

APPENDIX I
HEALTH EFFECTS

This appendix describes the models used to estimate health risks to the public from exposures to chemical and radioactive waste materials following the implementation of each remedial alternative. The appendix divides the modeling methodology into its component parts and describes each to provide sufficient information for an understanding of the application of risk assessment to the remedial alternative selection process.

Risk assessment has three major components: (1) hazard assessment, consisting of hazard identification and dose-response assessment; (2) exposure assessment; and (3) risk characterization (King et al., 1987). These components are common to all assessments of the risk of exposure to hazardous substances, regardless of the substance under investigation, the species, the population or environmental systems at risk, the medium in which exposure occurs, the route of exposure, or the adverse effects under consideration.

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Hazard assessment involves the identification of substances of concern (i.e., as subjects of the risk assessment) and an initial determination of the intrinsic toxicity of these materials (dose-response assessment). Exposure assessment is the process of measuring or estimating the intensity, duration, and frequency of exposure to these pollutants, including the identification of routes of exposure and the determination of human receptors at risk; Appendix H describes this element of risk assessment. Risk characterization is the process of estimating the incidence of an adverse effect under the various conditions of exposure described in the exposure assessment; it involves combining the results of the exposure and hazard (dose-response) assessments.

I.1 HAZARD ASSESSMENT

I.1.1 HAZARD IDENTIFICATION

Hazard identification is the process of determining whether exposure to an agent can cause an increase in the incidence of a health condition (cancer, birth defects, etc.). According to the National Research Council, hazard identification involves characterizing the nature and strength of the evidence of causation (National Research Council, 1983). The Savannah River Plant (SRP) health risk analysis identified certain chemical and radioactive waste materials as hazardous on a site-by-site basis. An indepth evaluation of these materials, using transport modeling and risk calculations, forms the basis of the risk assessment.

The hazard evaluation process was divided into two parts. First, the available data - including soil characterization studies, groundwater analyses, influent records, and process chemical usage - were analyzed to determine what chemicals might have been disposed of at each site. Second, the concentration of each chemical was compared to a "selection criterion" listing. If the groundwater or soil concentration exceeded the selection criterion, the material was selected as a part of the transport modeling and risk calculation

TE | studies. In addition, if large amounts of specific chemicals were believed to have been released to the site (based on inventory or process usage), those materials were included for assessment, even if the soil or groundwater characterization data did not indicate their presence (Looney et al., 1987).

TE | Soil and groundwater concentration criteria for selection of radioactive and chemical wastes and sites for evaluation were based on toxicological and modeling information published by the U.S. Environmental Protection Agency (EPA). Additionally, the South Carolina Department of Health and Environmental Control (SCDHEC) regulations governing groundwaters of the State were considered in setting selection criteria (Looney et al., 1987).

TC | The selection of a radionuclide from an SRP site for environmental assessment and dose-risk calculations was based on detection of that radionuclide in soils or groundwater at levels that exceed the guideline activity concentrations listed in Table I-1 (Looney et al., 1987). These concentrations correspond to those that would be "below regulatory concern" (Guimond and Galpin, 1984) or "de minimis" (NRC, 1984); that is, they would produce a negligible increase in societal risk of adverse health effects (10^{-5} to 10^{-7} lifetime risk increment). The groundwater concentrations correspond to 0.5 times the EPA Interim Primary Drinking Water Standard of 4 millirem per year for beta-gamma emitters or 0.5×15 picocuries per liter for alpha-emitting radionuclides (EPA, 1976). The soil concentrations are derived by considering all soil-derived dose pathways, both external and internal, that would result in a dose to the maximally exposed individual that does not exceed 30 millirem per year. This value provides a margin of safety below the DOE standard of 100 millirem per year when combined with the annual exposures from the drinking-water and airborne pathways of 4 and 25 millirem, respectively.

Groundwater and soil criteria for selection of chemical waste constituents and sites for evaluation were also established. In determining whether a given nonradioactive compound present in groundwater at SRP waste sites was the subject of a risk or environmental assessment, measured levels in groundwater were compared with maximum contaminant limits (MCLs) or other health-based standards. If the observed levels exceeded 0.5 times the MCL (or, in the absence of the MCL, 1 times other relevant health criteria or guidelines), the compound was included in the assessment. This approach resulted in the assessment of a larger number of chemicals present in groundwater, and, therefore, was more conservative than a comparison made solely on the basis of EPA delisting guidelines (Looney et al., 1987).

TE | The approach for the selection of compounds for risk assessment based on soil contaminant concentrations was similar to that developed by EPA in the final rule on identification and listing of hazardous waste (EPA, 1985a). Using a 20-fold dilution factor based on EP toxicity testing procedures (EPA, 1984) and assuming a dilution factor of 10 to account for hydrodynamic dispersion in a saturated groundwater system (EPA, 1985a), Looney et al. (1987) developed the following soil constituent concentration criterion:

$$\text{Soil criterion } (\mu\text{g/g}) = \text{MCL } (\mu\text{g/L}) \times 10 \times 20 \frac{1}{1000\text{g/L}}$$

Table I-1. Selection Criteria for Radioactive Constituents^a

Constituent	Groundwater concentration guideline (pCi/L)	Soil concentration guideline (pCi/g)
Americium-241	8	2.6×10^1
Americium-243	8	7.9
Antimony-125	150	NA
Carbon-14	NA	4.9
Cesium-134	10,000	4.2
Cesium-135	NA	4.7×10^1
Cesium-137	450	1.1×10^1
Cobalt-60	50	2.9
Curium-243	8	1.9×10^1
Curium-244	8	6.0×10^1
Curium-246	8	7.6
Hydrogen (tritium)	10,000	2.7×10^4
Iodine-129	0.5	2.9
Iron-55	NA	4.1×10^2
Neptunium-237	NA	4.3×10^{-1}
Nickel-59	NA	2.6×10^2
Nickel-63	NA	1.1×10^4
Niobium-94	NA	2.7
Plutonium-238	8	3.3×10^1
Plutonium-239	8	3.3×10^1
Plutonium-240	8	3.3×10^1
Plutonium-241	NA	1.9×10^3
Plutonium-242	8	3.2×10^1
Sodium-22	200	NA
Strontium-90	4	3.4×10^1
Technetium-99	450	2.0×10^2
Uranium-233	NA	6.5×10^1
Uranium-235	NA	1.5×10^1
Uranium-238	NA	2.2×10^1

^aSource: Looney et al., 1987.

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where:

- MCL = the maximum contaminant level (or other health-based criteria of relevance in the absence of the MCL)
- 10 = dilution factor due to mixing in groundwater
- 20 = dilution factor due to leaching in the unsaturated zone

This criterion represents the level of a given constituent in soil that would result in a concentration equivalent to the MCL in water at a receptor well 152 meters downgradient, based on the VHS model used by EPA for screening purposes.

Table I-2 lists the groundwater and soil criteria developed for each nonradioactive waste constituent identified by sampling and analysis at the various sites. The hazard assessment component of the health risk assessment model was accomplished by the selection of nonradioactive constituents based on (1) exceeding concentration criteria, (2) exceeding the soil criteria, or (3) indicating that a particularly hazardous constituent was present in the site waste. In some cases, background concentration information and analytical protocol information were factored into the selection process.

I.1.2 DOSE-RESPONSE ASSESSMENT

TE | Health impacts associated with exposure to radionuclides usually are treated separately from impacts associated with nonradioactive materials (King et al., 1987). Similarly, risk characterization for carcinogens and noncarcinogens usually is considered separately. This is due to a fundamental difference in the way organisms typically respond to these classes of compounds. For noncarcinogens, toxicologists recognize the existence of a threshold of exposure below which there is only a small likelihood of adverse health effects in an exposed population. Exposure to carcinogenic compounds, however, is not characterized by the existence of a threshold. Rather, all levels of exposure are considered to carry a risk of adverse effect (risk per unit dose). Carcinogenic risks are associated with radionuclides and some nonradioactive materials.

I.1.2.1 Radiological Risks

Health impacts from radiation exposure, whether from sources external or internal to the body, generally are identified as "somatic" (affecting the individual exposed) or "genetic" (affecting descendants of the exposed individual). At low doses, the somatic risks of most importance are the induction of cancers; these risks are greater than genetic risks.

TC | For a uniform irradiation of the body, the incidence of cancer varies among organs and tissues; the thyroid and skin demonstrate a greater sensitivity than other organs. However, such cancers also produce relatively low mortality rates, because they are relatively amenable to medical treatment. A consideration of somatic risks must distinguish between cancer incidence and cancer mortality rates; the evaluation described in this section uses projections for the latter.

Increased cancer incidence has been observed in humans only after exposures to radiation at doses and dose rates that are at least several orders of magnitude greater than those of interest in this assessment. Thus, risks are estimated for effects at low doses and dose rates by extrapolation downward from risks observed to occur at high doses and dose rates. The factors involved in such extrapolations can produce risk estimates that vary by factors as great as about 4.

Table I-2. Selection Criteria for Nonradioactive Constituents^a

Constituent	Groundwater concentration guideline (µg/L) ^b	Soil concentration guideline (µg/g) ^c	TE	
Aluminum	NS ^d	NS	TE	
Arsenic	25	10		
Barium	500	200		
Beryllium	NS	NS		
Cadmium	5	2		
Chloride	NS	NS		
Chromium	25	25		
Copper	1,000	200		
Cyanide	100	40		
Fluoride	2,000	800		
Iron	NS	NS		
Lead	25	10		
Mercury	1	0.4		
Manganese	NS	NS		
Nickel	175	70		
Nitrate (as N)	5,000	2,000		
Phosphate (as P)	10	150		
Selenium	5	2		
Silver	25	10		
Sodium	10,000	4,000		
Sulfate	400,000	80,000		
Zinc	5,000	1,000		
Endrin	0.1	0.04		TE
Lindane	2	0.8		
Methoxychlor	50	20		
Silvex	5	2		
Toxaphene	2.5	1		
2-4,D	50	20		
Trichloroethylene	2.5	1		
Carbon tetrachloride	2.5	1		
Vinyl chloride	0.5	0.2		
1,2-dichloroethane	2.5	1		
Benzene	2.5	1		
1,1-dichloroethylene	3.5	1.4		
1,1,1-trichloroethane	100	40		
p-dichlorobenzene	375	150		
Formaldehyde	15	3		
Dichloromethane	60	12		
Chlorobenzene	1,000	200		
Chloroform	0.5	0.1		
Ethyl benzene	3,500	700		

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Table I-2. Selection Criteria for Nonradioactive Constituents^a
(continued)

C-14	Constituent	Groundwater concentration guideline (µg/L) ^b	Soil concentration guideline (µg/g) ^c
	Tetrachloroethylene	0.7	0.14
	Toluene	10,000	2,000
	1,1,2-trichloroethane	0.6	0.12
	Di-n-butyl-phthalate	44,000	8,800
	Bis(2-ethylhexyl)phthalate	20,000	4,000
	Diethyl-phthalate	500,000	100,000
	Methyl ethyl ketone	2,000	400
	Trichlorofluoromethane	10,000	2,000
	1,2-dichloroethylene	350	70
	Phenol	3,500	700
	Dichlorobenzenes	3,000	600
	Trifluorotrichloroethane	955	191
	Fluoroanthene	5	1
	Naphthalene	5	1
	Xylene	NS ^d	7
	Tetrachlorobiphenyl	NS	1
	Pentachlorobiphenyl	NS	1
TC	Hexachlorobiphenyl	NS	1
	TOH (total organic halogen)	10	NS

^aSource: Looney et al., 1987.

^bGroundwater concentration guidelines are 0.5 x EPA Primary Drinking Water Standards. National Secondary Drinking Water Regulations are generally not listed because they are based on aesthetic characteristics rather than a quantitative effect on human health. However, 1 x secondary standards are used for sulfate, zinc, and sodium based on sensitive subpopulations. Copper and phosphate groundwater concentrations are included based on ecological considerations.

TC ^cSoil concentration criteria are based on EPA guidance (EPA, 1985a). Values are based on the following assumptions: (1) all of the constituents present in the soil will leach into water, (2) the ratio of soil to water is 1:20, as specified in the EPA EP Toxicity Leach Test, and (3) calculation using the EPA VHS model can be used to determine the concentration at a receptor 152 meters from the site. A dilution factor of 10 at the receptor well was chosen (actual VHS model runs resulted in a dilution range of 8 to 30). Thus, soil concentration guidelines were conservatively chosen using the formula Soil concentration (ppm) = DWS (ppb) x 10 x [20/1000].

^dNS = No standard.

One such factor involves the nature of the cancer induction risk; that is, whether the excess cancers observed to occur in a defined exposed population are best represented by either a defined fractional increase in the natural cancer incidence or mortality rates per unit dose (a "relative risk" estimate), or by a defined number of excess cancers per unit dose (an "absolute risk" estimate).

Another factor involves the nature of the relationship between (or the shape of the curve relating) dose and effect in the dose region below that for which data exist. The National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation (BEIR, 1980) examined three dose-effect relationships:

- Linear - effects proportional to dose at all dose values greater than zero
- Linear-Quadratic - effects essentially proportional to dose at very low doses and to the square of the dose at higher doses
- Quadratic - effects increase as the square of the dose at all dose levels

A majority of the BEIR Committee felt that the linear-quadratic relationship provides the most probable representation of the true dose-effect relationship, because it is similar in form to observed biological system responses in studies of other effects. The committee accepted the linear (nonthreshold) dose-effect relationship as an upper-limit, conservative basis for extrapolation of observed effects to low doses.

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The BEIR study provided estimates of excess cancer deaths per million person-rem of low LET (beta-gamma) radiation from 67 to 226, depending on the dose response and risk function assumed. The linear-response, absolute risk-model estimate is 158 cancer deaths per million person-rem. The International Commission on Radiological Protection (ICRP, 1977) postulated about 125 fatal individual organ risks per million person-rem; however, ICRP rounded the overall fatal cancer risk factor to 100 per million person-rem. The United Nations Scientific Committee on Effects of Atomic Radiation (UNSCEAR, 1977) also presented a value of 100 fatal malignancies per million person-rem.

In contrast to the somatic risk that occurs in an exposed individual, genetic risks are expressed for the descendants of the exposed individual, potentially for several generations. These risks, which might or might not result in death, have been estimated primarily from the results of animal studies. The BEIR Committee estimated a risk of 5 to 65 disorders per million liveborn offspring per rem of preconceptual parental exposure (i.e., over a 30-year "generation") in addition to the present incidence rate of about 107,000 cases of such disorders per million live births (BEIR, 1980). If the parental exposure were to continue in each generation, the ultimate increase in such disorders would be in the range of 60 to 1100 per million liveborn offspring.

In its 1982 report, UNSCEAR reduced its genetic risk estimates to 20 first-generation and 150 total serious hereditary disorders per million

liveborn children per rem of parental exposure (over 30 years) (UNSCEAR, 1982). The corresponding total genetic risk proposed by ICRP (1977) is about three times that expressed in the first two generations (4×10^{-5} per rem), or about 1.2×10^{-4} per rem.

TC This evaluation assumes that a linear (nonthreshold) absolute risk model applies to the radiological risks. Further, to permit a simplified presentation of radiological risk estimates in this EIS, the evaluation considers such risks to include both those from cancer in the exposed individual and those from serious genetic disorders in that individual's descendants, as described above. These risks range from 1.65×10^{-4} to 2.8×10^{-4} fatal effects per person-rem of collective dose. This analysis uses the upper limit of this range to estimate radiological risks; the upper limit includes all fatal stochastic (probabilistic) somatic and genetic effects.

I.1.2.2 Nonradioactive Carcinogenic Risks

TE The procedure for calculating risk of exposure to carcinogenic compounds used in the SRP risk assessment is well documented (National Research Council, 1983; EPA, 1983; Roderick, 1984; King et al., 1987). A nonthreshold dose-response model was used to calculate a unit risk value (risk per unit dose) for each chemical; Table I-3 lists unit cancer risks (UCRs) for a select list of SRP waste constituents. The risk per unit dose (UCR) was multiplied by the estimated average daily lifetime dose experienced by the exposed population, to derive an estimate of risk as follows:

$$R = D \times \text{UCR}$$

where:

D = average daily lifetime dose (milligrams per kilogram of body weight per day)

UCR = unit cancer risk estimate [(milligrams per kilogram of body weight per day)⁻¹]

R is an explicit estimate of risk and will have a value between 0 and 1. In evaluating the risk of exposure to more than one carcinogen, the risk values (R) for each compound were summed to give an overall estimate of total carcinogenic risk (EPA, 1983; Roderick, 1984). This was done for each source of environmental release, for each associated pathway, and for each receptor group at risk of exposure.

I.1.2.3 Nonradioactive Noncarcinogenic Risks

The traditionally accepted practice of evaluating exposure to noncarcinogenic compounds has been to determine experimentally a no-observable-effect level (NOEL) and to divide this by a "safety factor" to establish an acceptable human dose [e.g., acceptable daily intake or ADI (National Research Council, 1983)]. Table I-4 lists values of ADIs used in this analysis. The ADI was

Table I-3. Toxicity Data for Potential Carcinogenic Effects^a

Chemical	Ingestion (mg/kg/day) ⁻¹	Inhalation (mg/kg/day) ⁻¹
Arsenic and compounds	1.50 x 10 ¹	5.00 x 10 ¹
Beryllium and compounds	-	2.60
Cadmium and compounds	-	7.8
Chromium VI and compounds	-	4.1 x 10 ¹
Nickel and compounds	-	1.20
Aldrin	1.10 x 10 ¹	
Benzene	4.45 x 10 ⁻²	2.60 x 10 ⁻²
Carbon tetrachloride	1.3 x 10 ⁻¹	
Chloroform	7.00 x 10 ⁻²	
1,2-dichloroethane	6.90 x 10 ⁻²	
1,1-dichloroethylene		1.50 x 10 ⁻¹
Dichloromethane (methylene chloride)		6.30 x 10 ⁻⁴
Lindane	1.33	
Polychlorinated biphenyls	4.34	
Polynuclear aromatic hydrocarbons	1.15 x 10 ¹	6.10
2,3,7,8 TCDD (dioxin)	1.56 x 10 ⁵	
1,1,2,2-tetrachloro- ethane	2.00 x 10 ⁻¹	
Tetrachloroethylene	5.10 x 10 ⁻²	1.70 x 10 ⁻³
Toxaphene	1.10	

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Table I-3. Toxicity Data for Potential Carcinogenic Effects^a (continued)

Chemical	Ingestion (mg/kg/day) ⁻¹	Inhalation (mg/kg/day) ⁻¹
1,1,1-trichloroethane	1.6 x 10 ⁻³	
1,1,2-trichloroethane	5.73 x 10 ⁻²	
Trichloroethylene	1.10 x 10 ⁻²	4.60 x 10 ⁻³
Vinyl chloride	2.30	2.50 x 10 ⁻²

TE | ^aSource: King et al., 1987.

compared to the average daily dose experienced by the exposed population to obtain a measure of risks as follows:

$$R = D/ADI$$

where:

D = average daily lifetime dose (milligrams per kilogram of body weight per day)

ADI = acceptable daily intake for chronic exposure (milligrams per kilogram of body weight per day)

The method of developing acceptable limits of exposure implies that the application of safety factors of various magnitudes to an experimentally derived NOEL will ensure minimal risk. The acceptable exposure levels (e.g., ADIs) typically are derived by making assumptions about the nature of dose-response relationships at low doses and by drawing inferences based on the available data (National Research Council, 1983).

The risk values derived for noncarcinogens will vary from less than 1 to more than 1. The smaller the value of R, the larger the margin of safety (MOS). The smaller the MOS, the larger the risk.

TE | The data base (King et al., 1987) for UCRs and ADIs for inhalation and ingestion pathways was derived from the EPA Superfund Public Health Evaluation Manual (EPA, 1983), which was designed to conform to EPA's proposed risk assessment guidelines (49 FR 46294-46331; 50 FR 1170-1176) and to serve as a framework for analyzing public health risks and for developing design goals for remedial alternatives.

I.1.2.4 Occupational Risks

Occupational risks due to workers' exposures to radioactive constituents were estimated with the use of the methodology outlined in Section I.1.2.1 for assessing public risk. The occupational risks are based on the assumption that the average worker is exposed for 40 hours per week for the period of

Table I-4. Toxicity Data for Noncarcinogenic Effects^a

Chemical	Ingestion (mg/kg/day)	Inhalation (mg/kg/day)
<u>INORGANIC</u>		
Arsenic and compounds	0.00	2.80×10^{-3}
Barium and compounds	5.10×10^{-2}	1.40×10^{-4}
Cadmium and compounds	2.90×10^{-4}	
Chromium III and compounds	1.50	5.10×10^{-3}
Chromium VI and compounds	5.00×10^{-3}	
Copper and compounds	3.70×10^{-2}	1.00×10^{-2}
Iron and compounds		8.60×10^{-3}
Lead and compounds	1.40×10^{-3}	4.30×10^{-4}
Manganese and compounds	2.20×10^{-1}	3.00×10^{-4}
Mercury and compounds (alkyl)	2.80×10^{-4}	1.00×10^{-4}
Mercury and compounds (inorganic)	2.00×10^{-3}	5.10×10^{-5}
Nickel and compounds	1.00×10^{-1}	1.20
Phosphoric acid (H ₃ PO ₄)	5.10×10^{-3}	5.10×10^{-3}
Selenium and compounds	3.00×10^{-3}	1.00×10^{-3}
Silver	3.00×10^{-3}	
Sodium	5.70×10^{-1}	
Sulfuric acid (H ₂ SO ₄)	5.10×10^{-3}	5.10×10^{-3}
Zinc and compounds	2.10×10^{-1}	1.00×10^{-2}
Chloride	3.00×10^{-1}	

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Table I-4. Toxicity Data for Noncarcinogenic Effects^a (continued)

Chemical	Ingestion (mg/kg/day)	Inhalation (mg/kg/day)
<u>INORGANIC</u> (continued)		
Cyanides	2.00×10^{-2}	
Fluorides	5.00×10^{-2}	
Nitrate	2.86×10^{-1}	
Phosphate	3.0×10^{-1}	
Sulfate	3.5×10^{-1}	
<u>ORGANIC</u>		
Bis-2ethylhexyl phthalate	6.00×10^{-1}	
Carbon tetrachloride	7.00×10^{-4}	
Chlorobenzene	2.70×10^{-2}	5.70×10^{-3}
Dibutyl phosphate	2.55×10^{-2}	2.55×10^{-2}
1,2-dichlorobenzene	9.00×10^{-2}	
1,1-dichloroethane	1.20×10^{-1}	1.40×10^{-1}
trans-1,2- dichloroethylene	4.03	4.03
Dichloromethane (methylene chloride)	5.00×10^{-2}	
2,4-dichlorophenoxy- acetic acid (2,4D)	1.26×10^{-1}	
n-Dodecane	7.40	7.40
Endrin	1.00×10^{-3}	
Ethylbenzene	9.70×10^{-2}	
Freon	2.86×10^1	2.86×10^1

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Table I-4. Toxicity Data for Noncarcinogenic Effects^a (continued)

Chemical	Ingestion (mg/kg/day)	Inhalation (mg/kg/day)
<u>ORGANIC</u> (continued)		
Lindane	3.00×10^{-4}	
Methoxychlor	5.00×10^{-2}	
Methyl ethyl ketone	4.60×10^{-2}	
Naphthalene	2.60×10^{-1}	
Phenol	1.00×10^{-1}	2.00×10^{-2}
Silvex	9.00×10^{-3}	
Sym-trimethylbenzene	6.38×10^{-1}	6.38×10^{-1}
Tetrachloroethylene	2.00×10^{-2}	
Toluene	2.90×10^{-1}	
Tributyl phosphate	1.28×10^{-2}	1.28×10^{-2}
1,1,1-trichloroethane	5.40×10^{-1}	6.30
Xylene (mixed)	1.00×10^{-2}	4.00×10^{-1}

^aSource: King et al., 1987.

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cleanup. If a worker were exposed to the DOE annual occupational dose limit of 5 rem to the whole body, the increased risk to that worker would be 1.4×10^{-3} health effect. Occupational risks due to worker exposures to nonradioactive carcinogenic and noncarcinogenic constituents were estimated with the use of the methodologies outlined in Sections I.1.2.2 and I.1.2.3, respectively, for assessing public risk, with the following exceptions. The occupational risks are based only on worker exposure via the inhalation pathway and, assuming the average individual works at the site for 8 hours each day, for the period of cleanup.

I.2. RISK CHARACTERIZATION

I.2.1 GENERAL APPROACH

Risk characterization is the process of estimating the incidence of a health effect under the various conditions of human exposure described in the exposure assessment (National Research Council, 1983). It essentially combines the exposure and dose-response assessments.

Risks associated with exposure to radionuclides and nonradioactive carcinogenic waste materials are characterized as the probability of a health effect occurring in an exposed individual or the number of health effects in a population group.

The individual risks take on values ranging from 0 to 1. For example, a 10^{-6} cancer risk indicates that an individual incurs a one-in-a-million additional chance (i.e., above the normal likelihood) of cancer due to exposure to the waste material. In this analysis, cancer risk estimates have been added when concurrent exposure to more than one carcinogen occurs. For example, concurrent exposure to two waste constituents, each posing a 10^{-6} cancer risk, is assumed to yield an overall 2×10^{-6} additional cancer risk (i.e., two chances in a million, or one in 500,000) beyond the normal likelihood of cancer.

Risk characterization for exposure to noncarcinogens is estimated from the fraction of the ADI represented by the estimated dose. A fractional ADI less than 1.0 indicates that the estimated exposure dose is less than that recognized as constituting a health hazard. Consequently, some MOS exists at the estimated dosage if the fraction of ADI is less than 1. Under this system, the smaller the MOS, the larger the risk. For example, if the fraction of ADI is 0.1 for one contaminant, and 0.01 for another, the latter (0.01) has a larger associated MOS than the former (0.1) and, hence, a lower attendant risk of the associated health effect.

ADI fractions can be added when concurrent exposure to more than one noncarcinogen occurs to provide a means of evaluating the MOS resulting from exposure to a mixture of contaminants. In such cases, the Hazard Index (HI) (EPA, 1985b) of the mixture based on the assumption of dose additivity is defined as

$$HI = E_1/AL_1 + E_2/AL_2 + \dots + E_i/AL_i$$

where:

E_i = exposure level to the i^{th} toxicant

AL_i = maximum acceptable level for the i^{th} toxicant

Because the inverse of the acceptable level can be used as an estimate of toxic potency, the equation can be interpreted as a normalized weighted-average dose, with each component dose scaled by its potency. As this index approaches unity, concern for the potential hazard of the mixture increases. If HI is greater than 1, the concern for the potential hazard is the same as if an acceptable level were exceeded for an individual compound (i.e., if E_i/AL_i exceeded 1). If the variabilities of the acceptable levels are known, or if the acceptable levels are given as ranges (e.g., associated with different margins of safety), then HI should be presented with estimates of variation or as a range (EPA, 1985b).

The Hazard Index is not a mathematical prediction of incidence of effects or severity. Statistical properties of this index and its dependence on the shape of the dose-response curves for the components are not known. Much additional research is required to determine the accuracy of the Hazard Index

as a numerical prediction of toxic severity. The Hazard Index is only a numerical indicator of the transition between acceptable and unacceptable exposure levels and should not be overinterpreted (EPA, 1985b).

I.2.2 WASTE SITE RISK CHARACTERIZATION

To characterize the risks associated with potential exposure to hazardous materials at any SRP waste site, the dosages, as determined in the exposure assessment step, were evaluated in terms of their attendant carcinogenic and noncarcinogenic risks. Radioactive and nonradioactive carcinogenic risks were evaluated separately for the mixed waste sites.

Carcinogenic and noncarcinogenic risks were calculated for all exposure scenarios (subsurface and atmospheric) over the 1000-year time period for each remediation option. Risks were displayed in tabular or graphic format over appropriate time intervals, usually 100 years. Maximum risks and time of occurrence were also calculated and displayed. Additionally, summary estimates of risks for all exposure routes were computed by summing the carcinogenic risk estimates and ADI fractions. These risks are presented in Chapter 4 of this statement for each of the sites and remediation alternatives evaluated.

The methods for evaluating and characterizing carcinogenic and noncarcinogenic risks have been used only to assess the relative risk of adverse effects from alternative remediation options at a given site or from one site to the next on the SRP. These methods are not to be assumed to be a quantitative evaluation and prediction of the incidence of adverse effects in exposed populations, but are rather a tool for the assessment of relative risk (i.e., comparison across sites or across the different remediation options).

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