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.

NUCLEAR REGULATORY COMMISSION

Docket No. 50-352/353 Official Exh. No. 40-137

In the matter of Philadelphia Electric

Staff _____ IDENTIFIED ✓

Applicant ✓ _____ ADMITTED _____

Intervenor _____ REJECTED _____

Gov't Agency _____

Contractor _____ DATE 5/22/84

Other _____ Witness _____

Reporter Ann Riley

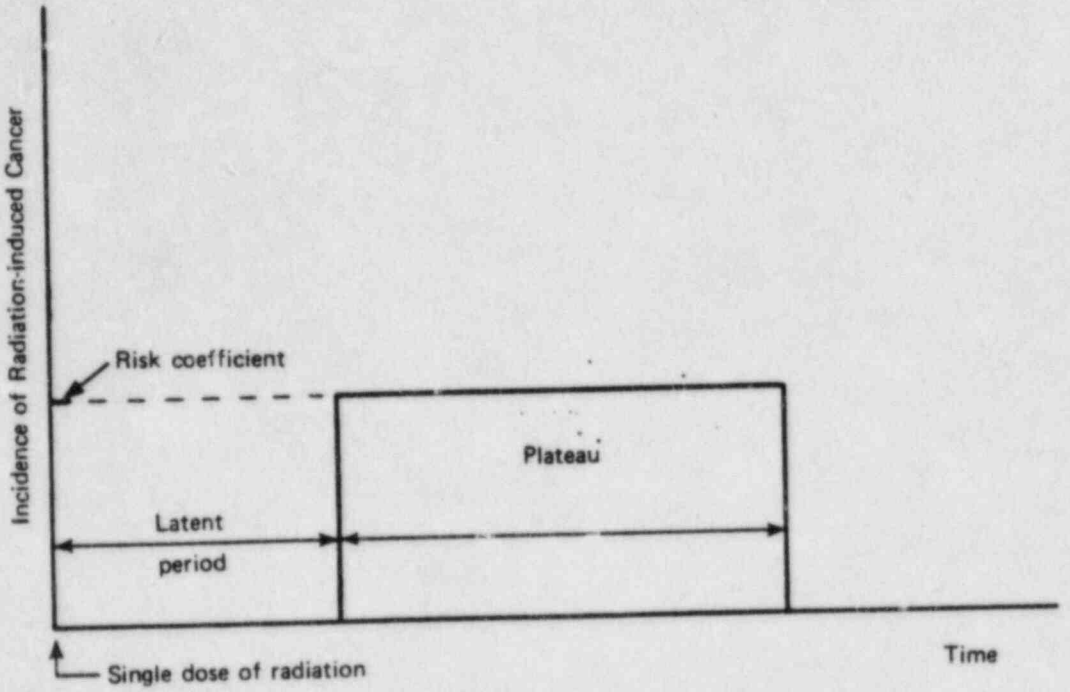

FIGURE VI 9-10 Basic model for latent cancer fatalities.

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 30 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 C, 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.

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[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 9-10 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 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.

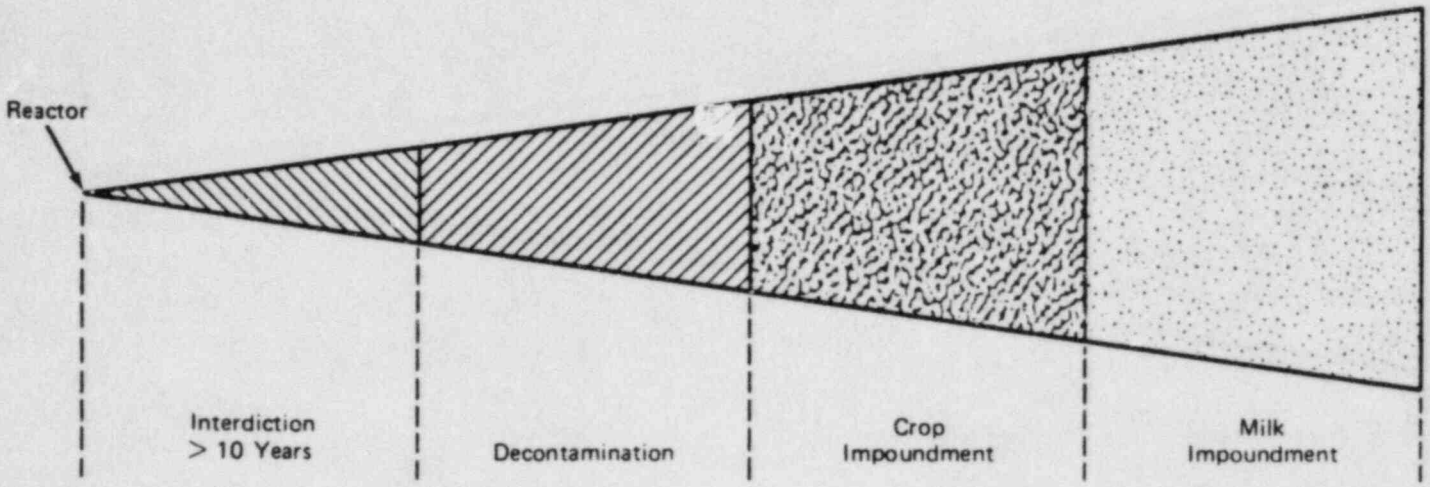

FIGURE VI 11-1 Simplified interdiction model.

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

Evacuation Category	Effective Speed (mph)		
	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.

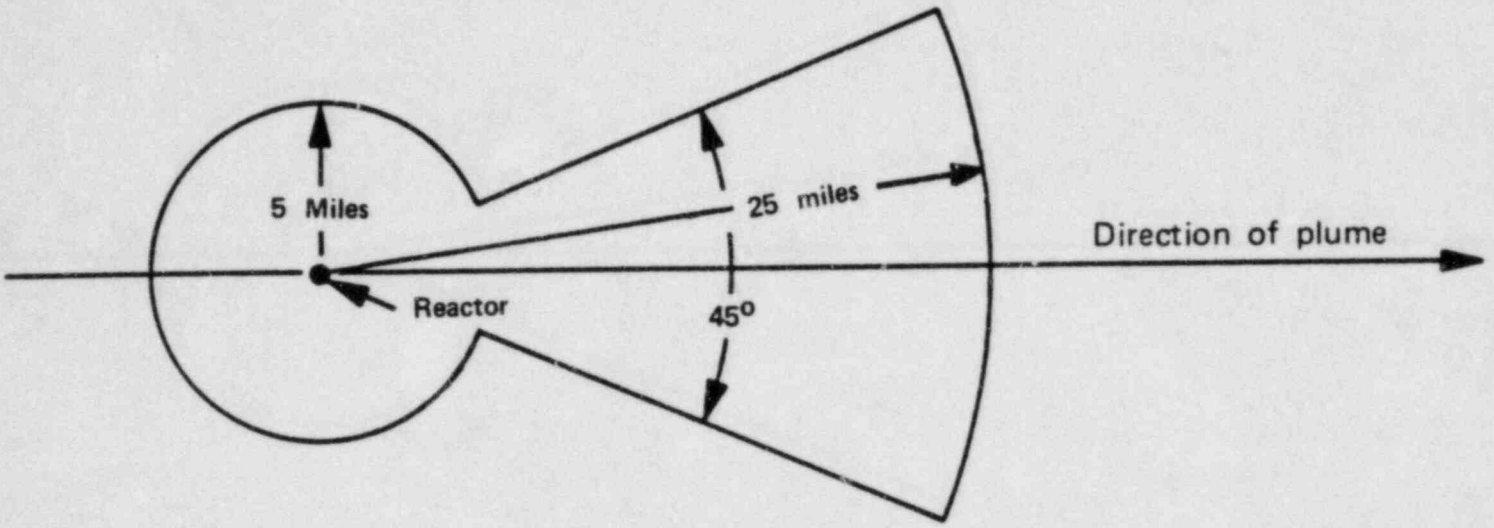

FIGURE VI 11-2 Evacuation area used for cost calculations.

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, $n = 0.9\bar{u}$, where u is in meters per second and n is in reciprocal units of 1 hour. For a cloud of constant air concentration, which would give a dosage external to the building of $\bar{V}_o$, in a time Δt, the dosage inside a shelter, $\bar{V}_i$, is given by (Slade, 1968)

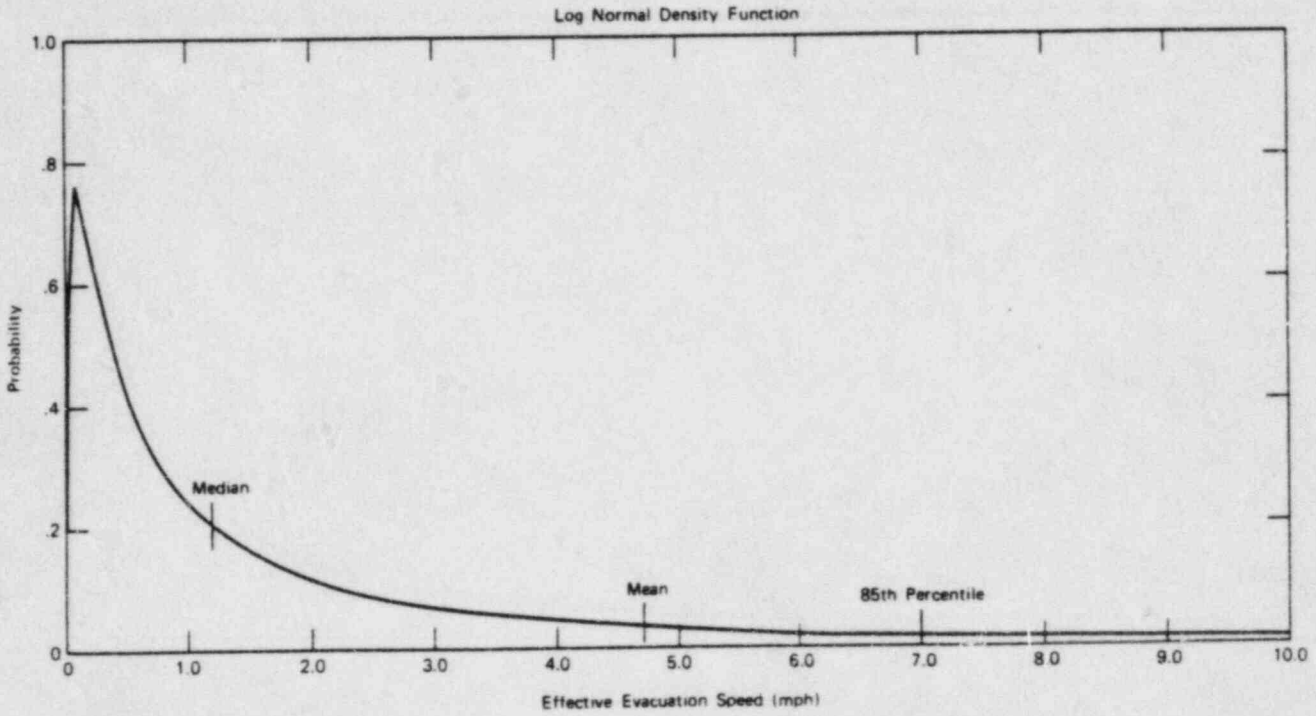

FIGURE VI 11-3 Log-normal distribution of evacuation speeds.

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

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.

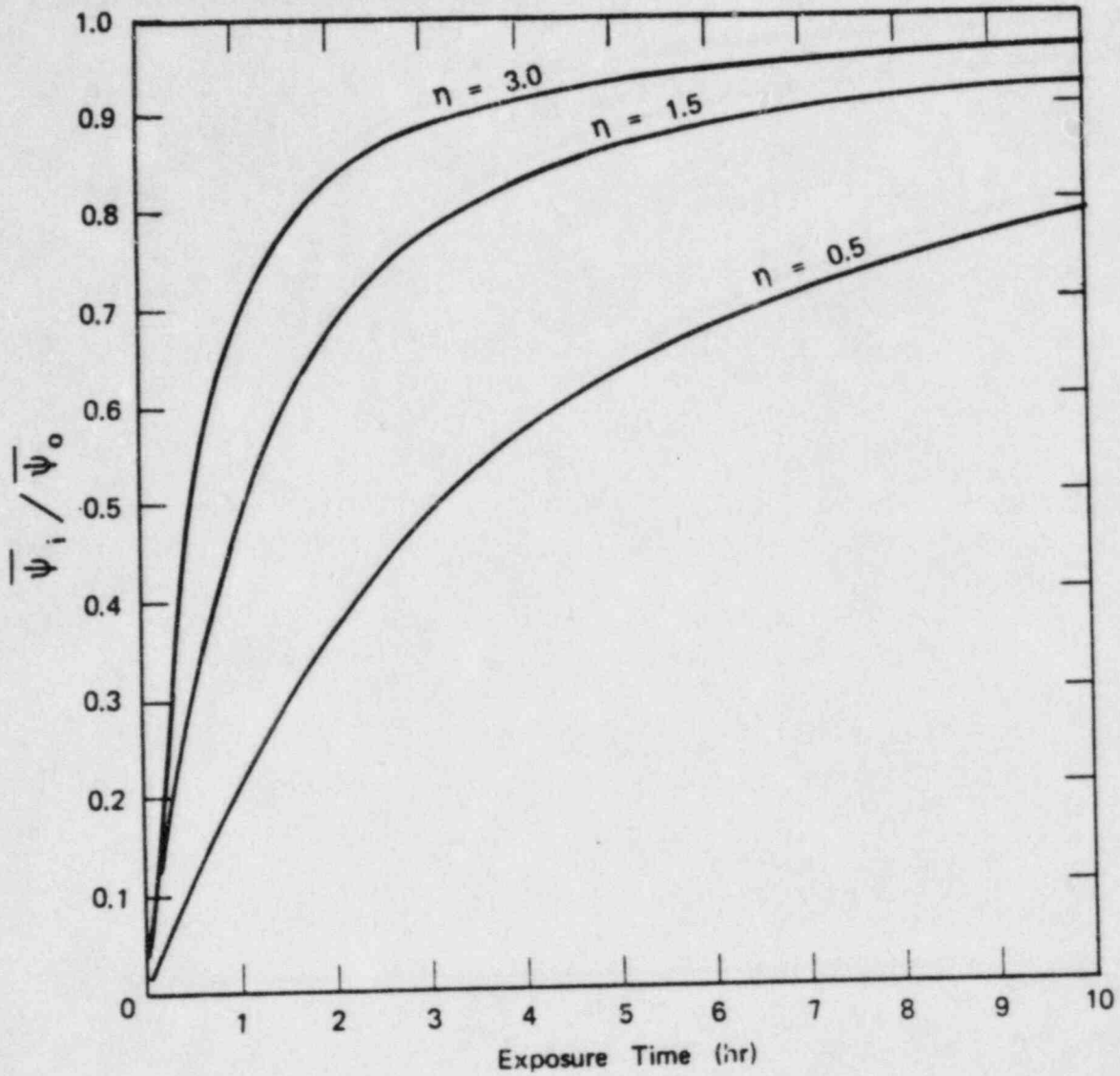

FIGURE VI 11-4 Ratio of the inhaled dose inside a shelter to that outside the shelter as a function of ventilation rate η.

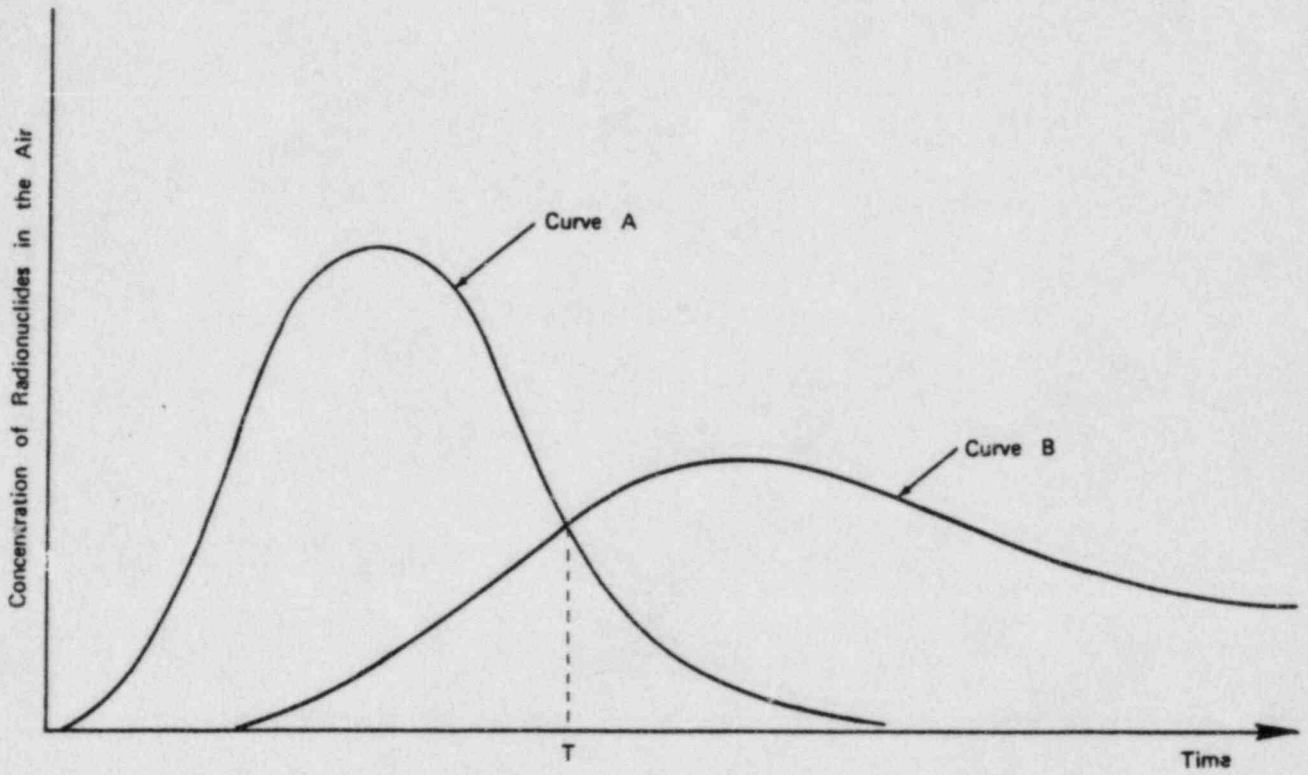

FIGURE VI 11-5 Concentration of radioactive material outdoors (curve A) and indoors (curve B) as a function of time during the cloud passage.

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
	3.0[a]
Skin, bone, thyroid	7.5
Hands and forearms; feet and ankles	1.5
Other single organs	

[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 dose 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 urlikely 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.

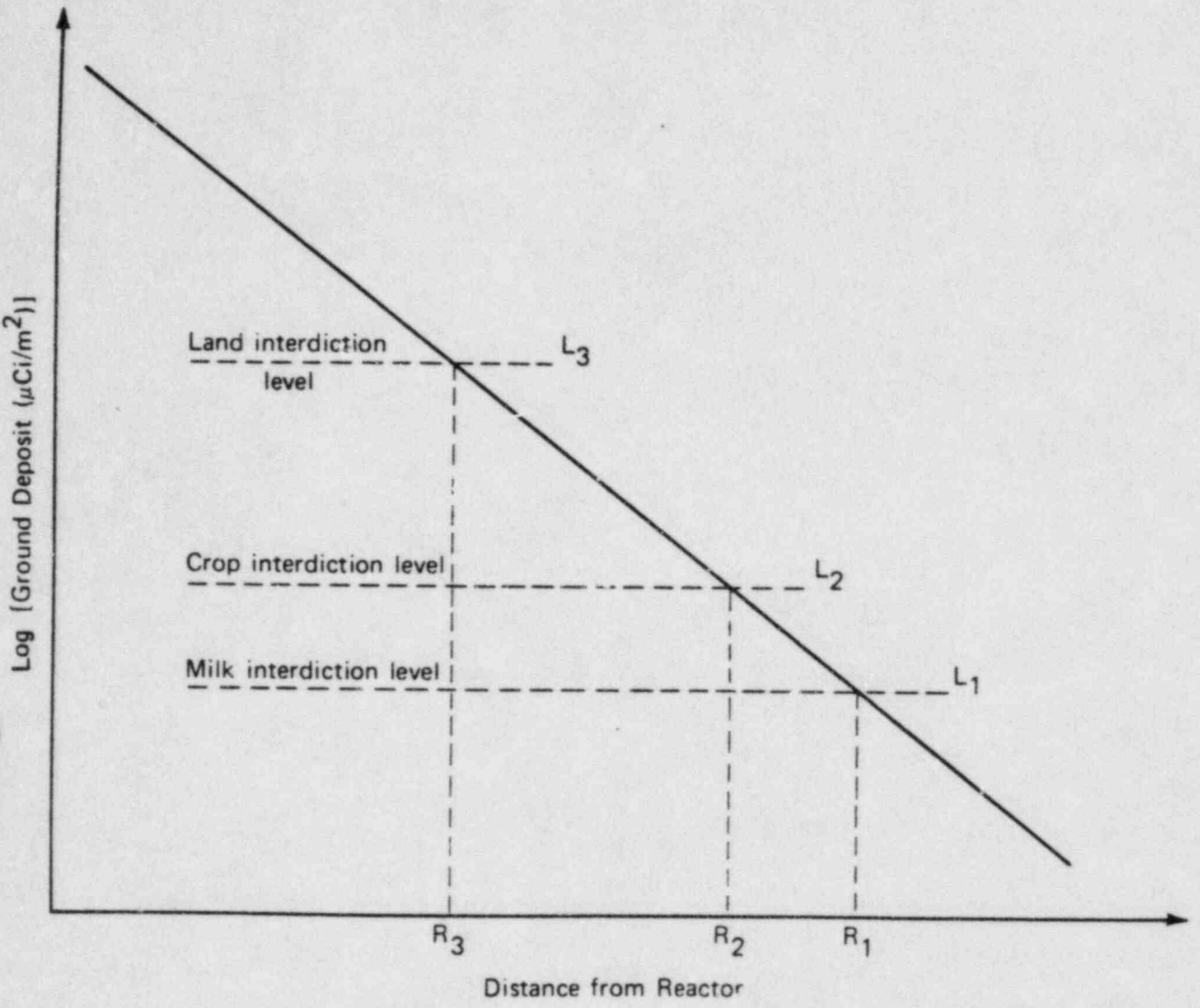

FIGURE VI 11-6 Illustrative interdiction model for ground level release.

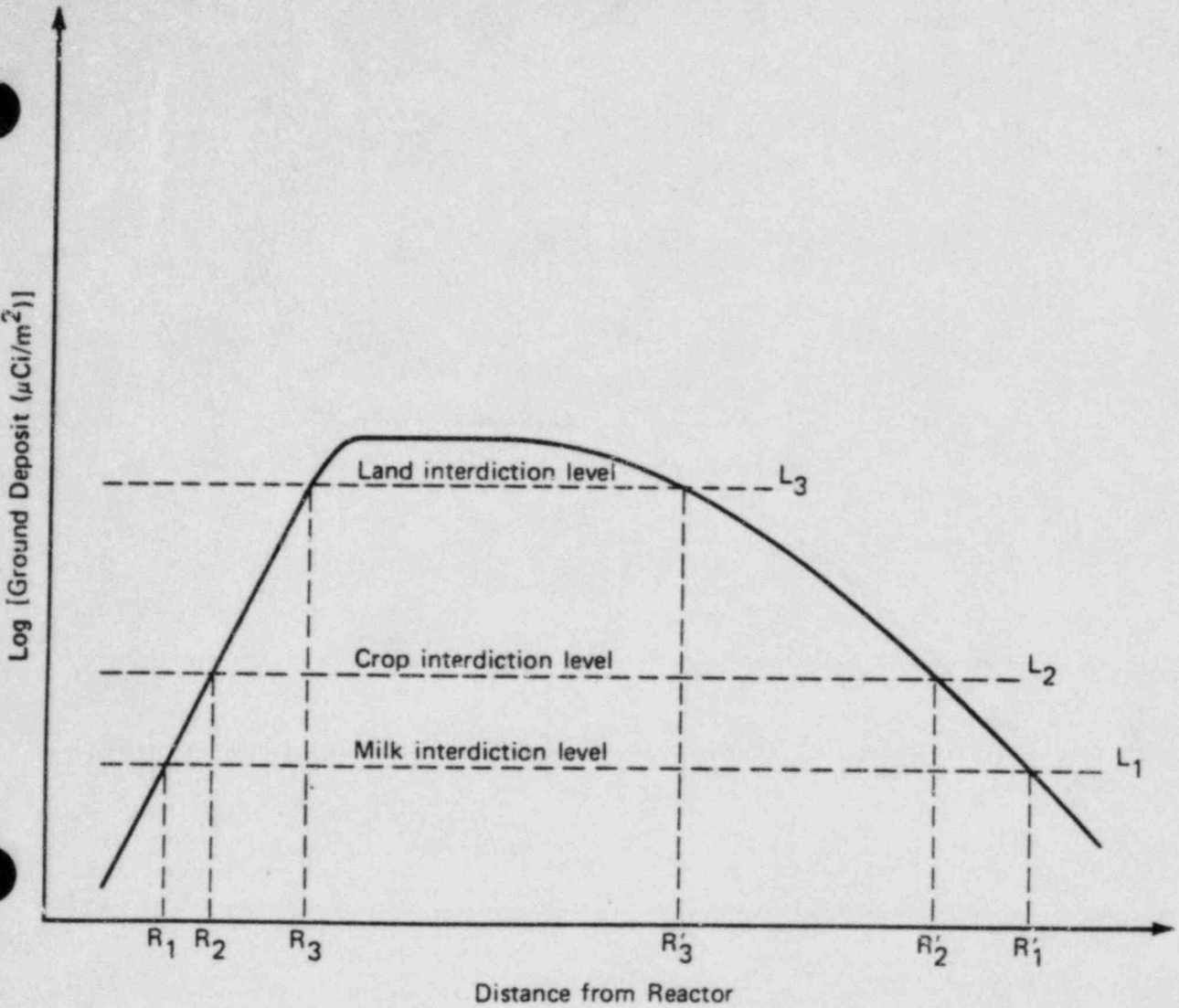

FIGURE VI 11-7 Illustrative interdiction model for elevated release.

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_4 in a matter of a few years, then the area between R_3 and R_4 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.

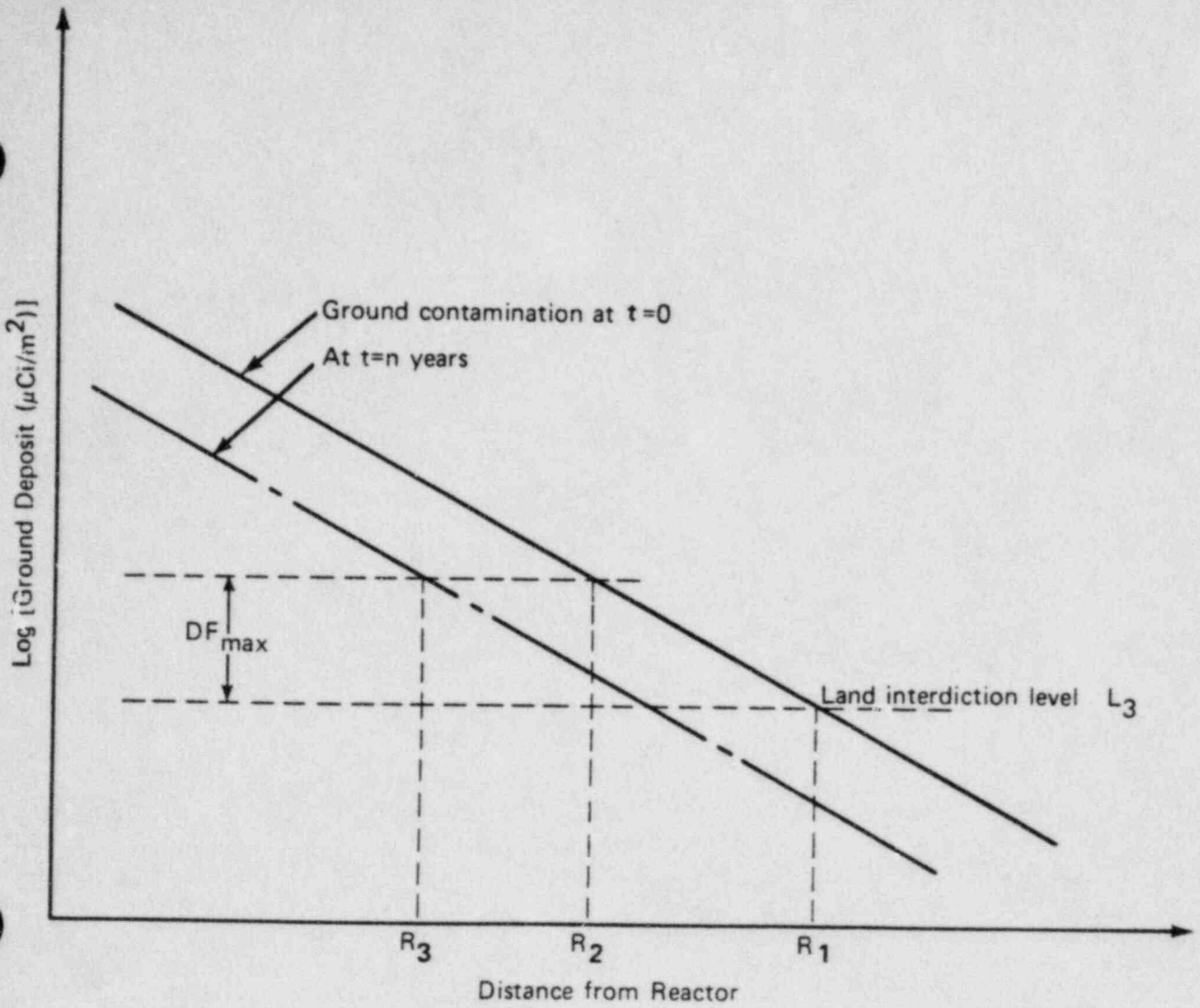

FIGURE VI 11-8 Illustrative decontamination model for ground level release.

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 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 (2[illegible]% 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.

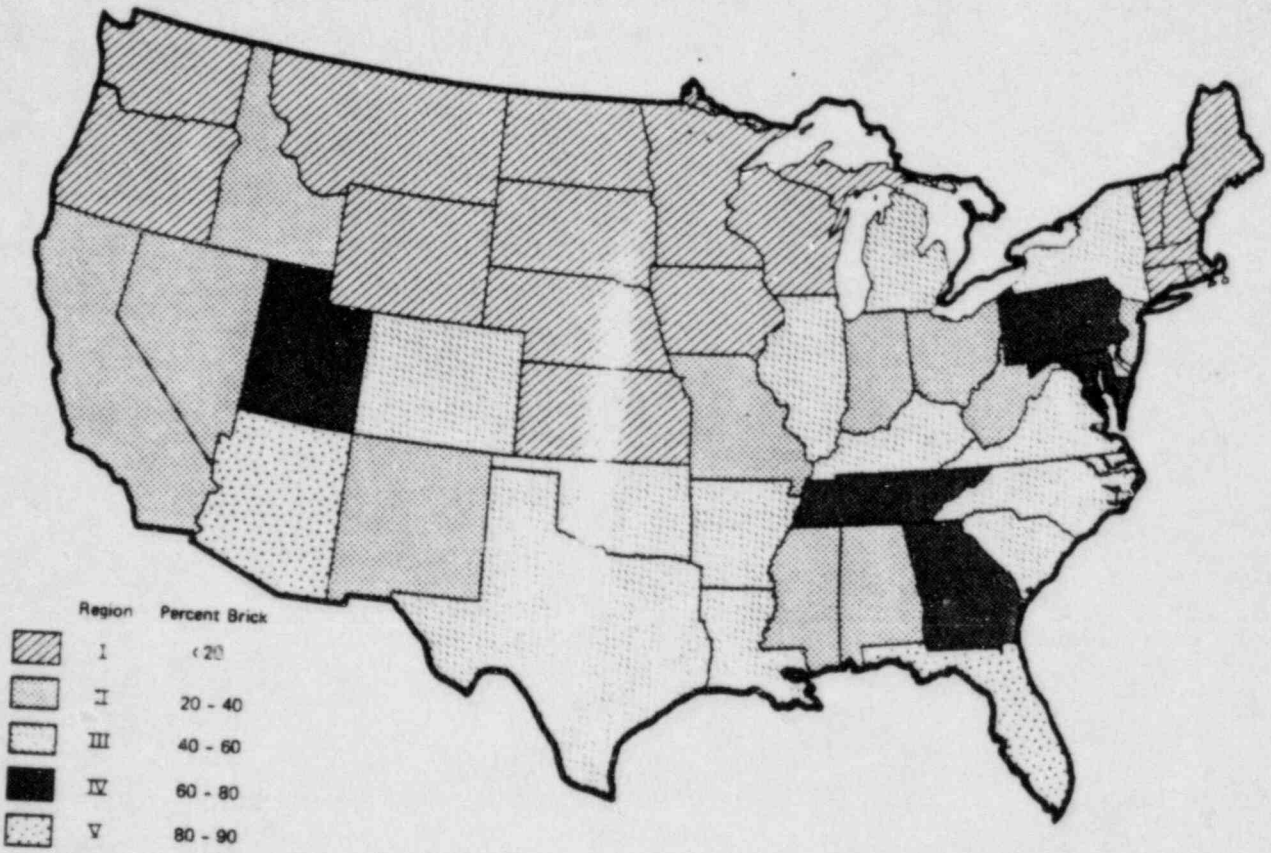

FIGURE VI 11-9 Percentage of brick housing units by region.

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

Region[a]	Frequency Distribution (%)						
	Home		School or Work			Commuting and Outdoors SF = 1.0	Average SF[b]
	Brick, SF = 0.6	Wood, SF = 0.9	Large Building SF = 0.2	Brick SF = 0.6	Wood, SF = 0.9		
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 (%)							
	Home		School or Work			Commuting, SF = 0.5	Outdoors, SF = 0.7	Average SF[b]
	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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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 offspring (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

Type of Disorder	BEIR Report			This Study					
	Normal Incidence per Million Live Births	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]			
		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.

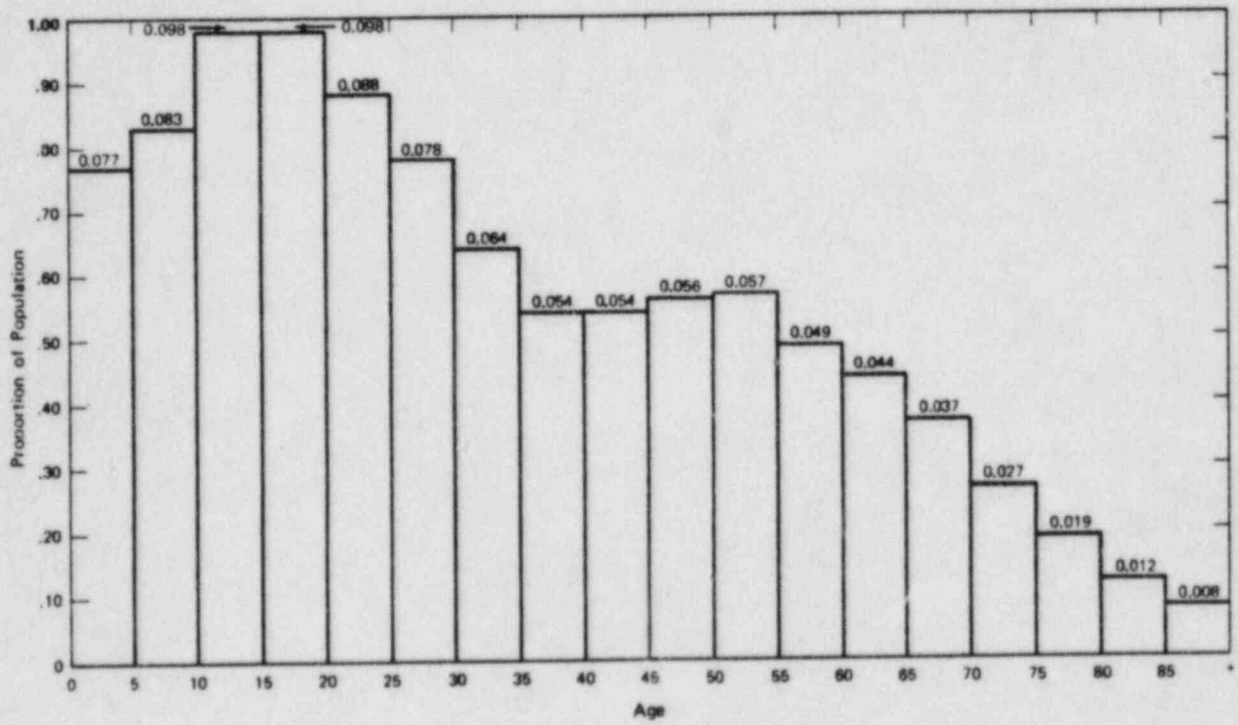

FIGURE VI I-1 Age distribution in U.S. population.

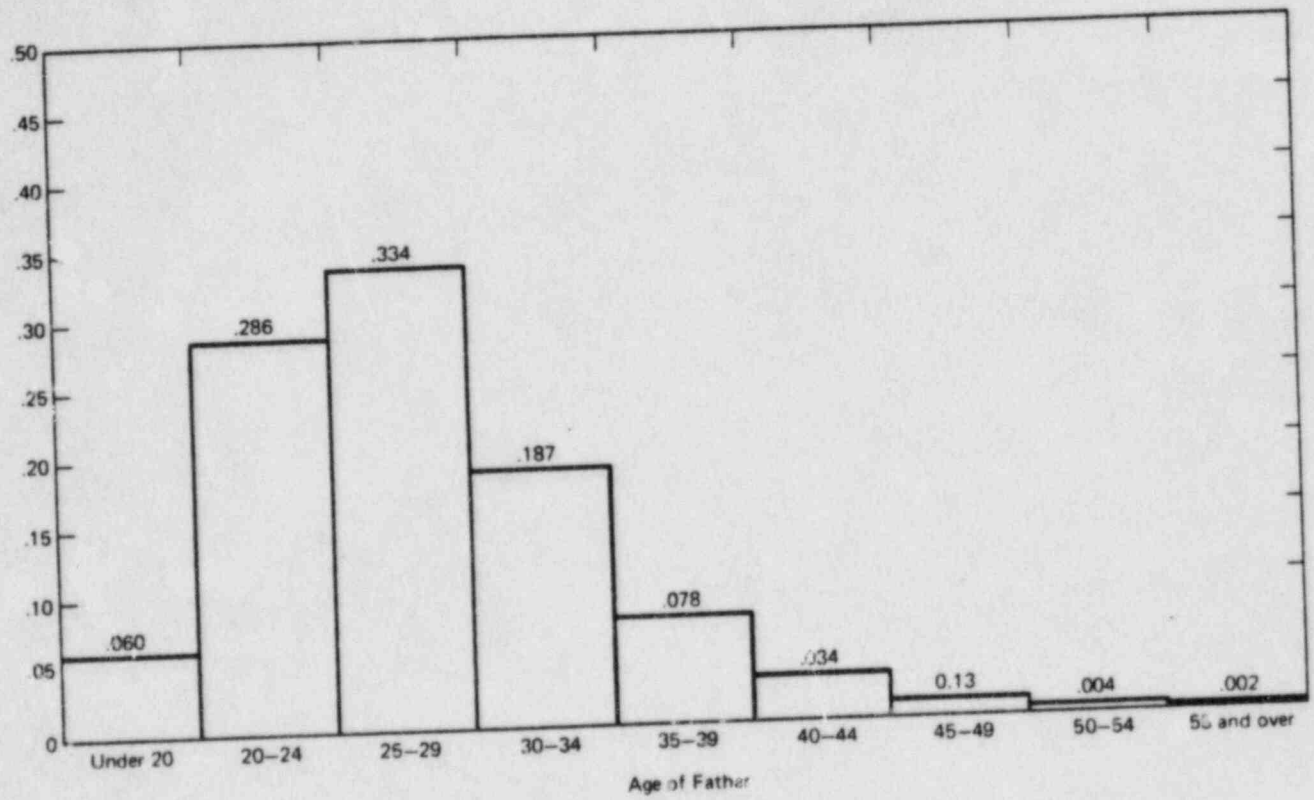

FIGURE VI I-2 Probability distribution of live births by age of father (1973 data).

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 $v = 0.283d^{0.91}$. 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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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	.2	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; 3/5[illegible]	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.

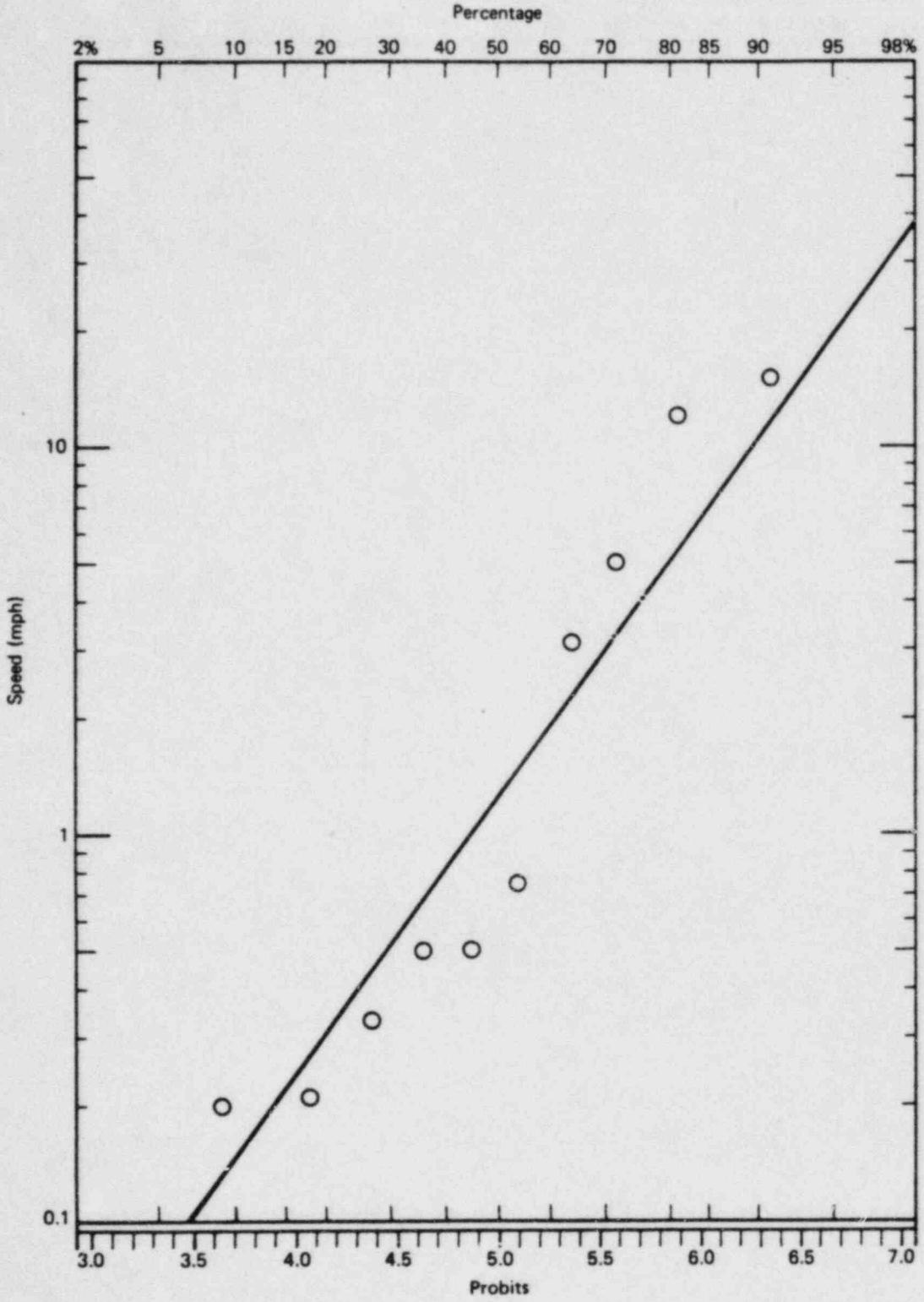

FIGURE VI J-1 Probability plot of transportation evacuation speeds.

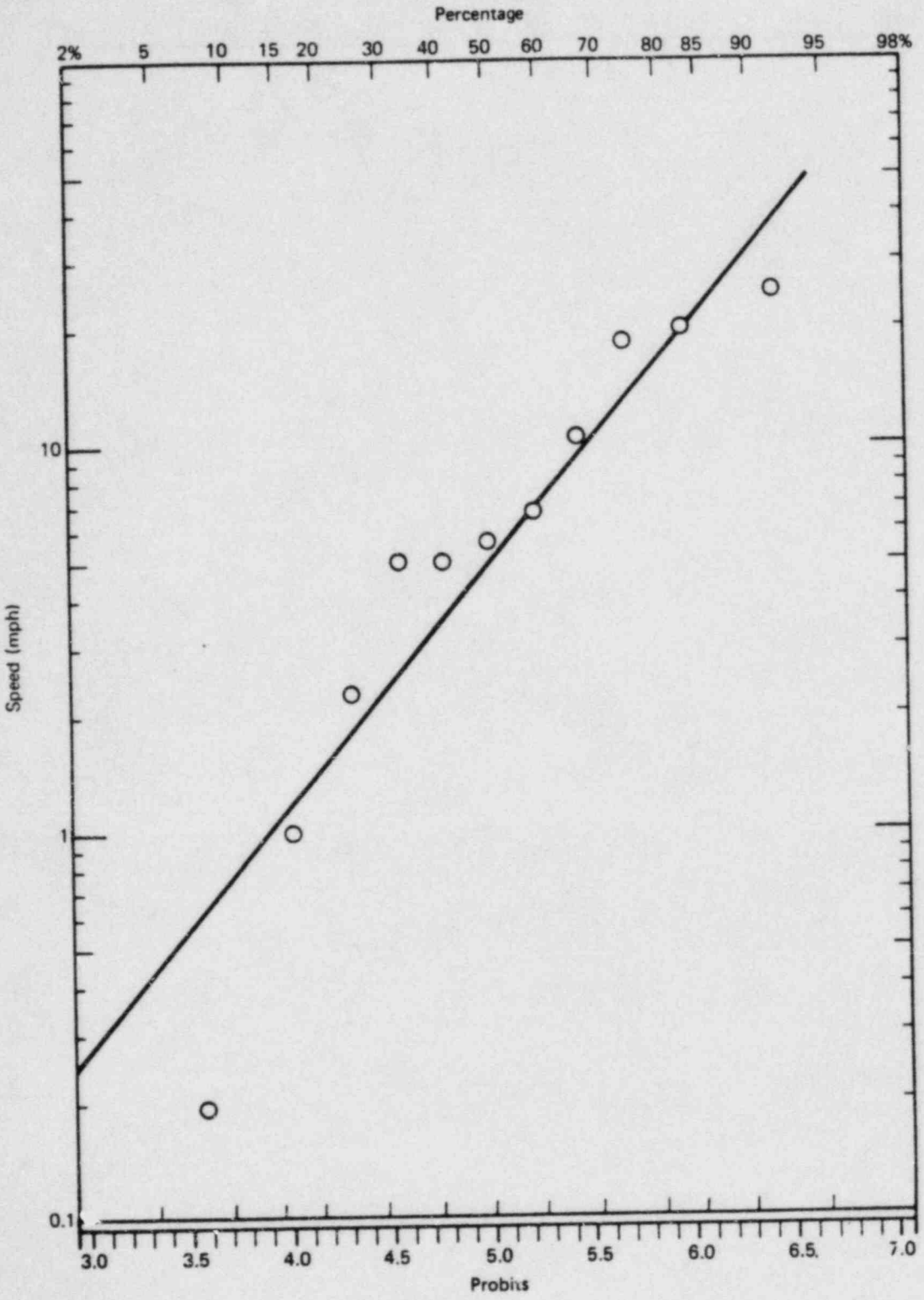

FIGURE VI J-2 Probability plot of hurricane evacuation speeds.

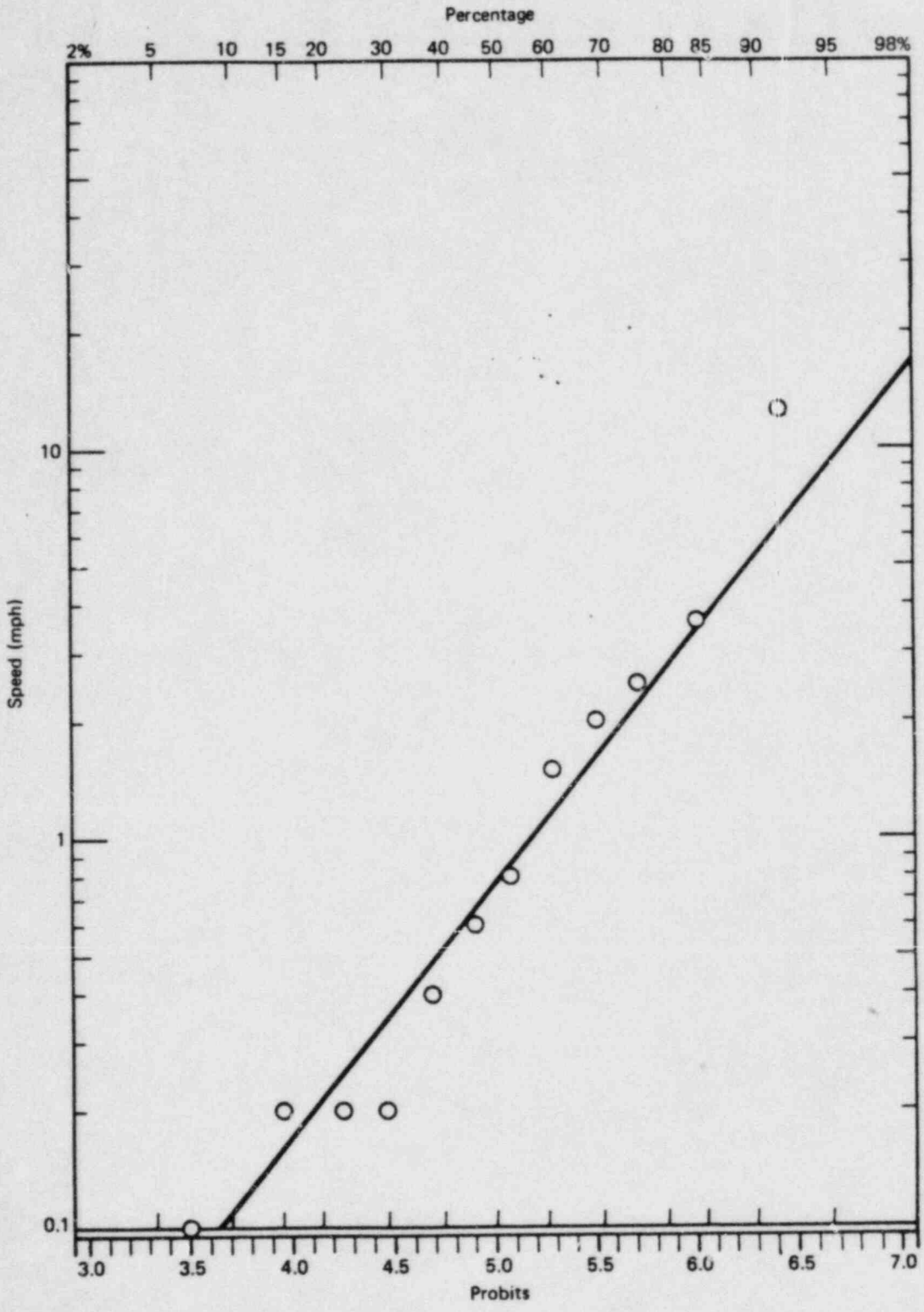

FIGURE VI J-3 Probability plot of flood evacuation speeds.

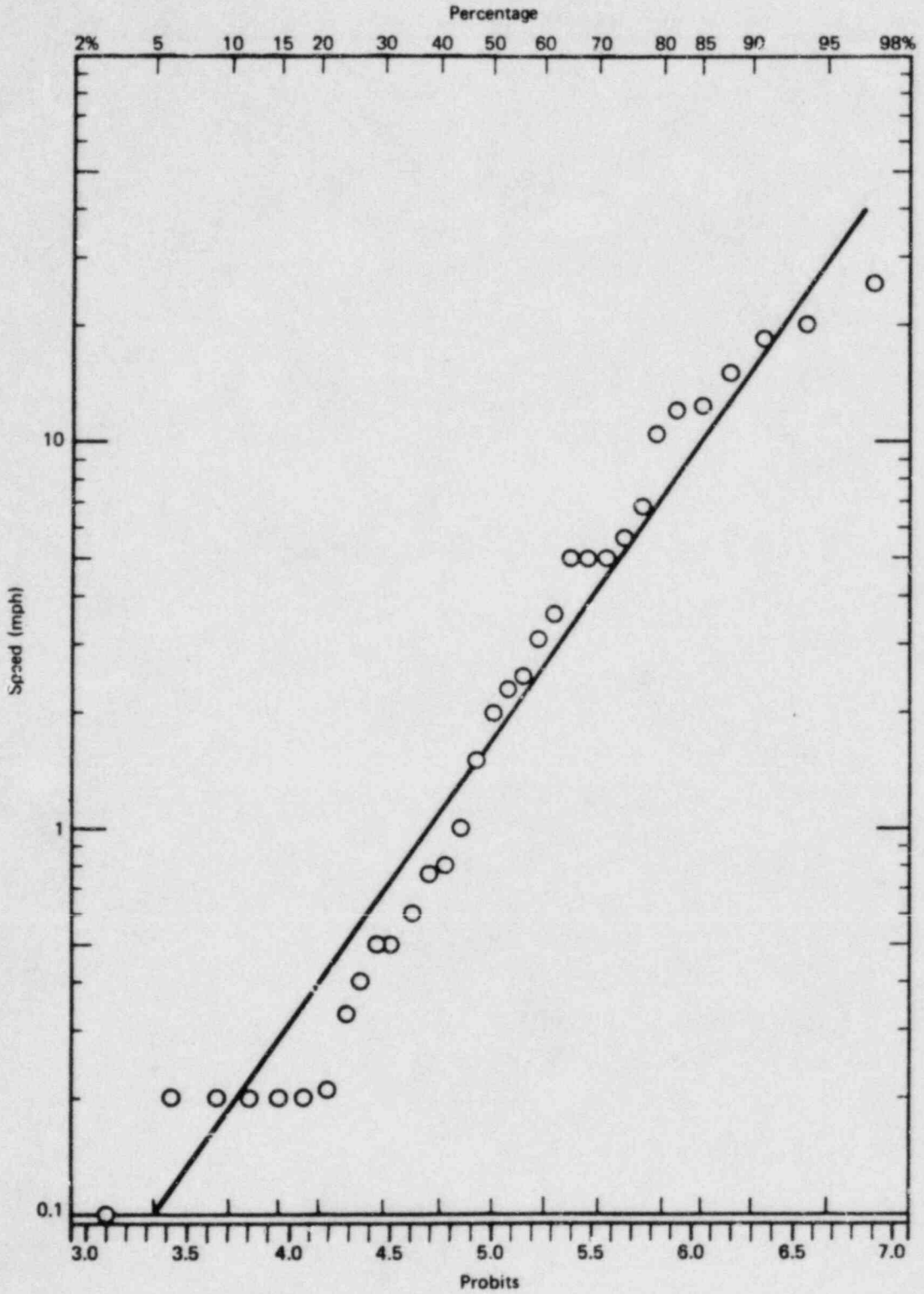

FIGURE VI J-4 Probability plot of general evacuation speeds.

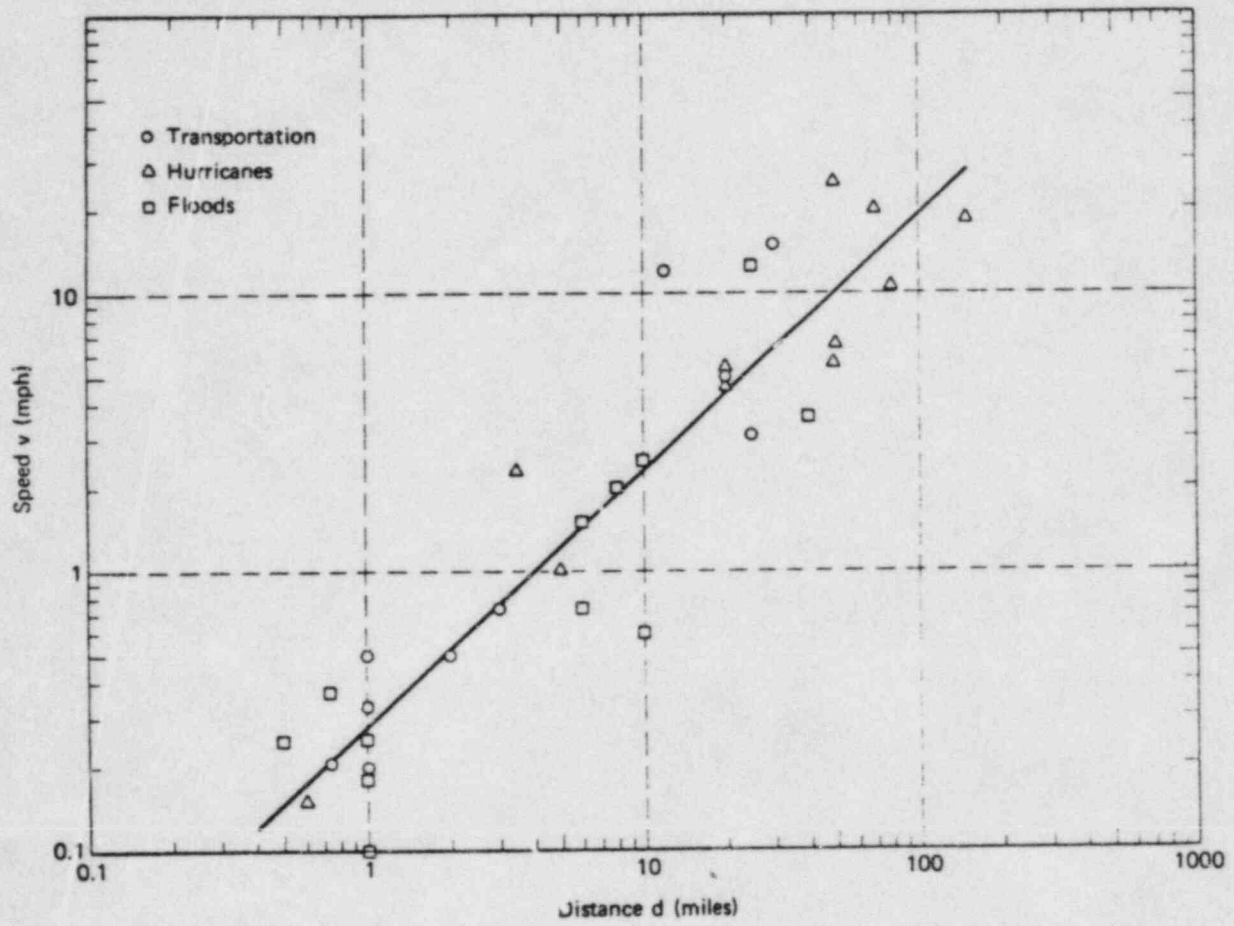

FIGURE VI J-5 Effective evacuation speed v versus distance of evacuation, d.

Best fit-line $v = 0.283d^{0.914}$