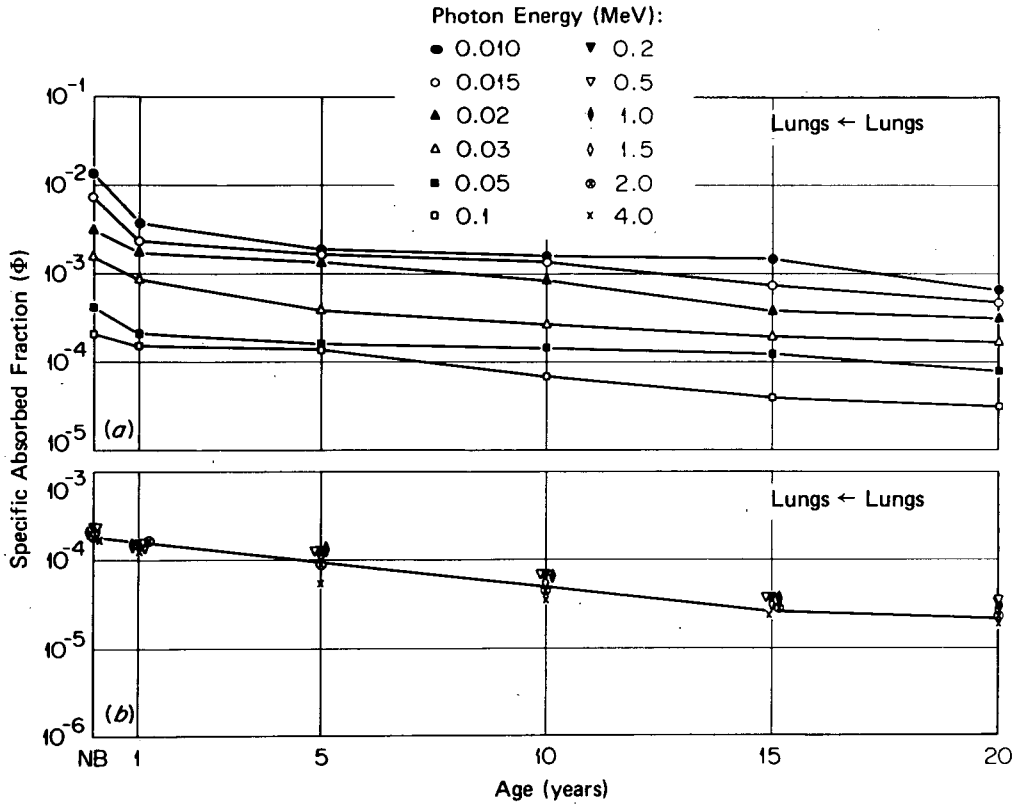


FIGURE VI D-3 Specific absorbed fraction (lungs←lungs) as a function of age and photon energy.

TABLE VI D- 3 GASTROINTESTINAL TRACT MODEL OF REFERENCE MAN

Portion of Gastrointestinal Tract	Mass of Wall (g)	Mass of Contents (g)	Average Time Food Remains (days)
Stomach	150	250	1/24
Small intestine	640	400	4/24
Upper large intestine	210	220	13/24
Lower large intestine	160	135	24/24

distinct. A similar result is shown in Fig. VI D-4, in which Φ (liver ← lungs) is plotted, except that now at the lowest photon energies the increase is by about two orders of magnitude. These results were obtained by Monte Carlo type calculations using phantoms of appropriate sizes. These results are typical of the many such calculations that have been reported (Snyder and Cook, 1971; Hilyer et al., 1972, 1973).

Generally, the energy carried by electrons and alpha particles is considered to be completely absorbed in the organ where the particle is emitted. In such cases the dose per particle is inversely proportional to the mass of the organ, which varies with age. Table VI D-4 gives the ratios of doses per particle for individuals aged 0 (newborn), 1, 5, 10, and 15 years as well as the dose per particle to the adult. This ratio is simply the inverse ratio of the organ masses, that is, organ mass of adult/organ mass of child. As age decreases, the ratio tends to rise, and the total increase is again by an order of magnitude (Snyder, 1974).

D6.2 METABOLIC CONSIDERATIONS

Generally, the child's intake of water, air, or food is smaller than that of the adult, and likewise the child's higher rate of metabolism often leads to a more rapid elimination of activity from his body than would be typical for adults. One cannot maintain the position that the geometric and metabolic factors cancel each other in any precise way, but generally available data indicate that the dose received by exposure to a radionuclide is not drastically different in the young and in the adult. However, there are some exceptions, especially those noted in discussing the model used for the gastrointestinal tract.

As an illustration of the general rules, data on naturally occurring radium-226 in the bones of various age groups are not widely different, although some difference by a factor of 2 may be present (Muth et al., 1960; Rajewsky et al., 1965). Likewise, if one consults the issues of Radiation Health Data over the past 10 years or more, one sees only a modest difference in the levels of strontium in children's bones and in adult bones. Similarly, stable strontium would be expected to be near equilibrium in the adult. This is not to say that no difference exists, but rather that the difference would not be expected to be greater than twofold or threefold in most cases. For example, Table VI D-5 lists typical total-body and thyroid weights, surface areas of the total body, and typical breathing rates as a function of age. The primary data are all from the Report of the Task Group on Reference Man (ICRP, 1975), except for the breathing rate at age 5 years, which is obtained by interpolation on weight and on surface area. Since these give rather different results, the average of the two values is used. The last lines show the ratios of the breathing rate to the mass of the total body or to the mass of the thyroid. Other things being equal, one would expect the inhaled activity of a radionuclide to be approximately proportional to the breathing rate, and thus the dose rate should be approximately proportional to the ratio of breathing rate to body or organ mass. It is seen that this ratio does not vary greatly for individuals of different ages, although factors of 2 to 3 do occur. The mass of the skeleton is approximately 10% of the total body mass for various ages, and thus the ratio of the breathing rate to the total-body mass would indicate approximately the variation of dose per microcurie inhaled for bone-seeking radionuclides for the indicated ages, assuming no great difference in metabolism.

Actually other factors will be present and have some effect. For example, differences in metabolic activity may and do exist and cause differences in dose. However, these generally favor the younger individuals. Lloyd et al. (1968) and Zundel et al. (1969) have shown that the retention half-time of cesium in the body varies with age. These authors estimate that the retention half-time in the body of adults, pregnant women, and normal infants is 105 ± 25, 49 ± 16, and 19 ± 8 days, respectively. Similarly, iodine has a shorter retention half-time in the thyroid of the younger individuals than for adults (Cook and Snyder, 1965; Cuddihy, 1966; Karhausen et al., 1973; Kereiakes et al., 1966). The estimates range from approximately 140 days for the retention half-time for the adult (ICRP, 1959) to approximately 8 days for the infant. For iodine-131 these differences affect the dose by a factor of 2 or less because of its short radioactive half-life, but for other isotopes of iodine of longer radioactive half-life they may produce more substantial changes in the dose.

It is possible to define and calculate dose correction ratios for various ages. These are simply the ratios D_{ch}/D_{ad} by which the dose to the adult per unit intake must be

TABLE VI D-4 MASSES OF ADULT ORGANS AND RATIOS OF ORGAN MASSES TO TOTAL BODY MASSES OF REFERENCE MAN BY AGE

Organ	Organ Mass (g) for Adult	Ratio				
		15 years	10 years	5 years	1 year	Newborn
Kidneys	310	1.2	1.7	2.8	4.2	13
Liver	1,800	1.5	2.1	3.1	5.6	14
Lungs	950	1.4	2.2	2.7	6.1	18
Ovaries	10.7	2.3	4.1	7.1	15.0	32
Skeleton	10^4	1.3	1.7	2.2	10.0	30
Testes	35	2.2	18.0	22.0	29.0	41
Thyroid	16	1.4	2.2	4.5	9.1	16
Total body	7×10^4	1.2	2.1	3.7	7.0	20

TABLE VI D-5 ORGAN MASSES, SURFACE AREAS, AND BREATHING RATES FOR VARIOUS AGES

Parameter	Age (years)				
	0 (Newborn)	1	5	10	20 (Adult)
Total body mass (kg)	3.5	10	19	33	70
Thyroid mass (g)	1	1.8	3.6	7.4	16
Surface area of total body (m^4)	0.21	0.39	0.75	0.96	1.7
Breathing rate (10^4 ℓ/day)	0.08	0.38	0.96	1.5	2.3
Ratio of breathing rate to total body mass	0.023	0.038	0.051	0.045	0.033
Ratio of breathing rate to thyroid mass	0.08	0.21	0.27	0.20	0.14

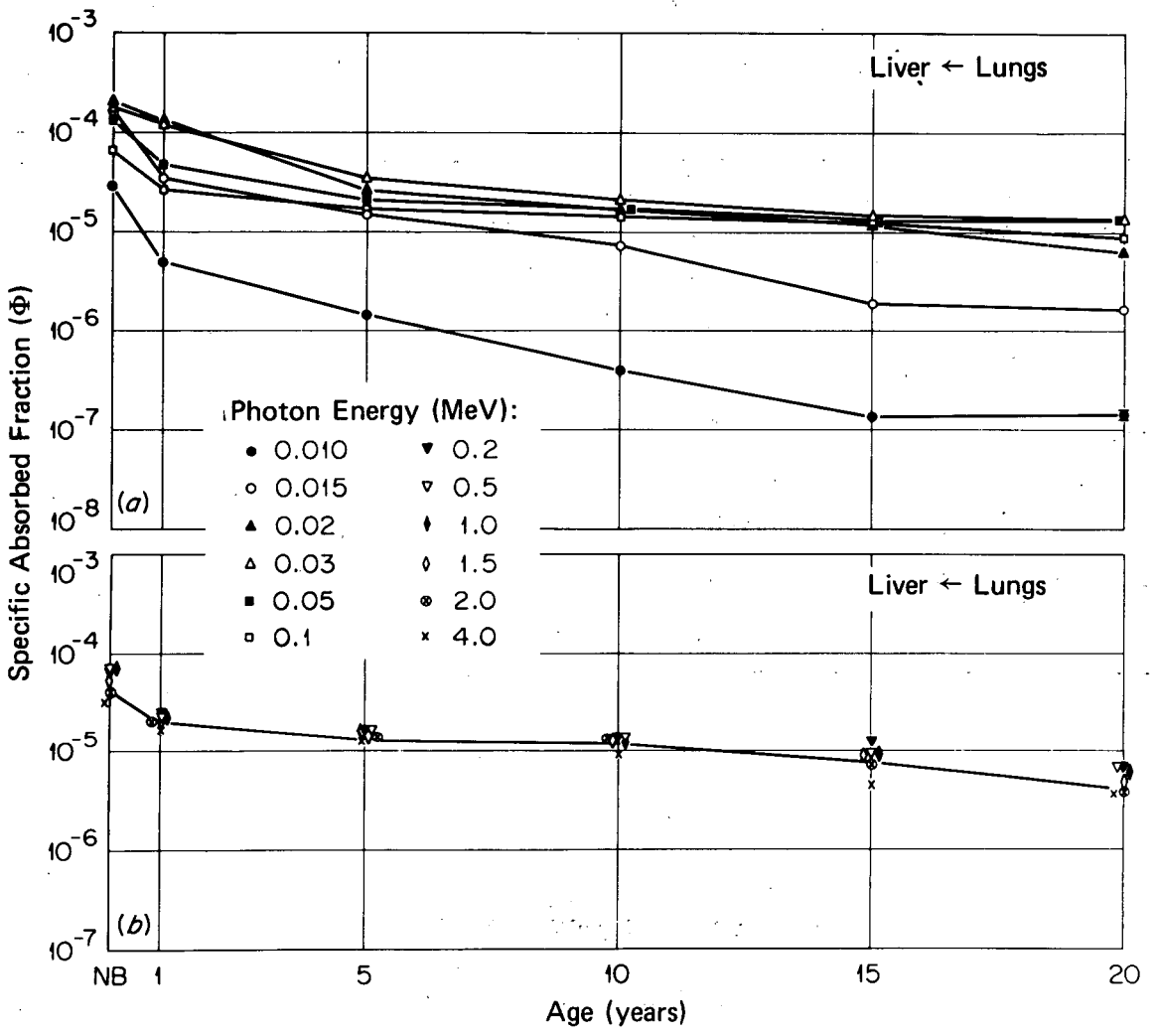


FIGURE VI D-4 Specific absorbed fraction (liver←lungs) as a function of age and photon energy.

ultiplied to obtain the dose to the child per unit intake. Substituting the expressions for the doses and simplifying, one obtains

$$\frac{D_{ch}}{D_{ad}} = \frac{U_{ch}\Sigma\bar{E}_i\Phi_{ch,i}}{U_{ad}\Sigma\bar{E}_i\Phi_{ad,i}} \qquad \text{(VI D-1)}$$

where U represents the microcurie-days accumulated in the organ of the body being considered, $\bar{E}_i$ is the mean energy of the radiation emitted, and Φ_i is the specific absorbed fraction (energy absorbed per gram). Since, for most organs, electron energies are considered as completely absorbed, one can separate the emissions into electrons and alpha particles, which are totally absorbed, and gamma rays, which are only fractionally absorbed.

In considering age effects, only four radionuclides are included: iodine-131, cesium-137, strontium-89, and strontium-90. In the case of iodine-131, the retention can be considered to be a single-exponential term, with the retention half-time in the thyroid varying from approximately 8 days for the infant (Cook and Snyder, 1965; Kereiakes et al., 1966; Karhausen et al., 1973) to approximately 140 days for the adult (ICRP, 1959). The uptake by the thyroid may also vary with age, particularly for infants up to about 1 year of age (Kearns et al., 1962; Morrison et al., 1963). Beyond about 1 year of age, however, the uptake remains practically constant for all age groups (Oliner et al., 1957; Fisher et al., 1962; Cuddihy, 1966).

To the degree of approximation attempted here, the microcurie-days in the thyroid are given by

$$A_{ch}\int_0^{\infty}\exp\left[-(\lambda_b + \lambda_r)\right]dt = A_{ch}/(\lambda_b + \lambda_r), \qquad \text{(VI D-2)}$$

where A_{ch} is the uptake to the child's thyroid per microcurie inhaled or ingested and λ_b and λ_r are constants for biological elimination and radioactive decay respectively. Thus, $\lambda_b = (0.693/T_b)$ and $\lambda_r = (0.693/T_r)$, where T_b and T_r are the retention half-time and radioactive half-life, respectively. A similar expression is valid for the adult. Thus,

$$A_{ch} = \left(A_{ch}^{bl}\right) \times \left(A_{ch}^{th}\right),$$

where A_{ch}^{th} is the activity in the child's thyroid at the peak of the retention curve per unit intake to blood and A_{ch}^{bl} is the activity going to blood per microcurie of iodine inhaled. Thus the microcurie-days accumulated by the child are given by

$$U_{ch} = \frac{\left(A_{ch}^{bl}\right)\left(A_{ch}^{th}\right)}{\lambda_b + \lambda_r}. \qquad \text{(VI D-3)}$$

A similar expression holds for the adult.

The energy term can be written as

$$\Sigma E_\gamma \Phi_\gamma + (\Sigma \bar{E}_e/M) \qquad \text{(VI D-4)}$$

since electron energy is considered as completely absorbed. Data on Φ_γ in the thyroid as a function of age have been published by Ford et al. (1975). Thus

$$\frac{D_{ch}}{D_{ad}} = \left(\frac{A_{ch}^{bl}}{A_{ad}^{th}}\right)\left(\frac{A_{ch}^{th}}{A_{ad}^{th}}\right)\left(\frac{\lambda_b^{ad} + \lambda_r}{\lambda_b^{ch} + \lambda_r}\right)\frac{\left[\Sigma E_\gamma \Phi_\gamma + \Sigma(\bar{E}_e/M)\right]_{ch}}{\left[\Sigma E_\gamma \Phi_\gamma + \Sigma(\bar{E}_e/M)\right]_{ad}} \qquad \text{(VI D-5)}$$

where the first two fractions are taken as being substantially equal to unity.

For cesium-137, the equations are simpler because the radioactive half-life is long and thus uptake to blood can be assumed to be unity. Thus the microcurie-days accumulated by the child per microcurie are given by

$$U_{ch} = (\lambda_b + \lambda_r)^{-1} \qquad \text{(VI D-6)}$$

as before with a similar relation for the adult. The data on λ_b are taken from Lloyd et al. (1968). The energy dependence is again that given by Equation (VI D-4), and data on Φ_γ are obtained from Fisher and Snyder (1966).

Strontium poses the most difficult problem because of its imperfectly known metabolism and its long radioactive half-life. This study follows Papworth and Vennart (1973), who allow for an increase by a factor of 5 during the first year of life. Thus the increase reflects an enhanced uptake to blood and to bone during the first year of life, but the indications are that after the first year the child's metabolism is essentially similar to that of the adult. Actually, there are indications that the uptake to blood and to bone declines during the first year, but it is taken as constant here. Correction ratios D_{ch}/D_{ad} computed in this way are listed in Table VI D-6 for iodine-131, cesium-137, strontium-89, and strontium-90 for ages 1, 5, and 10 years. These dose correction factors can be multiplied by the ratio of the breathing rates given in Table VI D-5 to represent substantially the ratio of doses to the critical organ of a child and an adult exposed to the same radioactive cloud. If they are used to calculate doses from exposure via ingestion, they need to be multiplied by the ratio of activities of these radionuclides in the total diet of the child and the adult.

However, there are many radionuclides for which there is little documentation of the effect of body size on the dose received by a given organ from a given quantity of radionuclide inhaled or ingested. Of course, it is necessary to recognize situations in which the infant's or child's daily intake of milk or of some other food is about the same as or greater than that of an adult, and a table of typical intakes should be used along with doses computed here, which are all based on a rad per microcurie, or rem per microcurie inhaled or ingested. Though there are differences in inhalation parameters that vary with age, the overall effect seems to be that the amount of air inhaled is proportional to body mass or perhaps to surface area and thus generally to the masses of the various organs. It is evident that the constancy of dose per unit intake with age is not exact, but no large variations are expected. It is difficult to estimate the uncertainty without being very precise concerning the individual and the conditions of exposure. Analyses such as that offered above suggest that for inhalation it might be by a factor of 2 or 3.

D6.3 INGESTION

The discussion of variation of inhalation dose with age requires some amendment for ingestion. There is much greater variation in intake of certain foodstuffs with age, sex, and other factors than in the amount of air breathed per day. Individuals of a given size normally inhale comparable quantities of air, and there is some correlation of dose and intake by inhalation. However, in the case of many foods this correlation is largely missing because one may substitute another food or source of supply that is uncontaminated, and this needs to be taken into account. For example, an infant may consume cow's milk, uncontaminated condensed milk, or milk from the mother's breast. Its milk consumption, on the average, will be nearly equal to that of an adult, but the infant's consumption of meat or leafy vegetables will be virtually zero at certain ages. Since practices may vary widely with circumstances, one must be careful to adjust the dose using representative intakes that reflect the intakes of the population under consideration. This can be done, but must take account of the specific pathways and sources of food involved as well as the characteristics of the population. These situations are discussed in more detail in Appendix E.

TABLE VI D-6 DOSE CORRECTION RATIOS D_{ch}/D_{ad} FOR VARIOUS AGES AND RADIONUCLIDES[a]

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.

D7 RADIOACTIVE DAUGHTERS

The dose evaluation takes account of all daughters produced in the body when a parent nuclide is inhaled. There is generally little information available concerning the retention of these daughter radionuclides in the organs where they are produced or on their transfer to other organs. Generally, it is assumed that the daughter will be retained in the organ in which it is produced and that the retention function will be the same as for a directly deposited daughter radionuclide. Exceptions to this rule will be noted in the detailed discussion of the metabolic models given below.

D8 CALCULATION OF RETENTION

A retention model is specified below for each element considered. The time integrals of activity (microcurie-days) are calculated by use of a computer program described by Snyder et al. (1975). The lung and gastrointestinal tract are standard parts of this program and represent the ICRP models for retention in these organs rather closely. The lung model is specified by the pathways and parameters in Fig. VI D-2, and the corresponding model for the gastrointestinal tract is specified in Table VI D-3. The retention models used for systemic organs are discussed below; however, they are always given for the stable element and thus need to be corrected for radioactive decay.

D8.1 ACTINIDES

Plutonium and the other actinide elements neptunium, americium, and curium have been the subjects of several fairly recent reviews (Durbin, 1971; ICRP Publication 19, 1972; Hodge et al., 1973). The metabolic model for these actinides used here is essentially that developed in ICRP Publication 19 (ICRP, 1972) with a few minor changes that reflect more recent experience and review.

The lung model is the usual one adopted by ICRP, with some changes in parameters, as discussed in section D4. The chemical forms that these elements would assume cannot be specified without very precise information about the type of accident involved. However, it seems likely that dose estimates based on the dioxides and the nitrates would be typical of what one might expect. Lung-clearance class Y is used for the dioxide, and class W is used for the nitrate; no reasonably expected compound of clearance class D is known.

The value of f_1 is always small, so that the actual value has little effect on the estimation of inhalation dose. The value of $f_1 = 3 \times 10^{-5}$ is used for plutonium of either form, and, because of the comparative paucity of experimental data, the value of f_1 for the other actinides is conservatively set at $f_1 = 10^{-3}$.

Once the material has entered the bloodstream, it is partitioned between the liver and bone, with smaller amounts deposited in other soft tissues or excreted. The amount going to bone may vary widely depending on the polymeric form and the complexing that has taken place. ICRP Publication 19 (ICRP, 1972) recommends that 10% of the activity entering the bloodstream be allowed for deposition in other soft tissues or for excretion and that the other 90% be equally divided between bone and liver. The report also recommends that the retention half-times in the liver and in bone be taken as 40 and 100 years, respectively. Thus, of the activity entering the bloodstream, the retention at t days after the intake is as follows:

Tissue	Retention R(t)
Bone	0.45 exp(-0.693t/36,500)
Liver	0.45 exp(-0.693t/14,600)
Kidneys	0.01 exp(-0.693t/14,600)
Testes	0.0001 exp(-0.693t/36,500)
Ovaries	0.00001 exp(-0.693t/36,500)
Other tissues	0.05 exp(-0.693t/36,500)

The kidneys, ovaries, and testes are assigned somewhat more activity (per unit mass) than the "other tissues." This represents a considered judgment based on experimental work with animals and also on autopsy data on humans with plutonium in their bodies (Richmond and Thomas, 1975). These metabolic models, with the values of f_1 noted above are used for all the actinides, even when they occur as daughter elements produced in the body.

Although there are known differences in the metabolism of the actinides, the experimental work has not been so exhaustive that one can predict human exposure with confidence. Thus the adoption of the plutonium model for the other actinides represents a further degree of conservatism since most experimental work indicates more rapid elimination for other actinides than is the case for plutonium. In assessing dose to bone, the ICRP formula is used for all the actinides. The dose in rads is averaged over the bone mass, 5000 grams, and a quality factor of Q = 10 and a distribution factor of N = 5 are then applied to obtain the dose in rem. The N factor is used

for only alpha and recoil energy or for energy from spontaneous fission. (Studies are now underway to estimate the dose to endosteal cells more precisely, taking account of the distribution of plutonium and other actinides in bone. The ICRP has designated these cells as the tissue at risk for exposure of bone. However, these studies have not yet reached a point at which extrapolation to man is possible.) The method of estimating the dose equivalent in rem that is used in this report rests largely on the demonstrated difference in carcinogenic effect in bone between plutonium and radium-226 (ICRP Publication 2, 1959; Mays and Dougherty, 1972).

The assessment of dose or dose equivalent to the various organs is then straightforward except for the contribution from spontaneous fission. This distribution is assessed using the methods developed by Dillman and Jones (1975) and by Ford et al., (1975). The quality and modifying factors used have been mentioned before.

There is very little information available on differences in metabolism that might be attributed to age. Thus the same metabolic model and calculation are used for the adult and the child. This is known to result in a considerable overestimate of intake, but it also involves the differences in organ mass which are of the same order of magnitude and tend to be compensatory.

D8.2 ANTIMONY

The total-body burden of antimony in the "reference man" (ICRP, 1975) is about 8 mg, corresponding to an average concentration of about 0.1 microcurie per gram of tissue. Of the eight specimen types for which there are data, hair shows a concentration that is 65 times the average and blood shows a concentration one-twentieth of the average. The other organs have about the average concentration.

For the reference man, excretion of antimony in urine is about 40 micrograms per day. Dietary intake is placed at 50 micrograms per day, but this is estimated from excretion. It implies an f_1 value of about 0.8. In its Publication 2, the ICRP (1959) gave an f_1 value of 0.03, determined by Hamilton et al. (1948) on an unspecified form of antimony fed to rats. From studies on pigs and dogs fed a gelatin capsule containing fallout debris from a nuclear explosion (Chertok and Lake, 1971), f_1 is indicated to be greater than 0.05 because urinary excretion is greater than 5%, and, as will be discussed, urinary excretion is the predominant pathway for excretion after injection into blood. Oral doses of antimony tartrate given to mice also gave results that suggest 5% uptake to blood (Waitz et al., 1965).

Some studies indicate a lower uptake to blood from the gut. Westrick (1953) fed rats antimony trioxide in a synthetic casein diet and, assuming a total-body retention half-time of about 40 days, estimated f_1 to be about 0.005. Rose and Jacobs (1969) studied human accidental exposure and found that in 10 days about 1% of the amount excreted in feces (~24 nanocuries) was excreted in urine.

There are no adequate human data for estimating retention for unit intake to blood, but a provisional retention function can be constructed from studies on rats and the reference man data. In rats Durbin (1960) found that about 95% of an injected dose was excreted in urine within 4 days. Sollman (1957, p. 1227) states that in man antimony compounds are rapidly excreted into urine, and of 1.7 grams of antimony tartrate injected during 6 weeks, about 50% was excreted in urine. Studies of organ and total-body retention in rats after the inhalation of antimony trichloride (Djuric et al., 1962) suggest that the long half-time for retention is about 140 days, with 10 to 20% of the initial body burden excreted with this long half-time. Djuric et al. found that the blood contained approximately 50% of the total-body retention, the rest being approximately uniformly distributed. In a followup study of lung retention in a human subject, Palmer (1962) found a retention half-time in lung of about 100 days.

From the above data, f_1 is taken to be 0.05, except for antimony trioxide, for which the value of 0.01 is used.

The sulfides, oxides, and halides of antimony are assumed to be in clearance class W. Studies of Felicetti et al. (1974) on dogs that had inhaled heated antimony tartrate indicated the possibility that a class Y compound also exists.

The total body retention is taken as

$$R(t) = 0.85 \exp(-0.693t) + 0.15 \exp(-0.693t/100),$$

and the distribution is assumed to be uniform.

Antimony-127 and antimony-129 both have radioactive half-lives of less than 1 day and decay to form isotopes of tellurium. These in turn decay to isotopes of iodine. For these daughters, the metabolic models for tellurium and iodine will be used.

D8.3 CERIUM, PRASEODYMIUM, NEODYMIUM, AND PROMETHIUM

For cerium, f_1 is taken as 10^{-4} (McClellan et al., 1965; Miller and Byrne, 1970). Although in these studies one compound was cerous chloride ($CeCl_3$) and the other an unspecified cerium oxide, essentially the same value for absorption was obtained in the cow and in the dog. However, Grigoryan (1966) found a value in rats indicated as being less than 10^{-3}. Inhalation studies on beagles have indicated that cerous chloride behaves as a class W aerosol (Boecker and Cuddihy, 1974), and this is true also for ceric oxide (CeO_2) inhaled by hamsters (Hobbs et al., 1974).

Durbin (1960) has discussed the distribution of the rare earths generally, indicating that the liver and bone would be expected to be "major deposition sites." The studies reported by Boecker and Cuddihy (1974) confirm this and give a range for retention in various organs of the beagle. On this basis, retention in the liver, bone, kidney, and other tissues is represented by

$$A \exp(-0.693t/5000),$$

where A has the values 0.5, 0.4, 0.025, and 0.05 for the liver, bone, kidneys, and other tissues, respectively (Boecker and Cuddihy, 1974), with the remainder representing early excretion.

Some isotopes of cerium decay to form radioactive isotopes of praseodymium. Since praseodymium is one of the rare-earth elements, as is cerium, the same metabolic model is used for both (Durbin, 1960). This is appropriate since the radioactive half-lives of these daughters are relatively short. One of these, praseodymium-144, decays to the radioactive neodymium-144, which has a radioactive half-life of 2.4×10^{15} years, and thus the dose contribution of this isotope as a daughter is negligible. However, neodymium-147 does occur as a parent radionuclide. Since the radioactive half-life is only 11 days and there are very scanty data on its metabolic behavior, the same metabolic model is used as for cerium.

The same metabolic model is used also for promethium. The clearance classes are class Y for the oxides, hydroxides, carbides, and fluorides; class W for the carbonates, phosphates, and halides (except for the fluorides); and class D for the sulfides, sulfates, and nitrates. A value of 10^{-4} is used for f_1 in all cases, by analogy with cerium.

D8.4 CESIUM

The metabolism and dosimetry of cesium when taken into the human body have been investigated due to the importance of cesium-137 as a constituent of weapons fallout. Practically all forms of cesium inhaled are absorbed readily into the blood (Stara et al., 1971). Here $f_1 = 0.95$ and clearance class D are assumed.

When absorbed into blood, cesium is widely distributed among the body tissues, muscle being higher in concentration (generally by a factor of about 2). Since muscle is so widely distributed in the body, cesium is generally considered to irradiate the

entire body. A two-exponential formula for retention has been found to represent retention adequately:

$$R(t) = 0.13 \exp(-0.693t/1.4) + 0.87 \exp(-0.693t/135).$$

The coefficients were selected on the basis of controlled studies on adult humans (Richmond et al., 1962). There has been speculation that there should be a component of longer retention half-time, perhaps associated with bone, and several such high values have been reported (Yamagata and Yamagata, 1960; Anderson and Gustafson, 1962; Takizawa and Sugai, 1971). However, Rundo (1964) has followed an individual case for over 1000 days without detecting any significant departure from the single-exponential model after the first few days. He calculates that such a component, if present, would not make a significant contribution to dose.

In individual cases there is considerable variability about the values selected above; for example, the longer elimination half-time ranged from 110 to 142 days for the four subjects studied by Richmond et al., (1962). There is also some build-up of the dose from photons in organs near the center of the trunk, with smaller values of dose for organs near the surface. This variation, however, is only by about a factor of 2 (Fisher and Snyder, 1966).

More importantly, the retention half-time seems to be shorter for children, pregnant women, and individuals suffering from certain diseases (Lloyd et al., 1968; Zundel et al., 1969). This tends to decrease the dose estimated for these individuals and for fetuses. For example, Lloyd et al., (1968) estimate that the retention half-time for cesium in the normal infant is about 19 days. There are also factors that may influence dose in the opposite direction. Generally, for a given concentration of cesium in the body, the dose to the child is somewhat smaller due to the greater escape of photon energies from the smaller body of the child.

Cesium-137 decays to form barium-137m, but the radioactive half-life of this daughter is only 2.5 min. Thus it should be in equilibrium with its parent in most tissues of the body. The model used assigns essentially the same microcurie-days for barium-137m as for cesium-137.

D8.5 COBALT

Data from several balance studies on humans indicate that the absorption of dietary cobalt from the gastrointestinal tract to blood is from 20 to 95% (Harp and Scoular, 1952; Hubbard et al., 1966; Engel et al., 1967; Valberg et al., 1969). The absorption of inorganic compounds, usually cobalt chloride, depends on the quantity administered and may vary from 2% at trace levels to 20% if carrier cobalt is present (Paley and Sussman, 1963; Smith et al., 1972). Studies with animals confirm the generally high absorption of organic complexes (Taylor, 1962), but there seems to be no data available on the absorption of insoluble cobalt compounds. Here f_1 is taken as 0.05, which would be appropriate for trace levels of inorganic, poorly soluble, compounds.

Oxides and hydroxides of cobalt are assumed to be in clearance class Y, and all other compounds are assumed to be in class W (Morrow, 1966).

Table 108 of Gross and Elemental Composition of Reference Man (ICRP Publication 23, 1975) indicates that the cobalt reaching the blood is distributed rather generally throughout the body, except that the concentration in the liver is approximately threefold. The retention half-time for stable cobalt after unit intake to blood is taken from Smith et al., (1972), who studied two human subjects for 1000 days:

$$R(t) = 0.5 \exp(-0.693t/0.5) + 0.3 \exp(-0.693t/6) + 0.1 \exp(-0.693t/60) + 0.1 \exp(-0.693t/800).$$

This retention is quite similar to that found by Letourneau et al., (1972), who studied more subjects but for a shorter time.

It is assumed that the activity reaching the blood is uniformly distributed in all tissues except the liver, in which it is assumed to reach a concentration three times that in the rest of the body. Further, it is assumed that the concentration in

all these tissues follows the same time course. Thus 0.95R(t) is taken to represent the fraction of activity that reaches the blood and is distributed uniformly in the total body, and 0.05R(t) is taken as the additional activity that is concentrated in the liver.

Neither of the isotopes of cobalt considered here decays to form a radioactive daughter.

D8.6 IODINE

Iodine has been used extensively in nuclear medicine and also has been studied when present in the environment from weapons fallout. Thus iodine retention in the human body is rather well documented.

After inhalation, iodine in all forms is virtually completely absorbed into the blood (Cuddihy, 1964; ICRP, 1959; NCRP, 1969). Nevertheless, a rather wide variation of doses per microcurie of intake is possible. The weight of the thyroid varies greatly among individuals and also with age, as shown in Fig. VI D-5. In addition to this rather large variation in thyroid mass, there is some indication that other parameters--uptake by the thyroid and retention half-time--may be affected, at least under chronic conditions of intake (Dolphin, 1971; ICRP Publication 23, 1975, pp. 196-199).

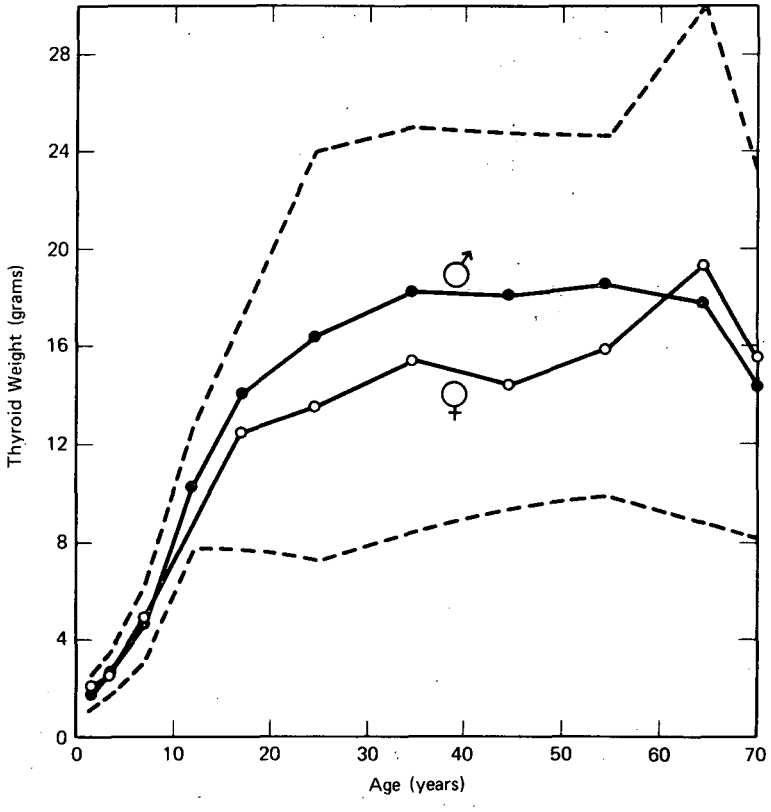


Figure VI D-5 Weight of the thyroid as a function of age and sex. (From Ford and Snyder, 1975).

Metabolic models for iodine retention have been given by many authors (Riggs, 1952; Cuddihy, 1964; Berman, 1968). The model of Riggs, which provides for an inorganic and an organic pool generally distributed in the body as well as for a special component in the thyroid, is generally adequate for the normal adult (Bernard, 1970;

Berman, 1971). Retention at time t days after the absorption in blood of one unit of stable iodine is given by

$$R_{th}(t) = -0.328 \exp(-0.693t/0.243) + 0.016 \exp(-0.693t/11.3) + 0.312 \exp(-0.693t/117)$$

and

$$R_{tb}(t) = 0.997 \exp(-0.693t/0.243) - 0.04873 \exp(-0.693t/11.3) + 0.0514 \exp(-0.693t/117),$$

where R_{th} represents retention in the thyroid and R_{tb}, the total-body retention, includes both the inorganic and organic pools referred to above. The standard ICRP lung model and the gastrointestinal tract model are used for the inhalation of a soluble form of iodine (solubility class D) with an f_1 value of 0.95. Radioactive decay, which is not included in the ICRP lung model, is accounted for by applying a multiplicative factor $\exp(-0.693t/T_r)$, where T_r is the radioactive half-life in days of the particular isotope of iodine.

In some cases an iodine isotope decays to form a daughter xenon that is also radioactive. Since xenon is a noble gas, its elimination would be expected to be rapid Conservatively, this report has used solubility class D, $f_1 = 0.95$ for absorption into blood from the gastrointestinal tract, and a retention half-time of 2 hours for that produced in the thyroid and in the total body. Thus, the retention equations used for xenon produced in the thyroid and in the total body are

$$R_{th}(t) = \exp(-0.693t/0.0833)$$

and

$$R_{tb}(t) = \exp(-0.693t/0.0833),$$

and these must be multiplied by $\exp(-0.693t/T_r)$ for the radioactive decay of the particular isotope of xenon, as before.

There is some evidence that the elimination half-time for iodine in the thyroid of children is considerably shorter than that for adults (Cook and Snyder, 1965; Karhausen, 1973). This somewhat compensates for the smaller mass. However, this reduction of dose is small for the short-lived iodine isotopes, particularly iodine-131, and the child will still be the critical individual both on the basis of dose per unit intake as well as on the basis of radiosensitivity. In the case of intake by inhalation, the child or infant will have a reduced intake of air and hence of the iodine contained in the air, as has been discussed above. Without pretending that there is any exact cancellation in the factors to be applied for child and infant versus the adult, the reduced intake is of the same order of magnitude as the reduction in thyroid mass, and these factors largely offset each other. Of course, this does not affect the question of the radiosensitivity of the child or infant.

For a fetal thyroid the above discussion does not apply since there will not be any reduction in the mother's intake. The fetal thyroid does not become functional until about the third month, and uptake even then is minimal (Chapman et al., 1948; Hodges et al., 1955). Uptake increases thereafter, but its net effect is a concentration that ranges from 2 to 10 times that found in an adult population (Bierwalters et al., 1960). Since dose is roughly proportional to the concentration, this would give an increase in dose per unit intake by the mother of about the same magnitude as that for the infant taking a similar amount of iodine (i.e., merely correcting for the mass of the thyroid). The data of Bierwalters et al. from 25 near-term pregnancies suggest that the mean concentration in the fetal thyroid is somewhat less than five times the adult concentration; the same conclusion can be drawn from the data reported by Chapman et al. and Hodges et al. Thus one can, on the basis of these data, obtain the dose to the fetal thyroid by multiplying the adult dose by a factor of about 5.

D8.7 KRYPTON AND XENON

There have been a number of studies on the retention of krypton-85 and xenon-133 under near-equilibrium conditions (Nussbaum, 1957; Kirk and Morken, 1975; Kirk et al., 1975), but there seems to be little information available on retention and distribution after a single intake of a noble gas with a fairly short radioactive half-life. Although the contribution to dose from the intake of these gases is not expected to be large, in the interest of completeness this exposure route should be considered.

The retention model is essentially that developed by Bernard and Snyder (1975) and the main features of the model will be indicated briefly here. The data on radon exhalation (Harley et al., 1958) after exposure to the gas for a period of more than 8 hours was analyzed and fitted with a formula of five exponential terms by those authors. This formula has been deconvoluted to arrive at retention of a brief intake of radon, with corrections for radioactive decay. The distribution of the gas in the body tissues has been established by a method given by Bernard (1973) and by further analysis in terms of the body organs and tissues (Bernard and Snyder, 1975). The model thus obtained has been adjusted for krypton and for xenon using data on the Ostwald coefficients for these gases (Lawrence et al., 1940). The organs and tissues of the body are grouped into three classes: one with high Ostwald coefficients, one with low Ostwald coefficients, and an intermediate class.

Here the data of Nussbaum on the distribution of radon in rats and his extrapolation to the distribution to be expected in man have been followed (Nussbaum, 1957).

These three groups of organs and tissues classified by Ostwald coefficient, are as follows:

1. High: separable fat (~3.5 kg).
2. Low: bone, heart, muscle, skin, testes and bladder (~40 kg).
3. Intermediate: all other organs and tissues (~17.5 kg).

The masses are assigned on the basis of the values given in the Report of the Task Group on Reference Man (ICRP Publication 23, 1975) and are typical for a 70-kilogram adult.

Retention of stable krypton for the three classes of tissues, for t in days, is given by

$$R_H(t) = 1.5 \times 10^{-3}\, e^{-2.2t} + 2.9 \times 10^{-3}\, e^{-14t} + 3.4 \times 10^{-4}\, e^{-73t} + 0.02e^{-670t} + 0.18e^{-8000t},$$

$$R_L(t) = 1.4 \times 10^{-6}\, e^{-2.2t} + 8.8 \times 10^{-4}\, e^{-14t} + 0.02e^{-73t} + 0.026e^{-670t} + 0.25e^{-8000t},$$

$$R_I(t) = 8 \times 10^{-7}\, e^{-2.2t} + 1.7 \times 10^{-3}\, e^{-14t} + 1.7 \times 10^{-4}\, e^{-73t} + 0.05e^{-670t} + 0.45e^{-8000t}.$$

The values are rounded and are for unit intake inhaled at the breathing rate of reference man, that is, 20 m^3/day. For the radioactive isotopes these equations should be multiplied by $\exp(-\lambda_r t)$, where λ_r is the radioactive decay constant.

For xenon, the corresponding equations for t in days, are the following:

$$R_H(t) = 3 \times 10^{-3}\, e^{-t} - 5 \times 10^{-6}\, e^{-7t} + 0.04\, e^{-36t} + 0.0275e^{-330t} + 0.26e^{-4000t},$$

$$R_L(t) = 3.6 \times 10^{-6}\, e^{-t} + 5.4 \times 10^{-3}\, e^{-7t} + 7.8 \times 10^{-3}\, e^{-36t} + 0.018e^{-330t} + 0.18e^{-4000t},$$

$$R_I(t) = 3.5 \times 10^{-6}\, e^{-t} + 2.3 \times 10^{-5}\, e^{-7t} + 0.02e^{-36t} + 0.046e^{-330t} + 0.44e^{-4000t}.$$

The above equations include the lung and gastrointestinal tract as well as the systemic organs, and thus retention by an inhalation model is already included in these equations. The terms with very short elimination half-times (on the order of 10 seconds or a few minutes) pertain to elimination from lungs and gastrointestinal tract. The lung

model used for particulate material is thus not necessary here and would be inappropriate for noble gases. The 1 microcurie of activity inhaled has been assumed to be retained according to the three retention functions given above, and this activity has been reapportioned to the various organs and tissues for the dose calculation. The model is in reasonable accord with clinical data on so-called normals, who generally show a retention half-time of approximately 30 seconds (Goddard and Ackery, 1975). The retention half-time of the most rapid component is about 15 seconds, and there are some reasons for expecting the clinical experience to indicate longer half-times. Of course, for non-normal cases the retention half-time is generally much longer.

D8.8 MOLYBDENUM AND TECHNETIUM

The only radionuclides of concern here are molybdenum-99 (T_r = 2.79 days) and technetium-99 (T_r = 0.25 day). Absorption from the gastrointestinal tract into blood is nearly complete for molybdenum trioxide, calcium molybdate, and ammonium molybdate (Comar, 1948; Underwood, 1971). A value of f_1 = 0.95 is used for the molybdates. The oxides appear to be in clearance class Y (Cuddihy et al., 1970), whereas the molybdates appear to be in class D (Morrow, 1966).

Retention after unit intake to blood, is given by (Rosoff and Spencer, 1964)

$$R(t) = 0.85 \exp(-0.693t) + 0.15 \exp(-0.693t/50) \qquad (t \leq 10 \text{ days}).$$

It is assumed that the liver, kidney, bone, and the other tissues have concentrations in the ratios of 100:60:10:1. These ratios are concentrations found at 8 days after inhalation in experiments with beagles (Cuddihy et al., 1970). This concentration is approximately realized if R(t) is multiplied by factors A_l = 0.57, A_k = 0.06, A_b = 0.15 and A_{tb} = 0.22 to obtain, respectively, retention in liver, kidneys, bone and a component distributed uniformly in the total body.

Molybdenum-99 decays to form technetium-99, which has a radioactive half-life of 0.25 day. Pertechnetate is used medically, and thus there are relatively good data on retention after absorption into blood. Beasley et al. (1966) give, for whole-body retention of a unit amount entering blood, the following equation:

$$R(t) = 0.76 \exp(-0.693t/1.6) + 0.19 \exp(-0.693t/3.7) + 0.043 \exp(-0.693t/22).$$

Since the radioactive half-life is about 0.25 day, the final term is of little significance and the activity is considered to be rather uniformly distributed in the body, except that the concentration in the thyroid may be high by a factor of about 20 (MIRD, 1975). On this basis we assign factors of A_{th} = 0.0054 for activity in the thyroid and A_{tb} = 1 for activity in the total body, and these are used with the R(t) given above to give the activity in the thyroid and in the total-body, respectively.

Pertechnetate is absorbed into blood almost completely (Beasley et al., 1966), but only about 50% of technetium chloride is absorbed into blood (Hamilton, 1948). Here f_1 = 0.95 is used for all compounds. Likewise, the clearance class is taken to be D for all compounds. This is probably a conservative assumption in that it tends to increase the estimate of dose received by the thyroid.

D8.9 RUBIDIUM

Absorption from the gastrointestinal tract to blood has been found to be approximately 100% (Richmond, 1958). Thus f_1 is taken as 0.95. Since rubidium is an alkali metal and most of its salts are readily soluble, it would be expected that absorption would be high for most compounds inhaled. All common compounds are indicated as being of clearance class D (Morrow, 1966).

Retention half-time is taken from studies on four adults who ingested the chloride in a saline solution (Richmond, 1958; Iinuma et al., 1967). Retention in the whole body, when corrected for decay and averaged, for these individuals yielded, for t in days,

$$R(t) = 0.1 \exp(-0.693t/4) + 0.9 \exp(-0.693t/95).$$

The activity is taken as uniformly distributed in the total body (Hamilton, 1948; Iinuma et al., 1967; Inaba et al., 1968). Rubidium-86, the only isotope of concern

here, decays to a stable element.

D8.10 RUTHENIUM AND RHODIUM

The metabolic behavior of ruthenium has been studied or reviewed by several authors (Burykina, 1962; Furchner et al., 1964; Thompson et al., 1965; Buldakov and Moskalev, 1968). All compounds of ruthenium are assigned to clearance class Y, and standard models for the lung and for the gastrointestinal tract are used. These assumptions are based on experimental data on the citrated complex of ruthenium, which was expected to be a reasonably soluble compound. However, in these studies, some 20% of the activity remained in the lung with a retention half-time of approximately 1 year. On this basis other compounds might also be expected to show a similar retention in the lung, and hence clearance class Y is used here for all compounds (Boecker and Harris, 1968).

The multispecies study by Furchner et al. (1964) yields an estimated absorption of ruthenium from the gastrointestinal tract of approximately 4%. The report of Thompson et al., (1956) explores in some detail the effect of pH on absorption into blood as well as absorption after chronic feeding experiments. Since the accident situation would not likely involve extended exposure by inhalation, the value of $f_1 = 0.04$ is adopted for this report.

The retention function for a single intake to blood has been studied by Furchner et al., (1964) in mice, rats, monkeys, and dogs. When the function is corrected for radioactive decay, one obtains the estimated retention for stable ruthenium. The retention in the total body is approximately

$$R(t) = 0.15e^{-2t} + 0.4e^{-0.07t} + 0.25e^{-0.012t} + 0.2e^{-0.0004t},$$

with t in days. The activity seems to be fairly uniformly distributed, kidneys being high by a factor of perhaps 3. The term of longest retention half-time might be considered to be the fraction present in bone and in muscle, but, since these tissues are rather generally distributed in the body and the radioactive half-life is 1 year or less, the activity reaching the blood is considered to be uniformly distributed throughout the body.

Several isotopes of ruthenium considered here decay to radionuclides of rhodium. These have radioactive half-lives on the order of minutes or hours except for rhodium-105, which has a radioactive half-life of approximately 1.5 days. All these isotopes of rhodium, except rhodium-105, are assumed to be essentially in equilibrium with the parent in all organs except the lungs and the gastrointestinal tract, for which the standard model is used.

Metabolic data on rhodium are quite limited, there being only one study. Hamilton (1947) injected rhodium into a few rats and studied its metabolism for 6 days. About one-third of the injected activity had a long retention half-time, the remaining two-thirds being excreted in 2 to 3 days. Because there is so few data and because of the short half-lives of these isotopes, the activity will be considered to be distributed uniformly in the body once it has reached the blood. Thus retention is given by

$$R(t) = 0.67 \exp(-0.693t/3) + 0.33 \exp(-0.693t/200)$$

for all rhodium isotopes reaching the blood. The standard models are used for the lungs and for the gastrointestinal tract. Because of the short radioactive half-life and by analogy with ruthenium, all compounds are assigned to clearance class Y. The value of $f_1 = 0.04$ is used, as in the case of ruthenium.

D8.11 TELLURIUM

Data on the uptake and distribution of stable tellurium are widely discrepant (Hollins, 1969; Soman, 1970; ICRP Publication 23, 1975). Fortunately most of the tellurium isotopes of concern here have a radioactive half-life of less than 4 days, but tellurium-129m (T_r = 33 days) and tellurium-127m (T_r = 105 days) have a possibility of longer retention in the body.

Hollins (1969) found $f_1 = 0.1$ in the rat for tetravalent tellurium, but Scott et al., (1947) found $f_1 = 0.25$ for uptake to blood after intramuscular injection in the rat. Scott and associates, (1947) also studied the absorption of hexavalent tellurium but did not cite a value for f_1 because of complications, although they did indicate that the uptake by blood was less than that for the tetravalent form. Because of the uncertainty of the results for the rat and because of the uncertainty of extrapolation from rat to man, $f_1 = 0.25$ is used here conservatively for all forms of tellurium.

Morrow (1966) indicates that the oxides would be expected to be of clearance class W, and this class is chosen here for all forms of tellurium.

Retention in the rat has been studied by Hollins (1969) and by Scott et al. (1947). Hollins has attempted to indicate the organs that might be represented by the various exponentials, and his prescription is adopted here--that is, merely assigning a long retention half-time to the final 2% which he indicated for bone. The retention function after unit intake to blood is given by

$$R(t) = 0.32 \exp(-0.693t/9.2) + 0.055 \exp(-0.693t/10.2) + 0.10 \exp(-0.693t/17.7) + 0.034 \exp(-0.693t/23) + 0.02 \exp(-0.693t/8000),$$

for t in days. The final term is assigned to bone, the kidneys are represented by the term with the coefficient 0.034, muscle by the 0.10 term, liver by the 0.055 term, and the term with the coefficient 0.32 is assumed to be distributed uniformly in the body. Though these are not exactly the assignments made by Hollins, they approximate his distribution and are somewhat simpler for dose estimation.

Several of the isotopes of tellurium decay to form a radioactive isotope of iodine. There is evidence that the iodine produced in the body, even in the rectum, is available to the thyroid and other organs (Revis and Muravei, 1959). It is assumed here that iodine produced in the lungs, the gastrointestinal tract, and in the systemic organs is available to the blood. For iodine produced in the lungs, stomach contents, and contents of the small intestine, absorption into blood had been included in the computer program. However, the program has been altered for use with tellurium so that iodine produced in the contents of the upper large intestine and the lower large intestine also reaches the blood to the extent of 95% of the activity of iodine present in those contents. Moreover, iodine produced in any of the systemic organs is assumed to reach the blood and follow the regular course of iodine metabolism.

D8.12 STRONTIUM-YTTRIUM AND BARIUM-LANTHANUM

The ICRP established a task group in 1968 to review the metabolism of the alkaline-earth elements, (ICRP Publication 20, 1973; Marshall et al., 1973). Essentially the report describes a computer program for estimating the accumulated activity (microcurie-days) in various organs per unit intake to the blood of an adult of any of the four elements calcium, strontium, barium, and radium and indicates the choice of parameters for each element. It also explains the choice of these parameters and describes the various tests that have been made to check the model. The model is in accord with all the major well-established biological data available for a normal adult. This model is adopted here for the adult. The microcurie-days for each of the time periods and iostopes used in this study, are listed in Table VI D-7.

The microcurie-days for each organ or tissue in Table VI D-7 are proportional to the number of disintegrations occurring in each of the source organs or tissues. To compute the average doses to the various tissues, these disintegrations are assumed to be uniformly distributed; this may seem to be somewhat at variance with the model, which does provide for some of the activity in bone being distributed in "hot spots." However, the model does not associate these higher concentrations of activity with any particular locations or structural features of bone, and for purposes of dosimetry the activity is considered to be uniformly distributed. This leads again to the uniform distribution as being representative of the average over many cases. With this uniform distribution, the dosimetry of Spiers (1968) is used to obtain the dose to the active marrow and also to the endosteal cells near bone surfaces. The data supplied by Marshall provide values of microcurie-days for blood, soft tissues, bone surfaces, and cancellous and cortical bone. These have been combined in

Table VI D-7 to produce microcurie-days in cancellous bone, in cortical bone, and in soft tissues. This accounts only for the microcurie-days accumulated after the activity enters the bloodstream. The standard ICRP lung model is used for all three clearance classes. However, the only compound of strontium definitely known to be in clearance class Y is strontium titanate, whereas the chloride, nitrate, sulfate, oxide, carbonate, phosphates, and similar ionic forms are all in class D. Following Marshall, a value of $f_1 = 0.2$ is used for absorption from the gastrointestinal tract into blood.

Similar remarks apply to barium, although the data on which the model is based are much more limited than those for strontium. Barium is chemically similar to strontium and to radium, which have been well studied in the human, and because the model is basically similar, it is adopted here. Barium is a constituent of fallout, and the available data on the behavior of barium in the environment tend to confirm the belief that it behaves similarly to strontium. Marshall has supplied the microcurie-days for barium-140 that are used in this report. The clearance classes for the barium compounds are the same as for strontium, and the value of $f_1 = 0.1$ is used for all compounds. This value is selected from the study of Leroy et al., (1966) and is appropriate for barium oxide, which was considered to be a simulant of environmental barium.

The only radioactive daughters of the radionuclides of strontium and barium considered here are isotopes of yttrium and lanthanum, respectively. When produced as daughters in an organ of the body, they are assumed to be retained in that organ as though they had just entered from blood or by inhalation. Their uptake by blood from the gastrointestinal tract to blood is assumed to 10^{-4}, and in the lung they are assumed to be of clearance class W. Yttrium reaching blood is assumed to behave as follows: 50% is deposited in bone, 15% in liver, and 10% in all other tissues; the remaining 25% is excreted. The deposition in bone is considered to be partitioned equally between cancellous and cortical bone. Retention is assumed to follow a single exponential with a retention half-time of 8000 days in all these tissues. This estimate of the distribution in tissues is based largely on data for yttrium-91 chloride inhaled by dogs (Lovelace Foundation, 1968a). However, the retention time in the whole body and in the organs is that given by Palmer et al. (1970) for promethium; that is, it is assumed that the retention of the trivalent yttrium ion will be similar to that of the lanthanides (Durbin, 1960).

For lanthanum, the distribution among organs is assumed to be that reported by the Lovelace Foundation (1968b, pp. 105-114) for inhalation studies with lanthanum-140 chloride in dogs: bone, liver, and muscle were high among the systemic organs in uptake, but in concentration liver, kidney, skeleton, and spleen were high in that order. In this report it is assumed that lanthanum retention in man is as follows: liver, 40%; bone, 40%; kidney, 5%; deposited in other soft tissues, 10%; and excreted, 5%. Since the radioactive half-life of lanthanum-140 is rather short, this model should be reasonably adequate.

Even when the microcurie-days accumulated in the various organs and tissues are known, there remains the problem of computing the microcurie-days for the daughter elements. This is done by an integration. If the retention of the daughter element in an organ due to 1 microcurie introduced into blood is given by

$$\sum_j A_j \exp\{-(\lambda_j + \lambda_r)t\},$$

then 1 microcurie of activity of a daughter radionuclide introduced into the organ by decay of the parent will produce a retention of

$$\sum_j A_j^* \exp\{-(\lambda_j + \lambda_r)t\}$$

at a subsequent time t, where the A_j^* are proportional to the A_j and $\Sigma A_j^* = 1$. The activity of the daughter produced in the various organs at different times is obtained by linear interpolation among the values supplied by Marshall. Since the daughter isotopes generally have a short radioactive half-life, this is believed to be sufficiently accurate for this purpose. A similar interpolation scheme is used to estimate the microcurie-days of the parent radionuclide and of its daughters accumulated in the various intervals of time for which dose equivalents are estimated. Though the data

of Marshall are available only for a single intake at time 0, the microcurie-days due to later intakes from the lung or from the gastrointestinal tract are assumed to vary linearly throughout these intervals of dose assessment. This interpolation procedure is probably conservative, but the discrepancy would not be expected to exceed a factor of at most 2 and even then only for radionuclides of short half-life.

Doses to fetuses, infants, and children, have not been as carefully studied, primarily because there is much less biological data available and because the individual is changing rather rapidly. Studies by Rivera (1967), Bennett (1972), and by Papworth and Vennart (1973) indicate that the dose to the red marrow or to the bone due to a short-term intake of the parent radionuclide might be higher by a factor of as much as 5 for the infant or child than for an adult. There is evidence of some placental discrimination, and the time during which the fractional uptake of the infant might significantly exceed that of the adult is limited. The analysis of data on fallout suggests that a factor of about 5 is conservative, but it is difficult to rule out a factor of about this magnitude for doses resulting from short-term intakes. However, in the case of infants this would apply only to the first year of life.

D8.13 ZIRCONIUM-NIOBIUM

Data on intake of zirconium and its concentration in some tissues of reference man (ICRP Publication 23, 1975) indicate a much higher absorption from the gastrointestinal tract into blood than has been found in experiments with small animals and a variety of chemical forms (Furchner et al., 1963; Schroeder and Balassa, 1965; Fletcher, 1969; Tipton et al., 1969). For inhalation exposure, this report assumes an f_1 value of 2×10^{-3} and a class W for all compounds except the oxides and hydroxides, which are class Y (Morrow, 1966).

Early work of Hamilton (1947) indicated that bone was a major site of retention after intake to blood. Fletcher (1969) and Furchner et al. (1963) have also given data on retention in rodents for periods of up to 14 months. For man retention is assumed to be given

$$R(t) = 0.5 \exp(-0.693t/7) + 0.5 \exp(-0.693t/8000).$$

This equation represents a recombination of the terms found by Fletcher and Furchner et al. and is consistent with the generally observed tendency for longer retention in larger animals. Ninety percent of the term for long retention half-time is assigned to bone; the other 10%, as well as the term for shorter retention half-time, is assigned to soft tissues.

Both radionuclides of zirconium considered in this report decay to form a radioactive isotope of niobium. Furchner and Drake (1971) have compared the metabolism of niobium in the mouse, rat, monkey, and dog; they have also reviewed the earlier literature. Their study showed that after the nuclide has reached blood, the bone has a concentration 10 times higher than the average concentration in the body and that kidneys, spleen, and testes have concentrations that are higher by a factor of about 3. All these organs seem to lose niobium at about the same rate. Thus retention in the total body following a unit intake to blood is given by

$$R(t) = 0.5 \exp(-0.693t/6) + 0.5 \exp(0.693t/200).$$

This formula is arrived at by adjusting the results on rodents as was done for zirconium. The short-term exponential is assumed to represent material distributed uniformly in all the tissues. The long-term exponential is separated into fractions present in bone, kidneys, spleen, testes, and other soft tissues by factors $A_b = 0.357$, $A_k = 0.855 \times 10^{-2}$, $A_s = 0.500 \times 10^{-2}$, $A_t = 0.128 \times 10^{-2}$, and $A_{ot} = 0.128$, respectively, each multiplied by $\exp(-0.693t/200)$.

Uptake from the gastrointestinal tract to blood is set at $f_1 = 0.01$, a value given by Fletcher (1969) and by Furchner and Drake (1971). The clearance class is taken as Y, since experimental work indicates long retention in the lungs (McClellan, 1968; Thomas et al., 1971).

TABLE VI D-7 MICROCURIE-DAYS ACCUMULATED IN CANCELLOUS BONE, IN CORTICAL BONE, AND IN SOFT TISSUES AFTER INTAKE OF 1 MICROCURIE OF THE INDICATED RADIONUCLIDE IN BLOOD[a,b]

Time (days)	Tissue	Strontium-89	Strontium-90	Strontium-91	Barium-140
2	Cancellous bone	0.151	0.153	0.0291	0.0745
	Cortical bone	0.161	0.164	0.0311	0.0817
	Soft tissue	1.18	1.20	0.410	0.614
7	Cancellous bone	0.609	0.636	0.0313	0.202
	Cortical bone	0.666	0.697	0.0334	0.227
	Soft tissue	2.23	2.30	0.416	0.907
30	Cancellous bone	1.97	2.31		0.401
	Crotical bone	2.30	2.71		0.479
	Soft tissue	4.09	4.58		1.37
60	Cancellous bone	3.09	4.20		0.467
	Cortical bone	3.70	5.08		0.565
	Soft tissue	5.21	6.45		1.49
180	Cancellous bone	5.17	12.8		0.487
	Cortical bone	6.34	16.0		0.591
	Soft tissue	6.37	10.6		1.51
365	Cancellous bone	5.57	24.1		
	Cortical bone	6.86	30.9		
	Soft tissue	6.47	13.1		
1825	Cancellous bone	5.60	82.7		
	Cortical bone	6.91	120.0		
	Soft tissue	6.47	17.1		
3650	Cancellous bone		119.0		
	Cortical bone		197.0		
	Soft tissue		17.9		
7300	Cancellous bone		147.0		
	Cortical bone		292.0		
	Soft tissue		18.2		
10,950	Cancellous bone		153.0		
	Cortical bone		347.0		
	Soft Tissue		18.3		
14,600	Cancellous bone		157.0		
	Cortical bone		376.0		
	Soft tissue		18.3		
18,250	Cancellous bone		158.0		
	Cortical bone		394.0		
	Soft tissue		18.3		

(a) Data supplied by courtesy of Dr. John C. Marshall.

(b) The entries indicated for a given time t represent the microcurie-days accumulated for the period 0 to t days. Blank spaces indicate the data are the same as the last entry in the column.

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Appendix E

Chronic Exposure Model

The deposition of radioactive material in the environment would result in a long-term exposure of man to both external and internal irradiation. Human exposure would be determined by personal habits and by the environmental factors that transport the radioactive material to man. These factors are best discussed under three headings:

1. External irradiation
2. Inhalation of resuspended radioactive particles
3. Ingestion of radioactive material

Each of these modes is discussed in this appendix, and appropriate models are derived for use in the calculation of long-term radiation doses to man.

E1 EXTERNAL IRRADIATION

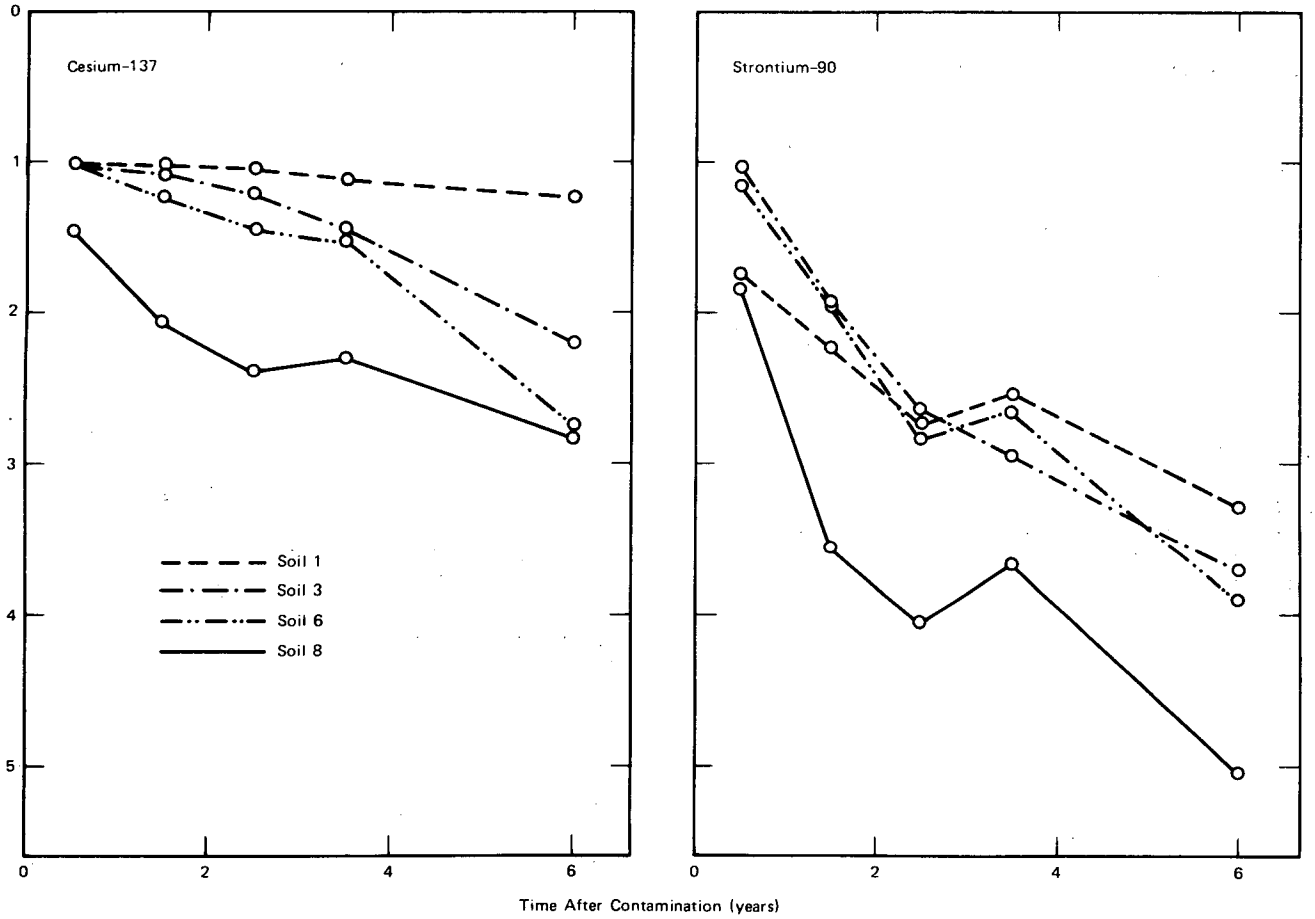
Radionuclides deposited from the air onto the ground or other surfaces would present a large, nearly uniform thin sheet of contamination. The hazards from this contamination, in the event the contamination were not subsequently inhaled or ingested, would be the gamma emissions and, for close body contact, the beta emissions of the radioactive contamination. The amount of radiation that a person would receive from a contaminated surface would depend on the amount of shielding between the contaminant and the receptor (human body).

There are a number of problems that must be adequately addressed to properly assess the level of external irradiation that the population would receive due to a particular level of contamination. Primary among these problems is an adequate description of the time-dependent behavior of the radionuclides deposited on the surfaces. This behavior would depend, of course, on the surface properties, the chemical and physical form of the radionuclides, and the external forces acting on the surfaces. For example, an important aspect of the behavior of radionuclides on the soil surface is the rate at which they would penetrate downward into the soil or at which they would be removed by erosion processes acting on the soil.

E1.1 BEHAVIOR OF RADIONUCLIDES IN SOILS

Movement of the radionuclides down into the soil would result in a reduction in the radiation flux above the soil due to the shielding effect of the soil. Because of its long radioactive half-life and the energy of the gamma radiation to which it gives rise, cesium-137 would be the dominant source of external radiation after short-lived radionuclides have decayed (Beattie, 1972). It is well established that cesium-137 is subject to considerable fixation in the soil as a result of its entrapment in the lattice structure of clay minerals (Jacobs and Tamura, 1960; Jacobs, 1963). The results of recent investigations of Squire and Middleton (1966) are compatible with the view of progressive fixation of cesium-137 on the clay minerals. Their results are reproduced in Fig. VI E-1. These results clearly show that cesium-137 does not penetrate rapidly into the soil. Even after 6 years, the median depth of cesium-137 was between 1 and 3 cm, depending on the soil characteristics.

Cline and Rickard (1972) measured soil profiles for cesium-137 at 8 years, and strontium-90 at 16 years, after surface contamination. In two separate experiments the soil profiles were measured in cultivated soil and in undisturbed (noncultivated) soil (see Figs. VI E-2a and b). In the cultivated field, the soil profile of both radionuclides assumed a fairly uniform distribution. In the undisturbed soil, the cesium-137 remained in the upper few inches of the soil.



Description of Soils

Soil	pH	Exchangeable Cations (mEq/100g) Total	Ca	K	Organic Matter (%)	Clay Content (%)
1. Acid clay	4.6	16.3	3.2	0.20	4.2	19.5
3. Sand	6.6	6.8	5.6	0.14	2.0	3.2
6. Lower greensand	6.0	13.2	8.5	0.28	2.1	11.0
8. Calcareous loam	7.6	26.0	28.5	0.95	5.5	16.8

FIGURE VI E-1 Changes in the median depth with time of cesium-137 and strontium-90 in different soils after contamination of the soil surface. From Squire and Middleton (1966).

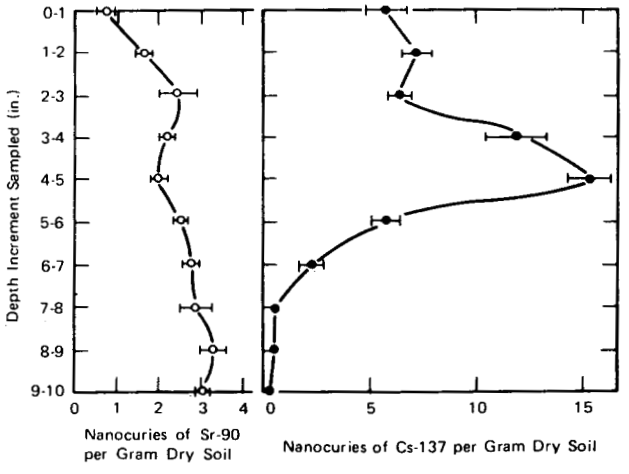


FIGURE VI E-2a Distribution of strontium-90 and cesium-137 in the soil profile of a tilled plot. From Cline and Rickard (1972).

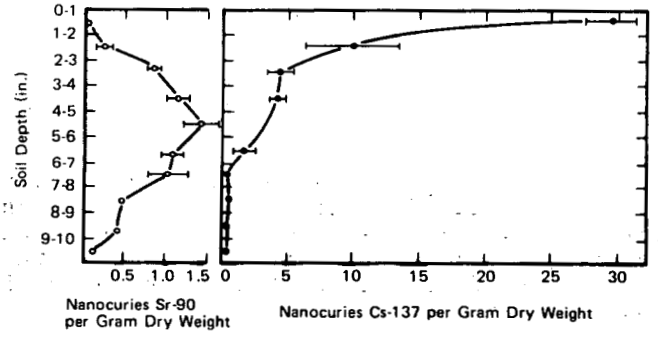


FIGURE VI E-2b Distribution of strontium-90 and cesium-137 in the soil profile of an untilled plot. The soil profile for strontium-90 was measured 16 years after surface contamination. The soil was silt loam: 66% sand, 28% silt, and 6% clay. The soil pH was 8.2, with an organic matter content of 1%. From Cline and Rickard (1972).

This same type of behavior is illustrated by data (see Table VI E-1) on the vertical distribution of fallout cesium-137 in several watershed areas in the United States (Ritchie and McHenry, 1973). Gale et al. (1964), who also examined a number of soil types, found that 50 to 80% of the cesium-137 was in the upper 5 cm of undisturbed soil about 5 years after contamination of the soil surface.

Similar types of observations have been made in a number of other investigations (Fredricksson et al., 1958; Peirson and Salmon, 1959; Walton, 1963; Essington et al., 1965; Kwaratskhelia et al., 1967; Pavlotskaya et al., 1967). The comparison of the radionuclide distribution characteristics in various soils shows, in general, the following mobility sequence:

$$^{90}Sr > ^{106}Ru > ^{144}Ce > ^{137}Cs.$$

In cultivated soils, all radionuclides show a fairly uniform distribution, whereas in noncultivated soil, their penetration more than a few centimeters into the soil is a relatively slow process.

Beck (1966) has shown that fallout radionuclides present in the soil for longer than 1 month can reasonably be assumed to be distributed exponentially as a function of depth, with a relaxation length of approximately 3 cm. Figure VI E-3 (from Beck, 1966) indicates that this exponential approximation is consistent with experimentally determined depth distributions.

Calculations of the dose rate from deposited radionuclides have been extensively performed by many investigators. The assumption utilized in these calculations is that the source is evenly distributed on a plane of zero thickness and infinite extent (Loutit et al., 1960; Gustafson et al., 1963; Beck and dePlanque, 1968). The effect of ground "roughness" is generally accounted for by assuming that the plane source is buried at some depth beneath the surface. For a widely distributed source and at distances of at least 1 meter above the surface, the assumption of an average exponential depth distribution should be more realistic and should also account for ground roughness (Beck and dePlanque, 1968).

Gale et al. (1964) have studied explicitly the cesium-137 gamma-ray dose rate reduction as a function of time after surface contamination of various soil types. The mean values of the gamma dose rates in air at 1 meter above the surface of the various soils was found to fit the time-dependent expression

$$D_g = D_o \left[0.63 \exp(-1.13t) + 0.37 \exp(-0.0075t)\right] \exp(-\lambda t), \qquad \text{(VI E-1)}$$

where D_0 is the dose rate from a plane source at zero time, λ is the radioactive decay constant of cesium-137, and t is measured in years. Gale et al. found that the dose rate decreased rather rapidly during the first 4 years after the contaminating event. Thereafter, however, the dose rate changed little apart from what could be attributed to radioactive decay. The results of their measurements of the profile of cesium-137 in the soil were consistent with those of other investigators (e.g., Squire and Middleton, 1966).

The results from Beck (1966), shown here in Table VI E-2, indicate that the ratio D_g/D_0 is essentially independent of gamma-ray energy. Therefore, Equation (VI E-1) can be used in a model for other radionuclides by substituting the appropriate half-life for λ in Equation (VI E-1).

TABLE VI E-1 VERTICAL DISTRIBUTION OF FALLOUT CESIUM-137 IN TILLED AND NONTILLED SOILS[a]

Depth (cm)	Cesium-137 Concentration (nCi/m^2)									
	Watershed A[b]		Watershed A[c]		Watershed B[d]		Watershed C[e]		Watershed D[f]	
	Tilled	Non-tilled	Tilled	Non-tilled	Tilled	Non-tilled	Tilled	Non-tilled	Tilled	Non-tilled
0 to 5...	29.3	135.2	31.4	133.9	27.3	100.7	11.4	51.0	47.5	85.1
5 to 10...	32.9	22.2	33.2	16.9	25.5	15.2	19.6	27.3	46.7	25.2
10 to 15...	23.4		31.5		26.9		16.2		22.7	
15 to 20...	10.1		20.3		27.3		12.6		6.7	
20 to 25...	5.0		3.4		17.2		4.3			
25 to 30...					4.0					

(a) From Ritchie and McHenry (1973).
(b) Holly Springs, Miss.
(c) Data from another series of measurements.
(d) Ashland, Mo.
(e) Durant, Okla.
(f) Gainesville, Tex.

TABLE VI E-2 RATIO OF GAMMA-RAY DOSE-CONVERSION FACTORS

Radionuclide	D_g/D_0 Ratio[a]
Manganese-54	0.42
Zirconium-95	0.40
Niobium-95	0.40
Ruthenium-103	0.41
Rhodium-106	0.39
Antimony-125	0.41
Cesium-137	0.43
Barium-140	0.40
Lanthanum-140	0.40
Cerium-141	0.41

(a) The ratio is the gamma-ray dose-conversion factor for a source distributed exponentially in soil, with a relaxation length of 3 cm, to the dose conversion factor for a uniform infinite plane source.

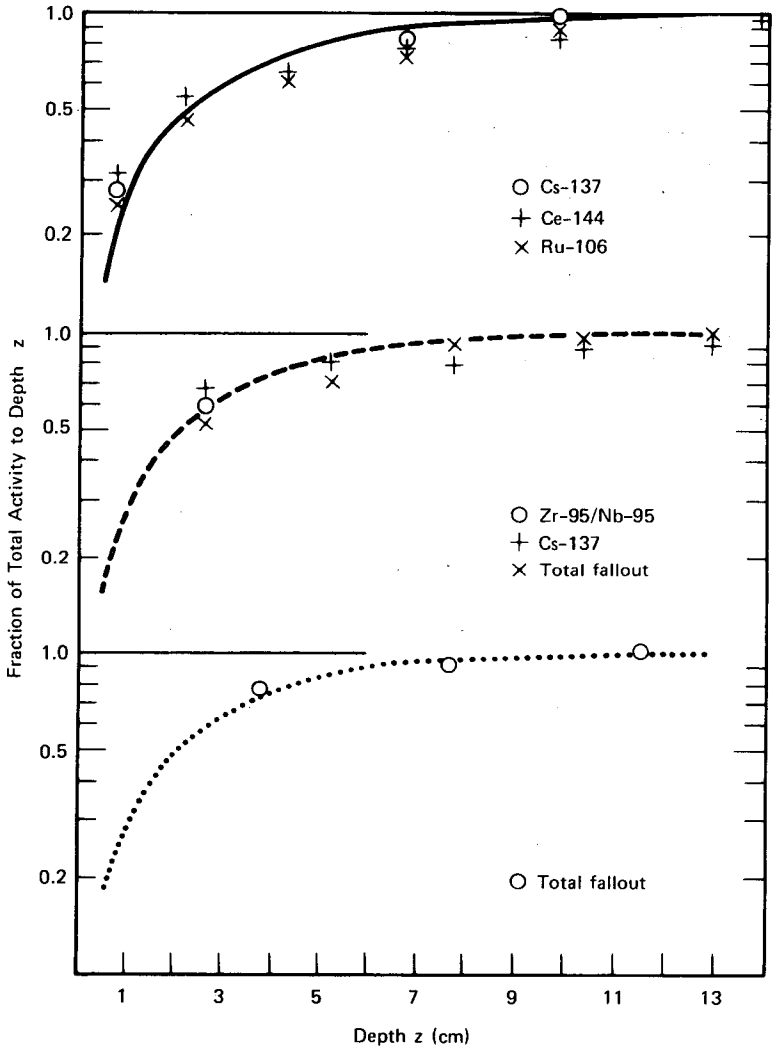


FIGURE VI E-3 Distribution of radioisotopes with depth in the ground. The curves give the fraction of the total activity to depth z [F = 1 - exp(-z/3)], assuming an exponential decrease in activity with a 3-cm relaxation length. This assumed exponential distribution is compared with the experimental data of Walton (1963) in curve A, with the data of Telfair and Luetzwlschwab (1974) in curve B, and with the measurements of Gustafson (1958) in curve C. From Beck (1966).

E1.2 BEHAVIOR OF RADIONUCLIDES ON PAVED AREAS AND STRUCTURE SURFACES (ROOFS AND WALLS)

The deposition and adherence of aerosol particles on hard surfaces, such as paved areas, are not well understood. Few of the factors that influence particle adherence can be quantitatively defined. However, one would expect that some of the deposited radionuclides would be washed from paved surfaces by rain. There is some evidence (Fumis et al., 1964) to suggest that this happens to weapons fallout, but it is not conclusive enough to support any guidelines on general behavior.

In a typical urban area, there is a significant amount of exposed ground surrounding the residential houses. Assuming that the paved areas (street, driveway, and sidewalks) were completely washed of deposited material, the dose received in the house would not be significantly reduced (Bryan et al., 1970). In addition, because of the large amount of shielding in the lower part of vehicles, irradiation directly from the street while driving would not contribute a large part of the dose received in the vehicle (Burson and Profio, 1975). Therefore, having streets that are naturally "cleaned" would not greatly reduce the total external radiation dose.

In accounting for the amount of protection afforded by structures, it is very important to be able to describe the deposition of radioactive aerosols on vertical surfaces. Experiments on the deposition of aerosols on vertical surfaces are essentially nonexistent for real buildings; however, measurements (Sehmel, 1973) in the laboratory have demonstrated significant deposition of small aerosol particles on vertical surfaces. The deposition on the vertical surfaces was on the order of 20% of that on horizontal surfaces under the same flow conditions. However, an important parameter of the problem--the time-dependent behavior of the radionuclides on the vertical surfaces--is not known.

E1.3 EXTERNAL IRRADIATION MODEL

This section discusses the methodology utilized in the consequence model to calculate the long-term external dose. The basis for the methodology is the work of Gale et al. (1964), as explained in section E1.1.

The initial dose rate in tissue from a uniform infinite plane source of radionuclide i is related to the surface deposition, or contamination level, by

$$RD_0^i = (SF)(DC^i)(SD_o^i), \qquad \text{(VI E-2)}$$

where SF is the shielding factor to account for the protection afforded by houses, vehicles, etc. (dimensionless), DC^i is the dose-conversion factor to relate surface deposition level to dose rate in the organ of interest (e.g., from the EXREM-III program), calculated in units of rem per year per curie per square meter, and SD_o^i is the initial surface deposition level, in curies per square meter.

The dose rate, due to radionuclide i, as a function of time is found by combining Equations (VI E-1) and (VI E-2).

$$RD^i(t) = (SF)(DC^i)(SD_o^i)\ [0.63\ \exp(-1.13t) + 0.37\ \exp(-0.0075t)]\ \exp(-0.693t/T_{\frac{1}{2}}^i). \qquad \text{(VI E-3)}$$

It is assumed that the shielding factor (SF) is independent of the gamma-ray energy and hence the radionuclide. The dose received by an individual over a period of t′ years from the contaminated ground is computed by integrating Equation (VI E-3) from 0 to t′. The assumption is that the individual would continuously reside on the contaminated ground from the time of radionuclide deposition.

$$RD^i_{t'} = (SF)(DC^i)(SD^i_0)\left\{ \frac{0.63}{1.13 + (0.693/T^i_{\frac{1}{2}})} \left[1.0 - \exp\left(-1.13\, t' - \frac{0.693}{T^i_{\frac{1}{2}}}\, t'\right)\right] \right.$$

$$\left. + \frac{0.37}{0.0075 + (0.693/T^i_{\frac{1}{2}})} \left[1.0 - \exp\left(-0.0075\, t' - \frac{0.693}{T^i_{\frac{1}{2}}}\, t'\right)\right] \right\} \qquad \text{(VI E-4)}$$

The dose commitment in Equation (VI E-4) can be inverted to find a surface-contamination level for radionuclide i that corresponds to a particular dose commitment received in t' years. This permits one to determine an acceptable level of surface contamination for radionuclide i when a permissible dose commitment is specified. The implicit assumption in the utilization of Equation (VI E-4) is that only radionuclide i contributes to the total dose received. In the hypothetical accident, however, many radionuclides would be released, all of which would contribute simultaneously to the total dose.

Let the acceptable dose commitment be denoted by RD_L and let the corresponding surface-contamination level of radionuclide i that delivers exactly RD_L be denoted by SD^i_L. If the actual surface-contamination level of radionuclide i is SD^i_{act}, which may be very different from SD^i_L, the dose that would be received can be calculated from the ratio of the actual to permitted surface-contamination level, multiplied by the dose commitment RD_L:

$$\frac{SD^i_{act}}{SD^i_L}\, RD_L = \text{dose received.} \qquad \text{(VI E-5)}$$

Therefore, to find whether an acceptable dose commitment would not be exceeded when there is a variety of radionuclides contaminating the ground, the following condition must be fulfilled:

$$\sum_{i=1}^{I} \frac{SD^i_{act}}{SD^i_L} \leq 1.0, \qquad \text{(VI E-6)}$$

where SD^i_L is derived from Equation (VI E-4) when $RD^i_{t'}$ is set to the total permissible dose commitment, SD^i_{act} is the actual surface contamination level of radionuclide i, and I is the total number of radionuclides contributing to the dose.

If the sum of the ratios is greater than 1.0, the acceptable dose commitment RD_L will be exceeded by the fraction that the sum exceeds 1.0, if one were to reside on the land continuously. For the acceptable dose commitment not to be exceeded, it is necessary either to decontaminate the land or to deny use of the land (interdiction) for a period of time. To calculate the time necessary for land interdiction (t_{land}), assuming no decontamination, one must solve for t_{land} such that

$$\sum_{i=1}^{I} \int_{t_{land}}^{t_{land}+t'} RD^i(t)\, dt = RD_L \qquad \text{(VI E-7)}$$

where t' is the time period over which a dose RD_L is received.

Since this equation contains I unknowns, an iterative procedure is necessary to find t_{land}. The problem can be somewhat simplified by bounding the solution and interpolating. Since a maximum period of t_{max} years is considered for the expenditures for land interdiction, the first test is to see if

$$R_{max} = (RD_L)^{-1} \sum_{i=1}^{I} \int_{t_{max}}^{t_{max}+t'} RD^i(t) \quad dt \leq 1.0 \qquad \text{(VI E-8)}$$

is satisfied. If this condition is not fulfilled, then the time period for land interdiction is given as $t_{land} = t_{max}$. On the other hand, if the above condition is satisfied two additional trials are made with $t = 1$ and $t = 2$ years in

$$R_t = (RD_L)^{-1} \sum_{i=1}^{I} \int_{t}^{t+t'} RD^i(t) \quad dt. \qquad \text{(VI E-9)}$$

The periods of 1 and 2 years is used because a significant portion of the radionuclides that would contribute to external irradiation have half-lives on the order of 1 year or less. Therefore, during the first year after deposition, the dose rate would decrease appreciably. In the event that $R_{2.0} > 1.0$, an exponential interpolation is performed to calculate t_{land}:

$$t_{land} = 2.0 + (t_{max} - 2.0) \frac{\ln(1.0/R_{2.0})}{\ln(R_{max}/R_{2.0})}. \qquad \text{(VI E-10)}$$

To calculate the dose commitment to people living on the contaminated land, assuming that $t_{land} < t_{max}$ has been satisfied, the time integration of Equation (VI E-3) is carried out over several time periods and summed for each radionuclide considered.

E2 INHALATION OF RESUSPENDED RADIOACTIVE PARTICLES

E2.1 REVIEW OF EXPERIMENTAL DATA

Resuspension is the entrainment of surface particulates into a fluid layer adjacent to a surface. As a process in nature, it is ubiquitous, occurring in deserts, streams, oceans, grasslands, homes, highways, etc. It is a term generally applied, at least in the nuclear context, to radioactive particles that were originally airborne but deposited on the surface--hence resuspension. This process may be an important component in calculating the hazards arising from deposits of fission products and actinides.

Much work has been devoted to quantifying the long-term hazards from resuspended radioactive particles. Table VI E-3 indicates the quantity of work and its principal areas of impact. It is clear that resuspension from arid lands has received most attention. This emphasis has occurred primarily because fallout fields from nuclear weapons tests are located in arid areas.

Most of the work shown in Table VI E-3 was devoted to measuring a "resuspension factor" K, defined as

$$K = \frac{\text{resuspended air activity } (Ci/m^3)}{\text{deposited ground activity } (Ci/m^2)}. \qquad \text{(VI E-11)}$$

TABLE VI E-3 Experimental Resuspension Factors

Arid/Desert	Semiarid/Grassland	Urban/Suburban	Interiors
4×10^{-7} near road graded in fallout field[a]	5×10^{-6} (ZnS) walking and loading boxes[f]	2.5×10^{-6} sandblasting for I-131 removal[g]	1×10^{-5} heavy work[i] ZnS (3.1 μm)
3×10^{-5} in Land Rover during travel to work site and outside during work; fission products (5-18 hr)[a]	1×10^{-5} ($\pm0.5\times10^{-5}$) limestone, rock, sand, grass, bushes; fission products[a]	2×10^{-6} 0- to 4-μm U_3O_8 particles on concrete slabs[a]	7×10^{-4} light work[i] CuO (2 μm)
3 to 10×10^{-4} downwind of crater[a]	0.8 to 2×10^{-7} crater of tower shot; fission products; road graded from soil disturbance[a]	1.5×10^{-7} 0-to 12-μm U_3O_8 particles on concrete[a]	2×10^{-3} operations[j]
1×10^{-3} mechanical disturbance[a] — UO_2	2×10^{-5} to 4×10^{-6} in vehicle on road graded through fallout[a]	10^{-4} to 10^{-2} fraction resuspended per pass through particles[h]	0.4 to 7×10^{-3} clothes change and other activity[k]
5×10^{-4} dust from vehicles	8×10^{-6} 0 to 4 μm U_3O_8	K = 2×10^{-2} to 2×10^{-4}; initially very rapid half-time of a few days	1.2×10^{-4} people walking in change room[k]
10^{-4} to 10^{-6} dust from pedestrians[a] — PuO_2	5×10^{-7} 0 to 12 μm U_3O_8 — undisturbed sandy soil with desert grasses[a]	$\sim2\times10^{-5}$ to 2×10^{-3} at 4 days	5×10^{-5} PuO_2, many steps[l]
3×10^{-7} particles of 7 μm MMD on 6-m circles (sandy) furrowed and rocky with sand base (dry)[b]	8×10^{-9} to 10^{-11} Y-91 aqueous solution on sandy soil-rain weathering small plot[a]		3×10^{-6} $PuNO_3$, many steps[l]
1×10^{-7} to 3×10^{-7} (wet)[b]	1×10^{-7} for particles of 7 μm MMD on 6-m circles, grass plot (dry)[b]		2×10^{-4} to 4×10^{-5} in enclosed space, I-131 in brick dust[a,g]
1.2×10^{-7} in 1000-μg/m^2 isopleth	4×10^{-8} for particles of 7 μm MMD on 6-m circles, grass plot protected by snow fence (dry)[b]		
1.4×10^{-7} in 100-μg/m^2 isopleth	2×10^{-7} both cases, above, wet[b]		
1.6×10^{-6} in 10-μg/m^2 isopleth — PuO_2 Safety Test[c]			
~35 day half-time air concentration decay 1 to 3-μm median aerosol size			
38-day half-time for resuspension of fission products from Schooner[d]			
76-day half-time for resuspension of fission products from Baneberry[d]			
3×10^{-10} for 8- to 10- year Pu aged deposit[e]			
2×10^{-9} for direction of strongest wind[e]			

(a) Stewart (1964).
(b) Healy and Fuguoy (1959).
(c) Wilson et al. (1960).
(d) Anspaugh et al. (1973).
(e) Anspaugh (1973).
(f) Schwendiman (1958).
(g) Chamberlain et al. (1951).
(h) Anspaugh et al. (1974).
(i) Fish et al. (1965).
(j) Bailey and Rohr (1953).
(k) Brunskill (1964).
(l) Jones and Pond (1964).

Values for K were determined by dividing measured air concentrations by measured ground or surface deposit. Thus, the values listed in the table, which range from 10^{-2} to 10^{-10} m^{-1}, imply that a surface deposit of one unit (e.g., curie) per square meter would give an air concentration of 10^{-2} to 10^{-10} units per cubic meter. While this approach has the advantage of simplicity, it lacks any semblance of universality. Any measurement of K is likely to hold only for the place and the instant at which the measurements were performed. It should be clear that the resuspension factor, at the very minimum, is a function of the surface condition, the particle deposited, the inactive particles present on the surface, the local meteorology, and the height at which concentrations were measured.

Another variable of importance is the length of time since the particles were deposited. Some attempts to account for deposit age have been made by using an availability half-time that acknowledges the decrease in measured air concentration with time after deposit. Kathren (1968) and Langham (1969) have each formulated predictive resuspension models for the resuspension factor of the form

$$K(t) = K_o \exp(-\lambda t), \qquad \text{(VI E-12)}$$

with values of λ corresponding to a half-time of 35 to 45 days. This formulation appears to agree reasonably well with the results of experimental measurements made up to several weeks after deposition. At long deposition periods the implied half-times become long and seem to range into periods of years. Figure VI E-4 is a graphical representation of several models used to describe the time dependence of the resuspension factor (Anspaugh et al., 1974a). The obvious conclusion is that there is a large uncertainty associated with the long-term behavior of the resuspension factor.

Another method of accounting for resuspension is to assume that, when particles are removed from the surface, the fractional makeup of the suspended material is the same as it was in the particulate matter on the surface. Thus, if one knows the concentration of trace particles on the surface material and the concentration of surface material in air, the concentration of trace material is found directly. This "mass loading method" is likely to apply especially to "aged" deposits, where the trace materials initially deposited on the surface, have had time to be mixed thoroughly into the upper few centimeters of the surface material. The concept ignores the important effects of particle size distribution, the attachment of radioactive particles to soil particles, and the relative suspendability of various size fractions of surface material. However, it would permit the use of the large volume of knowledge amassed by Bagnold (1954), Chepil (1965), and others on the dependence of total suspended soil mass on meteorological and soil factors.

For an urban environment in which artificial surfaces are very common, the mass loading method may not be useful since there is no depth of material into which the contaminant particle could disperse. In fact, the recent work of Sehmel (1973b) on resuspension from roads subject to vehicular traffic is stated in terms of resuspension factors.

The problem with either method of accounting for resuspension is that the basic physical principles governing the process remain largely unknown. A few questions that might be asked in the interest of putting resuspension prediction on a firm footing are the following:

- To what extent does attachment of active particles to inert particles affect results?
- What is the mechanism by which particles are removed from the surface and transported to a sampler at a typical height of one meter?
- To what extent is resuspension of small particles dependent on the large-particle fraction of the soil size distribution?
- How does resuspension occur in an urban environment or in buildings where winds may be of little or no importance?

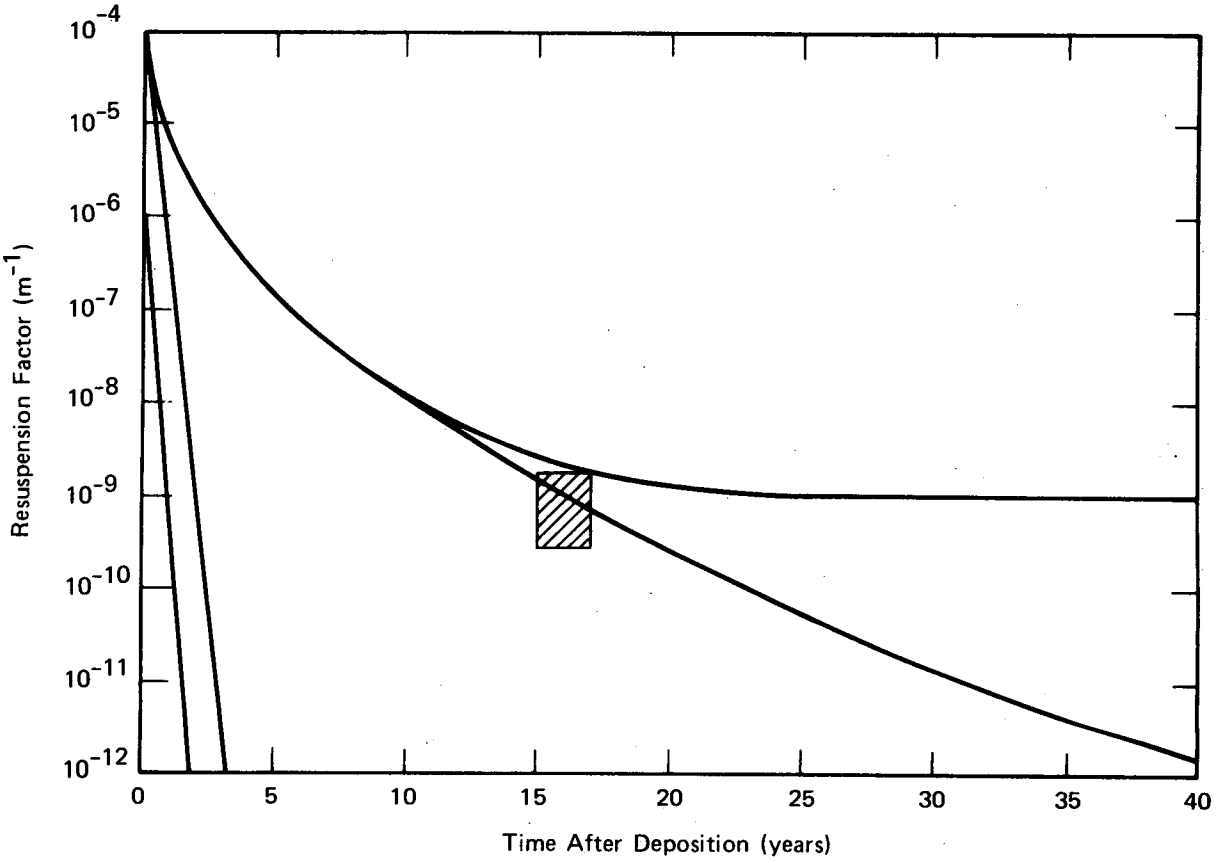


FIGURE VI E-4 Graphical representation of several time-dependent resuspension factor models. From Anspaugh et al. (1974a).

Until such questions are answered, the empirical data summarized in Table VI E-3 must be used only for guidance purposes.

E2.2 RESUSPENSION AND INHALATION MODEL

In spite of the difficulties in assigning appropriate resuspension factors to particular situations, it is still necessary to utilize the concept for the calculation of inhalation hazards from resuspended radioactive material. A number of investigators have recommended use of various resuspension values in hazard calculations. For example, Stewart (1964) recommended a value of 10^{-6} m^{-1} as appropriate for quiescent conditions outdoors and 10^{-5} m^{-1} for conditions of moderate activity. Langham (1971) suggested a value of 10^{-6} m^{-1} as a reasonable average value. Kathren (1968) used 10^{-4} m^{-1} as a conservative value for standard-setting purposes.

These recommendations are for the most part based on experimental evidence that is not directly applicable to the particular situation being considered for a nuclear reactor accident (i.e., well-vegetated areas). It could be argued that the initial resuspension factor in well-vegetated areas would be higher than an arid region, primarily because of the deposition on foliage with a higher susceptibility for removal than from the ground. However, as reviewed in the section dealing with the ingestion of deposited radionuclides, only some 50% of the depositing material would be retained on the vegetation, and for this fraction the mean resident time on the vegetation would be only 20 days.

For radioactive material not depositing on the vegetation and for material depositing on the vegetation and later transferred to the soil, it is believed that the vegetation and root mat would prevent resuspension. Therefore, as a reasonable estimate, a resuspension factor of 10^{-5} m^{-1} can be used as an initial value for conditions shortly after the initial deposition.

The only long-term studies of the resuspension factor are those reported by Anspaugh et al (1974b) and Volchok (1971). Resuspension factors for plutonium of 10^{-9} and 10^{-10} m^{-1} were measured at the Nevada Test Site in an area contaminated 17 years previously, as reported by Anspaugh et al. (1974b). Measurements of plutonium in the vicinity of the Rocky Flats plant (Volchok, 1971) several years after deposition, indicate a resuspension factor of 10^{-9} m^{-1}. Therefore, it is assumed that an equilibrium resuspension factor 10^{-9} m^{-1} would be reached in a time period of 17 years after initial deposition of the radioactive material. Since the explicit time behavior of the resuspension factor is not known, an exponential decrease from 10^{-5} to 10^{-9} m^{-1} in 17 years is assumed; this results in a resuspension factor half-life of about 1 year. As there are no data to support any estimates of the resuspension factor after 17 years, it is assumed that it would remain constant at 10^{-9} m^{-1} for the life of the radionuclide.

To summarize, it is assumed that the time-dependent resuspension factor is described by

$$K(t) = K_0 \exp(-\lambda t) + K_e \qquad \text{(VI E-13)}$$

where $K_0 = 10^{-5}$ m^{-1}; $K_e = 10^{-9}$ m^{-1}; $\lambda = \ln 2/0.977 = 0.677$ yr^{-1}.

This time-dependent resuspension factor is utilized to calculate the quantity of radioactive material inhaled and therefore the dose.

The inhalation of resuspended radionuclides is the product of man's inhalation rate, the surface contamination level, and the resuspension factor:

$$I^i(t) = (IHR)(SD_0^i) \exp(-0.693t/T_{\frac{1}{2}}^i) K(t), \qquad \text{(VI E-14)}$$

where i is the radionuclide index, $I^i(t)$ is the rate of inhalation of radionuclide i (curies per year), IHR is the inhalation rate of man (7300 m^3/yr for standard man), SD_0^i is the initial surface deposition level of radionuclide i (curies per square meter), $\exp(-0.693t/T_{\frac{1}{2}}^i)$ is a factor to account for the radioactive decay of radionuclide i

having a half-life of $T_{\frac{1}{2}}^i$ years,[1] and K(t) is the time-dependent resuspension factor (m^{-1}).

The total quantity of radionuclide i that is inhaled is found by integrating Equation (VI E-14) over all time.

$$\hat{I}^i = \int_0^\infty I^i(t)\, dt = (IHR)(SD_0^i)\left[\frac{10^{-5}}{0.677 + (0.693/T_{\frac{1}{2}}^i)} + \frac{10^{-9}}{0.693/T_{\frac{1}{2}}^i}\right]. \qquad \text{(VI E-15)}$$

An examination of Equation (VI E-15) reveals that only for very long half-lives ($T_{\frac{1}{2}} > 10^4$ years) does the second term approach the first in magnitude. Therefore, most of the material would be inhaled within the first couple of years after deposition.

The use of Equation (VI E-15) implies the addition of radionuclides inhaled by many successive generations. Therefore, to appropriately assign risk, the integration of Equation (VI E-15) for dose commitment calculations is performed over consecutive time periods after deposition to give $\hat{I}_k^i$, where k is the time interval index; that is,

$\hat{I}_1^i$ = quantity of radionuclide inhaled in time 0 to 10 years after deposition

$\hat{I}_2^i$ = quantity of radionuclide inhaled in time 10 to 20 years after deposition

and so forth.

The relation between inhaled radioactive material and the radiation dose received by an individual is

$$RD_j = \sum_{k=1}^{j} \sum_{i=1}^{I} DC_k^i \hat{I}_{j+1-k}^i \qquad \text{(VI E-16)}$$

where RD_j is the radiation dose delivered to a particular organ in the time interval j (rem), DC_k^i is the dose-conversion factor in the time interval k for a unit quantity of radionuclide i inhaled during the first time interval after deposition (rem per curie inhaled), and $\hat{I}_k^i$ is the quantity of radionuclide i inhaled during the time interval k (curies inhaled).

E3 INGESTION OF RADIOACTIVE MATERIAL

The deposition of radioactive material in the environment could result in radiation being received from later ingestion of water or foodstuffs grown in the contaminated environment. This mode of exposure is often the one that provides the most stringent criterion for acceptable levels of contamination and affects the greatest area of land.

The contamination could result either from direct deposition of the radioactive materials on vegetation or from incorporation of the radioactive materials into vegetation. Because of the many uncertainties in the deposition process and the chemical and biological features of food chains, direct estimates of the potential ingestion of the radioactive material by man are difficult and uncertain. Two recent extensive reviews of these areas (Russell, 1966a; Garner, 1972) provide an excellent overview of the problems.

The problem of direct deposition on vegetation is transitory in nature since it affects only a single crop; however, the incorporation of the radioactive material into vegetation is a long-term problem since plants can take up the radioactive material in the

[1] In the event that the radionuclide has a precursor in the environment (e.g., curium-242 decays to plutonium-238), an additional factor is included to account for the source of the radionuclide.

soil through their roots over many growing seasons.

E3.1 DIRECT DEPOSITION OF RADIOACTIVE MATERIAL ON VEGETATION

There are a number of important parameters relevant to the transport of deposited material to man. The parameters of interest are considered to be:

1. Initial retention of radioactive material (aerosols and vapors) on vegetation.
2. Removal of deposited material from vegetation.
3. Transport of deposited material from vegetation to man.

E3.1.1 Initial Retention of Radioactive Material on Vegetation

As one might expect, the deposition process plays an important role in the interception and initial retention by vegetation of airborne radioactive material. Precipitation washes out material from the atmosphere with an efficiency that depends on the precipitation rate, the height of the air layer involved, and the physical and chemical properties of the vapor or aerosols. Only a fraction of this wet deposition is retained on vegetation. Studies of iodine retention from 1961 weapons fallout in Britain (Chamberlain and Chadwick, 1965) showed that 40 to 60% was retained on vegetation during wet deposition. In experiments with simulated rainout, as described by Middleton (1960), initial retentions of carrier-free strontium-90 and cesium-137 were found to be as follows: wheat, 20 to 60%; cabbage, 8 to 30%; potatoes, 25 to 65%; sugar beet, 12 to 30%. When dense grass was sprayed, about 60% of the applied material was removed with the grass that was removed by close mowing. These percentages can be expected to be upper limits of retention since the solutions were applied as a fine spray. Rain would usually be of longer duration and would wash away variable amounts of already deposited material. In most studies, rain was found to be a major factor leading to loss of material from foliage. The retention of fallout strontium-90 and cesium-137 on plants in East Germany (Bose, 1969) (where wet deposition can be expected to predominate) has been estimated to be between 23 and 49%. In Britain, a series of field experiments (Milbourn and Taylor, 1965) were carried out on various types of grassland by lightly spraying strontium-89. The extent of retention ranged from about 15 to 30% on those plant parts that might be eaten by grazing cattle. The cited retention values should not be considered constants. One would expect the values to vary with precipitation rate and with the properties of the depositing material.

The dry deposition of gases, vapors, and aerosols on vegetation is a complex mechanism. For vapors and gases, and for particles so small (~1 micron) that their settling velocity is negligible, the deposition is assumed to occur through filtering effects when the ground layer of air comes in contact with vegetation and ground. The filtering efficiency is influenced by the amount and by the properties of the vegetation, as well as by the characteristics of the airborne material, of the ground, and the atmospheric conditions. For example, early studies on the deposition of fallout nuclides indicate that deposition rates on grass could be up to five times higher than on horizontal filter papers (Megaw and Chadwick, 1965). These effects are illustrated quite clearly in the results of Pelletier and Zimbrick (1970), reproduced here in Fig. VI E-5. The figure summarizes the measurements of the deposition rate of gaseous molecular radioiodine made during a field release. The correlation between the deposition velocity and a real grass density is significant and seems to indicate that the quantity of radioiodine deposited is directly proportional to the surface area available for deposition. Their data indicate a proportionality up to about 300 gm/m^2, above which the proportionality decreases. Hawley and Markee (1965) also observed the strong influence of the mass per unit area of grass on the amount of radioiodine deposited per unit area in controlled field-test release of gaseous molecular iodine-131. Chamberlain (1970) assumes the relationship between initial retention of deposited vapors and aerosols and herbage yield to take the form

$$p = 1 - \exp(\nu w), \qquad \text{(VI E-17)}$$

where p is the fraction initially retained, w is the herbage density (in kilograms of dry matter per square meter), and ν is an uptake coefficient (expressed in square meters per kilogram). Values for ν, experimentally derived, lie in the range 2.3 to

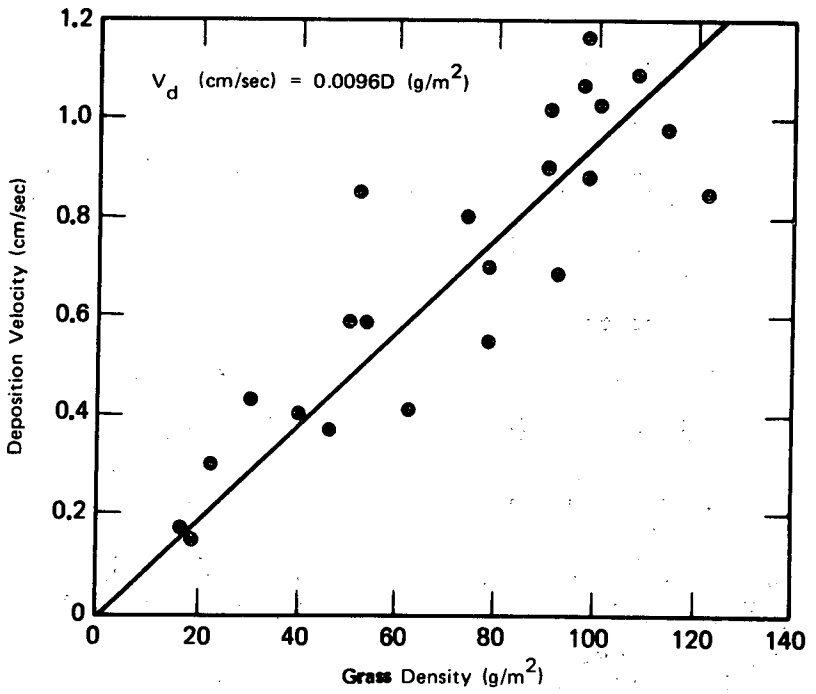


FIGURE VI E-5 Measured deposition velocities of gaseous molecular radioiodine as a function of areal grass density (dry weight). From Pelletier and Zimbrick (1970). Reprinted by permission from Charles C Thomas, Publisher.

3.3 m^2/kg. Chamberlain's data, supporting Equation (VI E-17), are given in Figs. VI E-6a and b. Although there is considerable scatter in the data points, the general trend is quite evident. For an average pasture having $250g/m^2$ of plant dry matter, the above expression yields a figure of 44 to 56% for initial retention. In dense herbage, as might be expected, the contamination is confined largely to the upper parts of the foliage.

Measurements made after the Windscale accident of average levels of iodine-131 in milk were compatible with predictions made on the basis that 25% of the deposit was originally retained on edible tissue (Loutit et al., 1960). This value is frequently used in the assessment of milk contamination with iodine-131 after a large accidental release (Bruce and Russell, 1969; Bryant, 1969; Garner, 1960). However, estimates of the iodine-131 retention factor range from 6 to 85% (Cline et al., 1965).

Few data are available on the retention of aerosol deposits for 1-micron particles. Observations by Martin (1965) of the behavior of fallout from a nuclear cratering explosion suggest that particles smaller than 5 microns were retained by sagebrush and shadscale to the extent of 8 to 23%. It is obvious, however, that the form of vegetation determines to a considerable degree the extent to which it retains small particles.

In light of the large variations in initial retention of deposited material by vegetation, by both wet and dry processes, it is probably adequate to utilize a single value for vapors and aerosols. In the following calculations a universal retention factor of 50% is assumed. As an average over very large areas with various vegetation types and densities, and accompanying seasonal variations, this value is probably conservative. However, there are local conditions under which 50% is clearly too low. If one takes 0.25 kg/m^2 as a reasonable average for major forage crop yields (Koranda, 1965) and utilizes the middle value of ν, the uptake coefficient derived experimentally by Chamberlain (1970), the empirical formula for initial retention gives

$$p = 1 - \exp(-2.8 \times 0.25)$$

$$p = 0.50.$$

It is felt, therefore, that a retention factor of 50% represents a good compromise.

E3.1.2 Removal Rate of Deposited Material from Vegetation

The removal of deposited radioactive material from foliage has been studied quite extensively. Some of the mechanisms responsible for removal of the material are rainfall, wind, plant death and subsequent loss of leaves, sloughing of the leaf surface, and dilution of activity by plant growth. Conversely, retention may be prolonged by absorption into the plant. When allowance is made for radioactive decay, it has been found that the contamination over a period of 6 to 8 weeks, expressed either as the content in foliage per unit area or per gram dry matter, could be approximately described by an exponential relationship of the form $f = \exp(-kt)$, where t is the number of days since deposition and f is the fraction of the initially retained activity. Russell (1966b) suggested that a half-period of 14 days would be appropriate for most nuclides for the first 1 or 2 months following deposition. This half-life, which is commonly known as the "weathering half-life," is determined from the expression

$$T_{eff} = \frac{T_w T_r}{T_w + T_r}, \qquad \text{(VI E-18)}$$

where T_{eff} is the effective residence time on herbage, T_r is the radioactive half-life of the particular radionuclide, and T_w is the weathering half-life. If T_{eff} is based on contamination per unit area of ground surface, it and T_w are independent of plant growth. The value for T_w suggested by Thompson (1965), after reviewing available evidence, was 13 days. In addition, he showed that this empirical expression held for a number of nuclides, including strontium-90, zirconium-95, iodine-131, and cesium-137. Chamberlain (1970) derived an average value of 14 days for the weathering half-life on pastures during the growing season. However, Chamberlain warns that

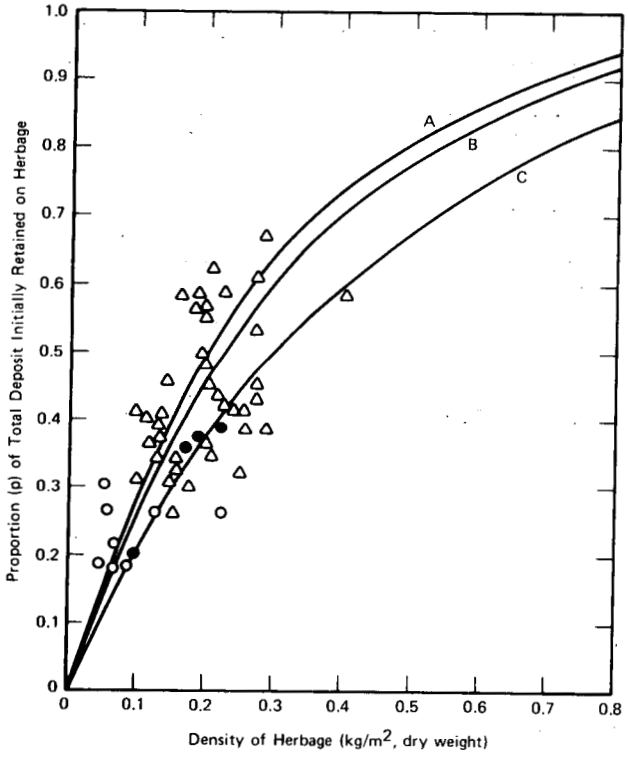


FIGURE VI E-6a Deposition of 30-micron spores (triangles) and spray droplets (open and solid circles) on grassland. Curve A, $\nu = 3.33\ m^2/kg$; curve B, $\nu = 3.08\ m^2/kg$; curve C, $\nu = 2.30\ m^2/kg$. From Chamberlain (1970). Reprinted by permission of Pergamon Press.

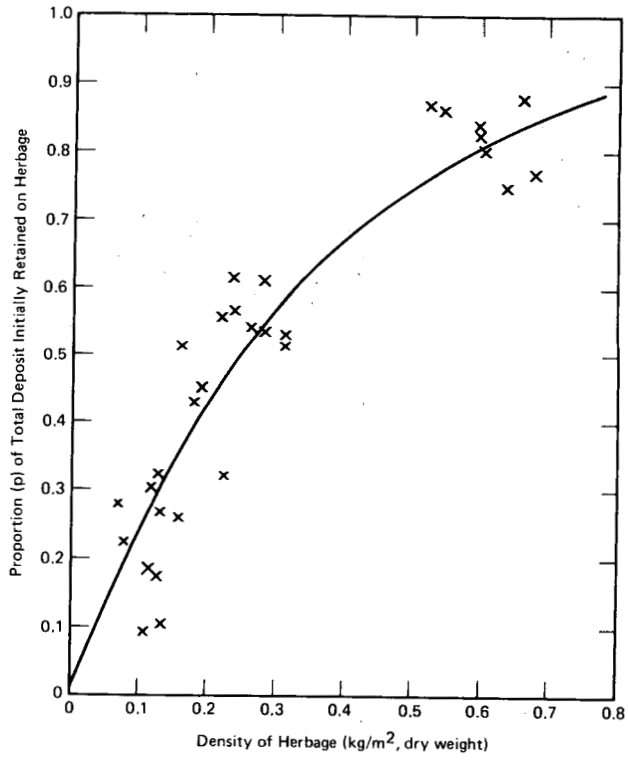


FIGURE VI E-6b Deposition of radioiodine vapor on grassland. The data points represent values measured in field experiments; the curve shows values calculated with $\nu = 2.78\ m^2/kg$. From Chamberlain (1970). Reprinted by permission of Pergamon Press.

the weathering half-life may be longer under poor growing conditions (e.g., winter). In fact,there is some doubt as to the applicability of a weathering half-life beyond the first week or so even after deposition of material in aerosol form. Under arid conditions Martin (1964) found that the weathering half-life was about 30 days for nonvolatile particulates. Recent studies by Krieger and Burmann (1969) have shown that there are two components governing the loss of strontium-85 and cesium-134 from ungrazed pastures. For the first component, the weathering half-life was 12 to 15 days, and for the second it was 25 to more than 50 days. The results of these experiments are shown in Figs. VI E-7a and b. The increasing half-life of retention is clearly evident. In fact, in some cases it appears to approach the radioactive half-life of the nuclide. Investigations (Witherspoon and Taylor, 1970) on the retention of large particles (44 to 88 microns) by agricultural plants indicated that the weathering loss was quite rapid initially. The average half-life on several different plant types was 2.8 days during the first 1.5 days after deposition and increased to 7.6 days for the next 2 weeks. After 1 month, the half-life was roughly 40 days, except for particles deposited on sorghum, for which it was 273 days. It was found that rain was most effective in removing deposited material at early times but had little effect after the third week.

The results of Miller (1966), obtained from the study of volcanic particle (10 to 200 microns) retention, indicate that a proportion (2 to 10% of the initial deposit on most grasses and vegetables) is not removable by weathering. For example, as has been known for some time, the inflorescence of grain crops apparently serves as an excellent trapping device for deposited material (Russell, 1966c). Much of the data illustrated in Figs. VI E-7a and b suggest that a fraction of the initially retained material is not removed by weathering. This fraction is roughly 15%.

The general conclusion to be drawn from the above is that the use of a simple exponential to describe the time-dependent retention of deposited radioactive material, both as vapor or small particles, is only adequate for a few weeks after deposition. For longer periods it is necessary to use a variable weathering half-life and to assume that a significant fraction of the deposited material is not affected by weathering effects.

For the purpose of the calculation model to be utilized for assessing the transport of the deposited radionuclides to the human diet, a time-dependent retention factor

$$f = \exp(-0.693t/14) \qquad \text{(VI E-19)}$$

is assumed. The reason for using this simple relation, rather than one with multiple weathering half-lives, is that the total intake by man of deposited material is calculated by an integration of the time-dependent equation over all time. This integration introduces conservatism into the end result to such an extent that it is not necessary to include a more refined weathering relationship. However, in the event that the contamination level is found to be unacceptably high, the time-dependent retention factor

$$f = 0.85 \exp(-0.693t/14) + 0.15 \qquad \text{(VI E-20)}$$

is used to calculate the time at which the cows can be returned to the pasture or the crops may be utilized.

E3.1.3 Vegetation to Man Transport

The accurate prediction of the total content of radioactive material in a mixed diet is extremely difficult. The problem of predicting for a major release from a nuclear reactor is complicated further because only a limited area would be contaminated. Many foodstuffs consumed by an individual would have been produced before the contaminating event and/or in areas not affected by the release. Therefore, it will be necessary to make a number of conservative assumptions.

Burton et al. (1966) suggested that, if deposition were to occur during the grazing season and milk were a major component of the diet, it would be reasonable to assume that the total intake of radioactive iodine, strontium, and cesium can be judged from the levels in milk. This estimate can be made through correlations established from radioactivity in diets resulting from nuclear weapons testing.

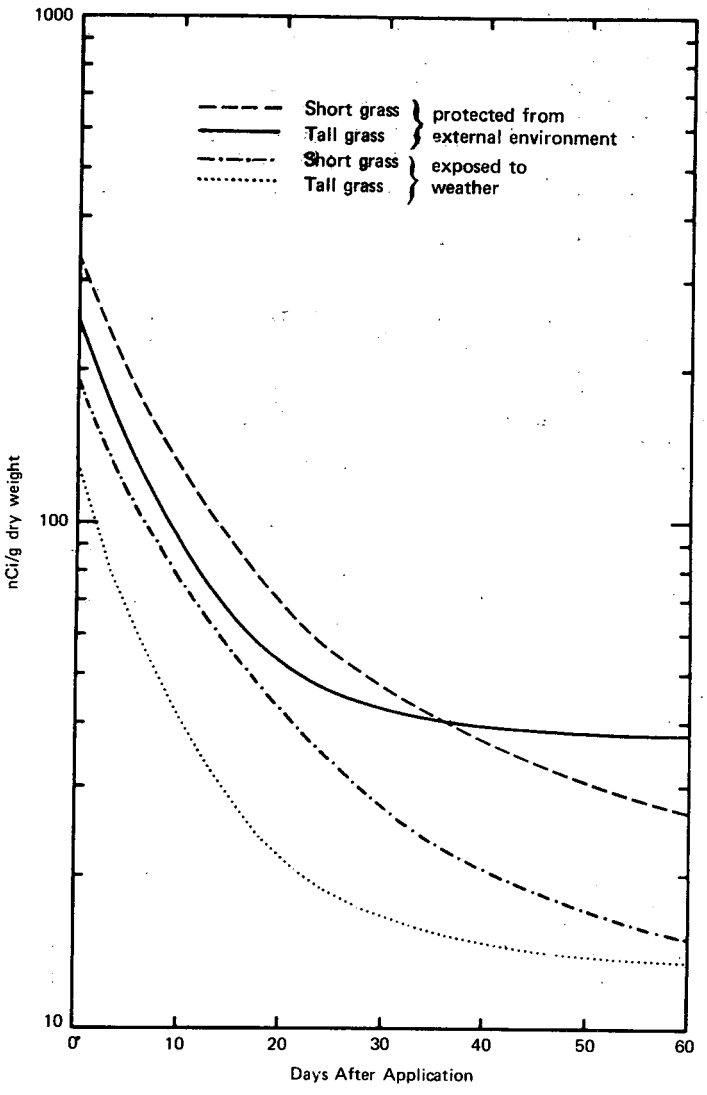


FIGURE VI E-7a Effective half-life of strontium-85 on simulated pasture grass. From Krieger and Burmann (1969).

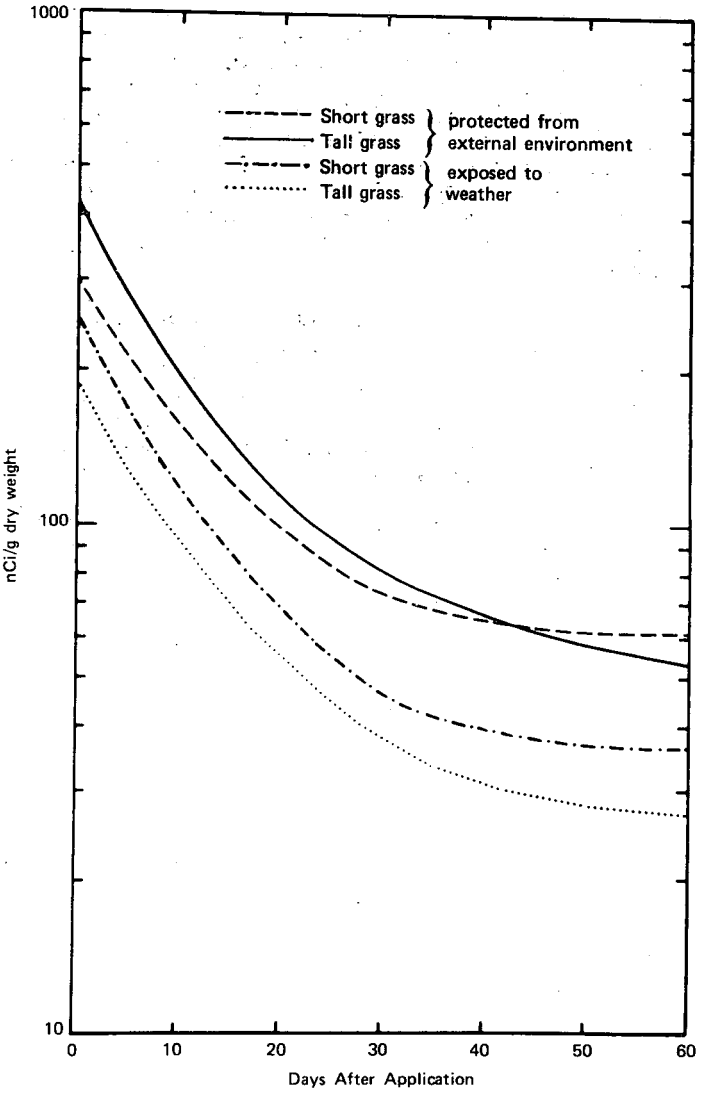


FIGURE VI E-7b Effective half-life of cesium-134 on simulated pasture grass. From Krieger and Burmann (1969).

The dairy cow at pasture occupies a unique position in that its grazing habits endow it with the properties of a rather efficient carpet sweeper. The dairy cow may ingest radioactive material deposited over many square meters of surface and transfer it rapidly to a product that is consumed soon after collection. In addition, measurements made on representative U.S. diets have shown that milk and other dairy products provide a major contribution of cesium-137 and strontium-90 to man's diet from fallout (Gustafson and Miller, 1969; Hardy and Rivera, 1969; Langham and Anderson, 1959). The half-life of I-131 is too short for contamination of vegetation ingested directly by man to be a major route of entry into the food chain because of the interval that occurs between harvesting and consumption.

Additional arguments for the use of milk as the mechanism for calculating radionuclide transport to man are the following:

1. Milk is frequently consumed close to the site of production, and the probability of its dilution with uncontaminated sources is reduced by the relatively short delay between production and consumption.

2. Milk may be the major food source of young children.

3. Direct contamination of grain crops (strontium-90) is only important during a relatively short period when grain is maturing.

This report considers the nuclides of iodine, strontium, and cesium as being important in the ingestion route. Therefore, the following sections describe models for the transport of several radionuclides of these elements from pasture grass to man and through other food chains.

E3.1.3.1 Iodine

The metabolism of iodine in milk-producing animals has been widely and extensively studied (Bustad, 1964; Lengemann and Comar, 1964; Garner and Russell, 1966). A number of working models have been proposed for the prediction of iodine concentrations in milk as a function of time after a single contaminating event (Lengemann, 1966; Tamplin, 1965; Peterson and Smith, 1970). The readily identifiable factors that influence the amount of radioiodine consumed at a given time by an individual can be expressed by the equation

$$I(t + d) = C \cdot R \cdot L \cdot A \cdot S \cdot V, \qquad \text{(VI E-21)}$$

where t is the days from start of ingestion of radioiodine by the cow, d is the days from milk production to consumption (assumed to be 3 days), I(t + d) is the amount of radioiodine consumed by an individual on day t + d after start of ingestion of radioiodine by cow, C is the first days' intake of radioiodine by an average cow, R is a factor to account for radioactive decay occurring between deposition on pasture and t, L is a factor to account for loss of radioiodine from pasture grass due to "weathering," A is the fraction of radioiodine ingestion by the cow that is secreted into a particular milk sample, S is a factor to account for decay of radioiodine during the time from production to consumption, and V is the volume of milk consumed daily by an average individual of the population.

To calculate C, the first days' intake of radioiodine by an average cow, the data of Koranda (1965) were used. Koranda defined a "utilized area factor" (UAF) as the actual area producing the forage a cow will consume in 1 day. He found that a UAF of 45 m^2/day was a reasonable median value for dairy cows grazing on continuously used pastures and on rotational or strip pastures. The range for UAF was 24 to 84 m^2/day, where the lower value reflects early summer conditions and the higher value late summer conditions. If the value of 0.25 kg/m^2 is used for the density of major forage yields for grazing cows and it is assumed that the average cow consumes 11.8 kg/day (Koranda, 1965), UAF is calculated to be

$$UAF = \frac{11.8 \text{ kg/day}}{0.25 \text{ kg/m}^2}$$

$$= 47.2 \text{ m}^2\text{/day}.$$

Therefore, using a UAF of 45 m^2/day is compatible with the assumed forage density of 0.25 kg/m^2. For a reference deposition value of 1 Ci/m^2 and utilizing the initial retention factor of 0.5, the value of C is

$$C = (45\ m^2)(1\ Ci/m^2)(0.5)$$

$$= 22.5\ Ci.$$

The factor R is

$$R = \exp(-0.086t) \quad \text{for iodine-131}$$

and

$$R = \exp(-0.792t) \quad \text{for iodine-133.}$$

Since a weathering half-life of 14 days for iodine has been assumed, the factor L is

$$L = \exp(-0.0495t).$$

The fraction of daily intake of radioiodine secreted per liter of milk has been determined experimentally by Lengemann (1966). The equation that fitted the data points was determined to be

$$A = 0.0091 \exp(0.021t) \left[1-\exp(-0.292t)\right] \text{ liter}^{-1}.$$

The experimental data and the above equation are shown in Fig. VI E-8, reproduced from Lengemann (1966). The factors that influence the concentration of radioiodine in the milk (e.g., milk yield, breed, season, stage of lactation, and stable iodine in the diet) have been discussed by Tamplin (1965). It is assumed that the deposited radioiodine is 100% biologically available.

To account for radioactive decay of the iodine in the milk during the period from production to consumption the factor S is included. Assuming this delay to be, on the average, 3 days, one obtains

$$S = \exp\left[3(-0.086)\right] = 0.772 \quad \text{for iodine-131}$$

and

$$S = \exp\left[3(-0.792)\right] = 0.093 \quad \text{for iodine-133}$$

The final parameter in the equation for the consumption of radioiodine is the volume of milk consumed daily by an average individual. In the past, the practice has been to assume a milk consumption rate of 1 liter per person per day for the explicit purpose of estimating the intake of radionuclides from milk. However, it is generally felt that this value is too conservative. Analyses of milk consumption patterns have shown that in the United States age, sex, geographical locale, and season are important parameters. The Bureau of the Census (1963) found that the average per capita milk consumption in July 1962 was 0.297 liter. Thompson (1966) points out that, on analysis of available data, there is no group having an average daily consumption level over 0.6 liter. Table VI E-4 shows the average monthly fluid milk consumption in 1964 for the cities from the Federal Milk Marketing Order Network (these cities are located in areas covered by the Public Health Service Pasteurized Milk Network). From these figures the average daily consumption is 0.3 liter. The English, in the derivation of limits of contamination in the vicinity of nuclear energy establishments, use a daily milk consumption rate of 0.7 liter for children and 0.5 liter for adults (Bryant, 1969). A very detailed analysis of the daily volume of milk products consumed by the average U.S. infant month by month from birth through the second year was performed by Durbin (1970), who found that the milk intake increases rapidly from about 0.35 liter per day at birth to a maximum of about 0.84 liter per day at the end of the second month. This intake level is maintained through the sixth month before a decline begins. By the tenth month, milk consumption has fallen to 0.67 liter per day and by 2 years, to 0.53 liter per day. These consumption rates are shown in the data of Fig. VI E-9. It should be noted that there are four sources of milk in the infant's diet: whole cow's milk, dried or evaporated milk, prepared formulas, and human milk. Only fresh milk of animal origin would contribute radioiodine to the infant diet. As shown in Fig. VI E-10, infants more than 6 months old consume mainly this kind of milk, and their daily milk intake from 6 months to 2 years averages about 0.7 liter.

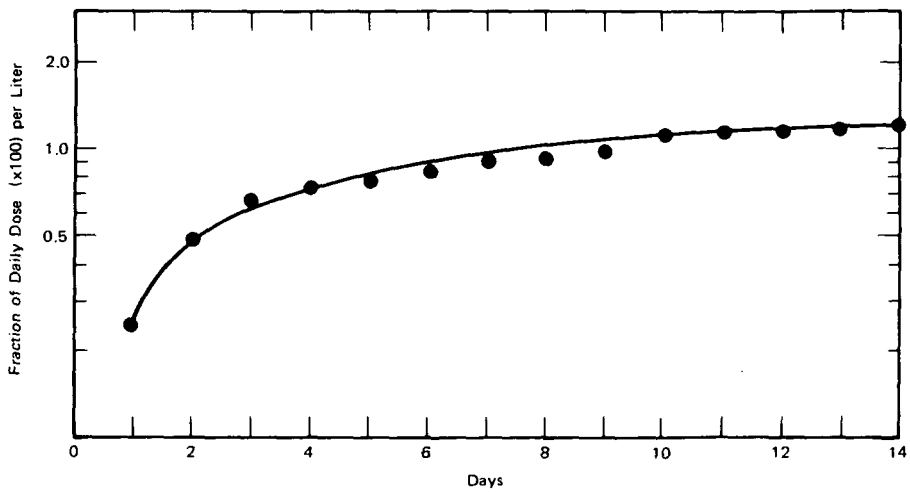


FIGURE VI E-8 Radioiodine concentration in milk during daily dosing experiments with 15 cows. The points are the actual data, and the curve represents the value calculated by the equation derived from these points. From Lengemann (1966).

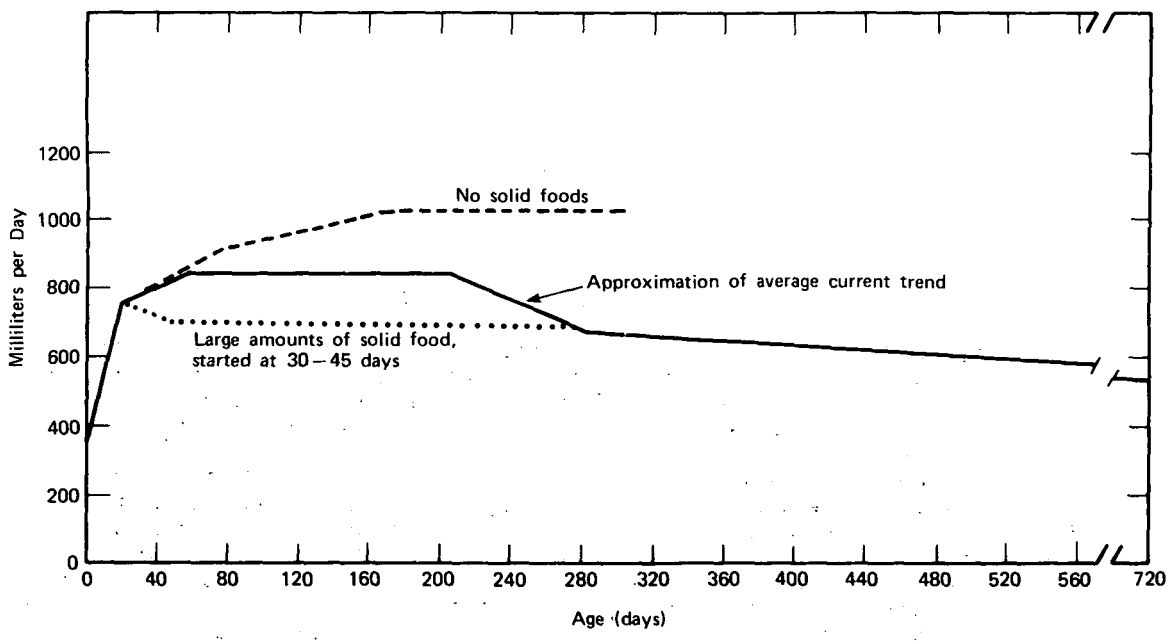


FIGURE VI E-9 Daily volume of milk drunk by infants given human milk, whole cow's milk, evaporated milk, or low mineral prepared formulas. Data from Durbin (1970).

TABLE VI E-4 MONTHLY FULID MILK CONSUMPTION BY GEOGRAPHIC REGIONS OF THE UNITED STATES(a)

Month	Milk Consumption per Capita per month				
	North-east	North Central	South	West	Average
January	11.1	10.0	8.5	8.5	9.4
February	11.0	10.0	8.5	8.5	9.4
March	10.8	9.6	8.2	8.1	9.1
April	10.9	9.7	8.3	8.3	9.2
May	10.6	9.1	7.9	7.9	8.7
June	10.5	8.7	7.6	7.5	8.4
July	10.2	8.6	7.6	7.3	8.3
August	10.2	8.7	7.6	7.4	8.3
September	11.0	9.8	8.7	8.3	9.3
October	11.3	10.2	9.0	8.6	9.7
November	10.8	9.6	8.5	8.1	9.1
December	10.8	9.5	8.2	8.0	9.0
Average	10.8	9.4	8.2	8.0	9.0

As the most sensitive receptor (in terms of total thyroid dose per unit intake) for iodine ingestion is the young child, it is important to utilize a daily milk consumption rate that reflects this. Hence V = 0.7 liter per day is the value used here. This is adequate for the small child but conservative for the average of the population by a factor of roughly 2.

Putting all of the components of Equation (VI E-21) together, we have

$$I(t) = 0.111\left[\exp(-0.115t) - \exp(-0.407t)\right] \quad \text{for iodine-131}$$

and

$$I(t) = 0.0133\left[\exp(-0.821t) - \exp(-1.11t)\right] \quad \text{for iodine-133,}$$

where I(t), in curies per day, is the radioiodine intake as a function of time for an initial deposition level of 1 curie per square meter. Integration of these equations over all time gives the total amount of radioiodine that would be ingested by an individual, assuming the cow were to continuously graze on the contaminated pasture. Therefore

$$CF = \int_0^\infty I(t)\, dt = 0.692 \text{ Ci} \quad \text{for iodine-131}$$

$$= 0.0042 \text{ Ci} \quad \text{for iodine-133.}$$

It should be recognized that the above integration gives a conservative final result because it implies that the cow never returns to graze on a previously grazed area (i.e., new ungrazed areas are always available for grazing). This, of course, is not true, as a cow returns many times in a season to the areas previously grazed.

It is useful to relate the calculated values of total radioiodine ingestion to available experimental evidence from nuclear weapons fallout and measurements made after the Windscale accident. There is, however, a recognized difficulty in converting the estimates made in these studies to a common basis and relating them to estimates, presented here Nevertheless, as summarized in the paper by Peterson and Smith (1970), the range of values obtained from experimental measurements on the total iodine-131 secretion into milk as a fraction of the ingestion by the cow on the first day is found to be 0.035 to 0.178 Ci-day liter^{-1} Ci^{-1}. The average value is on the order of 0.055 Ci-day liter^{-1} Ci^{-1}. Utilizing the value of 22.5 curies for the first day's ingestion by the cow (based on an initial deposition level of 1 curie per square meter), a per capita consumption rate of 0.7 liter per day, and accounting for an average 3-day delay between milk production and consumption, the total quantity

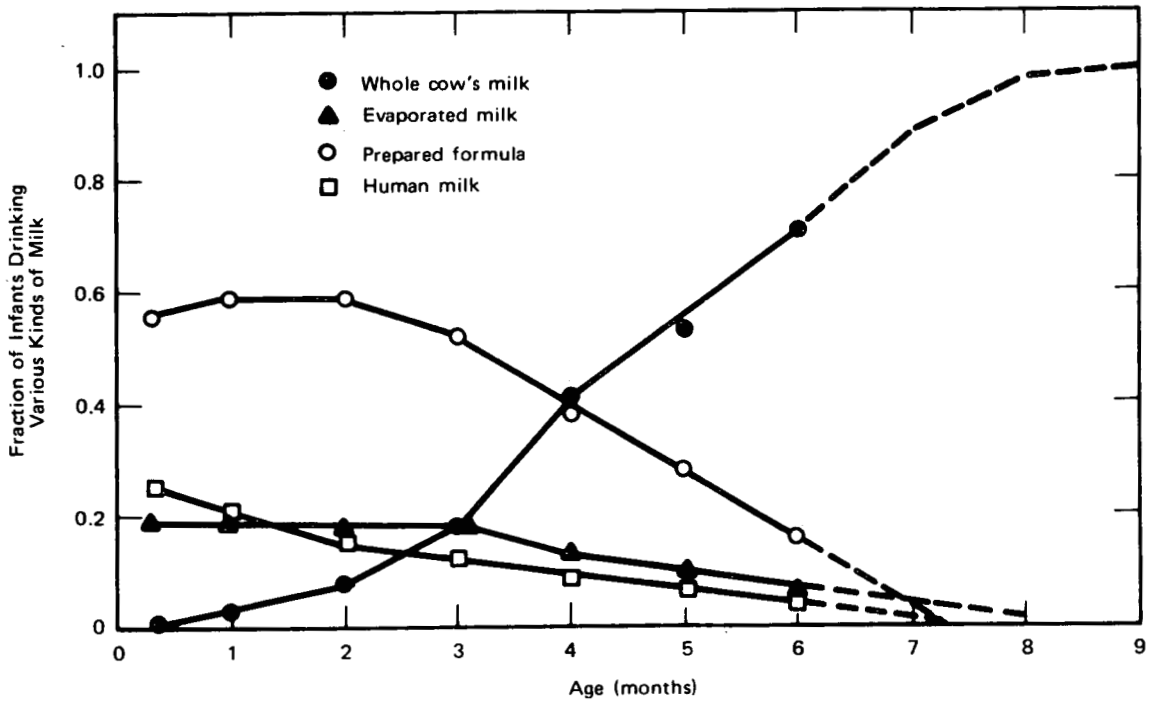


FIGURE VI E-10 Fraction of total number of infants fed various types of milk from birth through 10 months of age during calendar year 1965. From Durbin (1970).

radioiodine ingested is found to be 0.42 to 2.16 curies, with the average being 0.67 curie.

Bruce (1973) reports a value of 1.4 curie-days per liter as a reasonable estimate, based on available evidence, of the total contamination of milk after a single deposition of iodine-131 with an initial surface deposition level of 1 curie per square meter. With a per capita consumption rate of 0.7 liter per day and an average 3-day delay, the total quantity of iodine-131 ingested by an individual would be 0.76 curie.

The above results agree fairly well with the results of the models utilized in this study.

E3.1.3.2 Strontium

The procedure for calculating radiostrontium intake by man via milk is similar to that used above for radioiodine. Data on the transfer of radiostrontium from a cow's diet to milk are available from a number of experiments (Garner, 1960; Comar, 1966; Comar and Lengemann, 1966).

Within the context of the formulation used for radioiodine, the pertinent factors are as follows:

C = 22.5 curies for a reference deposition level of 1 curie per square meter,

$R = \exp(-0.0137t)$ for strontium-89,

$= \exp(-6.6 \times 10^{-5}t)$ for strontium-90,

$L = \exp(-0.0495t)$,

$A = 0.0013 \exp(0.017t)\left[1-\exp(-0.45t)\right]$ liter^{-1} (determined by Comar and Lengemann, 1966, by fitting to experimental data),

S = a factor that accounts for radioactive decay during the period from milk production to consumption and is essentially 1.0 because of the relatively long half-lives of the two strontium isotopes, strontium-89 and strontium-90,

V = 0.7 liter per day.

Combining these factors into a single equation yields

$$I(t) = 0.0205\left[\exp(-0.0462t) - \exp(-0.496t)\right] \quad \text{for strontium-89}$$

and

$$I(t) = 0.0205\left[\exp(-0.0325t) - \exp(-0.483t)\right] \quad \text{for strontium-90.}$$

Where $I(t)$, in curies per day, is the radiostrontium intake as a function of time for an initial deposition level of 1 curie per square meter. To find the total intake of radiostrontium via milk it is necessary to integrate these equations over all time. The assumption implied in the integration is the same as that mentioned for radioiodine. Integration of the above equations gives

$$CF = \int_0^\infty I(t)\,dt = 0.402 \text{ Ci} \quad \text{for strontium-89 via milk}$$

$$= 0.588 \text{ Ci} \quad \text{for strontium-90 via milk.}$$

These calculated results are in good agreement with those of Bruce (1973). His results, based on available experimental evidence, are as follows:

Isotope	Curie-days / liter	Curies
Strontium-89	0.4	0.28
Strontium-90	0.5	0.35

assuming an initial deposition level of 1 curie per square meter and a milk consumption rate of 0.7 liter per day.

As an additional check on the results calculated here it is possible to use the strontium-90 sampling data of Bennett (1972). The quarterly samplings of representative food items in New York City and San Francisco have been conducted by the Health and Safety Laboratory since 1960. The results of strontium-90 analyses, together with estimates of the average consumption of these food items, yield estimates of the total dietary intake of strontium-90 for residents of the two cities.

The year of highest annual deposition of strontium-90 was 1963: 0.024 microcurie per square meter. The estimated daily intake of strontium-90 in New York City was 29.6 picocuries, of which 56% was from dairy products. Therefore, the ingestion from dairy products was 6050 picocuries per year. Bennett (1972) estimates that about 22% of the strontium-90 in milk in 1963 was attributable to plant uptake from the accumulated deposit in the soil. Therefore the intake of strontium-90 in milk as a result of direct deposition was 4720 picocuries per year for a deposition level of 0.024 microcurie per square meter. For a reference deposition of 1 curie per square meter, this results in a strontium-90 intake of 0.20 curie, which agrees fairly well with the results presented here.

Because of the long radioactive half-lives of strontium-90 and strontium-89, it is important to consider the radiostrontium contamination of other foods, such as grain products and vegetables. A reasonable estimate can be derived from the data of Bennett (1972), who found that, during a period of high deposition, slightly less than 50% of the total strontium-90 intake was from sources other than milk. Therefore as one estimate it may be assumed that the intake via contaminated vegetation ingested directly by man would be the same as the estimates presented here for milk.

An alternative approach is to use a simple model proposed by Fish et al., (1972). The time-dependent concentration of deposited radioactive material on crops can be expressed as

$$C(t) = C_0 \exp(-0.693t/T_{eff}), \qquad \text{(VI E-22)}$$

where

C_0 = initial concentration of a particular radioactive nuclide on the vegetation (in curies per kilogram),

$T_{eff} = \frac{14\ T_r}{14 + T_r}$ = effective removal half-life (in days),

and

T_r = radioactive half-life of the radionuclide (in days).

The concentration of the radionuclide on the vegetation is related to the total surface deposition by the crop yield,

$$C_0 = 0.5(SD/CY), \qquad \text{(VI E-23)}$$

where

SD is the total initial surface deposition level (in curies per square meter), CY is the crop yield (in kilograms per square meter), and 0.5 accounts for a 50% initial retention of deposited material.

If the average per capita consumption of vegetation is K (kg/day), the time-dependent intake of the radionuclide is

$$I(t) = KC(t) = K(0.5)(SD/CY) \exp(-0.693t/T_{eff}). \qquad \text{(VI E-24)}$$

The total intake of the radionuclide, normalized to a unit surface deposition value, is found by integrating the above equation over all time:

$$CF = (SD)^{-1} \int_0^{\infty} I(t)\, dt = (0.5K/CY)(T_{eff}/0.693). \qquad \text{(VI E-25)}$$

The assumptions inherent in the above equation are the following:

1. The delay between crop harvesting and consumption has no effect on the quantity of radioactive material transported to man.

2. Consumption of the contaminated crops occurs continuously by the same individual from the time of deposition.

3. There is no loss of deposited material on harvesting or in food preparation.

The U.S. Department of Agriculture (1972) statistics on the average per capita consumption of major food commodities show that in recent years fresh-vegetable consumption is 0.12 kg/day. Crop yields are quite variable, depending on the crop type. Large leafy vegetables (lettuce, cabbage, etc.), which generally are not cooked before consumption, have yields of roughly 2.4 kg/m^2. Therefore, Equation (VI E-25) becomes

$$CF = 0.0361 T_{eff}. \qquad \text{(VI E-25)}$$

Computed values of CF for strontium intake by man via vegetation are 0.397 and 0.505 for strontium-89 and strontium-90, respectively. These values appear to be in excellent agreement with those estimated from fallout data (Bennett, 1972), as discussed above.

E3.1.3.3 Cesium

Cesium is the one element of major importance among all the ingested radionuclides. Its metabolism and behavior in food chains have been reviewed by Garner (1960) and Frederiksson et al. (1966). Because of its behavior in soils, cesium reaches man almost entirely via direct deposition despite the long radioactive half-life of cesium-134 and cesium-137.

The formula used to calculate the amount of radiocesium ingested by man via contaminated milk is the same as that used for iodine and strontium. Within the context of that formulation, the pertinent factors are as follows:

C = 22.5 curies for a reference deposition level of 1 curie per square meter,

R = 1.0 for cesium-134 and cesium-137 because of their long radioactive half-life,

= exp (-0.0537t) for cesium-136,

L = exp (-0.0495t),

A = (0.0138 + 0.000073t) [1 - exp (-0.3t)] liter^{-1} (determined by Comar and Lengemann, 1966, by fitting to experimental data),

S $\simeq$ 1.0 for cesium-134 and cesium-137,

S = exp [3(-0.0537)] = 0.851, cesium-136

V = 0.7 liter per day.

Combining these factors into a single equation yields

$$I(t) = (0.217 + 0.00115t)\,[\exp(-0.0495t) - \exp(-0.3495t)]$$

for cesium-134 and cesium-137,

$$I(t) = (0.185 + 0.00098t)\,[\exp(-0.103t) - \exp(-0.403t)]$$

for cesium-136. Here I(t), in curies per day, is the radiocesium intake as a function of time for an initial deposition level of 1 curie per square meter. To find the total intake of radiocesium via milk, it is necessary to integrate these equations over all time. As mentioned previously, this gives a conservative final result. Integration of

the above equations give

$$CF = \int_0^\infty I(t)dt = 4.22 \text{ Ci} \quad \text{for cesium-134 and cesium-137 via milk}$$

$$= 1.42 \text{ Ci} \quad \text{for cesium-136 via milk.}$$

The results of Bruce (1973) give
10(Ci-days/liter) (0.7 liter/day) = 7.0 Ci for cesium-137 via milk.

This value is larger than that calculated in this report, but Bruce's (1973) result significantly overestimates the experimental observations. It appears that the results calculated here fit the experimental data much better.

A parametric analysis of cesium-137 intake from contaminated food (Ng et al., 1973), resulting from nuclear weapon's fallout, yields cesium-137 intake via milk of 3.0 Curies for a deposition level of 1 curie per square meter. This value is fairly close to the result calculated in this report.

In addition to milk, grain products and meat have been major contributions of cesium-137 to the diet. In the years 1961-1968, milk products contributed from 25 to 40% of the cesium-137 in the U.S. diet (Gustafson, 1969). In 1963, a year of particularly high cesium-137 deposition, milk contributed a proportion on the order of 33% of the total cesium-137 in the diets of Chicago residents (Gustafson et al., 1965). Therefore, a reasonable estimate would be that twice as much cesium is ingested by man via meat, grain products, etc., as by the grass-cow-milk pathway. This gives CF values of 8.44, 2.84, and 8.44 curies for the intake of cesium-134, cesium-136, and cesium-137, respectively, via the "other pathway." An alternative approach is to use the result of the simple model developed here for radiostrontium contamination of vegetables, that is, Equation (VI E-25)

$$CF = 0.0361 T_{eff}, \qquad \text{(VI E-26)}$$

which gives CF = 0.496, 0.242, and 0.505 curies for cesium-134, cesium-136, and cesium-137, respectively, for vegetables consumed directly by man.

The data from Gustafson et al. (1965) show that 4.1% of the total cesium-137 in the diet of Chicago residents was from contaminated vegetables. Therefore, by this calculation, the total intake of cesium-137 is (0.505 Ci)/(0.041) = 12.32 curies, and, correcting for the contribution solely from milk, one obtains (12.32 Ci)(2/3) = 8.21 curies. This is in very good agreement with the value calculated above.

E3.1.4 Summary and Ingestion Dose Model

The following is a reiteration of the results from the preceding sections:

1. Initial retention of radioactive material on vegetation: 50% of deposited material is intercepted and initially retained by vegetation of all types.

2. Removal rate of deposited material from vegetation: weathering half-life of 14 days is assumed. Therefore, for the calculation of total intake by man of radioactive material a time-dependent retention factor of $f = \exp(-0.693t/14)$ is used. For calculating time of pasture or crop interdiction the relationship $f = 0.85 \exp(-0.693t/14) + 0.15$ is used.

3. Transport from vegetation to man: Table VI E-5 gives the estimates of the total intake by a critical individual of the various radionuclides as a result of direct deposition.

TABLE V1 E-5 TOTAL INTAKE BY A CRITICAL INDIVIDUAL AS A RESULT OF DIRECT DEPOSITION[a]

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.

4. Model development: For radionuclide i, the relationship between total deposition level and received radiation dose from ingestion of the radionuclides is given by

$$RD^i = (SD^i)(CF^i)(DC^i), \qquad \text{(VI E-27)}$$

where RD^i is the dose commitment (rem) received from ingesting the radionuclide, SD^i is the total deposition level (curies per square meter), CF^i is a concentration factor relating a given total deposition level to the amount of radionuclide ingested by an individual (curies per curie per square meter), and DC^i is a dose-conversion factor relating the total amount of ingested material to the dose received by an organ (rem per cuie ingested).

The CF factors in the list above are derived for the respective critical segment of the general population, for example, iodine ingestion via milk is based on data for a young child. Therefore, the CF factors are appropriate for utilization in calculating criteria for acceptable deposition levels. For the calculation of dose commitments to large population groups these CF factors will introduce some conservatism. By fixing the value of an acceptable dose to a critical organ from ingestion, it is possible to calculate the corresponding acceptable total deposition level. Therefore

$$SD_L^i = \frac{RD_L}{(CF^i)(DC^i)}, \qquad \text{(VI E-28)}$$

where RD_L is the acceptable dose (rem) to the critical organ and SD_L^i is the corresponding deposition level for radionuclide i. The index for the radionuclide has been dropped here since an acceptable dose to an organ is specified only as a total. With more than one radionuclide contributing dose to the organ as a result of deposition on vegetation, it is necessary to account for this fact. If the actual deposition level is SD_{act}^i for the i^{th} radionuclide, it is necessary that the sum of the ratios, actual to acceptable deposition levels, be less than 1.0 if the acceptable dose commitment RD_L is not to be exceeded, that is,

$$R_0 = \sum_i \frac{SD_{act}^i}{SD_L^i} \leq 1.0, \qquad \text{(VI E-29)}$$

where the summation is over all radionuclides that contribute dose to the same organ. If the sum of the ratios exceeds 1.0, the acceptable dose RD_L will be exceeded by the fraction that the sum exceeds 1.0.

When the contamination of the vegetation is such that an acceptable dose will be exceeded, it is necessary to calculate the time over which condemnation should be practiced. For the milk ingestion pathway, the time from accident occurrence until the end of the grazing season is calculated. This time span is used to determine if the direct contamination of the pasture grass has decreased (radioactive decay and weathering) to such an extent that the dose criteria would not be exceeded if the cows were returned to the pasture and the milk produced were consumed. The fractional decrease in the direct contamination of the pasture grass is calculated with

$$f^i = [0.85 \exp(-0.0495t') + 0.15] \exp(-0.693t/T^i_{1/2}) \qquad \text{(VI E-30)}$$

for radionuclide i with a half-life of $T^i_{1/2}$ (days). The time t' is the period from accident occurrence to the end of the grazing season. With this fractional decrease (f^i) and the ratios of the initial deposition level to acceptable deposition level [SD^i_t from Equation (VI E-28)], the sum of the relative contributions of all radionuclides at time t' is calculated:

$$R_{t'} = \sum_i \frac{SD^i_{act}\, f^i}{SD^i_L} \qquad \text{(VI E-31)}$$

If $R_{t'} \geq 1.0$, the cows would not be returned to the pasture until the next grazing season. In the event $R_{t'} < 1.0$, an exponential interpolation is performed to calculate the time period over which the pasture would be denied the cows for grazing. This time is calculated as

$$t_{pasture} = t' \frac{\ln(1.0/R_0)}{\ln(R_{t'}/R_0)} . \qquad \text{(VI E-32)}$$

The dose commitment received by a particular organ from ingestion of milk is calculated by

$$RD_{DC} = \sum_i (CF^i)(DC^i)(SD^i_{act})[\exp(-0.693t_{pasture}/T^i_{\frac{1}{2}})] \times [0.85 \exp(-0.0495\, t_{pasture}) + 0.15] . \qquad \text{(VI E-33)}$$

The dose-conversion factors DC^i relate the total ingested quantity of radionuclide i to a dose commitment RD_{DC} received by a particular organ.

For CF values (via "other" pathways) that pertain primarily to harvested crops, the procedure utilized is essentially the same as in the milk calculation. The one exception is the time available for natural decrease in the direct contamination of the vegetation. Here the time period is from accident occurrence to harvest time. If the contamination level would not be sufficiently reduced by harvest time, a total loss of crops is assumed.

E3.2 INCORPORATION OF RADIOACTIVE MATERIAL FROM SOIL INTO VEGETATION

After deposition of radioactive material on the soil surface and subsequent mixing in the soil, the only important route, under normal circumstances, by which the material can enter food chains is absorption from the soil. In this respect the soil is important as a reservoir of long-lived radionuclides. Factors affecting the uptake of radionuclides by plants from soils under both laboratory and field conditions have been the subject of a number of reviews (Menzel, 1965; Nishita et al., 1965; Russell, 1966a).

It is necessary to have information on the long-term behavior of radionuclides in the soil and their availability to plants. As the radionuclides will be competing with nonradioactive ions, it is important to have information on the effect of soil characteristics on the uptake of the radionuclides by plants. In addition, radionuclides are found not to be evenly distributed throughout vegetation. Therefore, information is needed on the fraction of radionuclides typically found in the edible portion of the vegetation. Unfortunately a complete data base of this type of information has not yet been established. There is, however, sufficient information from which to make some reasonable estimates.

A significant fraction of radionuclides deposited on soils is found to remain within the upper layers of soil and within reach of most plant roots. The behavior of radionuclides in soils is reviewed in section E1.1 of this appendix.

If the radionuclides are to be transferred through food chains, they must either be deposited in a soluble form or be rendered soluble in the soil or in the gastrointestinal tracts of animals. The results of many experiments indicate that there is a rather large reduction in the annual uptake by plants of deposited radionuclides. For example, Squire and Middleton (1966) found that the concentration of cesium-137 in rye grass grown on various soils decreased markedly during the first 3 years after contamination, as shown in Fig. VI E-11a. The same data are shown in Fig. VI E-11b but are expressed as the mean concentration for each year relative to that in the final year. An analysis of the results supports the view that the decrease in absorption was due mainly to the progressive fixation of cesium-137 on the clay minerals. It appears that roughly 3 years after contamination the fixation process is largely complete since from all soils the cesium-137 content of grass decreased thereafter by only about one-third. From all soils the amount of cesium-137 absorbed was much less than that of strontium-90, and the ratio in which the two nuclides were absorbed decreased with time. In the first year, the ratios ranged from 0.04 to 0.2, whereas corresponding values in the fifth year were 0.01 to 0.04. The high mobility of strontium-90 in the soil is expected to play a large part in the reduction in plant uptake. The results in Figure VI E-12 are explained by the fact deep penetration of strontium-90 in the soil caused the mean ratio of stronium-90 to calcium in grass to decrease steadily during the first 5 years of the experiment, the average reduction being 10 to 15% per year (Squire, 1966).

Van der Stricht's (1970, 1971) survey results from Ispra in Northern Italy gave an annual reduction in strontium-90 uptake from the soil of about 13%. Bennett (1972) estimates that the annual reduction in strontium-90 uptake by grass in New York State is 11%.

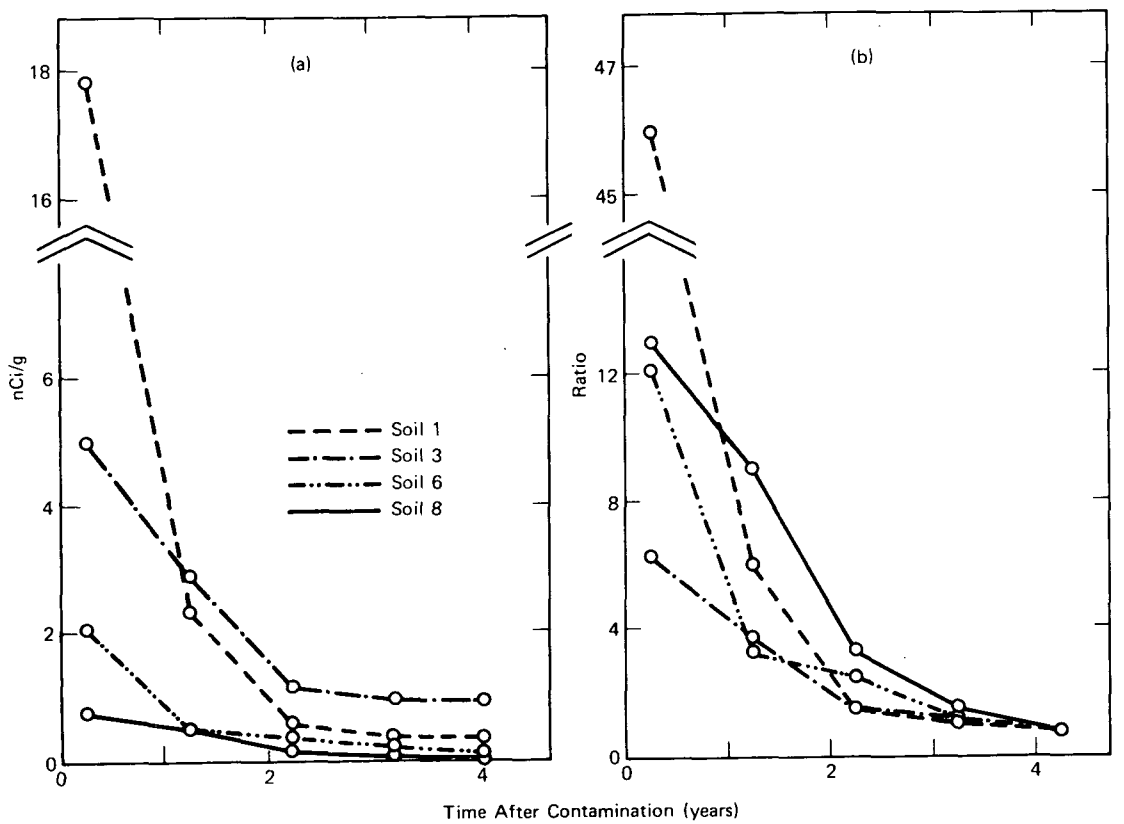
The percentage of strontium-90 removed from soil by different crops (mustard, cheat grass, alfalfa) at different time periods after surface contamination is illustrated in Fig. VI E-13. The low initial point in Fig. VI E-13 is probably due to the fact that little of the strontium-90 had penetrated the soil to a sufficient depth to allow much root contact with the contaminant. Experiments in the United Kingdom have shown that pasture grasses can remove 2 to 5% of recently introduced strontium-90 from the soil in a single summer, (Russell et al., 1970). Beyond this the downward movement of strontium-90 in the soil by only a few centimeters will frequently cause an appreciable reduction in absorption since the roots of pasture plants draw nutrients largely from the upper soil layers.

The relative uptake of many different chemical elements is found to vary widely. An extensive review of available data (Menzel, 1965) gave the values shown in Table VI E-6, which lists relative concentration factors (ppm in dry plant material/ppm in dry soil) derived from experiments in which the elements were applied in water-soluble form and mixed into the surface soil.

TABLE VI E-6 RELATIVE CONCENTRATION OF ELEMENTS IN FIRST-CROP PLANTS COMPARED WITH SOIL[a]

Concentration Factor	Element
10-1000 (strongly concentrated)	K, Rb, N, P, S, Cl, Br, Na, Li
1-100 (slightly concentrated)	Mg, Ca, Sr, B, Se, Te, Mn, Zn, Mo
0.1-10 (not concentrated)	Ba, Ra, Si, F, I, Co, Ni, Cu
0.01-1 (slightly excluded)	Cs, Be, Fe, Ru
<0.01 (strongly excluded)	Sc, Y, Zr, Ta, W, Ce, Pm, Pb, Pu, Sb

[a] Data from Menzel (1965).



Description of Soils

Soil	pH	Exchangeable Cations (mEq/100g) Total	Ca	K	Organic Matter (%)	Clay Content (%)
1. Acid clay	4.6	16.3	3.2	0.20	4.2	19.5
3. Sand	6.6	6.8	5.6	0.14	2.0	3.2
6. Lower greensand	6.0	13.2	8.5	0.28	2.1	11.0
8. Calcareous loam	7.6	26.0	28.5	0.95	5.5	16.8

FIGURE VI E-11 Changes with time in the concentration of cesium-137 in rye grass grown on different soils after contamination of the soil surface: (a) annual means for grass grown on each soil in the absence of fertilizers; (b) mean values expressed relative to those in the final year. From Squire and Middleton (1966). Reprinted by permission from Pergamon Press.

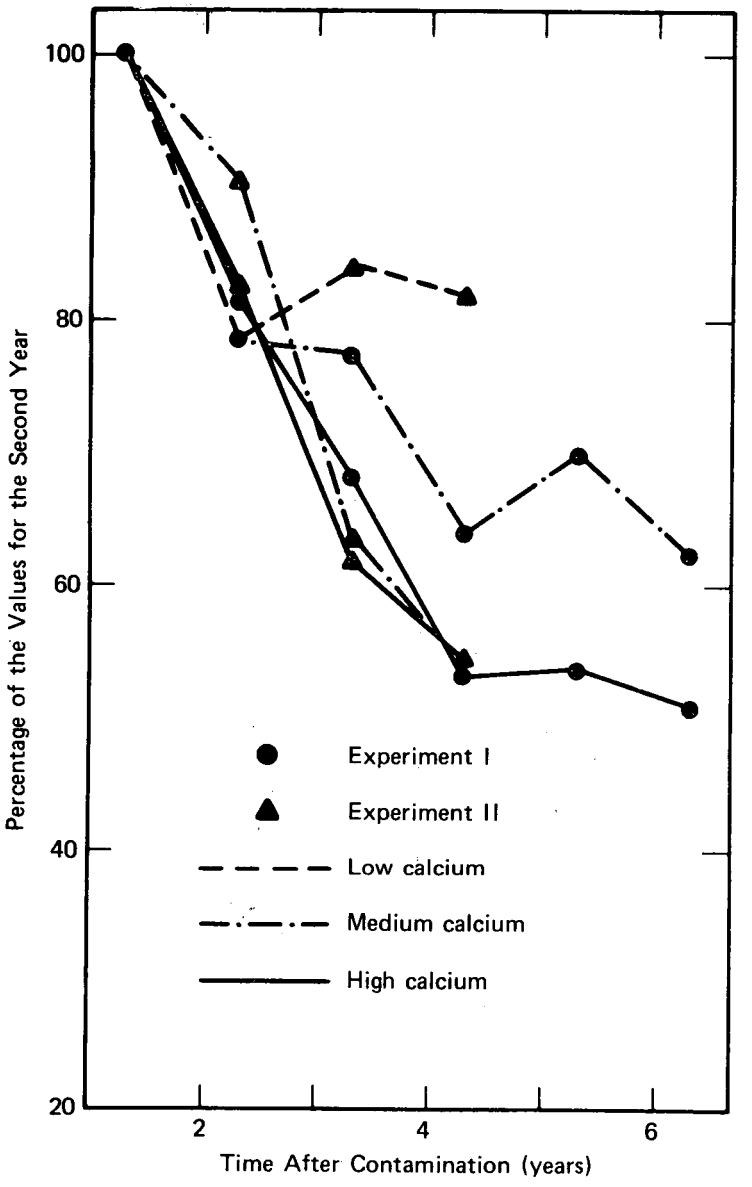


FIGURE VI E-12 Changes with time in the strontium-90/calcium ratio in grass growing on soils that varied in calcium content and were fertilized annually. From Squire (1966). Reprinted by permission from Pergamon Press.

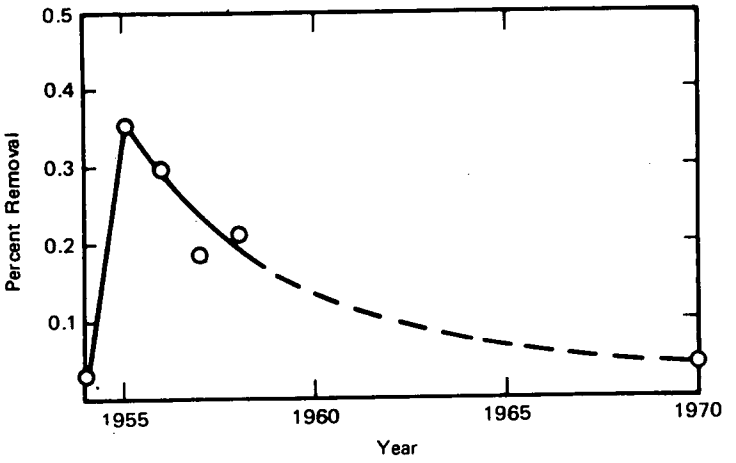


FIGURE VI E-13 Percent of the total strontium-90 applied to the soil removed annually by plant harvests. From Cline and Rickard (1972).

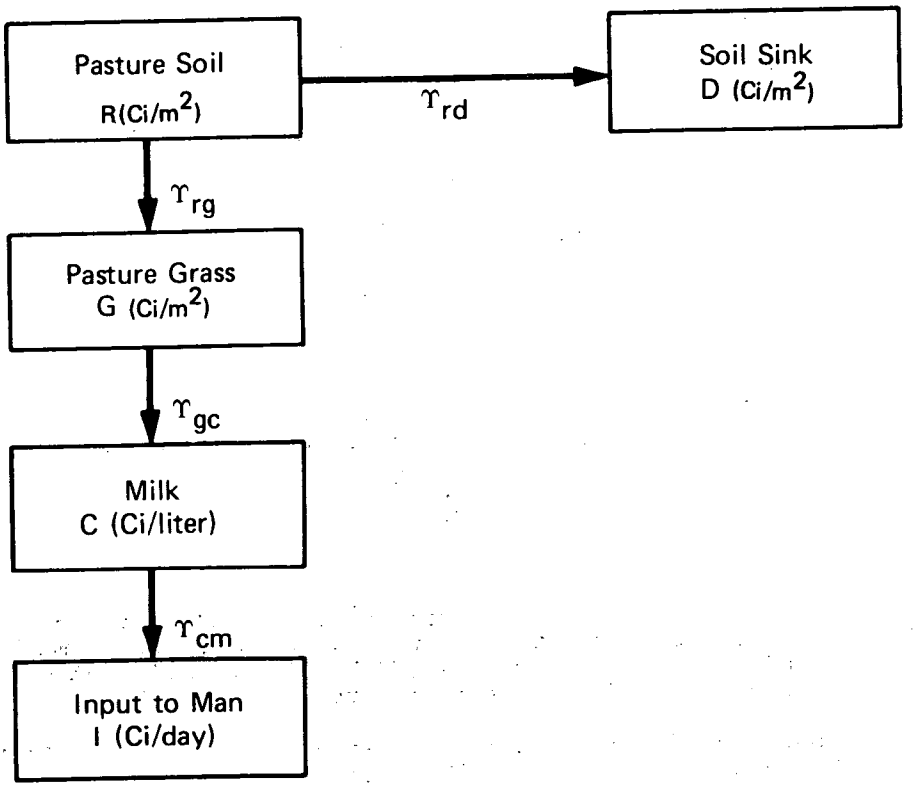


FIGURE VI E-14 Compartment model describing radionuclide movement to man via the plant root-milk pathway.

The variability in the values of Table VI E-6, as well as the overlapping ranges that occur, indicates the influence of differences in the chemical state of the element and soil type.

To account for the behavior of radionuclides in the soil and the transport mechanisms to man, a simplified model, fashioned from the work of Booth et al. (1971), can be utilized. This model is obviously an oversimplification of the actual situation. However, given the available data, a more complex model is probably not justified.

The model used is a single-compartment model shown in Fig. VI E-14. The source of radioactive material contamination is assumed to be the pasture soil. The level of contamination is given as the total quantity of deposited material per square meter, R (in curies per square meter). The loss of availability of the material for uptake is represented by the soil sink compartment with a transfer coefficient of T_{rd}. Uptake of the material by pasture grass is governed by a transfer coefficient T_{rg}. The milk compartment, C (in curies per liter), is the concentration of the material in milk produced by a cow continuously grazing on the contaminated pasture. The input to man is governed by the quantity of milk consumed daily.

The compartment model of Fig. VI E-14 does not include a return loop--that is, the return from pasture grass via cattle excretions to the pasture soil. The inclusion of this feedback loop would complicate the model to the point that an analytical solution would not be possible. The effect is, however, not judged to be of major importance, since the transfer coefficient T_{rd} is sufficiently large to dominate the problem.

The differential equations for the compartment model are the following:

$$dR/dt = -(\lambda_R + T_{rg} + T_{rd})R,$$

$$dG/dt = -\left[\lambda_R + (V/AD)\right] G + T_{rg}R,$$

$$dC/dt = T_{gc}\, G - (\lambda_R + T_{milk})C,$$

and

$$I = T_{cm}C,$$

where $\lambda_R = 0.693/T_{1/2}$ ($T_{1/2}$ = radioactive half-life of the radionuclide in days), V/AD is the loss term for the pasture grass compartment due to grass consumption by a cow, V is the dry weight of grass consumed by a cow each day (11.8 kg/day), A is the pasture area utilized per cow (8500 m^2/cow), D is the dry weight areal grass density (0.25 kg/m^2), T_{milk} is the loss of milk from the cow's udder (2.0 day^{-1}), and T_{cm} is the average daily consumption of milk by an individual (0.7 liter per day).

The transfer coefficient T_{gc} governs the fraction of a total intake of a particular radionuclide by a cow that is transferred to the udder per liter of milk in the udder. The surface area grazed by a cow is given as (V/D). For a unit contamination of this surface area, a fraction f_m/liter is transferred to the milk.

Under equilibrium condition f_m has been determined by Comar (1966) for strontium ($f_m = 0.0008$) and by Fredricksson et.al. (1966) for cesium ($f_m = 0.012$). Therefore,

$$T_{gc} = (V/D)\ f_m = 3.78 \times 10^{-2}\ (m^2/\text{liter-day}) \quad \text{for strontium}$$

$$= 5.66 \times 10^{-1}\ (m^2/\text{liter-day}) \quad \text{for cesium.}$$

Based on the results discussed previously, the uptake per year by pasture grass is assumed to be 5% per year for strontium ($T_{rg} = 1.41 \times 10^{-4}\ day^{-1}$) and 0.23% per year for cesium ($T_{rg} = 6.31 \times 10^{-6}\ day^{-1}$). The loss of availability of the elements, excluding radioactive decay, is taken to be 10% per year for strontium ($T_{rd} = 2.89 \times 10^{-4}\ day^{-1}$) and 61% per year for cesium ($T_{rd} = 2.60 \times 10^{-3}\ day^{-1}$).

Solution of the coupled differential equation yields

$$CF(t) = I(t)/R_0 = (T_{cm}T_{ge}T_{rg})/(\lambda_2 - \lambda_1)\ \{(\lambda_3 - \lambda_1)^{-1}\ [\exp(-\lambda_1 t) - \exp(-\lambda_3 t)] + (\lambda_3 - \lambda_2)^{-1}\ [\exp(-\lambda_2 t) - \exp(-\lambda_3 t)]\}, \qquad \text{(VI E-34)}$$

where R_0 is the initial level of contamination (in curies per square meter) and CF represents the intake rate per unit contamination level, (Ci ingested)/(Ci/m^2),

$$\lambda_1 = \lambda_R + T_{rg} + T_{rd},$$

$$\lambda_2 = \lambda_R + V/AD,$$

$$\lambda_3 = \lambda_R + T_{milk}$$

Integration of the CF(t) over all time gives total intake of a particular radionuclide:

$$CF = \int_0^\infty CF(t)\ dt = \frac{T_{cm}\ T_{ge}\ T_{rg}}{\lambda_1\ \lambda_2\ \lambda_3} \qquad \text{(VI E-35)}$$

Utilizing the above data and Equation (VI E-35), the CF values given in Table VI E-7 can be calculated.

TABLE VI E-7 INGESTION OF RADIONUCLIDES RESULTING FROM ROOT UPTAKE

Radionuclide	CF via Milk (Curies)
Strontium-89	0.00682
Strontium-90	0.669
Cesium-134	0.0547
Cesium-137	0.0835

The calculated result for strontium-90 is in fairly good agreement with the results calculated by Bruce (1973).

The contribution of radionuclides in man's diet from sources other than milk can be included by using the data of Bennett (1972) for strontium. In recent years, with the low deposition level of strontium-90 from nuclear weapons testing, most of the strontium-90 in man's diet is a result of uptake from the soil by vegetation. Bennett's (1972) results show that for the last few years, 1969-1971, roughly 28 to 40% of the estimated ingestion of strontium-90 was from dairy products. Using an average value of 33%, it can be assumed that the ingestion of strontium-90 from sources other than milk will be twice the values given in Table VI E-7.

Gustafson and Miller's (1969) data indicate that, during a period of low deposition of cesium-137 from nuclear weapons fallout, milk contributes roughly 25% of the total cesium-137 in man's diet. Therefore, as an estimate, it can be assumed that the ingestion of cesium-137 from sources other than milk would be three times the values in Table VI E-7.

The total ingestion of strontium and cesium from uptake by vegetation from the soil is given in Table VI E-8.

TABLE VI E-8 TOTAL INGESTION OF STRONTIUM AND CESIUM RESULTING FROM ROOT UPTAKE[a]

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.

These values are utilized in the consequence model to calculate a dose commitment in the following manner. A dose commitment resulting from radionuclide i in the soil is

$$RD^i = (SD^i)(CF^i)(DC^i), \qquad \text{(VI E-36)}$$

where RD^i is the dose commitment (rem), SD^i is the contamination level of the soil per surface area (curies per square meter), CF^i is the concentration factor relating the total ingested quantity of radionuclide i, over all time, per unit contamination level (curies ingested per curie per square meter), and DC^i is the dose-conversion factor for an organ relating dose commitment to total ingested quantity of radionuclide i (rem per curie ingested). For the same organ, the values of RD^i are summed to give a total dose commitment via ingestion:

$$RD_T = \sum_i RD^i. \qquad \text{(VI E-37)}$$

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Appendix F

Early and Continuing Somatic Effects

F1 BONE MARROW

F1.1 INTRODUCTION

Bone marrow damage is the dominant event in the production of the hematologic form of the acute radiation syndrome. The sequence of events has been reviewed in detail in many publications (Wald et al., 1962; Bond et al., 1965; International Atomic Energy Agency, 1971).

When the bone marrow is damaged by external irradiation, the lymphoid tissue is also severely injured, resulting in a further reduction of defenses against infection; moreover, the vascular system is damaged under such circumstances, enhancing the hazard of bleeding. In comparison with external bone marrow irradiation, internal irradiation due to the incorporation of radioactive isotopes with a predilection for the bone or marrow cells (e.g., strontium-90, gold-198, or phosphorus-32) produces less damage to lymphoid tissue and the vascular system.

F1.2 Dose-Response Relationships

Fig. VI F-1 presents three curves showing the relationship between radiation dose and 50% mortality in 60 days. The data points shown on this figure are derived from the literature (Thoma and Wald, 1959; UNSCEAR, 1962; Langham, 1967; Rider and Hasselback, 1968; E. D. Thomas et al., 1971a; NCRP, 1974; Wald, 1975) and a "best estimate" calculated by the Biomedical and Environmental Assessment Group at the Brookhaven National Laboratory.

An important consideration in the preparation of the dose-mortality curves in Fig. VI F-1 was that a dose-response relationship based on the absence of any treatment would be somewhat unrealistic and less than useful in evaluating the health hazards of reactor accidents. For this reason curves were developed for three levels of treatment: minimal (curve A), supportive (curve B), and heroic (curve C).

The term "supportive treatment" indicates such procedures as reverse isolation (measures to protect the patient from pathogenic bacteria and viruses, such as the use of sterile garments and masks by entering personnel, sterilization of all objects in the patient's room, use of portable or permanent laminar-air-flow systems, etc.), large doses of antibiotics, and transfusions of whole-blood packed cells or platelets. This type of treatment can be provided by most large urban and some rural acute medical and surgical hospitals, particularly those with facilities for kidney transplants. The term "minimal treatment" indicates the absence of any of these measures, whereas "heroic treatment" represents extraordinary procedures, such as bone marrow transplantation. The curves developed for these three levels of clinical management obviously are not based on large numbers of cases studied under ideal conditions. Rather, they are the best interpretations that can be drawn from the various clues obtained from the available sources of data. The specific features of each curve are explained below.

F1.2.1 Minimal Treatment

It is considered that death might occur in rare instances beginning at about 150 rads, in spite of the absence of any fatalities in the Marshall Islanders, who supply the first actual data point at about 175 rads. This conservative approach was based on the recognition that the exposure in the Marshallese extended over approximately 50 hours, which may have ameliorated some of its effects. The selection of 250 rads as an LD_{10} (i.e., the dose that would be lethal to 10% of the exposed population) is based largely on clinical observations in the Ewing's sarcoma patients treated by Rider (Rider and Hasselback, 1968). It seems likely that, without the supportive therapy given these patients, several more fatalities would have occurred, and therefore that somehwat lower exposures would also be lethal to some. When the LD_{10} and LD_{100} (ie., 100% mortality) points were connected, a median lethal dose (LD_{50}, the dose that would be lethal to 50% of the exposed population) of 340 rads resulted. Accepting the uncertainty of ±10%

suggested by NCRP Report 42, this provides a range of 314 to 374 rads, which overlaps the best estimate figure of the Biomedical and Environmental Assessment Group at the Brookhaven National Laboratory and the UNSCEAR Report of 1962 at 360 rads and is close to the 300 rads suggested in NCRP Report 42 and NAS Publication 1487 (Langham, 1967). In addition, it provides a span of 230 rads from LD_5 to LD_{95}, which is in fairly close agreement with the span of about 200 rads found experimentally in dogs (Cronkite and Bond, 1960; Perman et al., 1962) and other animals. Finally, this dose range is in keeping with the analysis of statistical features of radiation injury in NCRP Report 42, which suggests that the LD_5 in large animals and man is about half the LD_{50}.

F1.2.2 Supportive Treatment

The assumptions used in developing curve B in Fig. VI F-1 included the following: (1) the shape of the curve should be approximately the same as that of curve A and (2) the experimental dog studies cited above suggest that supportive treatment increases the LD_{50} by a factor of approximately 1.5. In addition, evaluation of the patients treated by Rider (data point 4) showed only one postirradiation fatality in 22 cases.[1] Another supportive-treatment data point (data point 5) was located on the basis of the lack of mortality in accident patients (Thoma and Wald, 1959, with newer cases added) who received a mean exposure of at least 450 but less than 600 rads (NCRP, 1974). The clinical category to which these patients were assigned was group III of the acute radiation syndrome (i.e., severe hematologic injury).

F1.2.3 Heroic Treatment

The same shape was used for curve C as for curves A and B. The main data points are a 600-rad Pittsburgh accelerator accident patient (data point 6), who survived after receiving bone marrow transplants from an identical twin, and the radiation therapy series of E. D. Thomas et al. (data point 7), which showed 60-day postirradiation mortality of 20% in 37 leukemic patients treated with 1000-rad whole-body irradiation followed by bone marrow transplantation from close-matching siblings. The illness that justified this form of therapy was severe, and this suggests that data point 7 is conservative for normal individuals.

The dose-mortality curves in Fig. VI F-1 are based on the assumption that rads of external and internal radiation are equivalent in effect, and hence the curves are somewhat conservative for lower total doses. However, if the dose is sufficiently high, the bone marrow suppression alone may produce the characteristic radiation-induced depletion of all blood cell lines (pancytopenia) with its clinical consequences.

Recovery from hematologic radiation injury has been correlated with the fraction of stem cells of the blood cell lines that survive. In experimental animals given uniform whole-body exposure, in which energy absorbed by the hematologic cells is the same, the fraction of stem cells surviving the exposure can be calculated from the survival curves for each particular cell line.

Extrapolation to man indicates that after 700 rads only about 0.1% of the stem cells may survive. After 300 rads, about 5% of the stem cells may be expected to survive (Cronkite, 1967). In the absence of the clinical complications of blood cell depletion, the regenerative capability of blood cells and their doubling time accounts for the pattern of recovery about 30 days after such an exposure. To suppress human marrow regeneration permanently appears to require a minimum of about 1700 to 2300 rads delivered in 2 to 4 weeks (Sykes et al., 1964). About half as much may be about as effective in a single exposure, since 1000 rads from cobalt-60 in 5 hours has sufficed to prevent recurrent leukemia in 17 of 19 leukemic patients who received successful marrow transplants from an identical twin after the "conditioning" irradiation and in 48 of 63 patients receiving successful marrow transplants from a close-matching sibling in the same procedure (E. D. Thomas et al., 1975).

[1] The patient fell 1 week after the 300-rad whole-body irradiation and sustained a pathological fracture of her sarcomatous leg, which required operative intervention. Complications of bleeding and infection ensued. These included petechiae, nosebleed (epistaxis), and vaginal bleeding as well as bilateral bronchopneumonia.

F1.3 MODELS OF THE DOSE-RESPONSE RELATIONSHIP

More sophisticated approaches to calculating the relationship between dose rate and hematologic injury have been proposed by Bateman (1968) and Yuhas et al. (1972) and recently summarized by Lushbaugh (1974).

Using data from about 2000 clinical case records, Yuhas et al. (1972) developed a multifactorial regression model for human blood cell responses to single and multiple whole-body therapeutic radiation exposures. The percent of white cells left at the lowest observed blood count was found to be related to a power function on total exposure and the duration of therapy in days. Using this approach, Yuhas et al. found that the bone marrow effect produced by a given dose in 2 days required two times that dose to be produced when the dose was spread over 7 days, about four times the dose over 21 days, and about nine times the dose over 3 months.

Bateman (1968) empirically fitted dose rate data for a number of different endpoints as a linear function of the reciprocal cube root of dose rate. This model appears to be appropriate for biological systems in which a significant dose rate effect is present. More recent studies also suggest that it should be limited to dose rates below 100 rads per minute and to exposures of less than 100 days in duration. Bateman's model indicates that the marrow damage produced in normal persons by a dose delivered in 1 day would require twice that dose if protracted over 1 to 3 months and three times that dose if protracted over a year. Since Bateman's model gives a more conservative result, it has been used for computational purposes in this study.

F2 LUNGS

F2.1 INTRODUCTION

The reaction of the lungs to intense irradiation from external sources or from internally deposited radionuclides is characterized by (1) an early stage of radiation pneumonitis and (2) a late stage of pulmonary fibrosis. The severity of these reactions and their time course are determined by the total radiation dose, dose rate, fraction of lung irradiated, and the condition of the lung before exposure.

The relevant literature on the effects of lung irradiation, with emphasis on internal radionuclide exposure, has been recently reviewed by Hahn (1975).

F2.2 EXTERNAL EXPOSURE

Hemorrhagic or other lesions are commonly found in the lungs of patients exposed to total-body irradiation, but the changes appear to result primarily from various systemic disturbances. However, direct radiation-induced damage to the lungs may also occur; for example, Fleming et al. (1960) reported hyaline membrane formation in two patients given total-body irradiation.

Available information on radiation pneumonitis in humans is based primarily on clinical experience in radiation therapy for cancer. The clinical data have demonstrated the importance of (1) lung volume and (2) dose protraction and fractionation in the development of clinically apparent damage.

Fatal reactions are expected after radiation doses to both lungs of 2500 to 3000 rads (Rubin and Casarett, 1973). Higher doses are tolerated when only a portion of the lungs is exposed; for example, no mortality and varying degrees of fibrosis are produced at the 4000 rad level. After doses higher than 6000 rads, clinically important reactions invariably occur, even when small portions of the lungs are involved.

The tolerance of pulmonary tissues to irradiation is increased by protraction and fractionation. The severity of radiation pneumonitis decreases with dose rate. However, pulmonary fibrosis is more dependent on the total dose than on dose rate (Rubin and Casarett, 1968).

In the fractionated-dose schemes used in radiation therapy, clinically important reactions are unusual at total doses below 2000 rads. Varying degrees of injury occur at the 4000 rad level, the severity depending on the lung volume involved.

There are several reports on the irradiation of large lung volume in cancer patients (see, for example, Margolis and Phillips, 1969; Cox et al., 1972; Baeza et al., 1975). The results emphasize the importance of the time-dose relationship with regard to the production of symptomatic radiation pneumonitis. Fatal pulmonary complications have occurred in cancer patients after 3000 rads delivered in 2 weeks to both lungs. Fractionated doses of 1000 rads per week produce acute radiation pneumonitis, whereas a decrease to 700 rads per week significantly reduces the incidence. Midplane doses of 1500 rads delivered in 10 fractions over 2 weeks and 2000 rads delivered in 15 fractions over 3 weeks to sizable lung volumes are generally tolerated. However, Baeza et al. (1975) reported a 23% incidence (in nine out of 39 patients) of radiation pneumonitis with tumor doses, which approximate lung doses, between 1500 and 2000 rads to both lungs in 2 to 3 weeks. The average time of onset after exposure was 2.7 months.

A quantitative evalaution of time-dose factors with regard to the production of radiation pneumonitis has been carried out by Wara et al. (1973). The authors established a dose-response curve for radiation pneumonitis based on clinical symptoms and radiographic changes for a series of 51 patients treated with irradiation to 75% of one lung or more for metastatic lung cancer. The nominal single dose (NSD) that was calculated to produce radiation pneumonitis in 50% of those exposed was about 1050 rets.[1] For total lung irradiation, Wara et al., suggest a treatment regimen of 2500 rads delivered in 20 fractions, to ensure that acute and chronic lung damage is not produced.

Radiation pneumonitis has been observed in a number of animal species after external irradiation (reviewed by Weir and Michaelson, 1971). Studies in mice (Phillips and Margolis, 1972), rats (Jennings and Arden, 1961), hamsters (de Villiers and Gross, 1967), rabbits (Engelstad, 1940), and dogs (Michaelson and Schreiner, 1971) indicate that the chronological development of both acute and chronic pulmonary lesions after chest irradiation is qualitatively similar to that observed clinically in humans.

Phillips and Margolis (1972) determined that, in mice given x-irradiation of the chest, the dose producing 50% mortality within 160 days was 1350 $\pm$ 50 rads. Using fractionated exposures approximating the schedules followed clinically for humans, they found that a dose of 2650 rads delivered in 20 fractions to both lungs produced a 5% incidence of pneumonitis and a dose of 3050 rads in 20 fractions produced a 50% incidence. Field and Hornsey (1974) reported that 1170 $\pm$ 100 rads produced 50% mortality within 180 days in mice exposed to chest irradiation.

In dogs, the dose causing 50% mortality in 30 days after a single upper body exposure is approximately 1700 rads (Hansen et al., 1961). Autopsy showed pulmonary congestion and fibrinous pneumonia, but other complications, such as neuroendocrine abnormalities and infection, had developed. Sweany et al. (1959) studied the effect of fractionated thoracic x-irradiation on pulmonary function in dogs. The calculated nominal single doses were 930 to 1500 rets. Four of seven dogs died within 6 months of irradiation. No changes in pulmonary function were found in the first few weeks after exposure, but pulmonary diffusing capacity, lung compliance, and functional residual volume decreased progressively thereafter. A similar reduction in pulmonary compliance has been demonstrated in rats within 6 weeks of receiving single doses of more than 1500 rads to both lungs (Shrivastava et al., 1974). Tyree et al. (1966) reported 100% mortality in five dogs after 1250 rad midline doses to the chest. However, secondary infections were a complicating factor in these deaths.

F2.3 INTERNAL EXPOSURE

Limited data are available on the acute effects of inhaled radionuclides in man. Exposures have resulted either in no clinically apparent pulmonary damage (Roeder, 1968; Ross, 1968) or in the production of late effects related to metaplasia and neoplasia (BEIR Report, 1972; UNSCEAR, 1972). The development of radiation pneumonitis has been reported in a radium plant worker, presumably caused by inhalation of radon and radon daughters (Rajewsky, 1939), but the report contains inadequate details to permit a quantitative evaluation of the dose.

[1] The NSD, expressed in units of rads equivalent therapeutic (rets), is determined by extrapolating the dose-time relationship back to a single fraction, taking into account the number of fractions and the total time separately (Ellis, 1968). It is used to compare doses from multiple exposures with those of single exposures.

Only a few experimental animal studies have reported radiation pneumonitis after the inhalation of radionuclides. Typical lesions have been observed in dogs after the inhalation of $^{144}CeO_2$ (Stuart et al., 1964) and $^{239}PuO_2$ (Clarke and Bair, 1964) and in rats after the inhalation of $^{144}CeO_2$ (Thomas et al., 1972) and intratracheal injection of $^{144}CeCl_3$ (Cember and Stemmer, 1964). The radiation pneumonitis induced by these internally deposited radionuclides is, in general, similar to that produced by external irradiation. However, due to the localization of the radionuclide in the lung, the penetrating ability of the emitted radiation, and the residence time of the material in the lung the onset and tissue distribution of these lesions differ from those produced by external irradiation.

Marked acute pulmonary damage has been produced by inhaled plutonium in experimental animals (Bair et al., 1973). The deposition of large quantities of plutonium (in excess of 0.5 microcurie per gram of lung) causes death within a short period (weeks) after exposure, with the lungs showing the exudative and inflammatory changes characteristic of acute radiation pneumonitis. With somewhat lower doses, down to about 0.1 microcurie per gram of lung, a gradually developing fibrosis eventually causes cardiopulmonary insufficiency and death after more prolonged periods (years). Dogs exposed to insoluble $^{239}PuO_2$ particles at levels greater than 0.1 microcurie per gram of lung died within a year from cardiopulmonary insufficiency. At the lowest $^{239}PuO_2$ levels producing fibrosis, the total radiation dose was between 2000 and 12,000 rads delivered at dose rates of 0.5 and 8 rads per day, respectively.

Quantitative dose-response studies with dogs that had inhaled various beta-and gamma-emitting radionuclides have been reported (McClellan et al., 1970; Hobbs et al., 1972; McClellan and Rupprecht, 1974). Groups of beagle dogs received single, nose-only exposures of yttrium-90, yttrium-91, cerium-144, or strontium-90 incorporated into aerosols of relatively insoluble, aluminosilicate particles to give graded initial lung burdens of radioactivity. The varying radioactive half-lives of the four radioisotopes resulted in a variety of effective half-lives in the lung, ranging from 2.6 days for yttrium-90 to about 400 days for strontium-90. Because of the protracted irradiation of the lung with cerium-144 and strontium-90, very high pulmonary radiation doses were accumulated by the time of death. Doses ranged from 9300 rads for the short-lived isotope yttrium-90 to 140,000 rads for the long-lived isotope cerium-144.

Animals dying within 500 days of inhalation had varying degrees of radiation pneumonitis and pulmonary fibrosis, and succumbed because of respiratory insufficiency. Pulmonary lesions induced by all four radionuclides were qualitatively similar. In general, dogs exposed to yttrium-90 and yttrium-91 had shorter survival times than those exposed to cerium-144 or strontium-90. The doses that produced death from radiation pneumonitis and/or pulmonary fibrosis are given in Table VI F-1.

Table VI F-2 shows the cumulative mortality in dogs within 1 year of exposure to yttrium-90 in fused-clay particles. Cumulative 365-day lung doses (infinite-time doses) of 20,000 rads or more resulted in 100% cumulative mortality from radiation pneumonitis and/or pulmonary fibrosis. A similar analysis of mortality in dogs exposed to yttrium-91 aerosols (Fig. VI F-3) shows that pulmonary doses of 30,000 rads or more resulted in 100% cumulative mortality. Dose-response curves derived from these data are given in Fig. VI F-4 for yttrium-90 and yttrium-91.

Mauderly et al. (1973) have shown that subclinical (i.e., not apparent) impairment of lung function is produced in dogs after the inhalation of relatively low levels of yttrium-90 in fused-clay particles. Two to three months after exposure, three animals with yttrium-90 levels giving lung doses of 4900 to 5700 rads developed functional impairment that could be detected only by dead-space tolerance or treadmill exercise testing. All three dogs survived to 1 year after exposure. These results suggest that dogs surviving after pulmonary doses of 5000 to 9900 rads and more (Fig. VI F-2) have a mild, subclinical, radiation pneumonitis, despite their prolonged survival. (The data indicate a 100% incidence of morbidity for doses in the 5000- to 9900-rad range.) Comparable studies with pulmonary doses appreciably smaller than 5000 rads have not been conducted.

F2.4 COMPARISON OF HUMAN AND ANIMAL DATA

The development of pulmonary changes leading to radiation pneumonitis and pulmonary fibrosis is qualitatively similar in humans and experimental animals. Furthermore, the doses required to induce these changes in various species do not vary by more than a factor of 2. Doses producing 50% mortality after external chest irradiation vary from 1170 to 1350 rads in mice to about 1700 rads in dogs. Reported nominal single doses producing a 50% incidence of radiation pneumonitis in mice, dogs, and humans are 1040, 930 to 1500, and 1050 rets, respectively. Despite the apparent differences in dose calculation and biological endpoints involved (i.e., clinical radiation pneumonitis versus mortality), these similarities suggest that animal data can serve as a basis for reasonably predicting the effects of lung irradiation in humans.

F2.5 ANALYSIS OF THE HYPOTHETICAL ACCIDENT

The fractional buildup of the 365-day pulmonary dose resulting from a maximum-release accident would be as follows:

Days after Exposure	Fraction of 365-Day Dose
2	0.26
7	0.42
30	0.56
60	0.64
180	0.84
360	1.00

These data are plotted in Fig. VI F-5.

There are no human data for predicting the effect of the temporal and spatial pattern of the pulmonary dose resulting from the hypothetical accident. It is assumed that the radiation response of the lungs of dogs and humans is comparable. The dose-buildup patterns obtained in dogs after exposure to yttrium-90 and yttrium-91 in fused-clay particles are compared in Fig. VI F-5 with the dose-buildup pattern estimated to result from the hypothetical accident. The curves for yttrium-90 and yttrium-91 generally bound the accident curve, with the initial rapid accumulation of pulmonary dose to day 7 after the release being approximated by the curve for yttrium-90 and more closely following the curve for yttrium-91 at later times.

Fig. VI F-4 indicates that a cumulative 100% mortality occurs after lung doses on the order of 15,000 rads or more from yttrium-90 and of 30,000 rads or more from yttrium-91. Thus, individuals receiving pulmonary doses of this magnitude from a maximum-release accident would succumb to radiation pneumonitis and/or pulmonary fibrosis at the end of 1 year. Estimates of the mortality incidence resulting from lower doses can be made from the dose-response curves in Fig. VI F-4.

Limited data exist on the incidence of morbidity produced by internal pulmonary irradiation in experimental animals. Radiation therapy experience in humans indicates that clinically important reactions are invariably produced by doses on the order of 6000 rads and are unusual after doses of 2000 rads (Rubin and Casarett, 1968). Wara et al. (1973) demonstrated that no acute or chronic lung damage was produced after doses of 2500 rads in 20 fractions to the whole lungs of radiotherapy patients. Limited dog data show 100% morbidity after doses of 5000 to 9900 rads from internally deposited radionuclides (Manderly et al., 1973) and are thus in close agreement with the clinically suggested dose of 6000 rads.

These data suggest that pulmonary morbidity can be reasonably estimated by assuming a 100% incidence after 6000 rads and a 5% incidence after 3000 rads. The dose-response curve is shown in Fig. VI F-6.

F3 GASTROINTESTINAL TRACT

F3.1 INTRODUCTION

Lethal radiation-induced gastrointestinal injury and the associated symptoms are consequences of the disruption of the normal proliferation of the intestinal epithelium (lining). The small intestine, because of its more rapid cell turnover, is the most radiosensitive portion of the intestinal tract, and the stem cells in the crypts of the villi are the most radiosensitive cells. The stem cells continuously supply mature, functional cells to the tips of the villi, the rate of delivery normally equaling the rapid and continual shedding of old cells. Reduction in the stem-cell population and/or the cell-renewal rate after irradiation results in a diminished flow of cells to the tips of the villi.

Despite the high radiosensitivity of the small intestine, lethal damage is produced only by radiation doses in the kilorad range (Bond et al., 1965). Single doses of this magnitude delivered in a short period of time produce marked cell killing and completely inhibit cell division. This causes denudation of the intestinal lining, which in turn leads to loss of vital intestinal function and the development of diarrhea, dehydration, electrolyte imbalance, hemorrhage, and infection.

Sublethal doses produce only temporary changes in the stem-cell population, and complete regeneration occurs, even after relatively severe initial damage. Thus, the critical factor is the survival of adequate numbers of stem cells to resume cell production in time to prevent complete denudation of the villi.

The high tolerance of the intestine to acute radiation damage, in comparison to other rapid-cell-renewal tissues, is attributed to a number of protective mechanisms (reviewed by Hagemann and Concannon, 1975). Stem cells possess a high capacity to rapidly repair sublethal radiation damage--as well as an ability to shorten their life cycle, thus accelerating cell production--in response to decreases in the stem-cell population. In addition, compensatory changes occur in the villi themselves. These changes, which include a broadening in the shape of the surviving epithelial cells and a shrinkage in villus size, serve to maintain an intact intestinal lining until stem-cell recovery can occur. The combined effect of these mechanisms is lower cell mortality at low single doses and an increased tolerance to protracted or fractionated irradiation. Thus, larger total doses are required to produce the same degree of intestinal damage as that resulting from a single brief exposure.

The radiation response of the stomach and large intestine is similar to that of the small intestine, but damage develops more slowly because of their lower rate of cell renewal.

Local irradiation of the gastrointestinal tract may occur from external exposure to the abdominal region and/or from internal radionuclide contamination by inhalation and/or ingestion.

F3.2 EXTERNAL EXPOSURE

Available human data are insufficient to establish dose-response relationships for either the gastrointestinal form of the acute radiation syndrome (Lushbaugh, 1974) or for mortality after local intestinal irradiation.

F3.2.1 Gastrointestinal Response to Total-Body Irradiation

The development of the classical gastrointestinal syndrome requires that the functional efficiency of both the gastrointestinal and the blood-forming (hematopoietic) systems be altered (Bond et al., 1965). Although intestinal changes are produced by total-body radiation doses sufficient to cause the hematologic syndrome, the intestinal recovery mechanisms prevent the development of acute gastrointestinal symptoms. The treatment of advanced cancer patients with total-body doses of up to 1000 rads in efforts to prolong survival or to prepare individuals for bone marrow transplants has not resulted in severe gastrointestinal complications (King, 1961; Saenger et al., 1973; E. D. Thomas et al., 1975). Similarly, 300 rad lower-body irradiation, either alone or in combination with 200-rad total-body doses, did not produce significant effects (Saenger et al., 1973). The bone marrow transplantation studies of E. D. Thomas and co-workers (1975) indicate that, in the absence of hematological complications, the human total-body dose for producing early mortality from gastrointestinal injury is above 1000 rads.

F3.2.2 Gastrointestinal Response to Local Irradiation

Radiation therapy experience indicates that the severity of the gastrointestinal response to local irradiation varies with the total dose, dose rate, and volume and segment of tract (small intestine, large intestine, or stomach) irradiated. The tolerance is determined primarily by the radiosensitivity of the small intestine.

Early mortality from local intestinal irradiation has been shown only in experimental animals. The response differs from the gastrointestinal syndrome produced by acute total-body irradiation (which characteristically causes death in 3 to 4 days in the mouse, rat, and dog) in that higher doses are required and death occurs later (Bond et al., 1965). For example, the mean survival time in rats after a brief total-body exposure of 1500 rads was 3.6 days, versus 7.1 days after a similar dose to the exteriorized intestine[1] (Sullivan et al., 1959). In irradiation of the lower abdomen, the median lethal dose (LD_{50}) was found to be 1620 rads, the mean survival time being 5.7 days. Microscopic examination showed complete recovery of the rat jejunal mucosa within 1 week after a 1000-rad dose to the exteriorized intestinal surface (Wiernik and Plant, 1970).

Survival time also varies with the proportion of total intestine irradiated. Rats given 3000 or 4000 rads to 20% of the exteriorized small intestine survived for about 12 days, whereas comparable doses to 50 or 80% of the intestine caused death in about 5.5 days (Osborne, 1956). Animals given whole-body and abdominal irradiation lived an average of 3.2 and 3.5 days, respectively. When short (2.5- or 7.5-centimeter) segments of exteriorized rat ileum were irradiated with 3000 or 5000 rads, the survival time was considerably longer: the majority of the animals died after 10 to 100 days, with some living longer than 200 days (Osborne et al., 1970). A lesion characterized by serosal fibrosis, apparently secondary to mucosal ulceration, was produced in all animals, causing death due to bowel obstruction.

The mouse and dog show similar increases in mean survival time after abdominal irradiation in comparison to total-body exposure (reviewed by Bond et al., 1965). However, the data are more limited than those for the rat. Furthermore, because of the early termination of the experiments, no assessment of "delayed" death or morbidity is possible.

F3.2.3 Tolerance to Protracted or Fractionated Irradiation

Several recent studies have demonstrated the marked tolerance of the intestinal tract to protracted or fractionated irradiation. Krebs and Leong (1970) showed that, in mice exposed to total-body cobalt-60 radiation, the dose causing 50% mortality in 5 days increased steadily from 1653 to 3133 rads as the exposure rate was reduced from 111.3 through 7.45 roentgens per minute. The change was attributed to rapid repair during exposure, the lethal dose being reached when the sum of the damage reached a critical value. A similar repair effectiveness was observed after fractionated x-irradiation (exposure rate: 68 roentgens per minute) of the abdominal region of mice (Hagemann and Concannon, 1975). When delivered in a single exposure, the dose causing 50% mortality in 6 days was 1610 rads At exposure rates of 1000 roentgens per week, the cumulative doses producing 50% mortality ranged from 1800 to 11,500 rads, depending on the schedule of exposure. The single-fraction extrapolate derived from these data was 1270 rets.

No acute gastrointestinal symptoms were observed in monkeys and dogs after total-body cobalt-60 irradiation delivered in unequal fractions to total doses of 1048 rads (Spalding et al., 1969) or in dogs given abdominal doses of 1820 to 2970 rads fractionated in 12 equal daily fractions (Concannon et al., 1970).

F3.2.4 Summary

Local external irradiation of the intestinal tract in the kilorad range can result in significant mortality or morbidity in exposed individuals. The lack of acute effects in man after doses of up to 1000 rads and the available animal data suggest that single brief doses in excess of 1500 rads to the abdomen are required to produce such damage.

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[1] In this procedure, the intestine is lifted out of the abdominal cavity and directly irradiated, the rest of the body being shielded.

Ret doses calculated from radiotherapy data, though derived from different endpoints and radiation conditions, support this conclusion. Dose protraction or fractionation markedly increases the total dose tolerated.

F3.3 INTERNAL EXPOSURE

No acute gastrointestinal injury from internal radionuclide contamination has been reported in humans. The available information is limited to effects produced in experimental animals after exposure to individual radionuclides.

Internal radionuclide contamination of the gastrointestinal tract occurs after ingestion and inhalation. The ingested nuclides irradiate segments of the tract during passage, exposure being maximal from nuclides that are poorly absorbed because of their chemical properties or their presence in an insoluble form. The radiation dose to any particular segment of the tract is determined by the residence time of the contaminated ingesta in that segment. The lower large intestine is the gastrointestinal region of major concern because of its prolonged occupancy factor (Eve, 1966).

The large intestine differs from the small intestine in that it contains no villi (Bloom and Fawcett, 1975). The mucosa has a smooth surface and is about 0.5 to 0.7 mm thick. The glands of Lieberkühn are straight tubules about 0.5 mm long. A mucous layer about 1 mm thick covers the mucosal surface. These anatomical features make it difficult to relate surface dose to the intestinal wall and acute intestinal damage, particularly for radionuclides emitting radiations of low penetrating power.

F3.3.1 Exposure by Ingestion

F3.3.1.1 Single Exposure

The intragastric (i.e., directly into the stomach) administration to rats of large quantities of plutonium-239 did not produce acute pathologic effects, despite surface doses to the intestinal wall of hundreds of thousands of rads (Sullivan and Thompson, 1957; Sullivan et al., 1960), reflecting the inability of alpha radiation to reach the critical cells. A similar ineffectiveness in producing damage would be expected for unabsorbed radionuclides emitting beta particles comparable in range to alpha particles (Unnikrishnan et al., 1973).

Early mortality from intestinal damage has been observed in rats that had ingested poorly absorbed radionuclides emitting high-energy beta radiation. The median lethal dose in rats given yttrium-91 (average beta energy: 0.606 MeV) was 17 millicuries per kilogram of body weight, the average survival time being 8.4 days (Sullivan et al., 1960). This ingested dose was calculated to have delivered surface doses of 1150 rads to the small intestine, 1287 rads to the ascending colon, and 3929 rads to the cecum. Only minimal intestinal damage was produced by a sublethal dose of 10 millicuries per kilogram, calculated to have delivered 1650 to 2765 rads to the large intestine.

The acute toxicity of the beta-emitting nuclide complex ruthenium-106/rhodium-106 (average beta energy: 1.43 MeV) has been studied in rats given single exposures (Sullivan et al., 1975a and b). The median lethal doses for adult, weanling, and newborn animals were 9, 20, and 2 millicuries per kilogram, the average survival times being 8.4, 4.5, and 10 days, respectively. Adult rats died from severe damage to the cecum after the surface mucosa had received doses on the order of 4500 rads. The greater lethality in the newborn was attributed to the incorporation of ruthenium-106 into the lining of the lower small intestine, where it caused complete denudation in 8 days.

Unpublished experiments of M. F. Sullivan have shown that in rats the median lethal dose (single ingestion) of promethium-147 (average beta energy: 0.062 MeV) is 5 curies per kilogram of body weight (see Cross, 1975a and b). The pathological changes were similar to those produced by ruthenium-106/rhodium-106.

Cross (1975a and b) has analyzed Sullivan's data on acute mortality in the rat after a single ingestion. Depth-dose data for nine beta-emitting radionuclides ranging in average energy from 0.062 to 1.43 MeV were computed for a cylinder of the same diameter as the rat intestine (Table VI F-2). The analysis indicates that the critical tissues in the rat cecum lie at or near a depth of 0.0175 cm. Table VI F-3 gives the median lethal dose extrapolation for the intestinal surface and at 0.0175-cm depth for the nine radionuclides.

Using the above LD_{50} large-intestine data for ruthenium-106/rhodium-106, yttrium-91, and promethium-147, Cross (1975c) applied the dose reduction factors to the critical tissue(s) at 0.0175 cm and calculated the doses to the segments of the large intestine (see Table VI F-4). Despite the wide variation in surface dose for the three radioisotopes, the mean and standard deviation of the dose to the critical depth in the rat cecum is 3514 ± 437 rads. Thus, in the rat the large-intestine dose for acute mortality is on the order of 3500 to 4000 rads to critical tissues in the cecum.

Sullivan et al. (1975a and b) characterized the acute lethality of ruthenium-106/rhodium-106 in adult dogs. The acute LD_{50} was estimated to be 3.5 millicuries per kilogram, the average survival time being about 10 days. The resulting surface dose to the mucosa was estimated to be 2000 rads in the ileum and 8000 rads in the midcolon. The majority of deaths resulted from damage to the midcolon. Dogs given smaller doses (2.75 to 3.0 millicuries per kilogram) survived for up to 6 months after exposure. The animals showed severe gastrointestinal symptoms, including persistent vomiting, anorexia, and bloody diarrhea; death was attributed primarily to midcolon damage. The delayed death observed in dogs was not seen in rats after comparable exposures to ruthenium-106/rhodium-106.

Analysis of the dog mortality data indicated that the critical tissue in dog colon is 0.050 to 0.075 cm below the mucosal surface. Tables VI F-5 and VI F-6 give the extrapolated doses producing 50% mortality in 10 and 180 days, respectively. Table VI F-7 shows the doses obtained by direct measurements with thermoluminescence dosimeters implanted into the intestinal wall (Cross, 1975e).

F3.3.1.2 Repeated Ingestion

The effect of repeated daily ingestion of yttrium-90 (average beta energy: 0.937 MeV) has been measured in rats (Sullivan et al., 1963). One group of 52 animals ingested an average of 410 microcuries per day for 60 days, which resulted in a total dose of about 55,000 rads to the large intestine. Another group was given an average of 91 microcuries per day for 60 days and thus received a total dose of about 12,000 rads. Nine deaths occurred in the first group during exposure, primarily from radiation-induced intestinal damage, and all 52 rats experienced severe weight loss, which was regained within 2 weeks after the last ingestion of yttrium-90. No gross symptoms developed in the second (low level) group. All animals, except the nine from the first group, survived for long periods after treatment, which demonstrates the marked tolerance of the intestine to protracted irradiation and its ability to recover after large doses.

Nold et al. (1960) fed 24 millicuries of yttrium-90 to mongrel dogs in daily doses of 5 millicuries per day. Colon doses above 2200 rads produced relatively minor histopathologic injury until they exceeded 6000 rads. All dogs were killed 7 days after treatment, so lethality was not determined. However, the relatively minor nature of the intestinal damage, in comparison to the damage caused by comparable doses delivered after a single ingestion exposure (Sullivan et al., 1975a and b), suggests a reduced effectiveness due to the protracted dose schedule.

F3.3.2 Exposure by Inhalation

Inhalation exposure contributes a significant gastrointestinal component due to the transfer of deposited material from the nasopharyngeal and tracheobronchial regions. The relative percentage of inhaled material that is deposited in these regions is highly dependent on particle size. Particles with a mass median aerodynamic diameter (MMAD) of 0.01 to 2 microns are deposited predominantly in the deep lung compartment, whereas smaller and larger particles are deposited primarily in the nasopharyngeal and tracheobronchial regions. The clearance time for the nasopharyngeal and tracheobronchial regions determines the rate at which the material is transferred to the intestinal tract.

The gastrointestinal effects produced in 10 beagle dogs after the inhalation of yttrium-90 in fused-clay particles were studied by Hahn et al. (1975). The particle characteristics (MMAD ranging from 1.9 to 2.6 microns, with geometric standard deviations of 2.0 to 2.2) and exposure conditions resulted in initial lung burdens ranging from 9 to 35 millicuries and subsequent gastrointestinal burdens of between 8 and 34 millicuries. No deaths attributed primarily to intestinal injury occurred. Nonlethal focal ulcerations were induced in the colon by yttrium-90 at levels giving measured surface doses of 3200 to 5700 rads in 196 hours. Six of seven dogs killed at 27 to 29 days after exposure had severe radiation pneumonitis and radiation dermatitis

of the nostrils and surrounding skin. The extent of intestinal damage suggested that repair had occurred. These findings show that yttrium-90 in insoluble particles in the 1 to 3-micron MMAD range does not cause an acute, life-shortening irradiation of the intestinal tract. This ineffectiveness in producing lethal gastrointestinal damage, despite the high dose rate and the dose levels attained, suggests that a dose protracted over 196 hours is less effective than the same dose delivered in a shorter time.

Cuddihy (1974) has analyzed the factors affecting the radiation dose distribution between the lung and the lower large intestine after inhalation exposure. In general, time-integrated total doses to lung are much greater than gastrointestinal doses unless very short-lived radionuclides are involved. When very short-lived nuclides (e.g., yttrium-90) are inhaled in the form of very large or very small insoluble particles (i.e., more than 2 and less than 0.01 micron in MMAD, respectively), gastrointestinal injury becomes more important than pulmonary injury. For radioisotopes with longer half-lives (e.g., yttrium-91), particle sizes greater than 10 microns MMAD would be required before gastrointestinal injury became the dominant consideration.

F3.3.3 Summary

Internal irradiation from ingested beta-emitting radionuclides can produce acute injury to the lower large intestine. The severity of such damage is highly dependent on the average energy of the emitted radiation. Studies in dogs indicate that an acute dose on the order of 3500 to 5000 rads at a critical depth in intestinal tissue is required for early lethality.. Lower doses to critical tissue, on the order of 2500 to 4000 rads, cause significant morbidity and delayed death. The temporal distribution of the dose markedly affects the response. The induction of acute gastrointestinal injury after inhalation exposure requires that both the radionuclide half-life and particle size be such that the transfer of material to the intestines and the dose delivery are maximized.

F3.4 ANALYSIS OF THE HYPOTHETICAL ACCIDENT

The fractional buildup of the 365-day dose to the lower large intestine from combined external and internal sources after a maximum release would be as follows:

Days after Exposure	Fraction of 365-Day Dose
2	77.1
7	96.4
30	97.6
60	98.8
180	99.4
360	100.0

The temporal radiation dose pattern indicates that the period of major concern with regard to the production of intestinal damage would be the first 7-day period after exposure, when the dose would be accumulated fairly rapidly. Based on the documented tolerance of the intestines to protracted irradiation from both external and internal sources, the rate and level of dose accumulation subsequent to this time should be readily tolerated.

There are no human data for predicting the acute gastrointestinal damage that would result from the above projected temporal and spatial radiation dose pattern. Experimental data in dogs most closely approximate the projected conditions. It is assumed that the intestinal response of the dog and the human is comparable and that the results of Sullivan et al. (1975a and b) and Cross (1975a, d, and e) on mortality in dogs may be directly applied to the projected situation.

Early mortality in dogs after the ingestion of ruthenium-106/rhodium-106 was produced by mucosal-surface doses above 8000 rads, which resulted in critical-tissue doses on the order of 5000 rads, with a range of 3500 to 7000 rads (Cross, 1975e). Although no data are available to estimate the upper or lower bounds, the curve shown in Fig. VI F-7 is suggested.

It should be noted that the direct application of these data, obtained with a high-average-energy-emitting radionuclide administered in an acute single exposure, will overestimate the early mortality incidence due to the 7-day dose buildup. Protraction of the dose has been shown to be more readily tolerated (Sullivan et al., 1963; Nold et al., 1960; Hahn et al., 1975). Thus, it appears highly unlikely that early mortality from intestinal damage will be a significant factor under the hypothetical accident conditions.

Significant morbidity leading to delayed death was produced in dogs with ruthenium-106/rhodium-106 after critical-tissue doses on the order of 4000 rads, with a range of 2500 to 5500 rads (Cross, 1975e). Assuming a 100% mortality incidence at a 2500-rad dose to critical tissue and a 5% incidence at 1000 rads, the curve shown in Fig. VI F-8 is suggested. The application of these data should similarly overestimate the morbidity incidence for the reasons given above.

The dose buildup in the intestinal tract over 7 days provides adequate time to initiate the use of mild laxatives to accelerate the excretion of contaminated ingesta. Such supportive treatment, which reduces the average intestinal dose by a factor of 2 to 4 (Nold et al., 1960), is suggested for individuals receiving significant inhalation and/or ingestion exposure.

The following point should be noted. If exposure of the lower large intestine is used as the prognostic indicator of gastrointestinal lethality, the resulting estimate will be conservative because the radiosensitivity of the upper large intestine (i.e., mid-colon), at least in the dog, is greater than that of the lower large intestine (see Table VI F-7).

F4 OTHER ACUTE EFFECTS

In addition to the major acute effects described in the preceding sections, there are some noteworthy but nonlethal forms of radiation damage that may occur as a result of localized as well as whole-body exposure delivered from external or internal sources. The varied manifestations of the more significant of these are described in this section.

F4.1 PRODROMAL SYMPTOMS

The most consistent of the nonlethal effects of whole-body irradiation are the so-called prodromal symptoms: anorexia, nausea, vomiting, and diarrhea. The most definitive study involves a review of 504 cases of patients in 33 hospitals who underwent total-body irradiation because of life-threatening cancer (Lushbaugh et al., 1968). The initial analysis of some of these data was summarized in Radiobiological Factors in Manned Space Flight, NAS-NRC Publication 1487 (Langham, 1967).

The incidence of vomiting within 2 days as a function of dose was also plotted in report NAS-NRC Publication 1487 (see Fig. VI F-9). There is some divergence between the data based on clinical patients (straight line) and that based on accidental exposure cases (dashed line). However, both lines fit within the 95% confidence limits for the clinical patient data. In this connection it is of interest that, of the seven patients treated by E. D. Thomas et al. (1971b) with 1000-rad cobalt-60 whole-body irradiation and subsequent allogeneic bone marrow grafting, all demonstrated vomiting within 48 hours of irradiation. This data point, which was added to the NAS-NRC graph, falls right on the projection of the accidental exposure line. This suggests that data on radiation therapy patients are meaningful for accidentally exposed populations as well.

Figure Vl F-10 is based on more recent data on radiation therapy patients as well as information presented in NAS-NRC Publication 1487. The curve drawn represents a reasonable conclusion drawn from the data and clinical judgment.

The effect of dose protraction on such prodromal symptoms as nausea and vomiting have been studied by Lushbaugh (1974). Data from 1085 patients given small daily total-body exposures suggest that 30 or more daily exposures of 20 to 30 roentgens per day are required to induce these symptoms. The dose inducing vomiting in 50% of the patients was 182 rads when delivered in a single dose, 495 rads when protracted over 1 to 8 days, and 594 rads when protracted over more than 8 days. Thus an approximately threefold increase in dose was required to produce the same effect when its delivery was spread over a week or more as compared to a single exposure in less than a day.

F4.2 SKIN

The clinical manifestations of acute radiation injury to the skin vary in severity, depending on the total dose, the type and energy of the radiation and its depth of penetration. The endpoints of significance from both a diagnostic and a clinical standpoint are as follows (NCRP, 1974):

F4.2.1 Loss of Hair

Loss of hair (epilation) generally becomes apparent within 2 to 3 weeks after doses in in excess of 300 rads to the hair follicles. It serves as a reliable indicator of exposure to both external radiation and to mixed radiation from fallout. Permanent hair loss is produced by doses of 600 rads and above.

F4.2.2 Radiation Dermatitis

Radiation dermatitis consists of three early clinical stages in order of increasing severity: erythema (redness), transepidermal injury, and dermal radionecrosis.

Erythema (equivalent to a first-degree thermal burn) occurs after doses on the order of several hundred rads. The major redness generally appears 2 to 3 weeks after exposure, but the intensity of the reaction and its time of appearance vary widely with the dose.

Dry desquamation (scaling) follows. Medical care is not necessary.

Transepidermal injury or moist desquamation (equivalent to a second-degree thermal burn) occurs after brief skin doses between 1000 and 2000 rads. Confluent blisters (bullae) appear within 1 to 2 weeks, depending on the dose. The blisters may rupture, leaving open painful lesions vulnerable to infection. The need for medical care depends on the size, location, and severity of the lesion.

Dermal radionecrosis (equivalent to a third-degree thermal burn) occurs after brief skin doses in excess of 2000 rads. Pain occurs promptly and is intense and the areas are generally slow in healing. Medical abatement of pain is urgently needed and surgical resection and skin grafting may be required.

F4.3 LENS OF THE EYE

Although insufficient data are available for quantification, the dose-response relationship in the induction of cataracts severe enough to impair vision is considered to be sigmoidal for low-LET radiation (ICRP, 1969). The human threshold dose varies between 200 to 500 rads for a single brief exposure.

Progressive cataracts are formed primarily after doses of 500 rads and more, the probability of progression increasing with dose (Langham, 1967). The threshold dose increases on fractionation or protraction; two to three times as much radiation is required if the dose is delivered over a period of several months as compared to a brief single exposure. However, there is a high probability of cataract formation after doses in the 1100- to 1400-rad range, irrespective of the duration of exposure. The latent period varies in relation to the dose and the dose rate, ranging from 6 months to 35 years, with an approximate average of 2 to 3 years (Merriam, et al., 1972). A child at the age of 1 year manifests the same type of response as the adult.

Because of the high doses required, cataract formation would be a consideration only in individuals receiving a nonuniform exposure involving the lens of the eye without a correspondingly high whole-body exposure.

F4.4 IMMUNOLOGICAL IMPAIRMENT

The drastic effect of radiation exposure on lymphoid tissue and lymphocytes was one of the earliest radiobiological observations, made by Heineke in 1903. The effect on the immune system was described in 1908 by Benjamin and Sluka. A growing interest in the immune response in recent years has led to a marked increase in information on this subject. An excellent summary was published in the 1972 Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (United Nations, 1972), and useful information is contained in Manual on Radiation Haematology (International Atomic Energy Agency - World Health Organization, 1971).

It is not yet possible to make quantitative estimates of the risks to man from radiation-induced lesions in the immune system. Much of the information has come from animal experiments, and problems of species variation must therefore be recognized. Three main areas of study have been (1) the effect of radiation on resistance to infection and on antibody formation, (2) its role in preventing or facilitating tissue transplantation, and (3) the relationship to cancer induction. The first two are relevant to a consideration of acute radiation injury and its early manifestations.

Among the clearest evidences of radiation-induced immunologic injury is a decreased resistance to disease-producing microorganisms. This effect, which has been found in many irradiated species, is most striking in the exposure range of 200 to 600 roentgens. Experimental data and results are varied, at least in part due to the great variety of test situations and endpoints utilized in studying this complex system. In general, the radiation-induced decreased resistance to infection occurs several days after exposure rather than immediately.

The immune response involves both cellular and humoral factors. The effect of radiation on many of these factors has been studied in isolated test systems, and some of the results of interest are described below. For example, lymphocytes show effects in culture from a dose as low as 10 rads. The ability of cells to incorporate and degrade an antigen (e.g., a bacterium) is relatively resistant to radiation, requiring doses on the order of 1000 rads for inhibition. There is, however, some indication that, after doses as

low as 150 rads, irradiated macrophages that have incorporated the test antigen may not be able to process it in the required manner to initiate the immune response.

The production of antibodies after an antigen injection may show a shortened lag phase and higher antibody levels after relatively low doses (e.g., 25 rads). As the dose increases, the lag phase is increased due to the radiation effect on the hematopoietic stem cells whose D_{37} values (dose to give 1/e survival) are in the range of 60 to 150 rads. The carrying of antibody by the cells that are present does not appear to be impaired at doses of up to 2000 rads.

The logarithmic phase of antibody production is only moderately radiosensitive because it involves a mixture of highly radioresistant, mature, nondividing, antibody-synthesing cells as well as proliferating immature plasmablasts. The mature plasma cells show no significant depression of antibody secretion after doses of up to several thousand rads.

For these reasons, although the cells involved in the primary immune response and the secondary type may be equally affected and reduced in number by irradiation, responses differ in the number of cells actually needed. Thus a comparable radiation-induced reduction in the percentage of cells involved in primary and secondary immune response will still leave many more surviving cells in absolute numbers in the secondarily stimulated animal. This gives the appearance of greater radioresistance to the secondary antibody response.

The immune system is actively involved in the rejection of foreign materials, including tissues grafted from a foreign donor, and also the unique case in which the transplanted marrow, a component of the immune system, may "reject" the host. The dose of radiation necessary to suppress the graft-rejection mechanism is in the range that would be lethal without heroic treatment. This radiation effect makes it possible to make successful bone marrow transplants after accidental or therapeutic radiation exposure for otherwise fatal illness. However, there is a major immunological obstacle known as the graft-versus-host reaction, or secondary disease. It is characterized by apparently successful initial takes of the transplanted marrow, which flourishes until it recognizes the host tissue as foreign and attempts to destroy it.

The use of identical or close-matching cells from an immunologic standpoint as well as the use of the new immunosuppressive drugs has made possible the apparently successful long-term transplantation of bone marrow in some humans. Unfortunately, induced immunological tolerance, whether by radiation or other techniques, may also impair the immune system's complex functions, which are not yet clearly understood, in the area of self-tolerance and surveillance for deviations in the composition of the host's own cells. These in turn may lead to so-called autoimmune diseases or to cancer.

F4.5 FERTILITY IMPAIRMENT

Another nonlethal effect of radiation exposure occurs in the gonads, which are among the more radiosensitive organs of the human body.

Because of the much greater facility with which male spermatogenesis can be studied, most available experimental human data pertain to the male. Two recent studies in particular provide contemporary information on the effects of radiation on human spermatogenesis. In one of these studies, 64 male volunteers received a single midorgan dose to both testes ranging from 7.5 to 400 rads.[1] The cell-killing median effective dose (ED_{50}) was 75 rads, and the mitosis-halting ED_{50} was 27 rads. The maximum observed sterile period was 501 days, with eventual recovery observed in each individual for whom the followup was complete. (Thorsulund and Paulsen, 1972).

The second study involved exposures to the testes of human volunteers of single doses between 8 and 600 rads. Transient sterility was produced in the period from 46 to 67 days postexposure at all dose levels above 100 rads. Seventy five and 50 rads produced markedly reduced sperm counts, while lower doses including 20 rads showed moderate reductions. The first increase in sperm count ranged from 6 months for 20 rads to 24 months for 600 rads. Complete return to preirradiation level was within 9 to 18 months for low doses, 30 months for intermediate doses and 5 or more years for high doses. (Rowley et al., 1974).

To summarize, in the human male, radiation doses beginning above 10 rads and extending to 600 produce a decrease or absence of sperm beginning at least 6 to 7 weeks after

[1] Thorsulund and Paulsen quote an f factor of 0.95.

exposure and continuing for a few months to several years with subsequent recovery ensuing within this dose range. The magnitude of the depression and the rate of return of sperm count are related to the magnitude of the exposure. It is noteworthy however, that even the dose at the high end of the range, which would be in the potentially lethal area if administered to the whole body, is not sufficient to produce permanent sterility.

It is also to be noted that the biological mechanisms for expressing libido and potency are not affected in this dose range, although psychologic factors may affect sexual capacity. This is of importance because the relative radioresistance of spermatozoa allows a period of about 46 days before the deficiency of the more radiosensitive precursor cells results in a decrease in sperm count. The directly irradiated spermatozoa therefore can be involved in conception within the first six weeks postexposure unless measures are taken for its prevention.

Eventual recovery of spermatogenesis is due to the comparative radioresistance of the primitive spermatogonium (type A) which has an LD_{50} of about 600 rads. The more mature (type B) spermatognium is much more radiosensitive, being affected by as little as 15 rads. In man, unlike some other species, maturation of type A cells into type B and these into spermatocytes occurs even though replenishment by mitosis of the full complement of type A cells has not been completed. This makes for the very slow return to normal sperm levels following exposure. It also accounts for the fact observed by Heller and Rowley (Rowley, personal communication, 1975), that 11 fractional doses of 5 rads to the human testes produce a more rapid fall in the sperm and a longer recovery time requirement than single exposures to the same total dose of 55 rads. Different patterns of fractionation would, of course, produce different results.

The data on dose-response relationships for depression of sperm count from these two studies, presented in Fig. VI F-11, show reasonably good agreement.

Radiation effects on the human ovary differ from those in the testes because unlike the testes, the ovaries contain their entire supply of germ cells or oocytes early in life and lack stem cells capable of replacing those that are lost thereafter. Since the oocytes are relatively radiosensitive, loss of such cells due to radiation damage irreversibly reduces the reproductive potential of the woman exposed. The age of the ova and their stage of development affect their radiosensitivity.

Although human experimental data is lacking, there is information from studies of the effects of localized radiation therapy as well as followup studies of the Japanese atomic bomb survivors and Marshallese women exposed to radioactive fallout. Neither of the population studies have shown any apparent effect on fertility although there were no adequate controls.

Radiation therapy data have been reviewed in detail by Upton (1968), Rubin and Casarett (1968) and Lushbaugh and Ricks (Lushbaugh, 1972). Generalizations that emerge are that single exposures of 125 to 250 roentgens to the ovaries may produce prolonged or permanent amenorrhea in about 50% of women while about 600 "tissue roentgens" or rads are required to produce permanent amenorrhea in virtually all women so exposed. One to two thousand rads may be needed to reach this endpoint if the dose is fractionated over about two weeks and the subjects are young women, who are more radioresistant. If the dose is delivered over six weeks at 100 rads daily five days a week, Lushbaugh has estimated that the 50% probability level for permanent sterility would be about 2000 rads in young women.

F 5 SPECIAL EFFECTS ON PRENATAL GROWTH AND DEVELOPMENT

The mammalian organism is particularly sensitive to radiation damage during the embryonic and fetal stages. The embryonic period (i.e., the period in humans from fertilization to approximately the eighth week of pregnancy) is characterized by a rapid proliferation of cells and by organ differentiation and development. The fetal period (from the end of the eighth week of pregnancy to birth) is characterized primarily by the growth and maturation of the system, although the development of some organ systems (e.g., the cerebral and gonadal systems) continues.

The principal acute effects are prenatal death; neonatal death, congenital malformations; and growth retardation, both before and after birth (Brent and Borson, 1972, NCRP Report No. 42, 1974).

For each specific effect there is usually a "critical period" during which the effectiveness of irradiation is highest and the probability of producing that specific effect is highest. Some effects are evident soon after irradiation, but others only much later, often after birth.

Brief exposures to penetrating radiation delivered at a high dose rate at various stages of prenatal development produce the effects described in the following sections.

F5.1 Prenatal Death

The incidence of radiation-induced prenatal death is highest after irradiation in the early stages of development, particularly during the preimplantation stage--that is, before the fertilized ovum becomes attached to the uterine wall. During this period the organism is more sensitive to radiation than at any other time of life, and, in general, irradiation either causes death or the embryo develops normally, at least in a morphological sense.

The high radiosensitivity of the early embryo has been established primarily in rodents. A dose of 150 rads on the first day of gestation killed approximately 70% of the rat embryos exposed (Brent and Gorson, 1972). An 80% mortality was produced in early mouse embryos after doses of 200 rads (Russell and Russell, 1952), and increased mortality has been reported at doses below 10 rads (Rugh and Grupp, 1961). Comparable doses given at later times during gestation are less effective in causing death.

Embryos surviving exposures given early in gestation are not grossly abnormal. Intrauterine growth retardation occurs only in embryos irradiated after implantation.

There are no quantitative data for humans on radiation-induced prenatal death. In the human female, implantation occurs within about 10 days after fertilization, and major organ formation in the embryo begins at approximately the second week. The lack of human information is related to the high percentage of zygotes (Saxen and Rapola, 1969, estimate 20%) that are lost early and are unnoticed.

Prenatal death occurs consistently after doses of about 50 to 100 rads and more, with a reduced incidence being produced by lower doses. Brent and Gorson (1972) estimated the acute minimal lethal dose is approximately 9.5 rads and median lethal dose as approximately 67 to 95 rads for the human embryo on the first day after conception.

The highest incidence of developmental abnormalities is produced by irradiation during organogenesis. Susceptibility to prenatal death decreases during organogenesis, but neonatal mortality increases, presumably due to an increased incidence of the types of abnormalities that are lethal as the fetus approaches term.

Extensive animal data are available defining the critical period and exposure conditions for inducing a wide variety of developmental abnormalities (see, for example, Russelland Russell, 1952; Brent and Gorson, 1972; Rugh, 1973). The majority of experiments have used doses (in the range of 100 to 200 rads) selected to give significant numbers of abnormalities with maximal survival to term. However, specific developmental defects have been produced by doses as small as 10 to 20 rads. Most gross abnormalities, however, have followed doses of 50 rads or more (BEIR Report, 1972).

Most defects produced involve the nervous system (UNSCEAR, 1969; Rugh, 1973). This is attributed to the high radiosensitivity of neuroblasts during their transitional stage, their abundance in the developing organism, and their persistence throughout gestation. (In man, neuroblasts first appear at 18 days after conception, and are present in the fetus through gestation and in the newborn for several weeks.) In general, gross anomalies of the nervous system are produced by irradiation during organogenesis, when the neuroblasts are concentrated, whereas more subtle effects are produced at later fetal stages, when these cells are more diffuse. The latter defects tend to be more functional than structural and may not be clinically apparent until after birth. Growth retardation, apparent at birth and even persisting into adulthood, may accompany these changes.

Studies on atom bomb survivors and irradiated patients show the main effects of prenatal irradiation on humans development to be impairment of growth, microcephaly, and mental retardation (BEIR Report, 1972; Brent and Gorson, 1972). The incidence of microcephaly was increased in children born to women who received doses as low as 10 to 19 rads at Hiroshima before the seventeenth week of pregnancy (Miller and Blot, 1972). The incidence of this defect increased with dose and was often accompanied by mental retardation. At Nagasaki no effect was observed until maternal doses of 150 rads or greater. Mental retardation generally increased significantly at doses greater than 50 rads at Hiroshima and greater than 200 rads at Nagasaki (Blot and Miller, 1973).

The differences between the cities are attributed to differences in the quality of radiation: gamma rays plus neutrons at Hiroshima and mainly gamma rays at Nagasaki. The greater environmental disturbance at Hiroshima is also a factor. No evidence of delayed skeletal maturation attributable to the atom bomb radiations has been found (Russell et al., 1973).

The defects found in children born to mothers irradiated for medical reasons are similar to those found in children of the survivors: microcephaly, with concomitant mental retardation and general impairment of body growth, as well as defects of the eye. The reports generally predominate in the early literature, however, making dose estimation difficult (BEIR Report, 1972; Brent and Gorson, 1972).

Therapeutic abortions have been induced with a 96% efficiency in women irradiated primarily during the first trimester with doses of approximately 370 rads to the pelvic region (Rubin and Casarett, 1968). The majority of cases aborted between 19 and 35 days after exposure, with death of the embryo being attributed to direct radiation it received.

Irradiation during the fetal stage causes cellular damage and loss (which can alter the structure and function of adult organs and tissues), associated functional defects (which may become apparent only later in life), and reduced growth (Rubin and Casarett, 1968; Brent and Gorson, 1972). Exposure during late pregnancy usually gives the greatest permanent growth retardation and the smallest rate of neonatal death and developmental malformation (except those defects produced in late-developing organs).

No effects in humans other than the defects in growth and development discussed previously have been attributed to fetal exposure. The susceptibility of the fetus to killing by radiation approaches that of the adult (NCRP Report No. 42, 1974). The median lethal dose has been estimated to be on the order of 285 to 380 rads (Brent and Gorson, 1972).

F5.2 EFFECTS OF INTERNAL EXPOSURE

Radionuclides ingested by the mother may cross the placental barrier and enter the embryo or fetus. The probability of producing damage depends on the total dose to the fetus or a particular fetal tissue, the dose rate and its variation over time, and the stages of gestation when irradiated (Brent and Gorson, 1972).

Individual materials vary widely in their ability to cross the placenta. Specialized mechanisms control the transfer of essential metabolites and restrict the passage of other substances on the basis of configuration and/or charge. Most simple ions cross the placenta by simple diffusion, but other (e.g., calcium, iron, and phosphate) are found at higher concentration in fetal than maternal serum, a fact that suggests active transport. The uptake of essential elements often shows age-dependent differences during development. For example, iodide is taken up readily and at a continuously increasing rate by the fetal thyroid from the tenth week of gestation to term (Brent and Gorson, 1972). Calcium uptake is relatively constant until the final trimester of gestation, during which it increases markedly because of fetal calcification (Mays and Lloyd, 1966).

In general, the transfer of a nuclide to the fetus correlates with its availability in the maternal bloodstream. Nuclides with a high affinity for maternal serum proteins, cells, and tissues are generally transferred poorly, although this may vary significantly with the chemical form; for example, plutonium-239 in the monomeric form shows higher transfer to fetal tissues than does the polymeric form (Sikov and Mahlum, 1972). Studies of the uptake and distribution of a number of radionuclides in the developing organism have been published (Sikov and Mahlum, 1969).

The inadvertent administration of therapeutic iodine-131 levels to pregnant women, has caused fetal damage with one case showing complete thyroid destruction and a marked arrest of brain development (Sternberg, 1970). With these exceptions, effects on the unborn of radionuclides ingested by the mother have been reported only in experimental animals. The doses have been high, and the overall response corresponds generally with that seen after a brief external exposure.

For example, intravenous strontium-90 given on day 2 after conception gave 58% mortality and 18% gross defects. On day 10, during organogenesis, the same dose (382 microcuries) gave 6% mortality and 48% gross defects (Hopkins, 1972). The average absorbed dose to the embryo declined from about 12 rads per day shortly after injection to about 0.3 rad per day after 3 days. The dose increased to 23 rads per day during calcification. In general, only relatively small amounts of the injected material reached the embryo, so that after oral ingestion, even less of the isotope would be available for fetal assimilation.

There are no other studies of the effect on the embryo or fetus of a specific quantity of a radionuclide as a function of time since conception. Specific defects can be induced during critical periods (Sikov and Mahlum, 1969; Sikov and Lofstrom, 1962; Finkel and Biskis, 1969).

Plutonium-239 given to pregnant rats may kill embryos but does not cause defects (Sikov and Mahlum, 1972a). Ten microcuries per kilogram given nine days after conception killed many embryos within 3 to 5 days. At 15 and at 19 days after conception no fetuses were killed, even by doses (150 microcuries per kilogram) sufficient to kill the mother. The effect at 9 days was attributed to plutonium incorporation into fetal membranes and, to a lesser extent, into the placenta, which produced alterations in their function. Similar results have been obtained with other transuranic elements (Ovcharenko, 1972), although americium-241 and curium-244 appear to be much less efficient than plutonium-239 in causing prenatal death (Sikov and Mahlum, 1975).

Limited data are available comparing the prenatal effects produced by varying levels of radionuclides. Female rats have been maintained at equilibrium levels of tritium that gave approximately 3, 15 and 30 rads per day total-body irradiation to the embryo and fetus throughout gestation (Cahill and Yuile, 1969). The 15 and 30 rads per day levels produced measurable effects, including growth retardation, gonadal changes in both sexes,

and evidence of microcephaly. No damage was detected following exposure to 3 rads per day. Similarly, phosphorus-32 administered to pregnant rats at 14 days of gestation has produced marked changes in skeletal development after a 2-millicurie dose, less severe damage after 1 millicurie, and no effects after a dose of 0.6 millicurie (Sikov and Lofstrom, 1962). These results show that a specific minimum dose must be administered to an organ during its critical period for development before defects are produced. This is in agreement with results obtained from external exposures and suggests a nonlinear dose-response relationship.

The transfer of iron-59 and iodine-131 to the human fetus has been observed directly after intravenous administration to the mother and prior to therapeutic abortion (Dyer and Brill, 1969). Fetal doses, normalized to the number of millicuries of radionuclide that were transferred from the mother, have been calculated from these data. These calculations deal with the gestation stages from 9 to 22 weeks. For iron-59 the total fetal dose is about 38 rads per millicurie and the critical fetal organ is the liver, which receives a dose of 331 to 536 rads per millicurie. For iodine-131, the total fetal dose varies from about 0.8 to 3 rads per millicurie, and the fetal thyroid dose varies from 715 to 5900 rads per millicurie.

The relationship between fetal dose and quantity of radioactivity ingested is needed for assessing prenatal hazard from radionuclides. The above values for iron-59 and iodine-131 are directly applicable in this context. The case of iodine is particularly important because of the efficient transfer of this element to the blood after either inhalation or ingestion. However, available data are too limited for meaningful estimates to be made for most radionuclides.

F5.3 EFFECTS OF DOSE PROTRACTION

No embryopathologic effects have been observed in animals exposed *in utero* continuously throughout gestation at dose rates less than about 1 rad per day (BEIR Report, 1972). In general, lower dose rates gave fewer defects for a given dose (Sikov and Lofstrom, 1962a), in keeping with the results obtained through internal exposure by radionuclides. However, a variety of developmental defects can be produced by divided exposures delivered during gestation at high dose rate.

F5.4 SUMMARY AND CONCLUSIONS

Irradiation of the developing organism causes a variety of effects on growth and development. Many of these endpoints are not sharply defined. This fact and the changes in radiosensitivity during prenatal life make it extremely difficult to establish dose-response relationships. However, the available information, reviewed above, suggests that certain conclusions can be drawn.

Table VI F-8 gives the approximate minimal and median lethal doses for human fetuses at various times after conception. These doses were estimated by Brent and Gorson (1972).

Dose-response curves for the specified time intervals during the first trimester of gestation are given in Fig. VI F-12. A 1% incidence of mortality has been assumed for the minimum lethal doses specified. Throughout the first trimester, the curves change in accord with the changing radiosensitivity, which is greatest before implantation.

The mortality incidences predicted in Fig. VI F-12 are probably conservative with regard to the exposure conditions predicted for the reference accident. Human pregnancies have been terminated with an efficiency greater than 95% by *in utero* doses on the order of 370 rads given during the first trimester (Rubin and Casarett, 1968). However, in the atom bomb survivors, there was no significant increase in the termination of pregnancy among women exposed during the first trimester (Kato, 1973). Although the number of cases reported was small, probably reflecting incomplete reporting, prenatal mortality was the major effect in mothers with obvious symptoms of radiation sickness (Warren, 1969). Furthermore, the protracted nature of the predicted dose buildup pattern should lessen the degree of effect. Reductions in dose rate generally significantly reduce the incidence of embryopathologic damage (Sikov and Lofstrom, 1962a; Brent and Gorson, 1972; Rugh, 1973). However, available data are inadequate for quantitative assessments of the effect of dose protraction on lethality.

The susceptibility of the fetus to radiation death approaches that of the adult (Brent and Gorson, 1972; NCRP Report No. 42, 1974). Thus, curve A in Fig. VI F-1 (minimal

treatment) should be used to describe the mortality incidence in children irradiated in utero during the second and third trimesters.

The effect of irradiation on human growth and development can be estimated from data on the frequency of reduced head circumference in atom bomb survivors exposed in utero (Miller and Blot, 1972). The incidence of this abnormality at age 10 after exposure increased progressively with dose among those whose mothers were exposed before the eighteenth week of pregnancy. These children were exposed to a sizeable neutron component in addition to gamma radiation and were subjected to greater environmental disturbance than were corresponding children in Nagasaki. In Nagasaki, where gamma radiation predominated, no effect was observed for maternal doses lower than 150 rads. However, in both cities, at maternal doses of 150 rads or more, a reduced head circumference was usually accompanied by mental retardation.

Combining the data from Hiroshima and Nagasaki for all individuals exposed between 0 and 17 weeks of gestation (Miller and Blot, 1972) gives values shown in Table VI F-9.

These data are plotted in Fig. VI F-13. The curve bends at higher doses, presumably partly because of the small numbers involved, and partly because the examination time was 10 years after exposure, so that some individuals were not counted.

The use of the combined data from Hiroshima and Nagasaki helps to dilute the effects of neutrons and the environmental disturbances that prevailed in Hiroshima. However, the curve is conservative with regard to the dose conditions projected for the hypothetical accident because of the predominant weight of the Hiroshima data. Fig. VI F-13 has been extrapolated to a 1% incidence at a maternal dose of 10 rads. This extrapolation is based on the fact that developmental defects have been reported in experimental animals after doses on the order of 10 to 20 rads delivered during critical stages in organogenesis (BEIR Report, 1972).

The possibility of damage to the fetal thyroid after the uptake of radioiodines is of major concern under the hypothetical accident conditions. Iodide is taken up readily by the fetal thyroid from about the tenth week onward throughout gestation (Brent and Gorson, 1972). Directly measured values in the human fetus during the period from 9 to 22 weeks indicate a fetal thyroid dose of 715 to 5900 rads per millicurie of iodine-131 administered intravenously to the mother (Dyer and Brill, 1969).

Although there are no human data showing an increased radiosensitivity of the fetal thyroid on a per rad basis, animal data suggest that thyroid radiosensitivity changes markedly during development. For example, Sikov (1969a) reported that the radiosensitivity of the fetal and the adult thyroid in the rat differed by a factor of approximately 18. The endpoint used was a 50% reduction, compared with control levels, in the uptake of tracer iodine-131 given 4 months after birth to animals that had previously been exposed to graded damaging levels of iodine-131 late in gestation. For this criterion, radiation dose values of 18,000 and 970 rads were obtained for the adult and fetal thyroids, respectively, for the initial iodine-131 exposures to produce the effect. The endpoint was interpreted as indicating radiation-induced hypothyroidism. The data are limited, however -- particularly with regard to the possible influence of maternal factors on the fetus. Additional animal data reviewed by Sikov (1969a) give qualitative support to an increased radiosensitivity for the fetal thyroid but are inadequate for quantitative dose estimates.

If the limited rat data apply to humans, the most pessimistic interpretation would be that the fetal thyroid is 18 times more radiosensitive on a per rad basis than the adult thyroid. Given the paucity of actual human fetal data, one could also simply assume equal radiosensitivity of the thyroid from fetal to adult life.

F6 MODIFICATION OF RESPONSE

F6.1 EFFECTS OF VARIOUS EXPOSURE REGIMES

F5.1.1 Protracted Exposure

The preceding reviews of the available information on acute effects in most cases include the effects of dose protraction on a particular organ or system. It should be noted, however, that the reviews generally compared the effects produced by different regimes: (1) doses delivered in a single brief exposure; (2) doses protracted over various intervals; and (3) doses delivered by intermittent exposures (dose fractionation).

More pertinent to the exposure situation considered in this report is a changing rate of exposure where the initial component is delivered at a high dose rate while the remainder is gradually more and more protracted. There is a report on large animals exposed in this general manner. Page and Still (1972) have approximated this situation in the mode of exposure they designate as "acute followed by a chronic exposure." The experimental animals were sheep and pigs, and on the basis of various indices it was concluded that the response pattern of the sheep most closely resembles that of man. In the experiment, sheep were conditioned acutely with 155 roentgen cobalt-60 gamma irradiation at a rate of 510 roentgens per hour. They were then immediately exposed to predetermined graded doses at 3.8 roentgens per hour. The combined LD_{50} of acute and protracted exposure was 326 roentgens, whereas the LD_{50} of a single acute exposure was 314 roentgens. Since 45 to 50% recovery had been observed in previous experiments during chronic exposure, it was inferred that the acute exposure had suppressed the recovery mechanism that operated in the protracted-exposure study. In the sheep, therefore, the individual responses to both acute and low chronic exposures are additive when the exposures are delivered in that sequence. It is not clear how to apply this information to man without further study.

F6.1.2 Combined External and Internal Exposure

There are very few data on the effects of combined external and internal radiation exposure. Studies of sheep and livestock receiving cobalt-60 external irradiation and yttrium-90 fallout simulant for internal beta irradiation (Sasser et al., 1971) have provided some information, but the different functioning of the gastrointestinal tract in these animals limits the comparability of the results to man. In the case of sheep, it was apparent that the external gamma-radiation LD_{50} of approximately 200 rads (midline tissue dose) was reduced to 145 rads when the internal irradiation damage to the gastrointestinal tract was added. Since the transit time of the human gastrointestinal tract can be markedly reduced by the simple expedient of taking a laxative, it is unlikely that this situation would obtain in man. In man, it appears most likely that the augmentation of external-exposure bone marrow damage by the internal exposure from radionuclides deposited in the thyroid and lungs could produce a further increment of morbidity and mortality due to the hematologic syndrome. It is not possible to give any quantitative estimates of this effect.

F6.2 MODIFICATION OF MORTALITY AND MORBIDITY BY MEDICAL INTERVENTION

It is difficult to quantify the degree to which medical intervention may modify mortality and morbidity from radiation overexposure without being precise about the degree and type of intervention to be considered. For this reason only some generalizations can be offered.

The most dramatic evidence of the effect of medical intervention is provided by the work on bone-marrow transplantation recently reviewed by Thomas and co-workers (1975). In preparing leukemic patients for grafting, a 1000-rad midline tissue dose of total-body irradiation was delivered by opposing cobalt-60 sources at an exposure rate of 5.5 roentgens per minute. One hour before irradiation, the patients were given a barbiturate and an antiemetic. The characteristic irradiation effects were nausea and vomiting toward the end of the 5-hour irradiation period. In the first 48 hours, vomiting and diarrhea ranged from none to moderately severe. After the second day, vomiting was rare, and oral intake of liquid and soft food began. One to four daily liquid stools continued through the fifth day, when the gastrointestinal symptoms subsided.

In the majority of the 70 cases reviewed, bone marrow from an identical twin or a closely matching sibling, administered immediately after the irradiation, grew successfully in the recipient. Although there were many subsequent fatalities due to recurrent leukemia, graft vs. host syndrome, and infection, the classical gastrointestinal syndrome was not observed in these patients. Moreover, the classical changes associated with the hematologic form of the actue radiation syndrome did not materialize.

Given this form of medical intervention, it becomes exceedingly difficult to draw the dose-mortality or dose-morbidity curves as modified by the treatment in the absence of any clearly radiation-induced mortalities. Similarly, the successful treatment of accidental whole-body radiation doses of 600 rads, using isogeneic bone marrow transplantation (E.D. Thomas et al., 1971; Wald, 1975) as well as more conservative therapy in 400- and 300-rad whole-body accidental exposures only provide the basis for shifting the mortality curve above these respective levels.

Another significant series referred to earlier is that of Rider who treated at least 22 Ewing's sarcoma patients with 300-rad whole-body doses (Rider and Hasselback, 1967). The radiation used was cobalt-60 scanned over the patient at a source-to-skin distance of about 100 centimeters. Both sides of the patient were treated, and the dose was estimated at the midplane position; 300 rads were delivered in about 15 minutes.

The clinical management of these patients included a pretreatment antiemetic, hospitalization through the 48-hour prodromal-symptom period, and subsequent discharge for outpatient observation until about 3 weeks after exposure, when the signs of pancytopenia resulted in readmission. In the management of the pancytopenia, the general approach was to utilize barrier nursing; antibiotics for infections on their recognition, not prophylactically; and transfusions of red cells, platelets, and, rarely, white cells when indicated.

In view of the paucity of human data to provide more than minimal mortality points on the dose-response curve, reliance has generally been placed on studies of a variety of lower animals. In particular, data derived from untreated and treated dogs have generally been considered to be reasonably close to the response pattern in man. Reliance has been placed on studies designed at evaluating the extent of improvement in mortality that active treatment can make possible. Such studies as those of Cronkite and Bond (1960) and Perman et al. (1962) are examples (see section F1.2.1 for details). The results suggest that supportive therapy can raise the LD_{50} by a factor of about 1.5.

Supportive measures--including the use of laxatives to accelerate the transit of radionuclides through the gastrointestinal tract, strict reverse-isolation procedures, adequately managed major antibiotic therapy with appropriate microbiological laboratory support, and the ready availability of blood and blood products for transfusion--are available in most large acute medical and surgical hospitals. In particular, the availability of capabilities utilized in managing kidney transplant patients in a useful practical means of assessing the adequacy of hospital capabilities for dealing with acute radiation injury problems.

The utilization of more heroic treatment techniques may increase the LD_{50} even further. For example, bone marrow transplantation, as utilized by E.D. Thomas' leukemia therapy group, may well raise the LD_{50} by a factor of 3 or more. The techniques involved have been reported in the literature (E.D. Thomas and Storb, 1970). The type of medical facility alluded to in the preceding paragraph and the participation of specialists in hematology should suffice to make this procedure available when needed.

Another heroic measure, lung lavage, has been demonstrated to be an effective method for removing radioactive particles from the lungs of beagle dogs in studies at the Lovelace Foundation (Pfleger et al., 1969) and at Battelle-Northwest Laboratories. Multiple lung lavages (Boecker et al., 1974; Silbaugh et al., 1975) have shown that up to 50% of relatively insoluble particles can be removed from the lung, resulting in a substantial reduction in the daily dose rate and the cumulative absorbed dose to the lung. This approach has been effective in the prevention of radiation pneumonitis in beagle dogs that had inhaled substantial lung burdens of cerium-144 in aluminosilicate particles (Muggenburg, et al., 1975). The use of this technique in treating one human patient who had inhaled plutonium-239 successfully reduced the pulmonary burden of this radionuclide (McClellan et al., 1972).

The method of lung lavage used in the experimental studies and in the human plutonium-23 inhalation case was the method of Kylstra (1971). This procedure requires general anesthesia and placement of a double-lumen endotracheal tube with two cuffs into the trachea and one major bronchus. After the cuffs are inflated, the right and the left lungs are functionally separated. The functional residual volume of one lung is then filled with warmed isotonic saline solution. An approximate tidal volume of the same fluid is added to the lung and drained. This procedure is repeated several times. Finally the lung is drained of as much fluid as possible. Only a few hundred milliliters of saline solution remain in the lung at the end of the procedure.

Lung lavage has been used safely as a treatment for chronic obstructive lung diseases in man since the mid-1960s. The potential risk of the procedure is probably in the use of general anesthesia. Only mild and transient physiologic and pathologic changes have been demonstrated experimentally after a lung lavage. Although there are data from only one case of radionuclide removal in man, the safe use of lung lavage in man has been demonstrated in the treatment of chronic obstructive lung disease. No other method is presently available to remove inhaled insoluble particles from the lung. For optimal results, it would be advisable to call on one of the chest physicians who have had active experience with this technique.

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TABLE VI F-1 RADIATION DOSES TO LUNG AND TIME OF DEATH AFTER EXPOSURE FOR DOGS DYING WITH RADIATION PNEUMONITIS AND/OR PULMONARY FIBROSIS AFTER INHALATION OF VARIOUS BETA-EMITTING RADIONUCLIDES

Radionuclide[a]	Time of Death After Exposure (days)		Dose to Lung at Death (rads)	
	Median	Range	Median	Range
Yttrium-90	99	7-903	16,000	9,300-70,000
Yttrium-91	181	113-1011	29,000	8,300-60,000
Cerium-144	211	143-410	57,000	28,000-140,000
Strontium-90	262	159-477	60,000	40,000-95,000

(a) Inhaled in relatively insoluble form.

TABLE VI F-2 DOSE RATES AT VARIOUS DEPTHS IN THE WALLS OF CYLINDERS OF 0.75-cm RADIUS CONTAINING HOMOGENEOUS SOLUTIONS OF VARIOUS RADIONUCLIDES(a)

Radionuclide	Average Energy (MeV)	Dose Rate (μrad/hr)			
		0 cm	0.0175 cm	0.0500 cm	0.0750 cm
Rhodium-106	1.428	34.3	29.7	24.5	21.3
Yttrium-90	0.937	24.4	19.8	15.2	12.5
Phosphorous-32	0.695	18.6	14.3	9.89	7.60
Bismuth-210	0.394	11.0	6.85	3.78	2.38
Tellurium-204	0.243	6.87	3.30	1.24	0.570
Strontium-90	0.196	5.58	2.05	0.490	0.125
Lead-212	0.106	2.97	0.470	0.0373	$\sim 1.5 \times 10^{-3}$
Ruthenium-103	0.065	1.82	0.066	$\sim 8.2 \times 10^{-5}$	$\sim 4.3 \times 10^{-7}$
Promethium-147	0.062	1.79	0.053	$<10^{-5}$	

(a) Data from experiments by M. F. Sullivan, analyzed by Cross (1975a).
Specific activity of solutions: one beta particle per second per gram.

TABLE VI F-3 DOSE EXTRAPOLATION FOR RAT CECUM FOR ACUTE DEATH[a]

Radionuclide	Dose (mCi/kg) 0 cm	0.0175 cm
Ruthenium, Rhodium-106	9.0	9.0
Yttrium-90	13	14
Phosphorous-32	17	19
Bismuth-210	28	39
Tellurium-204	45	81
Strontium-90	55	1.3×10^2
Lead-212	1.0×10^2	5.7×10^2
Ruthenium-103	1.7×10^2	4.1×10^3
Promethium-147	1.7×10^2	5.1×10^3

[a] Based on Ruthenium-106/Rhodium-106 experiments (Cross, 1975a).

TABLE VI F-4 RELATIONSHIP BETWEEN SURFACE AND CRITICAL-DEPTH (0.0175-cm) DOSES IN VARIOUS SEGMENTS OF THE LARGE INTESTINE OF THE RAT[a]

Radionuclide and Tissue	Surface	0.0175-cm Depth[b]
Ruthenium-106/rhodium-106:[c]		
Cecum	4424	3798
Upper large intestine	1003	987
Lower large intestine	1616	1590
Yttrium-91:[d]		
Cecum	3929	3010
Upper large intestine	1287	986
Lower large intestine	2075	1589
Promethium-147:[e]		
Cecum	1.27×10^5	3733
Upper large intestine	5.50×10^5	1615
Lower large intestine	8.86×10^4	2603

[a] Data from experiments by M. F. Sullivan, analyzed by Cross (1975c).

[b] See Table 1 for the correction factors. For yttrium-91, the phosphorus-32 correction factor was used since these radionuclides emit beta particles of similar energy.

[c] LD_{50} = 9 millicuries per kilogram of body weight, 2.7 millicuries per rat.

[d] LD_{50} = 17 millicuries per kilogram of body weight, 5.1 millicuries per rat.

[e] LD_{50} = 5000 millicuries per kilogram of body weight, 1500 millicuries per rat.

TABLE VI F-5 DOSE EXTRAPOLATION FOR DOG COLON FOR ACUTE DEATH[a,b]

Radionuclide	LD_{50} (mCi/kg)			
	0 cm	0.0175 cm	0.0500 cm	0.0750 cm
Ruthenium-106/Rhodium-106	3.5	3.5	3.5	3.5
Yttrium-90	4.9	5.3	5.7	6.0
Phosphorous-32	6.5	7.3	8.7	9.8
Bismuth-210	11	13	28	31
Tellurium-204	17	32	69	1.3×10^2
Strontium-90	22	51	1.8×10^2	6.0×10^2
Lead-212	40	2.2×10^2	2.3×10^3	$\sim 5 \times 10^4$
Ruthenium-103	66	1.6×10^3	$\sim 1 \times 10^6$	$\sim 2 \times 10^8$
Promethium-147	67	2.0×10^3	$>10^7$	

[a] Based on M. F. Sullivan's experiments with ruthenium-106/rhodium-106; data analyzed by Cross (1975a).

[b] Acute death is defined here as death in 10 days.

TABLE VI F-6 DOSE EXTRAPOLATION FOR DOG COLON FOR DELAYED DEATH[a,b]

Radionuclide	LD_{50} (mCi/kg)			
	0 cm	0.0175 cm	0.0500 cm	0.0750 cm
Ruthenium-106/Rhodium-106	2.75	2.75	2.75	2.75
Yttrium-90	3.9	4.1	4.4	4.7
Phosphorous-32	5.1	5.7	6.8	7.7
Bismuth-210	8.6	9.8	22	25
Tellurium-204	14	25	54	1.0×10^2
Strontium-90	17	40	1.4×10^2	4.7×10^2
Lead-212	32	1.7×10^2	1.8×10^3	$\sim 4 \times 10^4$
Ruthenium-103	52	1.2×10^3	$\sim 8 \times 10^5$	$\sim 1 \times 10^8$
Promethium-147	53	1.6×10^3	$>10^7$	

[a] Based on M. F. Sullivan's experiments with ruthenium-106/rhodium-106; data analyzed by Cross (1975a).

[b] Delayed death is defined here as death in 180 days.

TABLE VI F-7 MEDIAN LETHAL DOSES OF RUTHENIUM-106/RHODIUM-106 IN VARIOUS SEGMENTS OF THE DOG INTESTINAL TRACT[a]

Tissue	Acute Mortality[b] Surface Dose (rads)	Acute Mortality[b] Critical Tissue[d] Dose (rads)	Delayed Mortality[c] Critical Tissue[d] Dose (rads)
Ileum	2033 ± 533	1457 ± 381	1145 ± 299
Upper colon	6509 ± 1536	4096 ± 967	3218 ± 760
Midcolon	8135 ± 2584	5120 ± 1626	4023 ± 1278
Lower colon	11,800 ± 8200	7426 ± 5161	5835 ± 4055

[a] Sullivan's data (1975a,b), analyzed by Cross (1975a,d,e).

[b] Mortality in 10 days after ingestion of 3.5 millicuries per kilogram of body weight.

[c] Mortality in 180 days after ingestion of 2.75 millicuries per kilogram of body weight.

[d] Estimated to be 0.050 to 0.075 cm below the mucosal surface.

TABLE VI F-8 APPROXIMATE MINIMAL AND MEDIAN LETHAL DOSE FOR HUMAN FETUSES AT VARIOUS TIMES AFTER CONCEPTION[a]

Time After Conception	Approximate Minimal Lethal Dose[b] (rads)	Approximate Median Lethal Dose[b] (rads)
Day 1	9.5	67 to 95
Day 14	24	133
Day 18	47	143
Day 28	>47	209
Day 50	>95	247
Late fetus to term	--	285 to 380

[a] Data from Brent and Gorson (1972).

[b] Conversion from roentgens to rads was made assuming an f factor of 0.95.

TABLE VI F-9 INCIDENCE OF REDUCED HEAD CIRCUMFERENCE IN HIROSHIMA AND NAGASAKI SURVIVORS EXPOSED BETWEEN 0 AND 17 WEEKS OF GESTATION[a]

Maternal Dose (rads)	Average Maternal Dose (rads)	Combined Hiroshima-Nagasaki Incidence of Reduced Head Circumference	
		Frequency	Percent
10 to 19[b]	15	6/61	10
20 to 29	25	6/29	21
30 to 49	40	9/29	31
50 to 99	75	9/29	31
100 to 149	125	2/6	33
150+		13/22	59

[a] From Miller and Blot (1972).

[b] Incidence occurring below 10 rads could not be attributed to radiation.

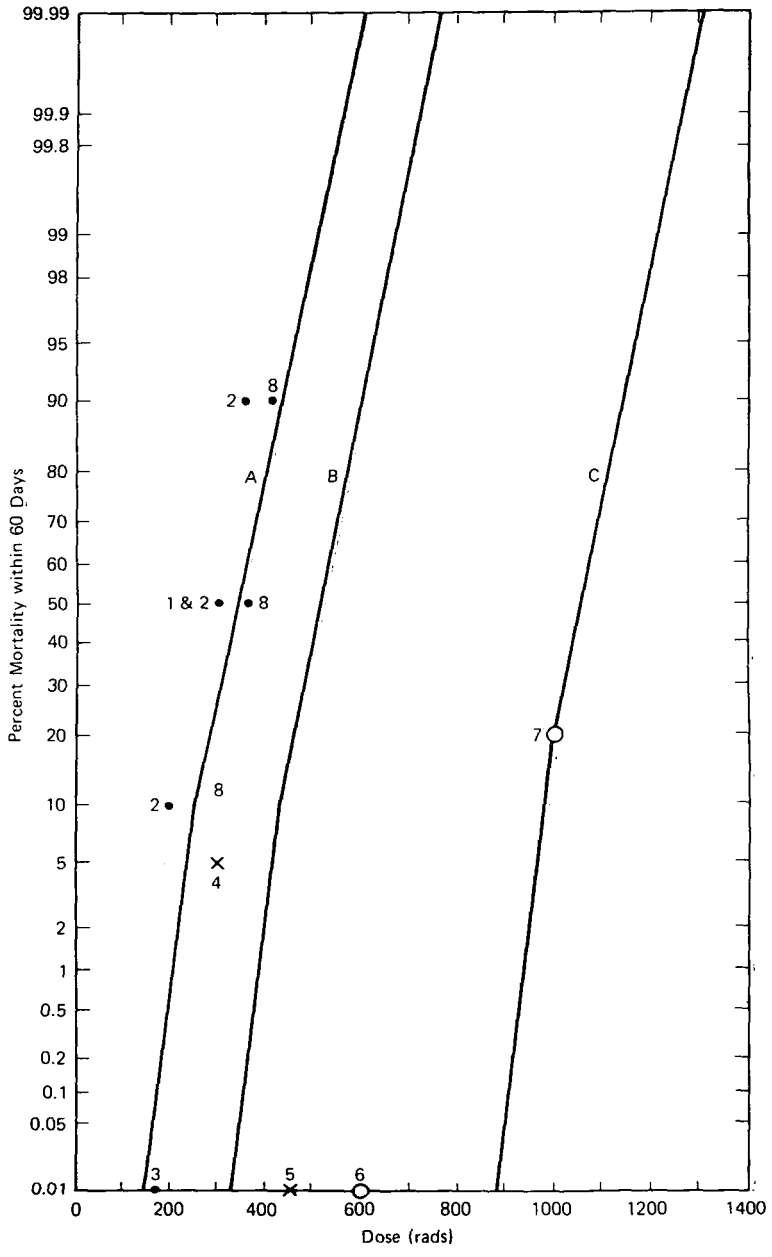

Figure VI F-1 Estimated dose-response curves for 50% mortality in 60 days with minimal treatment (curve A), supportive treatment (curve B), and heroic treatment (curve C). Origin of data points: 1, NCRP Report 42 (converted to rads using factor given in NCRP Report 42); 2, Langhorn (1967, Table 12, estimate for "normal man?"); 3, Marshall Islanders (protracted exposure); 4, radiation therapy series, 22 patients (Rider and Hasselback, 1968); 5, clinical group III accident patients (Thoma and Wald, 1959, with newer cases added); 6, Pittsburgh accelerator accident patient (E.D. Thomas, 1971; Wald, 1975); 7, 37 leukemia patients (E.D. Thomas, 1975); 8, "best estimate" of the Biomedical and Environmental Assessment Group at the Brookhaven National Laboratory.

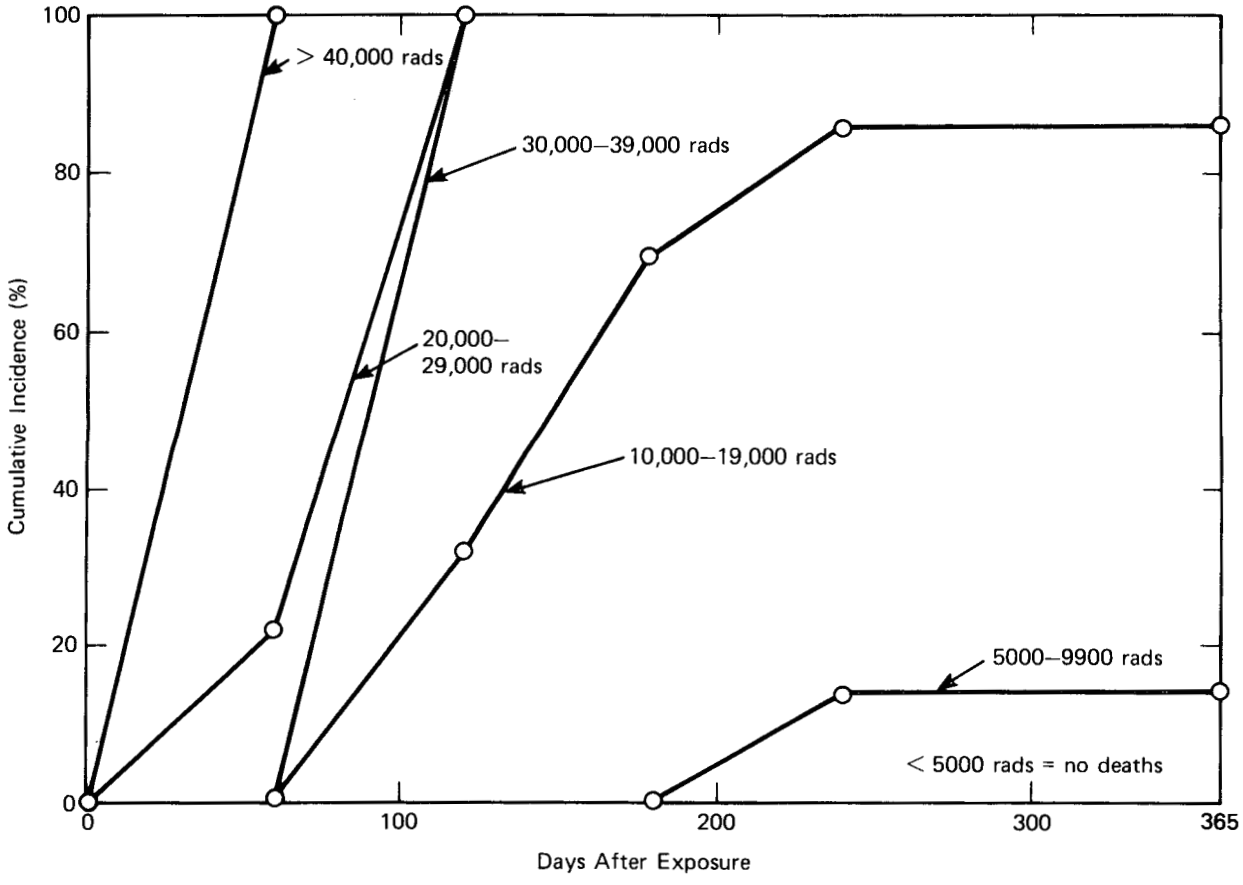

FIGURE VI F-2 Cumulative mortality in dogs from radiation pneumonitis and/or pulmonary fibrosis after inhalation of yttrium-90 in fused-clay particles (doses to lung at 365 days after exposure). After Hahn (1975).

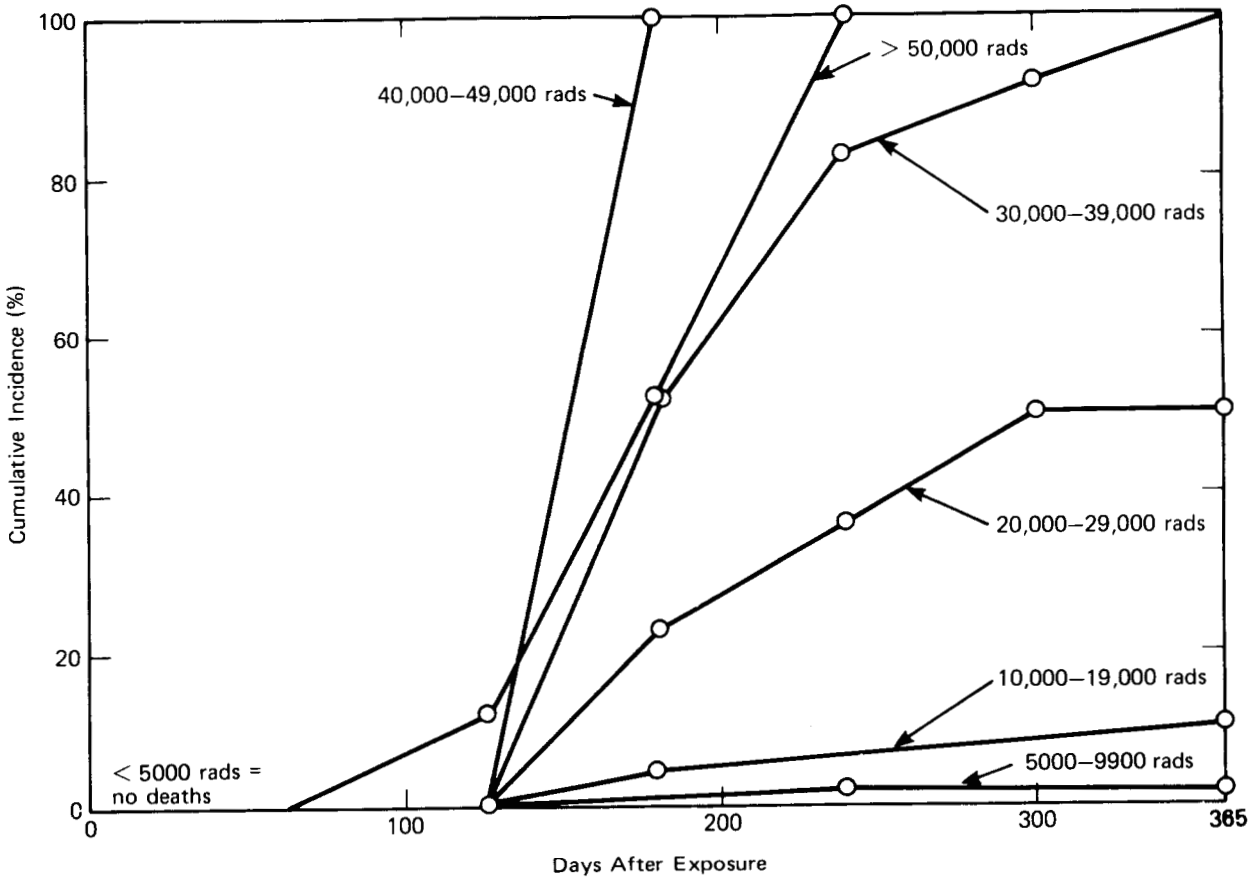

FIGURE VI F-3 Cumulative mortality in days from radiation pneumonitis and/or pulmonary fibrosis after inhalation of yttrium-91 in fused-clay particles (doses to lung at 365 days after exposure). After Hahn (1975).

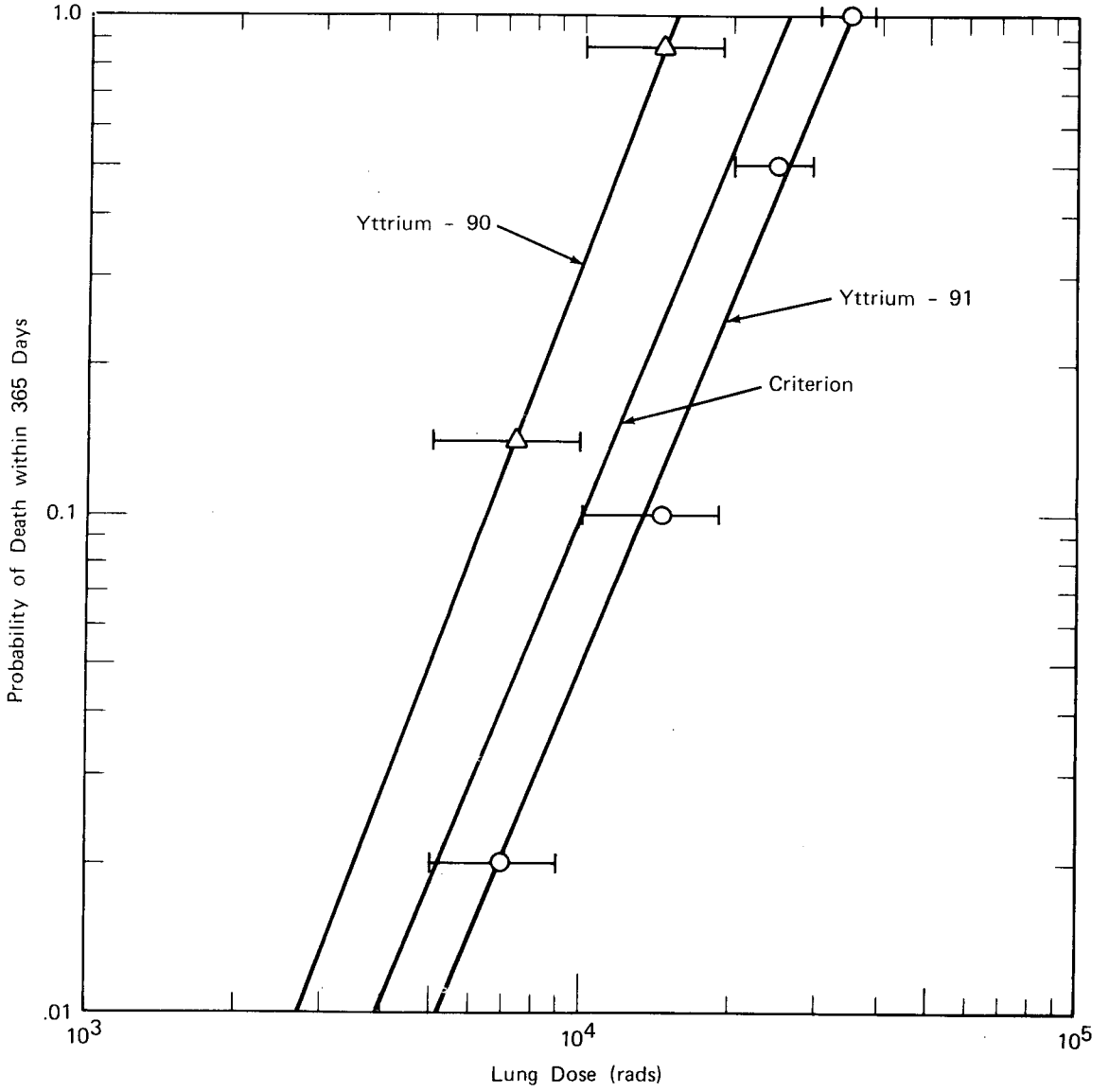

FIGURE VI F-4 Dose-response curves for yttrium-90 and yttrium-91.

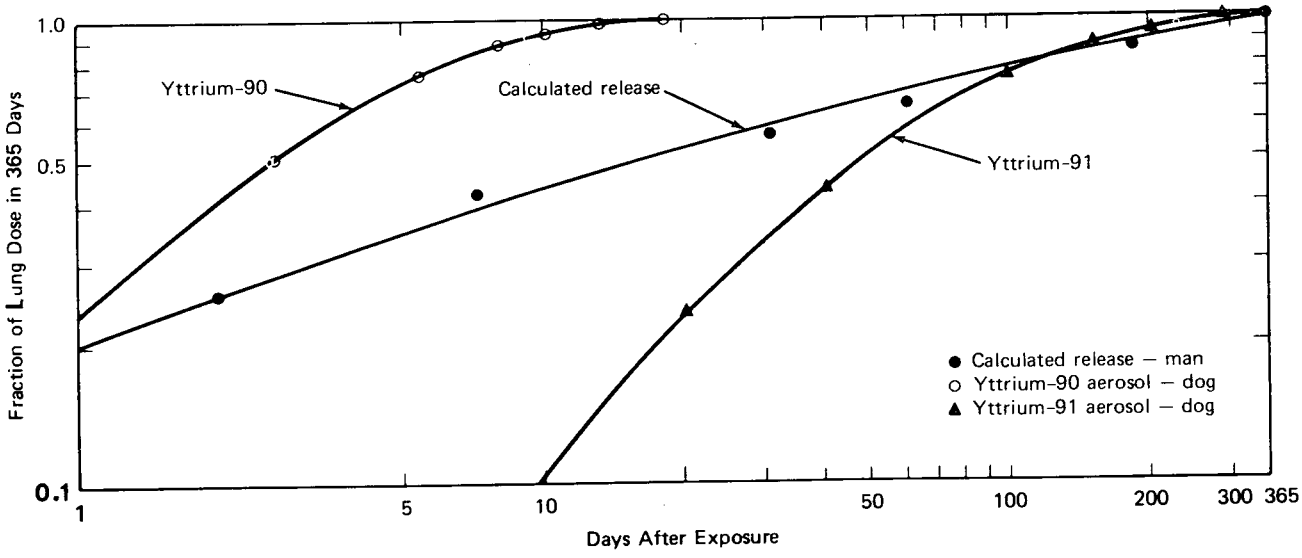

FIGURE VI F-5 Fractions of a 365-day dose to lung accumulated at various times after exposure.

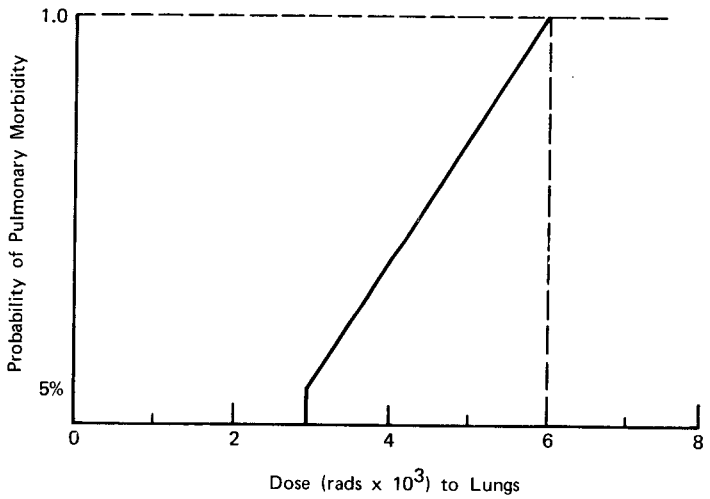

FIGURE VI F-6

Dose morbidity criterion for lung

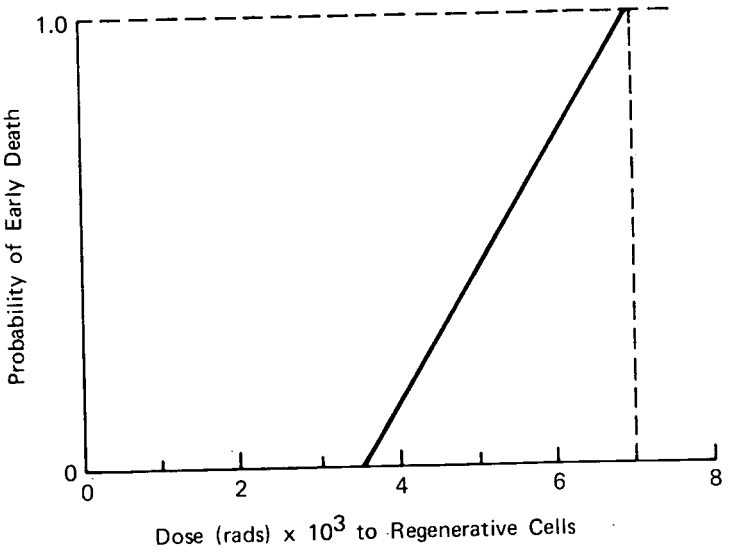

FIGURE VI F-7

Dose-morbidity curve for dogs from ingested ruthenium-106/rhodium-106

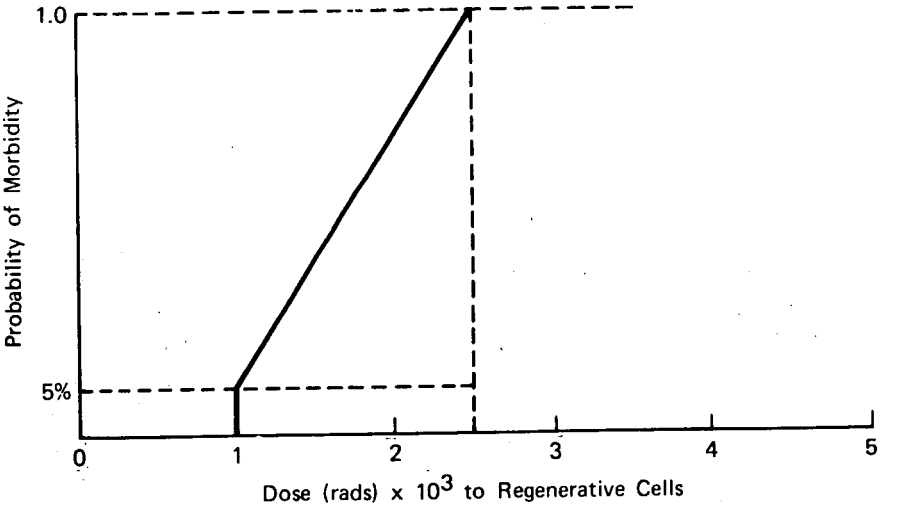

FIGURE VI F-8

Dose-morbidity criterion for the gastro-intestinal tract

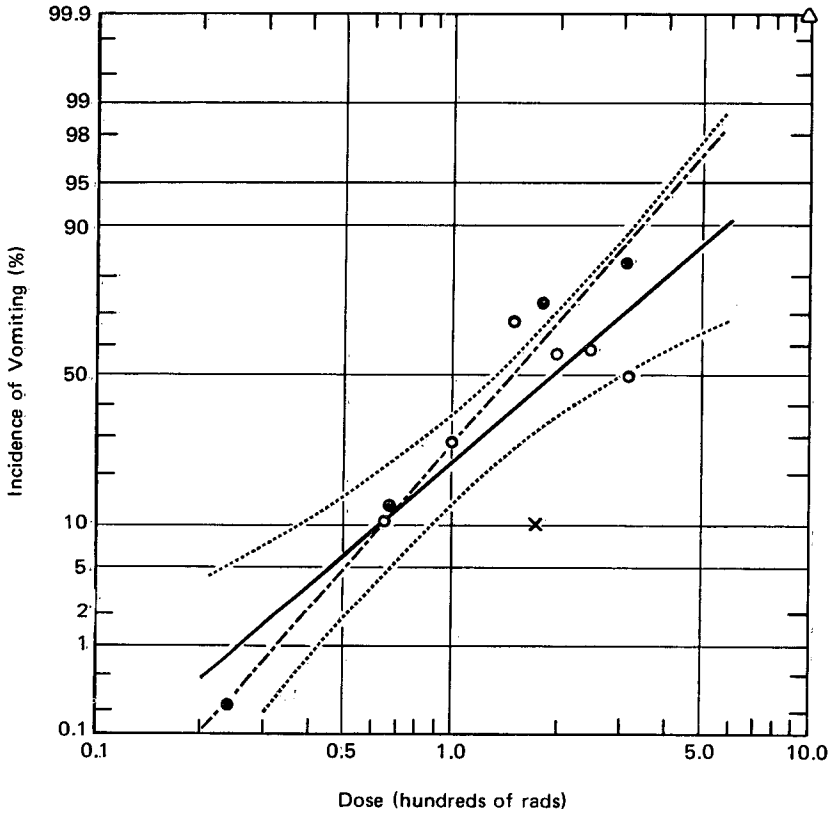

FIGURE VI F-9 Incidence of vomiting within 2 days as a function of dose assuming log-normal distribution of quantal response (open circles, clinical patient data; closed circles, accidental exposure cases; x, Rongelap natives). The dotted arcs represent the 95% confidence level for the clinical patient data. This is Fig. 10 from NAS-NRC Publication 1487 (Langham, 1967). The triangle is the data point for seven hematologic malignancy patients treated with 1000-rad whole-body irradiation (E. D. Thomas et al., 1971 b).

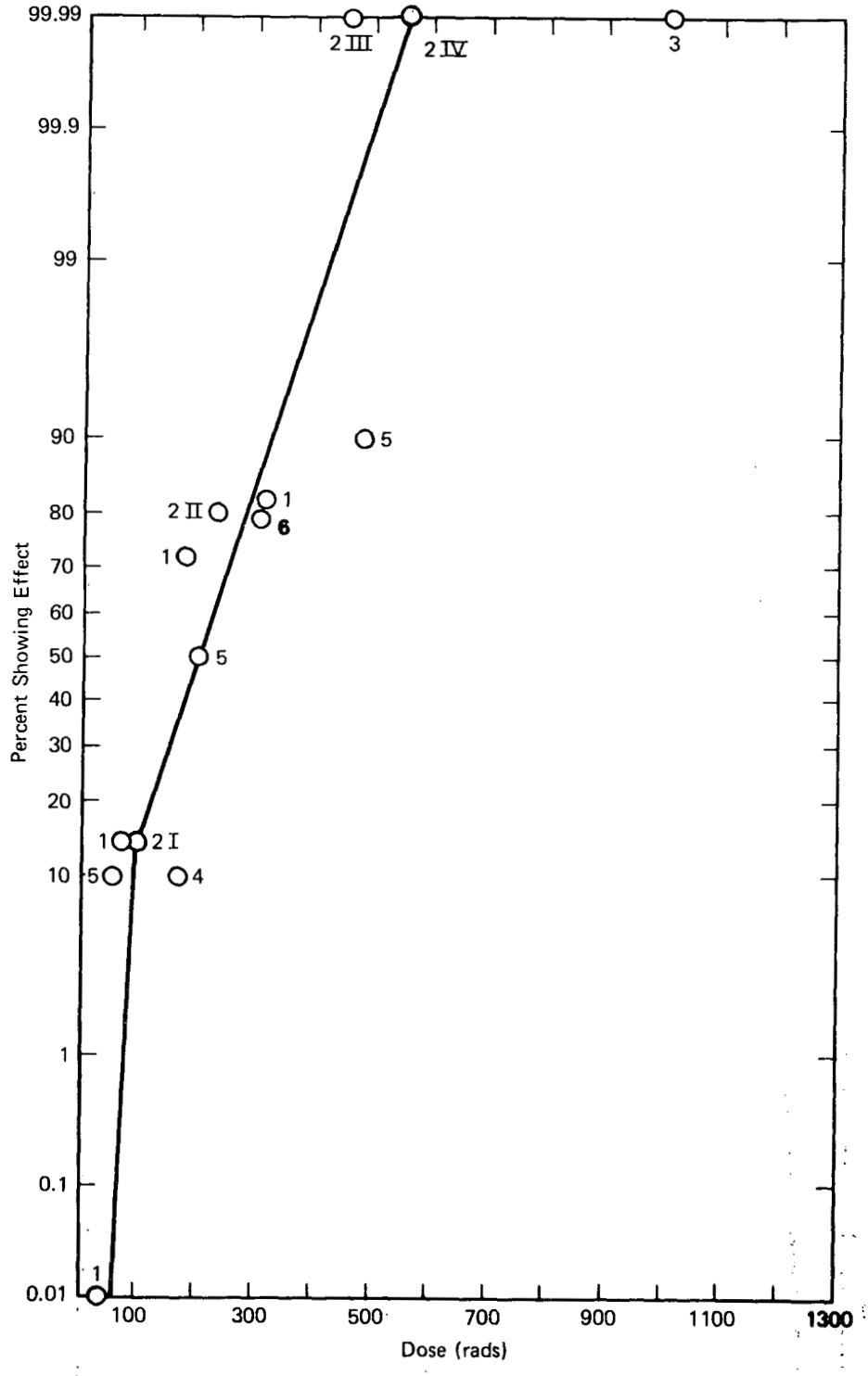

FIGURE VI F-10 Prodromal vomiting within 48 hours of exposure without pretreatment. Origin of data points: 1, Langham (1967), accidental exposure cases (see Fig. VI F-9); 2, accidental exposure cases (Thoma and Wald, 1959, updated); 3, therapy patients (E. D. Thomas, 1971); 4, Rongelap fallout cases, protracted 50-hour exposure (Langham, 1967); 5, half the difference between normal arithmetical and log-normal values given in Langham (1967); 6, Toronto therapy cases (11/14) with Gravol pretreatment. The numerals I, II, III, and IV identify the clinical categories of the acute radiation syndrome.

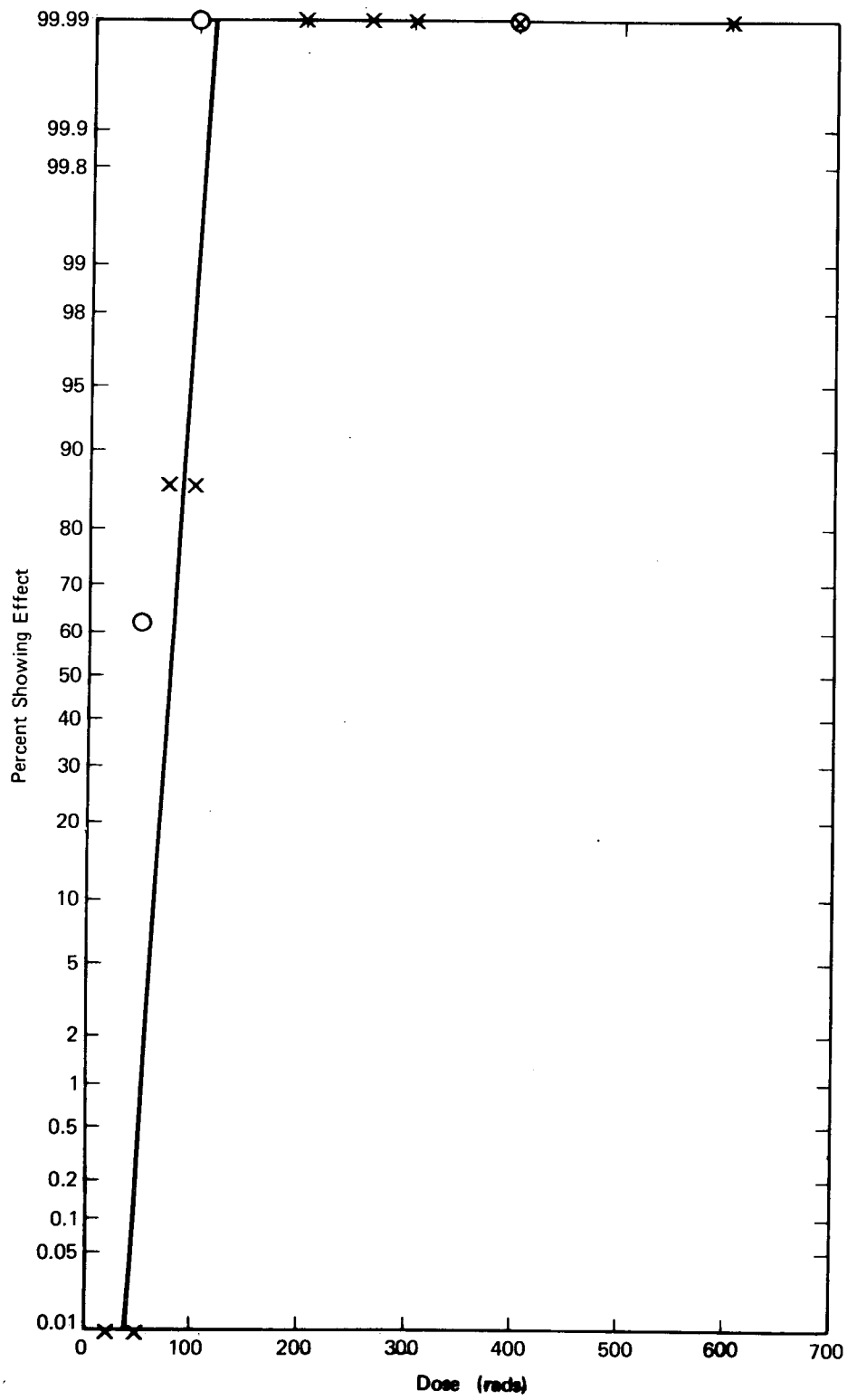

FIGURE VI F-11 Transient sterility (azoospermia) in males within 210 days after exposure. The circles are data from Thorslund and Paulsen (1972); the crosses are data from Rowley et al. (1974, 1975).

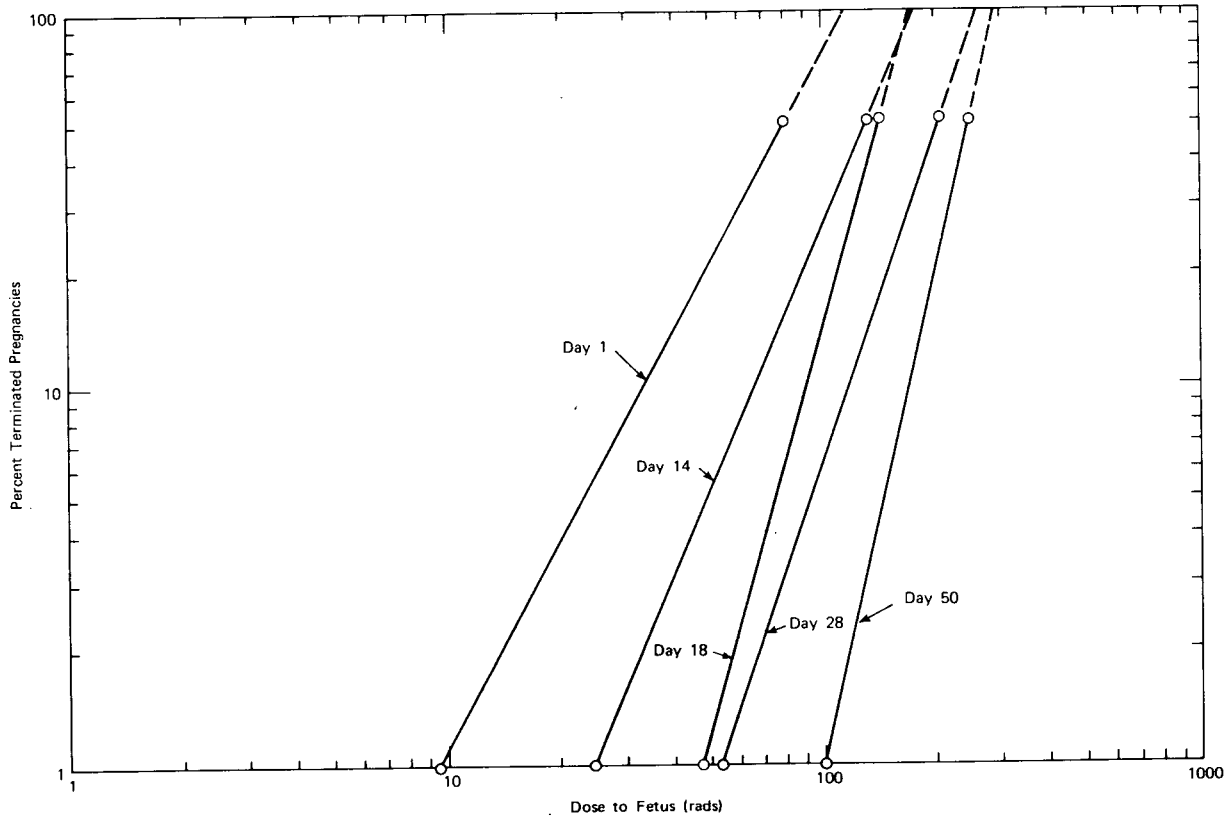

FIGURE VI F-12 Percent termination of pregnancy following brief external exposure during the first trimester of gestation.

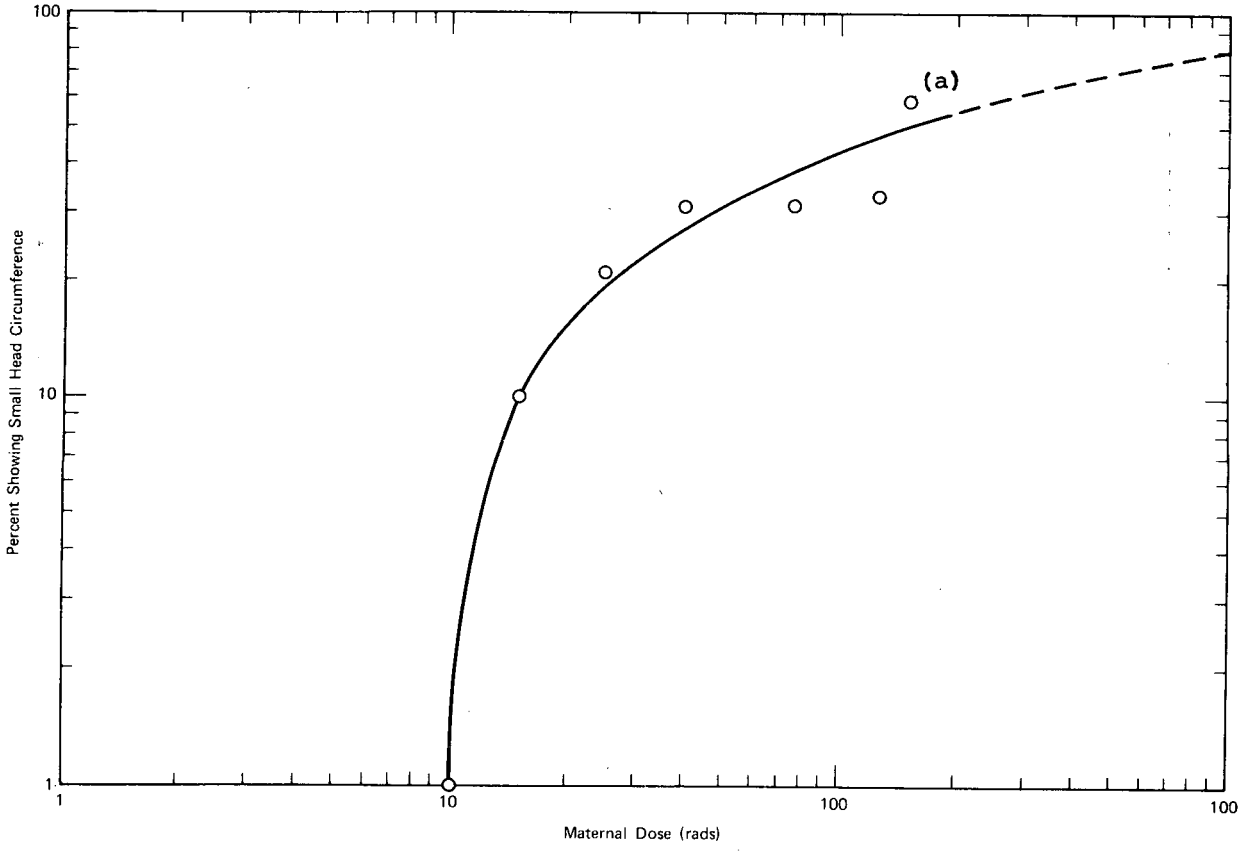

FIGURE VI F-13 Incidence of small head circumference versus dose for individuals exposed between the 0 and 17 weeks of gestation in Hiroshima and Nagasaki.

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

Appendix G

Latent Somatic Effects

G1 INTRODUCTION

The late somatic effects of radioactivity accidentally released from a light-water reactor are difficult to quantitate. Without comparable accident information, certain assumptions are necessary in using available human data accumulated over the past 70 years. The body of information on the assessment of radiation risks to humans has been extensively summarized in several recent reports, such as those of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1972), the National Academy of Sciences (1972,[1] 1974), the National Council on Radiation Protection and Measurements (NCRP, 1971, 1975), the International Commission on Radiological Protection (ICRP, 1966, 1968, 1969), Hutchinson (1972), Mole (1971), Mays et al. (1973), Storer (1969), Storer and Bond (1972), and Upton (1967, 1968). Though these sources review and assess much of the available data, additional specific data have been utilized for this appendix. The assessment of possible risks to a population exposed to the release of effluents from a postulated accident will pertain to those individuals who have survived the acute (early) radiation effects resulting from immersion in the radioactive cloud. Thus, this appendix is restricted entirely to an evaluation of health consequences that may appear months or, more likely, years after the postulated accident.

The spatial and temporal nature of the radiation dose commitment requires that adequate attention be paid to several distinct sources of radiation exposure. As discussed in other sections, the "source term" for radiation dose consists primarily of four components, the sum of which is the total dose commitment to individuals and to the population; (1) the short-term, predominantly external dose, resulting from the passing cloud containing radionuclides; (2) the dose resulting from early inhalation and ingestion of both short- and long-lived radionuclides from the radioactive cloud; (3) the long-term radiation dose commitment associated with later uptake and incorporation of long-lived radionuclides entering the body in food and water; and (4) the dose arising from residual ground surface contamination (additional total body dose). The fractional contributions of each released radionuclide can be computed in terms of these four dose components. They are shown schematically in Figs. VI G-1 and VI G-2.

Almost all radionuclides released as fission or activation products emit beta and gamma radiation, with an assumed quality factor and relative effectiveness of unity. Alpha-emitting radionuclides, which contribute a minute fraction of the total dose commitment and include the transuranic elements, are assumed here to have a quality factor and relative biological effectiveness of 10 (ICRP, 1969).

G1.1 RISK ESTIMATION

Several radiobiologic principles will be used here in assessing risk from radiation exposure. Over a wide range of exposures, a dose-rate effectiveness factor appears to influence the risk for late effects (NCRP, 1975). Thus, for doses below lethal levels, the same dose received acutely ($\leq$1 hour) is generally more damaging than is a similar, but protracted, dose. At exceedingly high doses, including those in the lethal range, there is so much cell destruction that a crude cancer incidence rate would appear to be lower than that for intermediate and lower dose rates from acute and chronic irradiation (Rowland et al., 1971). These considerations were not included in numerical estimates of cancer risk based on linear extrapolations used in recommendations for exposure standards and limits (NAS, 1974).

[1]Reference is made here to the report of the Advisory Committee on the Biological Effects of Ionizing Radiations of the National Academy of Sciences-National Research Council; hereinafter referred to as the BEIR Report.

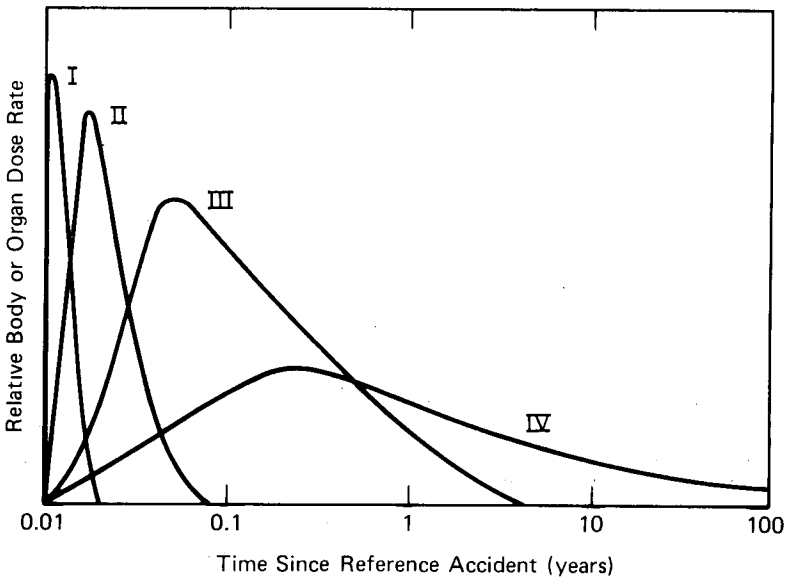

FIGURE VI G-1 Dose rates from unit exposure of individuals in the hypothetical reference accident. Curve I, acute external dose from the passing cloud; curve II,subacute body and organ dose from short-lived radionuclides; curve III, body and organ dose from long-lived radionuclides; curve IV, internal and external body and organ doses from residual environmental contamination by long-lived radionuclides.

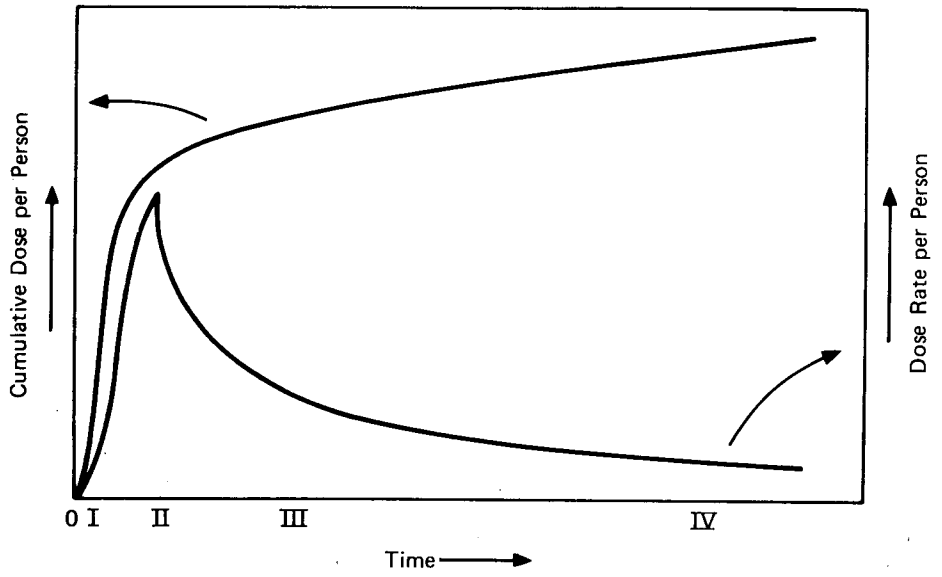

FIGURE VI G-2 Individual cumulative doses and rates consequent to the exposure patterns described in Fig. VI G-1

In many radiations carcinogenesis studies in experimental animals (Sanders et al., 1973), particularly with low-LET radiation (UN, 1972), there was a nonlinear relation between dose and effect.[1] In fact, there is little to indicate measurable risks to critical tissues ranging up to several hundred millirem per day (Sanders et al., 1973). This difficulty was recognized by NCRP (1975):

> "Risk estimates for radiologic cancers at low dose rates derived on the basis of linear (proportional) extrapolations from the rising portions of the dose incidence curves at high doses and high dose rates as described and discussed in subsequent sections of this report, cannot be expected to provide realistic estimates of the actual risks from low level, low LET radiations, and have such a high probability of overestimating the actual risks as to be of only marginal value, if any, for purposes of realistic risk-benefit evaluation."

NCRP further cautions as follows:

> "The linear dose-effect hypothesis has been coming into frequent use in analyses in which population exposures are expressed in the form of person-rem, including doses of 1 mrem/year or less to population groups and doses to individual organs, with linear extrapolation to damage estimates through the use of the NAS-BEIR committee report values. The indications of a significant dose rate influence on radiation effects would make completely inappropriate the current practice of summing of doses at all levels of dose and dose rate in the form of total person-rem for purposes of calculating risks to the population on the basis of extrapolation of risk estimates derived from data at high doses and dose rates.
>
> "The NCRP wishes to caution governmental policy making agencies of the unreasonableness of interpreting or assuming "upper limit" estimates of carcinogenic risks at low radiation levels, derived from linear extrapolation from data obtained at high doses and dose rates as actual risks, and of basing unduly restrictive policies on such an interpretation or assumption. The NCRP has always endeavored to ensure public awareness of the hazards of ionizing radiation but it has equally determined to insure that such hazards are not greatly overestimated. Undue concern, as well as carelessness with regard to radiation hazards, is considered detrimental to the public interest."

This general concern regarding extrapolations is further amplified by the BEIR Report (1972, p. 88), in which it is noted:

> "At background radiation levels, ionizing events in individual mammalian cell nuclei occur at a rate of much less than one per day, whereas at the higher dose rates mentioned, ionization events occur in cells at a frequency of the order of 2600/sec. This enormous difference may have important implications with respect to the production of radiation damage within cells and its repair at the molecular level. On the basis of the likelihood of such repair, the risk of cancer induction at low doses and low dose rates might be expected to be appreciably smaller per unit dose than at high doses and high dose rates, as has been observed to be the case in certain radiation induced tumors in experimental animals. Hence expectations based on linear extrapolations from the known effects in man from larger doses delivered at high dose rates and the range of the rising dose incidence relationship may well overestimate the risks of low-LET radiation at low dose rates and therefore may be regarded as upper limits of risk for low level, low-LET irradiation. The lower limit, depending on the shape of the dose incidence curve for low-LET radiation and the efficiency of repair processes in counteracting carcinogenic effects could be appreciably smaller (the possibility of zero is not excluded by the data)."

A further caveat was entered in the UNSCEAR overview (1972, Annex H) of radiation carcinogenesis in man. UNSCEAR noted that it had reviewed the evidence available on derived estimates for some malignancies:

> "The committee wishes to reemphasize that all the estimates apply to short term exposures at high dose rates and as discussed in annex G, are likely to be overestimates of the risks per unit dose that may result from protracted irradiation at low dose rates of low-LET radiation. The estimates given in this annex are all subject to revision, both because the total risk of any malignancy can only

[1] Linear energy transfer (LET) is the average amount of energy lost per unit of particle-track length and is usually expressed in thousands of electron volts per micron. Low-LET radiation includes beta particles and gamma rays.

be assessed by observing a cohort of irradiated people until extinction, and in no case has there been an opportunity for such prolonged observation yet, but also because of the basic uncertainties of the data."

The UNSCEAR report uses the linear hypothesis and offers cancer death risk estimations that range from about 0.6 to 10 per 10^6 persons per year per rad, subject to this caution regarding their use.

Despite the inadequacies of available data and the difficulties of extrapolating experimental information to possible human exposures, a quantitative estimation of radiation risk is essential. The application of scientific judgment is needed to bridge the gaps in existing knowledge. In many instances, such universally acceptable, interpretations are amenable to future revisions as further information becomes available. The NCRP (1975) recently considered areas of reasonable agreement with regard to the effects of radiation and, in summary, was concerned with such matters as the following:

"A. The likelihood of no absolute dose threshold for carcinogenic and genetic effects;

"B. The uncertainties regarding the shape of the dose effect curves overall from high dose and dose rate to low dose and dose rate;

"C. The likelihood that dose and dose rate influence effect per unit dose;

"D. The uncertainty concerning the influence of dose and dose rate on relative biologic effectiveness of high-LET radiation;

"E. The questionable scientific legitimacy of linear extrapolation from high dose and dose rate to low dose and dose rate in protracted exposure;

"F. The likelihood that such extrapolation from the rising part of the dose-effect curve (below the high cell-killing dose that reduce effectiveness) gives an upper limit of risk (even when zero risk is not excluded by the available data);

"G. Problems related to the inability to extrapolate the inverse relationship between carcinogenic latency and dose and dose rates;

"H. The uncertainty of the period of excess carcinogenic risk and of the excess and absolute incidence, owing to incomplete follow up of irradiated populations and their control populations;

"I. The many deficiencies of epidemiological data and their analyses, and of the control populations in the epidemiologic studies;

"J. The uncertainty of the influence of temporal and spatial variation of radiation exposure and dose;

"K. The many and difficult problems relating experimental animal data to human estimates."

Understanding that these caveats must not be ignored in undertaking risk estimates, we have estimated the late somatic effects. Analysis of all available data suggests that the principal effect is radiation-induced cancer (Storer and Bond, 1972).

G1.2 BEIR REPORT RISK ESTIMATES

The approach used in this study is to define risk estimates on the basis of the linear, no-threshold values generated in the BEIR Report (1972). This assumed that all risks of somatic effects are proportional to dose -- in other words, that each incremental increase in dose carries an equal increase in risk. The report adjusted the risk estimates to account for possible differences in the sensitivity of the fetus, child, or adult with respect to cancer induction by radiation. In addition, the concept of a latent period extending from radiation exposure to induced cancer death was addressed as well as the likelihood that the risk may be elevated for some finite interval (plateau) after the latent period. The factors used in the BEIR Report are summarized in Table VI G-1, with some minor modifications that are discussed below.

The absolute risk for persons 10 years old or older at the time of irradiation for all cancer excluding leukemia is broken down by organ in Table VI G-2.

The concept of a "plateau of risk" following a latent period after each increment of absorbed dose implies that risks are elevated during the plateau. In the case of leukemia risk after acute irradiation, it should be noted that the pleateau is not flat, but appears to decrease with time. By 25 years (BEIR Report, p. 171) the absolute risk has dropped to levels close to those of unirradiated persons. Lung-cancer risk in the ex-cigarette smoker also seems to decline with time when the "dosage" stops (Diehl, 1969). There is little documentation that the plateau is truly flat.

In this study, it has been assumed that each dose increment will have a specific latent period, for each cancer, followed by a flat plateau. The plateaus for nonleukemic cancers are assumed to persist for 30 years rather than for the duration of life. As noted elsewhere in this study, there do appear to be mechanisms of "repair" of certain steps in cancer induction and, as noted above, though latent periods may vary among cancers, it was deemed appropriate to use a fixed but long plateau.

As noted by the NCRP (1975), the linear hypothesis can be applied to population risk estimates, but data in support of this approach are limited. This assumption implies that the number of cancer deaths is determined by the products of the number of exposed individuals and the mean dose received and is independent of the size of the dose. For example, an equal number of cancer deaths would result from 10,000 people each receiving 100 rem or 10 million people each receiving 0.1 rem. The pertinent number is the number of man-rem.

G1.3 RELATIVE VERSUS ABSOLUTE RISKS

Risks are reported as either "absolute" or "relative". Relative risk is defined as "the ratio of the risk in those exposed to the risk to those not exposed (incidence in exposed populations to incidence in control populations)" (BEIR Report, 1972, p. 216). Absolute risk is defined as the "product of assumed risk times the total population at risk. The numbers of cases that will result from exposure of a given population" (BEIR Report, 1972, p. 213).

We have used the absolute risk approach in preference to the relative risk approach since the former provides a better indication of the impact in terms of the total number of deaths in a population due to a disease. Population cancer rates are variable and may be influenced by many geographic, socioeconomic, dietary, genetic, racial, and environmental factors among others.

The absolute risk approach does not assume that radiation risk at a given age is proportional to the spontaneous incidence of the disease at that age. The relative sensitivity of organs to radiogenic cancers is not proportional to spontaneous cancer rates nor to age-specific incidence rates. For example (American Cancer Society, 1974), stomach cancer death rates in males are 7.5 times higher in Japan than in the United States and at least 3 times higher than in Great Britain, yet the BEIR review shows an opposite trend in radiation risk (i.e., 0.04 per million per year per rem for Japanese atom bomb survivors and 0.64 for British patients with ankylosing spondylitis following X-ray therapy). The absolute-risk approach considers only the number of additional cases in an irradiated population, over the number of cases in an identical unirradiated population regardless of the magnitude of the latter. For this study, it was deemed appropriate to estimate only absolute radiation risks since there are many local spontaneous cancer rates (Mason et al., 1975).

The BEIR Report places heavy reliance on the ongoing study of the Japanese atom bomb survivors, who received very high dose rate exposures to gamma, neutron, and beta irradiation, quite different from the radiation source being evaluated in this report. Furthermore, risk estimations are for individuals whose doses were estimated to range from 10 to 300+ rem (those receiving 0 to 9 rem are used as controls). The results of other studies, to be mentioned later, were also used in the report.

TABLE VI G-1 BEIR RISK ESTIMATES[a]

Irradiation	Cancer	Duration of Latent Period (yr)	Duration of Plateau Region[b] (yr)	Absolute Risk[c]
In utero	Leukemia	0	10	25
	All other cancer	0	10	25
0-9 years	Leukemia	2	25	2
	All other cancer	15	30 Life	1
10+ years	Leukemia	2	25	1
	All other cancer	15	30 Life	5

(a) From the BEIR Report (1972), p. 171.
(b) Plateau region is the interval after the latent period during which risk remains elevated.
(c) Deaths per million population per year per rem.

TABLE VI G-2 ABSOLUTE RISK FOR ALL CANCER EXCLUDING LEUKEMIA FOR PERSONS 10 YEARS OLD OR OLDER AT TIME OF IRRADIATION[a]

Organ	Absolute Risk[b]	As %
Breast	1.5[c]	30
Lung	1.3	26
Gastrointestinal tract, including stomach	1.0	20
Bone	0.2	4
All other cancer[d]	1.0	20
Total	5.0	100

(a) From the BEIR Report (1972), p. 171.
(b) Deaths per million population per year per rem.
(c) Includes males and assumes a 50% cure rate.
(d) Includes thyroid and skin.

G1.4 RISK ESTIMATES

G1.4.1 Upper Bound Estimate

This section presents three different estimates of radiation risk: the upper bound estimate, the central estimate and the lower bound estimate. The upper bound estimate essentially uses the BEIR Report values with certain minor modifications relating to the gastrointestinal tract, bone, and thyroid gland. The total risk consists of the weighted sum of the risk for leukemia and for each specified cancer type adjusted for current age distributions in the population (US Department of Commerce, 1974), age-specific sensitivities, estimated "latencies," "plateaus," and life expectancies (US Department of Health, Education, and Welfare, 1975). Age intervals include the fetal period, 0 to 0.99 year, 1 to 10 years, and each decade to 80+ years of age in the analysis of absolute risk factors. Because we used 1974 and 1975 vital statistics and different age groupings, our estimates are not exactly the same as those of the BEIR Report (1972). It should be emphasized that risk as used in this report will be derived from calculations of doses to specific organs and the risk of death from cancer induced in that organ per unit dose such that the sum of these represents the total risk to the individual.

G1.4.2 Central Estimate

The central estimate modifies the upper bound estimate by correcting for risk reduction due to both the ameliorating effects of dose protraction and the lesser effectiveness of very small acute doses. There are many theories and much speculation regarding the spectrum and sequence of the events that occur between the absorption of ionizing radiation and the manifestations of a radiogenic cancer (Casarett, 1973; UNSCEAR, 1972; BEIR, 1972; NAS, 1974; Upton, 1967, 1968, NCRP, 1971; Storer, 1969). It is beyond the scope of this report to review all the factors, cofactors, and interrelations that have been proposed. One common theme, however, is found in almost any discussion of radiation carcinogenesis and the nature of dose-response curves. Where unequivocal data on these effects exists, the doses usually extend over a tenfold to fiftyfold range. As examples, the Atomic Bomb Casualty Commission usually considers data on persons receiving acute doses of ~10 to 300+ rem, and radionuclide-induced animal tumors of lung and bone are generally in a cumulative dose range of 1000 to 10,000+ rem (Bair et al., 1974; Bustad et al., 1972). As doses increase to very high but not acutely lethal levels, cell sterilization and cell killing as well as competing risks appear actually to reduce the apparent value of the crude incidence of specific tumors as a function of dose (Casarett, 1973). We do not compute the magnitude of this effect, nor do we employ any lowering of risk at these high doses, and to the extent that one might thus lower the estimate, we use a more conservative approach. In some cases reanalysis of data using epidemiological life tables and cumulative cause-specific incidence rates can produce different dose-response curves (Goldman et al., 1973).

Regardless of the method of data analysis, there is a consistent repetition of the statement that the protraction of dose in space and time increases the likelihood of repair of radiation damage (NCRP, 1971). This is especially evident in low-dose, low-dose-rate studies (NAS, 1974). A frequently quoted numerical value regarding radiogenic carcinogenesis is that low-dose-rate exposures are about one-fifth as effective as high dose rates for the same total (high) dose (Storer, 1969; Mays et al., 1973). Mays et al. have further defined a high dose rate as >10 rads/min ($\geq$14,000 rem/day) and low dose rates as <0.01 rad/min ($\leq$14 rem/day) for low-LET radiation and, on the basis of ten studies on animals derived a ratio of lower to higher effectiveness of about 0.2. If this is also true for the human, some intermediate value might also be obtained for the range from 0.01 to 10 rads/min. Mays et al. further estimate that this factor of 0.2 might range between 0.1 and 0.5 using a no-threshold linear estimate.

There is some radiobiological support for seriously considering the use of effectiveness factors for low doses and low dose rates (BEIR Report, 1972, pp. 379-401). It is not completely understood why *in vitro* studies of acute irradiation of cells invariably show that low-LET radiation is less lethal at doses below 100 rads than above that level (Hall, 1973). The appearance of a low-dose "shoulder" is frequently explained as a manifestation of repair of cell damage that might otherwise have caused cell death. It may be that some of the molecular lesions that cause cell death *in vitro* and *in vivo* are involved in the cancer-initiating steps in irradiated tissues. Other cell studies have shown that some enzymes (endonucleases) have the ability to repair radiation-induced molecular lesions in nuclear DNA and if these lesions are sparse with respect to space and time, efficient repair can result (Cleaver, 1970). Although these factors are

possibly only partially involved in the phenomena of radiation carcinogenesis, it may not be coincidental that similar ranges of doses are involved.

If one were to choose a working definition of the term "low dose," it might be 25 rem or between 10 and 50 rem (NAS, 1974). A low dose rate might be 14 rem/day (Mays et al., 1973) or more likely about 1 rem/day. A dose- and dose-rate-effectiveness factor applied below such levels might, for purposes of consequence estimation, utilize a crude approximation to some continuum that is close to zero at minute doses and unity at doses where the real risks are readily apparent. Thus the NAS upper bound limits are modified by the following dose-effectiveness factors:

	Dose-Effectiveness Factor		
	At <1 rem/day	At 1-10 rem/day	At >10 rem/day
<10	0.2	0.2	0.2
10-25	0.2	0.4	0.4
25-300	0.2	0.4	1.0

Note that the chronic factors present a crude approximation of a curvilinear dose response at low doses and dose rates. The upper "cutoff" for chronic radiation is derived primarily from animal experiments (Sanders et al., 1973; Upton, 1967, 1968; Storer and Bond, 1973) and a study of radium poisoning in dial luminizers (Evans, 1974). These factors do provide a numerical means of addressing the role of "repair" as an ameliorating effect in the case of low doses or protracted doses, in which the radiation energy is sufficiently spread in space and time (NCRP, 1971, p. 26; NAS, 1974). A tenfold rate difference could have been chosen rather than the fivefold factor of Mays et al. (1973) and Storer (1969) if one accepted a radiobiologic assumption that within the low dose domains defined above, even more repair is likely (NAS, 1974), and, in fact, the possibility of almost zero residual risk cannot be excluded from such an analysis (BEIR Report, p. 88). The dose-effectiveness factors used in this estimate are reinforced by the data and assessments discussed as well as other instances to follow and will be employed except where contrary evidence so dictates.

G1.4.3 Lower Bound Estimate

This section presents some factors and models for a lower bound estimate. It discusses nonlinear models, low dose thresholds, variable (dose-related) latencies, plateaus, and some experimental paradoxes.

A consistent nonlinear response has been observed in animal experiments employing graded doses and dose rates of external radiation and/or internally deposited low-LET radiations (Bustad et al., 1972; UNSCEAR, 1972). Considered singly, each study suffers from the fact that less than ideal numbers of animals were used at each exposure level. Considered as a whole, however, these experiments show a pattern in which acute doses below 25 rem (whole body) and chronic doses of about 1 rem per day (whole and partial body) show no observable difference from the unirradiated controls in terms of life shortening and specific cancer incidences (Upton, 1967; NAS, 1974). In some studies groups receiving low-level irradiation have, paradoxically, lived longer and manifested fewer cancers than did the controls. These trends are frequently attributed to the vagaries of "microstatistics" (Lorenz, 1954; Carlson et al., 1957, 1959; Bustad et al., 1965).

One interesting parallel human study relates to the analysis of the atom bomb survivors in Nagasaki, where the neutron, high-LET, radiation component was minimal in comparison to that received in Hiroshima (Mays et al., 1973). These data appear to be best fitted by a quadratic model such that the "probability would increase with the square of the X-ray or gamma-ray dose" (Jablon, 1974). Jablon (1974) has suggested that "such a model fits the data on leukemia mortality among the Japanese A-bomb survivors better than a model linear in both kinds of radiation, and the ratio of the numerical coefficients of the neutron and gamma ray terms is close to that reported for a number of effects in dissimilar biological systems." The support of the animal data by the human data from Hiroshima and Nagasaki argues strongly for a nonlinear approach to low doses of low-LET radiation (Rossi and Kellerer, 1974). (Note that the above passage refers to high dose rate, but "sparse" ionization density.)

A 1-rem dose of low-LET radiation might be so separated in space that only one nucleus in 100 cell nuclei would experience two ionizations and one in five might receive a single ionization. A large fraction of such "single-hit" molecular lesions would likely be reparable (Cleaver, 1970) and entail no further risk to the cell or tissue. If one further accepts the evidence suggesting that radiation carcinogenesis follows a multi-event sequence (Casarett, 1973), it is even more compelling to question the linear extrapolation of high-dose high-dose-rate data to all levels of radiation. The data do not support linearity for low levels of low-LET radiation (Frigerio et al., 1973), but the question is now more a quantitative one than a qualitative one (Brues, 1971). Mays et al. (1973) attempted to relate some high- and low-dose-rate data to derive a simple estimate of the numerical values of a dose-squared model. They suggested that four fatal cancers would be found for every fatal leukemia and that at a high dose rate (>10 rem/min), 0.1 leukemia + 0.4 fatal cancer might be found per million persons per rem squared. At low doses the factors are lower: 0.004 for leukemia and 0.016 for fatal cancers. They provide an example that predicts only 0.8% of a linear projection by using a quadratic model and a 10-rem exposure of a million persons. (The ratio of these two estimates drops by a factor of 10 for each tenfold dose reduction and would differ by almost a millionfold at a dose of 0.1 rem.) Thus, while finite risks can be derived for any exposure level, the risk becomes vanishingly small at millirem exposures.

A somewhat different approach at estimating cancer risks was derived from the twofold observation that after progressively decreasing doses, the latency appeared to lengthen and the incidence rate ("fraction exposed showing the effect per unit time") to decline (Jones and Grendon, 1975; Rosenblatt et al., 1971). These analyses of animal and human data predict that, at very low doses, latency is so extended as to exceed the individual's life expectancy and in this context constitutes a "practical threshold" (Evans, 1974). The relationship between exposure level (D) and latency (t) is nonlinear and can be fitted by an exponential or cubic function of the dose (Jones and Grendon, 1975):

$$t_i = t_o(D_o/D_i)^{0.33},$$

which again suggests that radiation carcinogenesis is a multi-event phenomenon that may involve effective repair of lesions. At low levels of exposure approximating 1 rem, we estimate that a linear, no-threshold, upper bound prediction and a lower bound estimate will probably differ by at least a factor of 1000 and at the millirem level by a factor of 10^6. Thus, as recognized by the BEIR Report (1972, p. 88), "the possibility of zero is not excluded by the data."

G2 LEUKEMIA

Although the mechanism by which radiation may induce leukemia, as well as other types of cancer, is not well understood, the literature on human leukemia and animal models of this disease suggest that susceptibility is greater in the young than in the adult (BEIR Report, 1972; UNSCEAR, 1972). The susceptibility of the fetus to radiogenic leukemia and cancer is uncertain. Studies in Great Britain and the United States suggest that children exposed shortly before birth are at high risk (Stewart and Kneale, 1970a,b; MacMahon and Hutchinson, 1964) but data from studies in Japan do not support these high estimates of risk (BEIR Report, 1972; UNSCEAR, 1972; Jablon, 1974). There is agreement that this disease manifests a short latent period, a fairly short plateau, and a decline of risk to negligible levels within a decade of irradiation (UNSCEAR, 1972, p. 408).

G2.1 UPPER BOUND ESTIMATE

The BEIR Report quotes an estimate of two leukemia deaths per million per year per rem for children irradiated at ages 0-9 years and one death per million per year per rem for individuals irradiated at 10+ years of age. The disease shows a 2-year latency and a 25-year plateau. The BEIR Report also uses a value of 25 deaths per million per year per rem in utero (1972, p. 171). This high value was derived primarily from the data of Stewart and Kneale (1970a), who later indicated (1970b) that the fetal dose estimates they had used were considerably lower than the doses actually received, so that the corresponding risk estimates had been overestimated by 60 to 100%. (It was confirmed by Draper at the meeting of the American Nuclear Society, Las Vegas, June 19, 1972, that the dose estimate originally used was too low by at least a factor of 2.) Thus it follows that the excess risk of death from leukemia derived from these data should be 15 cases per million per year per rem. The assumptions of no latency and a 10-year plateau are used for those irradiated in utero.

G2.2 CENTRAL ESTIMATE

Available data suggest that the risk of leukemia after fetal irradiation is not constant throughout life and does not persist for many years. The fetal leukemia risk of 15 deaths per million per year per rem is modified as above from the BEIR (1972) estimate (25 cases per million per year per rem) and is used in this analysis. Note that the UNSCEAR (1972) estimate is 10 cases per million per year per rem. We assume that childhood leukemia is always a fatal disease. The risk from fetal acute irradiation is assumed to be exhausted within 10 years after exposure (BEIR Report, 1972, p. 166):

> "Despite uncertainty about the oncogenic effects of intra-uterine exposure we presume for purposes of conservative overall risk evaluation that such exposures do increase the risk of cancer in the child until ten years of age but not thereafter."

In the case of a fetal dose resulting from long-lived radionuclides inhaled by the mother and other factors that might contribute to the total fetal radiation dose in a protracted or fairly continuous fashion, a dose-rate-effectiveness reduction factor of 5 is assumed as described earlier for the adult. This is to say, 5 rem of continuous fetal irradiation throughout gestation will have the same consequence as a 1-rem acute dose delivered at a randomly selected time during gestation. The absence of an increase in cancer among Japanese infants irradiated in utero (Jablon and Kato, 1970) casts some doubt on the numerical estimates derived by Stewart and Kneale (1970a, b) and MacMahon and Hutchison (1964).

Irradiation is thought to make juveniles more susceptible to late occurring health effects. A major reason for this belief is that juveniles have a large life expectancy in which such effects can be manifested. This would further assume that a plateau of risk would obtain through much of the life expectancy. In the BEIR summary, the latent period for juveniles was assumed to be about 2 years for leukemia and 15 years for all other cancers. This was assumed to be followed by a risk plateau lasting 25 to 30 years. The result was an absolute risk for death of about 2×10^{-6} and 1×10^{-6} per year per rem per individual for leukemia and for all other cancers, respectively. It should be noted that two-thirds of the absolute risk is associated with leukemia and one-third with all other cancers (BEIR Report, p. 171). This age category includes 16% of the U.S. population and has a life expectancy of 69 to 72 years (Table VI G-3). Most of the human experience is based on high-dose-rate exposures in either Japanese atom bomb survivors or children irradiated for medical reasons. Chronic exposure of experimental animals at daily doses below several hundred millirem have failed to show an increase in myeloproliferative disorders and leukemias. However, for juvenile experimental animals receiving doses above 1 rem/day, a nonlinear sigmoidal relationship of dose to absolute risk has been observed (Bustad et al., 1972). For chronic irradiation at the same total dose, the induction rate appears to diminish exponentially to a level that is 10 times lower than that for acute irradiation.

The reduction factors for acute and chronic low dose effectiveness are further modified by the above mentioned latency and plateau constraints before application. In this estimate we have used a risk to the fetus of 15 per million per year per rem. Children of ages 0-9 are assigned a risk of 2 cases per million per year per rem, and adults a risk of one case per million per year per rem.

G2.3 LOWER BOUND ESTIMATE

Jablon's analysis (1974) of the Japanese bomb survivors represents a comprehensive survey on the temporal pattern of leukemia risk. His analysis suggests a latency of 3 to 5 years, with a plateau or peak during the following 5 years and an exponential decline to a level that may not differ significantly from the control level by 40 years after exposure (Jablon, 1974). These data, based on the population assumed to have received more than 100 rad, further suggest that the dose-response relationship can best be quantified in terms of the absolute risk being proportional to the square of the dose. The data for the Nagasaki survivors is particularly germane to the reference accident model in that they are not obscured by problems relating to the relative biologic effectiveness of the large neutron component in the Hiroshima weapon. It should be noted that in almost 30 years of follow up, no excesses in leukemia incidence among Nagasaki survivors have been attributed to doses below 100 rem.

TABLE VI G-3 POPULATION STATISTICS[a]

Age (yr)	Fraction	Life Expectancy (yr)
In utero	0.011	71.0
0-0.99	0.014	71.3
1-9.9	0.146	69.4
10-19.9	0.196	60.6
20-29.9	0.164	51.3
30-39.9	0.118	42.0
40-49.9	0.109	32.6
50-59.9	0.104	24.5
60-69.9	0.080	17.1
70-79.9	0.044	11.1
80+	0.020	6.5

(a) Data from the U.S. Department of Commerce (1974) and the U.S. Department of Health, Education and Welfare (1975).

The follow up studies on patients receiving partial body irradiation for therapeutic purposes (phosphorus-32 for polycythemia vera and spinal irradiation for ankylosing spondylitis) are generally in agreement with the results of the Japanese studies (UNSCEAR, 1972, pp. 404-415). In the absence of direct observation of chronic continuous irradiation of human bone marrow and subsequent leukemia induction, one study that is applicable relates to the consequences of marrow irradiation following continual strontium-90 incorporation into the skeleton throughout the first 1.5 years of life in groups of beagles. The lowest levels at which an increase in leukemia (myeloproliferative disorder) was observed were dose rates in excess of about 500 mrem/day, totaling 500 to 1000 rem to the bone marrow. On the basis of a comparison of dog and man it would appear that a dose-protraction factor of between 5 and 10 might be appropriate for leukemia risk (Goldman and Bustad, 1972). Similar studies on inbred mice support the extrapolation from species to species with regard to the consequences of dose protraction (Upton, 1967). Thus, if the leukemia risk in adults after high-dose-rate exposure is one case per million per year per rem, the risk from low dose rates might be 0.1 to 0.2 per million per year per rem for all adult "ages" at exposure.

If the animal data cited above are assumed to be relevant to man, they suggest that chronic marrow irradiation at a rate lower than about 0.2 rem/day (~75 rem/year) imposes minimal risk for leukemia. Mays et al (1973) have further speculated that a low dose rate (<0.01 rem/min) might impose a population lifetime risk of 0.004 leukemia death per million per year per rem^2 using an average dose of 1 rem and a dose-squared model.

G3 LUNG CANCER

Risk estimates for radiation-induced human lung cancer have been prepared by the BEIR committee (1972) and UNSCEAR (1972). The influence of the inhalation of particulates emitting high-LET radiation on that risk has also been assessed (Bair et al., 1974). Most estimates of low-LET radiation effects are derived from atom bomb survivors (correcting for neutron contributions) and from patients with ankylosing spondylitis. In addition, high-LET exposures of fluorspar miners, uranium miners, and patients who had been administered Thorotrast have been studied. The estimates of absolute risk for lung cancer range from 0.2 to 9 per million per year per rem (BEIR Report). The UNSCEAR (1972) summary includes the same basic information and estimates a range of 0.6 to 3 per million per year per rem. From the American Cancer Society's statistics (1974) the mortality rate for lung cancer seems to be 89%.

G3.1 UPPER BOUND ESTIMATE

The BEIR Report (1972, p. 150) risk estimate for lung cancer deaths is 1.3 per million per year per rem for adults and includes a bronchial cancer risk in adults of 1 per million per year per rem. A latency of 15 years followed by a 30-year plateau of elevated risk is assumed in calculating the risk estimate.

G3.2 CENTRAL ESTIMATE

The BEIR values are adjusted by the low-dose and low-dose-rate factors presented earlier For example, an acute lung dose of 75 rem would yield a central estimate for deaths in adults of 1.3 x 0.4 = 0.5 per million per year per rem. It should be noted that to date no significant excess lung-cancer incidence has been reported for the Nagasaki (low-LET) survivors (UNSCEAR, 1972, p. 418).

Lung irradiation at low dose rates--for instance, by inhalation of particles emitting low-LET radiation--requires some further comment before quantification of risk. Recently the special question of plutonium particulates has been studied and comparisons made with available information from experimental animal research on low-LET lung irradiation (Bair et al., 1974). When the cumulative average pulmonary dose is related to the fractional incidence of lung cancer in rodents and dogs, a nonlinear increase in effect from about 1000 to 30,000 rem cumulative, has been seen. There is little in the way of quantification for absorbed cumulative doses below 100 (and possibly 500) rem. Over the range of available data, the average lung dose for alpha-emitting particulates within the lung appears to be 5 to 20 times more effective than the same total dose for gamma emitters. For mixtures of beta and gamma emitters in lung, the average dose equivalent will likely not reflect a local dose distribution factor, and it is expected that the results would be quantitatively similar to comparable dose rates from chronic external total lung irradiation. It should be pointed out that in the case of inhaled radioactive particles, the initial dose rate will be maximal and will continually decline.

Thus the dose-rate effect might be of greater significance than slight nonuniformity of dose distribution within the lung (Jones et al., 1974). Available experimental information provides no justification for altering the quality factor for alpha particles from the value of 10 already employed (ICRP, 1966). Furthermore, the observed relative sensitivity of pulmonary tissue with respect to tumor induction does not provide evidence for an especially greater radiosensitivity than that observed for other tissues similarly irradiated by deposited radionuclides. For both beta-gamma and alpha emitters, the evidence strongly suggests that the less spatially and temporally uniform the organ dose, the lower the total risk (Bair et al., 1974).

The possible role of cigarette smoking and other environmental factors in human pulmonary carcinogenesis has made difficult any comparison of experimental animal data with the limited amount of available human information. There may be synergistic effects of cigarette smoking and pulmonary irradiation that are not yet completely understood (Mole, 1971). Furthermore the histological type and location of tumors in experimental animals do not always resemble those seen in people. The most reasonable explanation available is that these may be more a reflection of the additional cofactors associated with environmental pollution and cigarette smoking rather than species dependencies. The occurences of "repair" of some of the "factors" involved in lung carcinogenesis can be inferred from the analysis by Hammond, in which ex-cigarette smokers were shown to have a continually declining excess risk for lung cancer and, by 10 years after the cessation of the smoking habit, were not significantly different from age-adjusted controls, who had never smoked cigarettes (Diehl, 1969). To the extent that this risk reduction parallelism may be extended, it may be reasonable to assume that, in the case of a declining dose rate from decaying and metabolized pulmonary deposits of radioactive particulates, the risk, if not constant, may actually decline.

G3.3 LOWER BOUND ESTIMATE

If no major changes in the trends among the Japanese atom bomb survivors are noted, it might well be that acute low-LET pulmonary doses below about 400 rem or 100 rem per year of chronic irradiation invoke minimal additional lung cancer risk. Furthermore, the latency may be about 15 years, followed by a 10-year risk plateau and a linear decline in risk to control levels in 10 years. A dose-squared model could use varying estimates of deaths, ranging from 0.03 to 1 per million per year per rem^2. In animal experiments, lung-cancer death rates rise exponentially at doses above ~500 rem (chronic) but show an extended "latency" as the cumulative dose declines (Bair et al., 1974). At low dose rates the lung appears to be a radioresistant organ.

G4 CANCER OF THE GASTROINTENTINAL TRACT

Most of the available information regarding radiation-induced cancer of the gastrointestinal tract is derived from acute or fractionated high-dose-rate exposures in man. Animal experiments in which radionuclides were ingested, have failed to yield information regarding increased risk for radiogenic cancers of the gastrointestinal tract. One (15-year) study on beagles fed up to 36 microcuries per day of strontium-90/yttrium-90 (an energetic beta emitter) for 1.5 years has failed as yet to show pathologic change in these tissues (dose rates to the colon ranged up to 5 rads per day) although marked skeletal and hematopoietic effects were found (Goldman, 1974).

Most estimates of risk to man are derived from the Japanese atom bomb survivors and from ankylosing spondylitis (male) patients receiving 250 to 2750 rads of partial-body high-dose-rate X-irradiation (BEIR Report, pp. 157-159).

G4.1 UPPER BOUND ESTIMATE

The BEIR Report (p. 171) estimate of absolute risk for death from gastrointestinal cancer including the stomach is 1 per million per year per rem. The estimate is apparently derived primarily from the ankylosing spondylitis patients, using a value of 250 rem as the mean exposure. Tables g1 and g2 in the BEIR Report (pp. 158 and 159) further subdivide this value to 0.64 (0.30 to 1.1) for stomach and 0.44 (0.20 to 0.76) for the rest of the gastrointestinal tract. There has been one additional stomach cancer case in the Japanese study of 250,000 person-years of followup, for an estimated mean dose of about 82 rads, from which an estimated risk of 0.04 per million per year per rem (0.0 to 0.86) for this disease is derived. The estimate for the rest of the gastrointestinal tract

is 0.48 (0.0 to 1.2). It is significant that the 20 gastrointestinal cancer deaths seen in the spondylitic patients consisted of 5 pharyngeal, 3 esophageal, and 12 pancreatic cancers. None was attributed to the large intestine. Furthermore, "the possibility remains that the excess number of cases was not due to radiation but arose from selective factors associated with the disease process or its treatment" (BEIR Report, p. 157). In particular, the possible influence of medications given these patients cannot be excluded nor can "the possible relation of ankylosing spondylitis to ulcerative colitis and, consequently to colon cancer" (UNSCEAR, 1972, p. 423).

No excess cases of cancer of the large intestine have been observed (UNSCEAR, 1972, p. 433), and the risk relative to the "control group" would appear to decline slightly, if anything, with increasing dose. A similar trend seems to hold for pancreatic cancer.

The absence of evidence of radiation-induced large-intestine cancer and the problems associated with interpreting the BEIR values (i.e., the influence of the pancreas data on the BEIR estimate) suggest that the present assessment of risk be confined to the alimentary tract and that a separate estimate be used for the pancreas. Thus, instead of a value of 1 per million per year per rem, the value of 0.6 for the stomach plus 0.2 for the remainder of the gastrointestinal tract is used (i.e., 0.8 per million per year per rem for the entire gastrointestinal tract of irradiated adults. A separate estimate of 0.2 is used for the pancreas. A latency of 15 years followed by a plateau of 30 years is assumed for individuals over 10 years of age.

G4.2 CENTRAL ESTIMATE

The upper bound values are used for this estimate, but the low-dose, low-dose-rate modification factors are applied to those tissues in order to account for the extreme likelihood that much of the tissue injury (molecular lesions) thought to play a role in risk will be successfully repaired after low doses of low-LET radiation. These estimates also have additional conservatism included as the doses are calculated to the regenerative cells of the lower large intestine, which is likely to receive the highest dose, but appears to be less sensitive than the rest of the gastrointestinal tract.

G4.3 LOWER BOUND ESTIMATE

The lower limit for risk derived by the BEIR Report for gastrointestinal cancers in Japanese atom bomb survivors is zero. The mucosal cells in the intestinal tract have a high renewal rate, but the absence of data suggests that they are quite resistant to radiogenic cancer. Alternatively, their latency may be very long (over 30 years) and may be further extended as dose and dose rate decline. Since there is so little data available, it is interesting to note that over 35,000 rem of local continuous irradiation at high rates was required to produce esophageal tumors in mice (Gates and Warran, 1968). A curvilinear or quadratic expression of risk can be used as an estimate. On the assumption that 16% of solid tumors are of this type and using the estimate for dose-squared model (Mays et al., 1973), the lifetime population risk from acute high-dose-rate exposure could be $0.4 \times 0.16 = 0.06$ per million per rem^2 and for chronic low-dose-rate, low-LET radiation it could be about $0.016 \times 0.16 = 0.003$ per million per rem^2.

If the mouse data presented above are applicable to people (i.e., within an order-of-magnitude of agreement between the species), it appears that there may be a "threshold" for the induction of some of these cancers (UNSCEAR, 1972, p. 397). Under such an assumption a gut threshold of 10^4 to 10^5 rem might apply. Following a gut dose of about 10^5 rem, however, it is quite likely that the dose to adjacent tissues would be so high as to substantially reduce life expectancy and thereby markedly reduce the incidence of latent radiogenic intestinal tumors.

G5 BREAST CANCER

The UNSCEAR (1972) review recommends an overall individual risk of 0.7 per million per year per rem for the induction of this disease in females. Morbidity estimates range between 1 and 6 per million per year per rem and are primarily derived from data on the Japanese atom bomb survivors. The estimate for latency is 15 to 20 years (BEIR Report, p. 136). There appears to be little evidence that fractionated irradiation reduces the effectiveness of breast-cancer induction. Most of the human experience has been associated with relatively high doses -- in excess of 80 rem.

It should be noted that breast-cancer mortality has been declining as therapy improves (Myers, 1973). The BEIR Report assumes a 50% cure rate.

G5.1 UPPER BOUND ESTIMATE

The BEIR mortality estimate is 1.5 per million per year per rem for the whole population, male and female, assuming a 50% cure rate. A 15-year latency and a 30-year plateau are also assumed for individuals over 10 years of age.

G5.2 CENTRAL ESTIMATE

The value of 1.5 deaths per million per year per rem is not modified for low doses and low dose rates, as in the case of other cancers. Evidence that fractionated high-dose-rate radiation shows minimal dose amelioration precludes the use of such risk reduction factors.

G5.3 LOWER BOUND ESTIMATE

The BEIR Report data appear to derive principally from breast doses in excess of 80 rem and suggest a latency of 20, rather than 15, years. If true, this would suggest a cutoff of about 80 rem, below which the risk is minimal. Furthermore, it seems reasonable to exclude females below puberty (~10 to 15 years) from the analysis. A dose-squared model might be calculated using $(1.5/5)(0.016) = 0.005$ per million per year per rem^2 for a 1-rem population dose (Mays et al., 1973).

G6 BONE CANCER

The BEIR Report summary of information on bone cancer relies heavily on data for ankylosing spondylitis patients (X-ray data), patients receiving radium-224 treatments for bone tuberculosis (high dose and high dose rate of alpha particles to endosteal cells), and a group of painters of luminous dials who ingested quantities of radium-226 some four to five decades ago. No significant changes in mortality for this rare cancer form have yet been observed in the Japanese atom bomb survivors. The fairly extensive animal studies using bone-seeking radionuclides that have been performed support the general impression that skeletal tissues are relatively resistant to low doses of low-LET radiation, especially at low dose rates (UNSCEAR, 1972, p. 385).

G6.1 UPPER BOUND ESTIMATE

Bone-cancer risk is difficult to define precisely, although the BEIR Report presents a mean value of 0.2 per million per year per rem. For children below 20 years of age a value five times higher is used (i.e., 0.96 per million per year per rem; BEIR Report, p. 128), assuming radium-224 to be twice as potent in juveniles as in adults. However, if the weighted adult value (0.2) is one-half of the juvenile estimate, the latter might best be estimated as 0.4 per million per year per rem. Note that the BEIR Report (p. 171) ascribes a total 0- to 9-year risk coefficient of 1 for all cancers, and a 10+ year risk coefficient of 0.2 for bone cancer with a 15-year latency for both age groups. Thus a risk coefficient of 0.4 for the 0 to 20-year age cohort may be more conservative value than that used by the BEIR Report. In this study a latency estimate of 10 years has been used for all ages rather than the 15-year value quoted by the BEIR Report. This more conservative estimate is based on the data summarized by UNSCEAR (1972, p. 421) and is assumed to be the best estimate available.

G6.2 CENTRAL ESTIMATE

Radiogenic bone cancer in man has been extensively studied. There have been some epidemiological studies of patients given X-ray therapy, although most of the information has been derived from the deposition of bone-seeking radionuclides within the skeleton. The UNSCEAR (1972) report suggests that in radiation-induced bone cancers (primarily sarcomas) a latency of 4 to 42 years may obtain, with an average value of 11 years, after initial exposure. This study has used a latency-period of estímate of 10 years. For spondylitic patients treated with high dose rate X-irradiation, the BEIR Report (p. 128) quotes a bone-cancer absolute risk of 0.1 per million per year per rem, which agrees with the value of 0.11 derived from the study of radium-226 patients. It would appear that high-dose-rate endosteal cell irradiation from short-lived radium-224 treatment of tuberculosis patients in Germany provides an absolute estimate of 0.5 per million per year per rem. Estimates for bone-cancer risk of 0.2 and 0.4 per million per year per rem for adults and children, respectively, modified by the previously defined low-dose and low-dose-rate factors, were used in the analysis.

G6.3 LOWER BOUND ESTIMATE

For the case of chronic irradiation from radium-226 deposited in the skeletons of painters of luminous dials, no excess cancer mortality has been observed for cohorts whose cumulative radiation doses are below about 700 to 1000 rads (i.e., 7000 to 10,000 rem), averaged over the skeleton (Evans, 1974). Chronic skeletal irradiation from low-LET sources, such as strontium-90, manifests a similar "threshold" in beagles. Furthermore, the shape of the dose-response curve for low-LET radiation effects on the skeleton definitely appears to be nonlinear (Goldman, et al., 1972). It should be emphasized that all of the available data refer to populations of experimental animals whose cumulative skeletal dose has been on the order of several hundred rems, delivered at rates > 1 rem per day. If one were to extrapolate linearly downward from the dose domain in which effects have been noted, it would appear that an absolute risk on the order of 0.15 per million per year could be reasonably defended. There is strong evidence (BEIR Report, p. 128) that the true risk for low-LET skeletal irradiation is related to the square of the dose. Although bone cancers are exceedingly rare, they are lethal.

G7 OTHER CANCERS

For all cancers other than leukemia, the BEIR Report suggests a latency period of approximately 15 years and plateau of 30 years or more, with a resultant absolute risk of 5 per million per year per rem. The risk is distributed as follows: 30% for female breast cancer, 26% for lung cancer, 20% for gastrointestinal tract and stomach cancers, about 4% for skeletal tumors, and the remaining 20% (i.e., 1 per million per year per rem) for all other cancers. Thus relative to the 0- to 9-year-old child, the risk for adults is assumed to be fivefold greater for nonleukemic cancer deaths (see Tables VI G-1 and VI G-2).

In this appendix thyroid cancer is not included in the "all other category." This residual category includes a variety of unspecified cancers, data for which are fragmentary, and often lack a clear dose-effect relationship.

Skin cancer is one disease that the BEIR committee recognized as a possible contributor to overall risk, but it was reluctant to speculate on numerical risk estimates for low-level irradiation (BEIR Report, p. 133):

"Although evidence suggests that the probability of radiation-induced skin cancer is greatly increased in the presence of radiodermatitis, the data are insufficient to document the induction of skin cancer at doses below the level required to cause radiodermatitis, suggesting that the susceptibility of skin to radiation carcinogenesis may be lower than that of certain other tissues such as the thyroid and the bone marrow. The possibility remains, however, that the absence of recorded cases may be attributable to unusually long latency or to underreporting of neoplasms. In the absence of further data, numerical estimates of risk at low dose levels would not seem to be warranted."

In rodents exposed to high-dose-rate electron irradiation, doses of more than 1000 rads were required to produce an increase in skin cancer (BEIR Report, p. 133). In the absence of adequate documentation it may be reasonable to assume that a "threshold" exists for skin-cancer induction at a level of about 500 rem. We suggest that a latency of at least 10 years and a plateau of 30 years may be appropriate for this disease. In the absence of specific data, and recognizing the possibility of a threshold, an absolute-risk estimate of 0.1 per million per year per rem might be applied for purposes of risk estimation. Mortality is quite low for most skin cancers (<5%, mainly from melanomas) (American Cancer Society, 1975).

Fetal irradiation increases the risk of leukemia and solid cancer but no studies link fetal irradiation with any specific solid tumor (BEIR Report, p. 165). In the absence of specific data, a conservative estimate of a doubling of the leukemia rate is assumed for the induction of fatal cancers. Tumors of the central nervous system, kidney, and of the lymphoid and reticular tissues might predominate after fetal irradiation, if one assumes an analogy to the spontaneous rates of early childhood cancers for these tissues (Mason et al., 1975).

The risk from childhood radiation exposure for the induction of cancer in tissues other than the bone marrow (i.e., leukemia), does not appear to be specifically age-sensitive, with two exceptions: the child's thyroid gland and skeleton appear to have a greater sensitivity (by a factor of at least 2) than those of adults after the same total organ exposure (BEIR Report, 1972). The apparent increase in risk for the induction of skeletal tumors is based primarily on the observation of children in Germany following radium-224 therapy for skeletal tuberculosis. On a total-absorbed-dose basis, these fractionated exposures appeared to be twice as effective in inducing osteosarcomas as comparable doses administered to adults. It should be recalled that this experience was with alpha irradiation and that a relative biological effectiveness (RBE) of 10 was assumed. In the absence of specific quantification, it would appear that the BEIR estimate of a 15-year latent period for children followed by a 30-year excess-risk plateau is appropriate, if applied to the total absolute risk of 1 per million per year per rem for all solid cancers estimated in the BEIR Report. The study has used a value of 0.6 per million per year per rem for children 0 to 10 years old for all cancers other than leukemia, bone, and thyroid.

It may be inappropriate to assume that the distribution of childhood cancers resembles that of cancers in adults. A more realistic approach would be to omit numerical partitioning by cancer type, since some childhood cancers (e.g., neuroblastoma, Wilms' tumor) inducible by irradiation may not have counterparts in adults (BEIR Report, p. 165). Although these estimates are derived principally from data on high-dose-rate, acute exposure of adults, additional information from animal experimentation can be applied for the chronic (radionuclide) component of such radiation. If the evidence derived from the exposure of beagles to strontium-90 throughout early life can be a guide, it would appear that continual skeletal irradiation in excess of about 0.5 rem per day is necessary to demonstrate an excess leukemia and sarcoma risk and that the risk rises very steeply above this dose, in proportion to the square of the dose. Total skeletal exposures of 500 to 1000 rem appear to be required to manifest this effect (Goldman and Bustad, 1972).

G7.1 UPPER BOUND ESTIMATE

The upper bound estimates of mortality from all other cancers for adults, children 0 to 10 years old, and fetuses are 1, 0.6, and 15 per million per year per rem, respectively.

G7.2 CENTRAL ESTIMATE

The upper bound estimates are used, modified as before for low dose and low dose rate.

G7.3 LOWER BOUND ESTIMATE

A dose-squared estimate of "all other" cancer risks might use the BEIR value multiplied by the low-dose-rate factor (0.016) of Mays et al. (1973) to predict, for a 1-rem dose, a fatal cancer rate of 0.008 per million per rem^2. For acute (high-dose-rate) exposure, the comparable value would be 0.02 per million per rem^2. The possibility of dose-related extensions of latency, discussed in the introduction, may be quite germane to this class of cancer. If their incidence is very low, it is possible that, at low doses and low dose rates, the numerical risk is not significant, and its omission would not alter the overall total estimate.

G8 CANCER PREVALENCE, INCIDENCE AND MORTALITY

Risk estimation for radiation-induced cancer is usually discussed in terms of cancer mortality. For the purposes of this study, however, it is important to estimate the number of additional cases, as well as mortality. It is assumed that, once recognized or diagnosed, a specific cancer -- whether "spontaneous" or radiation-induced -- will have the same prognosis with regard to curability and survival. A complete discussion of cancer statistics is beyond the scope of this study. There are demographic, age-related, regional, socioeconomic, racial, and environmental and temporal variations and trends in the data (Mason et al., 1975). Over the years, cancer classifications, the reporting efficiency, and treatment effectiveness have also changed (Levin et al., 1974). This section summarizes some recent cancer statistics for the United States, derived from the National Cancer Institute's Third National Cancer Survey, 1969-1971, (U.S. Department of Health, Education and Welfare, 1974), with extrapolations for 1975 by the American Cancer Society (1974). Of particular significance to estimates of cancer mortality is the relationship between net prevalence and mortality, the estimate of the fraction of additional radiation-induced cases that will be fatal, and some data for survival for each cancer type. Age-adjusted cancer rates were used to account for each cancer's specific age-frequency distribution. Also discussed are estimates of median survival and 5-year relative survival rates for some specific cancers (i.e., the percentage of patients alive 5 years after diagnosis, adjusted for "normal" mortality) (Levin et al., 1974).

Cases of acute leukemia have a median survival of only 0.3 year and a 5-year relative survival rate of only 1%. The prognosis for chronic leukemia is somewhat better, with a 5-year survival rate of about 27% and a median survival of 1.6 years. Since radiation-induced leukemias are expected to be of the acute type, we have assumed that the mortality rate is equal to the prevalence of radiation-induced leukemia.

The prognosis for cancer of the lung, bronchus, and trachea is poor, with a median survival of only 0.4 year and a 5-year relative survival rate of only 8%. Thus for purposes of this study the additional cases produced will be assumed to equal the mortality rate.

If radiation-induced stomach cancer has the same prognosis as spontaneous cancer, a median survival of about 0.4 year would be expected, with a 5-year relative survival rate of 12%. Thus for computational purposes, the additional prevalence rate from radiation will again be assumed to be equal to the mortality rate. Cancer of the rest of the alimentary canal is estimated on the basis of health statistics for cancer of the colon and rectum. For this disease the median survival is estimated to be 2.2 years, with a 5-year relative survival rate of 40%. It has been assumed that there will be two additional cases for each cancer death. For radiation-induced pancreatic cancer, the median survival is estimated to be 0.2 year and the 5-year relative survival rate is only 1%; thus the additional cases will be equal to the mortality rate. Statistics on the prognosis for breast cancer are changing due to earlier recognition and improved therapy. Current estimates suggest that the median survival for breast cancer is 6 years and the 5-year relative survival rate is about 62%. For each cancer death due to this disease an additional case ratio of 2.5 could be assumed from these data. The study however has conservatively used the 50% cure rate given in the BEIR Report (p. 169).

Median survival for bone cancer is 1.7 years, and the 5-year relative survival rate is 35%. For purposes of this study it has been assumed that 80% of the cases will be fatal.

A recent estimate for all cancers throughout the United States would suggest a median survival of 1.7 years and 5-year relative survival rate of 40%, taking into account the relative distribution of each type of cancer among the population. For all unspecified additional cancers it has been assumed that the number of radiation-induced cancers will be twice the radiation cancer mortality rate.

G9 SUMMARY AND CONCLUSIONS

The late somatic effects of irradiation have been reviewed and used to provide three levels of risk estimation for late occurring radiogenic cancers, the most significant biomedical parameter. For the upper bound the approach of the BEIR Report was used, which assumes a linear, no-threshold relationship of dose to risk and is summarized in Table VI G-4. No numerical corrections for low dose or low dose rates were included, although different values for the gastrointestinal tract and stomach are presented. The central estimate that is presented modifies the upper bound values to account for risk reduction at low total doses and low dose rates. Also provided are lower bound estimates which are based on nonlinear responses. Where possible, some numerical estimates of the range of risk expectations have been provided for perspective. A recapitulation of the BEIR Report estimates is included in the introduction, and, for additional background, the reader is referred to the summary of risk estimates in the UNSCEAR report (1972, Table 22). The population statistics used in the analysis are shown in Table VI G-3.

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TABLE VI G-4 UPPER BOUND RISK FACTORS FOR CANCER MORTALITY[a]

Type of Cancer and Age at Irradiation	Latency (yr)	Plateau (yr)	Risk[b]
Leukemia:			
In utero	0	10	15
0-9.9 years	2	25	2
10+ years	2	25	1
Lung cancer:			
10+ years	15	30	1.3
Gastrointestinal tract:			
Stomach, 10+ years	15	30	0.6
Rest of gastrointestinal tract,[c] 10+ years	15	30	0.2
Pancreas,[d] 10+ years	15	30	0.2
Breast cancer,[e] 10+ years	15	30	1.5
Bone Cancer:			
0-19.9 years	10	30	0.4
20+ years	10	30	0.2
Other cancers:[f]			
In utero	0	10	15
0-9.9 years	15	30	0.6
10+ years	15	30	1

[a] Data from the BEIR Report (1972), modified where appropriate.

[b] Number of deaths per million population per year per rem.

[c] Including the lower bowel.

[d] See text.

[e] Includes male population correction.

[f] Includes all other cancers; corrects for juvenile bone cancers; assumes fetal leukemia risk equals that for all other cancers.

Appendix H

Thyroid Effects

H1 INTRODUCTION

In developing estimates of the possible effects of ionizing radiation on the normal human thyroid gland, it was decided to use principally information from human exposure to iodine-131 and to external radiation. To prepare the current estimates, data from several different populations irradiated under different circumstances have been combined. Many of these data have not yet been published, but the sources are referenced.

In evaluating the effects of iodine-131, the consequences of a single dose administered within a time span of about 1 day or less were considered. The lesser effects of environmental recycling were excluded. Since many of the available data have been drawn from diseased populations, an attempt has been made to adjust for the spontaneous thyroid effects expected in a similar population that is diseased but not irradiated. Data drawn from populations without thyroid disease were adjusted for the spontaneous prevalence of thyroid disease by age in the general population, or when comparison populations were available, from those groups.

Multiple exposures over varying time intervals complicate the study of the effects of external radiation doses to the thyroid. Under these circumstances, and when appropriate, the correction formulas applied in similar circumstances for radiation therapy have been employed (see below). When sufficiently detailed information was lacking, thyroid dose estimates of the particular authors were employed as if the dose were administered at a single time.

In evaluating thyroid neoplasms, estimates have been developed for thyroid cancer and for total thyroid nodules. The reason for discussing total nodularity rather than benign lesions is that diagnostic procedures and therapy, either surgical or medical, are directed to nodules. Only after the procedures are performed is it known whether the nodule is benign or malignant.

It has been difficult to settle the question of the latent period, defined as the amount of time elapsed between radiation exposure and the development of the lesion. Thyroid neoplasms detected within the first year have not been attributed to the irradiation. When specific data have not been available, the latent period has been assumed to be the time from exposure to followup. In cases of multiple exposures over long periods, the mean time between the first and last radiation exposures has been taken as the time at which exposure occurred.

Risk estimates have all been made, on the basis of the latent period described, by using the linear model. When a portion of the time elapsed since radiation exposure has been excluded (e.g., the first year after radiation exposure in the case of thyroid neoplasms), the excluded period of time has been subtracted from the mean latent period figure in the calculation of risk.

In discussing thyroid neoplasms, a linear, no-threshold risk model has been used since the possibility that damage to a single cell could eventually result in a neoplasm cannot be excluded. For hypothyroidism, a linear model with a threshold has been postulated since a large number of cells would probably have to be altered to result in hypothyroidism because of the large functional reserve capacity of the thyroid gland.

H2 ACUTE EFFECTS OF IONIZING RADIATION ON THE THYROID

Radiation thyroiditis is an acute condition characterized by inflammation of the thyroid gland. It usually occurs 3 to 10 days after the ingestion of radioiodine and results in the eventual necrosis of some or all cells. Beierwaltes et al. (1956) have reported that, although it may occur in 4 to 5% of patients with thyrotoxicosis treated with iodine-131, it is usually so mild that patients had to be carefully questioned in order to establish the presence of symptoms. These authors also found that symptoms of increased thyrotoxicosis associated with radiation thyroiditis were

usually associated with higher doses of iodine-131 and were apparently considered to be less likely below single doses of 13 millicuries or approximately 17,400 rem to the thyroid, assuming a mean 45-gram gland size, a mean uptake at 24 hours of 65%, and an effective half-life of 6 days.

Segal (1958) evaluated 65 euthyroid patients with severe ischemic heart disease treated by thyroid ablative doses of iodine-131. Three of the 65 patients (4.6%) died shortly after therapy, with radiation thyroiditis as a contributing factor (Segal, 1958). The estimated thyroid doses in those three patients, assuming a 20-gram thyroid and a 6-day effective half-life, were in the range of 70,000 to 125,000 rem. None of the patients who received less than 32,000 rem (7 millicuries of iodine-131 retained in an assumed 20-gram gland with a 6-day effective half-life) developed radiation thyroiditis.

On the basis of the above data, radiation thyroiditis would seem to be highly unlikely in euthyroid individuals at radiation doses of iodine-131 below 25,000 rem. (The value of 25,000 rem is the average of 17,400 rem (Beierwaltes et al., 1956) and 34,000 rem (Segal, 1958).) At that level, mild symptoms might occur in 4 to 5% of the population, or in approximately 4500 persons in a population of 100,000.

Unpublished data from the University of Cincinnati and the University of Michigan suggest that very high doses (more than 200,000 rem) of iodine-131, administered for the ablation of residual thyroid tissue after thyroidectomy for thyroid cancer, may induce radiation thyroiditis in 90% of such patients. The symptoms were found to be severe in 2 of 67 patients (3%) so treated in Cincinnati.

Thus, there appears to be a threshold for radiation thyroiditis in euthyroid individuals at about 25,000 rem. At slightly above that dose level, approximately 4.5% of patients will develop symptoms. An additional 5% of exposed individuals are estimated to develop thyroiditis for each 10,000-rem increment above 25,000 rem.

Thyroid storm was found to occur in 21 of 2329 (0.9%) patients hospitalized for thyroid problems (Nelson et al., 1969) and was most commonly associated with infection. Death has been found to occur in approximately 20% of cases of thyroid storm (Ingbar et al., 1974). Although thyroid storm has been observed after exposure to iodine-131 (Shafer et al., 1971; Krishnamurthy et al., 1974), it is extremely rare.

Data from the National Health Survey (U.S. Dept. of Health, Education and Welfare, 1974) suggest, on the basis of in-patient utilization of short-stay hospitals, that 29 cases of thyrotoxicosis occur per 100,000 population. Even if all of these highly susceptible individuals were accidentally exposed to sufficient iodine-131 to induce radiation thyroiditis, the resultant number of cases would be only 29 per 100,000 population. If thyroid storm occurred in all of these 29 cases per 100,000 population, an extremely unlikely circumstance, a maximum of 20%, or 5.8 in 100,000, might be expected to die.

All of the patients treated by Segal (1958) had severe heart disease, the prevalence of which was approximately 328 cases per 100,000 population in 1972, based on mortality figures (U.S. Bureau of Census, 1974). Even if this highly selected group were exposed to more than 25,000 rem from iodine-131, the mortality attributable to the ensuing radiation thyroiditis complicating the underlying disease would probably not exceed 4.6%, or 15 deaths in 100,000 persons exposed to those doses.

It also seems likely that most deaths due to accelerated thyroid hormone release would be limited to patients with preexisting thyrotoxicosis or severe ischemic heart disease. Total deaths within these two groups would not be expected to exceed 20.8 per 100,000 population exposed to thyroid doses of more than 25,000 rem from iodine-131. Symptoms of radiation thyroiditis could be treated in most cases by aspirin; in the most severe instances adrenocortical steroids and beta-adrenergic-blocking agents would be required.

No human data on radiation thyroiditis after acute or fractionated external radiation therapy or accidental exposure have been reported. The absence of such findings may be due to relatively small doses or dose fractionation permitting recovery.

H3 CONTINUING EFFECTS OF IONIZING RADIATION ON THYROID FUNCTION--HYPOTHYROIDISM

H3.1 CRITERIA FOR HYPOTHYROIDISM

In analyzing data in which "clinical hypothyroidism" was used as the criterion for thyroid hypofunction, it can only be assumed that the physicians reporting such judgments were equally competent and precise in making the diagnosis. Patients who were given thyroid hormone replacement are also assumed to have shown a definite need for that medication.

Abnormally high levels of thyroid stimulating hormone (TSH) and/or depression of other parameters of thyroid function (T_4, protein-bound iodine, response to TSH stimulation) have been accepted as biochemical evidence of thyroid dysfunction. If the results of such tests were found to be abnormal in the absence of clinical hypothyroidism, the patient was classified as being biochemically hypothyroid.

H3.2 SPONTANEOUS FREQUENCY OF HYPOTHYROIDISM

Tunbridge et al. (1975a) found that in England clinical hypothyroidism was present in 1% of 2000 randomly selected individuals out of a population of approximately 20,000. Their mean age was 46.4 years. Additional unpublished data presented in Boston at the June 1975 Annual Meeting of the American Thyroid Association suggest that hypothyroidism is present in 0.017% (17 cases per 100,000 live births) of the newborn population in North America. Assuming that a linear curve fitted to these two points (0.017% at age 0 and 1% at age 46 years) gives a reasonable estimate of the prevalence in the entire population, these data permit an estimate of the prevalence and incidence of spontaneous hypothyroidism in the general population by age (see Table VI H-1).

H3.3 HYPOTHYROIDISM IN THE FIRST YEAR FOLLOWING RADIATION TO THE THYROID

H3.3.1 External Exposure

There are no reported cases of hypothyroidism occurring within the first year after external doses to the thyroid of less than approximately 2000 rem. Since a 2000-rem dose to the whole body is lethal, exposures of this magnitude from external sources do not appear to represent a realistic thyroid problem.

H3.3.2 Iodine-131

Total ablation of the thyroid within the first year after exposure has been shown to require at least 27,000 rem (Segal, 1958; Goolden et al., 1963) and is always associated with hypothyroidism.

Hypothyroidism without total thyroid ablation can also occur within 1 year of exposure to lower doses from iodine-131. In the Cooperative Thyrotoxicosis Follow-up Study (Becker et al., 1971), clinical hypothyroidism was noted to occur in many cases within the first year following therapy, as shown in Figure VI H-1.

The Cooperative Thyrotoxicosis Follow-up Study (Becker et al., 1971) collected data on 6000 patients treated solely with a single dose of iodine-131. These data have been used in estimating the radiation dose to the thyroid by multiplying the thyroidal concentration of iodine-131 by 91 rem/μCi-g. This calculation assumes a 6-day effective half-life, which may slightly overestimate the dose in patients with thyrotoxicosis but is reasonably close to the half-life in euthyroid individuals. The dose-response relationship for the development of hypothyroidism 1 and 5 years after treatment is shown in Table VI H-2. The 5-year data reflect the analysis presented in section H.3.4.2 below.

There is no evidence to suggest that patients with Graves' disease are more resistant to radiation than are normal persons. Indeed, because of the more rapid iodine turnover rate in persons with thyrotoxicosis, and because of the remote possibility that a patient would become hypothyroid without therapy within the first year after onset, these estimates are assumed to give a maximal dose-response estimate of the magnitude of the problem of hypothyroidism in the first year after iodine-131 ingestion.

H3.4 HYPOTHYROIDISM DEVELOPING MORE THAN 1 YEAR AFTER EXPOSURE TO RADIOIODINE

H3.4.1 Hypothyroidism After Low-Dose (<2500 rem) Exposure to Iodine-131

There are very few data on thyroid doses from iodine-131 of less than 2500 rem. Preliminary results of a follow-up survey of individuals regarded as having normal thyroids after diagnostic iodine-131 tests at ages of less than 16 years (Hamilton and Tompkins, 1975) suggest that 8 of 443 (1.8%) subsequently became hypothyroid. Two additional patients were excluded because they had a goiter before exposure to iodine-131. One was hypothyroid with a dose of less than 10 rem, and the other was not with a dose of 200 to 300 rem. In the hypothyroid subjects the mean time elapsed since exposure to iodine-131 was 14 years and the mean age at followup was 25 years. A summary of these preliminary data is presented by cohort in Table VI H-3.

When available information on the individual data points constituting the groups shown in Table VI H-3 was examined using a linear regression plot of dose versus hypothyroidism, it suggested a slope of 0.000069 case per rem. Assuming a mean latent period of 14 years, the slope was used to estimate a risk of hypothyroidism of approximately 4.9 cases per million persons per rem per year. Since $0.02\%\ yr^{-1}$ x 14 yr = 0.28% and 0.28% x 443 = 1.2, then 1.2 cases would have been expected to occur spontaneously in a nonexposed population of this size; furthermore, the possibility cannot be excluded that the study population may not have been entirely normal before the iodine-131 exposure. Thus it would seem probable that the risk of hypothyroidism at dose levels of less than 2500 rem is not significantly different from the risk estimated from data on patients with Graves' disease treated at dose levels of more than 2500 rem. Because of the larger numbers in the latter group, risk estimates derived from data on patients with Graves' disease will be used to apply to all dose ranges (see section H3.4.2 below).

It also seems likely that a large number of thyroid cells would have to be significantly damaged before clinical hypothyroidism would occur. For example, in many people, half or more of the thyroid can be surgically removed without causing clinical hypothyroidism. For this reason, a realistic estimate of the risk of hypothyroidism from iodine-131 should probably include a threshold below which any effect is negligible.

The data presented in Table VI H-3 indicate that no cases of hypothyroidism attributable to radiation occurred in the 146 patients with a mean dose of 18 rem at least 14 years previously. Rallison et al. (1974) found only two cases of overt hypothyroidism in 1378 children exposed to iodine-131 fallout compared to no cases in 3453 nonirradiated controls (Rallison, 1975). The average follow-up time was 16 years, and the mean dose to the thyroid was considered to be either 18 or 46 rem by two different estimates (Rallison et al., 1974). Statistical analysis of these data shows that the difference in hypothyroidism incidence in the irradiated and nonirradiated groups is not statistically significant ($p = .15$) (Fleiss, 1973). Thus, it would seem reasonable, in making a realistic estimate of the consequences of exposure to iodine-131, to consider that the threshold for the induction of hypothyroidism is 20 rem.

H3.4.2 Hypothyroidism After High-Dose (>2500 rem) Exposure to Iodine-131

The most comprehensive data on this subject are those reported by Becker et al. (1971) for a series of 6000 patients who received single doses of iodine-131 as therapy for Graves' disease. Becker and associates presented their data in terms of the cumulative probability of hypothyroidism at different dose levels (in terms of microcuries of iodine-131 per gram) over a 15-year follow-up period. The curves are shown in Figure VI H-1.

The 5-year follow-up data have been selected for analysis, and the mean amounts of iodine-131 in microcuries per gram have been converted to rem by multiplying by 91 rem/μCi-g, as noted in section H3.3.2.

Furthermore, in analyzing the outcome of 5200 surgically treated patients more than 2 years after surgery, Becker et al. (1971) found that the cumulative probability of becoming hypothyroid increases in a linear manner. Their figure of 0.7% per year probability of hypothyroidism after surgery has been used to estimate the rate of spontaneous hypothyroidism in the Graves' disease population. This interpretation has been based on the assumptions that the linear portion of the curve reflects spontaneous hypothyroidism in the Graves' disease population and that none of the patients was hypothyroid at the time of entry into the study. A cumulative probability of 3.5% (0.7% yr^{-1} x 5 yr) has thus been subtracted from the iodine-131 cumulative probability dose-response curves at an arbitrary 5-year follow-up time to give an estimate of the probability of hypothyroidism from iodine-131 exposure alone (see Table VI H-2).

Analysis of the 5-year data suggests a linear correlation (Figure VI H-2) between the radiation dose to the thyroid from iodine-131 and the probability of hypothyroidism above a lower limit of approximately 2500 rems--the lowest dose for which data are available in the study by Becker et al. (1971); it also suggests that a dose of approximately 57,000 rem would render all individuals hypothyroid by 5 years after the exposure.

Five-year follow-up data on 28 adult euthyroid patients treated with a mean dose of 32,000 rem from iodine-131 for cardiac disease revealed that 80% of them were clinically hypothyroid (Chapman, 1975). Segal (1958) found that a mean dose of 49,000 rem was required to render 65 euthyroid adult cardiac patients hypothyroid by iodine-131 therapy. Since they were all judged to be euthyroid before entry into these two studies and since the follow-up times were so short, no "adjustment" for spontaneous hypothyroidism has been made. Although they represent smaller and older series of patients, these data suggest that, if the dose-response curve for euthyroid individuals exposed to iodine-131 is linear, it is very similar to that indicated by the dose-response curve derived from the Graves' disease population. Therefore, in calculating risk estimates for the general population, data derived from patients with Graves' disease are used since they come from a larger population and would appear to provide an accurate predictor of outcome.

In the Cooperative Thyrotoxicosis Follow-up Study, the bias of age at the time of diagnosis on choice of therapy and frequency of follow-up was so strong at certain of the participating medical centers that it prevented any final conclusions regarding the relationship between age at exposure and outcome. Nevertheless, every analysis the investigators performed failed to demonstrate a relationship between age at exposure and subsequent hypothyroidism (Tompkins, 1975).

On the basis of the data derived from the Cooperative Thyrotoxicosis Follow-up Study, the absolute risk of hypothyroidism after treatment of Graves' disease with iodine-131 in doses greater than 2500 rem is 4.6 cases per million persons per rem per year.

H3.4.3 Hypothyroidism from Exposure to a Mixture of Radioiodines

Chapman et al. (1946) have published data on 22 patients treated for thyrotoxicosis with a mixture of radioiodines (approximately 90% iodine-130 and 10% iodine-131). The mean dose was 55.6 millicuries of iodine-130, with an estimated additional dose of 5.6 millicuries of iodine-131. Converting these values to rem yields a mean total of 20,300 rem, of which 12,400 rem was due to iodine-130 and 7900 rem was due to iodine-131. The mean age of patients at the time of therapy was 41.6 years, and the mean follow-up time was 20 months. Four of the patients had become hypothyroid during that time.

Based on these 22 cases, the absolute risk of developing hypothyroidism is approximately 5 cases per million per rem per year. This value is not significantly different from the 4.6 cases per million per rem per year from iodine-131 alone. This similarity of risk estimates suggests that for induction of hypothyroidism the short-lived iodine-130 (physical half-life 0.51 days) is no different from that for iodine-131 (physical half-life 8 days).

Summary

Exposure to iodine-131 appears to be linearly correlated with the induction of clinical hypothyroidism, with an estimated absolute risk of 4.6 cases per million per rem per year. Limited data support the concept that there is a threshold for this effect at approximately 20 rem.

H3.4.4 Clinical Hypothyroidism After Exposure to External Sources of Ionizing Radiation

In a 24-year followup of patients who had received a mean dose of 399 rem to the thyroid in early childhood for the treatment of benign disease, Hempelmann (1967) found that none of 105 patients who were examined was clinically hypothyroid. The mean age of the patients was 24 years. Similarly, at a mean follow-up period of 28 years, Refetoff et al. (1975) found no clinical hypothyroidism in 100 patients exposed to incidental external irradiation of the thyroid in childhood. Precise thyroidal doses are not available from that publication, but seem to have been less than 1000 rem.

Shafer (1975) found that 2 of 29 patients (6.9%) who received external radiation therapy alone for head and neck tumors were clinically hypothyroid at a mean follow-up time of 18 months. When the Ellis correction (Hall, 1973) was applied to the data to more closely approximate the estimate of a single-dose exposure, the mean dose to the thyroid was estimated to be 1700 ret. For the type of radiation involved, ret values are assumed to be roughly equal to a single exposure in rem.[1] The mean age of the patients at treatment was 59 years.

Rogoway et al. (1966) reported that none of 50 patients (4 to 52 years old) receiving external radiotherapy for lymphoma over a 3-year period became overtly hypothyroid. Their dose range of 4000 to 6000 rem to the neck would be roughly equivalent to 1570 ret, assuming maximal doses to the thyroid (Einhorn et al., 1972).

When the data of Shafer (1975) and Rogoway et al. (1966) are combined, they suggest that at a mean follow-up period of about 18 months after thyroid exposure to approximately 1640 ret the prevalence of hypothyroidism was 2.5% (2 cases in 79) and the incidence was 1.67% per year. The expected incidence of spontaneous hypothyroidism would be 0.02% per year. Therefore, the incidence of radiation-induced hypothyroidism would be 1.65% per year at this dose level.

It is of interest that Markson (1965) also documented the occurrence of hypothyroidism after external irradiation at relatively high total doses (3000 to 5000 rem). According to Einhorn and associates (1972), this range is equivalent to approximately 1350 ret. Incidence values are not calculated because the size of the population from which Markson derived his cases was not specified.

Hempelmann et al. (1952) reported the case of a 34-year-old man (case 4) accidentally exposed to fast neutrons and gamma rays. Ten years later he became clinically hypothyroid, and at a post-mortem approximately 20 years later, only microscopic remnants of thyroid tissue were found (Lushbaugh, 1975). Recalculation of the radiation doses (Los Alamos Scientific Laboratory) indicated whole-body doses of approximately 195 rads from fast neutrons and approximately 13 rads from gamma rays. Due to nonuniform irradiation to the body, the dose to the thyroid was approximately twice the average dose to the body (Lushbaugh, 1975), and the quality factor for the neutrons was 4 (Bond, 1975). Therefore, the radiation dose to the thyroid would have been 195 x 2 x 4 = 1560 rem from the neutrons and 13 x 2 = 26 rem from the gamma rays, or approximately 1586 rem total dose to the thyroid.

[1] The ret (rad equivalent therapy) values were determined by using the Ellis equation (Hall, 1973), $D = (NSD)T^{0.11}N^{0.24}$, where D is the total dose that will produce a given biological effect (tolerance) when delivered according to a treatment regime defined by the overall treatment time T(days) and the number of fractions, N. The nominal standard dose, NSD, may be thought of as approximating the dose that, given in a single exposure, would produce the given biological effect. The designated unit for NSD is the ret.

Summary

External radiation to the thyroid appears to be associated with the induction of clinical hypothyroidism at dose levels above 1000 rem and has not been reported to induce hypothyroidism at dose below 1000 rem. Very few data are available to permit an estimate of risk because of the small number of cases and short followup times. Nonetheless, from the data presented above, the absolute risk estimate for the induction of hypothyroidism by external irradiation, assuming a linear model, is 10 cases per million per rem per year. Although this is approximately twice the risk estimated for iodine-131, the numbers are too small and the assumptions in the estimate too great to establish this ratio with certainty. On a purely radiobiological basis, the more uniform distribution within the thyroid of the dose from external irradiation might well increase the efficiency of inducing clinical hypothyroidism, but further data are needed to establish or to refute this point.

H4 LATENT EFFECTS: THYROID NODULES AND RADIATION EXPOSURE

H4.1 PREVALENCE OF THYROID NODULES IN THE GENERAL POPULATION

Recent data from works in progress by Tunbridge et al. (1975b) suggest that the prevalence of clinically detectable thyroid nodules is as shown by age group in Table VI H-4.

The prevalence rates in this British study can be compared to similar data from the Framingham study (Vander et al., 1968) (see Table VI H-5). In the latter study, the population was surveyed at ages 30 to 50 years and again 15 years later. The totals in Table VI H-6 reflect only the prevalences in the 45-65 age groups.

The Framingham study was made on residents of the Northeastern United States, which is not considered to be within the "goiter belt." In reviewing the paper of Mortensen et al. (1955) from the Mayo Clinic (whose patients are assumed to come primarily from the Midwest "goiter belt"), it was observed that in 887 persons whose median age was approximately 60 years and who were considered to have had adequate thyroid evaluation there were 44 patients (5%) with palpable nodules.

These data are consistent with the values of Tunbridge et al. (1975b) and were used in conjunction with the latter in estimating the spontaneous prevalence of thyroid nodules in persons older than 18 years.

A recent survey (Trowbridge et al., 1975) of 7785 American children between the ages of 9 to 16 years found early and/or definite thyroid nodules in 17 subjects, or approximately 0.22%. These subjects were from the states of Michigan, Kentucky, Georgia, and Texas, and presumably represent a cross-section of the U.S. population.

For the current calculations, a linear regression line was fitted to these data points (Fig. VI H-3) as the best approximation. A maximum nodular malignancy rate of 10% was assumed in calculating the number of expected cancers from the number of total thyroid nodules in patients below age 20 and a rate of 12% in patients over age 20 (Messaris et al., 1973) (Fig. VI H-4).

The estimates that follow will be used in all subsequent discussions of nodules where comparison with the general population seems appropriate. Only nodules diagnosed after the first year after radiation exposure will be considered as possibly related to that exposure (Table VI H-6).

H4.2 EFFECTS OF HIGH-DOSE (>2,500 REM TO THE THYROID) EXPOSURE TO IODINE-131

In the Cooperative Thyrotoxicosis Follow-up Study (Dobyns et al., 1974) the smallest dose given to patients with Graves' disease was approximately 2500 rem at 25 μCi/g of iodine-131 in the gland. This group of patients is therefore considered to represent high-dose exposure. It should also be noted that over 98% of this population was 20 years or older at the time of treatment.

H4.2.1 "Spontaneous" Nodularity in Patients With Graves' Disease

Dobyns et al. (1974) reported that 1553 of 10,013 (15.5%) patients with Graves' disease had palpable thyroid nodules prior to surgery. Their mean age was 40.1 years, the incidence of palpable nodules thus being 0.39% per year. Nine of 1553 nodules (0.6%) were subsequently proved to be thyroid cancer; the remainder were benign. Of 19,186 similar patients who were subsequently given iodine-131 as the primary treatment, 3144 (16.4%) had palpable thyroid nodules prior to therapy (Dobyns et al., 1974). Their mean age was 49.7 years, and the incidence of palpable thyroid nodules was therefore 0.33% per year. There is no significant difference between these two incidences.

From these data, the spontaneous incidence of benign and malignant nodules in patients with untreated Graves' disease can be estimated as follows: the incidence of total palpable nodularity in patients with Graves' disease is $(0.33 + 0.39)/2 = 0.36\%$ per year. Of these, 0.6% will be malignant, resulting in an expected incidence of cancer of $0.36 \times 0.006 = 0.002\%$ per year. The expected incidence of benign nodules will be $0.36 \times 0.994 = 0.358\%$ per year.

H4.2.2 Thyroid Nodules Occurring After Iodine-131 Therapy

H4.2.2.1 Adults

Dobyns et al. (1974) found that 86 of 16,042 patients with Graves' disease without palpable nodules at the time of radioiodine therapy were subsequently operated for palpable nodules developing more than 1 year after iodine-131 therapy. Nine (10.5%) had cancer, and 77 (89.5%) had benign lesions. An additional 494 in 16,042 were found to have developed palpable nodules more than 1 year after iodine-131 therapy, but had not undergone surgery by the end of the study. Though the 494 unoperated patients might be assumed to be less likely to have cancer than the 86 selected for surgery, a conservative estimate would be to assume that the prevalence of cancer and benign lesions was the same in the 494 unoperated as in the 86 operated patients. This would result in predictions of $10.5\% \times 494 = 52$ additional cases of cancer and $89.5\% \times 494 = 442$ additional benign nodules. Therefore, the number of palpable nodules occurring at more than 1 year after iodine-131 therapy is estimated to be $52 + 9 = 61$ cancers per 16,042 patients for a prevalence of 0.38% and $442 + 77 = 519$ benign nodules per 16,042 for a prevalence of 3.2%. The mean follow-up time was 8 years, but the first year was excluded by definition of post-occurring cases, resulting in a 7-year risk period (Dobyns et al., 1974). These data indicate an observed incidence of cancer of 0.054% per year and an observed incidence of benign nodules of 0.457% per year for an observed incidence of total nodules of $0.054 + 0.457 = 0.511\%$ per year. The mean radiation dose in these patients was approximately 8755 rem to the thyroid.

H4.2.2.2 Children

Safa et al. (1975) have reported on 273 patients treated between the ages of 1 and 20 years with iodine-131 for Graves' disease. There were 31 additional cases of children aged 16 years or less who were treated with iodine-131 in the Cooperative Thyrotoxicosis Follow-up Study (Tompkins, 1975). Pooling of these observations reveals two cases of thyroid cancer ($2/304 = 0.66\%$) and 17 benign nodules ($17/304 = 5.6\%$) in the combined population followed after iodine-131 therapy. Estimates of thyroid dose and follow-up period were available from 271 of 304 subjects and suggested a mean radiation dose of about 9000 rem and a mean follow-up time of about 11 years. These values are assumed to apply to the entire group of 304 subjects.

Thus, the two cancers that were found in 304 patients result in an observed prevalence of 0.66% and an observed incidence of 0.06% per year. Seventeen of the 304 patients had benign nodules, for an incidence of 0.51% per year. The corresponding observed incidence of total nodules would be $0.51 + 0.06 = 0.57\%$ per year.

H4.2.3 Euthyroid Cardiac Patients Treated with High-Dose Radioiodine Therapy

E. M. Chapman (1975) and Freedberg et al. (1952) found no nodules attributable to iodine-131 "thyroid ablative" therapy in a total of 201 adult patients so treated. In Chapman's series, the mean dose was 32,000 rem and the mean follow-up time was 2.2 years. No such data exist for patients younger than 20 years of age.

H4.2.4 Risk Estimates for the Induction of Thyroid Nodules by Iodine-131

Based on the observed and expected incidence noted above, in the adult Graves' disease population, the absolute risk for thyroid cancer appears to be 0.06 case per million persons per rem per year, and the risk for total palpable thyroid nodules appears to be 0.18 case per million per rem per year.

Similarly, in persons younger than 20 years of age at the time of therapy for Graves' disease, the absolute risk of thyroid cancer appears to be 0.064 case per million per rem per year, and for total palpable nodules it appears to be 0.23 case per million per rem per year. In this instance, the total population is small, and it is possible, though unlikely, that the cases occurred by chance.

The risk for thyroid cancer after exposure to therapeutic quantities of iodine-131 for Graves' disease seems linear and independent of age at the time of exposure. Its value is approximately 0.062 case per million per rem per year [(0.06 + 0.064)/ 2 = 0.062].

All of these estimates make the assumption of linearity and other assumptions as noted above.

In considering the development of thyroid neoplasms by quartile age distribution of the population of Graves' disease patients who developed thyroid nodules more than 1 year after iodine-131 therapy, Dobyns et al. (1974) found a significantly higher ($p < .05$) proportion of benign neoplasms in patients treated at or below age 35 than in older groups of similar size. There was no such association between the development of malignant neoplasms and age at exposure to iodine-131. It should be noted that in the study by Dobyns et al. (1974) there was no evidence that benign lesions would become malignant (Tompkins, 1975).

H4.3 EFFECTS OF LOW-DOSE (<2500 REM) EXPOSURE TO IODINE-131

In a survey of 5179 children, of whom 1378 had been exposed to a maximum of 120 rem with a mean value of 18 rem to the thyroid from iodine-131 fallout in the western United States, Rallison et al. (1974) could find no significant differences between irradiated and nonirradiated subjects in the prevalence of thyroid nodules, benign or malignant.

Preliminary data (Hamilton and Tompkins, 1975) suggest that at slightly higher doses (mean doses of 94 rem to the thyroid, with a range of less than 10 to 1900 rem), 6 individuals in 443 were found to have benign thyroid nodules and none in 443 were found to have cancer of the thyroid at least 16 years later. Both the prevalence of total thyroid nodules (6/443 = 1.4%) and the prevalence of cancer (0/443 = 0%) were lower than would have been expected in the general population aged 20 to 39 years (see Table VI H-6). Furthermore, there was no correlation ($p > 0.8$) between dose and the incidence of nodules.

To date there has been only one case report of thyroid cancer after low-dose iodine-131 exposure (Pilch et al., 1973). That patient was a 20-year old woman who developed papillary cancer of the thyroid after a total of approximately 240 rem from diagnostic iodine-131 at ages 4 and 12 years. No clear association between the exposure and the cancer could be made because of a lack of any adequate control or study group.

Although these data suggest that there might be a threshold for the induction of thyroid nodules by iodine-131, the numbers involved are so small that the absence of cases might have occurred by chance at the 95% confidence level.

H4.4.1 Low Doses (<1500 rem) to the Thyroid

A recent examination of 1192 persons treated with external irradiation for benign diseases in childhood revealed a relationship between the dose received by the thyroid and subsequent thyroid neoplasia (Colman, 1975). The mean follow-up time was 28 years. These data were used to estimate the prevalence of detectable clinical nodules and projected thyroid cancers at different dose levels. These estimates were adjusted for an expected prevalence in the general population aged 20 to 39 years of 0.31% for thyroid cancer and 2.6% for all thyroid nodules. The results are shown in Table VI H-7.

Hempelmann et al. (1975) recently reported a survey of 2872 young adults given x-ray therapy for benign disease in early childhood and of 5055 nonirradiated siblings. The mean number of years at risk was 24.2 for the irradiated and 22.9 for the nonirradiated subjects. Table VI H-8 shows the estimates obtained from this data after adjusting for findings of no thyroid cancers and of six benign thyroid neoplasms in the control population [0% cancers and 0.12% benign (in this case total) nodules].

There is no apparent difference between the slopes of linear regression lines fitted to these two sets of cancer incidence data (slope for Colman's data = 0.00029; slope for the data of Hempelmann et al. = 0.00036).

At lower doses, Hempelmann (1968) found one surgically proved cancer of the thyroid in 985 (0.10%) patients in the Ann Arbor series who received an estimated dose of 20 rem to the thyroid. He also estimated that only one in three clinically detectable nodules was surgically removed, which would result in an estimate of cancer in 0.30% of the cases. Similar analysis of his data would predict that 2.7% would have benign neoplasms, for an overall prevalence of 3% with nodules. The resulting incidence would be 0.011% per year for thyroid cancer and 0.11% per year for total thyroid nodules. After adjustment for the expected incidence in the general population, these data suggest that the incidence of thyroid cancers and nodules induced by a dose of 20 rem would be 0.001 and 0.023% per year, respectively. The mean follow-up time was approximately 28 years.

Modan et al. (1974) found 10 more cases of thyroid cancer in 10,902 patients who had received x-irradiation in childhood than in the same number of controls (12 in irradiated and 2 in nonirradiated groups). The mean latent period was 12.3 years, and the estimated thyroid dose was 6.5 rem. These data suggest a thyroid cancer prevalence of 0.09% (10/10,902) at 6.5 rem.

Albert et al. (1968) found two cases of benign thyroid nodules in 2043 patients who received 6 rem to the thyroid during radiation therapy for tinea capitis and none in 1413 nonirradiated controls. The mean follow-up period was 13.1 years, and the mean age at exposure 7.4 years. Their data suggest a prevalence of benign thyroid nodules of 0.10% (2/2043) at 6 rem. No thyroid cancers were identified in this study. Thus, the incidence of benign thyroid nodules and, in this case, of total thyroid nodules, appears to be 0.007% per year at 6 rem.

The combined data of Modan et al. (1974) and of Albert et al. (1968) suggest a thyroid cancer prevalence of 0.08% (10/12,945) at a thyroid dose of approximately 6 rem from external irradiation in childhood. With a mean follow-up time of 12.4 years, the resulting incidence of thyroid cancer is 0.006% per year.

Using the data from the above studies, an estimate of risk was obtained, based on a linear dose-response curve using 18 points for total nodules and 14 points for cancer. The equation of the line is as follows: incidence = slope x dose + constant. The constant (or intercept) was not significantly different from zero in either case. Though other methods of calculation are possible, this method gave consistent estimates. From these slopes, the absolute risk for thyroid cancer and total nodules was found to be 4.3 and 12.4 cases per million per rem per year, respectively.

H4.4.2 High Doses (>1500 rem) to the Thyroid

In a review of 4673 patients treated with external irradiation in childhood, Beach et al. (1962) found that none of the 23 patients with carcinoma had received more than 1270 rem to the thyroid. Although only 12 patients could be shown definitely to have received more than that dose, it was suggested that, at doses above 2000 rem, thyroid neoplasms would probably not be induced by external irradiation of the thyroid.

Hanford and associates (1962) found thyroid cancer in 7 of 162 patients who had been irradiated for tuberculous adenitis. None of eight patients who received more than 1600 rem had developed thyroid cancer at a mean follow-up time of 17 years. At a mean follow-up time of 22.5 years, DeLawter et al. (1963) similarly found no thyroid cancers in 222 patients who had received a mean dose to the thyroid of approximately 2100 rem. The majority of these patients had been treated for diffuse goiter. Markson (1965) was also unable to demonstrate, at follow-up 1.8 years later, any nodularity in five patients who had received a mean thyroid dose of 3500 rem. Although Wilson et al. (1958) reported two cases of thyroid carcinoma after doses possibly higher than 2000 rem, accurate dosimetry was not available.

It would appear, then, that external irradiation of the thyroid at doses higher than 1500 rem is not clearly associated with the induction of thyroid cancer.

If the protective mechanism is complete destruction of thyroidal cells, then benign neoplasms would theoretically be as unlikely as malignant lesions, although there are almost no data on the incidence of benign nodules after external irradiation doses of more than 1500 rem to the thyroid.

Summary

External irradiation of the thyroid in doses lower than 1500 rem appears to be linearly related to the induction of thyroid nodules, both benign and malignant. Limited data suggest that the induction of thyroid neoplasms falls off rapidly at doses higher than 1500 rem. Absolute risk estimates from linear regression lines fitted to these data suggest that at doses lower than 1500 rem the absolute risk for total thyroid nodules is 12.4 cases per million persons per rem per year; the risk for thyroid cancer is 4.3 cases per million persons per rem per year.

H4.5 LATENT PERIOD AND INDUCTION OF THYROID NEOPLASMS BY EXTERNAL IRRADIATION

Beach et al. (1962) and Raventos et al. (1964) examined 660 cases of thyroid cancer occurring after external irradiation in childhood. The cumulative frequency showed a rapid rise and then approached a plateau 15 to 25 years after exposure. There is a statistically significant difference in the average latent period between the data of Beach (8.9 years) and the data of Raventos (10.9 years). However, for the purposes at hand and their clinical implications, these data can be combined since the biases in the collection of data outweigh the statistical variation. It appears from these data that the latent period has a log-normal distribution. If data on individual patients are combined, the distribution has a mean value of 10.5 years for the 660 cases, with 2σ limits of 3.6 to 30.8 years. If the studies are weighted equally and combined, the mean latent period is 9.9 years, with 2σ limits of 3.4 to 28.7 years.

More recent data from the Chicago area (Colman, 1975), collected at a mean follow-up time of 28 years, suggest that even after approximately 20 years, there is still an excess number of cases of thyroid cancer after external irradiation in childhood. This observation is consistent with the findings of Hempelmann et al. (1975), which suggest that the latent period for radiation-induced thyroid neoplasms may be longer than 30 years. Goolden (1958) has reported thyroid cancer as long as 40 years after irradiation. It should be noted that the latent period (measured as time from irradiation to detection) is probably longer than the interval between exposure to radiation and the appearance of the neoplasm. In a similar group of patients, Winship et al. (1970) found that the average interval between early clinical evidence of a neoplasm and its confirmation at surgery was almost 2 years.

H4.6 MIXED EXPOSURE TO EXTERNAL AND INTERNAL IRRADIATION

H4.6.1 Relationship Between Age at Exposure and Induction of Thyroid Neoplasms

Conard et al. (1975) have recently reported a 20-year follow-up study of Marshall Islanders accidentally exposed to radioactive fallout. The data for the combined exposed populations from the islands of Utirik, Ailingnae, and Rongelap are presented in Table VI H-9.

The difference between the prevalence of total thyroid nodules in the two age groups is probably significant ($p < .1$) and suggests that children may be twice as susceptible as adults to the induction of benign thyroid nodules by mixed external and internal (radioiodine) exposures. There is no significant difference in the thyroid cancer data.

H4.6.2 Comparison of Actual Findings of Nodularity with Predictions from Risk Factors Developed from Children Exposed to Iodine-131 or x-Rays

James (1974) suggested that in the Rongelap population approximately 36% of the internal radiation dose to the thyroid was due to iodine-131 and approximately 64% was due to other isotopes of iodine. Cole (1972) suggested that exposures from the other iodine isotopes were best considered to be equivalent to external irradiation in the induction of thyroid neoplasms. When these considerations are applied to the combined exposed populations from Rongelap, Ailingnae, and Utirik (Conord et al., 1975), the mean radiation dose appears to be approximately 46 rem from iodine-131 and 143 rem from the combination of external sources and internal sources of radioiodines other than iodine-131.

Of the 239 exposed persons, 4 were noted to have developed thyroid cancer 20 years later, for an incidence of 0.083% per year. None of 331 unexposed Marshallese in Rongelap and Likiep was found to have thyroid cancer over the same period. Thus, all four cancers appear to be attributable to the radiation exposure, although the numbers are small. Based on the risk factors outlined in Table VI H-10, three cancers would have been predicted at the radiation doses outlined above over a 20-year period.

When a similar analysis is applied to total nodules, the difference between 25 observed lesions attributable to radiation (34 in exposed minus 9 in a theoretical unexposed population of the same size based on incidence values obtained from the actual unexposed population at 20-year followup) and the 9 predicted from risk factors in Table VI H-10 is significant. This is probably due to the fact that the risk factors are based on clinically palpable nodules, whereas in the Marshallese a "high degree of scrutiny and suspicion led to the detection and removal of nodules when they were much smaller than nodules usually encountered in general clinical practice" (Conard et al., 1975).

H5 ESTIMATES OF RISK

The calculation of risk, in terms of the number of cases per million persons per rem per year, has traditionally been done according to the formula

$$\text{risk} = \text{number of cases } \left(\frac{10^6 \text{ persons}}{\text{persons at risk}}\right) \text{ (average number of years at risk/person)}^{-1} \text{ (average dose/person)}^{-1}$$

This definition has the effect of considering all of the cases occurring in the study population to be attributed to the mean dose received. By this method, risk is forced to be linear with dose as determined by the line connecting the origin and the mean dose point.

Whenever possible, dose-response curves have been based on data compiled from many sources. For these cases, risk appeared to be most accurately defined by analysis of the linear regression fit of the data. Absolute risks are applicable in all cases because appropriate corrections have been made for the expected number of cases in similar, nonirradiated populations. Risk estimates are shown in Table VI H-10.

H6 CONCLUSIONS

From the data presented above, it has been possible to postulate a model for predicting the thyroid effects that could result from exposure to iodine-131 and to external radiation. These estimates are presented below. The dividing point in age for all thyroid disease is taken to be 20 years since at that point the thyroid gland has passed the rapid-growth phase of childhood and adolescence. For convenience, persons younger than 20 years at exposure will be referred to as children, and all others will be called adults.

Acute Effects--Radiation Thyroiditis

Age at exposure. There is no evidence that age at exposure is related to thyroiditis, assuming equal doses in all age groups.

Threshold. Radiation thyroiditis appears to have a threshold of 25,000 rem to the thyroid.

Risks. Risks are outlined in the text.

Continuing Effects--Hypothyroidism

Age at exposure. There is no apparent relationship between age at exposure and subsequent hypothyroidism, assuming equal radiation doses.

Risk. The risk of hypothyroidism would appear to be best expressed using a linear estimate of 4.6 cases per million persons per rem per year above a 20-rem threshold. Until proved otherwise, all sources of radiation will be considered to be equally effective as a central bound estimate. For an upper bound estimate, external radiation might be considered to be at least two to three times as effective as iodine-131 based on the risk factors given in Table VI H-10. The length of time over which this effect would occur in a euthyroid population is unknown. In the majority (90%) of Graves' disease patients treated with iodine-131 this effect would have occurred within ~25 years.

Latent Effects--Thyroid Neoplasia

Age at exposure. From the data on the Marshall Islanders and on children with thyrotoxicosis given high-dose (>2500 rem) iodine-131 therapy, it appears that children are twice as susceptible as adults to the induction of benign neoplasms. Children and adults appear to be equally susceptible to the induction of cancer from iodine-131, mixed radioiodines, and external irradiation.

Iodine-131. More than 50,000 rem to the thyroid appears to cause thyroid ablation with no subsequent risk of neoplasm in either age group.

Risk estimates for the induction of thyroid nodules, including cancer, by iodine-131 have been presented in the text. The estimated risks for children are 0.064 and 0.23 per million persons per rem per year for cancer and for total nodules, respectively, whereas the estimated risks for children from external irradiation are 4.3 and 12.4 per million persons per rem per year for cancer and for total nodules respectively. Thus the ratios of iodine-131 risks to external x-ray risks are 1/67 for cancer and 1/53 for total nodules. These factors are smaller than the 1/10 to 1/20 derived by other workers (Klassovskii, 1967) from animal data. It would therefore seem reasonable to use a factor of 1/10 for an upper bound estimate, 1/20 for a central estimate, and 1/60 for a lower bound estimate of the efficacy of iodine-131 compared to x-rays in the induction of thyroid neoplasia. These factors could then be used to convert doses from iodine-131 to "equivalent" doses of external radiation in calculating the risk of thyroid nodules. The risk factors based on the large and more extensively studied population whose thyroids were incidentally exposed to external radiation could then be used.

External radiation. Above 2500 rem, there is no evidence of the induction of neoplasms, benign or malignant. At doses lower than 1500 rem, a linear no-threshold model suggests an absolute risk in children of 4.3 and 12.4 per million persons per rem per year for thyroid cancer and for total nodules, respectively. Assuming that adults are only half as sensitive as children to the induction of benign thyroid nodules by external irradiation, the risk for adults would be 4.4 and 8.4 per million persons per rem per year for cancer and for total nodules, respectively (benign = total minus cancer = 12.4 - 4.4 = 8. 8 ÷ 2 = 4. Total = benign + cancer = 4 + 4.4 = 8.4 for adults).

At doses of 1500 to 2500 rem there appears to be a gradual decrease, the average absolute risk in this region being assumed to be approximately half of that in the region below 1500 rem.

A comparison of these risk calculations with other estimates obtained from recent surveys is given in Table VI H-11.

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TABLE VI H-1 ESTIMATES OF SPONTANEOUS HYPOTHYROIDISM IN THE GENERAL POPULATION

Age Range (yr)	Prevalence of Hypothyroidism (%)	Incidence of Hypothyroidism (% yr^{-1})
0-9	0.10	0.02
10-19	0.31	0.02
20-29	0.52	0.02
30-39	0.74	0.02
40-49	0.95	0.02
50-59	1.17	0.02
60-69	1.38	0.02
70-79	1.59	0.02

TABLE VI H-2 PROBABILITY OF HYPOTHYROIDISM AFTER EXPOSURE TO IODINE-131

Estimated Thyroidal Concentration of Iodine-131 (μCi/g)	Estimated Dose to Thyroid (rem)	Estimated Probability of Hypothyroidism Within	
		1 Year	5 Years
25-50	3,400	0.06	0.13
51-75	5,750	0.10	0.17
76-100	8,000	0.14	0.20
101-125	10,300	0.16	0.24
126-150	12,600	0.17	0.18
151-175	14,900	0.19	0.31
176-200	17,150	0.21	0.24
201-225	19,400	0.22	0.45

TABLE VI H-3 RELATIONSHIP BETWEEN LOW-DOSE EXPOSURE TO IODINE-131 IN CHILDREN AND SUBSEQUENT HYPOTHYROIDISM(a)

Number of Subjects	Thyroid Dose Range (rem)	Estimated Mean Thyroid Dose (rem)	Number Hypothyroid	Incidence of Hypothyroidism (% yr^{-1})
146	10 to 30	18	0	0
146	31 to 80	52	3	0.15
151	81 to 1900	233	5	0.23

(a) Preliminary results (Hamilton and Tompkins, 1975).

TABLE VI H-4 PREVALENCE OF PALPABLE THYROID NODULES IN AN ADULT ENGLISH POPULATION[a]

Age (years)	Number of Subjects	Number of Subjects with Nodule(s)	Percent of Subjects with Nodules
Males:			
18-34	341	0	0
35-54	544	7	1.3
55-74	340	3	0.9
75+	57	0	0
Subtotal	1282	10	0.8
Females:			
18-34	446	17	3.8
35-54	511	23	4.5
55-75	447	31	6.9
75+	77	7	9.1
Subtotal	1481	78	5.3
Total Population:			
18-34	787	17	2.2
35-54	1055	30	2.8
55-74	787	34	4.3
75+	134	7	5.2
Total	2763	88	3.2

(a) Data from Tunbridge et al. (1975b).

TABLE VI H-5 PREVALENCE OF PALPABLE THYROID NODULES IN AN ADULT NORTH AMERICAN POPULATION[a]

Age (years)	Number of Subjects	Number of Subjects with Nodules[b]	Percent of Subjects with Nodules
Males:			
30-50	2282	35	1.5
45-65		55	2.4
Females:			
30-50	2845	183	6.4
45-65		230	8.1
Total	5127	285	5.6

(a) Data from Vander et al. (1968)

(b) Cases at 45-65 include cases at 30-50. Total is therefore of cases at 45-65.

TABLE VI H-6 ESTIMATED OCCURRENCE OF THYROID NEOPLASMS IN THE GENERAL POPULATION

Age (years)	Estimated Prevalence of Nodularity (%)	Estimated Incidence of Nodularity (% yr^{-1})	Estimated Prevalence of Cancers (%)	Estimated Incidence of Cancer (% yr^{-1})
0-19	0.8	0.080	0.08	0.008
20-39	2.6	0.087	0.31	0.01
40-59	4.3	0.086	0.52	0.01
60-80	6.1	0.087	0.73	0.01

TABLE VI H-7 ESTIMATES FROM THE DATA OF COLMAN (1975)

Estimated Mean Dose to Midplane of Neck (rem)[a]	Estimated Prevalence of Cancer (%) After Adjusting for Spontaneous Prevalence	Estimated Incidence of Cancer (% yr^{-1})	Estimated Prevalence of All Nodules (%) After Adjusting for Spontaneous Prevalence	Estimated Incidence of All Nodules (% yr^{-1})
57			7.5	0.27
150	7.2	0.26	12.5	0.45
400			4.5	0.16
600	12.2	0.44	19.5	0.70
730	8.2	0.29	25.0	0.89
800	9.2	0.33	25.5	0.91
876	6.2	0.22	32.0	1.14
1050			26.0	0.93
1250			55.5	1.98
1430	19.2	0.69	48.5	1.73

[a] The estimated mean dose to the midplane of the neck is assumed to be the mean thyroidal dose.

TABLE VI H-8 ESTIMATES FROM THE DATA OF HEMPELMANN ET AL. (1975)

Mean Thyroid Dose (rem)	Estimated Prevalence of Thyroid Cancer[a] (%) After Adjusting for Prevalence in Controls	Estimated Incidence[b] of Cancer (% yr^{-1})	Estimated Prevalence of Total Thyroid Nodules (%) After Adjusting for Prevalence in Controls	Estimated Incidence of Total Nodules (% yr^{-1})
17.2	0.061	0.0029	0.430	0.0201
137	0.474	0.0188	3.671	0.1457
220	1.263	0.0528	2.405	0.1006
346	1.123	0.0330	7.745	0.2278
426	3.955	0.1280	7.790	0.2521
648	7.447	0.2334	19.029	0.5965

[a] 268 patients are excluded because the thyroid dose was not known.

[b] Based on mean number of years at risk (age) for each dose group.

TABLE VI H-9 AGE AT EXPOSURE AND INDUCTION OF THYROID NEOPLASMS--20-YEAR FOLLOW-UP OF THE MARSHALL ISLANDERS[a]

Age at Exposure	Total Thyroid Nodules	Thyroid Cancers
Less than 18 years	22/117	2/117
More than 18 years	12/122	2/122

[a] Data from Conard et al. (1975).

TABLE VI H-10 ABSOLUTE RISK[a] OF THYROID ABNORMALITIES AFTER EXPOSURE TO IONIZING RADIATION

Type of Abnormality and Population Surveyed	Mean Dose or Dose Range (rem) for Which Data Were Available	Absolute Risk[a]	Statistical Risk Range[b]
	Internal Irradiation (Iodine-131)		
Thyroid nodularity:			
Children	9000	0.23	0 to 0.52
Adults	8755	0.18	0.13 to 0.23
Thyroid Cancer:			
Children	9000	0.06	0 to 0.158
Adults	8755	0.06	0.044 to 0.075
Hypothyroidism:			
"Low dose"--children	<10 to 1900	4.9[c]	3.9 to 22.9
"High dose"--Adults[d]	2500 to 20,000	4.6[c]	2.8 to 7.8[e]
	External Irradiation		
Thyroid nodularity in children	0 to 1500	12.4	4 to 47.4[e]
Thyroid cancer in children	0 to 1500	4.3	1.6 to 17.3[e]
Hypothyroidism in adults	1640	10.2	0 to 24.8

[a] In number of cases per million persons per rem per year.

[b] Unless otherwise indicated, the risk range was determined by using the assumption that the number of cases, n, out of the population at risk represents the true mean of a Poisson distribution. The range is then estimated by using $\pm 2\sqrt{n}$ as the 95% confidence level.

[c] Threshold of 20 rem.

[d] See Figure VI H-2.

[e] In these cases, the risk was determined from the slope of the linear regression line. The range was estimated from the extreme data points, which provide the lowest and highest slopes.

TABLE VI H-11 COMPARISON OF RISK ESTIMATES FOR THYROID CANCER IN CHILDREN AND ADULTS

Source	Population	Type of Exposure and Radiation	Mean Tissue Dose or Dose Range (rads)	Risk (cases 10^{-6} rem^{-1} yr^{-1})
BEIR Report (1972)	Children	Thymic x-ray, general population	229	2.5
		Thymic x-ray, high-risk group	329	9.3[a]
	Children	Japanese atom bomb survivors, gamma and neutron	143	2.6
UNSCEAR (1972)	Male adults and children	Japanese atom bomb survivors, gamma and neutron	25 to 200	1 to 2
	Female adults and children	Japanese atom bomb survivors, gamma and neutron	25 to 200	2 to 4
	Male and female infants	X-ray to neck	50 to 600	2.5
This report (1975)	Adults and children	External irradiation	0 to 1500	4.3
	Adults and children	External irradiation	1500 to 2500	2.2

[a] Modified recently (Hempelmann et al., 1975) to a value of 4.0.

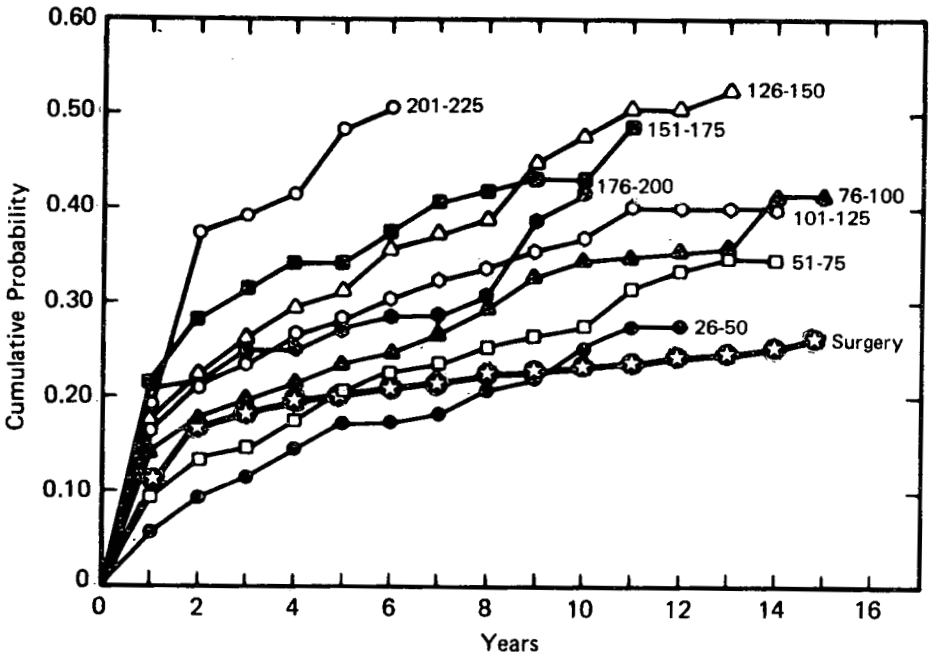

FIGURE VI H-1 Probability of becoming hypothyroid with a single treatment of iodine-131. The numbers on the curves show the iodine-131 dose in microcuries per gram of thyroid.
From the Cooperative Thyrotoxicosis Follow-up Study, Becker et al. (1971)

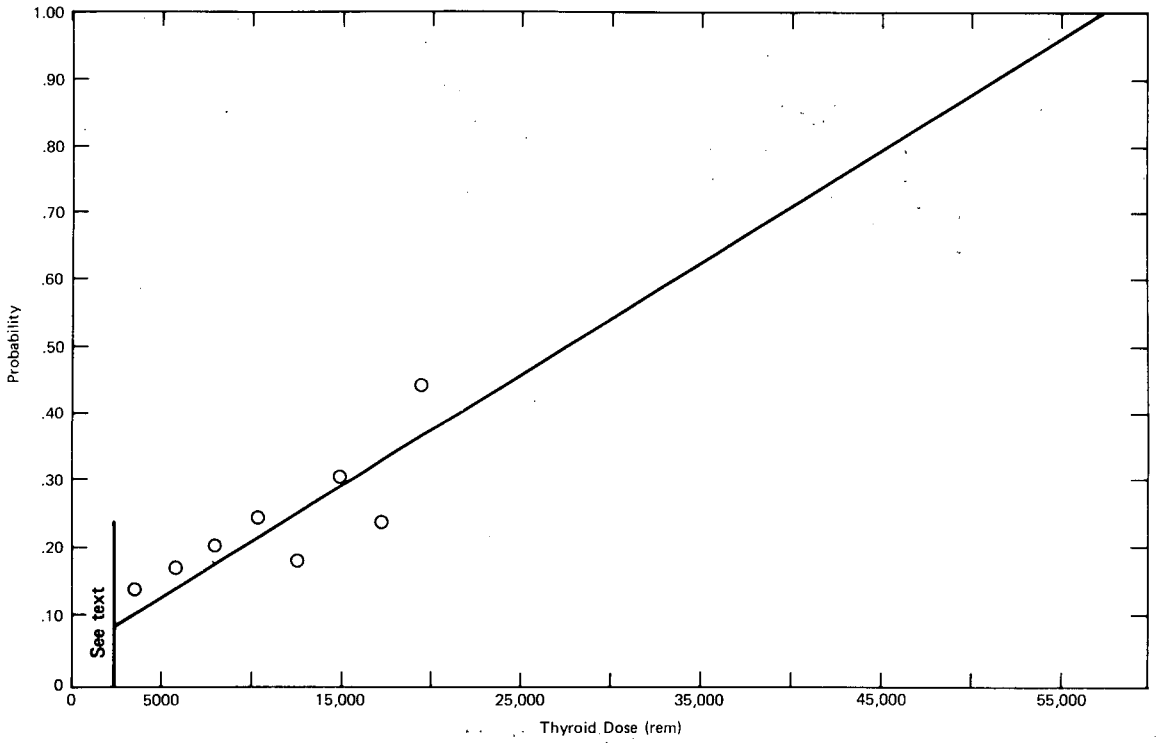

FIGURE VI H-2 Probability of hypothyroidism induction by iodine-131 doses higher than 2500 rem. Data from the Cooperative Thyrotoxicosis Follow-up Study (Becker et al., 1971) at 5 years after exposure. See Table VI H-2.

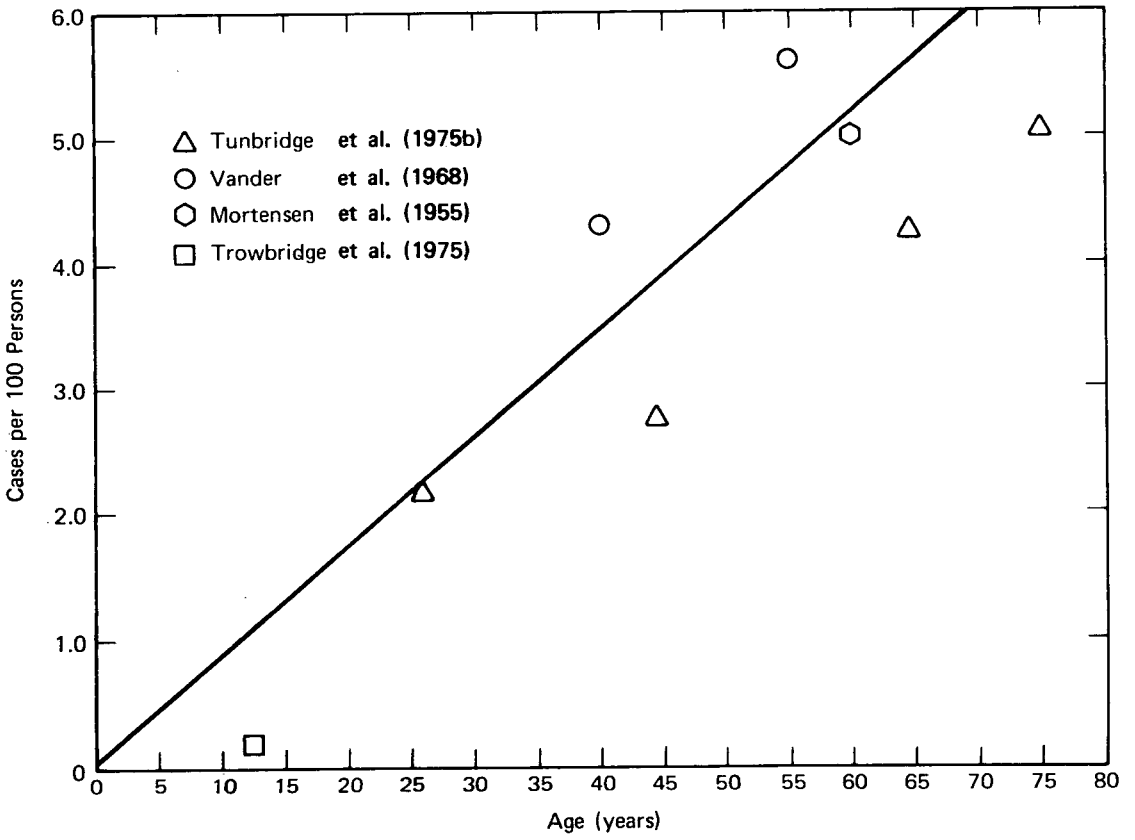

FIGURE VI H-3 Prevalence of spontaneous thyroid nodules in the general population.

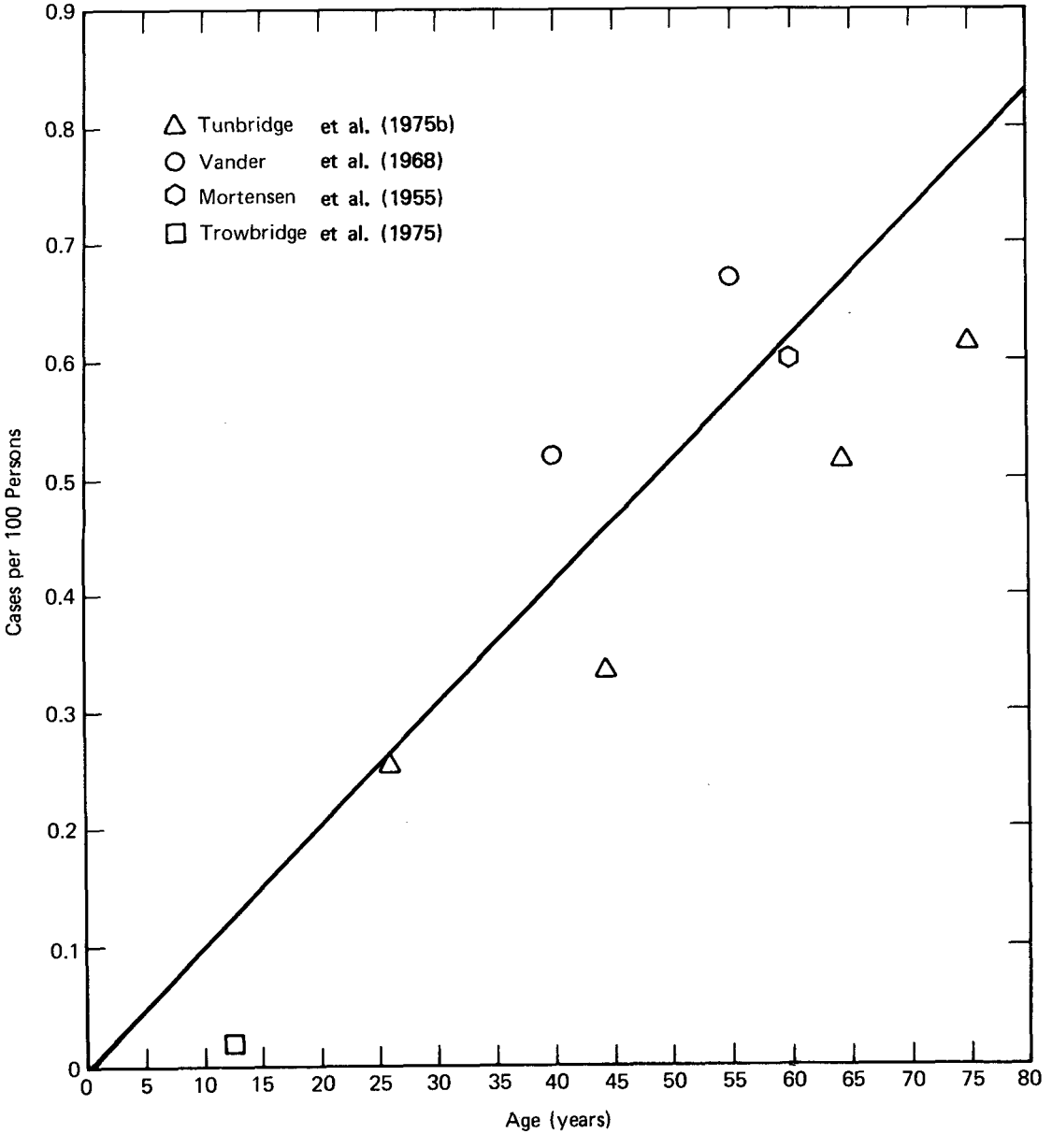

FIGURE VI H-4 Prevalence of spontaneous thyroid cancers in the general population.

Appendix I

Genetic Effects

I1 INTRODUCTION

This appendix discusses the assumptions and methods used to arrive at the calculations presented in section 9.4 and describes in some detail the principal types of genetic damage caused by ionizing radiation.

I2 POPULATION CHARACTERISTICS

There are many possible scenarios for exposure of the population, depending on such factors as the nature of the accident and the population density and distribution in the vicinity of the accident. Accordingly, the incidences of genetic disorders estimated in section 9.4 are per million of population and per rem of exposure. Estimates for future generations assume a stable population size and composition, with births and deaths in balance, and with negligible migration. (Changes in population size would affect the numbers of affected individuals, but not the probability of being affected. Migration would alter the spatial distribution of affected individuals, but not the numbers.) The estimated incidences can be applied to specific situations by multiplying by the appropriate factors for population size and exposure.

Age distributions in local populations may vary and may be accompanied by variations in birth rates, etc. The population assumed for the calculations is identical in such parameters as age distribution, sex, birth rate, and generation period with the current U.S. population as a whole and is based on census estimates for the year 1974 (Bureau of Census, 1974), the most recent available, as well as 1973 data on the distribution of live births by paternal age (National Center for Health Statistics, 1975). Figure VI I-1 shows the age distribution in the U.S. population, and I-2 Figure VI I-2 shows the distribution of live births by paternal age.

The paternal age was used in making the estimates because, in the mouse, male germ cells are much more sensitive to radiation than are female germ cells. For the base calculations, a period of 30 years was arbitrarily adopted for analysis of the effects of irradiation on successive generations; this is the generation period used by the BEIR Committee (1972) in its estimates of genetic damage.[1] The population data cited above show that the human generation period in the United States is presently about 28 years, and the final estimates were based on the real distribution of paternal ages. The probability of increases in the incidence of the various classes of genetic disorders was estimated in terms of the probable numbers of additional cases per year or per 30-year generation, per million in the population. Since generations overlap, this type of calculation makes it simpler to estimate effects whose expression will be summarized by 30-year intervals.

I3 TYPES OF RADIATION EXPOSURE AND DOSES

I3.1 EXTERNAL AND INTERNAL RADIATION EXPOSURES

In calculating the genetic effects of a reactor accident, external and internal radiation exposures were treated separately, and each was broken up into a number of time intervals over which the radiation dose was assumed to be accumulating. Both the population alive at the time of the accident and their descendants would be exposed to external radiation (mostly from contaminated ground). In contrast, only the population alive at the time of the accident would be exposed to internal radiation from incorporated radionuclides (almost exclusively through inhalation during the passage of the radioactive cloud).

[1] The committee referred to is the Advisory Committee on the Biological Effects of Ionizing Radiations (BEIR) of the National Academy of Sciences-National Research Council. Its report, The Effects on Populations of Exposure to Low Levels of Ionizing Radiation (1972), will be hereafter referred to as the BEIR Report.

I3.2 DOSES

The exposure to be anticipated from a reactor accident would occur at low intensities: less than 1 rem per minute. The individual integrated testis dose over all time is assumed not to exceed 50 rem. The fraction of man-rem to whole-body (testes) contributed by individuals receiving total doses in excess of 50 rem is 13% as stated in section 13.1.

Although there would be considerable variation in individual exposures, only the average exposure of the population (or segment of the population), expressed as the dose (in rem) to the testis of the male, was considered. The reasons for selecting the testis as the target organ are explained in section I4.

It is obvious that not all individual exposures are equally significant genetically: a young child is expected to have more offspring in the future than an aged adult. Estimates of genetic effects must be based, therefore, on the doses received by the reproductive cells of individuals and weighted according to the expected numbers of future offspring. The effect of weighting the dose, known as the genetically significant dose, was arrived at by estimating the fractional contribution of each 5-year age group of fathers to the population of infants born in each of twelve 5-year time intervals after the accident. The estimates were made for 5-year intervals because the data on paternal ages (shown in Figure VI I-2) were so grouped. In the final tabulations, however, the data are presented for the two 30-year periods immediately following the accident, after summing the effects in the six 5-year intervals in each.

For reasons explained below in the discussion of differential sensitivity, fetal exposures were not considered separately. However, fetal exposures are implicitly included in the calculations by selection of the generation period.

I4 DIFFERENTIAL SENSITIVITY

Germ cells may differ greatly in their responses to radiation, depending on the type and stage of development. The total damage to the genetic material in germ cells will depend on the fraction of the total radiation dose experienced at each of the developmental stages leading to the mature, functional germ cell. It is obvious, therefore, that the cell types and stages involved in the greater part of the germ-cell life cycle are of greatest concern in estimating hazards.

The selection of the testis as the target organ was based on a large body of experimental data on the mouse showing that the male germ cells are much more sensitive to radiation than are the female germ cells (see, for example, Russell, 1965; BEIR Report, 1972; UNSCEAR, 1972).

Fetal exposure was not considered separately, since fetal germ cells do not differ greatly in sensitivity from the spermatogonial cells of the male (see literature summary in the Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1972). Primordial germ cells in the female fetus would account for only a small fraction of the total effective exposure and would contribute perhaps 1% of the total estimated effects.

Taking these factors into account, the BEIR Report (1972) averaged the male and female mutation rates in the mouse to arrive at the value of 0.25×10^{-7} as the average per locus, per rem mutation rate for low-intensity irradiation. The assumption that human female germ cells behave like those of the mouse could introduce an error of perhaps a factor of 2.

I5 PRINCIPAL TYPES OF RADIATION-INDUCED GENETIC DAMAGE

I5.1 GENE MUTATIONS

The genetic material (deoxyribose nucleic acid, DNA) is organized in structures called chromosomes, which consist of a large number of genes, aligned in linear sequence. Each individual gene is a portion of the DNA involved in its own unique function, usually served by its specifying some biologically important molecule (a protein). The unique informational properties of the gene depend on the unique sequence of smaller molecular components (nucleotides) that make up its structure.

There are four possible nucleotides that can occupy any given position in the gene. Substituting one nucleotide for another at a specific position in the gene can change the informational content of the gene, just as a change in one letter may alter the meaning of a word. The effect may be to change the nature of the protein that the gene specifies or to change the quantity, so that there might be more, less, or none at all. Individual genes or parts of genes may also be lost, and resultant loss of function may have deleterious consequences.

The effects of a gene mutation will depend on (1) the type of chromosome that carries the gene (sex chromosome or autosome) and (2) the manner in which gene interaction leads to the development of a specific trait. Hereditary characters, including genetic disorders, may result from the substitution of a mutant gene at a single genetic locus (single-gene diseases or characters), or they may involve variation at more than one genetic locus (multifactorial, or polygenic, inheritance). Much more reliable predictions can be made for the effect of a changed mutation rate on the incidence of single-gene disorders than for its effect on the incidence of multifactorial disorders.

I5.1.1 Single-Gene Disorders

Single-gene disorders can be classified into three main groups: (1) Autosomal dominant, (2) autosomal recessive, and (3) sex-linked (i.e., X-chromosome-linked).

Autosomal dominant mutants are located on an autosome and produce effects when present singly (i.e., inherited from only one parent). Autosomal recessive genes, on the other hand, must be inherited from both parents for the mutant effect to be seen. In other words, with autosomal dominant traits, if one member of a pair of genes is normal and the other is defective, the mutant effect is seen. With recessive mutants, both members of a gene pair must be mutant, otherwise there is no evident effect. The situation with sex-linked genes is somewhat more complicated, but they behave more like the autosomal dominants than the autosomal recessives with respect to the time and degree of expression.

Autosomal dominant mutations will bring about increases in genetic disorders much more rapidly than will autosomal recessive mutations. This is to say, the incidence of autosomal dominant traits will be most clearly dependent on the mutation rate. A recent survey of the British Columbia population by Trimble and Doughty (1974) shows the more common autosomal dominant disorders to be such conditions as chondrodystrophy; osteogenesis imperfecta; neurofibromatosis; eye anomalies, including congenital cataract; and polydactylism. This abbreviated list accounts for about one-half of all dominant disorders observed in about 750,000 live births from 1952 through 1972.

Traits dependent on autosomal recessive mutant genes will show the slowest increases in incidence when the mutation rate is elevated. Any one kind of recessive mutant gene will be present in the population at a very low frequency, and the incidence of the corresponding trait will be approximately the square of the frequency of the recessive mutant gene. Hence, when small changes are made in mutant gene frequency [q increased to $(q + \Delta q)$], changes in the incidence of the recessive disorder will be very small [q^2 increased to $(q + \Delta q)^2$]. Since the spontaneous mutation rate will be of the order of magnitude of q^2 rather than of q, at least for the more serious disorders, it can be seen that a one-generation increase in mutation rate, even one that exceeded the spontaneous rate, would not result in an appreciable change in the incidence of this class of genetic diseases. Disorders due to autosomal recessive mutations would be expected, therefore, to increase imperceptibly. They include cystic fibrosis, phenylketonuria, albinism, deafness and impairment of hearing, and some forms of progressive muscular dystrophy. This abbreviated list accounts for about one-half of all cases listed by Trimble and Doughty (1974).

Sex-linked traits would show increases similar to those of the autosomal dominants when the mutation rate is increased. Among the more common abnormalities that are sex-linked in inheritance are hypogammaglobulinemia, color blindness, and some forms of progressive muscular dystrophy. These conditions account for about three-fourths of all sex-linked disorders listed by Trimble and Doughty (1974).

I5.1.2 Multifactorial Diseases

Multifactorial traits have a more complex pattern of inheritance than that of single-gene traits, since they depend on variation at more than one genetic locus. These diseases include a variety of congenital malformations and constitutional and degenerative diseases, such as spina bifida; ventricular and atrial septal defects, patent ductus arteriosus, and other heart and circulatory disorders; pyloric stenosis; cleft palate and/or cleft lip; hypospadias, and undescended testis; congenital dislocation and juvenile osteochondrosis of the hip; diabetes mellitus; various degrees of mental retardation; convergent strabismus; various forms of epilepsy; and asthma. This list accounts for about two-thirds of all multifactorial-disorder cases reported by Trimble and Doughty (1974).

I5.2 CHROMOSOMAL ABERRATIONS

The most serious consequences of gross changes in chromosomes, such as changes in number (numerical aberration, or aneuploidy) and changes in structural sequence (structural aberration, usually translocation), result from having the wrong amount - too much or too little - of the genetic material, rather than from intrinsic changes. The most common anomaly of this type in the British Columbia survey (Trimble and Doughty, 1974) was Down's syndrome, which arises from having one extra chromosome (No. 21). Other types of aneuploidy occur, as do also unbalanced conditions arising from certain kinds of segregations in bearers of translocations. The consequences may vary from moderate, as in the case of some sex-chromosome imbalances, to more severe cases of malformations in live-born children, to those severe enough to be lethal to the fertilized egg or embryo.

I6 CALCULATION OF INCIDENCE OF DISORDERS STEMMING FROM RADIATION-INDUCED GENETIC DAMAGE

I6.1 GENERAL ASSUMPTIONS AND METHODS

In arriving at the calculations presented in section 9.4, use was made of the BEIR Report.

The BEIR estimates were based primarily on the current incidence of serious disabilitie and this appears to be the most reliable and meaningful way of making estimates, especially in the case of single-gene disorders. Given that a genetic trait occurs and is maintained exclusively by recurrent mutation, reliable estimates can be made of its increase following an increased amount of mutation. The distribution in time of the added cases of genetic disorders can also be estimated. Since virtually all of the exposure to be anticipated from a reactor accident would occur at low intensities (i.e., less than 1 rem per minute), the BEIR estimates are applicable. However, although the study adopted the BEIR assumptions and made use of BEIR calculations of the levels of risk, the methods necessarily differ in several ways, as explained below.

The BEIR estimates are for 5-rem exposure in each generation, whereas the study chose to estimate damage per rem, to facilitate calculations. Thus, the BEIR estimates must be divided by 5.

The BEIR estimates were made per million live births. In order to have a common denominator for the expression of somatic and of genetic risks, the study estimated risks in terms of the total population (i.e., the expectation per million population of all ages). Recent census data show that currently there are about 14,000 live births per million population, or about 420,000 per 30-year period. Thus, the numbers predicted per year or per 30-year period (estimated as if it were made up of a single generation) can be derived from the BEIR values by multiplying the expectation per million by the factors 0.014 and 0.42, respectively. The estimates are then rounded to reduce the number of significant figures in order to avoid implying great precision.

The BEIR estimates were limited to estimating first-generation effects and effects anticipated at equilibrium. The objective of the study was to summarize effects over two 30-year periods immediately following the accident and overlapping the continuing exposures arising from the accident. To do this, it is necessary to take into account that generations actually overlap and that in any given time period the newborn population may be made up of other than the first-generation offspring of exposed parents.

It is necessary to distinguish between internal exposures from radionuclides in the body (almost exclusively inhaled during the passage of the radioactive cloud) and external exposure (mostly from contaminated ground). Internal exposure is limited to the population alive at the time of the accident; the dose rate declines and the population ages with time. External exposure affects both the population alive at the time of the accident and their descendants: the dose rate declines with time, but the entire age range of the population is subject to exposure. The declining dose rate and, in the former case, the aging population require that doses be estimated for separate and consecutive time periods after the accident.

It is difficult to place the radiation-induced genetic effects into perspective by comparing them with the spontaneous ones. Therefore, for each exposure mode and period, the time dependence of the accident-related genetic effects is expressed for the first and second 30-year periods after the accident, roughly corresponding to the first and second generations.

The method used by the study was to estimate for each type of exposure the effects of exposures experienced during a limited period of time, estimated on a per rem basis for the fraction of the population involved. When the appropriate testis doses are multiplied by the factors given in Tables VI 9-11 and VI 9-12 of section 9.4 and the products are summed, the net genetic consequences of a particular accident are obtained on a per million population basis.

I6.2 ESTIMATES OF INCREASES IN SINGLE-GENE DISORDERS (POINT MUTATION)

An increase in background radiation will result in an increased rate of occurrence of mutation. The amount of radiation that would produce as many additional mutations as were already occurring spontaneously -- i.e., a doubling the mutation rate -- is called the doubling dose.

Luning and Searle (1971) have estimated the doubling dose for point mutation in the mouse to be about 100 rads for low-intensity exposures. By using the data from the Oak Ridge specific-locus tests (UNSCEAR, 1972; Searle 1974), the study arrived at an estimate of about 170 rem for the mouse. For the human, UNSCEAR (1972) adopted 100 rads as the doubling dose, whereas the most recent studies of the Hiroshima and Nagasaki populations suggest a doubling dose of not less than about 140 rads for the male, and not less than 1000 rads for the female, based on estimates of damage resulting in death of offspring of irradiated parents (Neel et al., 1974). The BEIR lower limit of 20 rem appears to be much too conservative, and, for the induction of autosomal dominant mutations, its upper limit of 200 rem could also be too low. The study adopted the hopefully more realistic value of 100 rem as the doubling dose for point mutations in humans, a value that is well within the BEIR range. The use of one value gives single values rather than ranges in the tables presented in section 9.4. The reader who prefers the wider range can reconvert by multiplying the incidences by the factors 5.0 and 0.5.

The doubling dose is used in the following manner to make estimates of genetic damage. If a hereditary disorder is maintained exclusively by recurrent mutation, then the frequency of the mutant gene in the population will depend on the mutation rate. For autosomal dominants, it is assumed that there is an equilibrium between the occurrence of new mutation and the elimination of old mutations from the population, so that the incidence of the corresponding disease remains constant from generation to generation. When the mutation rate is altered, a new equilibrium incidence of the corresponding trait is reached, and the increase in incidence is proportional to the increase in mutation rate. Since the doubling dose is the dose sufficient to produce an additional amount of mutation equal to that occurring spontaneously, a doubling dose of 100 rem would mean that the exposure of each generation for a number of generations to a given dose would increase the mutation rate by 1/100 per rem: the mutation rate would be increased to about 1.01 times its old value for a 1-rem exposure, and at equilibrium there would be a corresponding increase in the incidence of autosomal dominant disorders. If the current incidence were 1.0%, the effect of 1 rem in each generation would be to increase this incidence to about 1.01%.

In the case of an increase in background radiation for any reason, the incidence of mutations will rise to an equilibrium value at which the production of new mutations is equal to the elimination of old (preexisting) mutations.

In the case of a one-time dose of radiation, however, (e.g., a reactor accident), the incidence of disorders would rise to a peak and then decline toward the original level, so that a one-time dose would result in a probable specific number of cases. The estimate of the incidence is based on the expectation that only about 80% of all mutant genes responsible for significant autosomal dominant disorders will be transmitted to the next generation. An elimination rate of 20% would thus lead to an increase from the old incidence of 1.0% to an incidence in the first generation after exposure of 1.002%. In succeeding generations, the incidences would decline (1.0016, 1.00128, etc.), finally returning to the preaccident incidence of 1.0%. Any mutation that is expressed in the first or in the later generations will have been induced in the germ cells of the exposed generation and will have been transmitted to the first generation of descendants. Expression in later generations is dependent on the rate of mutant-gene elimination.

The figures for current incidence of genetic disorders are derived from a survey of the Northern Ireland population (Stevenson, 1959), as interpreted by UNSCEAR (1958). The total incidence of autosomal dominant traits, approximately 1%, appears to be too high, resulting from the inclusion of such conditions as internal obstructive hydrocephaly, alopecia areata, and senile cataract, which collectively account for 40% of the incidence ascribed to autosomal dominants. On the other hand, the new British Columbia survey (Trimble and Doughty, 1974) estimates the total incidence of autosomal dominant traits to be about 0.1%. However, this list appears to have omissions (e.g., Huntington's chorea, polycystic renal disease), and it appears that dominant degenerative diseases appearing in adults have been underestimated. It seems likely that the true value for the incidence of autosomal dominant traits lies somewhere between these two estimates.

I6.3 ESTIMATES OF INCREASES IN MULTIFACTORIAL DISORDERS

Because of the involvement of multiple loci, it is difficult to assess the impact of changing the mutation rate on the incidence of multifactorial disorders. While there is uncertainty as to the extent of the effect of increased mutation, there is unanimity of opinion that there is no simple relation of multifactorial disorders to mutation, and that increases would be less than proportional to the dose. The BEIR Report (1972) recognized this uncertainty by asserting that the "mutational component" (i.e., the proportion of the incidence that could be considered to be proportional to the mutation rate) might lie between 5 and 50%, and this uncertainty was retained by the study in its estimate.

The BEIR estimate assumed that the first-generation expression would be one-tenth of that expected at equilibrium. This is equivalent to a rate of elimination such that the incidence due to radiation-induced mutation will decline by 10% in each succeeding generation, and this method was used to calculate the BEIR-type expectations for each succeeding generation. As with the autosomal dominant mutations, this assumes the elimination of mutant genes to be independent of frequency.

I6.4 ESTIMATES OF DISORDERS STEMMING FROM CHROMOSOMAL ABERRATIONS

The majority of chromosomal aberrations lead to spontaneous abortion, which often occurs so early in a pregnancy as to be undetectable. Unrecognized human abortion is difficult to quantify and even more difficult to assess from the standpoint of societal impact. However, there is no experimental evidence that the undetectable abortions induced by parental irradiation would be any more frequent than the detectable abortions from the same cause occurring in the first trimester of pregnancy. If the abortions occurring before and after implantation of the ovum in experimental mammals can be equated with undetectable and detectable abortions in the human, then there is experimental support for this opinion (see UNSCEAR, 1972).

Extensive studies of human abortuses show a large fraction to be associated with major abnormalities of the chromosomes, simple aneuploidy and unbalanced rearrangement being the two major categories. However, there is strong reason to believe that in experimental mammals virtually all of the postimplantation abortions following high-dose irradiation of the father are due to chromosome damage, and the estimates arrived at by the study are based on this premise.

The BEIR 1972 estimates of cytogenetic effects were used as the basis of calculations. It was recognized, as in the BEIR Report, that little of the effect seen in later generations will occur in the offspring of persons showing major effects in the first generation. Most of the affected individuals will be infertile, so that chromosomal imbalance seen in later generations will arise from adjacent segregation in the carriers of translocations, producing imbalance in the offspring. It was assumed that the damage remaining to be expressed, either in future spontaneous abortions or in viable individuals showing anomalies due to chromosomal imbalance, will decline by one-half in each succeeding generation. This assumption supposes that about one-half of all segregations give rise to gametes that are balanced.

I6.5 CALCULATED EFFECTS OF SPECIFIC EXPOSURE REGIMES

Table VI I-1 presents estimated incidences of disorders due to radiation-induced genetic damage. For convenience to the reader, the table also includes the BEIR Report data on which the Reactor Safety Study's estimates are based. For convenience of the reader, the principal differences between the BEIR estimates and the present ones are as follows.

The BEIR Report presented calculations based on 1 million live births. The present study uses a population base of 1 million, with a distribution that corresponds exactly in composition and characteristics to the present population of the United States. This corresponds to a live-birth rate of 14,000 per year, or 420,000 live births per million persons per 30 years.

The dose for which the BEIR figures were calculated was 5 rem. The present study uses a reference dose of 1 rem, and the BEIR estimates of effects are accordingly divided by a factor of 5 for this reason.

The doubling-dose range considered in the BEIR Report for genetic change is 20 to 200 rem per individual, and the values are presented as ranges. In the present study the doubling dose is taken to be 100 rem per individual, and the BEIR estimates of effects are adjusted by multiplying the lower value (for 200 rem) by 2 or the higher value (for 20 rem) by 0.2.

The values given in the BEIR Report for multifactorial disorders are based on a range of 5 to 50% for the "mutational component." This uncertainty factor is retained in the values used in the present study.

In calculating the effect of internal radiation, account must be taken of the fact that only the population alive at the time of the accident would be exposed and only their descendants would be affected by this dose. People born after the accident would not receive any significant internal exposure. The distribution of live births by fathers' age is presented in Fig. VI I-2. Table VI I-2 shows the division of each successive 30-year group of newborns among the successive generations. (Generation 1 is composed of those born after the accident to parents alive at the time of the accident.)

The values in Table VI I-2 are listed in two groups, according to the period over which the dose was accumulated. For internal dose, the periods used were the following: year 0-1, year 1-10, and 10 year periods thereafter. For external dose, the periods used were year 0-1, year 1-30, and years 31-60. The dose rates used are those pertinent to the midpoint of each period (except for year 0-1, which is conservatively calculated for time 0).

The net effect of unit dose over all future generations would depend on the probability of transmission for each type of disorder. These transmission probabilities for each type of disorder considered here are presented in Table VI I-3, together with the corresponding effects, calculated for a 30-year period as though it were made up of first generation øffspring (the numbers in the eighth column of Table VI I-1).

From Tables VI I-2 and VI I-3 the effect of unit dose for a given 30-year period is obtained from

$$E\,[a + (1 - s)b + (1 - s)^2c \ldots], \qquad \text{(VI I-1)}$$

where

a, b, c, d (shown in the column headings in Table VI I-2) are the fractions of newborns that are first, second, third, and fourth generation descendants of the exposed generation; E is the expected effect for a 30-year period, calculated as if it were made up exclusively of first-generation offspring; s is the probability of elimination, per generation (average rate of elimination); and (1 - s) is the probability of transmission, per generation. For example, the effect per rem on dominant disorders during the first 30 years of 1 rem accumulated during year 0-1 is

$$E\,[a + (1 - s)b]$$

$$8.4\,[0.87 + (1 - 0.2)0.124] = 8.15.$$

I7 SUMMARY AND CONCLUSIONS

Genetic damage induced in a parental generation will achieve its maximum expression in the immediate offspring of those parents, unless there is continuing exposure over one or more additional generations. Damaged genes and chromosomes will be transmitted to future generations, but the likelihood of transmission will be reduced according to the nature and severity of the effect. Transmission requires survival and reproduction, and adverse effects on these will reduce the transmission of genetic material by affected persons. Some damage, particularly chromosomal damage, is eliminated rapidly, whereas other types may take many generations to be eliminated from the population. Taking all kinds of damage into account, it would be perhaps 20 generations before the damage is reduced below the 1% level of the first generation.

The projected increases have been made by using the BEIR (1972) estimates, and there are reasons to believe that these may have been too high. One such reason is the new British Columbia survey of the incidences of handicapping conditions, which indicates that the incidence of autosomal dominants is lower by a factor of 10 than had previously been believed (Trimble and Doughty, 1974). Another reason is the belief of some that multifactorial traits may not be increased appreciably by changes in the mutation rate. There is further reason to believe that the estimated increases in chromosomal disorders may be far too high. These estimates have been made on the assumption that, at low doses effects are proportional to the dose. While it is true that any effect that is produced at very low doses must result from single-track events, it is by no means clear that this component of chromosomal damage is large enough to be significant, and it is possible that at low doses the induction of chromosomal aberrations would be negligible.

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TABLE VI I-1 NUMBER OF NATURALLY OCCURRING AND RADIATION-INDUCED DISORDERS, ACCORDING TO THE BEIR ANALYSIS AND ACCORDING TO THE PRESENT ANALYSIS

	BEIR Report			This Study					
		Effect of Radiation per 5 Rem Over One Generation per Million Live Births[a]		Normal Incidence for Total Population of 1 Million (14,000 Live Births per Year)		Effect of Radiation per Rem on Total Population of 1 Million (14,000 Live Births per Year)[b]			
Type of Disorder	Normal Incidence per Million Live Births	First Generation	Equilibrium	Per Year	Per 30 Years	Per Year	30 Years[c]	First Generation	Sum for All Generations
Single-gene disorders:									
Autosomal dominants	10,000	50-500	250-2500	140	4200	0.28	8.4	8.4	42
X-Chromosome linked	400	0-15	10-100	6	170	0.01	0.25		
Autosomal recessives	1500	Very few	Very slow increase	20	600	(d)	(d)	(j)	(f)
Multifactorial disorders	40,000	5-500	50-5000	560	16,800	0.03-0.28	0.84-8.4	0.84-8.4	8.4-84
Effects of chromosome aberrations:									
Congenital disorders from:									
Unbalanced rearrangements	1000	60	75	15	430	0.17	5	5.4	6.4
Aneuploidy[e]	4000	5	5	60	1700	0.013	0.4		
Spontaneous abortions from:									
Aneuploidy and polyploidy[f]	35,000	55	55	500	15,000	0.15	5	36	42
XO[g]	9000	15	15	130	40,000	0.05	1.3		
Unbalanced rearrangements	11,000	360	450	150	45,000	1	30		

[a] Doubling dose taken to be 20 to 200 rem.

[b] Doubling dose taken to be 100 rem.

[c] This method of calculation is an artifice that permits multiplying later by a fraction that shows the contribution of the first generation to any 30-year production of newborns.

[d] Negligible in comparison with the other effects.

[e] Having the wrong number of chromosomes, usually one extra.

[f] Having extra sets of chromosomes.

[g] Lacking one sex chromosome (Turner's syndrome).

TABLE VI I-2 COMPOSITION OF 30-YEAR POPULATIONS OF NEWBORNS*

Period of Accumulation of Exposure	30-year Post-Accident Period	Generation†			
		1 (a)	2 (b)	3 (c)	4 (d)
30-Year Intervals (used to calculate effects of external dose)					
0-1 year	First	0.871	0.124		
	Second	0.062	0.674	0.259	0.002
1-30 years	First	0.5	0.5		
	Second	0.431	0.544	0.021	
31-60 years	First	--			
	Second	0.871	0.124		
10-Year Intervals (used to calculate effects of internal dose)					
0-1 year	First	0.871	0.124		
	Second	0.062	0.674	0.259	0.002
1-10 years	First	0.705	0.039		
	Second	0.062	0.594	0.141	-
11-20 years	First	0.372			
	Second	0.062	0.301	0.018	
21-30 years	First	0.081			
	Second	0.062	0.054		
31-40 years	First	--			
	Second	0.024	0.002		
41-50 years	First	--			
	Second	0.001			

*The fractions assume the exposure behaves as if administered in a single, low-intensity exposure, as follows:

1. For doses accumulated over first year, as if at time zero.
2. For doses accumulated over 30-year periods, as if at year 15 or at year 45.
3. For doses accumulated over 10-year periods, as if at year 5, 15, 25, 35, or 45.

†See Equation (VI I-1).

TABLE VI I-3 TRANSMISSION PROBABILITIES FOR THE VARIOUS TYPES OF DISORDERS[a]

Disorder	Effect for a 30-Year Period as Though It Were Made up Exclusively of First-Generation Individuals (E)	Probability of Elimination, per Generation(s)
Dominant	8.4	20%
Multifactorial	0.84 to 8.4	10%
Chromosomal aberrations	5.4	50%[a]
Spontaneous abortions	36	50%[b]

(a) For children of parents with balanced rearrangements.

(b) For conceptions by parents with balanced rearrangements.

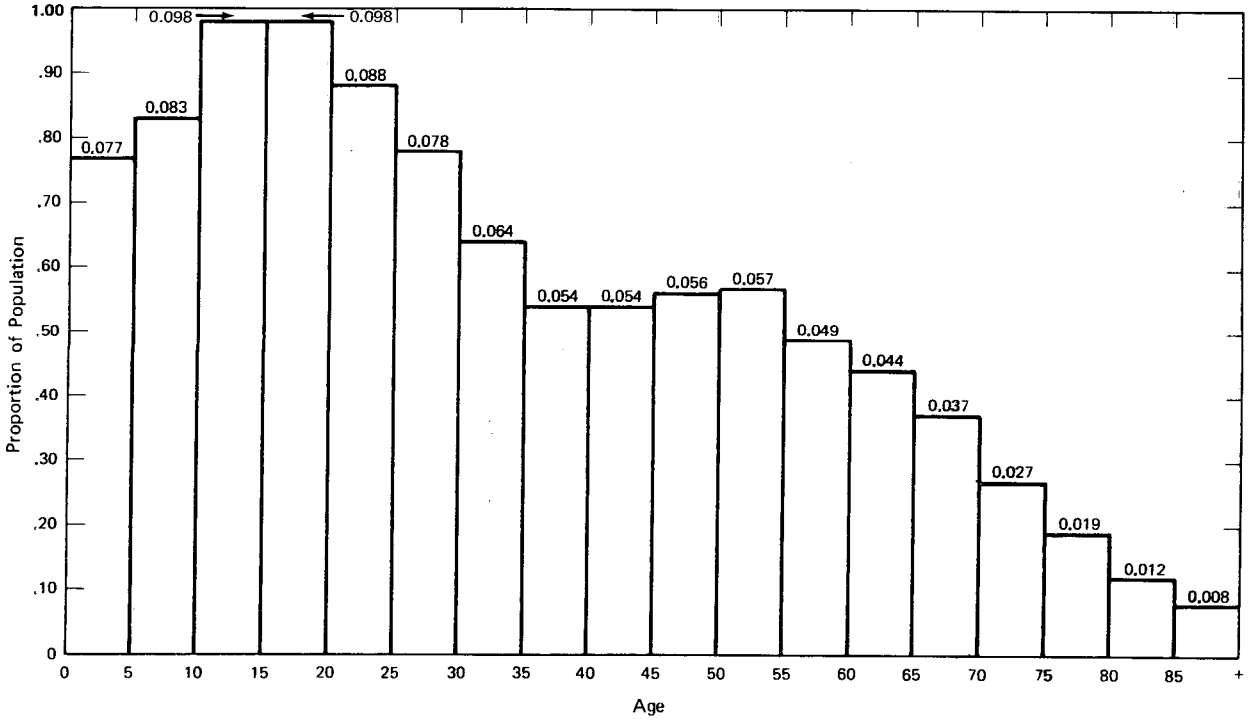

FIGURE VI I-1 Age distribution in U.S. population.

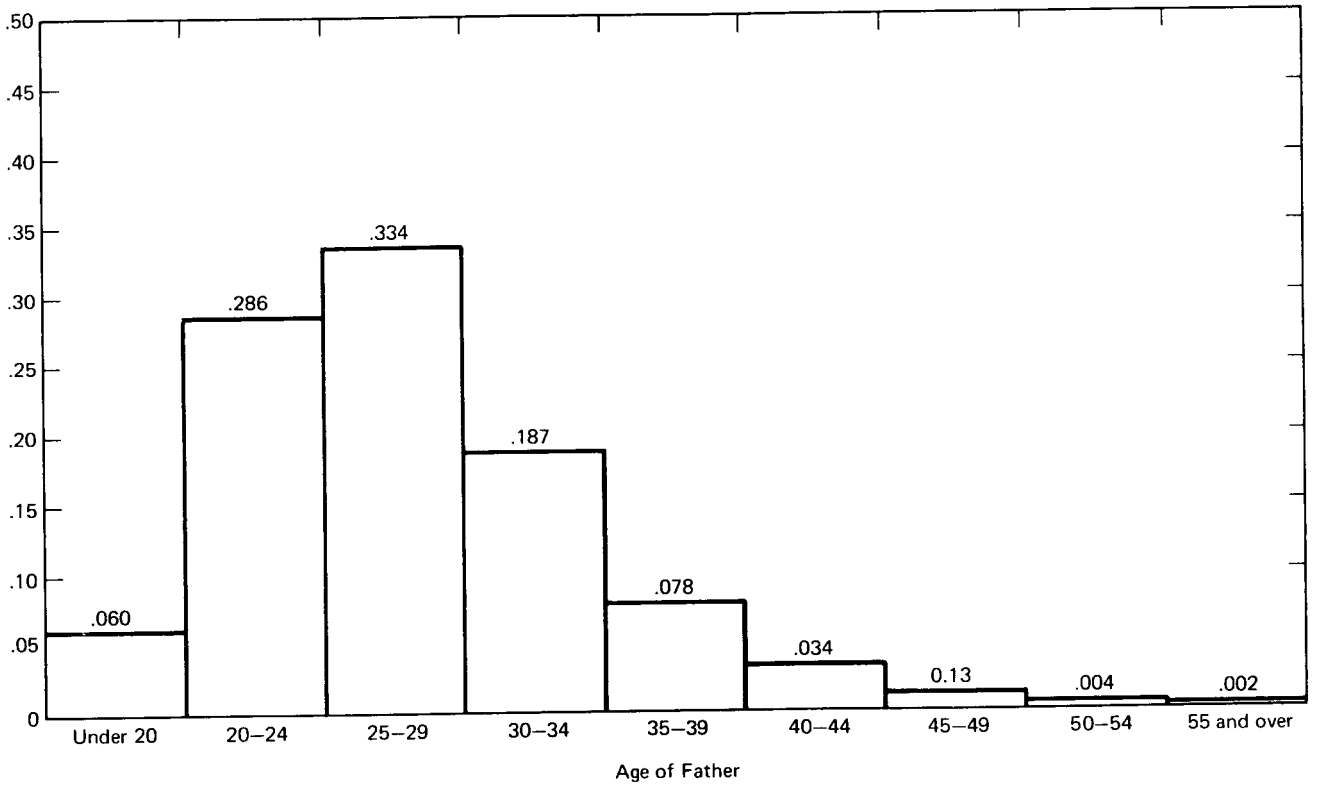

FIGURE VI I-2 Probability distribution of live births by age of father (1973 data).

Appendix J

Evacuation

J1 INTRODUCTION

In this appendix, evacuation data published by the U.S. Environmental Protection Agency (EPA) in the report Evacuation Risks - An Evaluation (Hans and Sell, 1974) are statistically analyzed to investigate mass evacuation behaviors. Evacuations in three categories -- transportation accidents, hurricanes, and floods -- are analyzed individually and also jointly to determine general behaviors. It is of interest that the evacuations described by EPA were carried out predominantly by private vehicles.

The only events considered are those for which there is sufficient information for statistical analysis. The nature of the analyses is such that this selection of data should not cause significant bias in the analyses; however, the analyses are to be interpreted within the framework of the data used.

Both random-variable and standard regression analyses are used. Various models are developed, depending on the amount of detail considered and the amount of a priori information assumed to be known in prediction-making. The models and techniques are discussed along with their accompanying results. Application of the results to prediction and risk analysis is also described.

J2 BASIC EVACUATION DATA

Tables VI J-1, VI J-2 and VI J-3 present the basic evacuation data used. Each entry represents an actual evacuation and the pertinent data characterizing it, the numbers for time and distance constituting a data point. The evacuations span the period from 1959 through 1973. The tables were compiled directly from the EPA data, originally obtained from surveys and personal questionnaires. The event numbers in the tables are those assigned in the EPA report; the other headings are self-explanatory. By their very nature, the data are somewhat rough, being derived from personal recollections and general descriptions. Nonetheless, these data can give information on general trends and behaviors.

J3 BASIC CONSIDERATIONS

Two important questions in the analysis of evacuation behavior are

1. The mean distance that can be traveled in a specified time

2. The effective time required to travel a specific distance.

The variables considered here are the mean distance traveled by evacuees (d), the elapsed time from evacuation signal to the arrival of evacuees at the destination (t) and the effective speed (v), defined by

$$v = \frac{d}{t}. \qquad \text{(VI J-1)}$$

The effective speed is lower than the vehicle speed because it includes, in "elapsed time," the effects of hesitation and delay.

J4 STATISTICAL TREATMENTS AND MODELS

This section presents a statistical analysis of five models under two general approaches: (1) the distance approach, where the distance d is treated as the random variable; and (2) the time approach, where the time period t is treated as the random variable. Although the two approaches are theoretically related, the statistical analysis of the data is performed somewhat differently. Specific models for both approaches will be discussed.

J4.1 DISTANCE APPROACH

J4.1.1 The Mixed Model

In a completely random model, all the evacuation speeds v_i in an evacuation category are considered as coming from the same statistical population. The speed is thus treated as being entirely random, and any dependencies on the particular evacuation are treated as being part of the random variation. The completely random model is termed the "mixed model" since the speeds for particular evacuations in a category are effectively mixed together to determine their combined probability distribution.

Because the speed is treated as being random within a category, the mixed model is applicable to predictions made when little information is available about the evacuation to be carried out (e.g., the precise number of people to be evacuated or the precise area). The model is also applicable to cases where, even though one does have more information, the speed is not strongly correlated with the particular evacuation characteristics. The validity of this latter application will be examined in subsequent models.

Because of its basic statistical properties, a log-normal distribution is postulated as adequately describing the variation in speed. Figures VI J-1 through VI J-4 are the log-normal probability plots of the speeds v_i for the four different categories. The straight lines are fitted to the points by the maximum-likelihood method. If the speed follows a log-normal distribution, the points should generally lie about the lines. A reasonably linear behavior is observed. A Lilliefors test on the fit of the points does not reject the log-normal distribution, with significance levels ranging from about 25 to 50%.

With the log normals identified, the distributions plotted in Figures VI J-1 through VI J-4 can be used in various applications, in standard probability plot fashion. The straight lines in the figures give the speed percentiles.

As an example of the use of the figures, consider a transportation accident. The question concerns the distance that the evacuees can travel within a 4-hour evacuation time period. From Figure VI J-1, the median (50th percentile) speed is approximately 1.2 mph, and hence the median distance that can be traveled in 4 hours is 1.2 x 4 = 4.8 miles. If the tenth percentile speed is associated with the minimum (conservative) speed that can be assumed (there is a 90% probability that the speed will be greater), then the minimum (conservative) distance that can be assumed is 0.15 x 4 = 0.6 miles. By using the median distance of 4.8 miles or the conservative distance of 0.6 mile, it is possible to make decisions on such topics as imposed risks, planning, and roads to use.

The maximum-likelihood calculations that are performed for the probability plots also yield the parameter values that define the speed distribution. The log-normal distribution has a probability density function given by

$$f(v) = \frac{1}{\sqrt{2\pi}\ \sigma\ v}\ \exp\left[-\frac{(\ln v - \mu)^2}{2\sigma^2}\right], \qquad \text{(VI J-2)}$$

where v is a particular speed value and μ and σ are the log-normal parameters.

Table VI J-4 gives the parameter values and characteristic speeds for the four categories as determined by fitting by the maximum-likelihood method.

For general evacuation behaviors, a number of observations can be made from Table VI J-4 and Figs. VI J-1 through VI J-4. Thus, it is found that evacuation speeds are quite low. The most probable speed is about 0.1 mph for transportation and flood evacuations and about 0.6 mph for hurricane evacuations.

The mean evacuation speeds in Table VI J-4, though still low, are significantly higher than the most probable speeds, being on the order of 2, 5, and 14 mph for flood, transportation, and hurricane evacuations, respectively.

The skewness of the log-normal distributions implies a large variability in attainable evacuation speeds. The large variability is perhaps best seen in the 90% ranges obtained from the probability plots. The 90% ranges for each category are also stated in Table VI J-4. The ratio of maximum to minimum speed in these ranges is about 100 to 200.

Because of the differences among categories, care must be taken in using the combined results. The combined-category distribution can be used only if gross results are desired. If more accurate results are of interest, then the category should be identified, or the relative probability of a given category of evacuation occurring should be determined and the individual distributions combined according to these relative probabilities.

One additional analysis that was performed on the mixed model should be mentioned. To attempt to obtain a better fit, the variance of v was made to be inversely proportional to the number of evacuees, which would be applicable if the individual evacuee movements were independent of one another. There was no significant improvement in the model fit; in fact, there was some lack of fit (i.e., lower significance levels), thus indicating that the evacuees moved as a mass instead of as independent individuals.

J4.1.2 The Correlated Model

The mixed model used no detailed a priori information about the evacuation. A better prediction of the speed can sometimes be obtained if the evacuation can be characterized by additional parameters correlated with the evacuation speed. For example, if the speed of evacuation is related to the number of evacuees, then using this information will yield better speed predictions. For no correlations to be found is also an important result since it implies that within the model framework the process can be viewed as being random and having few and small dependencies. The model that considers possible correlations is termed the "correlated model."

In the correlated model of a particular evacuation category, the speed v is treated not as entirely random but as having possible dependencies on certain parameters. The parameters that are most easily identified and most likely to be known a priori are the number of evacuees N and the evacuated area A. Since time is also a parameter, A, N, and t are considered to be the three parameters that identify an evacuation.

A log-normal regression approach is used since it is straightforward and yet of sufficient generality. Within the log-normal framework, the general equation for v, incorporating possible relationships with A, N, and t, is

$$v = \delta A^{\alpha} N^{\beta} t^{\sigma} n, \qquad \text{(VI J-3)}$$

where α, β, γ, and δ are coefficients and n is a log-normal noise variable.

Other general forms could be postulated for v; however, Equation (VI J-3) is of a standard log-normal regression form and incorporates a spectrum of possible relationships.

To determine the values of α, β, σ, and δ, standard regression analyses are performed. Taking the natural logarithms of Equation (VI J-3) gives

$$\ln v = \ln \delta + \alpha \ln A + \beta \ln N + \sigma \ln t + \ln n, \qquad \text{(VI J-4)}$$

For the regression calculations, for each point in an evacuation category, logarithms were thus taken of the speed $v_i = (d_i/t_i)$, the area A_i, the number of evacuees N_i, and the time t_i (columns 4, 5, 6 and 7 in Tables VI J-1, VI J-2, and VI J-3).

The set of values was used as input to a standard multiple-regression program, DCRT Mathematical and Statistical Program Package of the National Institutes of Health.

No significant correlation of the speed with any of the parameters A, N, and t was found for any evacuation category. For example, the values of the F-statistic were not significant at the 10% level. The coefficients α, β, and σ can therefore be taken to be zero and the correlated model is equivalent to the mixed model.

This result may be due, at least partly, to the character of the data analyzed. The recorded evacuation times varied only over a small range, so that data errors could mask some correlation and any long-range effects could be hidden.

J4.1.3 The Weiner, or Brownian, Model

The Weiner, or Brownian, model is sometimes applied to descriptions of mass transport and of drifting phenomena (e.g., molecular movement and instrument driftings). In the Weiner model, the evacuation is taken to resemble Brownian motion with a net forward movement (away from the evacuated area).

Even through it has been successfully applied to a class of problems and has some intuitive basis, the Weiner model makes the strong assumptions that distances traveled in successive intervals of time are independent of one another and follow a normal distribution. These assumptions are not true for the evacuation process, since the speed of an evacuee at one time is correlated to his speed at another time and the distance behavior is log-normal, not normal. Because of the limiting assumptions, the Weiner model was not as good a description of the evacuation data as the mixed model. Because of its inadequacies, the Weiner model will not be developed; however, a brief outline of its results and the basis for the decision that it was inadequate are presented below.[1]

The results of the Weiner model gave roughly the same behavior as the mixed model. The mean speeds for transportation, hurricanes, floods, and the general category were 2.62, 8.91, 1.44, and 4.18 mph, respectively. Even though the behaviors were roughly similar, when predictions were compared with observed values, the residuals of the Weiner model (observed minus predicted) were generally larger and showed greater lack of fit than did the mixed model. Similarly, the Lilliefors test showed poorer fitting properties. Furthermore, the Weiner residuals showed systematic errors; for example, large values were generally underestimated and small values were generally overestimated.

Besides being applied to the mean distance, the Weiner model can also be applied to the distances traveled by individual evacuees. Here again the Weiner model proved to be inadequate with regard to its prediction capability (larger residuals and systematic error). As a final point, correlations can be incorporated into either of the Weiner model applications by allowing the speed to be a function of the evacuation parameters, such as A and N. Regression analysis gave no significant improvement in the models.

J4.2 TIME APPROACH

J4.2.1 The Mixed Model

The distributions obtained for the distance approach are directly applicable to the time approach. They can be used in the analysis and prediction of times, with attention to the proper transformation of parameters. They can also be used in decision investigations and risk analyses.

[1]For development of the Weiner model, see, for example, Parzen (1967).

J4.2.2 The Correlated Model

This section describes investigations of possible dependencies, again using the regression approach. As before, one considers correlation of the evacuation speed with the parameters A and N, where A is the evacuated area and N is the number of evacuees. The distance d is now included as the third parameter.

The log-normal formula for v, incorporating possible relationships to A, N, and d, is

$$v = \delta A^{\alpha} N^{\beta} d^{\sigma} n, \qquad \text{(VI J-5)}$$

where α, β, σ, and δ are coefficients and n is a noise variable.

Taking the natural logarithms of Equation (VI J-5) gives

$$\ln v = \ln \delta + \alpha \ln A + \beta \ln N + \sigma \ln d + \ln n, \qquad \text{(VI J-6)}$$

which is the standard regression equation. The values for each evacuation were taken as input to the regression computer program as before.

The regression analyses showed that in general only the distance significantly affected the evacuation speed. The evacuation parameters A and N had minor or negligible effects compared to the distance effect.[1]

Table VI J-5 gives the results of the regression analysis for Equation (VI J-5) with α and β equal to zero.

Table VI J-5 gives the regression estimates and the 90% confidence bounds of the parameters δ and σ. The quantity s given for each category is the standard error of estimate, which may be used as an estimate of the standard deviation for ln v in determining confidence bounds for predicted speeds.

Thus the 90% confidence limits are obtained by multiplying the best estimate of speed, $v = \delta d^{\sigma}$, by $e^{-1.64s}$ and $e^{+1.64s}$. (Instead of the normal value 1.64, actual t-values can also be used, where the degree of freedom is N - 2, N being the number of data points (Tables VI J-1, VI J-2, and VI J-3).

Table VI J-5 shows that for all evacuation categories the correlation of speed with distance is quite pronounced: all the regressions were significant at 99.9% (0.1% rejection level). Since α is close to unity, the evacuation speed is approximately directly proportional to the distance.

Because the coefficients α and β are not significantly different for the various evacuation categories (e.g., the corresponding confidence bounds overlap), the general relationship, in which all evacuations are combined, can be usefully applied as giving a general evacuation behavior. Figure VI J-5 is a plot of speed versus distance for all the evacuations. The straight line in the figure is the regression best fit for all the data points in Table VI J-1, VI J-2, and VI J-3. As observed, all the evacuations lie fairly well along the regression best-fit lines. Statistical tests on the regressions showed that no significant loss of fit resulted from combining all the evacuations.[2] This result would be expected from Table VI J-5 since the coefficients δ and σ are similar for different evacuation categories and their confidence intervals overlap. Thus, the general relationship can be usefully applied to predicting evacuation behavior.

[1] For transportation and flood evacuations, the regression t-values for α and β ranged from 0.4 to 1.3. For hurricane evacuations the area coefficient α was barely significant at the 5% individual t level. The area coefficient (negative) was only 15% of the value of the distance coefficient. Since the hurricane evacuation area and distances are comparable in value, the area effect was treated as being minor. Standard regression F tests (residual sums with and without A and N) gave equivalent results.

[2] For example, the F-statistic formed from the individual residual sums of squares and the combined (general case) sum of squares was not significant at the 10% level.

Figure VI J-5 illustrates the strong apparent dependency of speed on distance. In this respect, the time-approach correlated model differs from the distance-approach correlated model, in which little dependency was observed. Since v is the effective evacuation speed, the initial delay and confusion become less important as distance increases and a greater portion of the evacuation time is spent in actual travel. With increasing distance the effective speed thus approaches the actual travel speed and the effective speed increases as distance increases, in agreement with Fig. VI J-5.

Even though a strong dependency is shown and has a certain physical rationale, care must be taken in interpreting and using the results, as in any regression analysis. The evacuation distance has been treated as a parameter that characterizes the evacuation, and the recorded distances are thus treated as having negligible data errors. Since the range of distance data points is large, reasonable errors in the recorded distances (say 10%) should not significantly affect the regression results; however, larger errors can influence the results.

The discussion of the regression-analysis limitations can be summarized by saying that the regression results must be interpreted within the framework of the data and the definitions of speed and distance. Within this framework, the dependency of speed on evacuation distance is quite pronounced, with the best-estimate general formula given by $v = 0.283d^{0.914}$. Moreover, the general relationship is applicable to the various evacuation categories, and further analyses have shown little dependency on any other additional evacuation characteristic.[1]

In ending this section, the possible uses of the speed formula in prediction modeling should again be mentioned. If the distance associated with an evacuation is known with reasonable accuracy, then this distance can be substituted into the particular evacuation type formula or into the general formula $v = 0.283d^{0.914}$ to yield the best estimate of the evacuation speed. The best estimate of the time required for the evacuation is then $t = d/v$. (Alternatively, by direct substitution, $t = 3.53d^{0.086}$ for the general formula). Confidence bounds on v obtained from the regression results can be used to determine the confidence bounds on the predicted time period t (an upper bound on t, for example, will be obtained by using the lower bound on v).

In comparison to the mixed model, the regression estimates will in general have smaller variability and smaller uncertainty because knowledge of the evacuation distance is now utilized. The regression estimates are, however, dependent on knowledge of the distance. If the distance is not accurately known, then several possible distance values can be used to determine the spreads and sensitivities, or alternatively the mixed model can be used.[2] The speed and time predictions, however obtained, can then be utilized in investigating decision alternatives and in calculating evacuation risks.

J5 CONCLUSIONS

In the distance approach, because of little correlation, the mixed model and the correlated model were found to be equivalent. In both models the distribution of effective speeds is log normal.

In the time approach, the log-normal distributions of the distance approach were found to be applicable to the mixed model. In the correlated model, a significant correlation was found to exist between evacuation speed and distance. It was determined that a general regression formula,

$$v = 0.283d^{0.914},$$

is applicable to the evacuations of any type and any characteristic.

[1] In addition to the parameters A and N, the other parameters in Tables VI J-1, VI J-2, and VI J-3 (weather, day, etc.) had generally negligible or minor effects on the speed. These effects were investigated using the standard residual sum-of-squares F-test.

[2] Because of the log-normal transformation, the regression best estimates correspond to the median values of the mixed model.

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Parzen, E., 1967, Stochastic Processes, Holden-Day, San Francisco

TABLE VI J-1 BASIC EVACUATION DATA - TRANSPORTATION

Event Number	Location and Date	Type of Area Evacuated	Area Evacuated (sq. miles)	Number of Persons Evacuated	Distance Evacuated (Miles)	Evacuation Period (hrs)	Population Density (number per sq. mile)	Road and Conditions (a)	Weather	Time of Day	Evacuation Plans (b)	Remarks
12	Downington, PA; 2/5/73	Suburban	0.25	700 of 800	1.0	2.0	3200	Dry S	Cloudy	Night	PU	Private vehicles
16	Creve Coeur, MO; 8/1/61	Rural residential; suburban; urban	15	7,500	12	1.0	500	Dry S	Fog	Night	Pu	Private vehicles
18	Chadbourne, NC; 1/13/68	Suburban	0.5	350	1.0	5.0	700	Dry S	Cloudy	Dusk Night	NP	Private vehicles
33	Wetanka, OK; 4/4/69	Rural residential	3	2,000	25	8	667	Dry S	Cloudy	Day	PU	Private vehicles
34	Louisville, KY; 3/19/72	Urban	0.35	4,000	1	3	11,400	Wet U	Rain	Day	Pu	Private vehicles; chlorine barge; no chlorine release
35	Urbana, OH; 8/13/63	Suburban	3.1	4,000	0.75	3.5	1,300	Dry S	Clear	Dawn	N.D.	Private vehicles
36	Baton Rouge, LA; 8/65	Urban	8	150,000	30	2.0	19,000	Dry U, EU	Clear	Day	PU	Private vehicles chlorine barge; no chlorine release
38	Morgan City, LA; 1/19/73	Urban	1.8	3,000 of 3,300	2	4	1,800	Ice U	Snow	Day	PU	Private vehicles; chlorine barge; no chlorine release
39	Texarkana, TX; 8/27/67	Suburban	9.0	5,000	3	4	550	Dry U	Clear	Night	NP	Private vehicles
44	Glendora, MS; 9/11/69	Rural farming; rural residential suburban urban	1,200	35,000	20	4	29	Dry S	Cloudy	Night	P	Private vehicles

(a) Key: U - urban road;
S - suburban road;
R - rural road;
EU - express way (unlimited access);
EL - express way (limited access).

(b) Key: P - plan available (not used);
PU - plan used
NP - no plan
N.D. - no data

TABLE VI J-2 BASIC EVACUATION DATA - HURRICANES

Event Number	Location and Date	Type of Area Evacuated	Area Evacuated (sq. miles)	Number of Persons Evacuated	Distance Evacuated (miles)	Evacuation Period (hrs)	Population Density (number per sq. mile)	Road and Conditions[a]	Weather	Time of Day	Evacuation Plans[b]	Remarks
19	Port Aransas, TX; 9/61	Urban	1.3	2,800 of 4,000	50	2.0	3,100	Dry R	Cloudy	Day	PU	Private vehicles
20	Robestown, TX, 7/3/70	Urban	0.08	450	3.5	1.5	5,600	Wet R	Rain	Dusk	PU	Supplied Vehicles
22	Chambers Co. TX; 8/3/71	Rural farming	336	10,000 of 10,200	50	7.5	30	Wet UR	Rain Gale	Day Night	PU	Private vehicles
30a	Port Arthur, TX; 9/3/61	Hospital	N.D.	80	20	4	N.D.	Dry R	Clear	Day	PU	Hospital evacuation of ambulatory patients by private vehicles
30b	Port Arthur, TX; 9/3/61	Hospital	N.D.	20	20	4	N.D.	Dry R	Clear	Day	PU	Hospital evacuation of non-ambulatory patients by ambulances
	Jefferson Co., TX; 9/3/61	Suburban; urban; industrial	945	108,600 of 113,600	80	7.5	120	Dry S	Clear	Day	PU	Private vehicles; predominantly large-scale urban evacuation
37	St. Mary Parish, LA; 9/64	Rural residential; urban	1,036	40,500 of 45,000	150	8	43	N.D. U	N.D.	N.D.	PU	Private vehicles
41	Grand Isle, LA; 9/3/61	Rural residential; industrial	1.8	2,200 of 2,300	70	3.5	1,300	Wet R	Rain	Day Dusk	NP	Private vehicles
43	Seabrook Island, SC; 1/19/59	Suburban	4.5	208	0.6	4	46	N.A.	Rain	Day Dusk Night	PU	Boat evacuation
47	Lafourche Parish, LA; 9/11/61	Rural farming	100	23,000 of 37,000	50	9	370	Wet R	Rain	Night	PU	Private vehicles
49	Biloxi, MS; 9/11/61	Urban	7.7	15,000 of 20,000	5	5	2600	Dry S	Clear	Dawn Dusk	PU	Private vehicles

(a) Key: U - urban road;
S - suburban road;
R - rural road;
EU - express way (unlimited access);
EL - express way (limited access).

(b) Key: P - plan available (not used);
PU - plan used;
NP - no plan;
N.D. - no data;
N.A. - not applicable.

TABLE VI J-3 BASIC EVACUATION DATA - FLOODS

Event Number	Location and Date	Type of Area Evacuated	Area Evacuated (sq. miles)	Number of Persons Evacuated	Distance Evacuated (miles)	Evacuation Period (hrs)	Population Density (number per sq. mile)	Road and Conditions[a]	Weather	Time of Day	Evacuation Plans[b]	Remarks
6a	Ferndale, WA; 1/8/71	Rural farming; fishing	30	60	10	4.0	6.7	Wet R	Rain	Day Dusk	PU	Private vehicles; Indian Reservation
6b	Ferndale, WA; 1/8/71	"	30	140	8.0	4.0	6.7	Wet R	Rain	Day Dusk	PU	Supplied vehicles
6c	Ferndale, WA; 1/8/71	"	30	25	1.0	4.0	6.7	N.A.	Rain	Day Dusk	PU	Boat evacuation
7	Chehalis Indian Reservation, WA; 12/22/72	Rural farming	8.0	38	25	2	N.D.	Wet R	Rain	Night	Pu	Private vehicles
9	Port Angeles WA; 6/16/61	Surburban	1.0	100	0.5	2	N.D.	Wet U	Rain	Night	P	Private vehicles
17	Wilkes Barre, PA; 6/23/72	Urban	5.0	75,000 of 78,000	1.0	5.0	15,600	Wet U	Rain	Dawn Day	PU	Hospitals and jail evacuated
21	Payson, AZ; 9/70	Rural residential; recreation	20	160	1.0	12	8	Wet R	Rain	Day	PU	Private vehicles
25	Isleton, CA; 6/21/75	Suburban	11	1,200	40	11	109	Dry EU	Clear	Day	NP	Private vehicles
27a	Glenn Co., CA; 2/73	Rural farming	20	30	6	4.0	N.D.	N.A.	Rain	Day Night Dawn Dusk	N.D.	Helicopter evacuation
28	King Co. WA; 3/59	Rural farming	20	500 of 512	10	18	26	Wet R	Rain	Day Dusk Night	PU	Private vehicles
45b	Anderson, SC; 7/9/68	Suburban	0.09	150	0.75	2	1,700	Wet U	Rain	Night	NP	Supplied Vehicles
53a	Florence Co., SC; 2/3/73	Rural residential	6	90	6	8	15	Wet R	Clear	Night	PU	Private vehicles

(a) Key: U - urban road;
S - suburban road;
R - rural road;
EU - express way (unlimited access);
EL - express way (limited access).

(b) Key: P - plan available (not used);
PU - plan used;
NP - no plan;
N.D. - no data;
N.A. - not applicable.

TABLE VI J-4 LOG-NORMAL PARAMETERS AND CHARACTERISTIC EFFECTIVE EVACUATION SPEEDS

Category	μ	σ	Effective Evaluation Speed (mph)[a] Modal	Mean	5th%	95th%
Transportation	0.202	1.64	0.08	4.7	0.10	20
Hurricanes	1.57	1.50	0.64	13.8	0.45	55
Floods	-0.241	1.44	0.09	2.3	0.06	9
Combined	0.498	1.68	0.10	6.7	0.10	30

[a] The 5th and 95th percentiles are approximate values, taken from Figs. VI J-1 through VI J-4.

TABLE VI J-5 REGRESSION RESULTS FOR THE EQUATION $v = \delta d^{\sigma}$

Category	δ Regression Estimate	δ 90% Confidence Bounds Lower	δ 90% Confidence Bounds Upper	σ Regression Estimate	σ 90% Confidence Bounds Lower	σ 90% Confidence Bounds Upper	s[a]
Transportation	0.30	0.18,	0.50	1.02	0.77,	1.28	0.62
Hurricanes	0.41	0.22,	0.77	0.81	0.63,	1.00	0.52
Floods	0.23	0.14,	0.39	0.89	0.62,	1.15	0.72
Combined	0.28	0.21,	0.38	0.91	0.80,	1.03	0.63

[a] The standard error of estimate.

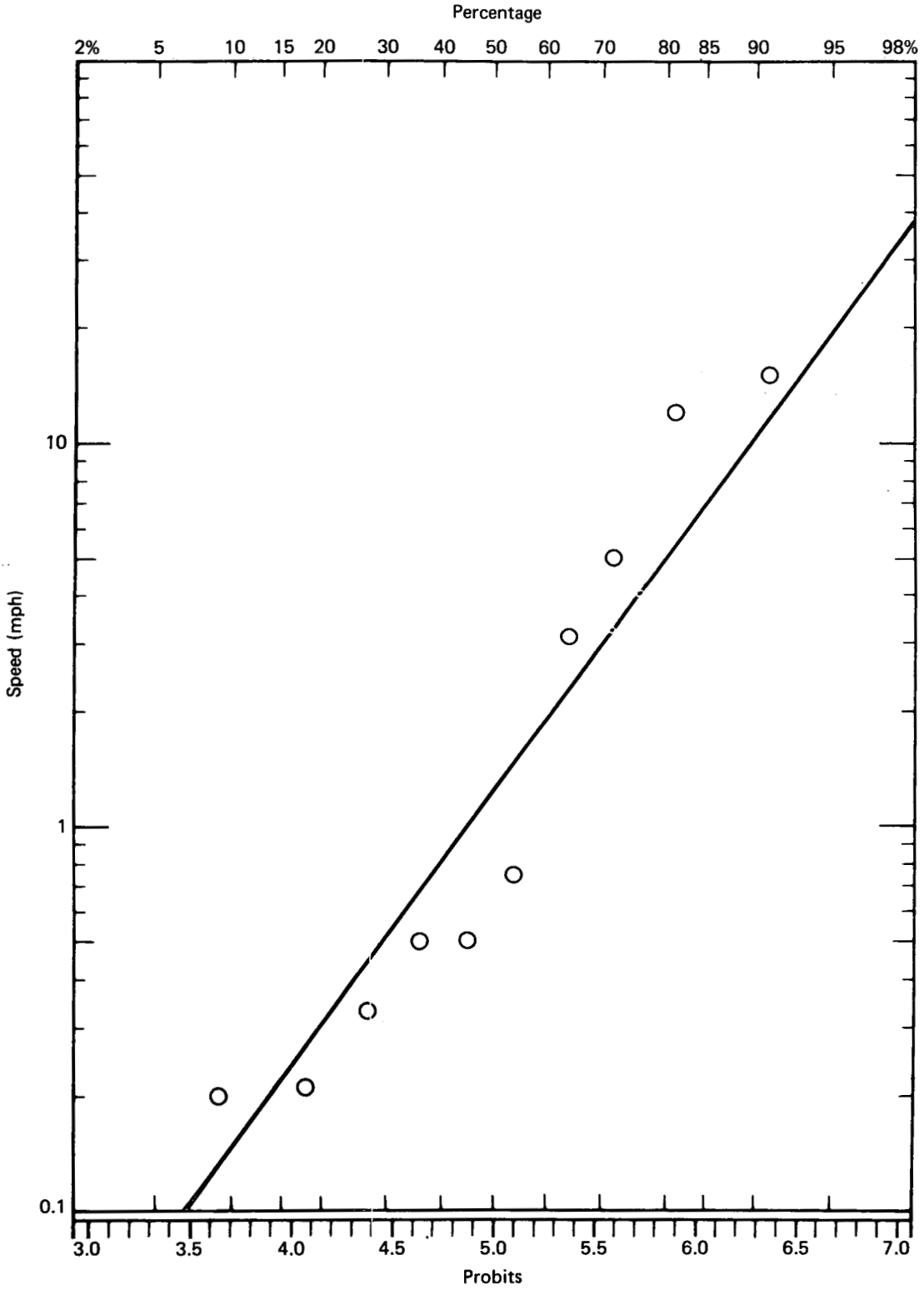

FIGURE VI J-1 Probability plot of transportation evacuation speeds.

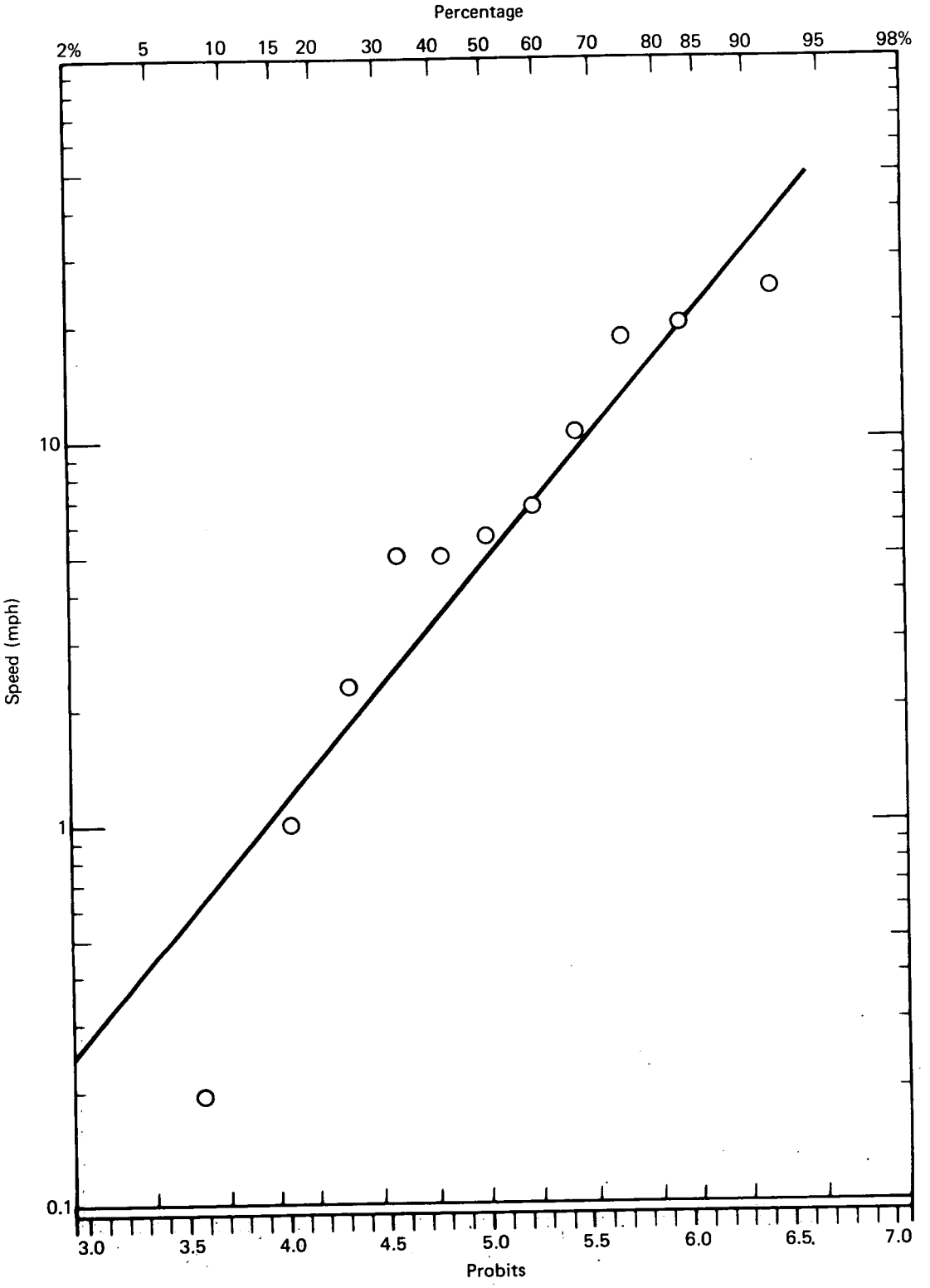

FIGURE VI J-2 Probability plot of hurricane evacuation speeds.

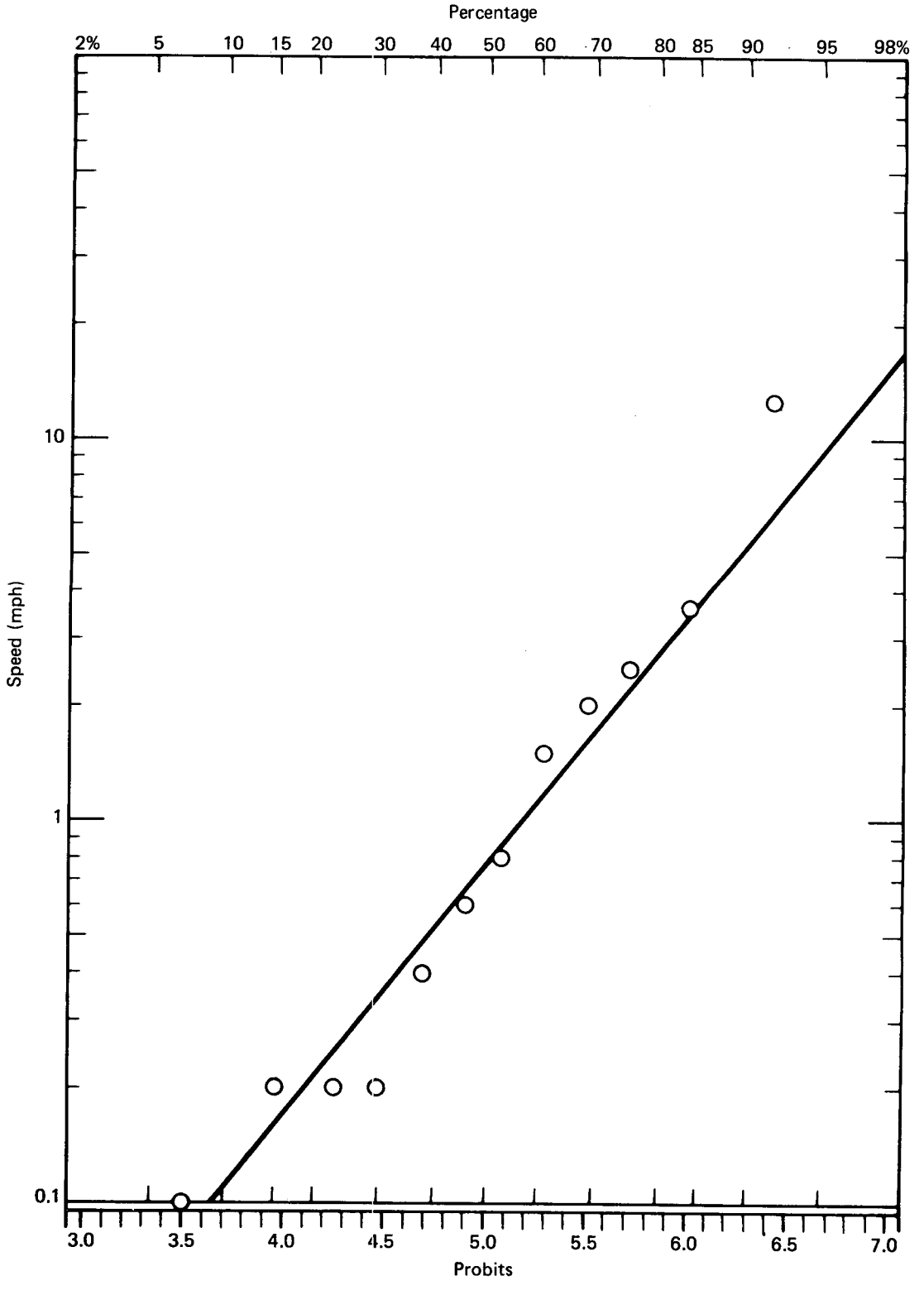

FIGURE VI J-3 Probability plot of flood evacuation speeds.

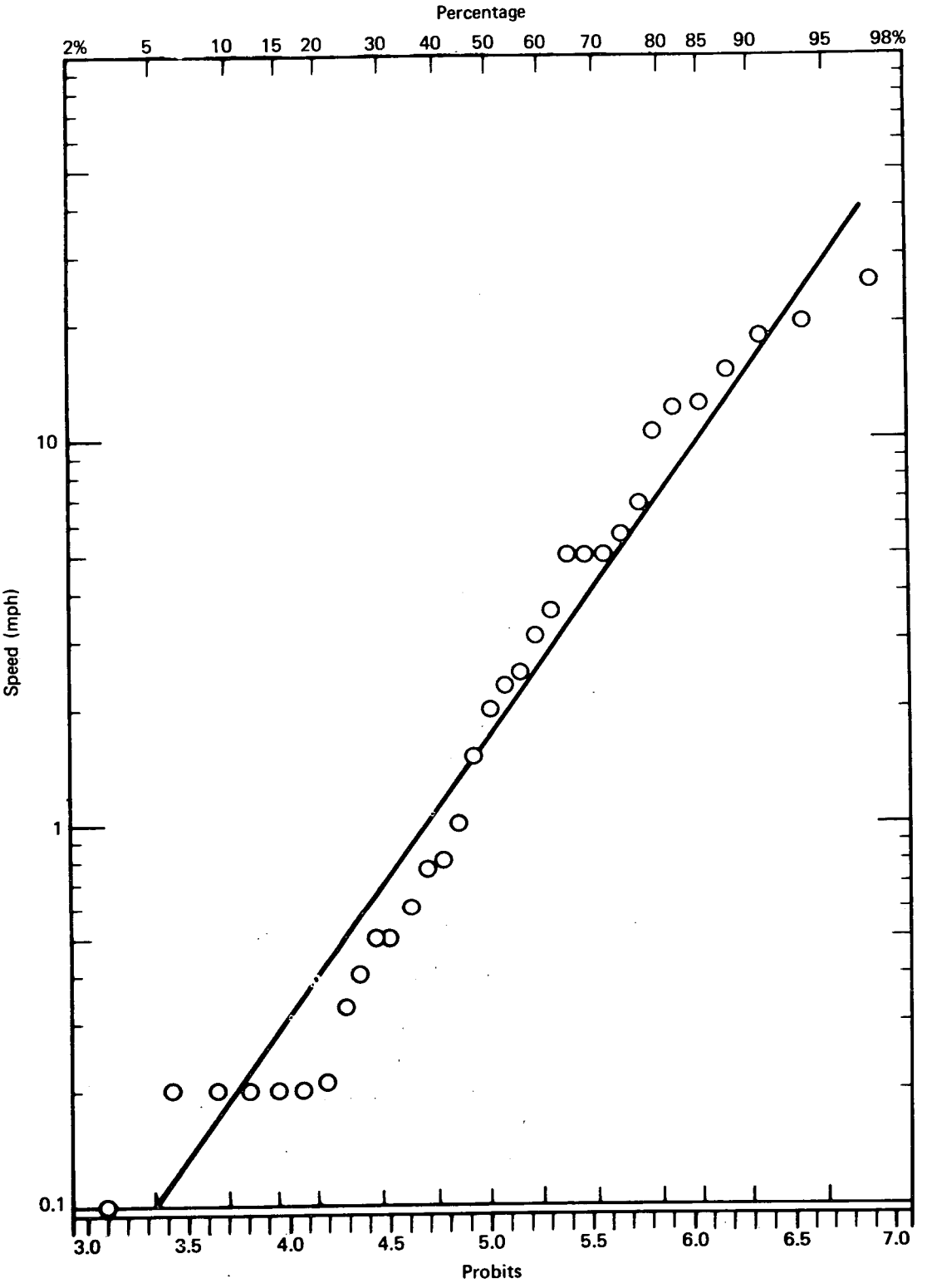

FIGURE VI J-4 Probability plot of general evacuation speeds.

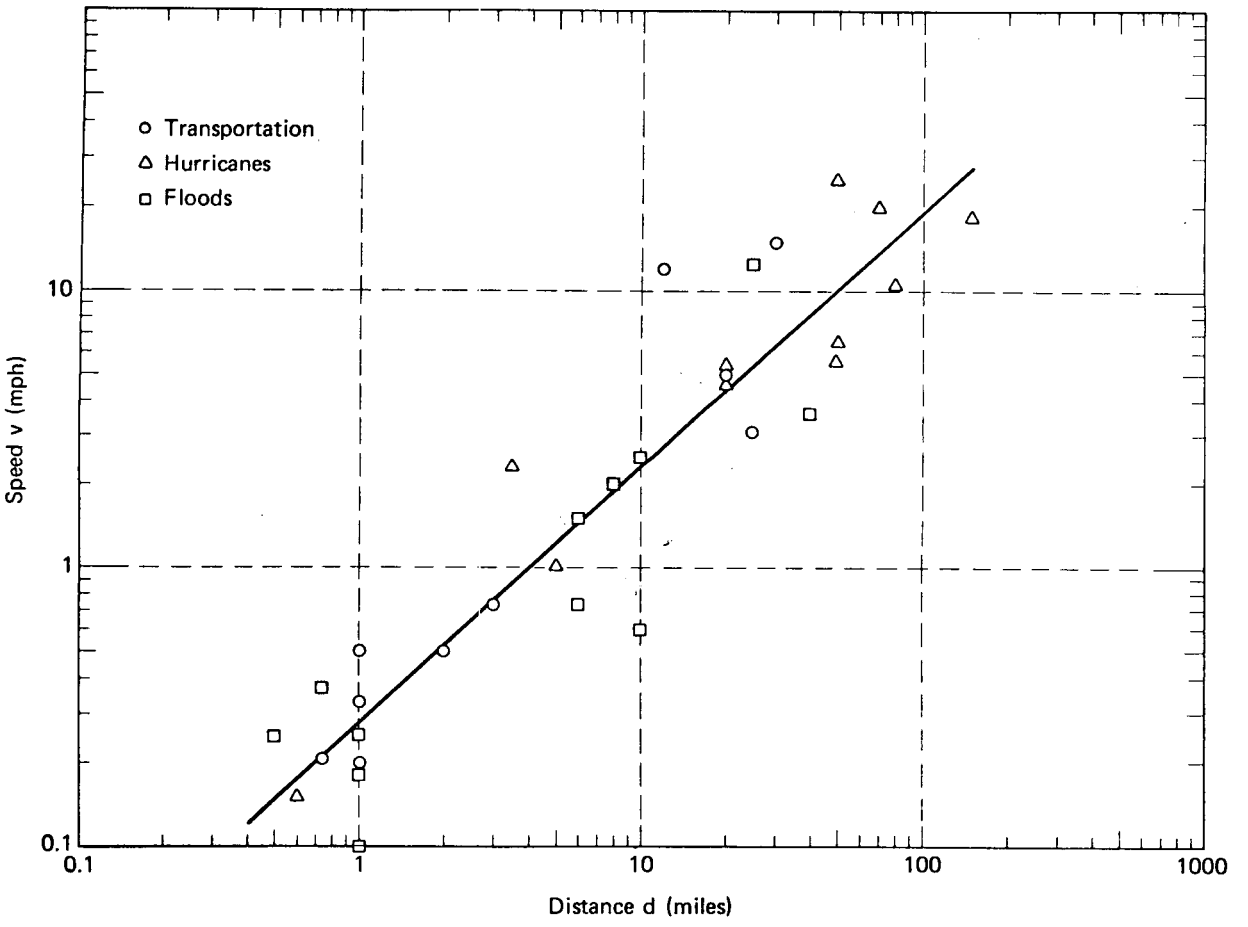

FIGURE VI J-5 Effective evacuation speed v versus distance of evacuation, d.

Best fit-line $v = 0.283d^{0.914}$

Appendix K

Decontamination

K1 INTRODUCTION

The problem addressed here is the radiological cleanup of environments contaminated by radionuclides released as a result of a major nuclear reactor accident (core meltdown and rupture of containment vessel). Radiological cleanup implies the utilization of procedures for the reduction of human exposure (external and internal) to the deposited radionuclides. Therefore, the possibilities include physical removal of the radionuclides, stabilization of the radionuclides in place, and environment management. The particular procedure utilized in a given case depends on many factors. For example, consideration must be given to the type of surface contaminated, the external environment the surface is exposed to, the possible radiological hazards to the operators, the costs involved, the level of decontamination required, and ultimately the consequences of the decontamination operation.

There is a large body of experimental data on the decontamination of structures, pavements, and land. These data were generated for the planning of radiological reclamation operations in the event of a nuclear war. Because of differences in contaminant particle size and decontamination criteria, some of these experimental data are not directly applicable to the particular case considered in this study. The particle size from weapons fallout can be of the order of 100 microns or more, whereas the aerosols that would be expected to be released from a reactor core meltdown would be at most a few microns in diameter. The objectives of radiological cleanup in a nuclear war are to reduce gamma radiation from these sources to an acceptable level with the least possible expenditure of labor and material and the least possible exposure of operating personnel. In general, the decisions would be to maximize short-term gains, a goal that would not be acceptable in the peaceful utilization of nuclear energy.

Despite these differences, this appendix discusses the decontamination problem one would reasonably expect to result from a nuclear reactor accident. The bases for the discussion are the available experimental evidence and past experience.

K2 CONSIDERATIONS FOR DECONTAMINATION OPERATIONS

The criteria by which decisions would be made concerning decontamination are not uniform nor precise. There are four general areas to consider, each of which would be important in the evaluation of a decision to decontaminate:

1. Radiological. Radiological considerations involve an estimation of the likely exposure of population groups to the radionuclide contaminants and possible effects on the biota.

2. Environmental. It is possible, depending on the particular situation, that the decontamination operation could be responsible for far greater environmental damage than would result if the radioactive contaminant were left in place. For example, one is reminded of the saying, "the cure was effective, but the patient died." Therefore, it is prudent to consider all the environmental factors as they relate to the effects of a decontamination operation.

3. Economics. A decontamination operation could be a very expensive undertaking. Therefore the benefits to be derived from the operation must be clearly delineated and weighed against the costs. Allowances must be made for the availability of adequate machinery to perform the operation, and the transportation and disposal of the large volumes of generated wastes if certain types of decontamination are to be performed.

4. Political. In the event that a large area became contaminated, various political forces would exert pressure on the body responsible for the incident and the decontamination operation. The political forces would range from the local level to, possibly, the international level. Therefore, political considerations have been and will be important in any decontamination operation.

A decision to decontaminate and the desirable level of residual contaminant would be based on considerations in any, or all, of the four above general areas.

Care must be exercised in the selection of a particular decontamination for a given area. For example, the types of procedures one would utilize on highly controlled land (agricultural land) would also be effective in wilderness areas but not very useful because of subsequent soil erosion and watershed control problems. Replanting would be possible but not always economically feasible, depending on the area.

K3 CLASSIFICATIONS OF DECONTAMINATION OPERATIONS

Decontamination operations can be classified as wet methods and dry methods. Wet methods are the most effective means for removing the particulate material from hard, smooth surfaces, such as asphalt or concrete. The methods include firehosing and motorized street flushing. The effectiveness of the methods relies on the high velocity of water streams to move the particulate matter from the areas of concern. Unless there were subsequent collection of the water and particulate matter, all that would be accomplished with the method is a simple displacement of the contamination. In addition, some of the soluble radionuclides in the particulate matter may be leached out and chemisorbed on surfaces. Therefore, in the evaluation of wet decontamination one must consider that radionuclide pickup and disposal may also have to be performed subsequent to the cleaning of the hard surfaces.

Dry decontamination methods include sweeping, scraping, grading, and bulldozing. Sweeping may be used for the decontamination of hard-surface areas where firehosing or flushing methods are not available or if the weather or terrain is unsuitable. Scraping, grading, and bulldozing techniques would be of principal use on unpaved areas such as lawns or fields. As with the wet methods, some of the dry methods involve only a simple displacement of the contaminants. However, in this case the contaminants would become mixed with a large volume of the native surface material. The dilution can easily be as large as 500 parts of surface material for every part of contaminant. If pickup and disposal are required, very large volumes of material would have to be reckoned with.

A measure of decontamination effectiveness is the decontamination factor (DF). The decontamination factor is defined as the amount of contaminant per unit surface area before decontamination, divided by the amount of contaminant after decontamination. Therefore, the larger the DF, the better the decontamination method. For example, a 90% removal of contaminants from a surface represents a DF value of 10, and a 99% removal a DF of 100.

K4 EXTERNAL INFLUENCES ON DECONTAMINATION EFFICIENCIES

There are a number of factors that effect the efficiency of decontamination operations. These are factors that would not generally be under the control of the persons performing the decontamination operations. although the influence of some of the factors can be minimized by the speed with which decontamination is undertaken and completed.

Wet and Dry Deposition. The distribution of the contamination on surfaces and its adherence to the surface can depend on whether the deposition was wet or dry.

Major Weather Changes. After initial deposition of the radioactive contaminant, major weather changes could severely affect the efficiency of decontamination. For example, significant amounts of snow or rainfall and variations in air temperature from temperate to subfreezing after deposition would hamper efficient removal of the contaminant.

Aging. In all environments there are normal physical and chemical forces operating that could render decontamination operations more difficult with the passing of time. Therefore, the length of time between deposition and decontamination is an important parameter in the efficiency of decontamination.

Particulate Matter Size. The removal efficiency of some decontamination procedures is a strong function of the size of contaminant particles. The smaller the particle, the more difficult it is to efficiently remove it from the surface.

Surface Characteristics. A hard, smooth surface is relatively easy to clean in contrast to a rough, porous surface.

Operator or Team Skill and Incentive. The ability of the personnel to operate their machinery efficiently and minimize recontamination is paramount for effective decontamination.

K5 CONTAMINATION CATEGORIES

In the event of a major release of radioactive material from a nuclear reactor, a wide variety of surfaces could become contaminated. These surfaces fit into eight different categories, which are described in this section. The composite of the environment would be made up of a combination of these contamination categories, the exact proportions of which would depend on the specific reactor site.

The importance of categorizing the potentially contaminated environments is that the surface characteristics and the behavior of the radionuclides on these surfaces are quite different. For many reasons, there would be a delay between initial deposition of radionuclide contaminants and initiation of any decontamination operation. During this period the nature and distribution of the contamination could change and thereby affect decontamination operations. Most of the information available, in this respect, has to do with the fallout from nuclear weapons. Therefore, caution must be exercised in the application of this information to the nuclear reactor contaminating event.

K5.1 STRUCTURE CONTAMINATION

The influence of urban area geometry on deposition is not well understood. However, eaves, troughs, the lee side of roof peaks, crevices in the roof surfaces, and any roof areas protected from the wind are locations where deposited particles would tend to accumulate. Certainly because of its particulate nature, the contaminant would tend to collect on horizontal surfaces, although some vertical surface contamination would be expected.

K5.2 PAVED-AREA CONTAMINATION

There are essentially no data available on the decontamination of streets and roads by rain or vehicular traffic. It is expected that light rains would facilitate the leaching of soluble radionuclides from the deposited particles and the transport of these radionuclides to pavement surfaces where they could be chemisorbed.

K5.3 VEHICLE AND EQUIPMENT CONTAMINATION

A variety of portable man-made objects could become contaminated. Depending on the economic incentives, some could be decontaminated and some could be disposed of with other contaminated wastes. The main objects of concern typically have hard, smooth surfaces, but have catching points that would be very efficient for the accumulation of the radioactive contaminants.

K5.4 LAND AREA AND SOIL CONTAMINATION

Particulate matter deposited on open land areas and on bare soils is not moved significantly by winds. The rate of penetration of radionuclides into soils, under natural forces, is slow. For example, measurements by Squire and Middleton (1966) show that the median depth of cesium-137 in various soil type was between 1 and 3 cm 6 years after a surface contamination. For strontium-90 the median depth achieved was between 3 and 5 cm after 6 years.

Deposition in heavy rain would result in fractional runoff of soluble radionuclides (Frere and Menzel, 1960), the rate of movement being governed by their solubility and the degree to which they undergo adsorption on and/or exchange processes with the constituents of the soil.

K5.5 WATER CONTAMINATION

Water could be directly contaminated by water surface deposition and by runoff from adjacent land areas. For example, analysis of river waters and of the deposition of strontium-90 in worldwide fallout from the Ohio River basin indicates that between 4 and 12% of the strontium-90 deposited in 1959 was carried into river water (Straub et al., 1960). An analysis of the watershed of the Providence, Rhode Island, water supply indicates that the water-supply contamination problem may be increased by a factor of 10 to 25 over that of direct water surface deposition when surface runoff from the watershed is considered (Grune et al., 1966).

The behavior of nuclides in surface water would be greatly affected by the mineral and organic matter content of the water (and, of course, by the chemical form of the released material). Sediments and suspended matter would exert a protective effect in that they readily adsorb many radionuclides. Therefore, some radionuclides would tend to be swept away and highly diluted by the surface water while others would move with the sediments. Sediment transport and dilution would therefore be an important radionuclide transport mechanism in flowing surface water.

For very large bodies of water, the Great Lakes and the oceans, the particularly important receptor region is that near the shoreline. The behavior of radionuclides would be especially complex in near-shore environments for a number of reasons. The affinity of the sediment for the radionuclide depends to a large extent on the particle size of the sediment, the amount of organic material present, and the degree of mixing. Currents and wave action would cause substantial mixing of sediment, both vertically within a given sediment mass and horizontally. If sediment accumulation is fairly rapid, as, for example, at some river mouths, a single layer of contaminated sediment would be mixed with, or buried by, later sediment layers. Near-bottom currents on the continental shelf are little known, but they would probably play a major role in transporting sediment-associated radionuclides. In contrast to the Atlantic coast of the United States, with its broad, shallow continental shelf, the Pacific coast has a very narrow mainland shelf. The inner shelf may be divided into two different hydraulic environments. Wave and wave-induced currents are the most improtant driving forces in the beach and surf zone. On the adjacent shelf the relative role of waves diminishes with increasing depth, and the role of tidal, density, and wind-drived currents becomes relatively more important. Under certain conditions, one could assume that the movement of radioactive particles would be close to that of sediment-associated radionuclides.

Another area of uncertainty is that of dispersion of radioactive material when the plume crosses terrain boundaries. In addition, the deposition velocity for particulate matter under these circumstances has not been defined.

K5.6 PLANT CONTAMINATION

Both wet and dry deposition of radionuclide particulate matter would temporarily leave films on vegetation. In addition, the soil can act as a secondary source of direct contamination of above-ground parts of plants through splashing by rain or resuspension by wind.

Chamberlain (1970) assumes the relationship between initial retention of deposited vapors and aerosols and herbage yield to take the form

$$1 - p = \exp(-\mu w),$$

where p is the fraction initially retained, w is the herbage density (kg/m^2 dry matter) and μ is an uptake coefficient (expressed in m^2/kg). For an average pasture having 250 g/m^2 dry matter, the above expression yields a figure of 44 to 50% for

initial retention. In dense herbage, contamination would be confined largely to the upper parts of the foliage. In addition, the form of vegetation determines to a considerable degree the extent to which it retains particulate matter (Witherspoon and Taylor, 1970). Retention is also highest when deposition occurs under conditions of high humidity.

One of the major factors in the removal of deposited radioactive materials would be washoff by precipitation. Wind, plant death, and subsequent loss of leaves, and removal by grazing animals are also factors to be considered.

In order to describe removal of deposited material by processes other than radioactive decay the concept of a "weathering half-life" has been introduced. This "weathering half-life" is experimentally derived from the expression

$$T_{eff} = \frac{T_W T_R}{T_W + T_R},$$

where T_{eff} is the effective residence time on herbage, T_R is radioactive half-life of the nuclide, the T_W is the weathering half-life. The quantity T_{eff} is based on contamination per unit area of ground surface so that it and T_W are independent of plant growth. A review of available evidence showed that this empirical expression held for a number of nuclides, including strontium-90, zirconium-95, iodine-131, and cesium-137; the mean value for T_W was 13 days (Thompson, 1965).

Chamberlain (1970) has similarly derived an average value for the half-life of 14 days on pastures during the growing period. However, it was found that for non-volatile particulates on fallout-contaminated shrubs at and near the Nevada Test Site a better value for the weathering half-life was $T_W \approx 30$ days (Martin, 1964, 1965).

There is some doubt as to the applicability beyond the first week or so for this form of loss rate. Krieger and Burmann (1969) observed two components of the loss of strontium-85 and cesium-134 from ungrazed pasture on which these nuclides had been sprayed; for the first week weathering half-life was 12 to 15 days, and for the second, 25 to more than 50 days.

K5.7 ANIMAL CONTAMINATION

There is no reliable method for estimating the degree of the contact hazard for animals exposed during deposition. Johnson and Lovaas (1970) studied the problem of initial retention of fallout on cattle. They found that the initial retention of near-in fallout-simulant sand (88 to 175 or 175 to 350 microns in diameter) on the backs of cattle averaged 50%. The retention half-time of simulant deposited on the animals' backs averaged 9 days for cattle kept under feedlot conditions.

K5.8 HUMAN CONTAMINATION

The major historical reference incident in which the effects of contact hazards were evidenced was in the exposure of the Marshall Islanders in 1954 (Cronkite et al., 1956). In this event, significant skin contact and adherence did occur. However, the particle size (60 to 200 microns) and the chemical nature (calcium oxide and calcium carbonate) of the fallout material are conditions that would not be expected from a reactor accident.

K6 EXPERIMENTAL DATA ON THE DECONTAMINATION OF ENVIRONMENTS

A large body of experimental data exists for the decontamination of environments contaminated by fallout from nuclear weapons. The three primary agencies responsible for the generation of these data were the following:

U.S. Naval Radiological Defense Laboratory
San Francisco, California

U.S. Army Nuclear Defense Laboratory
Edgewood Arsenal, Maryland

U.S. Agricultural Research Service
Beltsville, Maryland

As these data pertain primarily to nuclear weapons fallout, some of the data are not directly applicable to the nuclear reactor release contamination. In particular, many of of these data pertain to decontamination operations in which the native contaminated surface is not removed as a result of the decontamination (e.g., flushing or sweeping of the surface).

For these types of operation the efficiency and effort required of the decontamination operation are a strong function of (1) the contaminating particle size and (2) the initial surface mass loading of the contamination (weight per unit surface area). Since the expected contaminant particle size and initial surface mass loading are strikingly different for the nuclear reactor case, extrapolation of available data was performed when possible. However, the derived results should be used with caution. For the nuclear reactor case, interest is centered around particles on the order of 10 microns or less and an initial surface mass loading of a few grams per square foot and less.

For the experiments performed, simulated fallout was generally prepared by mixing a radioactive tracer with a short half-life and a bulk carrier material. The bulk carrier was generally a silica sand of specified particle size or local soil. The particles sizes generally ranged from 50 to 600 microns.

The decontamination results are discussed within two major groups, the temperate-climate (temperature > 40°F) and cold-weather results.

K6.1 TEMPERATE-CLIMATE RESULTS

K6.1.1 Hard Surfaces

K6.1.1.1 Pavements--Firehosing

The use of firehosing to clean paved areas is fast and utilizes readily available equipment. In general, however, its effectiveness decreases with decreasing particle size. Figures VI K-1 and VI K-2 show data from two sets of experiments (Wiltshire and Owen, 1965 and 1966) performed with a standard fire nozzle on roughly textured and smoothly textured pavements, respectively. In these experiments, several initial surface mass loadings were utilized, the lowest being 5 g/ft^2. The particle size of the contaminant was 44 to 88 and 300 to 600 microns. The results plotted in Figures VI K-1 and VI K-2 are for an initial surface mass loading of 25 g/ft^2 since the most experimental data existed for this value. The limited data for the initial surface mass loading of 5 g/ft^2 are shown in Figures VI K-3 and VI K-4. The two sets of data for each particular condition represent two passes with the firehose over the surface.

Due to the wide range in particle sizes for each experimental result, indicated by the horizontal bars, there is a great deal of uncertainty involved in extrapolating the results.

Figures VI K-1 through VI K-4 clearly show two very important points. First, with decreasing particle size, the additional effectiveness gained by a second pass of the firehose over the area decreases significantly. Second, based on the results and the extrapolation of the data, this decontamination method will have practically no effectiveness for particle sizes of 10 microns or less. One would expect, however, that the water-soluble radionuclides, not chemisorbed on the pavement surface, would be removed, although this would probably not represent a large fraction of the total contamination.

Examination of all the data shows that for a given particle size the decontamination effectiveness of the method was not very sensitive to the initial surface mass loading.

K6.1.1.2 Pavements--Mechanized Flushing

Figures VI K-5 and VI K-6 show the performance characteristics of mechanized street flushers for roughly textured pavements (Clark and Cobbin, 1964). In these experiments three consecutive passes over the area were performed. The experimental results indicate that the decontamination effectiveness of the methods is sensitive to the initial surface mass loading as well as the particle size. Therefore, plots of the results are given as a function of particle size in Figure VI K-5 and as a function of initial surface mass loading in Figure VI K-6.

As with the firehosing method, the additional effectiveness gained by multiple passes decreases significantly with both decreasing particle size and decreasing initial surface mass loading. In addition, the effectiveness of the decontamination method, it appears, would be marginal for particles of 10 microns and initial surface mass loadings of 1 g/ft^2 or less.

For smoothly textured asphalt (or concrete) the improvement in the results over those for the roughly textured asphalt is by a factor of about 3, as shown in Figure VI K-7. It appears that on sufficiently smooth surfaces some decontamination with the mechanized flushing is possible for particles of the 10-micron size. A reasonable estimate of the efficiency would be a decontamination factor of 5. Multiple passes over the surface would have little additional effect.

K6.1.1.3 Pavements--"Vacuumized" Sweeping

The two methods discussed in the preceding sections essentially displace the radioactive contaminants from the surface being cleaned. A more attractive procedure would be one that, in addition to cleaning the surface, would also pick up the removed contaminants. Such a method involves the use of a vacuumized sweeper (Clard and Cobbin, 1963). The values plotted in Figures VI K-8 and VI K-9 are from various tests performed with a vacuumized sweeper, for optimum operating conditions. They indicate that the efficiency of the method is sensitive to particle size and initial mass loading.

The data seem to indicate that the method would not be effective for particles smaller than 20 microns and initial surface mass loadings of less than 1.0 g/ft^2.

The general conclusion from the data is that the "vacuumized" sweeping method would have marginal effectiveness on smoothly textured asphalt or concrete. A reasonable estimate of the decontamination factor for this method for small particle sizes would be between 2 and 4.

One important advantage of the sweeper method is that temperature has little or no effect on its performance, as long as there is no snow, ice or water on the surface.

K6.1.1.4 Sloped Roofs--Firehosing

Experimental results from some limited firehosing tests (Wiltshire and Owen, 1965) are shown in Figure VI K-10. The results demonstrate that the performance of firehosing on these roofing materials (composition shingles and corrugated metal) exhibit the trends noted on pavements: removal effectiveness decreases as particle size decreases and surface mass loading. A reduction in nozzle pressure from 120 to 60 psi results in a reduction in decontamination efficiency by a factor of at least 2.

Test data on composition roll roofing and wood shingles are not sufficient to permit derivation of any meaningful results. However, it appears that firehosing roll roofing can be expected to be at least as effective as on composition shingles, while the results on wood shingles will be only about half as effective.

The conclusions to be drawn from the results is that firehosing these types of roofs appears to be only marginally effective, except for the very smooth surfaces. For corrugated metal a conservative estimate is that a decontamination factor of 5 could be achieved for particles of the 10-micron size.

K6.1.1.5 Flat Tar and Gravel Roofs--Firehosing

Table VI K-1 presents the evaluation of the decontamination of flat tar and gravel roofs by firehosing (Owen et al., 1960, 1962). It was found that the decontamination effectiveness was insensitive to the present of loose or fixed gravel, or to the properties of the simulated fallout.

It is not possible to judge from Table VI K-1 the effectiveness of the procedure for particles smaller than those used in the experiment (44 to 74 microns). One would expect, however, that the effectiveness would be somewhat lower than those given.

K6.1.1.6 Various Hard Surfaces

Some valuable information was gathered from small-scale decontamination exercises performed after nuclear weapons safety experiments. In one particular set of experiments a wide variety of surface types were exposed to fallout contamination from plutonium dispersed by a conventional high explosive (Dick and Baker, 1961). The results of these experiments are given in Table VI K-2. The contaminating particles (plutonium) in these experiments were determined to have an average diameter of 0.8 micron, with 99% of the particles being equal to or less than 2.5 microns.

The experiment surfaces were, except where indicated in Table VI K-2, small pads measuring 2 by 2 ft. Because of their small size, the possibility of recontamination during the decontamination operation is minimal. Therefore, the results should be viewed as upper limits achievable under ideal conditions, which rarely exist.

The decontamination of the surfaces was achieved by the following methods:

Wet Methods. A water truck capable of producing a water stream at a pressure of 400 to 750 psi was used for (1) plain water hosing, (2) water hosing and scrubbing, (3) hosing with water and detergent solution, and (4) hosing with a water and detergent solution, followed by scrubbing and rinsing.

Sandblasting. A standard sandblasting unit was used with no subsequent pickup of the contaminated sand.

Vacuuming. Industrial vacuum cleaners were modified to accept suitable filters over the exhaust to prevent resuspension of the contaminant blowing through the cleaner.

Steam Cleaning. A standard steam generator cleaner was used with an outlet pressure of approximately 90 psi of pure steam.

From these experiments it was observed, in general, that the effectiveness of decontamination by these methods was a function of the method rather than a function of the type of surface.

Sandblasting is not recommended for general use primarily because of its creation of a dust cloud bearing contaminant particulates.

K6.1.1.7 General Remarks Concerning Decontamination of Hard Surfaces

Many of the interpretations in the preceding sections were made with total neglect of important physical and chemical forces that affect the adhesion of particles to surfaces. Few of the factors that influence particle adhesion can be quantitated. Some of the factors known to influence the strength of these bonds are particle material, size, shape, and surface roughness, relative humidity of the ambient air, the presence of electrostatic charge, and the nature and physical characteristics of the substrate (Corn, 1961; Corn and Stein, 1964).

Equally important is the utility of using simulated contaminants for decontamination experiments. For example, as pointed out by Walker and Fish (1964), "there is justification for anticipating some difference in the adhesion of radioactive and of nonradioactive solid particles to solid surfaces. The radiation of the particle may affect the properties of the surface layers of the adhering particles and of the adherent surface. First, there may be sufficient energy deposited locally to desorb oxygen or other sorbed contaminants in the vicinity of the particle. This could greatly change the nature of the surface molecules in contact. In addition, the energy absorbed in the capillary layer of moisture between the particle and the surface may increase the rate of loss of moisture from the layer, potentially affecting a major factor of adhesion. Finally, radiation may produce a change in the electrostatic charge of the particle and of the adherent surface."

K6.1.2 Large Open-Land Areas

There are basically two types of treatment available for the decontamination of large open-land areas. The first type is physical removal of the radioactive contaminant, which generally involves removal of some portion of the native surface material. The second type is treatment of the land to reduce uptake by crops in agricultural land and to stabilize the radioactive contaminants in place or significantly below the ground surface. In a true sense the second type of treatment does not involve a decontamination of the land area, but rather it is a method to reduce exposure of man to the deposited radionuclides.

For the methods discussed in this section, the particle size of the contaminant is not a very important parameter. In addition, most of the simulated contaminants utilized in these experiments were 18 to 40 microns in size, which is not very much larger than the particle size of interest in the nuclear reactor case. Experiments have also shown that the amount of contaminant (initial surface mass loading) does not significantly alter the effectiveness of the decontamination methods. Therefore it is assumed that the results are directly applicable to the type of contamination problem possible from a major reactor accident.

An excellent review of treatment procedures and the feasibility of their application is provided by Menzel and James (1971). Valuable information has also recently been obtained from experiments performed by the Agricultural Research Service of the U.S. Department of Agriculture (James and Menzel, 1973).

K6.1.2.1 Removal of Crops, Crop Residues, and Other Low Vegetation

In general it has been found that the removal of contaminated vegetation is an ineffective method of decontaminating the land. The actual efficiency of the method depends on the density and type of vegetation and on the nature of the contaminant. The experimentally obtained efficiencies generally ranged between 25 and 75% removal (DF = 1.3 to 4.0). The removal of mulch from the surface was quite effective if the quantity per unit area of mulch was high enough and deposition occurred under wet conditions. Removal efficiencies greater than 95% (DF > 20) are achievable.

For subsequent treatment of the soil surface, it may be necessary to remove surface vegetation in any event. This would depend on the amount of vegetation per unit surface area.

K6.1.2.2 Removal of Surface Soil

Many common types of earth-moving equipment have been used in decontamination tests. These include graders, scrapers, and bulldozers. The effectiveness of any given procedure depends greatly on the terrain type. If the contamination lies on the surface of the soil, then careful removal of a layer of surface soil to a depth just slightly greater than the irregularities in the surface should remove all of the contamination. However, if the irregularities and fissures in the surface were deeper than the surface layer removed or if some of the surface layer material were spilled, then complete removal of the contamination would not be attained.

The amount of spillage is an important parameter in determining the overall effectiveness of surface soil removal. The ability of equipment to make clean cuts and efficiently pick up or otherwise remove a layer is directly related to the cohesiveness of the soil. Spills occur under the cutting blade, off the end of the blade, and occasionally over the top of the blade. The amount of spillage is also directly related to the skill of the equipment operator.

In most experiments on surface soil, removal of from 80 to 99% of the radioactive surface contamination was usually attained when 2 inches of soil were removed. In addition to the USDA Agricultural Research Service experiments (James and Menzel, 1973), a number of experiments have been performed by the U.S. Naval Radiological Defense Laboratory (Lee et al., 1959) and the U.S. Army Nuclear Defense Laboratory (Meredith and Maloney, 1964) on the decontamination effectiveness of removing surface soil. The results of these experiments are given in Table VI K-3.

Experiments on surface soil removal by bulldozing have demonstrated it to be the least effective method. Precise control of the equipment (bulldozer) is more difficult, and the bulldozer tends to gouge out more native surface material than necessary, thereby significantly increasing the waste disposal problem.

The cumulative-effort values in Table VI K-3 do not include support operations nor the time necessary to go to the disposal point and return. The grading operation leaves windrows of contaminated soil that may have to be removed by other equipment.

K6.1.2.3 Surface Soil Burial in Place

The most effective methods of placing the contaminated surface soil at a significant distance below the surface are deep plowing and grading. Typically, deep-plowing would place most of the surface contamination deeper than 30 inches. Table VI K-4 shows the profile of radioactivity after some deep plowing experiments (James and Menzel, 1973). This form of land treatment is effective in reducing the potential of direct contact to the radioactive contaminant and the external irradiation by the contaminant, but it is ineffective in reducing the uptake of radioactivity by vegetation.

To plow to depths of 39 inches, two large crawler tractors are generally necessary (James and Wilkins, 1969). About 1 acre can be plowed per hour of operating time, requiring two tractor drivers and one man at the controls of the plow.

The type of effect achieved by deep plowing can also be obtained by a grader-burial procedure. In this procedure a grader makes adjacent scraping passes to move windrows of contaminated soil into one large row. For a cut of 2 inches, no more than three adjacent passes can be made before objectionable spillage of contaminated soil occurs over the top of the blade. After the last pass a ditch is excavated beside the row of contaminated soil. The contaminated soil is scraped into the ditch and backfilled with clean soil. Under optimum operation conditions 100,000 square feet (2.3 acres) can be decontaminated in 3.3 hours by a grader with a 12-foot blade (James and Menzel, 1973). The fraction of contamination remaining on the surface soil after the operation is generally on the order of 10% (DF = 10).

K6.1.2.4 Sweeping Surface Soil

Street sweepers using vacuum or rotary brooms have been studied for removal of radioactive contamination from soil surfaces (Menzel, 1962; James and Menzel, 1973). For the procedure to be effective, heavy vegetation must first be removed from the surface. The primary advantage of the method is that it would leave the topsoil relatively undisturbed and large volumes of contaminated soil for disposal would not be generated.

Results from recent experiments are given in Table VI K-5 for several types of brooms and two soil types.

K6.1.3 Small Land Areas

A number of methods suitable for the decontamination of ground areas not accessible to large earthmoving equipment have been tested (Maloney and Meredith, 1962a; Cobbin and Owen, 1965). Lawns are the only surfaces that have been investigated with any thoroughness. Around structures considerable contamination can result from decontamination operations on the structure.

Because it gives greatest control of spillage, the most effective method tested so far on lawns is sod cutting and removal. The effort required was found to range from 1.6 to 3.2 man-hr/10^3 ft^2, depending on the soil type and condition. The residual fraction of contamination after decontamination was on the order of 1.5% (DF = 67).

K6.2 COLD-WEATHER RESULTS

Decontamination exercises performed at temperatures below 40F qualify as cold weather results. Only a very limited amount of data exists for cold-weather decontamination. These are principally from U.S. Army Nuclear Defense Laboratory experiments (Maloney and Meredith, 1962b; Maloney et al., 1962; Meredith and Maloney, 1964) and the plutonium contamination at Thule, Greenland (USAF, 1970).

In cold weather, the removal of surface contamination would usually be more difficult than in warm weather. If the soil surface were frozen, it could not be removed by scraping. Vacuuming or sweeping might be useful if the contaminant were not frozen into the surface.

The presence of snow can present several different problems, depending on whether the contaminant is beneath it, mixed with it, or deposited on top of it.

The experiments performed to date have been with simulated fallout with a particle size of 150 to 300 microns. Therefore, interpretation of the data must be performed with care for those operations that attempt to remove the contaminant from a hard surface. The decontamination effectiveness as a function of particle size would be expected to show the same type of behavior as seen for the results obtained under temperate conditions (firehosing or sweeping hard surfaces).

K6.2.1 Hard Surfaces

K6.2.1.1 Pavements and Frozen Ground Without Snow

Firehosing has been utilized in decontaminating hard surfaces of temperatures above 0F. It is believed, however, that the method would be ineffective at temperatures below 0F. The experimental results, for the same particle size, are very compatible with the temperate results shown in Figures VI K-1 and VI K-2 for firehosing pavements. As shown with these results, one would expect the method to have marginal effectiveness for particles 10 microns in size. Conventional or vacuumized mechanical sweeping of pavements would be expected to also show the same effectiveness as that for temperate climates.

It appears that on frozen ground the conventional street sweeper is the most effective piece of equipment. For example, for the particles of 150 to 300 microns and a temperature of 6 to 27F, three consecutive passes left residual fractions of 5.5, 0.55, and 0.30%, respectively (DF = 18, 180, and 333). The effort required for each pass was 2.0 equipment-minutes/10^3 ft^2. Note that these results fit well with the data for the vacuumized mechanized sweeper (Figure VI K-8).

K6.2.1.2 Pavements and Frozen Ground With Snow

On packed snow the conventional street sweeper has proved to be a most effective piece of equipment for contaminant removal. Since the surface layer of snow is removed during the sweeping operation, the effectiveness of the method would not be expected to be very sensitive to the contaminant particle size. Table VI K-6 presents the results of decontamination tests with a conventional sweeper.

Decontamination operation involving snow removal would not be very sensitive to particle size. The results should be directly applicable to the nuclear reactor accident.

For undisturbed snow the important parameters that would determine the effectiveness and effort required of a decontamination operation are snow depth and cohesiveness. In many of the experiments no snow depths were reported. Therefore, it is difficult to interpret many of the available data, and for a given procedure one finds a fairly large spread in experimental results. The available results are summarized in Table VI K-7.

Sufficient experimental information exists for the rotary snow blower to show clearly the influence of snow depth on the decontamination effectiveness. The experimental data have been plotted in Figure VI K-11 as a function of snow depth.

K6.2.2 Yards, Small Paved Areas, and Roofs

The results for the decontamination of yards, small paved areas, and roofs are summarized in Table VI K-8. Most of the methods have only marginal effectiveness. Many of the comments concerning cold-weather results in the preceding sections are also applicable here.

K7 CONCLUDING REMARKS

From a quick survey of the decontamination procedures explained in this appendix, it is evident that not all tested methods have been included in the discussion. For example, there have been many experiments performed on the utilization of irrigation and leaching to remove radionuclides from plant root zones and on the application of lime, fertilizers, and other soil amendments to reduce plant uptake of the radionuclides. These and other methods not discussed have either been found to be not practicable or not effective for the purpose intended.

It is evident from the interpretation of the results that without literally destroying the contaminated environment large decontamination factors are not attainable. A realistic obtainable value, averaged over large areas with many different surface types, appears to be DF = 20. This decontamination factor would be obtained by utilizing a combination of the methods discussed. For example, a higher decontamination factor on exposed ground would offset a lower decontamination factor obtained on paved areas.

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TABLE VI K-1 DECONTAMINATION OF FLAT TAR AND GRAVEL ROOFS BY FIREHOSING[a,b]

Number of Passes	Water Required (gal/ft^2)	Effort (nozzle-min/10^3 ft^2)	Residual Fraction (%)	DF
1	1.1	12	11.0	9.0
2		24	6.0	16.7
1	2	22	2-6	50-16.7
1	3.3	36	0.5-2	200-50

[a] From Owen et al. (1960, 1962).

[b] Particle size 44 to 74 microns; nozzle at 75 psi (three men per nozzle).

TABLE VI K-2 HARD SURFACE DECONTAMINATION EFFICIENCIES IN PERCENT[a,b]

Material	Vacuum (D + 2)	High-Pressure Water (D + 3)	High-Pressure Water with Scrub (D + 12)	High-Pressure Water and Detergent (D + 4)	High-Pressure Water and Detergent with Scrub (D + 5)	Sandblasting (D + 9)	Steam Cleaning (D + 14)
Glass	98.95	98.85	97.79	100.00	99.76	100.00	97.86
Stucco	48.00	97.94	95.22	100.00	99.59	100.00	27.00
Painted wood	99.28	98.43	96.77	99.69	99.97	100.00	91.61
Unpainted wood	36.00	85.00	93.18	99.54	95.54	99.90	85.00
Aluminum	89.00	99.45	97.33	99.62	100.00	98.49	84.00
Plate Steel	93.04	97.26	94.19	100.00	93.83	99.72	91.46
Asbestos shingles	61.00	99.97	98.91	96.89	99.36	100.00	63.00
Unpainted wood shingles	61.00	97.16	90.49	95.01	57.93	99.82	71.00
Brick	29.00	99.46	99.32	99.14	99.56	99.92	97.50
Tarpaper	55.00	98.66	95.04	95.32	95.83	99.51	52.00
Galvanized roofing	89.00	99.36	97.19	99.73	99.86	100.00	85.00
Highway asphalt	32.00	99.90	96.25	90.82	99.48	99.90	44.00
Highway asphalt (10 x 10 ft)	72.00	92.45	94.95	98.85	96.34	92.73	22.00
Sealed Asphalt	71.00	98.67	90.00	100.00	99.72	99.61	84.00
Sealed asphalt (10 x 10 ft)	64.00	90.00	82.00	96.31	97.54	90.42	48.00
Steel trowel concrete	74.00	98.94	--	96.91	99.53	100.00	--
Steel trowel concrete (10 x 10 ft)	--	73.00	97.34	--	98.58	98.96	27.00
Wood float concrete	--	98.00	92.03	100.00	97.47	100.00	65.00
Wood float concrete (10 x 10 ft)	56.00	97.84	--	98.09	98.28	98.78	85.00
Average of all surfaces	65.40	96.12	94.59	98.61	98.64	98.83	67.80

[a] From Dick and Baker (1961)

[b] Decontamination factor (DF) = 100/[100 - decontamination efficiency (%)];
(D + n) = number of days between contamination and decontamination.

TABLE VI K-3 DECONTAMINATION OF OPEN LAND AREAS BY SURFACE SOIL REMOVAL[a]

Method and Equipment	Soil and Condition	Number of Passes	Cumulative Effort Equipment-min/ 10^3 ft^2)	Residual Fraction (%)	DF
Scraping 2 to 4 inches deep (8 cubic yard capacity)	Moist turf	1	3.3	0.16	625
	Tilled soil (dry or moist)	1	4.0	1.0	100
		2	8.0	0.01	10^4
	Hard ground (dry or thawing)	1	5.0	3.5	29
		2	10.0	0.1	10^3
Grading 2 inches deep, only narrow strips (8- to 12-foot blade)	Moist turf	1	2.3	7.5	13
		2	4.6	0.5	200
	Tilled soil (dry or moist)	1	2.1	6.0	17
		2	4.2	0.4	250
	Hard ground (dry or thawing)	1	2.5	4.3	23
		2	5.0	0.1	10^3
Grading and scraping 2 inches deep	Moist turf	1	7.0	2.0	50
		2	14.0	0.04	2.5×10^3
	Tilled soil	1	5.0	4.5	22
		2	10.0	0.2	500
	Hard ground (dry or thawing)	1	6.0	1.7	59
		2	12.0	0.03	3.3×10^3

[a] From James and Menzel (1973).

TABLE VI K-4 PERCENTAGE OF RADIOACTIVITY AT VARIOUS DEPTHS AFTER DEEP PLOWING[a,b]

Sampling Depth (in.)	Radioactivity of High-Clay Content Pullman Soil (%)	Radioactivity of Sandy Laom Elkton Soil (%)
3	0.5	0.5
9	0.3	0.5
15	1.2	0.7
21	1.7	4.2
27	6.2	29.2
33	27.4	62.6
39	61.4	2.0
45	1.0	--

(a) From James and Menzel (1973).

(b) The plow broke up and increased the volume of the soil removed from the furrow. As a result, the plowed field was at a higher elevation than before plowing. While the plow was opening a 39-inch-deep furrow as measured from the former ground surface, the fluffed-up soil measured as much as 45 inches.

TABLE VI K-5 CUMULATIVE PERCENTAGE OF RADIOACTIVITY REMOVED BY REPEATED PASSES OF A ROTATING-BRUSH, MECHANICAL STREET SWEEPER[a]

Broom Material and Sweeping Procedure	Cumulative Percent Removed by Successive Passes								
	Sassafras Sandy Soil					Elkton Silt-Laom Soil			
	1[b]	2	3	4	10	1[b]	2	3	4
Steel:									
Normal pass first	74	86	91	92	--	80	89	75	92
Suction pass first	73	86	92	94	100	84	95	85	94
Steel and gutter broom:									
Normal pass first	73	84	92	96	99	78	90	95	94
Suction pass first	52	75	93	90	--	50	54	77	78
Plastic:									
Normal pass first						38	51	70	90

[a] From James and Menzel (1973).

[b] Number of passes.

TABLE VI K-6 DECONTAMINATION OF SURFACES WITH PACKED SNOW WITH A CONVENTIONAL SWEEPER[a,b]

Snow Condition	Temperature (°F)	Effort (Equipment-min/ 10^3 ft^2)	Residual Fraction (%)	DF
Packed	-6 to -4	1.2	6.6	16
(No depth reported)	+20 to ±24	2.0	5.8	17

(a) From Maloney and Meredity (1962b).

(b) Elgin sweeper with 72-inch broom. Particle size 150 to 300 microns.

TABLE VI K-7 DECONTAMINATION OF SURFACES WITH UNDISTURBED SNOW [a]

Method	Work Rate (ft /min)	DF
Snowplow (blade)	5500	10-35
Grading	2085	2-20
Scraping	1200	5-7
Snowplow (rotary)	883	7-50

[a] From Horan and Cunningham (1969).

[b] Particle size 150 to 300 microns.

TABLE VI K-8 DECONTAMINATION OF YARDS, SMALL PAVED AREAS, AND ROOFS [a,b]

Surface and Condition	Method and Equipment	Temperature (°F)	Effort (man-hr/10^3 ft^2)	DF
Ground (frozen)	Hand broom	22	0.78	5
	Vacuum cleaner	28	0.71	2
Ice (frozen lake)	Hand broom	28	0.55	29
	Vacuum cleaner	33	0.88	11
Loose snow:				
6.0 in. deep	Hand snow shovel	22 to 35	2.8 to 0.84	8 to 14
	Hand snow plow	28	0.28	8
	Garden tractor snow blade	28	0.073	4
Slope room (composition shingles)	Lobbing, 2.5-in. nozzle at 40 to 50 psi	2 to 14	5.5 [c]	27
	Hand broom	18 to 26	1.1	>5
	Forced air lance	30	0.18	6

[a] From Owen et al. (1967).

[b] Particle size 150 to 300 microns.

[c] Nozzle-minutes per 1000 ft^2, with three men per nozzle.

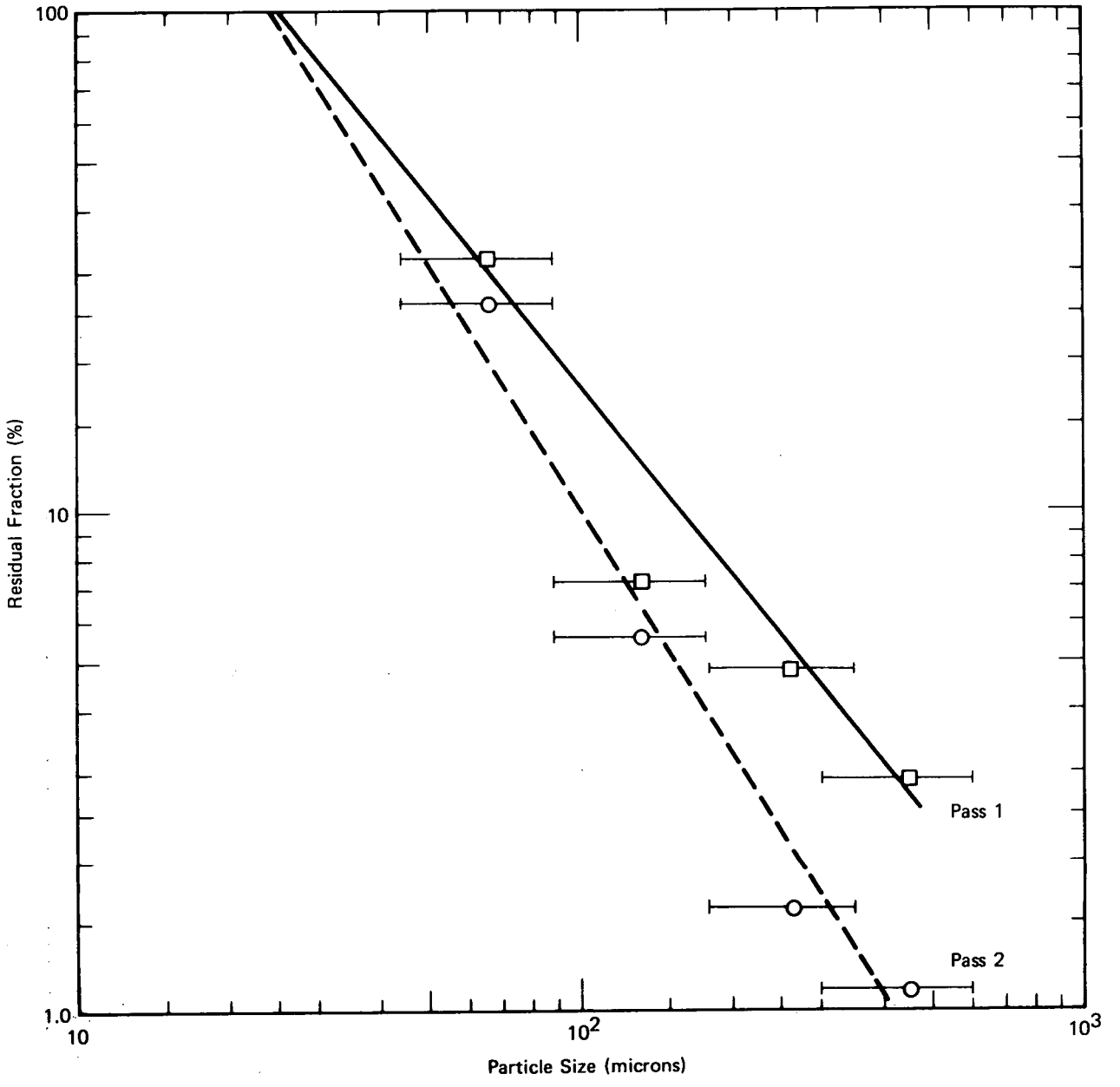

FIGURE VI K-1 Decontamination of roughly textured asphalt (or concrete) by firehosing (standard nozzle). Initial mass loading = 25 g/ft^2. [DF = 100/residual fraction (%).]

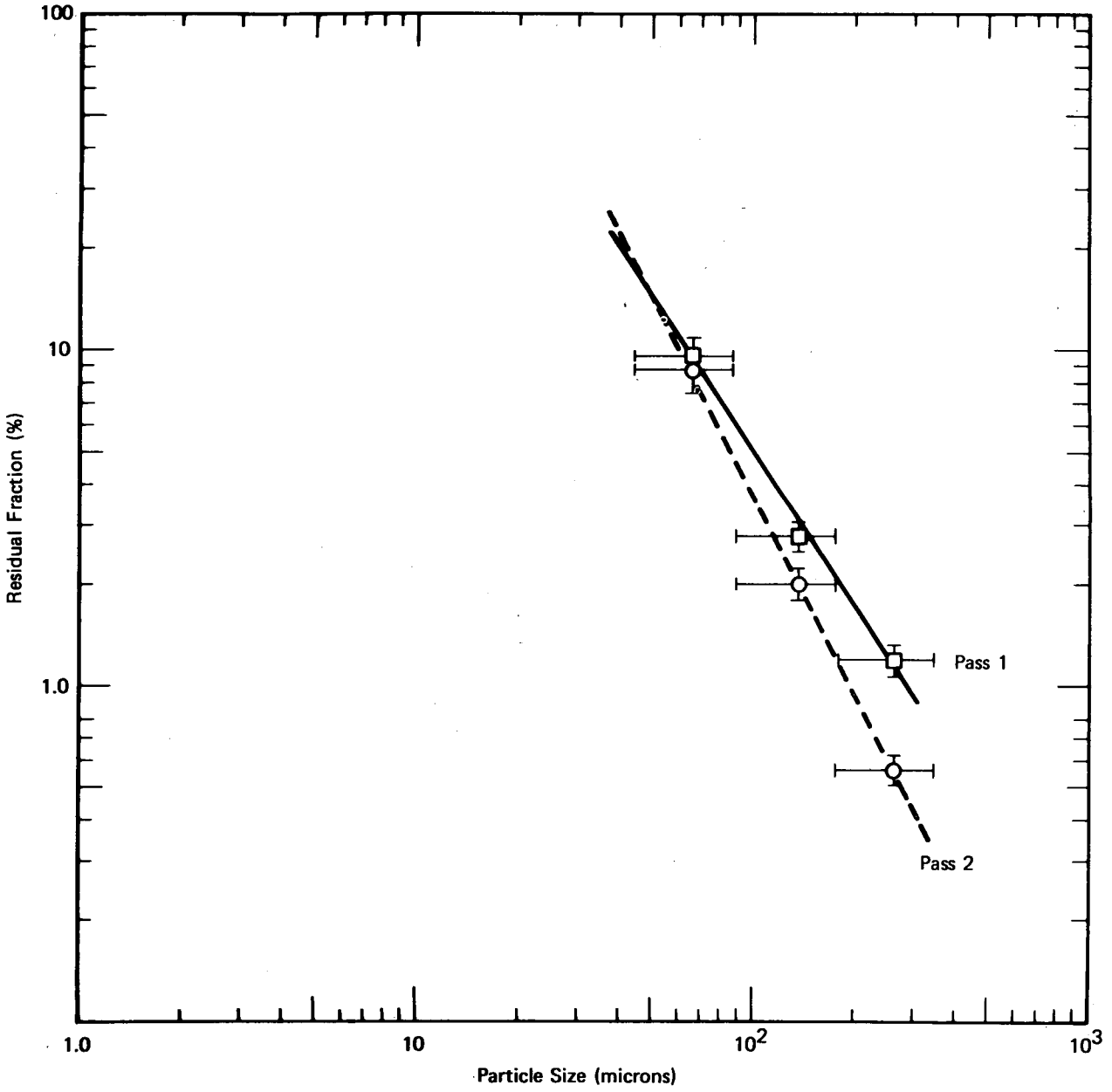

FIGURE VI K-2 Decontamination of smoothly textured asphalt (or concrete) by firehosing (standard nozzle). Initial mass loading = 25 g/ft^2. [DF = 100/residual fraction (%).]

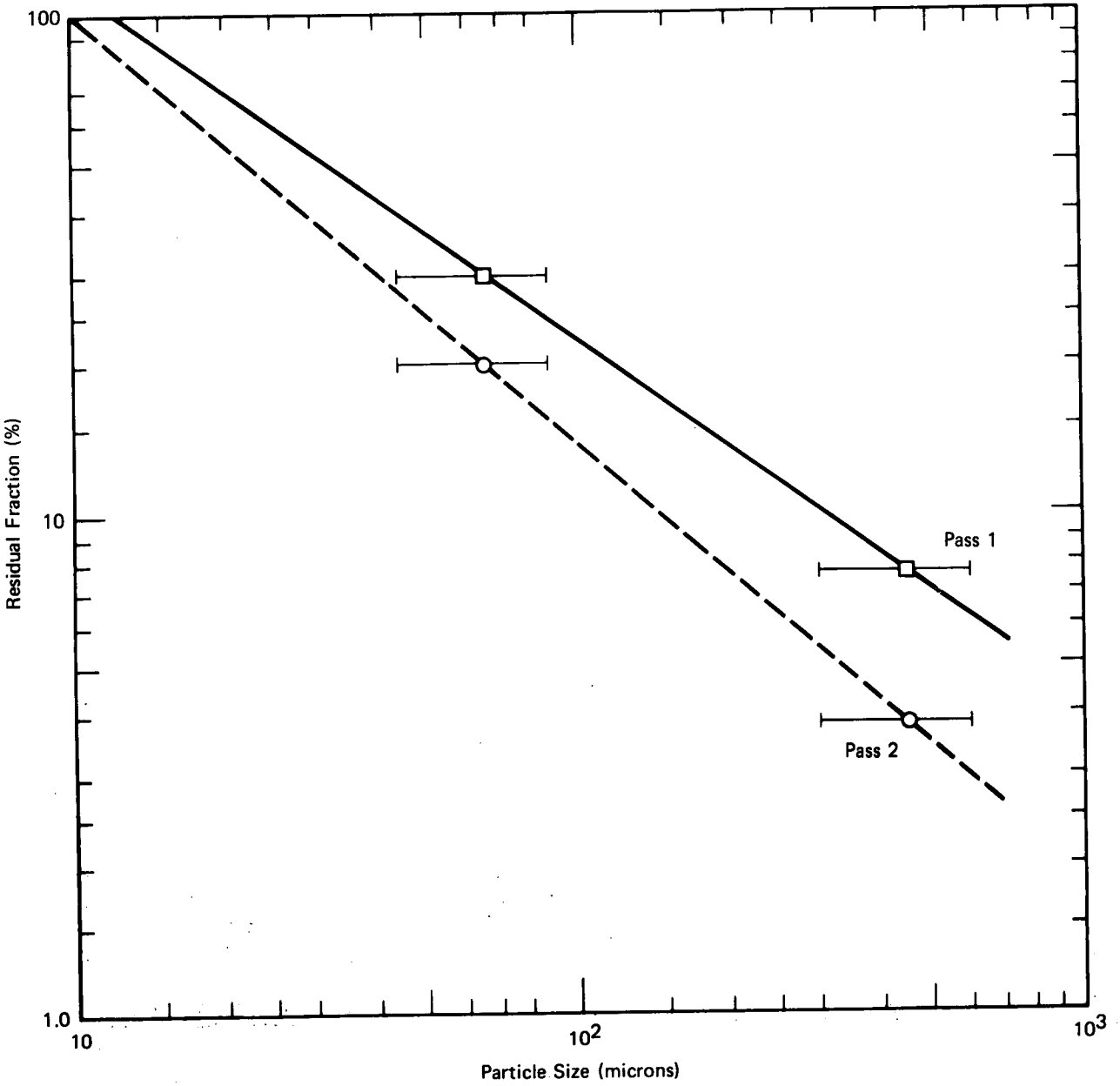

FIGURE VI K-3 Decontamination of roughly textured asphalt (or concrete) by firehosing (standard nozzle). Initial mass loading = 5 g/ft^2. [DF = 100/residual fraction (%).]

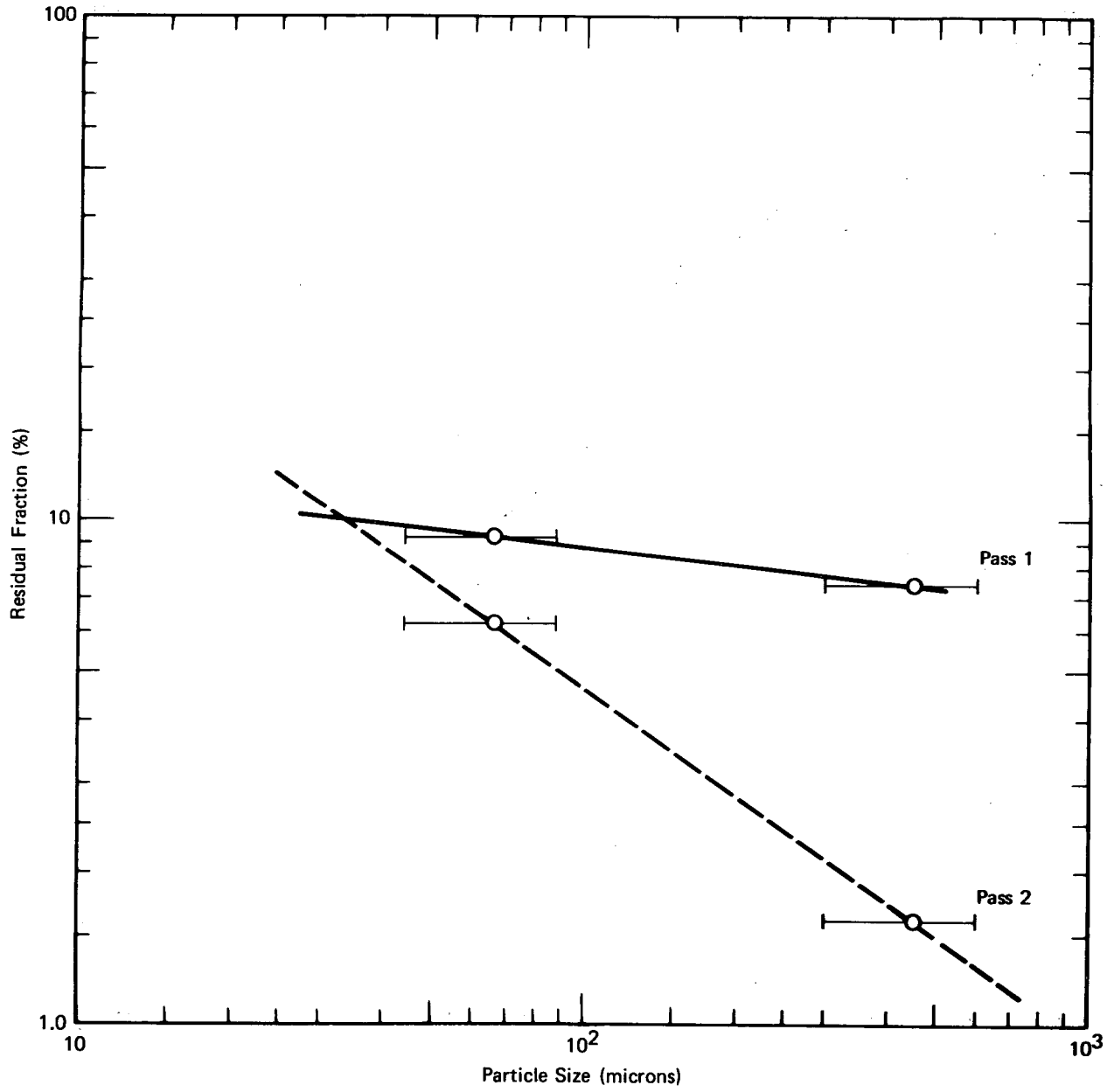

FIGURE VI K-4 Decontamination of smoothly textured asphalt (or concrete) by firehosing (standard nozzle). Initial mass loading = 5 g/ft^2. [DF = 100/residual fraction (%).]

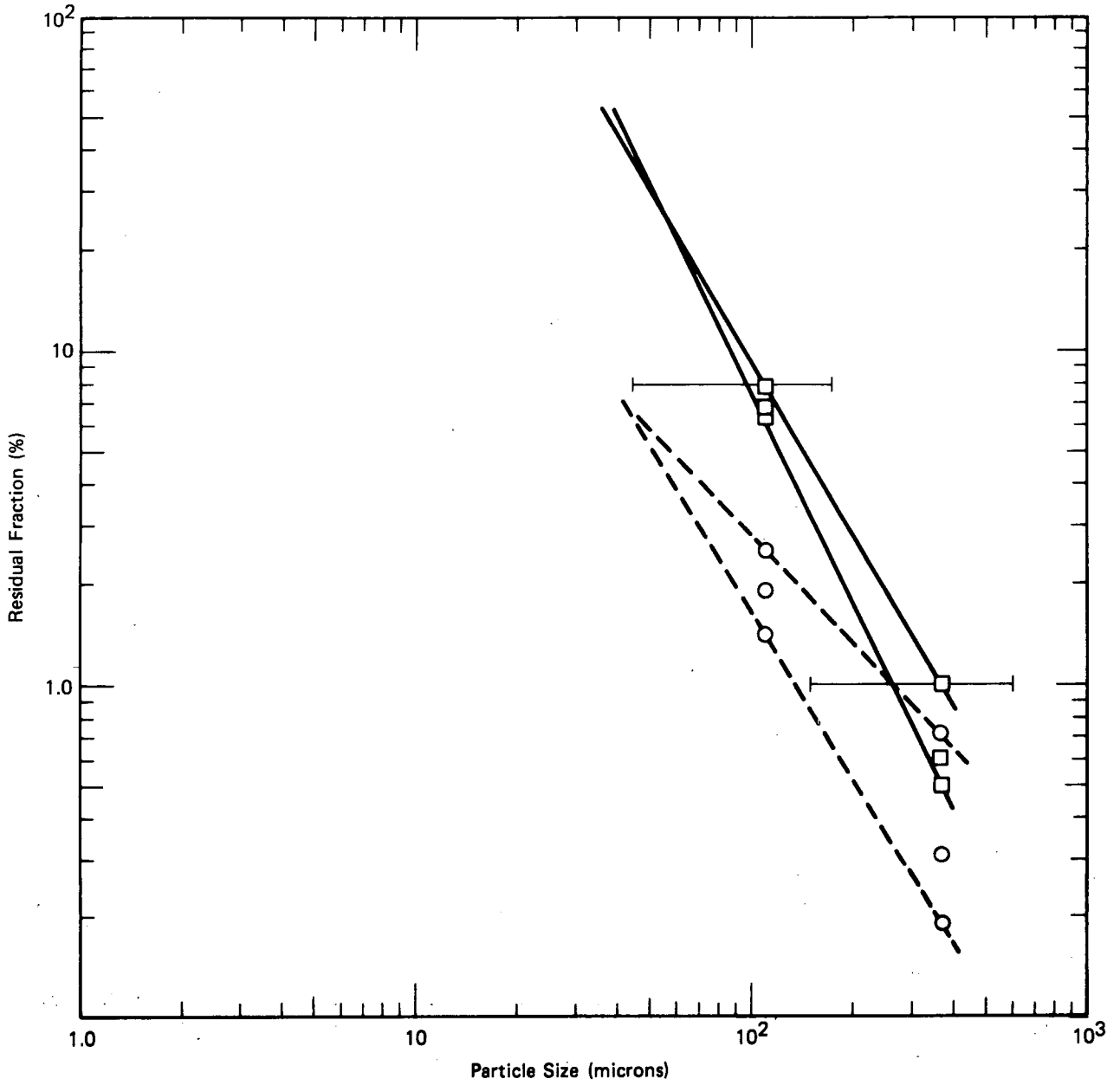

FIGURE VI K-5 Decontamination of roughly textured asphalt (or concrete) by mechanized flushing (three consecutive passes). Initial mass loading = 5 g/ft^2 (□) and 25 g/ft^2 (○). [DF = 100/ residual fraction (%).]

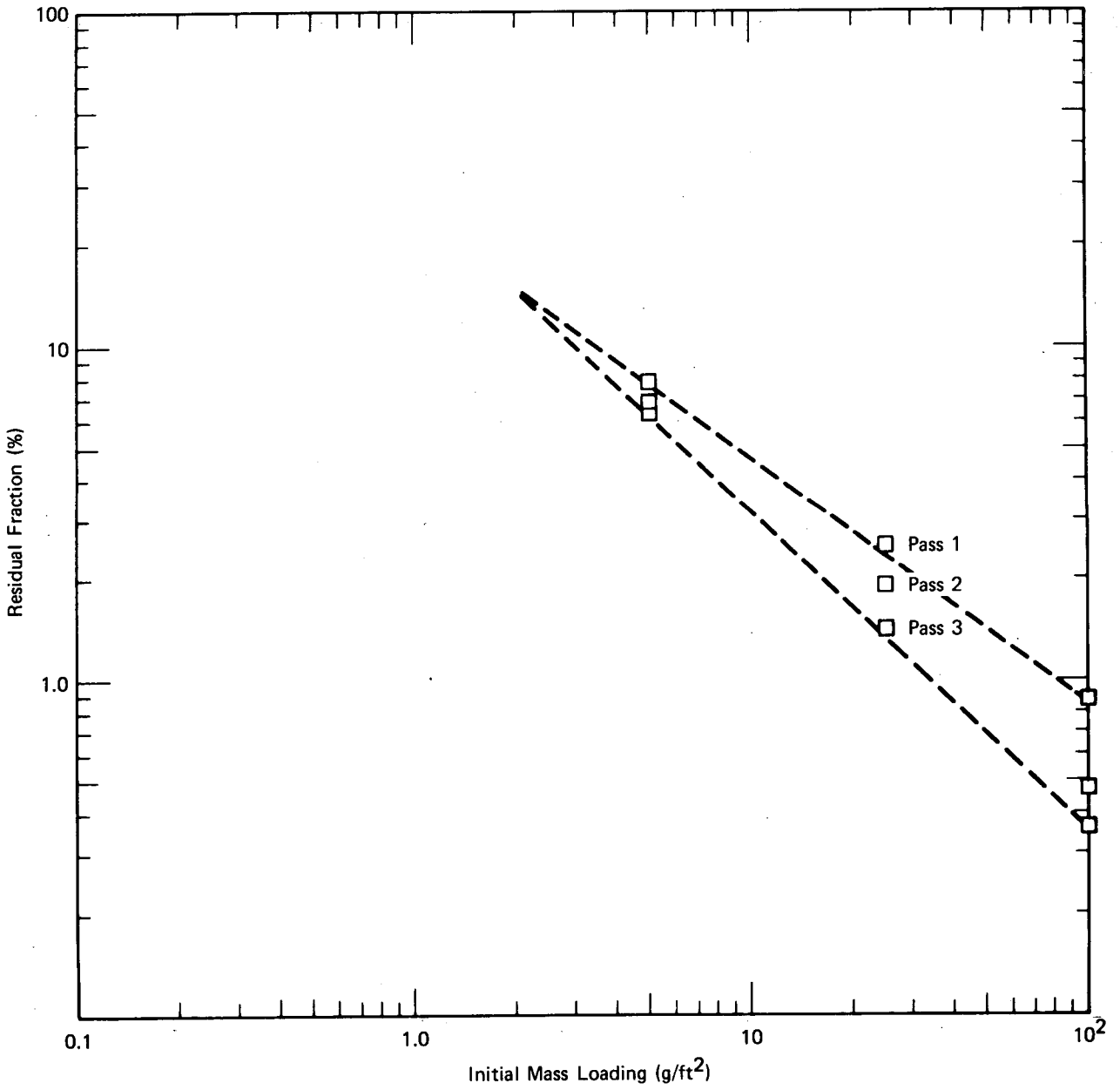

FIGURE VI K-6 Decontamination of roughly textured asphalt (or concrete) by mechanized flushing. Particle size = 44 to 100 microns. [DF = 100/residual fraction (%).]

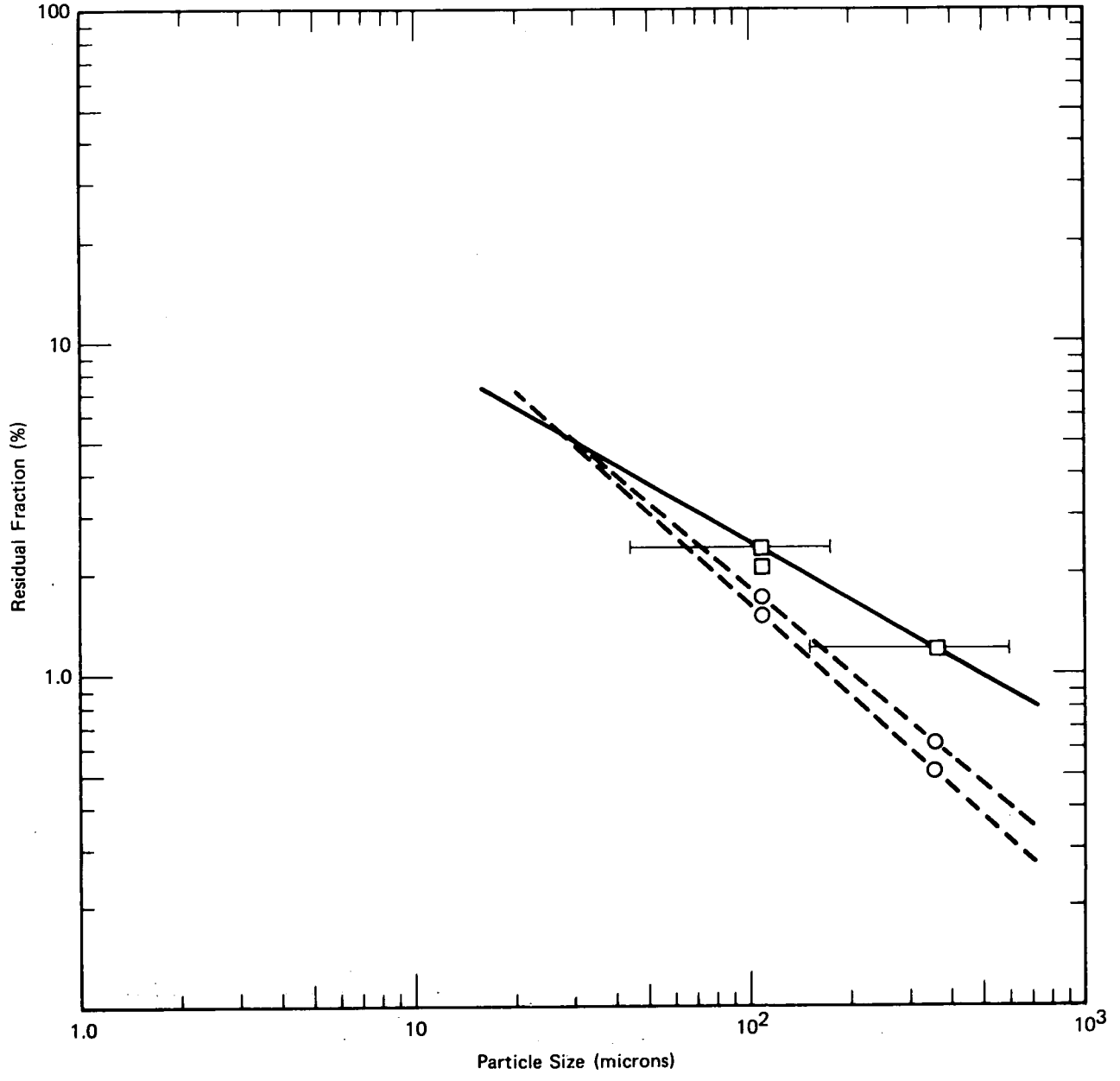

FIGURE VI K-7 Decontamination of smoothly textured asphalt (or concrete) by mechanized flushing (two consecutive passes). Initial mass loading = 5 g/ft^2 (□) and 12 g/ft^2 (○). [DF = 100/ residual fraction (%).]

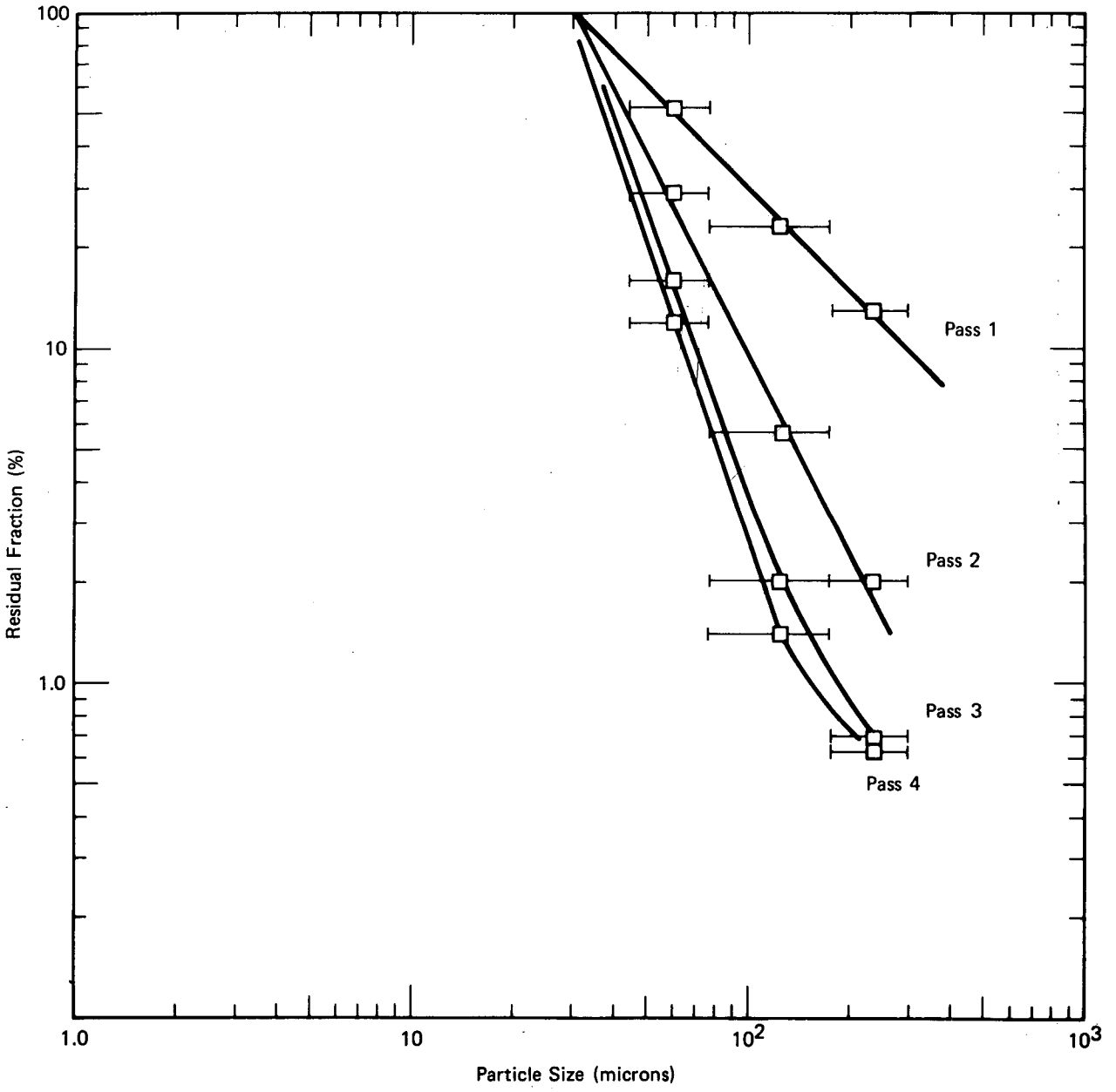

FIGURE VI K-8 Decontamination of roughly textured asphalt (or concrete) by "vacuumized" sweeper. Initial mass loading = 25 g/ft^2. [DF = 100/residual fraction (%).]

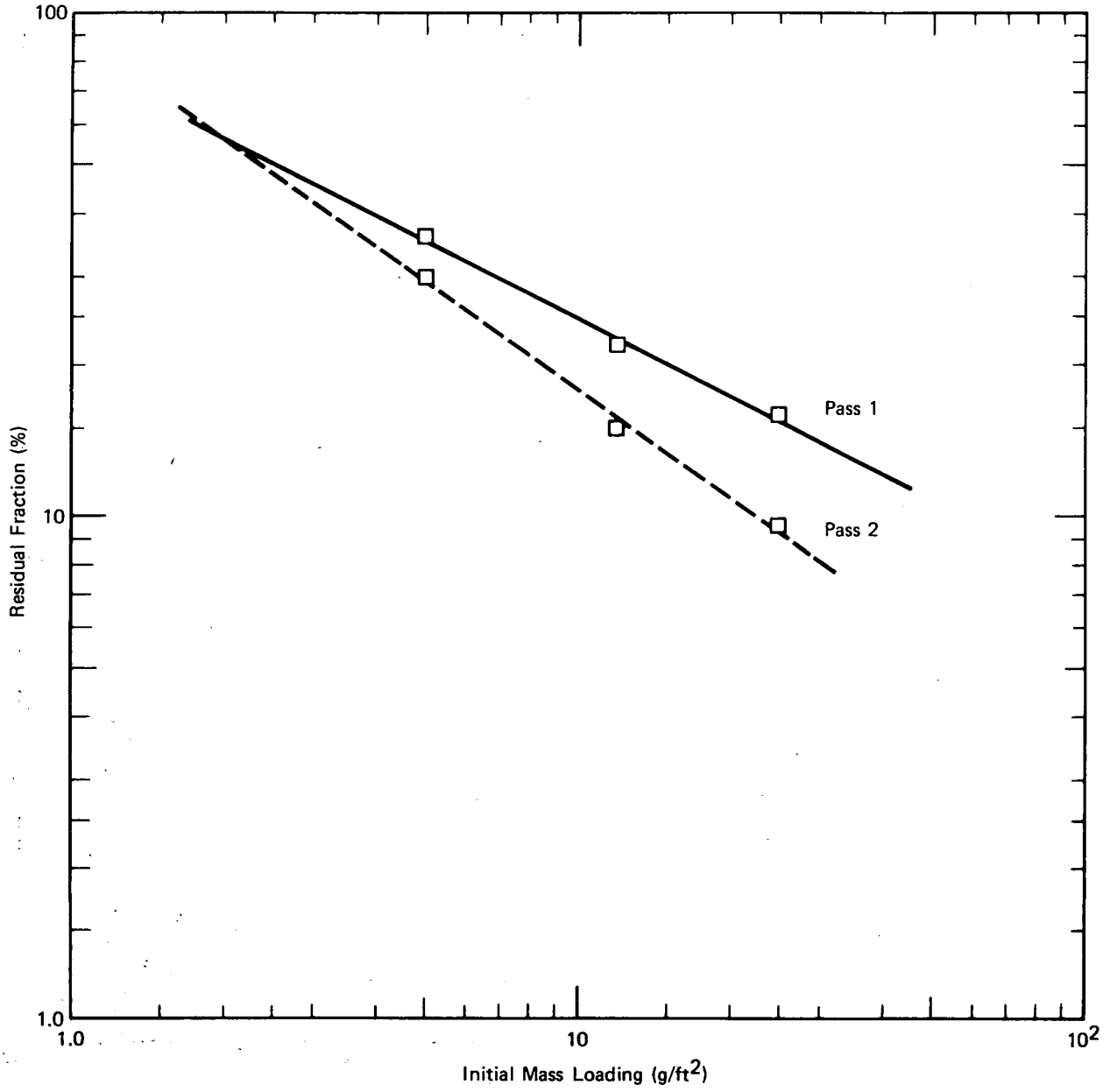

FIGURE VI K-9 Decontamination of roughly textured asphalt (or concrete) by "vacuumized" sweeper. Particle size = 44 to 74 microns. [DF = 100/residual fraction (%).]

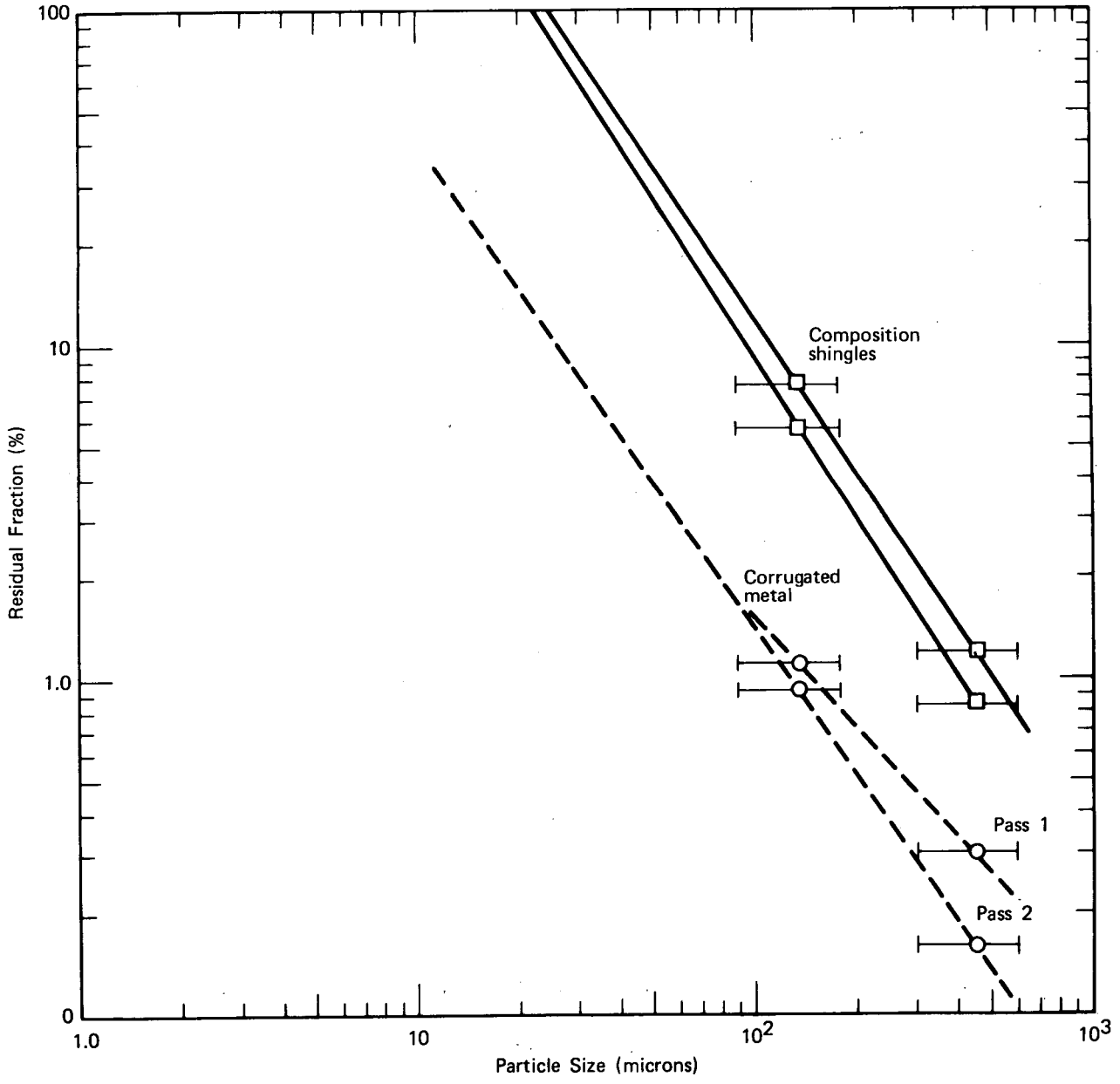

FIGURE VI K-10 Decontamination of sloped roofs by firehosing. Initial mass loading = 25 g/ft^2. [DF = 100/residual fraction (%).]

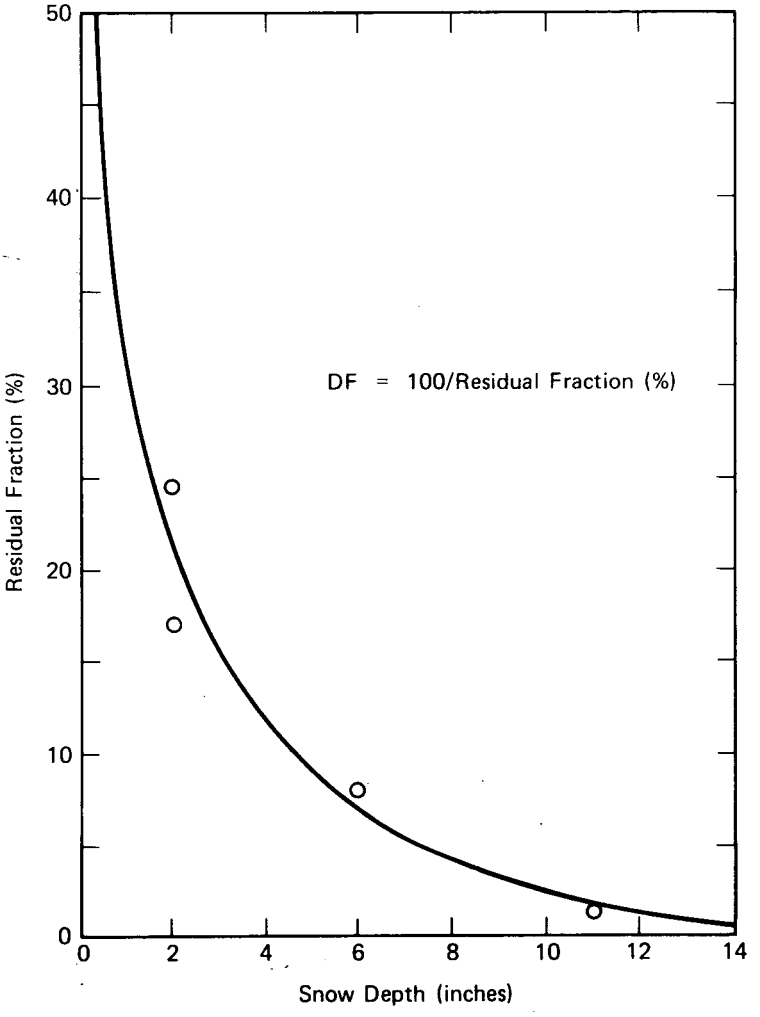

FIGURE VI K-11 Influence of snow depth on the decontamination effectiveness of the rotary snow blower. From Owen et al. (1967).

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

Reactor Safety Study
WASH-1400 (NUREG-75/014)
Errata sheet for Appendix VI

p 3-1 line 28	Should read "...26,400 thermal megawatt-days per..."
p 4-2 line 22	The sentence "If the effect...about 2.3 kilometers"[1] should be footnoted as follows: [1] A sensitivity study showed that this building wake effect always had small impact on the results and usually reduced their magnitude; it was therefore neglected.
p 4-2 line 35	Change to read "...where p is taken as 1/3."
p 4-3 Table VI 4-2	Column heading should read "Change with Height (K/100 m)"
p 4-3 Table VI 4-2	Last row in "Temperature" column replace 1.5 to + 4.0 with >1.5.
p 5-1 line 27 through p 5-2 line 10	In this paragraph, delete following material: "Figs. VI 5-1a through g show...flow is quite dominant. Actually," Paragraph should start with "For the six composite sites,..."
p 5-1	Second sentence of footnote should read "However, because one of the sites was..."
p 8-5 line 27	Change reference to Table VI 8-6 to Table VI 8-5.

p 8-15 line 24	Following the sentence "In general,..., oxide formation is expected.", remainder of paragraph should read as follows: The iodines can exist as elemental iodine, hydroiodic acid, or organic halides. The possible formation of hydroiodous acid may also lead to the presence of iodides and iodates. For several of the "transition" elements, the formation of oxygenated anions is also possible. Molybdenum oxides are assigned to class Y, whereas the molybdates are assigned to class W. All molybdenum compounds are treated as class Y aerosols in the inhalation model. Since molybdenum-99 has a short half life of 218 days, a small overestimate of the lung dose will result if a portion of the molybdenum is present as molybdates rather than oxides. Cerium is expected to be in an oxide form. Based on the recommendation of Morrow, the cerium compounds are assigned to class Y which may introduce a small overestimate of the lung dose attributable to cerium if the cerium compounds in fact behave as class W aerosols. The categorizing of the aerosol clearance from the pulmonary region does not include the noble gases.
p 8-17 Table VI 8-2	Mo-99 is assigned to lung clearance class Y.
p 8-17 Table VI 8-2	Add Pu-239 to last group of radionuclides
p 8-18 caption to Fig. VI 8-6	Change line 4 to "...the activity <u>or</u> mass median..."
p 9-11 line 27	Should read "As suggested in <u>Fig. VI 13-7</u>, at all distances..."
p 9-20	Modify first paragraph under 9.2.3.8 to read: "The study defines early morbidities as those requiring medical attention and possibly hospital treatment. Respiratory impairment and hypothyroidism clearly fall into this category, but prodromal vomiting, lasting only a short time and having no lasting effect on the individual, would be excluded under this definition. A small segment, (e.g. 5%) of the population might have a more serious reaction to prodromal vomiting. The number of early morbidities stated in section 13 are the cases of respiratory impairment plus 5% of the cases of prodromal vomiting."

p 9-22 line 52	Should read "...applies the incidence rate used for the central estimate."
p 9-25 line 9	Should read "...a 40-year-old man received 1.075 rem by age 10..."
p 10-3	Title of Table VI 10-3 should read "CONSTRUCTION OF A COMPOSITE REACTOR SITE"
p 11-16 line 17	Should read "...(more than 30 years)"
p 11-20	Change last paragraph to read: "In conjunction with decontamination, the consequence model also assumes limited land interdiction. Land interdiction is assumed to be required for the area where the maximum decontamination factor will not bring the ground contamination below the land interdiction criteria, L_3. This area would be located to the left of R_2. After some time period (n years), radioactive decay and weathering forces will reduce the ground contamination so that people are able to return to the land located between R_2 and R_3."
p 11-21	A corrected Fig. VI 11-8 is attached.
p 11-22	Title of Table VI 11-7 should read "REPRESENTATIVE SHIELDING FACTORS FROM SEMI-INFINITE GAMMA CLOUD SOURCE."
p 13-1 line 26	Should read "...for radionuclides deposited or inhaled and include."
p 13-7 Fig. VI 13-5	On the ordinate, 10^6 should be moved down to proper location.
p 13-24	A corrected Table VI 13-3 is attached.
p 13-25	A corrected Table VI 13-4 is attached.
p 13-26 line 2	Should read "...Meteorology is roughly a cyclical process."
p 13-33 Fig. VI 13-24	Correct footnote (c) as follows: "...zero within 25 miles."
p 13-37	A corrected Fig. VI 13-26 is attached.

p 13-39 — Title of Section 13.4 should be:

"RISK CALCULATION FOR ONE REACTOR"

p B-8 — Equation VI B-7 should read:

$$\bar{z} = \frac{1}{\chi(0)} \int_0^\infty \chi(z)dz = \ldots$$

p B-8 — Unnumbered equation below Equation VI B-7 should read:

$$f \simeq v_d t_i / \bar{z} .$$

p D-4 — Caption to Fig. VI D-1. Change line 4 to

"...the activity or mass median..."

p D-15 — Change title of Table VI D-4 to:

"MASSES OF ADULT ORGANS AND RATIOS OR ORGAN MASSES OF REFERENCE MAN TO CHILD ORGAN MASSES BY AGE."

p D-15 — Change third parameter in Table VI D-5 to:

"Surface area of total body (m^2)"

p D-21 line 21 — Should read "...concentration of about 0.1 microgram per gram of tissue."

p D-22 line 19 — Add the following sentence at the end of the paragraph:

"Morrow (1975b) has indicated that cerium compounds are likely to behave as class Y aerosols."

p D-22 line 38 — Change the sentence to read:

"The same metabolic model is used for all four elements."

p D-27 line 16 — Change to read "...molybdates appear to be in class W (Morrow, 1966)."

p D-29 line 21

Add the following sentence at the end of the paragraph:

"The remainder of the activity is indicated for early excretion."

TABLE VI 13-3 CONTRIBUTION OF DIFFERENT EXPOSURE MODES TO LATENT CANCER FATALITIES

	Percentages							
	Leukemia	Lung	Breast	Bone	GI Tract	All Other	Total	Whole Body[a]
External cloud	0.2	0.5	0.5	0.1	0.1	0.3	1	3
Inhalation from cloud	0.5	59.0	9.0	0.2	1.0	0.2	70	15
External ground (<7 days)	4.0	0.8	0.8	1.0	1.0	3.0	11	47
External ground (>7 days)	2.0	2.0	5.5	1.0	1.0	2.0	13	30
Inhalation of resuspended contamination	0.1	3.0	0.1	0.1	0.1	0	3	2
Ingestion of contaminated foods	0.2	0.2	0.5	0.1	0.1	0.2	1	4
Subtotals	7	66	16	2	3	6	100	100

(a) Whole body values are proportional to 50-year whole-body man-rem.

TABLE VI 13-4 EXPECTED CASES OF LATENT CANCER FATALITIES PER MILLION MAN-REM

Method	Upper Bound (BEIR)	Central Estimate	Threshold (Rem) 10	25
Whole body	122	48	46	31
Sum of individual organs	200	104	101	86