M.2.5 RADIOLOGICAL IMPACTS AT NEVADA TEST SITE

This section presents the radiological impacts of the various storage and disposition alternatives at NTS. Section M.2.5.1 presents the radiological releases and resulting impacts from facilities associated with No Action. Section M.2.5.2 presents the radiological releases and resulting impacts from the various alternatives.

For purposes of radiological impact modeling, NTS was divided into six separate areas which would release radioactivity in 2005. All release points in each area were aggregated into a single release point. Table M.2.5–1 presents the characteristics of each of the release points including location, release height, and minimum distance and annual average dispersion to the site boundary in each of the 16 directions. In order to calculate the maximum site boundary dose (that is, the dose ultimately incurred to the site MEI), the dose from each release point to the "maximum receptor" (that is, potential MEI) associated with each of the other release points has been calculated. For further clarification on the definition of "maximum receptor" refer to Section M.2.2.2. For example, the dose resulting from releases for Areas 5, 9, 12, 19, and Device Assembly Facility has been determined from the maximum receptor for Area 3. Figure M.2.5–1 illustrates the location of each maximum receptor in relation to each release point. The maximum dose to one of these maximum receptors. Table M.2.5–2 presents the direction, distance, and atmospheric dispersion from each release point to each of the maximum receptors. Annual radiological releases were assumed to remain constant during the full operational period.

The population and food stuffs distributions centered on each release area are provided in a Health Risk Data report, October 1996. The joint frequency distribution used for the dose assessment was based on the meteorological measurements for 1990 from the Desert Rock at the 10-m (33-ft) height and is contained in the Health Risk Data report.

Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables, and for the projected operational period in the text. Tables M.2.5–3 and M.2.5–4 include the radiological impacts to the public from atmospheric release for No Action and the storage and disposition alternatives.

Release Point ^a	Are	 a 3	Are	a 5	Are	a 9	Аге	a 12	Area	a 19	DA	
Latitude	37°2'52		36°51'1		37°7'4	0.938"	37°13'	9.788"	37°15'14.317"		6°53'37.824"	
Longitude	-116°0'2		-115°57'		-116°2'	5.827"	-116°9'2	-116°9'20.893"		'59.322"	-116°2'54.794"	
Release	Ground		Ground		Ground	l Level	Ground	l Level	Ground	d Level	Ground	l Level
Height		<u> </u>										
			I	Distance and	d Atmosphe					01:10	D'-4	Chi/Q
Direction	Distance	Chi/Q	Distance	Chi/Q	Distance	Chi/Q	Distance	Chi/Q	Distance	Chi/Q (s/m ³)	Distance (m)	(s/m^3)
	(m)	(s/m ³)	(m)	(s/m ³)	(m)	(s/m ³)	(m)	(s/m^3)	(m)			4.6x10 ⁻⁹
N	22,334	9.6x10 ⁻⁹	11,017	2.5x10 ⁻⁸	13,494	1.9x10 ⁻⁸	3,598	1.2x10 ⁻⁷	14,593	1.7x10 ⁻⁸	39,497	4.0X10
NNE	12,393	2.3x10 ⁻⁸	4,008	1.1x10 ⁻⁷	13,751	2.0x10 ⁻⁸	3,666	1.3x10 ⁻⁷	13,493	2.0x10 ⁻⁸	19,043	1.3x10 ⁻⁸
NE	8,331	5.3x10 ⁻⁸	2,719	2.7x10 ⁻⁷	11,367	3.5x10 ⁻⁸	4,317	1.3x10 ⁻⁷	13,488	2.8x10 ⁻⁸	12,827	2.9x10 ⁻⁸
ENE	7,092	5.7x10 ⁻⁸	2,290	3.0x10 ⁻⁷	9,692	3.7x10 ⁻⁸	6,382	6.6x10 ⁻⁸	11,870	2.8x10 ⁻⁸	10,926	3.2x10 ⁻⁸
E	6,970	5.6x10 ⁻⁸	2,249	2.9x10 ⁻⁷	9,518	3.7x10 ⁻⁸	17,654	1.6x10 ⁻⁸	11,646	2.8x10 ⁻⁸	10,734	3.1x10 ⁻⁸
ESE	7,124	4.6x10 ⁻⁸	2,307	2.4x10 ⁻⁷	9,727	3.0x10 ⁻⁸	20,667	1.1x10 ⁻⁸	35,224	5.4x10 ⁻⁹	10,975	2.5x10 ⁻⁸
SE	8,470	3.3x10 ⁻⁸	2,736	1.7x10 ⁻⁷	11,519	2.2x10 ⁻⁸	24,525	7.9x10 ⁻⁹	41,754	3.9x10 ⁻⁹	13,007	1.8x10 ⁻⁸
SSE	12,801	2.1x10 ⁻⁸	4,119	1.0x10 ⁻⁷	17,407	1.4x10 ⁻⁸	37,017	5.2x10 ⁻⁹	63,001	2.6x10 ⁻⁹	19,622	1.2x10 ⁻⁸
S	37,774	6.3x10 ⁻⁹	12,182	2.8x10 ⁻⁸	50,960	4.3x10 ⁻⁹	61,111	3.4x10 ⁻⁹	58,791	3.6x10 ⁻⁹	27,780	9.4x10 ⁻⁹
SSW	43,741	5.7x10 ⁻⁹	26,724	1.1x10 ⁻⁸	52,085	4.6x10 ⁻⁹	46,477	5.3x10 ⁻⁹	20,820	1.5x10 ⁻⁸	26,284	1.1x10 ⁻⁸
SW	47,205	6.4x10 ⁻⁹	26,537	4.0x10 ⁻⁹	44,107	7.0x10 ⁻⁹	31,082	1.1x10 ⁻⁸	15,876	2.7x10 ⁻⁸	30,390	1.1x10 ⁻⁸
WSW 1	40,053	5.6x10 ⁻⁹	38,554	5.9x10 ⁻⁹	37,377	6.1x10 ⁻⁹	27,130	9.3x10 ⁻⁹	15,771	1.9x10 ⁻⁸	36,372	6.3x10 ⁻⁹
W	39,283	8.1x10 ⁻⁹	44,204	6.9x10 ⁻⁹	36,657	8.8x10 ⁻⁹	27,851	1.3x10 ⁻⁸	16,416	2.6x10 ⁻⁸	35,698	9.1x10 ⁻⁹
WNW	40,055	8.1x10 ⁻⁹	45,100	7.0x10 ⁻⁹	40,648	8.0x10 ⁻⁹	28,395	1.3x10 ⁻⁸	18,231	2.3x10 ⁻⁸	36,413	9.2x10 ⁻⁹
NW	27,062	7.7x10 ⁻⁹	53,212	3.2x10 ⁻⁹	16,370	1.5x10 ⁻⁸	4,360	9.3x10 ⁻⁸	15,211	1.6x10 ⁻⁸	42,950	4.2x10 ⁻⁹
NNW	22,808	4.2x10 ⁻⁹	44,582	1.8x10 ⁻⁹	13,801	8.2x10 ⁻⁹	3,679	5.2x10 ⁻⁸	14,912	7.4x10 ⁻⁹	40,381	2.0x10 ⁻⁹

Table M.2.5-1.	Release Point Characteristics, Direction, Distance, and Chi/Q at Nevada Test Site

* See Figure M.2.5-1 for location of release points.

Source: HNUS 1996a.

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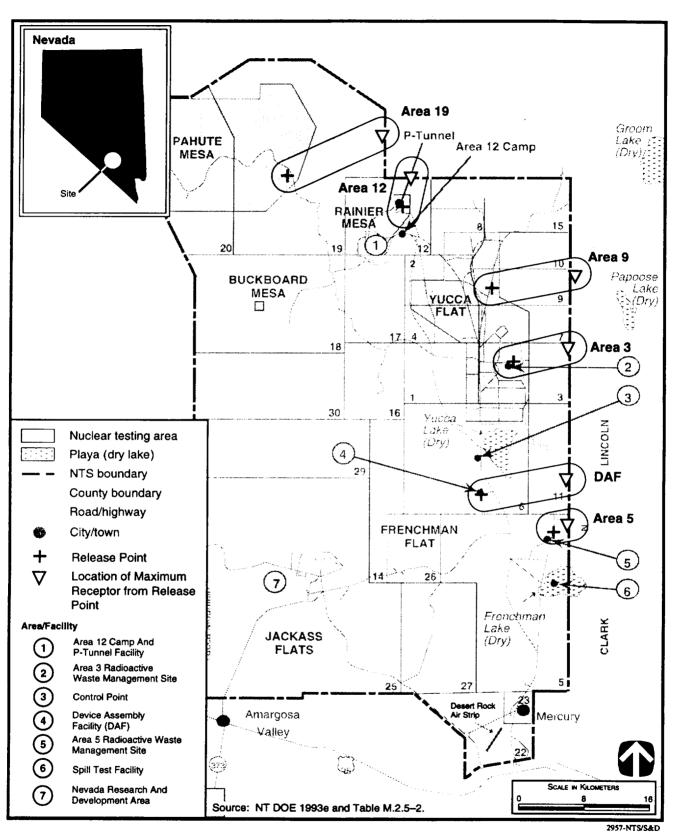


Figure M.2.5–1. Location of Release Points and Maximum Receptors at Nevada Test Site.

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<u> </u>	Direction	Distance	Atmospheric Dispersion Chi/Q
Maximum Receptor For		(m)	(s/m ³)
Release Point: Area 3		1	
Area 3	ENE	7,093	5.7x10 ⁻⁸
Area 5	SSE	22,117	1.0x10 ⁻⁸
Area 9	NNE	12,766	2.2x10 ⁻⁸
Area 12	NNW	25,015	3.8x10 ⁻⁹
Area 19	NNW	29,688	3.0x10 ⁻⁹
DAF	SSE	16,601	1.5x10 ⁻⁸
Release Point: Area 5			
Area 3	N	22,849	9.3x ¹⁰⁻⁹
Area 5	ENE	2,291	3.0x10 ⁻⁷
Area 9	N	32,178	6.0x10 ⁻⁹
Area 12	NNW	46,638	1.7x10 ⁻⁹
Area 19	NNW	50,866	1.5x10 ⁻⁹
DAF	NNE	6,740	5.3x10 ⁻⁸
Release Point: Area 9			•
Area 3	SE	12,141	2.0x10 ⁻⁸
Area 5	SSE	31,344	6.4x10 ⁻⁹
Area 9	ENE	9,692	3.7x10 ⁻⁸
Area 12	NNW	15,986	6.8x10 ⁻⁹
Area 19	NW	20,987	1.1x10 ⁻⁸
DAF	SSE	25,762	8.3x10 ⁻⁹
Release Point: Area 12			0
Area 3	SE	26,875	7.0x10 ⁻⁹
Area 5	SSE	44,834	4.0x10 ⁻⁹
Area 9	ESE	21,893	1.0x10 ⁻⁸
Area 12	NE	4,318	1.3x10 ⁻⁷
Area 19	NNW	6,614	2.3×10^{-8}
DAF	SSE	39,639	4.7×10^{-9}
Release Point: Area 19			<u>^</u>
Area 3	ESE	40,661	4.4x10 ⁻⁹
Area 5	SE	55,804	2.7x10 ⁻⁹
Area 9	ESE	36,584	5.1x10 ⁻⁹
Area 12	Е	16,711	1.7x10 ⁻⁸
Area 19	ENE	11,871	2.8x10 ⁻⁸
DAF	SE	51,278	3.0x10 ⁻⁹
Release Point: DAF			
Area 3	NNE	21,318	1.1x10 ⁻⁸
Area 5	SSE	11,411	2.4x10 ⁻⁸
Area 9	NNE	29,792	7.4x10 ⁻⁹
Area 12	N	40,290	4.5x10 ⁻⁹
Area 19	NNW	43,910	1.8x10 ⁻⁹
DAF	ENE	10,927	3.2×10^{-8}

 Table M.2.5–2.
 Direction, Distance, and Meteorological Dispersion to Various Maximum Individual

 Receptors at the Nevada Test Site Boundary

Source: HNUS 1996a.

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		Dose by Pat	hway (mrem)				·
Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (mrem)	Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
No Action (Total Site)	4.2x10 ⁻³	1.2x10 ⁻⁵	2.9x10 ⁻⁷	2.2x10 ⁻⁹	4.2x10 ⁻³	1.3x10 ⁻³	2.1x10 ⁻⁹
Consolidated Storage Facility (P-Tunnel)	5.5x10 ⁻⁶	9.6x10 ⁻⁹	2.1x10 ⁻¹⁵	4.4×10^{-12}	5.6x10 ⁻⁶	1.8x10 ⁻⁶	2.8×10^{-12}
Collocated Storage Facilities (P-Tunnel)	5.6x10 ⁻⁶	9.6x10 ⁻⁹	2.2x10 ⁻¹⁵	6.6x10 ⁻¹²	5.6x10 ⁻⁶	1.8x10 ⁻⁶	2.8x10 ⁻¹²
Consolidated Storage Facility	1.3x10 ⁻⁶	2.2x10 ⁻⁹	4.9x10 ⁻¹⁶	1.0x10 ⁻¹²	1.3x10 ⁻⁶	4.2x10 ⁻⁷	6.5x10 ⁻¹³
Collocated Storage Facilities	1.3x10 ⁻⁶	2.3x10 ⁻⁹	5.2×10^{-16}	1.6×10^{-12}	1.3x10 ⁻⁶	4.2×10^{-7}	6.5×10^{-13}
Pit Disassembly/ Conversion Facility	1.4x10 ⁻⁴	3.2x10 ⁻⁶	8.6x10 ⁻¹³	1.3x10 ⁻⁹	1.5x10 ⁻⁴	4.8x10 ⁻⁵	7.5x10 ⁻¹¹
Pu Conversion Facility	9.5x10 ⁻⁵	1.7x10 ⁻⁷	3.9x10 ⁻¹⁴	8.3x10 ⁻¹¹	9.5x10 ⁻⁵	3.0x10 ⁻⁵	4.8x10 ⁻¹¹
MOX Fuel Fabrication Facility	6.8x10 ⁻⁵	1.2×10^{-7}	2.6x10 ⁻¹⁴	1.3x10 ⁻¹⁰	6.8x10 ⁻⁵	2.2×10^{-5}	3.4×10^{-11}
Ceramic Immobilization Facility (Immobilized Disposition)	1.6x10 ⁻⁸	2.8x10 ⁻¹¹	6.4x10 ⁻¹⁸	1.3×10^{-14}	1.6x10 ⁻⁸	5.1x10 ⁻⁹	8.0x10 ⁻¹⁵
Deep Borehole Complex (Direct Disposition)	2.7x10 ⁻⁹	3.9x10 ⁻¹¹	1.0x10 ⁻¹⁷	1.5x10 ⁻¹⁴	2.7x10 ⁻⁹	8.6x10 ⁻¹⁰	1.4x10 ⁻¹⁵
Deep Borehole Complex (Immobilized Disposition)	3.3x10 ⁻⁹	5.8x10 ⁻¹¹	1.5x10 ⁻¹⁷	2.3x10 ⁻¹⁴	3.4x10 ⁻⁹	1.1x10 ⁻⁹	1.7x10 ⁻¹⁵
Vitrification Facility	6.6x10 ⁻⁶	4.3x10 ⁻⁷	1.3x10 ⁻¹⁰	7.1x10 ⁻⁸	7.2x10 ⁻⁶	2.3x10 ⁻⁶	3.6x10 ⁻¹²
Ceramic Immobilization Facility (Ceramic Immobilization)	1.8x10 ⁻⁸	8.5x10 ⁻⁸	2.5x10 ⁻¹¹	1.4×10^{-8}	1.2×10^{-7}	3.8x10 ⁻⁸	6.0×10^{-14}
Advanced Boiling Water Reactor	4.6x10 ⁻⁴	2.3x10 ⁻²	6.2×10^{-3}	5.7x10 ⁻⁴	3.0x10 ⁻²	9.6x10 ⁻³	1.5x10 ⁻⁸
CE System 80+ Reactor	1.4x10 ⁻³	2.6x10 ⁻²	5.7x10 ⁻⁴	2.2×10^{-4}	2.9×10^{-2}	9.3x10 ⁻³	1.5x10 ⁻⁸
[Text deleted.]					- .//10	2.5.10	1.5410
AP600 Reactor	7.4x10 ⁻⁴	2.2×10^{-2}	1.7x10 ⁻³	3.4x10 ⁻⁴	2.5x10 ⁻²	8.0x10 ⁻³	1.3x10 ⁻⁸
RESAR-90 Reactor	1.2×10^{-3}	3.2x10 ⁻²	7.0x10 ⁻⁴	2.8×10^{-4}	3.4×10^{-2}	1.1×10^{-2}	1.7x10 ⁻⁸

Table M.2.5-3. Doses and Resulting Health Effects to the Maximally Exposed Individual at Nevada Test Site From Atmospheric Releases Associated With Annual Normal Operation

^a Individual annual natural background radiation dose is equal to 313 mrem.

Source: HNUS 1996a.

		Dose by Pathw	ay (person-rem)				
	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (person-rem)	Percent of Background ^a	Estimated 1-Year Fatal Cancers
No Action (Total Site)	3.7x10 ⁻³	9.4x10 ⁻⁶	3.6x10 ⁻⁶	1.9x10 ⁻⁹	3.7x10 ⁻³	4.0x10 ⁻⁵	1.9x10 ⁻⁶
Consolidated Storage Facility (P-Tunnel)	1.7x10 ⁻⁶	1.8x10 ⁻¹¹	6.6x10 ⁻¹⁶	1.4×10^{-12}	1.7x10 ⁻⁶	1.8x10 ⁻⁸	8.5x10 ⁻¹⁰
Collocated Storage Facility (P-Tunnel)	1.7x10 ⁻⁶	1.9x10 ⁻¹¹	7.0x10 ⁻¹⁶	2.1×10^{-12}	1.7x10 ⁻⁶	1.8x10 ⁻⁸	8.5x10 ⁻¹⁰
Consolidated Storage Facility	2.6x10 ⁻⁶	5.8×10^{-11}	1.0×10^{-15}	2.1×10^{-12}	2.6x10 ⁻⁶	2.8x10 ⁻⁸	1.3x10 ⁻⁹
Collocated Storage Facilities	2.6×10^{-6}	6.2x10 ⁻¹¹	1.1x10 ⁻¹⁵	3.2×10^{-12}	2.6x10 ⁻⁶	2.8x10 ⁻⁸	1.3x10 ⁻⁹
Pit Disassembly/ Conversion Facility	2.9x10 ⁻⁴	8.4×10^{-8}	1.7×10^{-12}	2.6x10 ⁻⁹	2.9x10 ⁻⁴	3.2x10 ⁻⁶	1.5x10 ⁻⁷
Pu Conversion Facility	1.9x10 ⁻⁴	4.6x10 ⁻⁹	8.0x10 ⁻¹⁴	1.7x10 ⁻¹⁰	1.9x10 ⁻⁴	2.1x10 ⁻⁶	9.5x10 ⁻⁸
MOX Fuel Fabrication Facility	1.4×10^{-4}	3.4x10 ⁻⁹	5.4×10^{-14}	2.6x10 ⁻¹⁰	1.4x10 ⁻⁴	1.5x10 ⁻⁶	7.0x10 ⁻⁸
Ceramic Immobilization Facility (Immobilized Disposition)	3.3x10 ⁻⁸	7.2×10^{-13}	1.3×10^{-17}	2.7x10 ⁻¹⁴	3.3x10 ⁻⁸	3.6x10 ⁻¹⁰	1.7x10 ⁻¹¹
(Direct Disposition)	5.3x10 ⁻⁹	1.0×10^{-12}	2.1×10^{-17}	3.2x10 ⁻¹⁴	5.3x10 ⁻⁹	5.8x10 ⁻¹¹	2.7×10^{-12}
Deep Borehole Complex (Immobilized Disposition)	6.6x10 ⁻⁹	1.5x10 ⁻¹²	3.1x10 ⁻¹⁷	4.8x10 ⁻¹⁴	6.6x10 ⁻⁹	7.2x10 ⁻¹¹	3.3x10 ⁻¹²
Vitrification Facility	1.3x10 ⁻⁵	5.2×10^{-7}	2.6x10 ⁻¹⁰	1.4x10 ⁻⁷	1.4x10 ⁻⁵	1.5×10^{-7}	7.0x10 ⁻⁹
Ceramic Immobilization Facility (Ceramic Immobilization)	3.7x10 ⁻⁸	1.0x10 ⁻⁷	5.1x10 ⁻¹¹	2.9x10 ⁻⁸	1.7x10 ⁻⁷	1.8x10 ⁻⁹	8.5x10 ⁻¹¹
Advanced Boiling Water Reactor	7.3x10 ⁻⁴	2.0×10^{-2}	5.8x10 ⁻³	8.9x10 ⁻⁴	2.7×10^{-2}	2.9x10 ⁻⁴	1.4×10^{-5}
CE System 80+ Reactor	2.8x10 ⁻³	2.0×10^{-2}	8.3x10 ⁻⁴	4.5×10^{-4}	2.4×10^{-2}	2.6x10 ⁻⁴	1.2x10 ⁻⁵
[Text deleted.]							
AP600 Reactor	1.5×10^{-3}	1.7×10^{-2}	2.8x10 ⁻³	6.9x10 ⁻⁴	2.2×10^{-2}	2.4×10^{-4}	1.1x10 ⁻⁵
RESAR-90 Reactor	2.4x10 ⁻³	2.7x10 ⁻²	1.2×10^{-3}	5.8x10 ⁻⁴	3.2×10^{-2}	3.5×10^{-4}	1.6x10 ⁻⁵

Table M.2.5–4. Doses and Resulting Health Effects to the Population Within 80 Kilometers of Nevada Test Site From Atmospheric Releases Associated With Normal Operation in 2030

^a Dose to the population within 80 km from natural background radiation in year 2030 is equal to 9,190 person-rem. Source: HNUS 1996a.

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M.2.5.1 No Action

I

Atmospheric Releases and Resulting Impacts to the Public. For No Action, five of the six areas have radioactive releases to the atmosphere from normal operation. Table M.2.5.1–1 presents the estimated annual atmospheric radioactive releases.

Table M.2.5.1–1.	Annual Atmospheric Radioactive Releases From Normal
Opera	ution of No Action at Nevada Test Site (curies)

Isotope	Area 3	Area 5	Area 9	Area 12	Area 19
H-3	0	0.29	0	3.7	0
Kr-85	0	0	0	0	160
Pu-239	1.0x10 ⁻³	0	7.5x10 ⁻⁴	0	0

Source: NT DOE 1994b.

Tables M.2.5–3 and M.2.5–4, respectively, include the radiological impacts to the maximally exposed member of the public and offsite population within 80 km (50 mi). The MEI would receive an annual dose of 4.2×10^{-3} mrem. An estimated fatal cancer risk of 1.0×10^{-7} would result from 50 years of operation. The population within 80 km (50 mi) would receive a dose of 3.7×10^{-3} person-rem in 2030 (mid-life of operation). An estimated 9.3 $\times 10^{-5}$ fatal cancers could result from 50 years of operation.

Liquid Releases and Resulting Impacts to the Public. There are no radioactive liquid releases to the offsite environment associated with No Action. Therefore, there are no resulting impacts.

Worker Doses and Health Effects. Based on measured values during the time period of 1989 to 1992 (*Twenty-Second Annual Report Radiation Exposures for DOE and DOE Contract Employees-1989* [DOE/EH-0286P]) and subsequent yearly dose reports), the annual average dose to a badged worker at NTS was calculated to be 5 mrem. It is projected that in 2005 and beyond, there would be 619 badged workers involved in No Action activities at NTS. The annual average dose to these workers was assumed to remain at 5 mrem; the annual total dose among all these workers would then equal 3 person-rem. From 50 years of operation, an estimated fatal cancer risk of 1.0×10^{-4} would result to the average worker and 0.060 fatal cancer could result among all workers.

M.2.5.2 Storage and Disposition

Radioactive Releases and Resulting Impacts to the Public. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the incremental impacts from the storage or disposition facilities to determine the impacts from total site operation. For example, to determine the radiological impact for the addition of an AP600 reactor at NTS, the doses from No Action facilities have to be summed with the AP600 reactor doses. Estimated annual atmospheric radioactive releases from the facilities associated with the various alternative actions are given in Section M.2.3. Tables M.2.5–3 and M.2.5–4 include the radiological impacts by alternative facility. There are no radioactive liquid releases to the offsite environment associated with any alternative action.

The annual incremental doses associated with the different alternative facilities range from 2.7×10^{-9} to 0.034 mrem to the MEI and from 5.3×10^{-9} to 0.032 person-rem to the 80-km (50-mi) population in the year 2030. The associated health effects from annual operations are included in both tables.

Worker Doses and Health Effects. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the incremental impacts from the storage or disposition facilities to determine the impacts from total site operation (refer to the worker discussion under No Action, above, and to Table M.2.3.2-1).

M.2.6 RADIOLOGICAL IMPACTS AT IDAHO NATIONAL ENGINEERING LABORATORY

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This section presents the radiological impacts of various storage and disposition alternatives at INEL. Section M.2.6.1 presents the radiological releases and resulting impacts from facilities associated with No Action. Section M.2.6.2 presents the radiological releases and resulting impacts from the various alternatives.

For purposes of radiological impact modelling, INEL was divided into nine separate areas which would release radioactivity in 2005. All release points in each area were aggregated into a single release point. Table M.2.6–1 presents the characteristics of each of the release points including location, release height, and minimum distance to and annual average dispersion to the site boundary in each of the 16 directions. In order to calculate the maximum site boundary dose (that is, the dose ultimately incurred to the site MEI), the dose from each release point to the "maximum receptor" (that is, potential MEI) associated with each of the other release points has been calculated. For further clarification on the definition of the "maximum receptor" refer to Section M.2.2.2. For example, the dose resulting from releases from the Test Reactor Area, Argonne National Laboratory-West (ANL-W), Waste Experimental Reduction Facility/Power Burst Facility Area, and the other storage and disposition alternatives, has been determined for the maximum receptor from the Central Facilities Area. Figure M.2.6–1 illustrates the location of each maximum receptor in relation to each release point. The maximum dose to one of these maximum receptors. Table M.2.6–2 presents the direction, distance, and atmospheric dispersion from each release point to each of the maximum receptors. Annual radiological releases were assumed to remain constant during the full operational period.

The population and food stuffs distributions centered on each release area are provided in a Health Risk Data report, October 1996. The joint frequency distribution used for the dose assessment was based on the meteorological measurements for the year 1986 from the GRID III tower at the 10-m (33-ft) height and is contained in the Health Risk Data report.

Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables, and for the projected operational period in the text. Tables M.2.6–3 and M.2.6–4 include the radiological impact to the public from atmospheric releases for No Action and the storage and disposition alternatives.

				Sacoratory	Doundary					
Release Point ^a	SN	4C	TA	AN	T	RA	IC	PP	LWF	R Site
Latitude	43°51'2	27.741"	43°50'5	56.339"	43°35	8.244"	43°34'	16.091"	43°34'42.623"	
Longitude	-112°43	'47.366"	-112°42	2'14.153"	-112°57	7'46.840"	-112°56	5'4.083"		2'5.376"
Release Height	Ground	d Level	51.	4 m	76.	2 m		2 m	Ground Level	
		Di	istance and At	mospheric D	ispersion at S	ite Boundary				
Direction	Distance (m)	Chi/Q (s/m ³)								
N	18,016	1.6x10 ⁻⁸	18,938	5.2x10 ⁻⁹	19,099	3.3x10 ⁻⁹	20,722	3.1x10 ⁻⁹	22,328	1.2x10 ⁻⁸
NNE	16,399	3.7x10 ⁻⁸	12,650	1.8x10 ⁻⁸	21,737	6.8x10 ⁻⁹	24,282	6.0x10 ⁻⁹	44,886	9.8x10 ⁻⁹
NE	13,055	1.2x10 ⁻⁷	12,336	4.4×10^{-8}	42,901	7.3x10 ⁻⁹	42,405	7.4x10 ⁻⁹	37,705	2.9x10 ⁻⁸
ENE	12,005	9.9x10 ⁻⁸	9,884	3.4×10^{-8}	41,932	4.6x10 ⁻⁹	39,577	4.9x10 ⁻⁹	34,098	2.5x10 ⁻⁸
E	11,726	4.0x10 ⁻⁸	9,685	1.3x10 ⁻⁸	26,374	2.9x10 ⁻⁹	23,863	3.2x10 ⁻⁹	19,377	2.0x10 ⁻⁸
ESE	16,180	1.2x10 ⁻⁸	15,770	3.9x10 ⁻⁹	26,409	1.4x10 ⁻⁹	24,074	1.5x10 ⁻⁹	18,696	1.0x10 ⁻⁸
SE	26,221	3.3x10 ⁻⁹	23,757	1.3x10 ⁻⁹	19,093	1.0x10 ⁻⁹	16,409	1.2x10 ⁻⁹	18,261	5.2x10 ⁻⁹
SSE	35,151	3.2x10 ⁻⁹	33,821	1.2x10 ⁻⁹	15,967	1.6x10 ⁻⁹	14,337	1.7x10 ⁻⁹	14,690	1.0x10 ⁻⁸
S	35,319	9.6x10 ⁻⁹	33,731	3.8x10 ⁻⁹	15,538	5.2x10 ⁻⁹	13,952	5.8x10 ⁻⁹	14,635	3.1x10 ⁻⁸
SSW	46,586	9.7x10 ⁻⁹	44,899	4.5x10 ⁻⁹	15,753	1.0x10 ⁻⁸	14,144	1.1x10 ⁻⁸	15,028	4.3x10 ⁻⁸
SW	30,060	1.8x10 ⁻⁸	34,045	7.6x10 ⁻⁹	18,299	1.2x10 ⁻⁸	16,442	1.3x10 ⁻⁸	17,459	3.7x10 ⁻⁸
WSW	12,107	2.4x10 ⁻⁸	14,260	6.8x10 ⁻⁹	18,988	3.3x10 ⁻⁹	21,409	2.9x10 ⁻⁹	25,439	8.9x10 ⁻⁹
W	11,779	3.4x10 ⁻⁸	13,873	9.9x10 ⁻⁹	17,014	5.2x10 ⁻⁹	20,752	4.2x10 ⁻⁹	24,305	1.3x10 ⁻⁸
WNW	9,215	1.4x10 ⁻⁸	11,510	3.5x10 ⁻⁹	12,184	2.2x10 ⁻⁹	14,992	1.8x10 ⁻⁹	17,919	5.7x10 ⁻⁹
NW	10,005	2.0x10 ⁻⁸	12,462	4.4x10 ⁻⁹	11,503	2.9x10 ⁻⁹	14,283	2.3x10 ⁻⁹	17,908	9.2x10 ⁻⁹
NNW	14,481	1.3x10 ⁻⁸	18,035	3.4x10 ⁻⁹	12,204	3.4x10 ⁻⁹	15,365	2.7x10 ⁻⁹	20,732	8.2x10 ⁻⁹

Table M.2.6–1. Release Point Characteristics, Direction, Distance, and Chi/Q at the Idaho National Engineering Laboratory Boundary

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Release Point ^a	WE	 RF	CF		RWN		ANL	
Latitude	43°33'3	3.443"	43°32'4.386"		43°29'5		43°35'41.733" -112°39'18.744" 42.7 m	
Longitude	-112°51'	31.071"	-112°56'	10.073"	-113°2'13.843"			
Release Height	24.4		Ground Level		Ground	Level		
Kelease Height		Distance and A	tmospheric D	ispersion at S	ite Boundary			
	Distance	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)
Direction	(m)	<u>6.7x10⁻⁹</u>	24,783	1.1x10 ⁻⁸	17,201	1.7x10 ⁻⁸	32,639	8.3x10 ⁻⁹
N	25,458	7.5×10^{-9}	40,101	1.1x10 ⁻⁸	29,087	1.7x10 ⁻⁸	24,645	1.4x10 ⁻⁸
NNE	41,139	1.9x10 ⁻⁸	45,052	2.3×10^{-8}	53,829	1.8x10 ⁻⁸	19,642	1.6x10 ⁻⁸
NE	39,204	1.6x10 ⁻⁸	39,302	2.0x10 ⁻⁸	47,686	1.6x10 ⁻⁸	16,056	1.5x10 ⁻⁸
ENE	32,888	1.4×10^{-8}	23,842	1.5x10 ⁻⁸	32,039	1.0x10 ⁻⁸	14,469	9.4x10 ⁻⁹
E	17,582	6.7x10 ⁻⁹	18,765	1.0x10 ⁻⁸	11,265	2.0x10 ⁻⁸	9,005	1.5x10 ⁻⁸
ESE	17,857	4.5×10^{-9}	11,856	9.4x10 ⁻⁹	7,293	1.9x10 ⁻⁸	5,862	2.6x10 ⁻⁸
SE	14,508	4.5x10 8.4x10 ⁻⁹	10,161	1.7×10^{-8}	6,090	3.5x10 ⁻⁸	5,518	5.5x10 ⁻⁸
SSE	11,541	2.6x10 ⁻⁸	9,886	5.3x10 ⁻⁸	5,924	1.1x10 ⁻⁷	5,571	7.3x10 ⁻⁸
S	11,539	3.8x10 ⁻⁸	10,021	7.5x10 ⁻⁸	6,003	1.6x10 ⁻⁷	17,065	2.5×10^{-8}
SSW	11,937	-3.4×10^{-8}	11,653	6.4×10^{-8}	6,992	1.3x10 ⁻⁷	19,886	2.1x10 ⁻⁸
SW	13,872	7.9x10 ⁻⁹	16,966	1.5x10 ⁻⁸	10,193	3.0x10 ⁻⁸	28,926	1.1x10 ⁻⁸
WSW	20,227 26,937	7.5x10 ⁻⁹	20,726	1.6x10 ⁻⁸	12,661	3.1x10 ⁻⁸	35,298	6.1x10 ⁻⁹
W	20,937 21,124	3.1x10 ⁻⁹	19,192	5.2x10 ⁻⁹	12,803	8.9x10 ⁻⁹	32,525	5.0x10 ⁻⁹
WNW	20,318	4.9×10^{-9}	17,203	9.7x10 ⁻⁹	14,757	1.2x10 ⁻⁸	27,828	6.4x10 ⁻⁹
NW NNW	23,853	4.4×10^{-9}	17,397	1.0x10 ⁻⁸	16,111	1.1x10 ⁻⁸	31,167	8.5x10 ⁻⁹

Table M.2.6–1. Release Point Characteristics, Direction, Distance, and Chi/Q at the Idaho National Engineering Laboratory Boundary —Continued

^a See Figure M.2.6-1 for location of release points.

Note: TAN=Test Area North; TRA=Test Reactor Area; WERF=Waste Experimental Reduction Area. Source: HNUS 1996a.

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Storage and Disposition of Weapons-Usable Fissile Materials Final PEIS

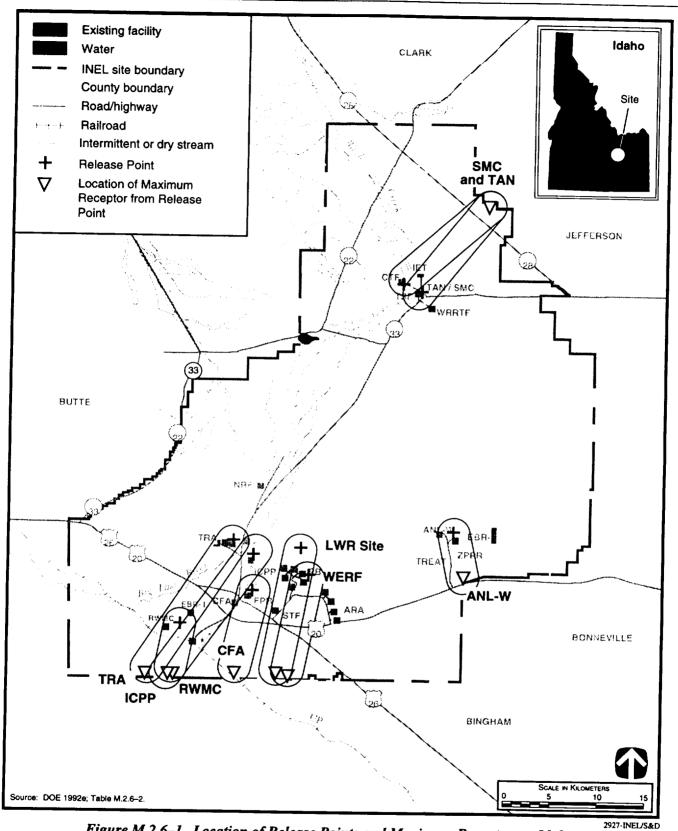


Figure M.2.6–1. Location of Release Points and Maximum Receptors at Idaho National Engineering Laboratory.

Maximum Receptor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
Release Point: SMC			
SMC and TAN	NE	14,481	1.1×10^{-7}
TRA	SSW	53,888	8.1x10 ⁻⁹
ICPP	SSW	52,249	8.4x10 ⁻⁹
LWR Site	SSW	47,838	9.4x10 ⁻⁹
WERF	SSW	47,497	9.5x10 ⁻⁹
CFA	SSW	49,319	9.0x10 ⁻⁹
RWMC	SSW	52,487	8.3x10 ⁻⁹
ANL-W	S	35,376	9.6x10 ⁻⁹
Release Point: TAN	-	, - · · -	
SMC and TAN	NE	12,337	4.4x10 ⁻⁸
TRA	SW	54,224	4.4x10 ⁻⁹
ICPP	SSW	52,464	3.8x10 ⁻⁹
LWR Site	SSW	47,566	4.2x10 ⁻⁹
WERF	SSW	47,165	4.3x10 ⁻⁹
CFA	SSW	49,252	4.1x10 ⁻⁹
RWMC	SSW	52,722	3.8x10 ⁻⁹
ANL-W	S	34,503	3.7x10 ⁻⁹
Release Point: TRA	-		
SMC and TAN	NE	48,269	6.4x10 ⁻⁹
TRA	SW	18,299	1.2×10^{-8}
ICPP	SSW	16,796	9.4x10 ⁻⁹
LWR Site	SSE	16,393	1.5x10 ⁻⁹
WERF	SSE	16,850	1.5x10 ⁻⁹
CFA	S	15,549	5.2x10 ⁻⁹
RWMC	SSW	16,992	9.3x10 ⁻⁹
ANL-W	Е	16,415	2.9x10 ⁻⁹
Release Point: ICPP			
SMC and TAN	NE	48,292	6.4x10 ⁻⁹
TRA	SW	18,395	1.2x10 ⁻⁸
ICPP	SW	16,443	1.3x10 ⁻⁸
LWR Site	S	14,251	5.7x10 ⁻⁹
WERF	SSE	14,570	1.7x10 ⁻⁹
CFA	S	14,059	5.8x10 ⁻⁹
RWMC	SW	16,712	1.3x10 ⁻⁸
ANL-W	Е	23,906	3.2x10 ⁻⁹
Release Point: LWR Site			
SMC and TAN	NNE	44,887	9.8x10 ⁻⁹
TRA	SW	22,811	2.6x10 ⁻⁸
ICPP	SW	20,426	3.0x10 ⁻⁸
LWR Site	SSW	15,029	4.3x10 ⁻⁸
WERF	S	14,873	3.0x10 ⁻⁸

Table M.2.6–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Idaho National Engineering Laboratory Site Boundary

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Maximum Receptor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
CFA	SSW	16,430	3.8x10 ⁻⁸
RWMC	SW	20,771	2.9x10 ⁻⁸
ANL-W	ESE	18,736	1.0x10 ⁻⁸
Release Point: WERF		, -	
SMC and TAN	NNE	47,288	6.3x10 ⁻⁹
TRA	WSW	21,626	7.3x10 ⁻⁹
ICPP	SW	18,954	2.3×10^{-8}
LWR Site	SSW	12,217	3.6x10 ⁻⁸
WERF	SSW	11,938	3.8x10 ⁻⁸
CFA	SW	14,170	3.3x10 ⁻⁸
RWMC	SW	19,347	2.2×10^{-8}
ANL-W	E	17,626	1.4×10^{-8}
Release Point: CFA		11,020	1.4×10
SMC and TAN	NNE	51,824	8.2x10 ⁻⁹
TRA	SW	15,468	4.3×10^{-8}
ICPP	SW	13,109	5.4x10 ⁻⁸
LWR Site	SSE	10,331	1.6x10 ⁻⁸
WERF	SSE	10,777	1.5×10^{-8}
CFA	SSW	10,021	7.5×10^{-8}
RWMC	SW	13,442	5.2×10^{-8}
ANL-W	Е	23,916	1.5×10^{-8}
Release Point: RWMC		20,710	1.5×10
SMC and TAN	NE	59,528	1.6x10 ⁻⁸
TRA	SW	7,019	1.3×10^{-7}
ICPP	S	5,943	1.1x10 ⁻⁷
LWR Site	ESE	12,605	1.7×10^{-8}
WERF	ESE	13,761	1.5x10 ⁻⁸
CFA	SE	8,791	1.4×10^{-8}
RWMC	SSW	6,004	1.6x10 ⁻⁷
ANL-W	Е	32,468	1.0x10 ⁻⁸
Release Point: ANL-W		,	1.0/10
SMC and TAN	Ν	38,094	6.8x10 ⁻⁹
TRA	WSW	38,408	7.9x10 ⁻⁹
ICPP	WSW	35,484	8.7x10 ⁻⁹
LWR Site	SW	25,870	1.5×10^{-8}
WERF	SW	24,903	1.5×10^{-8}
CFA	wsw	29,537	1.1x10 ⁻⁸
RWMC	wsw	35,923	8.6x10 ⁻⁹
ANL-W	S	5,572	7.3×10^{-8}

Table M.2.6–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Idaho National Engineering Laboratory Site Boundary---Continued

Note: WERF=Waste Experimental Reduction Facility; TAN=Test Area North; TRA=Test Reactor Area. Source: HNUS 1996a.

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	Dose by Path	way (mrem)				
Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (mrem)	Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
2.8.10-4	1.5×10 ⁻²	2.2×10^{-3}	2.9x10 ⁻⁵	1.8x10 ^{-2b}	5.2x10 ⁻³	8.9x10 ⁻⁹
					1.5x10 ⁻⁷	2.6x10 ⁻¹³
						8.0x10 ⁻¹³
						8.0x10 ⁻¹³
						9.0x10 ⁻¹¹
						6.0x10 ⁻¹¹
						4.4x10 ⁻¹¹
8.8x10 ⁻⁵						1.0×10^{-14}
2.0x10 ⁻⁸	3.6x10 ⁻¹¹	8.1x10 ⁻¹⁸	1.6x10 ⁻¹⁴			
3.3x10 ⁻⁹	4.9x10 ⁻¹¹	1.3x10 ⁻¹⁷	1.9x10 ⁻¹⁴	3.4x10 ⁻⁹	1.0x10 ⁻⁹	1.7x10 ⁻¹⁵
4.1x10 ⁻⁹	7.2x10 ⁻¹¹	1.9x10 ⁻¹⁷	2.9x10 ⁻¹⁴	4.2x10 ⁻⁹	1.2x10 ⁻⁹	2.1x10 ⁻¹⁵
	5.5x10 ⁻⁷	1.6x10 ⁻¹⁰	9.0x10 ⁻⁸	8.9x10 ⁻⁶	2.6x10 ⁻⁶	4.4×10^{-12}
2.3×10^{-8}	1.1x10 ⁻⁷	3.2x10 ⁻¹¹	1.7x10 ⁻⁸	1.5x10 ⁻⁷	4.4x10 ⁻⁸	7.5x10 ⁻¹⁴
10 10-3	6.2×10-2	1.3×10^{-2}	1.4×10^{-3}	7.8×10^{-2}	2.3×10^{-2}	3.9x10 ⁻⁸
					1.1×10^{-2}	1.9x10 ⁻⁸
1.9×10^{-5}	3.0X10 -	1.2810	5.0410	510410		
1 0x 10 ⁻³	2.9x10 ⁻²	2.2×10^{-3}	4.5x10 ⁻⁴	3.3x10 ⁻²	9.8x10 ⁻³	1.7x10 ⁻⁸
			3.8x10 ⁻⁴	4.6×10^{-2}	1.4×10^{-2}	2.3x10 ⁻⁸
	2.8x10 ⁻⁴ 5.1x10 ⁻⁷ 1.6x10 ⁻⁶ 1.6x10 ⁻⁶ 1.8x10 ⁻⁴ 1.2x10 ⁻⁴ 8.8x10 ⁻⁵ 2.0x10 ⁻⁸ 3.3x10 ⁻⁹ 4.1x10 ⁻⁹ 8.2x10 ⁻⁶	InhalationIngestion 2.8×10^{-4} 1.5×10^{-2} 5.1×10^{-7} 8.8×10^{-10} 1.6×10^{-6} 2.8×10^{-9} 1.8×10^{-4} 4.0×10^{-6} 1.2×10^{-4} 2.2×10^{-7} 8.8×10^{-5} 1.5×10^{-7} 2.0×10^{-8} 3.6×10^{-11} 3.3×10^{-9} 4.9×10^{-11} 4.1×10^{-9} 7.2×10^{-11} 8.2×10^{-6} 5.5×10^{-7} 2.3×10^{-8} 1.1×10^{-7} 1.2×10^{-3} 6.2×10^{-2} 1.9×10^{-3} 3.6×10^{-2} 1.0×10^{-3} 2.9×10^{-2}	InhalationIngestionImmersion 2.8×10^{-4} 1.5×10^{-2} 2.2×10^{-3} 5.1×10^{-7} 8.8×10^{-10} 1.9×10^{-16} 1.6×10^{-6} 2.8×10^{-9} 6.1×10^{-16} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.8×10^{-4} 4.0×10^{-6} 1.1×10^{-12} 1.2×10^{-4} 2.2×10^{-7} 5.0×10^{-14} 8.8×10^{-5} 1.5×10^{-7} 3.3×10^{-14} 2.0×10^{-8} 3.6×10^{-11} 8.1×10^{-18} 3.3×10^{-9} 4.9×10^{-11} 1.3×10^{-17} 4.1×10^{-9} 7.2×10^{-11} 1.9×10^{-17} 8.2×10^{-6} 5.5×10^{-7} 1.6×10^{-10} 2.3×10^{-8} 1.1×10^{-7} 3.2×10^{-11} 1.2×10^{-3} 6.2×10^{-2} 1.3×10^{-2} 1.9×10^{-3} 3.6×10^{-2} 7.2×10^{-4} 1.0×10^{-3} 2.9×10^{-2} 2.2×10^{-3}	InhalationIngestionPlume ImmersionGround Shine 2.8×10^{-4} 1.5×10^{-2} 2.2×10^{-3} 2.9×10^{-5} 5.1×10^{-7} 8.8×10^{-10} 1.9×10^{-16} 4.0×10^{-13} 1.6×10^{-6} 2.8×10^{-9} 6.1×10^{-16} 1.3×10^{-12} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.9×10^{-12} 1.6×10^{-6} 2.8×10^{-9} 6.5×10^{-16} 1.9×10^{-12} 1.8×10^{-4} 4.0×10^{-6} 1.1×10^{-12} 1.6×10^{-9} 1.2×10^{-4} 2.2×10^{-7} 5.0×10^{-14} 1.0×10^{-10} 8.8×10^{-5} 1.5×10^{-7} 3.3×10^{-14} 1.0×10^{-10} 2.0×10^{-8} 3.6×10^{-11} 8.1×10^{-18} 1.6×10^{-14} 3.3×10^{-9} 4.9×10^{-11} 1.3×10^{-17} 1.9×10^{-14} 4.1×10^{-9} 7.2×10^{-11} 1.9×10^{-17} 2.9×10^{-14} 8.2×10^{-6} 5.5×10^{-7} 1.6×10^{-10} 9.0×10^{-8} 2.3×10^{-8} 1.1×10^{-7} 3.2×10^{-11} 1.7×10^{-8} 1.2×10^{-3} 6.2×10^{-2} 1.3×10^{-2} 1.4×10^{-3} 1.0×10^{-3} 2.9×10^{-2} 2.2×10^{-3} 4.5×10^{-4}	$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$	$ \begin{array}{ c c c c c c c c c c c c c c c c c c c$

Table M.2.6–3. Doses and Resulting Health Effects to the Maximally Exposed Individual at Idaho National Engineering Laboratory From Atmospheric Releases Associated With Annual Normal Operation

^a Average individual annual natural background radiation is equal to 338 mrem.
 ^b The storage facility contributes 1.4x10⁻⁵ mrem/year.

[Text deleted.]

Source: HNUS 1996a.

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		Dose by Pathwa	ay (person-rem)				
Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (person-rem)	Percent of Background ^a	Estimated 1-Year Fatal Cancers
No Action (Total Site)	2.9×10^{-3}	2.4	2.1x10 ⁻²	3.0x10 ⁻⁴	2.4 ^b	2.7x10 ⁻³	1.2x10 ⁻³
Upgrade Storage	3.1x10 ⁻⁶	1.5x10 ⁻⁷	1.2x10 ⁻¹⁵	2.5×10^{-12}	3.2x10 ⁻⁶	3.5x10 ⁻⁹	1.6x10 ⁻⁹
Consolidated Storage Facility	1.7x10 ⁻⁵	9.1x10 ⁻⁷	6.5x10 ⁻¹⁵	1.4×10^{-11}	1.8x10 ⁻⁵	2.0×10^{-8}	9.0x10 ⁻⁹
Collocated Storage Facilities	1.7x10 ⁻⁵	9.2×10^{-7}	6.9x10 ⁻¹⁵	2.1x10 ⁻¹¹	1.8x10 ⁻⁵	2.0×10^{-8}	9.0x10 ⁻⁹
Pit Disassembly/ Conversion Facility	1.9x10 ⁻³	1.3x10 ⁻³	1.1x10 ⁻¹¹	1.6x10 ⁻⁸	3.2×10^{-3}	3.5x10 ⁻⁶	1.6x10 ⁻⁶
Pu Conversion Facility	8.6x10 ⁻⁴	3.1×10^{-4}	1.9x10 ⁻¹²	3.1x10 ⁻⁹	1.2x10 ⁻³	1.3x10 ⁻⁶	6.0x10 ⁻⁷
MOX Fuel Fabrication Facility	9.2x10 ⁻⁴	4.9x10 ⁻⁵	3.5×10^{-13}	1.7x10 ⁻⁹	9.7x10 ⁻⁴	1.1x10 ⁻⁶	4.9×10^{-7}
Ceramic Immobilization Facility (Immobilized Disposition)	2.2×10^{-7}	1.2x10 ⁻⁸	8.4x10 ⁻¹⁷	1.7×10^{-13}	2.3×10^{-7}	2.5×10^{-10}	1.2×10^{-10}
Deep Borehole Complex (Direct Disposition)	3.5x10 ⁻⁸	1.6x10 ⁻⁸	1.3x10 ⁻¹⁶	2.0x10 ⁻¹³	5.1x10 ⁻⁸	5.6x10 ⁻¹¹	2.6x10 ⁻¹¹
Deep Borehole Complex (Immobilized Disposition)	4.4×10^{-8}	2.3x10 ⁻⁸	2.0x10 ⁻¹⁶	3.0x10 ⁻¹³	6.7x10 ⁻⁸	7.4x10 ⁻¹¹	3.4x10 ⁻¹¹
Vitrification Facility	8.6x10 ⁻⁵	7.1x10 ⁻⁵	1.7x10 ⁻⁹	9.0x10 ⁻⁷	1.6x10 ⁻⁴	1.8x10 ⁻⁷	8.0x10 ⁻⁸
Ceramic Immobilization Facility (Ceramic Immobilization)	2.4x10 ⁻⁷	1.3x10 ⁻⁵	3.3×10^{-10}	1.9x10 ⁻⁷	1.4×10^{-5}	1.5×10^{-8}	7.0x10 ⁻⁹
Advanced Boiling Water Reactor	1.0×10^{-2}	13	7.6x10 ⁻²	1.2×10^{-2}	14	1.5x10 ⁻²	6.8x10 ⁻³
CE System 80+ Reactor	2.1×10^{-2}	8.6	5.4x10 ⁻³	3.4x10 ⁻³	8.6	9.5x10 ⁻³	4.3×10^{-3}
[Text deleted.]				UNATO	0.0	2.3810	4.5810
AP600 Reactor	1.1x10 ⁻²	6.9	1.9x10 ⁻²	5.1x10 ⁻³	6.9	7.6x10 ⁻³	3.5x10 ⁻³
RESAR-90 Reactor	1.8x10 ⁻²	9.6	8.2×10^{-3}	4.3×10^{-3}	9.6	1.1x10 ⁻²	4.8×10^{-3}

Table M.2.6–4. Doses and Resulting Health Effects to the Population Within 80 Kilometers of Idaho National Engineering Laboratory From Atmospheric Releases Associated With Normal Operation in 2030

^a Total dose to the population within 80 km from natural background radiation in the year 2030 is equal to 90,800 person-rem.

^b The storage facility contributes 7.6x10-5 person-rem/year.

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Source: HNUS 1996a.

M.2.6.1 No Action

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Atmospheric Releases and Resulting Impacts to the Public. For No Action, eight of the nine areas have radioactive releases to the atmosphere from normal operation. Table M.2.6.1-1 presents the estimated annual atmospheric radioactive releases.

Tables M.2.6–3 and M.2.6–4 include the radiological impacts to the MEI and to the offsite population within 80 km (50 mi), respectively. The maximally exposed individual would receive an annual dose of 0.018 mrem. An estimated fatal cancer risk of 4.4×10^{-7} would result from 50 years of operation. The population within 80 km (50 mi) would receive a dose of 2.4 person-rem in 2030 (mid-life of operation). An estimated 0.061 fatal cancers could result from 50 years of operation.

Liquid Releases and Resulting Impacts to the Public. There are no radioactive liquid releases to the offsite environment associated with No Action. Therefore, there are no resulting impacts.

Worker Doses and Health Effects. Based on measured values during the time period of 1989 to 1992 (*Twenty-Second Annual Report Radiation Exposures for DOE and DOE Contractor Employees–1989* [DOE/EH-0286P] and subsequent yearly data reports), the annual average radiation dose to a badged worker at INEL was calculated to be 30 mrem. It is projected that in 2005 and beyond, there would be 7,337 badged workers involved in No Action activities at INEL. The annual average radiation dose to these workers is assumed to remain at 30 mrem; the annual total radiation dose among all these workers would then equal 220 person-rem. From 50 years of operation, an estimated fatal cancer risk of 6.0×10^{-4} would result to the average worker and 4.4 fatal cancers could result among all workers.

	SMC	TA	N	TR	RA	IC	PP	WE	RF	CFA	RWMC	ANL	-W ^a
Isotope	All Releases	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	All Releases	All Releases	Monitored/ Non- monitored	
Ag-110m	0	0	0	0	0	5.1×10^{-12}	0	0	0	0	0	0	0
Am-241	0	0	0	0	5.8x10 ⁻⁷	2.4×10^{-11}	2.2×10^{-9}	0	0	0	2.0x10 ⁻⁶	0	0
Am-243	0	0	0	0	0	2.4x10 ⁻¹³	0	0	0	0	0	0	ů O
Ar-41	0	0	0	1.3x10 ³	0	0	0	0	0	0	0	17	ů
Ba-139	0	0	0	5.4×10^{-2}	0	0	0	0	0	0	0	0	ů 0
Ba-140	0	0	0	6.2x10 ⁻⁶	0	0	0	0	0	0	ů 0	2.7x10 ⁻³	0
C-14	0	0	0	0	0	9.6x10 ⁻³	0	0	0	0	0 0	0	0
Cd-113m	0	0	0	0	0	8.6x10 ⁻¹⁴	0	0	0	0	0 0	0	0
Ce-141	0	0	0	0	0	2.5x10 ⁻¹²	0	0	0	ů 0	0	0	0
Ce-144	0	0	0	0	0	3.8x10 ⁻⁶	9.2x10 ⁻⁹	0	0	0	0	0	0
Cm-244	0	0	0	0	3.0x10 ⁻⁵	0	0	0	0	0 0	ů O	0	0
Co-57	0	0	0	0	0	7.8x10 ⁻⁸	0	0	0	Õ	0	0	0
Co-58	0	0	0	8.0x10 ⁻⁷	0	2.4x10 ⁻⁷	0	0	0	0	0	8.8x10 ⁻⁶	0
Co-60	0	3.5x10 ⁻⁷	5.7x10 ⁻⁷	2.4x10 ⁻⁴	3.8x10 ⁻⁴	9.6x10 ⁻⁶	4.5x10 ⁻⁹	0	2.3x10 ⁻⁶	2.0x10 ⁻⁶	7.8x10 ⁻¹⁰	1.6x10 ⁻⁵	0
Cr-51	0	0	0	3.8x10 ⁻³	7.1x10 ⁻⁷	5.6x10 ⁻¹⁵	0	0	0	0	0	0	0
Cs-134	0	0	2.9x10 ⁻¹⁰	0	5.0x10 ⁻⁷	8.8x10 ⁻⁵	2.6x10 ⁻⁸	0	5.6x10 ⁻⁷	5.6x10 ⁻⁸	0	1.5x10 ⁻⁴	0
Cs-137	0	1.3x10 ⁻⁶	2.7x10 ⁻⁶	6.1x10 ⁻⁶	8.7x10 ⁻⁴	8.1x10 ⁻³	7.6x10 ⁻⁶	0	4.7x10 ⁻⁵	9.0x10 ⁻⁵	1.4x10 ⁻⁵	8.2x10 ⁻⁴	0
Cs-138	0	0	0	0.69	0	0	0	ů 0	0	0	0	0	0
Eu-152	0	0	0	0	7.9x10 ⁻⁷	8.7x10 ⁻⁵	1.1x10 ⁻⁶	ů 0	0 0	4.7x10 ⁻⁸	0	0	
Eu-154	0	0	4.1x10 ⁻¹⁰	0	3.7x10 ⁻⁷	6.0x10 ⁻⁵	0	ů 0	0 0	9.1x10 ⁻⁸	0	1.6x10 ⁻⁵	0 0
Eu-155	0	0	0	0	7.1x10 ⁻⁸	8.7x10 ⁻⁶	0 0	Õ	0	2.2x10 ⁻⁸	0	9.3x10 ⁻⁴	0
Fe-55	0	0	0	0	0	4.5x10 ⁻⁹	0	ů 0	0	0	0	9.3X10 0	-
Hg-203	0	0	0	8.8x10 ⁻⁵	0	0	õ	0	0	4.7x10 ⁻⁹	0		0
H-3	0	0	0	11	120	67	8.9x10 ⁻⁹	0	0	4.7210	0	0 34	0
I-129	0	0	0	0	0	9.8x10 ⁻²	3.8x10 ⁻⁸	0	0	4.4 0	0		7.9x10 ⁻²
I-131	0	0	0	1.1x10 ⁻⁴	ů 0	0	0	0	0	0	0	0	0
I-132	0	0	0	1.1x10 ⁻³	0	0	0	0	0	0	-	0	0
I-133	0	0	ů 0	4.3x10 ⁻⁴	0	0	0	0	0	0	0 0	0 0	0 0

Table M.2.6.1–1. Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Idaho National Engineering Laboratory (curies)

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	SMC	TAI	N	TRA	4	ICP	P	WEI	RF	CFA	RWMC	ANL-	Wa
Isotope	All	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	Monitored/ Non- monitored	Diffuse Area	All Releases	All Releases	Monitored/ Non- monitored	Diffuse Area
Kr-85	0	0	0	0	0	0	0	0	0	0	0	70	0
Kr-85m	0	0 0	0	8.0	0	0	0	0	0	0	0	44	0
Kr-87	ů	0	0	25	0	0	0	0	0	0	0	38	0
Kr-88	0	0	0	24	0	0	0	0	0	0	0	55	0
La-140	0	0	0	1.2x10 ⁻⁴	0	0	0	0	0	0	0	1.5x10 ⁻⁵	0
Mn-54	0	Õ	0	0	0	5.2x10 ⁻¹¹	0	0	0	0	0	5.0x10 ⁻⁵	0
Na-22	0	0	0	0	0	0	0	0	0	0	0	3.8x10 ⁻⁵	0
Na-22 Na-24	0	ů 0	0	5.2x10 ⁻³	0	0	0	0	0	0	0	1.0x10 ⁻⁷	0
Nb-94	0	0	0	0	0	1.1x10 ⁻¹⁵	0	0	0	0	0	0	0
Nb-95	0	0 0	0	0	0	4.8x10 ⁻⁷	3.1x10 ⁻⁸	0	0	0	. 0	0	0
Nb-95m	0	0	0	0	0	3.4x10 ⁻¹⁵	0	0	0	0	0	0	0
Ni-63	0	0	0	0	0	4.3x10 ⁻¹⁵	0	0	0	0	0	0	0
Np-239	0	Õ	0	0	0	3.8x10 ⁻¹⁶	1.6x10 ⁻⁸	0	0	0	0	0	0
Pa-234	2.2x10 ⁻⁸	ů 0	0	0	0	2.3x10 ⁻¹⁵	0	0	0	0	0	0	0
Pm-147	0	0	0	0	0	2.5x10 ⁻⁶	0	0	0	0	0	0	0
Pr-144	0	ů 0	Õ	0	0	4.5x10 ⁻⁶	0	0	0	0	0	. 0	0
Pr-144m	0	0	0	0	0	2.6x10 ⁻⁹	0	0	0	0	0	0	0
Pu-238	0	0	2.1x10 ⁻⁹	0	8.2x10 ⁻⁷	_	1.7x10 ⁻⁸		1.4x10 ⁻¹⁰	0	0	0	0
Pu-238 Pu-239	0	6.2x10 ⁻⁸	0	8.1x10 ⁻⁷	3.1x10 ⁻⁶		0	8.5x10 ⁻⁸	1.9x10 ⁻¹⁰	1.8x10 ⁻⁷	1.2x10 ⁻⁶		0
Pu-239 Pu-240	0	0	1.9x10 ⁻⁹	0	0	2.3x10 ⁻⁷	0	0	0	0	0	0	0
Pu-240 Pu-241	0	0	0	0	0	5.1x10 ⁻⁶	0	0	0	0	0	0	0
Rb-88	0	0	0	0.52	0	0	0	0	0	0	0	1.3x10 ⁻³	0
Rb-89	0	0	0	0.73	0	0	0	0	0	0	0	0	0
Ru-106	0	0	0	0	0	1.0x10 ⁻³	7.2x10 ⁻⁸	0	0	0	0	0	0
Sb-125	0	0	0	0	0	9.8x10 ⁻⁵	2.4x10 ⁻⁷	0	0	2.8x10 ⁻⁸	0	0	0
SD-125 Sn-199m	0	0	ů 0	0	0	2.4x10 ⁻⁸	0.0	0	0	0	0	0	0
Sn-199m Sr-90	0	2.9x10 ⁻⁷	1.9x10 ⁻⁷	3.6x10 ⁻⁴	8.6x10 ⁻⁵		9.5x10 ⁻⁹	4.1x10 ⁻⁸	6.8x10 ⁻⁶	7.8x10 ⁻⁷	3.0x10 ⁻⁸		
Sr-90 Tc-99m	0	0	0	2.2×10^{-3}		2.2x10 ⁻¹³	0	0	0	0	0	0	0

Table M.2.6.1–1. Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Idaho National Engineering Laboratory (curies)—Continued

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Health and Safety

	SMC	TA	N	TR	A	ICI	·Р	WE	RF	CFA	RWMC	ANL-	W ^a
		Monitored/		Monitored/		Monitored/		Monitored/				Monitored/	
Isotope	All Releases	Non- monitored	Diffuse Area	Non- monitored	Diffuse Area	Non- monitored	Diffuse Area	Non- monitored	Diffuse Area	All Releases	All Releases	Non- monitored	Diffuse Area
Th-228	0	0	0	0	2.0×10^{-7}	0	0	0	0	0	0	0	0
Th-230	0	0	0	0	1.0x10 ⁻⁷	0	0	0	0	0	0	0	0
Th-231	0	0	0	0	0	5.6x10 ⁻¹²	0	0	0	0	0	0	0
Th-232	0	0	0	0	9.5x10 ⁻⁹	0.0	0	0	0	0	0	0	0
Th-234	7.7x10 ⁻⁶	0	0	0	0	7.8×10^{-13}	0	0	0	0	0	0	0
U-232	0	0	0	0	1.7x10 ⁻⁷	0.0	0	0	0	0	0	0	0
U-233	0	0	0	0	0	1.2×10^{-14}	0	0	0	0	0	0	0
U-234	1.0x10 ⁻⁶	0	5.0x10 ⁻⁸	0	1.9x10 ⁻⁵	1.4x10 ⁻⁶	2.4x10 ⁻⁸	0	2.4x10 ⁻⁸	0	0	0	0
U-235	0	0	1.8x10 ⁻⁹	0	0	5.8x10 ⁻⁸	0	0	1.2x10 ⁻⁷	1.4x10 ⁻¹⁰	9.5x10 ⁻⁹	3.7x10 ⁻⁹	0
U-238	7.7x10 ⁻⁶	0	3.8x10 ⁻⁸	0	1.2x10 ⁻⁵	4.8x10 ⁻⁹	0	0	6.1x10 ⁻⁹	0	0	5.3x10 ⁻¹⁰	0
Xe-133	0	0	0	4.2	0	2.7x10 ⁻⁹	0	0	0	0	0	490	0
Xe-135	0	0	0	28	0	0	0	0	0	0	0	310	0
Xe-135m	0	0	0	14	0	0	0	0	0	0	0	11	0
Xe-138	0	0	0	71	0	0	0	0	0	0	0	22	0
Y-90	0	0	0	0.0	0	2.9x10 ⁻⁴	9.5x10 ⁻⁹	0	0	0	0	0	0
Y-91m	0	0	0	1.6x10 ⁻³	0	1.7x10 ⁻¹⁰	0	0	0	0	0	0	0
Zn-65	0	0	0	5.7x10 ⁻⁸	5.7x10 ⁻⁸	0	0	4.8x10 ⁻⁷	0	0	0	0	0
Zr-93	0	0	0	0.0	0.0	2.2x10 ⁻¹⁵	0	0	0	0	0	0	0
Zr-95	0	0	0	0.0	0.0	4.1×10^{-6}	0	0	0	0	0	0	0

Table M.2.6.1–1. Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Idaho National Engineering Laboratory (curies)—Continued

^a ANL-W reported that releases from the no action storage are not measurable. For calculation purposes, it was assumed that all the Pu released from ANL-W in 1993 was released from Pu storage. This is very conservative since EBR-II, a Pu-fueled reactor, was in operation in 1993. In October 1995, the EBR-II was defueled and is no longer in operation.
 Note: TAN=Test Area North; TRA=Test Reactor Area; WERF=Waste Experimental Reduction Facility.
 Source: IN DOE 1994c.

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M.2.6.2 Storage and Disposition

Radioactive Releases and Resulting Impacts to the Public. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the changes in impacts from the storage or disposition facilities to determine the impacts from total site operation. For example, to determine the radiological impact for the addition of an AP600 reactor at INEL, the doses from No Action facilities have to be summed with the AP600 reactor doses. Estimated annual atmospheric radioactive releases from the facilities associated with the various alternative actions are given in Section M.2.3. Tables M.2.6–3 and M.2.6–4 include the radiological impacts by alternative facility. There are no radioactive liquid releases to the offsite environment associated with any alternative action.

The annual incremental doses associated with the different alternative facilities range from 3.4×10^{-9} to 0.078 mrem to the MEI and from 5.1×10^{-8} to 9.6 person-rem to the 80-km (50-mi) population in 2030. The associated health effects from annual operations are included in both tables.

Worker Doses and Health Effects. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the incremental impacts from the storage or disposition facilities to determine the impacts from total site operations (refer to the worker discussion under No Action, above, and to Table M.2.3.2–1).

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M.2.7 RADIOLOGICAL IMPACTS AT PANTEX PLANT

This section presents the radiological impacts of the various storage and disposition alternatives at Pantex. Section M.2.7.1 presents the radiological releases and resulting impacts from facilities associated with No Action. Section M.2.7.2 presents the radiological releases and resulting impacts from the various alternatives.

For purposes of radiological impact modeling, Pantex was divided into six areas which would release radioactivity in 2005. All release points in each area were aggregated into a single release point. Table M.2.7–1 presents the characteristics of each of the release points including location, release height, and minimum distance and annual average dispersion to the site boundary in each of 16 directions. In order to calculate the maximum site boundary dose (that is, the dose ultimately incurred to the site MEI), the dose from each release point to the "maximum receptor" (that is, potential MEI) associated with each of the other release points has been calculated. For further clarification on the definition of "maximum receptor," refer to Section M.2.2.2. For example, the dose resulting from releases from Building 12-44 Cell 1 and the other storage and disposition alternatives, has been determined from the maximum receptor from the Burning Ground. Figure M.2.7–1 illustrates the location of each maximum receptor in relation to each release point. The maximum dose to one of these maximum receptors. Table M.2.7–2 presents the distance, direction, and atmospheric dispersion from each release point to each of the maximum receptors. Annual radiological releases were assumed to remain constant during the full operational period.

Descriptions of population and food stuff distributions centered on each release area are provided in a Health Risk Data report, October 1996. The joint frequency distribution used for the dose assessment was based on the meteorological measurements for 1989 from the National Weather Service at the 10-m (33-ft) height and is contained in the Health Risk Data report.

Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables and for the projected operational period in the text. Tables M.2.7–3 and M.2.7–4 include the radiological impacts to the public from atmospheric releases for the No Action and the storage and disposition alternatives.

Release Point ^a	Bldg. 12-	44 Cell 1	HE Burnir	ng Ground	Betv Zones 1		Strategic	Reserve	Immobi Fac		LWR	Site
Latitude	35° 18'		35° 20' -		35° 18' -	46.315"	35° 18'	22.415"	35° 19'	46.714"	35° 20'	25.520"
Longitude	-101° 33'		-101° 35	5' 4.249''	-101° 33'	' 53.239"	-101° 33	36.363"	-101° 34	' 14.606''	-101° 36	14.568"
Release Height	10.0) m	10.0) m	Ground	l Level	Ground	l Level	Ground	d Level	Ground	i Level
			I	Distance and	d Atmosphe	ric Dispersi	on at Site B	oundary				
Direction	Distance (m)	Chi/Q (s/m ³)										
N	5,176	1.6x10 ⁻⁷	931	1.7x10 ⁻⁶	4,482	2.1x10 ⁻⁷	5,224	1.7x10 ⁻⁷	2,614	4.8x10 ⁻⁷	1,380	1.4x10 ⁻⁶
NNE	2,790	2.4x10 ⁻⁷	950	1.0x10 ⁻⁶	4,095	1.5x10 ⁻⁷	3,315	2.1x10 ⁻⁷	2,660	2.9x10 ⁻⁷	1,406	8.1x10 ⁻⁷
NE	1,831	3.6x10 ⁻⁷	1,127	7.0x10 ⁻⁷	2,691	2.3x10 ⁻⁷	2,168	3.3x10 ⁻⁷	3,141	1.8x10 ⁻⁷	1,659	5.0x10 ⁻⁷
ENE	1,534	2.8x10 ⁻⁷	1,665	2.5x10 ⁻⁷	2,247	1.9x10 ⁻⁷	1,811	2.7x10 ⁻⁷	2,783	1.3x10 ⁻⁷	2,444	1.6x10 ⁻⁷
E	1,490	3.2x10 ⁻⁷	3,963	8.1x10 ⁻⁸	2,185	2.2x10 ⁻⁷	1,762	3.1x10 ⁻⁷	2,716	1.6x10 ⁻⁷	5,741	5.2x10 ⁻⁸
ESE	1,516	2.1x10 ⁻⁷	4,028	5.4x10 ⁻⁸	2,225	1.5x10 ⁻⁷	1,792	2.1x10 ⁻⁷	2,761	1.0x10 ⁻⁷	5,837	3.5x10 ⁻⁸
SE	1,781	2.1x10 ⁻⁷	4,719	5.6x10 ⁻⁸	2,604	1.5x10 ⁻⁷	2,091	2.1x10 ⁻⁷	3,224	1.1x10 ⁻⁷	6,827	3.6x10 ⁻⁸
SSE	2,577	9.5x10 ⁻⁸	6,942	2.4x10 ⁻⁸	3,465	7.1x10 ⁻⁸	2,625	1.1x10 ⁻⁷	4,776	4.4x10 ⁻⁸	7,587	2.3x10 ⁻⁸
S	2,607	1.8x10 ⁻⁷	7,473	4.2×10^{-8}	3,505	1.3x10 ⁻⁷	2,651	2.1x10 ⁻⁷	5,454	6.9x10 ⁻⁸	7,270	4.6x10 ⁻⁸
SSW	3,001	9.1x10 ⁻⁸	5,659	3.7x10 ⁻⁸	4,037	6.6x10 ⁻⁸	3,055	1.0x10 ⁻⁷	6,285	3.4×10^{-8}	2,333	1.5x10 ⁻⁷
sw	4,290	4.8x10 ⁻⁸	3,696	5.9x10 ⁻⁸	5,768	3.5x10 ⁻⁸	4,379	5.1x10 ⁻⁸	5,225	4.0x10 ⁻⁸	1,513	2.7×10^{-7}
wsw	5,643	4.8x10 ⁻⁸	3,083	1.1x10 ⁻⁷	4,925	6.4x10 ⁻⁸	5,366	5.6x10 ⁻⁸	4,368	7.6x10 ⁻⁸	1,257	5.3x10 ⁻⁷
w	5,495	5.0x10 ⁻⁸	2,999	1.1x10 ⁻⁷	4,795	6.5x10 ⁻⁸	5,223	5.8x10 ⁻⁸	4,254	7.8x10 ⁻⁸	1,223	5.5×10^{-7}
WNW	5,577	3.8x10 ⁻⁸	1,730	1.9x10 ⁻⁷	4,873	5.0x10 ⁻⁸	5,301	4.4x10 ⁻⁸	4,320	6.0x10 ⁻⁸	1,249	4.2×10^{-7}
NW	6,304	5.1x10 ⁻⁸	1,142	5.3x10 ⁻⁷	5,454	6.7x10 ⁻⁸	6,177	5.6x10 ⁻⁸	3,199	1.5x10 ⁻⁷	1,462	5.1x10 ⁻⁷
NNW	5,293	6.6x10 ⁻⁸	955	6.9x10 ⁻⁷	4,585	8.7x10 ⁻⁸	5,346	7.0x10 ⁻⁸	2,681	1.9x10 ⁻⁷	1,417	5.4x10 ⁻⁷

Table M.2.7–1. Release Point Characteristics, Direction, Distance, and Chi/Q at the Pantex Plant Boundary

^a See Figure M.2.7-1 for location of release points.

Source: HNUS 1996a.

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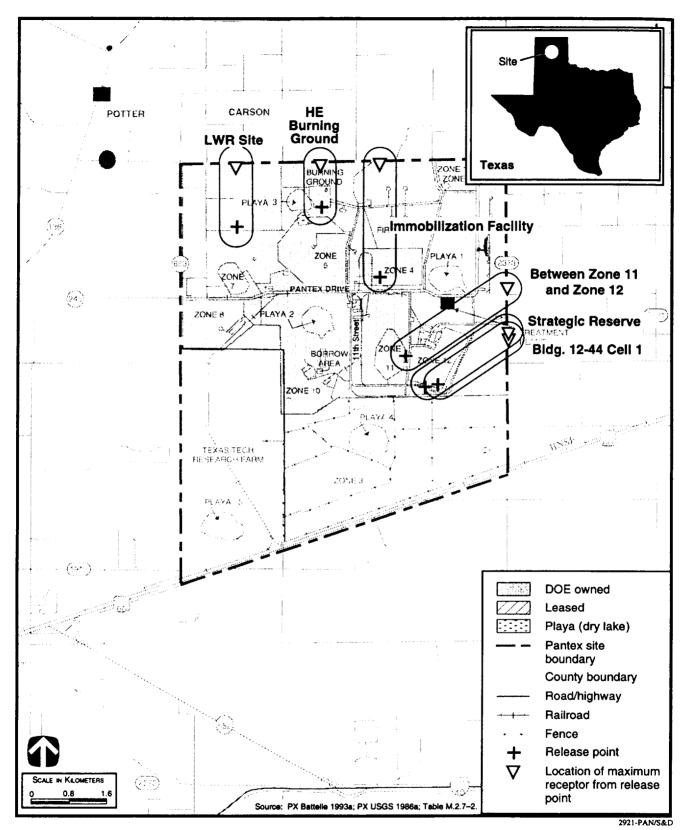


Figure M.2.7–1. Location of Release Points and Maximum Receptors at Pantex Plant.

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Maximum Decentor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
Maximum Receptor For		(111)	
Release Point: Bldg. 12-44 Cell 1	NE	1,831	3.6x10 ⁻⁷
Bldg. 12-44 Cell 1	NNW	5,728	5.9x10 ⁻⁸
HE Burning Ground	NE	2,707	2.0×10^{-7}
Between Zone 11 and 12	NE	1,922	3.3×10^{-7}
Strategic Reserve		5,315	6.5x10 ⁻⁸
Immobilization Facility	NNW		4.7×10^{-8}
LWR Site	NW	6,695	4.7210
Release Point: HE Burning Grou		5 0(0	5.0x10 ⁻⁸
Bldg. 12-44 Cell 1	SE	5,069	1.7x10 ⁻⁶
HE Burning Ground	N	932	4.7x10-8
Between Zone 11 and 12	ESE	4,424	4.7×10-8 5.2×10 ⁻⁸
Strategic Reserve	SE	4,977	5.2×10^{-7}
Immobilization Facility	NE	1,544	4.5×10^{-7}
LWR Site	WNW	2,032	1.5X10
Release Point: Between Zone 11			a a 10- ⁷
Bldg. 12-44 Cell 1	E	2,218	2.2x10 ⁻⁷ 8.1x10 ⁻⁸
HE Burning Ground	NNW	4,817	
Between Zone 11 and 12	NE	2,692	2.3x10-7
Strategic Reserve	ENE	2,248	1.9x10 ⁻⁷
Immobilization Facility	N	4,512	2.1x10 ⁻⁷
LWR Site	NW	5,722	6.3x10 ⁻⁸
Release Point: Strategic Reserve			- 7
Bldg. 12-44 Cell 1	ENE	2,085	2.1×10^{-7}
HE Burning Ground	NNW	5,661	6.4×10^{-8}
Between Zone 11 and 12	NE	2,905	2.1x10-7
Strategic Reserve	NE	2,169	3.3x10 ⁻⁷
Immobilization Facility	N	5,307	1.7×10^{-7}
LWR Site	NW	6,564	5.2x10 ⁻⁸
Release Point: Immobilization F	acility		_
Bldg. 12-44 Cell 1	ESE	3,098	8.7x10 ⁻⁸
HE Burning Ground	NNW	2,896	1.7×10^{-7}
Between Zone 11 and 12	Е	2,733	1.6x10-7
Strategic Reserve	ESE	3,029	9.0x10 ⁻⁸
Immobilization Facility	Ν	2,614	4.8×10^{-7}
LWR Site	NW	4,009	1.0×10^{-7}
Release Point: LWR Site			
Bldg. 12-44 Cell 1	ESE	6,344	3.1x10 ⁻⁸
HE Burning Ground	NE	2,243	3.1x10 ⁻⁷
Between Zone 11 and 12	ESE	5,935	3.4x10-8
Strategic Reserve	ESE	6,282	3.1x10 ⁻⁸
Immobilization Facility	ENE	3,314	1.0×10^{-7}
LWR Site	N N	1,380	1.4x10 ⁻⁶

Table M.2.7–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Pantex Plant Boundary

Source: HNUS 1996a.

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_		Dose by Pat	hway (mrem)				
Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (mrem)	Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
No Action (Total Site)	4.3x10 ⁻⁶	5.7x10 ⁻⁵	4.0x10 ⁻¹⁵	0.0	6.1x10 ⁻⁵	1.8x10 ⁻⁵	3.1x10 ⁻¹¹
Upgraded Storage Facility ^b	с	с	с	с	1.8x10 ⁻⁸	5.4x10 ⁻⁹	9.0x10 ⁻¹⁵
Upgraded Consolidated Storage Facility	9.5x10 ⁻⁶	1.7x10 ⁻⁸	3.6x10 ⁻¹⁵	7.6x10 ⁻¹²	9.5x10 ⁻⁶	2.8x10 ⁻⁶	4.7×10^{-12}
Consolidated Storage Facility	9.5x10 ⁻⁶	1.6x10 ⁻⁸	3.6x10 ⁻¹⁵	7.6x10 ⁻¹²	9.5x10 ⁻⁶	2.8x10 ⁻⁶	4.7x10 ⁻¹²
Collocated Storage Facility	9.6x10 ⁻⁶	1.7x10 ⁻⁸	3.8x10 ⁻¹⁵	1.2×10^{-11}	9.6x10 ⁻⁶	2.9×10^{-6}	4.7x10 4.8x10 ⁻¹²
Pit Disassembly/Conversion Facility	1.1x10 ⁻³	2.3×10^{-5}	6.3×10^{-12}	9.4x10 ⁻⁹	1.1x10 ⁻³	3.3×10^{-4}	5.5×10^{-10}
Pu Conversion Facility	6.9x10 ⁻⁴	1.3x10 ⁻⁶	2.9x10 ⁻¹³	6.1×10^{-10}	6.9x10 ⁻⁴	2.1×10^{-4}	3.5×10^{-10}
MOX Fuel Fabrication Facility	5.2×10^{-4}	8.9x10 ⁻⁷	2.0×10^{-13}	9.4×10^{-10}	5.2×10^{-4}	1.6x10 ⁻⁴	2.6×10^{-10}
Ceramic Immobilization Facility (Immobilization Disposition)	2.5×10^{-7}	4.3×10^{-10}	9.6x10 ⁻¹⁷	2.0×10^{-13}	2.5x10 ⁻⁷	7.5×10^{-8}	1.2×10^{-13}
Deep Borehole Complex (Direct Disposition)	4.1x10 ⁻⁸	5.9x10 ⁻¹⁰	1.5x10 ⁻¹⁶	2.4×10^{-13}	4.1x10 ⁻⁸	1.2×10^{-8}	2.1x10 ⁻¹⁴
Deep Borehole Complex (Immobilized Disposition)	5.0x10 ⁻⁸	8.8x10 ⁻¹⁰	2.3×10^{-16}	3.4x10 ⁻¹³	5.1x10 ⁻⁸	1.5x10 ⁻⁸	2.6x10 ⁻¹⁴
Vitrification Facility	9.8x10 ⁻⁵	6.7x10 ⁻⁶	1.9x10 ⁻⁹	1.1x10 ⁻⁶	1.1x10 ⁻⁴	3.3x10 ⁻⁵	5.5x10 ⁻¹¹
Ceramic Immobilization Facility (Ceramic Immobilization)	2.8×10^{-7}	1.3x10 ⁻⁶	4.0×10^{-10}	2.1×10^{-7}	1.8x10 ⁻⁶	5.4×10^{-7}	9.0×10^{-13}
Advanced Boiling Water Reactor	1.7x10 ⁻²	0.99	0.47	2.4x10 ⁻²	1.5	0.45	7.5x10 ⁻⁷
CE System 80+ Reactor	5.9×10^{-2}	1.1	2.7×10^{-2}	9.5×10^{-3}	1.5	0.36	6.0×10^{-7}
[Text deleted.]	-		2.7710	2.3710	1.4	0.50	0.0710
AP600 Reactor	3.2×10^{-2}	0.93	7.6x10 ⁻²	1.4x10 ⁻²	1.0	0.30	5.0x10 ⁻⁷
RESAR-90 Reactor	5.0x10 ⁻²	1.4	3.0×10^{-2}	1.2×10^{-2}	1.5	0.30	7.5×10^{-7}

 Table M.2.7–3.
 Doses and Resulting Health Effects to the Maximally Exposed Individual at Pantex Plant From Atmospheric Releases Associated

 With Annual Normal Operation

^a Individual annual natural background radiation dose is equal to 334 mrem.

^b For the three upgrade subaltematives including the Preferred Alternative, the dose to the MEI and the population within 80 km would decrease slightly from the No Action Alternative, although the differences are expected to be below detection limits. Therefore, the total site dose would decrease slightly but the change would be undetectable. The quantity of Pu pits at Pantex to be stored in upgraded facilities in Zone 12 would be slightly increased by the addition of RFETS pits (the Preferred Alternative) or by the addition of RFETS Pu and LANL Pu. The difference between these three subalternatives would be below detection limits. The AT-400A has both an inner container and an outer container that provides additional shielding material. The overall effect of moving Pantex and RFETS pits from Zone 4 to upgraded Zone 12 storage facilities would be lower potential releases of radioactive materials to the public, because the radiological impacts at Zone 4 would be reduced.

^c The committed effective dose equivalent for the storage facility is calculated to be 1.8x10⁻⁸ mrem based upon an analysis of measured dose. The dose shown here is for the Upgrade With RFETS Pu Pits Subalternative (Preferred Alternative). The dose for the Upgrade Without RFETS Pu or LANL Pu Subalternative would be slightly less and for the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative would be slightly greater. The differences are not measurable above background.

Note: The dose shown here is for the Upgrade with RFETS Pu Pits Subalternative (Preferred Alternative). The dose for the Upgrade Without RFETS Pu or LANL Pu Subalternative would be slightly less and for the Upgrade With All or Some RGETS Pu and LANL Pu Subalternative would be slightly greater. The differences are not measurable above background.

Source: HNUS 1996a.

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		Dose by Pathwa	ay (person-rem)				
-	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (person-rem)	Percent of Background ^a	Estimated 1-Year Fatal Cancers
Alternative/Facility	6.1x10 ⁻⁶	2.7x10 ⁻⁴	5.7x10 ⁻¹⁵	0.0	2.8x10 ⁻⁴	2.4x10 ⁻⁷	1.4x10 ⁻⁷
No Action (Total Site)	0.1X10 c	2.7X10 c	c	c	6.3x10 ⁻⁶	5.4x10 ⁻⁹	3.2x10 ⁻⁹
Upgraded Storage Facility ^b	-	5.4x10 ⁻⁷	2.1×10^{-14}	4.3x10 ⁻¹¹	5.5x10 ⁻⁵	4.7×10^{-8}	2.7x10 ⁻⁸
Upgraded Consolidated	5.5x10 ⁻⁵	5.4x10	2.1110	4.5710			_
Storage Facility	5 5 10-5	5 4. 10.7	2.0×10^{-14}	4.2x10 ⁻¹¹	5.2×10^{-5}	4.4x10 ⁻⁸	2.6x10 ⁻⁸
Consolidated Storage Facility	5.2×10^{-5}	5.4×10^{-7}	2.1×10^{-14}	6.3x10 ⁻¹¹	5.3x10 ⁻⁵	4.5x10 ⁻⁸	2.7x10 ⁻⁸
Collocated Storage Facilities	5.2×10^{-5}	5.4×10^{-7}	2.1x10 3.4x10 ⁻¹¹	5.1×10^{-8}	6.4×10^{-3}	5.5x10 ⁻⁶	3.2x10 ⁻⁶
Pit Disassembly/Conversion Facility	5.6×10^{-3}	7.5×10 ⁻⁴	3.4×10	3.4x10 ⁻⁹	3.8×10^{-3}	3.3x10 ⁻⁶	1.9x10 ⁻⁶
Plutonium Conversion Facility	3.8×10^{-3}	4.1×10^{-5}	1.6×10^{-12}		2.8×10^{-3}	2.4×10^{-6}	1.4x10 ⁻⁶
MOX Fuel Fabrication Facility	2.8×10^{-3}	2.9x10 ⁻⁵	1.1×10^{-12}	5.2×10^{-9}	6.3×10^{-7}	5.4×10^{-10}	3.1×10^{-10}
Ceramic Immobilization Facility	6.3x10 ⁻⁷	6.7x10 ⁻⁹	2.4×10^{-16}	4.9x10 ⁻¹³	0.5×10	J.4X10	5.1410
(Immobilized Disposition) Deep Borehole Complex (Direct	1.0x10 ⁻⁷	9.3x10 ⁻⁹	3.9x10 ⁻¹⁶	6.0×10^{-13}	1.1x10 ⁻⁷	9.4x10 ⁻¹¹	5.5x10 ⁻¹¹
Disposition) Deep Borehole Complex (With	1.3x10 ⁻⁷	1.4×10^{-8}	5.8x10 ⁻¹⁶	8.6x10 ⁻¹³	1.4×10^{-7}	1.2x10 ⁻¹⁰	7.0x10 ⁻¹¹
Immobilization)		. 5	4 - 10-9	2.7×10^{-6}	3.4×10^{-4}	2.9x10 ⁻⁷	1.7x10 ⁻⁷
Vitrification Facility	2.5×10^{-4}	8.7×10^{-5}	4.7×10^{-9}	5.3×10^{-7}	1.9×10^{-5}	1.6x10 ⁻⁸	9.5x10 ⁻⁹
Ceramic Immobilization Facility	7.0×10^{-7}	1.7x10 ⁻⁵	9.8x10 ⁻¹⁰	5.3×10	1.9210	1.0/10	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,
(Ceramic Immobilization)	<u>,</u>		.	1 0 10-2	8.5	7.3x10 ⁻³	4.3×10^{-3}
Advanced Boiling Water Reactor	1.5×10^{-2}	8.4	0.15	1.8×10^{-2}	8.3	7.0×10^{-3}	4.1×10^{-3}
CE System 80+ Reactor	5.1×10^{-2}	8.1	1.8×10^{-2}	8.4×10^{-3}	0.2	/.0.10	
[Text deleted.]			2		7.4	6.3×10^{-3}	3.7x10 ⁻³
AP600 Reactor	2.8×10^{-2}	7.3	5.8×10^{-2}	1.3×10^{-2}	7.4	7.6×10^{-3}	4.4×10^{-3}
RESAR-90 Reactor	4.5×10^{-2}	8.8	2.4×10^{-2}	1.1×10^{-2}	8.9	/.0X10	4.4710

Table M.2.7-4. Doses and Resulting Health Effects to the Population Within 80 Kilometers of Pantex Plant From Atmospheric Releases Associated With Normal Operation in 2030

^a Dose to the population within 80 km from natural background radiation in 2030 is equal to 116,900 person-rem.

^b For the three upgrade subalternatives including the Preferred Alternative, the dose to the MEI and the population within 80 km would decrease slightly from the No Action Alternative, although the differences are expected to be below detection limits. Therefore, the total site dose would decrease slightly but the change would be undetectable. The quantity of Pu pits at Pantex to be stored in upgraded facilities in Zone 12 would be slightly increased by the addition of RFETS pits (the Preferred Alternative) or by the addition of RFETS Pu and LANL Pu. The difference between these three subalternatives would be below detection limits. The AT-400A has both an inner container and an outer container that provides additional shielding material. The overall effect of moving Pantex and RFETS pits from Zone 4 to upgraded Zone 12 storage facilities would be lower potential releases of radioactive materials to the public, because the radiological impacts at Zone 4 would be reduced.

^c The committed effective dose equivalent for the storage facility is calculated to be 1.8x10⁻⁸ mrem based upon an analysis of measured dose. The dose shown here is for the Upgrade With RFETS Pu Pits Subalternative (Preferred Alternative). The dose for the Upgrade Without RFETS Pu or LANL Pu Subalternative would be slightly less and for the Upgrade With All or Some RFETS Pu and LANL Pu Subaltemative would be slightly greater. The differences are not measurable above background.

Note: The dose shown here is for the Upgrade with RFETS Pu Pits Subalternative (Preferred Alternative). The dose for the Upgrade Without RFETS Pu or LANL Pu Subalternative would be slightly less and for the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative would be slightly greater. The differences are not measurable above background.

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M.2.7.1 No Action

Atmospheric Releases and Resulting Impacts to the Public. For No Action, two of the areas have radioactive releases into the atmosphere from normal operation. Table M.2.7.1–1 presents the estimated annual atmospheric radioactive releases for No Action.

Table M.2.7.1–1.	Annual Atmospheric Radioactive Releases From Normal Operation of No Action at
	Pantex Plant (curies)

	Weapons Assembly/ Disassemb High Explosive				
Isotope	Bldg. 12-44 Cell 1	Burning Ground			
Tritium (H-3)	0.16	0.14			

Source: PX 1995a:1; PX DOE 1994a; PX DOE 1995d.

Tables M.2.7–3 and M.2.7–4 include the radiological impacts to the MEI and the offsite population within 80 km (50 mi), respectively. The MEI would receive an annual dose of 6.1×10^{-5} mrem. An estimated fatal cancer risk of 1.5×10^{-9} would result from 50 years of operation. The population within 80 km (50 mi) would receive a dose of 2.8×10^{-4} person-rem in 2030 (mid-life of operation). An estimated 7.0 $\times 10^{-6}$ fatal cancers could result from 50 years of operation.

Liquid Releases and Resulting Impacts to the Public. There are no radioactive liquid releases into the offsite environment associated with No Action. Therefore, there are no resulting impacts.

Worker Doses and Health Effects. Based on measured values during the time period from 1989 to 1992 (*Twenty-Second Annual Report Radiation Exposure for DOE and DOE Contractor Employees–1989*, DOE/EH-0286P) and subsequent yearly dose reports), the annual average dose to a badged worker at Pantex was calculated to be 15 mrem. It is projected that in 2005 and beyond, there would be 1,400 badged workers involved in No Action activities at Pantex (PX 1995a:1). The annual average dose to these workers was assumed to be 10 mrem; the annual total dose among all these workers would then equal 14 person-rem. From 50 years of operation, an estimated fatal cancer risk of 2.0×10^{-4} would result to the average worker and 0.28 fatal cancers could result among all workers.

M.2.7.2 Storage and Disposition

Radioactive Releases and Resulting Impacts to the Public. Total site radiological impacts during operation of storage or disposition facilities can be found by adding the impacts resulting from No Action facilities to the changes in impacts resulting from the storage or disposition facilities. For example, to determine the radiological impact for the addition of the AP600 reactor at Pantex, the No Action facilities doses have to be summed with the AP600 reactor doses. Estimated annual atmospheric radioactive releases for the different facilities are given in Section M.2.3. Tables M.2.7–3 and M.2.7–4 include the radiological impacts by alternative. There are no radioactive liquid releases into the offsite environment associated with any alternative action.

No change was reported in radioactive releases due to the upgrade of existing storage facilities for continued Pu storage at Pantex above those radioactive releases already included in No Action. Therefore, there are no changes in dose to the public from the upgrade of existing storage facilities at Pantex.

The annual doses associated with the different alternatives range from 0 to 1.5 mrem to the MEI and from 0 to 8.9 person-rem to the 80-km (50-mi) population in 2030. The associated health effects from annual operations are included in both tables.

Worker Doses and Health Effects. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the changes in impacts from the storage or disposition facilities to determine the impacts from total site operations (refer to the worker discussion under No Action, above, and to Table M.2.3.2-1).

M.2.8 RADIOLOGICAL IMPACTS AT OAK RIDGE RESERVATION

This section presents the radiological impacts of the various storage and disposition alternatives at ORR. Section M.2.8.1 presents the radiological releases and resulting impacts from facilities associated with No Action. Section M.2.8.2 presents the radiological releases and resulting impacts from the various alternatives.

For purposes of radiological impact modeling, ORR was divided into seven separate areas which would release radioactivity in 2005. All potential release points in each area were aggregated into a single release point. Tables M.2.8–1 and M.2.8–2 present the characteristics of each of the release points including location, release height, minimum distance, and annual average dispersion to the site boundary in each of 16 directions. In order to calculate the maximum site boundary dose (that is, the dose ultimately incurred to the site MEI), the dose from each release point to the "maximum receptor" (that is, potential MEI) associated with each of the other release points has been calculated. For further clarification on the definition of the "maximum receptor," refer to Section M.2.2.2. For example, the dose resulting from releases from the Oak Ridge National Laboratory (ORNL), Y–12 Plant (Y–12), High Flux Isotope Reactor Areas, and the other storage and disposition alternatives, has been determined for the maximum receptor from the K–25 Site (K–25) incinerator. Figure M.2.8–1 illustrates the location of each maximum receptor in relation to each release point. The maximum site boundary dose (that is, the dose ultimately incurred to the site MEI) is then determined by the maximum dose to one of those maximum receptors. Tables M.2.8–3 and M.2.8–4 present the distance, direction, and atmospheric dispersion from each release point to each of the maximum receptors. Annual radiological releases were assumed to remain constant during the full operational period.

Descriptions of population, foodstuffs distributions, and aquatic foods for each release area are provided in a Health Risk Data report, October 1996. The joint frequency distributions used for the dose assessment were based on 1990 meteorological measurements from five meteorological towers (Tower 1 for K-25, Tower 2 for ORNL, Tower 4 for the High Flux Isotope Reactor and Radiochemical Engineering Development Center, Tower 5 for Y-12, and Tower 6 for the proposed Tritium Supply Site location) at the 10-m (33-ft) height and are contained in the Health Risk Data report.

Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables and for the projected operational period in the text. Tables M.2.8–5 through M.2.8–8 include the radiological impacts to the public from both atmospheric release and from using the surface water for No Action and the storage and disposition alternatives.

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Release Point ^a	Immobilizat	ion Facility	K-	25	X-	10
Latitude	35°55'5'	9.139"	35°56'1	5.444"	35°55'39.169"	
Longitude	-84°20'5	5.855"	-84°22'	54.796"	-84°18'5	5.580"
Release Height	Ground Level		Ground	l Level	Ground	Level
		Distance and At	nospheric Dispersion a	t Site Boundary		
Direction	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)
N	3,200	2.2x10 ⁻⁷	3,037	1.7x10 ⁻⁷	4,218	2.1x10 ⁻⁷
NNE	2,996	5.8x10 ⁻⁷	3,919	3.2×10^{-7}	5,872	2.3x10 ⁻⁷
NE	4,624	6.2×10^7	4,360	5.0x10 ⁻⁷	8,512	2.0x10 ⁻⁷
ENE	9,494	2.9x10 ⁻⁷	4,633	4.8x10 ⁻⁷	3,935	4.4x10 ⁻⁷
E	6,806	1.5x10 ⁻⁷	9,767	1.1x10 ⁻⁷	4,337	2.3x10 ⁻⁷
ESE	6,782	1.2×10^{-7}	9,643	6.1x10 ⁻⁸	4,390	1.9x10 ⁻⁷
SE	5,900	6.9x10 ⁻⁸	4,931	1.1x10 ⁻⁷	4,029	2.5x10 ⁻⁷
SSE	3,558	6.1x10 ⁻⁸	2,313	4.0x10 ⁻⁷	4,367	2.0×10^{-7}
S	3,417	8.7x10 ⁻⁸	2,414	6.1x10 ⁻⁷	4,296	1.7x10 ⁻⁷
SSW	3,851	3.2x10 ⁻⁷	3,303	4.8x10 ⁻⁷	3,752	2.4x10 ⁻⁷
SW	2,903	1.1x10 ⁻⁶	3,897	2.6×10^{-7}	3,750	4.5x10 ⁻⁷
wsw	4,897	2.1x10 ⁻⁷	2,892	5.9x10 ⁻⁷	5,340	2.6x10 ⁻⁷
w	5,700	5.6x10 ⁻⁸	3,600	2.1×10^{-7}	8,677	↓ 4.5x10 ⁻⁸
WNW	4,299	4.7x10 ⁻⁸	2,775	1.2×10^{-7}	7,267	3.8x10 ⁻⁸
NW	4,788	3.9x10 ⁻⁸	2,374	1.3x10 ⁻⁷	4,474	8.1x10 ⁻⁸
NNW	4,767	4.7x10 ⁻⁸	1,856	2.6x10 ⁻⁷	3,900	9.4x10 ⁻⁸

Table M.2.8–1. Release Point Characteristics, Direction, Distance, and Chi/Q at the Oak Ridge Reservation Boundary (Without Presence of the Clinch River Breeder Reactor Site)

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Release Point ^a	Y-	12	MOX Fuel	Fabrication	Pit Disassemb	ly/Conversion	
Latitude	35°59'	3.409"		".676"			
Longitude	-84°15'38.488"		-84°15'43.725"		35°58'50.204" -84°16'13.244"		
Release Height	20.0 m		Ground Level		Ground Level		
		Distance and A	tmospheric Dispersion a				
	Distance	Chi/Q	Distance	Chi/Q	Distance	Chi/Q	
Direction	(m)	(s/m ³)	(m)	(s/m ³)	(m)	(s/m^3)	
N	675	7.7x10 ⁻⁷	824	1.9x10 ⁻⁶	839	2.1x10 ⁻⁶	
NNE	879	1.0x10 ⁻⁶	1,070	3.2x10 ⁻⁶	1,082	3.1x10 ⁻⁶	
NE	1,618	9.8x10 ⁻⁷	1,982	1.6x10 ⁻⁶	1,683	3.0x10 ⁻⁶	
ENE	2,360	6.6x10 ⁻⁷	2,671	8.3x10 ⁻⁷	3,396	1.3x10 ⁻⁶	
E	2,963	3.4×10^{-7}	2,765	8.4x10 ⁻⁷	2,970	5.2x10 ⁻⁷	
ESE	2,283	2.8x10 ⁻⁷	2,268	2.0x10 ⁻⁷	2,837	4.4×10^{-7}	
SE	2,329	2.1×10^{-7}	3,663	5.3x10 ⁻⁸	3,719	1.4×10^{-7}	
SSE	3,726	1.3x10 ⁻⁷	3,570	1.2×10^{-7}	4,276	4.6x10 ⁻⁸	
S	4,682	1.5x10 ⁻⁷	4,432	8.9x10 ⁻⁸	4,100	6.6x10 ⁻⁸	
SSW	9,589	7.2x10 ⁻⁸	9,563	5.7x10 ⁻⁸	10,586	7.5x10 ⁻⁸	
SW	11,872	3.8x10 ⁻⁸	11,602	1.6x10 ⁻⁷	10,901	1.7x10 ⁻⁷	
WSW	3,454	2.4×10^{-7}	3,733	7.3x10 ⁻⁷	3,306	3.8x10 ⁻⁷	
W	1,082	5.3x10 ⁻⁷	1,370	1.2×10^{-6}	1,349	5.7x10 ⁻⁷	
WNW	810	4.8x10 ⁻⁷	974	6.4x10 ⁻⁷	921	6.4x10 ⁻⁷	
NW	688	5.2×10^{-7}	862	6.6x10 ⁻⁷	801	7.8x10 ⁻⁷	
NNW	619	7.1x10 ⁻⁷	798	9.1x10 ⁻⁷	772	1.1x10 ⁻⁶	

^a See Figure M.2.8–1 for location of release points. Source: HNUS 1996a

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Release Point ^a	LWR	Site	Immobilizat	tion Facility	K-	and the second se	X-	
atitude	35°54'	9.137"	35°55'5	9.139"	35°56'1		35°55'3	
Longitude	-84°22'4		-84°20':	55.855"	-84°22'		-84°18':	
Release Height	Ground	i Level	Ground		Ground		Ground	
		D	istance and Atmo	spheric Dispersio	n at Site Bounda	гу		
	Distance	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)
Direction	(m)	2.5x10 ⁻⁶	3,199	2.2x10 ⁻⁷	3,041	1.7x10 ⁻⁷	4,206	2.1x10 ⁻⁷
N	930	2.5×10^{-6}	2,995	5.8x10 ⁻⁷	3,936	3.2x10 ⁻⁷	5,852	2.3×10^{-7}
NNE	1,209	2.7×10^{-7}	4,646	6.2x10 ⁷	4,362	5.0x10 ⁻⁷	8,512	2.0×10^{-7}
NE	8,444	2.0×10^{-8}	9,893	2.7×10^{-7}	4,634	4.8x10 ⁻⁷	5,162	2.9x10 ⁻⁷
ENE	11,141	9.5×10^{-7}	7,827	1.2×10^{-7}	10,817	9.6x10 ⁻⁸	4,863	2.0×10^{-7}
E	2,171	2.5×10^{-6}	7,133	1.1×10^{-7}	9,987	5.8x10 ⁻⁸	4,707	1.7x10 ⁻⁷
ESE	898	3.4×10^{-6}	6,077	6.7x10 ⁻⁸	5,089	1.0x10 ⁻⁷	4,385	2.2x10 ⁻⁷
SE	830	2.3×10^{-6}	4,081	4.9x10 ⁻⁸	2,306	4.0x10 ⁻⁷	4,586	1.9x10 ⁻⁷
SSE	979	5.0×10^{-7}	3,788	7.5x10 ⁻⁸	2,418	6.1×10^{-7}	4,483	1.6x10 ⁻⁷
S	2,154	7.2×10^{-7}	4,000	3.0x10 ⁻⁷	3,436	4.5x10 ⁻⁷	3,956	2.2x10 ⁻⁷
SSW	1,863	3.9x10 ⁻⁶	2,903	1.1×10^{-6}	3,897	2.6x10 ⁻⁷	4,134	3.9x10 ⁻⁷
SW	998		5,279	1.9×10^{-7}	2,892	5.9x10 ⁻⁷	5,340	2.6x10 ⁻⁷
WSW	897	4.5×10^{-6}	5,700	5.6x10 ⁻⁸	3,618	2.1x10 ⁻⁷	8,677	4.5x10 ⁻⁸
W	939	1.6×10^{-6}	3,700 4,294	4.7x10 ⁻⁸	2,782	1.2x10 ⁻⁷	7,259	3.8x10 ⁻⁸
WNW	854	1.2×10^{-6}	4,294 4,787	3.9x10 ⁻⁸	2,355	1.3x10 ⁻⁷	4,460	8.1x10 ⁻⁸
NW NNW	755 764	1.5x10 ⁻⁶ 1.4x10 ⁻⁶	4,787 4,769	4.7x10 ⁻⁸	1,855	2.6x10 ⁻⁷	3,900	9.4x10 ⁻⁸

Table M.2.8–2. Release Point Characteristics, Direction, Distance, and Chi/Q at the Oak Ridge Reservation Boundary (With Presence of the Clinch River Breeder Reactor Site)

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Release Point ^a	Y	-12	MOX Fuel Fabrication Pit Disassembly/Conv				
Latitude	35°59'8.409" -84°15'38.488"		35°59'.676"		Pit Disassembly/Conversion 35°58'50.204"		
Longitude							
Release Height	20 m		-84°15'43.725" Ground Level		-84°16'13.244"		
Bit					Ground Level		
	Distance		Distance and Atmospheric Dispersion at Site Boundary				
Direction	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q	
N	657	7.9x10 ⁻⁷	821	1.9x10 ⁻⁶	826	$\frac{(s/m^3)}{2 1 + 10^{-6}}$	
NNE	897	1.0x10 ⁻⁶	1,087	3.1×10^{-6}	1,067	2.1×10^{-6}	
NE	1,639	9.7x10 ⁻⁷	2,000	1.5×10^{-6}	1,658	3.1x10 ⁻⁶	
ENE	2,344	6.6×10^{-7}	2,658	8.3x10 ⁻⁷	3,380	3.0x10 ⁻⁶	
E	2,936	3.5×10^{-7}	2,772	8.4x10 ⁻⁷		1.3×10^{-6}	
ESE	2,286	2.8x10 ⁻⁷	2,273	2.0×10^{-7}	2,978	5.2x10 ⁻⁷	
SE	2,320	2.1x10 ⁻⁷	4,125	4.4×10^{-8}	2,844	4.4×10^{-7}	
SSE	4,229	1.1x10 ⁻⁷	4,085	9.5×10^{-8}	4,241	1.1x10 ⁻⁷	
5	5,423	1.3×10^{-7}	5,197	9.3×10^{-8}	5,014	3.6x10 ⁻⁸	
SSW	11,713	5.5x10 ⁻⁸	11,444		5,193	4.6x10 ⁻⁸	
SW	12,181	3.7x10 ⁻⁸	11,898	4.4×10^{-8}	10,902	7.2x10 ⁻⁸	
wsw	3,433	2.4×10^{-7}		1.5×10^{-7}	11,310	1.6x10 ⁻⁷	
N .	1,067	5.3×10^{-7}	3,712	7.4x10 ⁻⁷	3,330	3.8x10 ⁻⁷	
WNW	803	4.9×10^{-7}	1,353	1.2×10^{-6}	1,327	5.9x10 ⁻⁷	
W	687	4.9×10^{-7}	963	6.6x10 ⁻⁷	911	6.5×10^{-7}	
NNW	621		868	6.5x10 ⁻⁷	795	8.0x10 ⁻⁷	
See Figure M 2.8.1		7.1x10 ⁻⁷	805	9.0x10 ⁻⁷	773	1.1x10 ⁻⁶	

Table M.2.8–2. Release Point Characteristics, Direction, Distance, and Chi/Q at the Oak Ridge Reservation Boundary (With Presence of the Clinch River Breeder Reactor Site)—Continued

^a See Figure M.2.8–1 for location of release points. Source: HNUS 1996a.

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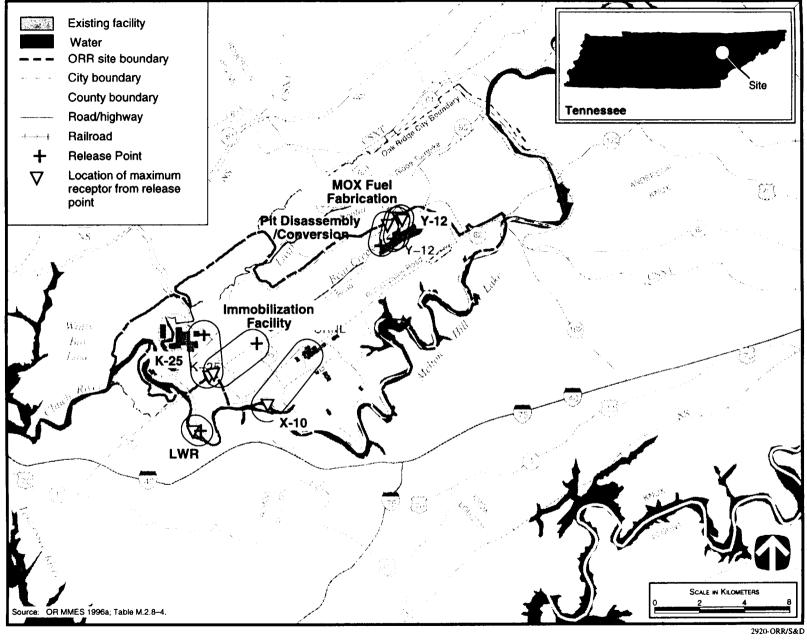


Figure M.2.8–1. Location of Release Points and Maximum Receptors at Oak Ridge Reservation.

Table M.2.8-3.	Direction, Distance, and Meteorological Dispersion to Various Maximum
Indiv	idual Receptors at the Oak Ridge Reservation Site Boundary
(₩	ithout Presence of the Clinch River Breeder Reactor Site)

Maximum Receptor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
Release Point: Immobilization		(111)	(s/m [*])
Immobilization Facility	SW	2,903	1.1x10 ⁻⁶
K-25	SW	3,189	9.7x10 ⁻⁷
X –10	SSE	3,582	6.0x10 ⁻⁸
Y-12	NE	10,548	1.9x10 ⁻⁷
MOX Fuel Fabrication	NE	10,449	1.9x10 ⁻⁷
Pit Disassembly/Conversion	NE	9,699	2.1×10^{-7}
Release Point: K-25		7,077	2.1110
Immobilization Facility	SSE	2,315	4.0x10 ⁻⁷
K25	S	2,415	4.0x10 6.1x10 ⁻⁷
X-10	SE	5,421	9.3x10 ⁻⁸
Y-12	ENE	12,739	1.1x10 ⁻⁷
MOX Fuel Fabrication	ENE	12,639	1.1x10 ⁻⁷
Pit Disassembly/Conversion	ENE	11,863	1.2×10^{-7}
Release Point: X-10		11,005	1.2810
Immobilization Facility	WSW	5,468	2.5×10^{-7}
K-25	WSW	5,735	2.3×10^{-7}
X-10	SW	3,750	4.5×10^{-7}
Y-12	NNE	8,933	1.2×10^{-7}
MOX Fuel Fabrication	NNE	8,842	1.3×10^{-7}
Pit Disassembly/Conversion	NNE	8,184	1.3×10^{-7}
Release Point: Y–12		0,101	1.4×10
Immobilization Facility	SW	12,769	3.4x10 ⁻⁸
K-25	SW	13,055	3.3×10^{-8}
X-10	SW	11,875	3.8x10 ⁻⁸
Y-12	NNE	879	1.0×10^{-6}
MOX Fuel Fabrication	N	812	6.4×10^{-7}
Pit Disassembly/Conversion	NW	772	4.5×10^{-7}

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Maximum Receptor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
Release Point: MOX Fuel Fabrication Facility		•	
Immobilization Facility	SW	12,523	1.4×10^{-7}
K-25	SW	12,809	1.4×10^{-7}
X-10	SW	11,606	1.6x10 ⁻⁷
Y-12	NN	1,143	2.8x10 ⁻⁶
MOX Fuel Fabrication	NNE	1,071	3.1x10 ⁻⁶
Pit Disassembly/Conversion	NW	863	6.6x10 ⁻⁷
Release Point: Pit Disassembly/ Conversion			
Immobilization Facility	SW	11,735	1.5x10 ⁻⁷
K-25	SW	12,021	1.5x10 ⁻⁷
X-10	SW	10,902	1.7x10 ⁻⁷
Y-12	NE	1,776	2.7x10 ⁻⁶
MOX Fuel Fabrication	NE	1,683	3.0x10 ⁻⁶
Pit Disassembly/Conversion	NNE	1,083	3.1×10^{-6}

Table M.2.8–3. Direction, Distance, and Meteorological Dispersion to Various MaximumIndividual Receptors at the Oak Ridge Reservation Site Boundary(Without Presence of the Clinch River Breeder Reactor Site)—Continued

Source: HNUS 1996a.

	Direction	Distance	Atmospheric Dispersion Chi/Q	
Maximum Receptor For		(m)	(s/m ³)	
Release Point: LWR Site				
LWR Site	WSW	897	4.5×10^{-6}	
Immobilization Facility	NNE	1,728	1.5x10 ⁻⁶	
K–25	Ν	1,518	1.1x10 ⁻⁶	
X–10	E	3,420	3.4×10^{-7}	
Y-12	NE	14,878	9.2x10 ⁻⁸	
MOX Fuel Fabrication	NE	14,780	9.2x10 ⁻⁸	
Pit Disassembly/Conversion	NE	13,996	1.0x10 ⁻⁷	
Release Point: Immobilization				
LWR Site	SSW	4,244	2.8x10 ⁻⁷	
Immobilization Facility	SW	2,903	1.1x10 ⁻⁶	
K-25	SW	3,198	9.7x10 ⁻⁷	
X–10	S	4,027	6.8x10 ⁻⁸	
Y-12	NE	10,571	1.9x10 ⁻⁷	
MOX Fuel Fabrication	NE	10,472	1.9x10 ⁻⁷	
Pit Disassembly/Conversion	NE	9,674	2.2x10 ⁻⁷	
Release Point: K–25 TSCA Incinerator				
LWR Site	S	4,244	2.6x10 ⁻⁷	
Immobilization Facility	SSE	2,306	4.0×10^{-7}	
K-25	S	2,419	6.1x10 ⁻⁷	
X-10	SE	5,747	8.5x10 ⁻⁸	
Y-12	ENE	12,761	1.1x10 ⁻⁷	
MOX Fuel Fabrication	ENE	12,663	1.1x10 ⁻⁷	
Pit Disassembly/Conversion	ENE	11,836	1.3x10 ⁻⁷	
Release Point: X–10 (ORNL)				
LWR Site	WSW	7,297	1.6x10 ⁻⁷	
Immobilization Facility	WSW	5,471	2.5×10^{-7}	
K-25	WSW	5,743	2.3x10 ⁻⁷	
X-10	SW	4,135	3.9x10 ⁻⁷	
Y-12	NNE	8,956	1.2x10 ⁻⁷	
MOX Fuel Fabrication	NNE	8,863	1.3x10 ⁻⁷	
Pit Disassembly/Conversion	NNE	8,163	1.4x10 ⁻⁷	

Table M.2.8-4.Direction, Distance, and Meteorological Dispersion to Various MaximumIndividual Receptors at the Oak Ridge Reservation Site Boundary
(With Presence of the Clinch River Breeder Reactor Site)

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	Direction	Distance	Atmospheric Dispersion Chi/Q	
Maximum Receptor For		(m)	(s/m ³)	
Release Point: Y-12			a a 10-8	
LWR Site	SW	14,978	2.8x10 ⁻⁸	
Immobilization Facility	SW	12,769	3.4x10 ⁻⁸	
K-25	SW	13,064	3.3x10 ⁻⁸	
X-10	SW	12,259	3.6x10 ⁻⁸	
Y-12	NNE	898	1.0×10^{-6}	
MOX Fuel Fabrication	Ν	827	6.2×10^{-7}	
Pit Disassembly/Conversion	NW	785	4.4×10^{-7}	
Release Point: MOX Fuel Fabrication Facility			-	
LWR Site	SW	14,726	1.1x10 ⁻⁷	
Immobilization Facility	SW	12,523	1.4×10^{-7}	
K-25	SW	12,818	1.4×10^{-7}	
X-10	SW	11,989	1.5x10 ⁻⁷	
Y-12	NNE	1,163	2.7x10 ⁻⁶	
MOX Fuel Fabrication	NNE	1,087	3.1x10 ⁻⁶	
Pit Disassembly/Conversion	NW	868	6.5x10 ⁻⁷	
Release Point: Pit Disassembly/ Conversion			-	
LWR Site	SW	13,950	1.2×10^{-7}	
Immobilization Facility	SW	11,735	1.5×10^{-7}	
K-25	SW	12,030	1.5×10^{-7}	
X-10	SSW	11,293	6.9x10 ⁻⁷	
Y-12	NE	1,798	2.7×10^{-6}	
MOX Fuel Fabrication	NE	1,705	2.9x10 ⁻⁶	
Pit Disassembly/Conversion	NNE	1,067	3.1x10 ⁻⁶	

Table M.2.8-4. Direction, Distance, and Meteorological Dispersion to Various MaximumIndividual Receptors at the Oak Ridge Reservation Site Boundary(With Presence of the Clinch River Breeder Reactor Site)—Continued

Source: HNUS 1996a.

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		Dose by Pat	hway (mrem)				
Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (mrem)	Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
No Action (Total Site)	1.4	1.7x10 ⁻²	3.2x10 ⁻²	5.3x10 ⁻⁴	1.5	5.0x10 ⁻¹	7.4x10 ⁻⁷
Upgrade HEU Storage	2.2x10 ⁻⁷	5.3x10 ⁻¹⁰	8.8x10 ⁻¹⁶	1.7x10 ⁻¹¹	2.2×10^{-7}	7.5x10 ⁻⁸	1.1x10 ⁻¹³
[Text deleted.]							
Collocated Storage Facility	4.4x10 ⁻⁵	8.0x10 ⁻⁸	1.8x10 ⁻¹⁴	5.5x10 ⁻¹¹	4.5x10 ⁻⁵	1.5x10 ⁻⁵	2.3x10 ⁻¹¹
Pit Disassembly/Conversion Facility	1.3x10 ⁻²	3.0x10 ⁻⁴	7.9x10 ⁻¹¹	1.2×10^{-7}	1.4x10 ⁻²	4.7x10 ⁻³	7.0x10 ⁻⁹
Pu Conversion Facility	9.1x10 ⁻³	1.7x10 ⁻⁵	3.9x10 ⁻¹²	8.2x10 ⁻⁸	9.2x10 ⁻³	3.1x10 ⁻³	4.6x10 ⁻⁹
MOX Fuel Fabrication Facility	6.8x10 ⁻³	1.2x10 ⁻⁵	2.6x10 ⁻¹²	1.2x10 ⁻⁸	6.8x10 ⁻³	2.3x10 ⁻³	3.4x10 ⁻⁹
Ceramic Immobilization Facility (Immobilized Disposition)	5.9x10 ⁻⁷	1.0x10 ⁻⁹	2.2×10^{-16}	4.6x10 ⁻¹³	5.9x10 ⁻⁷	2.0x10 ⁻⁷	3.0x10 ⁻¹³
Deep Borehole Complex (Direct Disposition)	9.3x10 ⁻⁸	1.4x10 ⁻⁹	3.7x10 ⁻¹⁶	5.6x10 ⁻¹³	9.4x10 ⁻⁸	3.2x10 ⁻⁸	4.7x10 ⁻¹⁴
Deep Borehole Complex (Immobilized Disposition)	1.2x10 ⁻⁷	2.0x10 ⁻⁹	5.4x10 ⁻¹⁶	7.9x10 ⁻¹³	1.2x10 ⁻⁷	4.1x10 ⁻⁸	6.0x10 ⁻¹⁴
Vitrification Facility	2.3x10 ⁻⁴	1.5x10 ⁻⁵	4.4x10 ⁻⁹	2.5x10 ⁻⁶	2.5x10 ⁻⁴	8.5x10 ⁻⁵	1.3x10 ⁻¹⁰
Ceramic Immobilization Facility (Ceramic Immobilization)	6.5x10 ⁻⁷	3.0x10 ⁻⁶	9.1x10 ⁻¹⁰	4.9x10 ⁻⁷	4.2x10 ⁻⁶	1.4x10 ⁻⁶	2.1x10 ⁻¹²
Advanced Boiling Water Reactor	4.9x10 ⁻²	3.2	1.5	6.7x10 ⁻²	4.8	1.6	2.4x10 ⁻⁶
CE System 80+ Reactor	1.1x10 ⁻¹	3.1	8.9x10 ⁻²	1.4x10 ⁻³	3.3	1.1	1.7x10 ⁻⁶
[Text deleted.]							
AP600 Reactor	2.5x10 ⁻²	2.1	2.5x10 ⁻¹	1.5x10 ⁻²	2.3	7.8x10 ⁻¹	1.2x10 ⁻⁶
RESAR-90 Reactor	9.0x10 ⁻²	3.2	1.0x10 ⁻¹	1.7x10 ⁻²	3.5	1.2	1.8x10 ⁻⁶

 Table M.2.8–5.
 Doses and Resulting Health Effects to the Maximally Exposed Individual at Oak Ridge Reservation From Atmospheric Releases

 Associated With Annual Normal Operation

^a Individual annual natural background radiation dose is equal to 295 mrem.

[Text deleted.]

Source: HNUS 1996a.

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		Dose by Pathw	ay (person-rem)				
- Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent in 2030 (person-rem)	Percent of Background ^a	Estimated 1-Year Fatal Cancers
No Action (Total Site)	26	0.41	2.3	5.7x10 ⁻²	29	7.7x10 ⁻³	1.5x10 ⁻²
Upgrade HEU Storage	3.4x10 ⁻⁶	8.0x10 ⁻¹⁰	1.4x10 ⁻¹⁴	2.7×10^{-10}	3.4x10 ⁻⁶	9.0x 10 ⁻¹⁰	1.7x10 ⁻⁹
[Text deleted.]							
Collocated Storage Facilities	8.7x10 ⁻⁴	1.4x10 ⁻⁷	3.5x10 ⁻¹³	1.0x10 ⁻⁹	8.7x10 ⁻⁴	2.3x10 ⁻⁷	4.4x10 ⁻⁷
Pit Disassembly/Conversion Facility	0.12	1.7x10 ⁻⁴	6.9x10 ⁻¹⁰	1.0x10 ⁻⁶	0.12	3.2x10 ⁻⁵	6.0x10 ⁻⁵
Pu Conversion Facility	7.4×10^{-2}	9.3x10 ⁻⁶	3.2x10 ⁻¹¹	6.6x10 ⁻⁸	7.4x10 ⁻²	2.0x10 ⁻⁵	3.7x10 ⁻⁵
MOX Fuel Fabrication Facility	4.8×10^{-2}	6.3x10 ⁻⁶	1.9x10 ⁻¹¹	9.1x10 ⁻⁸	4.8x10 ⁻²	1.3x10 ⁻⁵	2.4x10 ⁻⁵
Ceramic Immobilization Facility (Immobilized Disposition)	1.1x10 ⁻⁵	1.7x10 ⁻⁹	4.2×10^{-15}	8.5x10 ⁻¹²	1.1x10 ⁻⁵	2.9x10 ⁻⁹	5.5x10 ⁻⁹
Deep Borehole Complex (Direct Disposition)	1.8x10 ⁻⁶	2.3x10 ⁻⁹	6.9x10 ⁻¹⁵	1.1x10 ⁻¹¹	1.8x10 ⁻⁶	4.7x10 ⁻¹⁰	9.0x10 ⁻¹⁰
Deep Borehole Complex (Immobilized Disposition)	2.2x10 ⁻⁶	3.4x10 ⁻⁹	1.0x10 ⁻¹⁴	1.5x10 ⁻¹¹	2.2x10 ⁻⁶	5.8x10 ⁻¹⁰	1.1x10 ⁻⁹
Vitrification Facility	4.3x10 ⁻³	5.0x10 ⁻⁵	8.7x10 ⁻⁸	4.7x10 ⁻⁵	4.4×10^{-3}	1.2x10 ⁻⁶	2.2x10 ⁻⁶
Ceramic Immobilization Facility (Ceramic Immobilization)	1.2x10 ⁻⁵	9.8x10 ⁻⁶	1.7x10 ⁻⁸	9.6x10 ⁻⁶	3.2x10 ⁻⁵	8.4x10 ⁻⁹	1.6x10 ⁻⁸
Advanced Boiling Water Reactor	0.16	3.1	1.7	0.19	5.1	1.3x10 ⁻³	2.6×10^{-3}
CE System 80+ Reactor	0.39	2.8	0.21	4.9x10 ⁻³	3.4	9.0x10 ⁻⁴	1.7x10 ⁻³
[Text deleted.]							
AP600 Reactor	8.7x10 ⁻²	1.9	0.69	5.2×10^{-2}	2.8	7.4x10 ⁻⁴	1.4×10^{-3}
RESAR-90 Reactor	0.31	3.0	0.29	5.8x10 ⁻²	3.6	9.5x10 ⁻⁴	1.8x10 ⁻³

Table M.2.8–6.Doses and Resulting Health Effects to the Population Within 80 Kilometers of Oak Ridge Reservation From Atmospheric ReleasesAssociated With Normal Operation in 2030

^a Dose to the population within 80 km from natural background radiation in year 2030 is equal to 379,000 person-rem.

[Text deleted.]

Source: HNUS 1996a.

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			Dose by Path	nway (mrem)	· · · · · · · · · · · · · · · · · · ·				
Alternative/ Facility	Fish Ingestion	Other Food Ingestion	Drinking Water	Boating	Swimming	Shoreline	(mrem)	Percent of Background ^a	Estimated Risk of 1-Year Fatal Cancers
No Action (Total Site)	1.6	1.0x10 ⁻¹	1.2×10^{-2}	1.5×10^{-5}	3.0x10 ⁻⁵	6.1x10 ⁻³	1.7	5.8x10 ⁻¹	8.6x10 ⁻⁷
Advanced Boiling Water Reactor	2.7x10 ⁻²	1.0x10 ⁻³	3.2x10 ⁻²	2.6x10 ⁻⁶	5.3x10 ⁻⁶	2.9x10 ⁻⁴	6.0x10 ⁻²	2.0x10 ⁻²	3.0x10 ⁻⁸
CE System 80+ Reactor	9.0x10 ⁻²	6.7×10^{-3}	2.0×10^{-1}	5.1x10 ⁻⁶	1.0x10 ⁻⁵	8.0x10 ⁻⁴	3.0x10 ⁻¹	1.0x10 ⁻¹	1.5x10 ⁻⁷
[Text deleted.]									
AP600 Reactor	1.0x10 ⁻¹	1.1×10^{-2}	3.6x10 ⁻¹	5.7x10 ⁻⁶	1.1x10 ⁻⁵	8.5x10 ⁻⁴	4.7x10 ⁻¹	1.6x10 ⁻¹	2.4x10 ⁻⁷
RESAR-90 Reactor	8.0×10^{-2}	1.4×10^{-2}	4.5x10 ⁻¹	5.7x10 ⁻⁶	1.1x10 ⁻⁵	4.9×10^{-4}	5.4x10 ⁻¹	1.8x10 ⁻¹	2.4×10^{-7}

Table M.2.8–7. Doses and Resulting Health Effects to the Maximally Exposed Individual at Oak Ridge Reservation From Liquid Releases Associated With Annual Normal Operation

^a Individual annual natural background radiation dose is equal to 295 mrem.

Source: HNUS 1996a.

		D	ose by Pathwa	ay (person-rem)				Background ^a	Estimated 1-Year Fatal Cancers
Alternative/ Facility	Fish Ingestion	Other Food Ingestion	Drinking Water	Boating	Swimming	Shoreline	Committed Effective Dose Equivalent (person-rem)		
No Action (Total Site)	2.3	2.3	0	6.8x10 ⁻⁴	5.8x10 ⁻⁴	4.8x10 ⁻²	4.7	1.2×10^{-3}	2.3x10 ⁻³
Advanced Boiling Water Reactor	5.2x10 ⁻²	2.3x10 ⁻²	0	1.2x10 ⁻⁴	1.1x10 ⁻⁴	2.3x10 ⁻³	7.8x10 ⁻²	2.1x10 ⁻⁵	3.9x10 ⁻⁵
CE System 80+ Reactor	1.4x10 ⁻¹	1.5x10 ⁻¹	0	2.4×10^{-4}	2.1x10 ⁻⁴	6.4×10^{-3}	3.0x10 ⁻¹	7.9x10 ⁻⁵	1.5x10 ⁻⁴
[Text deleted.]			0						
AP600 Reactor	2.3x10 ⁻¹	2.6x10 ⁻¹	0	2.7x10 ⁻⁴	2.3x10 ⁻⁴	6.8x10 ⁻³	5.0x10 ⁻¹	1.3x10 ⁻⁴	2.5x10 ⁻⁴
RESAR-90 Reactor	1.5x10 ⁻¹	3.2x10 ⁻¹	0	2.6x10 ⁻⁴	2.3x10 ⁻⁴	3.9x10 ⁻³	4.8x10 ⁻¹	1.3x10 ⁻⁴	2.4x10 ⁻⁴

Table M.2.8–8. Doses and Resulting Health Effects to the Population Downstream of Oak Ridge Reservation From Liquid Releases Associated With Normal Operation in 2030

^a Total dose to the population within 80 km from natural background radiation in year 2030 is equal to 379,000 person-rem. Source: HNUS 1996a.

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Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables and for the projected operational period in the test.

M.2.8.1 No Action

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Atmospheric Releases and Resulting Impacts to the Public. For No Action, three of the five areas have radioactive releases into the atmosphere from normal operation. Table M.2.8.1–1 presents the estimated annual atmospheric radioactive releases.

Tables M.2.8–5 and M.2.8–6 include the atmospheric radiological impacts to the maximally exposed member of the public and the offsite population within 80 km (50 mi), respectively. The maximally exposed individual would receive an annual dose of 1.5 mrem. An estimated fatal cancer risk of 3.7×10^{-5} would result from 50 years of operation. The population within 80 km (50 mi) would receive a dose of 29 person-rem in 2030 (midlife of operation). An estimated 0.73 fatal cancers could result from 50 years of operation.

Liquid Releases and Resulting Impacts to the Public. For No Action, two of the five areas have radioactive releases to the offsite surface water from normal operation. Table M.2.8.1–2 presents the estimated annual liquid radioactive releases.

Tables M.2.8–7 and M.2.8–8 include the radiological impacts to the maximally exposed individual and the offsite populations using surface water within 80 km (50 mi) downstream of ORR, respectively. The maximally exposed member of the public would receive an annual dose of 1.7 mrem. An estimated fatal cancer risk of 4.3×10^{-5} would result from 50 years of operation. The population would receive a dose of 4.7 person-rem in 2030. An estimated 0.12 fatal cancers could result from 50 years of operation.

Worker Doses and Health Effects. Based on measured values during 1991 and 1992 (Dose Reports for 1991 and 1992), it is estimated that the average dose to a badged worker involved in No Action activities at ORR in 2005 and beyond would equal 2.6 mrem. It is projected that in 2005 and beyond, there would be 17,215 badged workers involved in No Action activities. The annual dose among all these workers would equal 44 person-rem. From 50 years of operation, an estimated fatal cancer risk of 5.2×10^{-5} would result to the average worker and 0.88 fatal cancers could result among all workers.

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Isotope	K-25	X-10	Y–12	No Action HEU Storage ²
H-3	0	2.4×10^2	0	0
Be-7	0	3.8x10 ⁻⁴	0	0
K-40	4.0×10^{-2}	0	0	0
Ar-41	0	1.8x10 ³	0	0
Co-57	1.2x10 ⁻⁴	0	0	0
Co-60	4.4×10^{-3}	2.6x10 ⁻⁶	0	0
Sr-90	0	3.8x10 ⁻⁴	0	0
Tc-99	0.12	0	0	0
Ru-106	4.5×10^{-3}	0	0	0
Cd-109	7.6×10^{-3}	0	0	0
I-129	0	2.5x10 ⁻⁴	0	0
I-129	ů 0	5.5x10 ⁻⁵	0	0
I-130	0	5.3×10^{-2}	0	0
I-131	ů 0	0.93	0	0
I-132	ů	0.20	0	0
I-135	ů 0	0.47	0	0
Xe-135	0	5.0x10 ¹	0	0
Xe-138	0	7.1×10^{1}	0	0
Cs-134	0	5.2×10^{-7}	0	0
Cs-137	5.0x10 ⁻³	5.1x10 ⁻⁴	0	0
Cs-138	0	7.1x10 ¹	0	0
Ba-140	0	4.9x10 ⁻⁴	0	0
Ce-141	2.0×10^{-4}	0	0	0
Eu-152	0	1.7x10 ⁻⁶	0	0
Eu-154	0	2.5x10 ⁻⁶	0	0
Eu-155	0	5.2x10 ⁻⁶	0	0
Os-191	0	0.17	0	0
Pb-212	0	0.37	0	0
Th-228	3.8x10 ⁻⁴	1.5x10 ⁻⁶	0	0
Th-230	5.9x10 ⁻⁵	5.7x10 ⁻⁸	0	0
Th-232	1.1x10 ⁻⁴	3.3x10 ⁻⁸	0	0
Th-234	1.8x10 ⁻²	0	0	0
U-234	4.0×10^{-3}	8.7x10 ⁻⁶	4.7x10 ⁻²	4.7x10 ⁻⁵
U-235	1.8x10 ⁻⁴	4.7x10 ⁻⁷	1.5x10 ⁻³	1.5x10 ⁻⁶
U-236	0	0	1.9x10 ⁻⁴	1.9x10 ⁻⁷
U-238	4.2x10 ⁻³	2.8x10 ⁻⁵	6.5x10 ⁻³	6.5x10 ⁻⁶
Np-237	5.7x10 ⁻⁴	0	0	0
Pu-238	2.5×10^{-4}	2.8x10 ⁻⁶	0	0
Pu-239	5.7x10 ⁻⁵	8.0x10 ⁻⁶	0	0
Am-241	0	4.6x10 ⁻⁶	0	0
Cm-244	0	7.3x10 ⁻⁵	0	0

 Table M.2.8.1–1.
 Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Oak

 Ridge Reservation (curies)

^a No Action HEU storage release is assumed equal to 0.001 of Y-12 releases. Source: OR DOE 1994c.

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 Isotope	K-25	X-10	
 Н-3	0	1.8x10 ⁻³	
K-40	0.019	0	
Co-60	0	0.55	
Sr-90	0	6.7	
Tc-99	0.030	0	
Ru-106	0.038	0	
Cs-137	1.2×10^{-3}	0.018	
Ce-143	0.20	0.040	
Th-228	0.20	0	
Th-230	2.4×10^{-5}	0	
Th-234	0.036	0	
U-234	7.7×10^{-3}	9.5x10 ⁻⁴	
U-235	0.014	0.056	
U-236	5.8×10^{-4}	0	
U-238	6.0×10^{-3}	4.5	
Np-237	1.2×10^{-3}	0	
Pu-238	1.6x10 ⁻⁴	0	

Table M.2.8.1–2. Annual Liquid Releases From Normal Operation of No Action at Oak Ridge Reservation (curies)

Source: OR DOE 1994c.

M.2.8.2 Storage and Disposition

Atmospheric Releases and Resulting Impacts to the Public. Total site radiological impacts during operation of storage or disposition facilities can be found by adding the impacts resulting from No Action facilities to the incremental impacts resulting from storage or disposition facilities. For example, to determine the radiological impact for the addition of the AP600 reactor at ORR, the No Action facilities doses would be summed with the AP600 reactor doses. Estimated annual atmospheric radioactive releases for the storage and disposition facilities are given in Section M.2.3. Tables M.2.8–5 and M.2.8–6 present the atmospheric radiological impacts by alternative facility.

The annual dose associated with the different alternative facilities range from 9.4×10^{-8} to 4.8 mrem to the maximally exposed member of the public and from 1.8×10^{-6} to 5.1 person-rem to the 80-km (50 mi) population in the year 2030. The associated health effects from annual operations are included in both tables.

Liquid Releases and Resulting Impacts to the Public. There are two disposition technologies that would release liquid discharges to the surface water surrounding ORR. These are the large and small evolutionary Advanced LWRs. The liquid releases for these technologies are given in Section M.2.3. As an example of determining the total site liquid radiological impact associated with the addition of an AP600 reactor at ORR, the No Action liquid doses must be summed with the AP600 reactor liquid doses. Tables M.2.8–7 and M.2.8–8 present the liquid radiological impacts for the applicable alternative facilities.

No change was reported in liquid radioactive releases due to the upgraded or new HEU storage facilities for continued HEU storage at ORR above those radioactive releases already included in No Action. Therefore, there are no changes in dose to the public from the upgraded or new HEU storage facilities at ORR.

The annual incremental doses associated with the different LWR's that have liquid releases range from 0.060 to 0.54 mrem to the maximally exposed member of the public, and range from 0.078 to 0.50 person-rem to the downstream population in 2030. The associated health effects from annual operations are included in both tables.

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Worker Doses and Health Effects. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the changes in impacts from the storage or disposition facilities to determine the impacts from total site operations, refer to the worker discussion under No Action, above, and to Table M.2.3.2–1).

M.2.9 RADIOLOGICAL IMPACTS AT SAVANNAH RIVER SITE

This section presents the radiological impacts of the various storage and disposition alternatives at SRS. Section M.2.9.1 presents the radiological releases and resulting impacts from facilities associated with No Action. Section M.2.9.2 presents the radiological releases and resulting impacts from the various alternatives.

For purposes of radiological impact modeling, SRS was divided into thirteen separate areas which would release radioactivity in 2005. All potential release points in each area were aggregated into a single release point. Table M.2.9–1 presents the characteristics of each of the release points including location, release height, and minimum distance and annual average dispersion to the site boundary in each of 16 directions. In order to calculate the maximum site boundary dose (that is, the dose ultimately incurred to the site MEI), the dose from each release point to the "maximum receptor" (that is, potential MEI) associated with each of the other release points has been calculated. For example, the dose resulting from releases from F-, H-, S-Areas, the K- and L-Reactors and other storage and disposition alternatives, has been determined for the maximum receptor from the Savannah River Technology Center Laboratory in A-Area. Figure M.2.9–1 illustrates the location of each maximum receptor in relation to each release point. The maximum dose to one of these maximum receptors. Table M.2.9–2 presents the direction, distance, and atmospheric dispersion from each release point to each of the maximum receptor," refer to Section M.2.2.2. Annual radiological releases were assumed to remain constant during the full operational period.

Descriptions of population and foodstuffs distributions centered on each release area are provided in a Health Risk Data report, October, 1996. The joint frequency distribution used for the dose assessment was based on the meteorological measurements for 1985 from the meteorological tower at SRS at the 61-m (201-ft) height and is contained in the Health Risk Data report.

Doses given in this section are associated with 1 year of operation because regulatory standards are given as annual limits. The health effects are presented on an annual basis in the tables and for the projected operational period in the text. Tables M.2.9-3 through M.2.9-6 include the radiological impacts to the public from both atmospheric releases and from using the surface water for No Action and the storage and disposition alternatives.

Release Point ^a	A-A	rea	HEU S	torage	C-A	rea	D-A		LWR		F-A		MOX F 33°14' 3	
Latitude	33°20' 2	4.303"	33°17' 4	4.436"	33°14' 5	59.126"	33°12' 1		33°15' 2		33°17' 1			
Longitude	-81°44'	6.652"	-81°37'	3.675"	-81°40' (37.760"	-81°44'	14.929"	-81°38'		-81°40'		-81°34' -	
Release Height	31	m	10		61		16		10		61	m	10	m
				Dista	ance and A	tmospher	ic Dispersi	on at Site	Boundary					
Direction	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)										
N	1,895	7.3×10^{-7}	11,484	2.1×10^{-7}	14,591	3.6x10 ⁻⁸	14,804	1.2×10^{-7}	15,162	1.5x10 ⁻⁷	10,898	4.8x10 ⁻⁸	17,092	1.2×10^{-7}
NNE	3,252	5.4x10 ⁻⁷	11,609	2.5×10^{-7}	17,178	3.7x10 ⁻⁸	20,525	9.3x10 ⁻⁸	16,006	1.6x10 ⁻⁷	12,665	5.0x10 ⁻⁸	15,559	1.7×10^{-7}
NE	5,443	3.4x10 ⁻⁷	13,248	2.4x10 ⁻⁷	20,171	3.3x10 ⁻⁸	25,502	8.1x10 ⁻⁸	17,442	1.7x10 ⁻⁷	14,770	4.5x10 ⁻⁸	12,020	2.7×10^{-7}
	12,398	1.5x10 ⁻⁷	13,622	2.6x10 ⁻⁷	18,137	4.6x10 ⁻⁸		1.1x10 ⁻⁷	14,346	2.4x10 ⁻⁷	18,525	4.5x10 ⁻⁸	8,089	5.3x10 ⁻⁷
ENE		8.7x10 ⁻⁸		3.4×10^{-7}	16,523	5.7x10 ⁻⁸	21,665	1.3x10 ⁻⁷	12,854	3.2x10 ⁻⁷	17,118	5.5x10 ⁻⁸	7,520	6.7x10 ⁻⁷
E	21,471	5.4×10^{-8}		2.5×10^{-7}	17,942	3.7×10^{-8}		1.3x10 ⁻⁷		1.8x10 ⁻⁷		4.0x10 ⁻⁸	9,794	3.3x10 ⁻⁷
ESE	23,860	2.6×10^{-8}		1.0x10 ⁻⁷	15,532	2.2x10 ⁻⁸	14,573	8.8x10 ⁻⁸		1.0x10 ⁻⁷		1.7x10 ⁻⁸	10,298	1.8x10 ⁻⁷
SE	27,210			5.6x10 ⁻⁸		1.7x10 ⁻⁸		1.1x10 ⁻⁷		7.2x10 ⁻⁸		1.4x10 ⁻⁸	10,942	1.1x10 ⁻⁷
SSE	25,918	1.8x10 ⁻⁸		5.5×10^{-8}		1.6x10 ⁻⁸		1.7x10 ⁻⁷		7.1x10 ⁻⁸		1.3x10 ⁻⁸	11,773	9.8x10 ⁻⁸
S	14,851	3.5x10 ⁻⁸		6.5×10^{-8}		2.8x10 ⁻⁸		3.4x10 ⁻⁷		9.1x10 ⁻⁸		2.4x10 ⁻⁸	13,372	1.2x10 ⁻⁷
SSW	7,325	1.2x10 ⁻⁷		1.4×10^{-7}	9,329	6.9x10 ⁻⁸		1.4x10 ⁻⁶		1.7x10 ⁻⁷		5.6x10 ⁻⁸	17,355	1.3x10 ⁻⁷
SW	5,305	3.1x10 ⁻⁷		_	9,329	7.7x10 ⁻⁸		2.3x10 ⁻⁶		2.4x10 ⁻⁷		7.4x10 ⁻⁸	17,206	1.6x10 ⁻⁷
WSW	3,421	5.8x10 ⁻⁷		1.9×10^{-7}	9,272	5.7x10 ⁻⁸		1.6x10 ⁻⁶		1.9x10 ⁻⁷		6.0x10 ⁻⁸		1.3x10 ⁻⁷
W	2,580	6.0x10 ⁻⁷	14,818	1.6×10^{-7}		4.7×10^{-8}		9.8x10 ⁻⁷		1.4x10 ⁻⁷		4.6x10 ⁻⁸		8.9x10 ⁻⁸
WNW	1,743	6.8x10 ⁻⁷		1.5×10^{-7}		-		1.8×10^{-7}		1.0x10 ⁻⁷		3.6x10 ⁻⁸		7.1x10 ⁻⁸
NW	1,603	5.6x10 ⁻⁷		1.3x10 ⁻⁷		2.9x10 ⁻⁸		1.9x10 ⁻⁷		1.0x10 ⁻⁷		3.5x10 ⁻⁸		7.7x10 ⁻⁸
NNW	1,385	6.3x10 ⁻⁷	11,505	1.4x10 ⁻⁷	12,763	2.7x10 ⁻⁸	7,897	1.9710	14,070	1.0110	,,,, <u>,</u>	0.00	,	

Table M.2.9–1.Release Point Characteristics, Direction, Distance, and Atmospheric Dispersion
at the Savannah River Site Boundary

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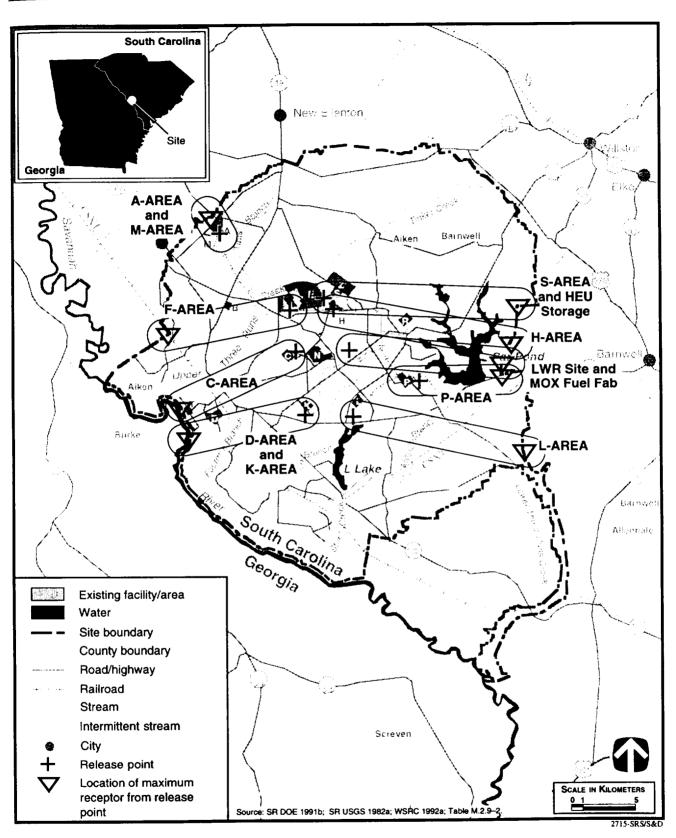
				at the Savannah River Site Boundary—Continued								
Release Point ^a	H-A	rea	K-A	rea	L-A	rea	M-A	Area	P-A	rea	S-A	rea
Latitude	33°17'	10.880"	33°12' 4	33°12' 42.145"		38.484"	33°20'	17.321"	33°13' 42.293"		33°17' 42.592"	
Longitude	-81°38'	25.118"	-81°39'	49.356"	-81°37'	26.480"	-81°44'	15.593"	-81°34'	53.420"		34.989"
Release Height	61	m	61	m	61	m	10	m	61	m	10	m
			Dista	nce and A	tmospher	ic Dispersi	on at Site	Boundary	,			
Direction	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)	Distance (m)	Chi/Q (s/m ³)
N	12,288	4.3x10 ⁻⁸	19,103	2.7x10 ⁻⁸	20,707	2.5x10 ⁻⁸	1,764	2.9x10 ⁻⁶	18,709	2.8x10 ⁻⁸	11,294	2.2x10 ⁻⁷
NNE	12,852	4.9x10 ⁻⁸	21,410	2.9x10 ⁻⁸	21,148	2.9x10 ⁻⁸	2,980	1.7x10 ⁻⁶	16,156	3.9x10 ⁻⁸	11,975	2.4×10^{-7}
NE	14,883	4.5x10 ⁻⁸	21,710	3.1x10 ⁻⁸	15,504	4.3x10 ⁻⁸	5,744	7.6x10 ⁻⁷	10,712	6.2x10 ⁻⁸	14,232	2.2x10 ⁻⁷
ENE	15,959	5.2x10 ⁻⁸	15,635	5.4x10 ⁻⁸	12,053	7.0x10 ⁻⁸	12,796	2.8x10 ⁻⁷	7,832	1.0x10 ⁻⁷	15,664	2.2x10 ⁻⁷
Ε	14,047	6.7x10 ⁻⁸	15,628	6.0x10 ⁻⁸	13,327	7.1x10 ⁻⁸	21,924	1.6x10 ⁻⁷	7,757	1.2x10 ⁻⁷	14,622	2.7x10 ⁻⁷
ESE	13,688	4.9x10 ⁻⁸	13,430	5.0x10 ⁻⁸	11,163	6.1x10 ⁻⁸	24,035	9.8x10 ⁻⁸	9,846	6.9x10 ⁻⁸	14,219	2.0x10 ⁻⁷
SE	17,629	2.0x10 ⁻⁸	11,432	3.0x10 ⁻⁸	9,888	3.5x10 ⁻⁸	26,982	4.9x10 ⁻⁸	9,253	3.7x10 ⁻⁸	18,437	8.1x10 ⁻⁸
SSE	17,662	1.5x10 ⁻⁸	10,837	2.5x10 ⁻⁸	9,295	2.9x10 ⁻⁸	25,603	3.4x10 ⁻⁸	9,658	2.8x10 ⁻⁸	18,667	5.1x10 ⁻⁸
S	18,109	1.3x10 ⁻⁸	11,120	2.1x10 ⁻⁸	9,588	2.5x10 ⁻⁸	14,346	7.5x10 ⁻⁸	10,160	2.4x10 ⁻⁸	19,114	5.1x10 ⁻⁸
SSW	18,481	2.0x10 ⁻⁸	10,680	3.5x10 ⁻⁸	12,155	3.1x10 ⁻⁸	7,012	2.8x10 ⁻⁷	11,769	3.2x10 ⁻⁸	19,045	7.3x10 ⁻⁸
SW	14,355	4.4×10^{-8}	10,612	6.1x10 ⁻⁸	12,500	5.1x10 ⁻⁸	5,099	7.2x10 ⁻⁷	15,824	4.0x10 ⁻⁸	14,549	1.7x10 ⁻⁷
WSW	14,212	5.0x10 ⁻⁸	9,142	7.8x10 ⁻⁸	13,517	5.3x10 ⁻⁸	3,289	1.6x10 ⁻⁶	16,741	4.2x10 ⁻⁸	12,874	2.4x10 ⁻⁷
W	12,763	4.4x10 ⁻⁸	8,855	6.3x10 ⁻⁸	12,507	4.5x10 ⁻⁸	2,500	1.9x10 ⁻⁶	16,724	3.3x10 ⁻⁸	12,465	2.0x10 ⁻⁷
WNW	12,643	3.6x10 ⁻⁸	12,325	3.6x10 ⁻⁸	15,669	2.8x10 ⁻⁸	2,277	1.7x10 ⁻⁶	18,799	2.3x10 ⁻⁸	11,487	1.7x10 ⁻⁷
NW	11,889	2.9x10 ⁻⁸	13,275	2.5x10 ⁻⁸	17,079	1.9x10 ⁻⁸	1,659	2.1x10 ⁻⁶	20,240	1.6x10 ⁻⁸	10,979	1.5x10 ⁻⁷
NNW	11,749	3.0x10 ⁻⁸	17,092	2.0x10 ⁻⁸	19,328	1.8x10 ⁻⁸	1,485	2.5x10 ⁻⁶	19,686	1.7x10 ⁻⁸	10,740	1.5x10 ⁻⁷

 Table M.2.9–1.
 Release Point Characteristics, Direction, Distance, and Atmospheric Dispersion at the Savannah River Site Boundary—Continued

^a See Figure M.2.9–1 for location of release points. Source: HNUS 1996a.

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Figure M.2.9–1. Location of Release Points and Maximum Receptors at Savannah River Site.

Maximum Receptor For	Direction	Distance (m)	Atmospheric Dispersion Chi/Q (s/m ³)
Release Point: A-Area		(11)	(8/111)
A-Area and M-Area	N	1,896	7.3x10 ⁻⁷
HEU Storage and S-Area	ESE	23,860	5.4x10 ⁻⁸
C-Area	SSW	14,089	5.6x10 ⁻⁸
D-Area and K-Area	S	15,711	3.3×10^{-8}
LWR Site and MOX Fuel Fab	ESE	24,185	5.3x10 ⁻⁸
F-Area	SSW	8,869	9.9x10 ⁻⁸
H-Area	ESE	24,206	5.3x10 ⁻⁸
L-Area	SE	24,200	2.4×10^{-8}
P-Area	ESE	28,504	5.2×10^{-8}
Release Point: HEU Storage	LSL	24,007	5.2210
A-Area and M-Area	WNW	13,188	1.5x10 ⁻⁷
HEU Storage and S-Area	E	12,267	3.4x10 ⁻⁷
C-Area	wsw	16,571	1.7x10 ⁻⁷
D-Area and K-Area	SW	16,844	1.4×10^{-7}
LWR Site and MOX Fuel Fab	ESE	12,193	2.4×10^{-7}
F-Area	W	15,195	1.6×10^{-7}
H-Area	ESE	12,254	2.4×10^{-7}
L-Area	SE	16,820	9.1x10 ⁻⁸
P-Area	ESE	12,637	2.3×10^{-7}
Release Point: C-Area	LSL	12,007	2.5×10
A-Area and M-Area	NNW	13,204	2.6×10^{-8}
HEU Storage and S-Area	ENE	18,313	4.5×10^{-8}
C-Area	WSW	9,273	7.7x10 ⁻⁸
D-Area and K-Area	SW	9,345	6.9x10 ⁻⁸
LWR Site and MOX Fuel Fab	E	16,526	5.7x10 ⁻⁸
F-Area	WNW	9,583	4.7×10^{-8}
H-Area	E	17,287	5.4x10 ⁻⁸
L-Area	ESE	19,141	3.5x10 ⁻⁸
P-Area	E	16,599	5.6x10 ⁻⁸
Release Point: D-Area	2	10,000	5.000
A-Area and M-Area	Ν	16,816	1.0x10 ⁻⁷
HEU Storage and S-Area	ENE	25,191	9.4x10 ⁻⁸
C-Area	WNW	3,112	8.0x10 ⁻⁷
D-Area and K-Area	WSW	1,991	2.3×10^{-6}
LWR Site and MOX Fuel Fab	ENE	22,651	1.1x10 ⁻⁷
F-Area	NNW	7,949	1.8x10 ⁻⁷
H-Area	ENE	23,721	1.0x10 ⁻⁷
L-Area	E	23,820	1.1x10 ⁻⁷
P-Area	Ē	22,520	1.2×10^{-7}
Release Point: LWR Site	2	22,720	1.2010
A-Area and M-Area	NW	14,555	1.0x10 ⁻⁷
HEU Storage and S-Area	ENE	14,510	2.4×10^{-7}
C-Area	wsw	13,026	2.3×10^{-7}
D-Area and K-Area	wsw	12,917	2.4×10^{-7}

Table M.2.9–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Savannah River Site Boundary

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Table M.2.9–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Savannah River Site Boundary—Continued

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	Direction	Distance	Atmospheric Dispersion Chi/Q
Maximum Receptor For		(m)	(s/m ³)
LWR Site and MOX Fuel Fab	E	12,855	3.2x10 ⁻⁷
F-Area	W	13,125	1.9x10 ⁻⁷
H-Area	Е	13,531	3.0x10 ⁻⁷
L-Area	ESE	16,000	1.7x10 ⁻⁷
P-Area	Е	12,995	3.2×10^{-7}
Release Point: F-Area			
A-Area and M-Area	NW	9,759	3.5x10 ⁻⁸
HEU Storage and S-Area	Е	17,703	5.3x10 ⁻⁸
C-Area	SW	11,589	5.5x10 ⁻⁸
D-Area and K-Area	SW	12,260	5.2×10^{-8}
LWR Site and MOX Fuel Fab	ESE	16,985	4.0×10^{-8}
F-Area	WSW	9,646	7.4×10^{-8}
H-Area	E	17,349	5.4x10 ⁻⁸
L-Area	ESE	20,708	3.2x10 ⁻⁸
P-Area	ESE	17,266	3.9x10 ⁻⁸
Release Point: MOX Fuel Fab			
A-Area and M-Area	NW	19,432	6.9x10 ⁻⁸
HEU Storage and S-Area	ENE	10,158	3.9x10 ⁻⁷
C-Area	W	17,750	1.3x10 ⁻⁷
D-Area and K-Area	WSW	17,210	1.6×10^{-7}
LWR Site and MOX Fuel Fab	E	7,538	6.7×10^{-7}
F-Area	W	18,577	1.2×10^{-7}
Н-Агеа	ENE	7,502	5.9×10^{-7}
L-Area	ESE	10,573	3.0×10^{-7}
P-Area	E	7,565	6.6×10^{-7}
Release Point: H-Area			
A-Area and M-Area	NW	12,076	2.8×10^{-8}
HEU Storage and S-Area	E	14,356	6.6x10 ⁻⁸
C-Area	WSW	14,239	5.0x10 ⁻⁸
D-Area and K-Area	SW	14,567	4.3x10 ⁻⁸
LWR Site and MOX Fuel Fab	ESE	13,766	4.9x10 ⁻⁸
F-Area	W	12,939	4.3x10 ⁻⁸
H-Area	E	14,047	6.7×10^{-8}
L-Area	SE	17,852	1.9×10^{-8}
P-Area	ESE	14,102	4.8×10^{-8}
Release Point: K-Area			-
A-Area and M-Area	NNW	17,560	2.0x10 ⁻⁸
HEU Storage and S-Area	ENE	18,629	4.5x10 ⁻⁸
C-Area	W	9,755	5.8x10 ⁻⁸
D-Area and K-Area	W	8,871	6.3x10 ⁻⁸
LWR Site and MOX Fuel Fab	ENE	15,793	5.3x10 ⁻⁸
F-Area	WNW	12,336	3.6x10 ⁻⁸
H-Area	ENE	16,942	4.9x10 ⁻⁸
L-Area	E	17,014	5.5x10 ⁻⁸
P-Area	Е	15,629	6.0x10 ⁻⁸

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		-	Atmospheric Dispersion
	Direction	Distance	Chi/Q
Maximum Receptor For		(m)	(s/m ³)
Release Point: L-Area			9
A-Area and M-Area	NNW	19,433	1.8x10 ⁻⁸
HEU Storage and S-Area	NE	15,504	4.3x10 ⁻⁸
C-Area	W	13,455	4.2×10^{-8}
D-Area and K-Area	W	12,529	4.5x10 ⁻⁸
LWR Site and MOX Fuel Fab	ENE	12,290	6.8x10 ⁻⁸
F-Area	WNW	15,677	2.8x10 ⁻⁸
H-Area	ENE	13,557	6.2×10^{-8}
L-Area	Ε	13,327	7.1x10 ⁻⁸
P-Area	ENE	12,058	7.0x10 ⁻⁸
Release Point: M-Area			
A-Area and M-Area	Ν	2,078	2.3x10 ⁻⁶
HEU Storage and S-Area	ESE	24,035	9.8x10 ⁻⁸
C-Area	SSW	13,829	1.1x10 ⁻⁷
D-Area and K-Area	S	15,468	6.8x10 ⁻⁸
LWR Site and MOX Fuel Fab	ESE	24,305	9.6x10 ⁻⁸
F-Area	SSW	8,574	2.2×10^{-7}
H-Area	ESE	24,347	9.6x10 ⁻⁸
L-Area	SE	28,576	4.5x10 ⁻⁸
P-Area	ESE	24,719	9.4x10 ⁻⁸
Release Point: P-Area		,	
A-Area and M-Area	NW	20,454	1.6×10^{-8}
HEU Storage and S-Area	NE	11,137	6.0×10^{-8}
C-Area	W	17,456	3.2×10^{-8}
D-Area and K-Area	W	16,737	3.3x10 ⁻⁸
LWR Site and MOX Fuel Fab	ENE	7,915	1.0x10 ⁻⁷
F-Area	WNW	18,800	2.3×10^{-8}
H-Area	ENE	9,137	9.1x10 ⁻⁸
L-Area	ESE	9,953	6.8x10 ⁻⁸
P-Area	E	7,758	1.2×10^{-7}
Release Point: S-Area	-	.,	
A-Area and M-Area	NW	11,264	1.4x10 ⁻⁷
HEU Storage and S-Area	E	14,623	2.7×10^{-7}
C-Area	sw	14,591	1.7x10 ⁻⁷
D-Area and K-Area	SW	15,046	1.6x10 ⁻⁷
LWR Site and MOX Fuel Fab	ESE	14,336	1.9x10 ⁻⁷
F-Area	WSW	12,875	2.4×10^{-7}
H-Area	ESE	14,510	1.9x10 ⁻⁷
L-Area	S	18,625	8.0x10 ⁻⁸
P-Area	ESE	14,719	1.9×10^{-7}
ource: HNUS 1996a.		14,/17	1.7210

Table M.2.9–2. Direction, Distance, and Meteorological Dispersion to Various Maximum Individual Receptors at the Savannah River Site Boundary—Continued

Source: HNUS 1996a.

			Pathway rem)				
- Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (mrem)	Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
No Action (Total Site)	4.4x10 ⁻²	3.9x10 ⁻¹	1.1x10 ⁻⁷	3.6x10 ⁻⁵	4.2x10 ⁻¹	1.4x10 ⁻¹	2.1x10 ⁻⁷
Upgraded Storage Facility ^b	c	с	с	c	6.2x10 ⁻⁶	2.2x10 ⁻⁶	3.1x10 ⁻¹²
Consolidated Storage Facility	1.4x10 ⁻⁵	2.5x10 ⁻⁸	5.5x10 ⁻¹⁵	1.1x10 ⁻¹¹	1.4x10 ⁻⁵	4.7x10 ⁻⁶	7.0x10 ⁻¹²
	1.4x10 ⁻⁵	2.4×10^{-8}	5.7×10^{-15}	1.7x10 ⁻¹¹	1.4x10 ⁻⁵	4.7x10 ⁻⁶	7.0x10 ⁻¹²
Collocated Storage Facilities	1.6x10 ⁻³	3.5x10 ⁻⁵	9.3x10 ⁻¹²	1.4x10 ⁻⁸	1.6x10 ⁻³	5.4x10 ⁻⁴	8.0x10 ⁻¹⁰
Pit Disassembly/Conversion Facility	1.0x10 ⁻³	1.9x10 ⁻⁶	4.4×10^{-13}	9.1x10 ⁻¹⁰	1.0x10 ⁻³	3.4x10 ⁻⁴	5.0x10 ⁻¹⁰
Pu Conversion Facility	1.5×10^{-3}	2.5×10^{-6}	5.6x10 ⁻¹³	2.7x10 ⁻⁹	1.5x10 ⁻³	5.0x10 ⁻⁴	7.5x10 ⁻¹⁰
MOX Fuel Fabrication Facility Ceramic Immobilization Facility (Immobilized Disposition)	1.8x10 ⁻⁷	3.2×10^{-10}	7.0x10 ⁻¹⁷	1.4×10^{-13}	1.8x10 ⁻⁷	6.0x10 ⁻⁸	9.0x10 ⁻¹⁴
(Direct Disposition)	2.7x10 ⁻⁸	3.9x10 ⁻¹⁰	1.0x10 ⁻¹⁶	1.6x10 ⁻¹³	2.8x10 ⁻⁸	9.4x10 ⁻⁹	1.4x10 ⁻¹⁴
(Direct Disposition) Deep Borehole Complex (Immobilized Disposition)	3.4x10 ⁻⁸	5.9x10 ⁻¹⁰	1.5x10 ⁻¹⁶	2.3x10 ⁻¹³	3.4×10^{-8}	1.1x10 ⁻⁸	1.7x10 ⁻¹⁴
Vitrification Facility	7.1x10 ⁻⁵	4.9x10 ⁻⁶	1.4x10 ⁻⁹	7.7x10 ⁻⁷	7.7x10 ⁻⁵	2.6x10 ⁻⁵	3.9x10 ⁻¹¹
Ceramic Immobilization Facility (Ceramic Immobilization)	2.0×10^{-7}	9.5x10 ⁻⁷	2.8x10 ⁻¹⁰	1.5x10 ⁻⁷	1.3x10 ⁻⁶	4.4x10 ⁻⁷	6.5x10 ⁻¹³
Advanced Boiling Water Reactor	3.1x10 ⁻³	2.3×10^{-1}	2.9×10^{-2}	3.7×10^{-3}	2.6x10 ⁻¹	8.7x10 ⁻²	1.3x10 ⁻⁷
CE System 80+ Reactor	8.1x10 ⁻³	2.0x10 ⁻¹	4.2×10^{-3}	1.0x10 ⁻⁴	2.1x10 ⁻¹	7.0×10^{-2}	1.1x10 ⁻⁷
[Text deleted.]							2
AP600 Reactor	1.8x10 ⁻³	1.5x10 ⁻¹	1.4×10^{-2}	1.1x10 ⁻³	1.6x10 ⁻¹	5.4×10^{-2}	8.0x10 ⁻⁸
RESAR-90 Reactor	6.5x10 ⁻³	2.1x10 ⁻¹	5.9x10 ⁻³	1.2×10^{-3}	2.3×10^{-1}	7.7x10 ⁻²	1.2x10 ⁻⁷

 Table M.2.9–3.
 Doses and Resulting Health Effects to the Maximally Exposed Individual From Atmospheric Releases Associated With Annual

 Normal Operation at Savannah River Site

^a Individual annual natural background radiation dose is equal to 298 mrem.

^b Dose and health effect results are based on a capacity of 5,000 Pu storage positions in the APSF (SR DOE 1995e). Because the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative and the Upgrade With RFETS Non-Pit Pu Subalternative both call for fewer than 5,000 Pu storage positions in the APSF, dose and health effects for these two subalternatives would be less. The dose shown here is for the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative. The dose for the Upgrade With RFETS Non-Pit Pu Subalternative would be slightly less, and would be below detection limits.

^c Number reflected as a component in the Committed Effected Dose Equivalent.

Source: HNUS 1996a.

-		•	Pathway on-rem)			Background ^a	Estimated 1-Year Fatal Cancer
Alternative/Facility	Inhalation	Ingestion	Plume Immersion	Ground Shine	Committed Effective Dose Equivalent (person-rem)		
No Action (Total Site)	3.9	3.6×10^{1}	2.5x10 ⁻⁵	5.1x10 ⁻³	40x10 ¹	1.5x10 ⁻²	2.0x10 ⁻²
Upgraded Storage Facility ^b	c	c	c	c	2.9x10 ⁻⁴	1.1x10 ⁻⁷	1.5×10^{-7}
Consolidated Storage Facility	9.2x10 ⁻⁴	2.6x10 ⁻⁶	3.6x10 ⁻¹³	7.4x10 ⁻¹⁰	9.2x10 ⁻⁴	3.5x10 ⁻⁷	4.6x10 ⁻⁷
Collocated Storage Facilities	8.8x10 ⁻⁴	2.7x10 ⁻⁶	3.6x10 ⁻¹³	1.1x10 ⁻⁹	8.8x10 ⁻⁴	3.3x10 ⁻⁷	4.4x10 ⁻⁷
Pit Disassembly/Conversion Facility	0.10	3.6x10 ⁻³	6.2x10 ⁻¹⁰	9.1x10 ⁻⁷	0.11	4.1x10 ⁻⁵	5.5x10 ⁻⁵
Pu Conversion Facility	6.6x10 ⁻²	2.0x10 ⁻⁴	2.8x10 ⁻¹¹	5.9x10 ⁻⁸	6.6x10 ⁻²	2.5×10^{-5}	3.3x10 ⁻⁵
MOX Fuel Fabrication Facility	4.4×10^{-2}	1.6x10 ⁻⁴	1.6x10 ⁻¹¹	7.8x10 ⁻⁸	4.4×10^{-2}	1.7x10 ⁻⁵	2.2x10 ⁻⁵
Ceramic Immobilization Facility (Immobilized Disposition)	1.2×10^{-5}	3.2x10 ⁻⁸	4.6x10 ⁻¹⁵	9.2x10 ⁻¹²	1.2×10^{-5}	4.5x10 ⁻⁹	6.0x10 ⁻⁹
Deep Borehole Complex (Direct Disposition)	1.7x10 ⁻⁶	4.7x10 ⁻⁸	6.6x10 ⁻¹⁵	9.8x10 ⁻¹²	1.7x10 ⁻⁶	6.4x10 ⁻¹⁰	8.5x10 ⁻¹⁰
Deep Borehole Complex (Immobilized Disposition)	2.1x10 ⁻⁶	6.8x10 ⁻⁸	9.5x10 ⁻¹⁵	1.5x10 ⁻¹¹	2.2x10 ⁻⁶	8.3x10 ⁻¹⁰	1.1x10 ⁻⁹
Vitrification Facility	4.7x10 ⁻³	2.3x10 ⁻⁴	9.1x10 ⁻⁸	5.1x10 ⁻⁵	5.0x10 ⁻³	1.9x10 ⁻⁶	2.5x10 ⁻⁶
Ceramic Immobilization Facility (Ceramic Immobilization)	1.3x10 ⁻⁵	4.4x10 ⁻⁵	1.9x10 ⁻⁸	1.0x10 ⁻⁵	6.7x10 ⁻⁵	2.5×10^{-8}	3.4x10 ⁻⁸
Advanced Boiling Water Reactor	1.8x10 ⁻¹	30	1.2	1.9x10 ⁻¹	32	1.2x10 ⁻²	1.6x10 ⁻²
CE System 80+ Reactor	5.1x10 ⁻¹	27	1.8x10 ⁻¹	5.9x10 ⁻³	28	1.1x10 ⁻²	1.4×10^{-2}
[Text deleted.]					20	1.1710	1.4410
AP600 Reactor	1.1x10 ⁻¹	23	6.5x10 ⁻¹	6.5x10 ⁻²	24	9.0x10 ⁻³	1.2x10 ⁻²
RESAR-90 Reactor	4.0x10 ⁻¹	29	3.0x10 ⁻¹	7.5×10^{-2}	29	1.1×10^{-2}	1.2×10^{-2}

Table M.2.9–4. Doses and Resulting Health Effects to the Population Within 80 Kilometers of Savannah River Site From Atmospheric Releases Associated With Normal Operation in 2030

^a Total dose to the population within 80 km from natural background radiation in year 2030 is equal to 266,000 person-rem.

^b Dose and health effect results are based on a capacity of 5,000 Pu storage positions in the APSF (SR DOE 1995e). Because the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative and the Upgrade With RFETS Non-Pit Pu Subalternative both call for fewer than 5,000 Pu storage positions in the APSF, dose and health effects for these two subalternatives would be less. The dose shown here is for the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative would be slightly less, and would be below detection limits.

^c Number reflected as a component in the Committed Effective Dose Equivalent. Source: HNUS 1996a.

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Storage and Disposition of Weapons-Usable Fissile Materials Final PEIS

				Pathway em)				Percent of Background ^a	Estimated 1-Year Fatal Cancer Risk
Alternative/Facility	Fish Ingestion	Other Food Ingestion	Drinking Water	Boating	Swimming	Shoreline	Committed Effective Dose Equivalent (mrem)		
No Action (Total Site)	0.27	8.1x10 ⁻²	2.3x10 ⁻²	2.0x10 ⁻⁶	4.0x10 ⁻⁶	5.9x10 ⁻⁴	0.37	0.13	1.9x10 ⁻⁷
• •	0.27 c	c	c	c	с	с	6.1x10 ⁻⁷	2.1x10 ⁻⁶	3.0x10 ⁻¹³
Upgrade Storage Facility ^b Advanced Boiling	1.4x10 ⁻²	4.3x10 ⁻⁴	1.3x10 ⁻⁴	6.1x10 ⁻⁷	1.2x10 ⁻⁶	1.3x10 ⁻⁴	1.5x10 ⁻²	4.9x10 ⁻³	7.3x10 ⁻⁹
Water Reactor	4.7x10 ⁻²	2.9x10 ⁻³	7.9x10 ⁻⁴	2.1x10 ⁻⁶	4.1x10 ⁻⁶	3.5x10 ⁻⁴	5.2×10^{-2}	1.7x10 ⁻²	2.6x10 ⁻⁸
CE System 80+	4.7810	£.7X10	7.5810	2.1.410					
[Text deleted.]	6.1x10 ⁻²	4.9x 10 ⁻³	1.4x10 ⁻³	2.0x10 ⁻⁶	3.9x10 ⁻⁶	3.7x10 ⁻⁴	6.7x10 ⁻²	2.3×10^{-2}	3.4x10 ⁻⁸
AP600 Reactor	•	_	1.4×10^{-3}	1.9×10^{-6}	3.8x10 ⁻⁶	2.1×10^{-4}	5.1x10 ⁻²	1.7×10^{-2}	2.5x10 ⁻⁸
RESAR-90 Reactor	4.3×10^{-2}	6.1x10 ⁻³	1.8x10 °	1.9X10 -	3.6810	2.1110	5.1110	1.7×10	2.5710

Table M.2.9–5.Doses and Resulting Health Effects to the Maximally Exposed Individual From
Liquid Releases Associated With Annual Normal Operation at Savannah River Site

^a Individual annual natural background radiation dose equal to 298 mrem.

^b Dose and health effect results are based on a capacity of 5,000 Pu storage positions in the APSF (SR DOE 1995e). Because the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative and the Upgrade With RFETS Non-Pit Pu Subalternative both call for fewer than 5,000 Pu storage positions in the APSF, dose and health effects for these two subalternatives would be less. The dose shown here is for the Upgrade With All Or Some RFETS Pu and LANL Pu Subalternative. The dose for the upgrade with RFETS Non-Pit Pu Subalternative would be slightly less.

^c Number reflected as a component in the Committed Effective Dose Equivalent.

Source: HNUS 1996a.

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Table M.2.9–6. Doses and Resulting Health Effects to the Population Downstream of Savannah River Site From Liquid Releases Associated With Normal Operation in 2030

	-	I	Oose by Pathwa (person-rem)					
Alternative/Facility	Fish Ingestion	Drinking Water	Boating	Swimming	Shoreline	Committed Effective Dose Equivalent (person-rem)	Percent of Background ^a	Estimated 1-Year Fatal Cancers
No Action (Total Site)	9.0x10 ⁻¹	2.7	2.2×10^{-5}	6.4x10 ⁻⁶	1.2x10 ⁻³	3.6	1.3×10^{-3}	1.8x10 ⁻³
Upgrade Storage Facility ^b	с	c	с	c	c	1.0x10 ⁻⁵	3.5x10 ⁻⁹	5.0x10 ⁻⁹
Advanced Boiling Water Reactor	8.2×10^{-2}	1.4×10^{-2}	6.8x10 ⁻⁶	2.0×10^{-6}	2.4×10^{-4}	9.6x10 ⁻²	3.4×10^{-5}	4.8×10^{-5}
CE System 80+	2.3x10 ⁻¹	9.2×10^{-2}	2.3×10^{-5}	6.7x10 ⁻⁶	6.7×10^{-4}	3.2×10^{-1}	1.1x10 ⁻⁴	16-10-4
[Text deleted.]				0.7710	0.7210	J.2XIU	1.1X10	1.6x10 ⁻⁴
AP600 Reactor	2.3×10^{-1}	1.6x10 ⁻¹	2.2x10 ⁻⁵	6.2x10 ⁻⁶	7.2x10 ⁻⁴	3.9x10 ⁻¹	1.4×10^{-4}	2.0x10 ⁻⁴
RESAR-90 Reactor	1.6x10 ⁻¹	2.0x10 ⁻¹	2.1×10^{-5}	6.2x10 ⁻⁶	4.1x10 ⁻⁴	3.6x10 ⁻¹	1.3×10^{-4}	1.8×10^{-4}

^a Natural background radiation dose to the population within 80 km plus the people who use the Savannah River for drinking water at the Port Wentworth and Beaufort-Jasper location is: 285,000 person-rem in the year 2030.

^b Dose and health effect results are based on a capacity of 5,000 Pu storage positions in the APSF (SR DOE 1995e). Because the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative and the Upgrade With RFETS Non-Pit Pu Subalternative both call for fewer than 5,000 Pu storage positions in the APSF, dose and health effects for these two subalternatives would be less. The dose shown here is for the Upgrade With All or Some RFETS Pu and LANL Pu Subalternative would be slightly less.

^c Number reflected as a component in the Committed Effective Dose Equivalent. Source: HNUS 1996a.

M.2.9.1 No Action

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Atmospheric Releases and Resulting Impacts to the Public. For No Action, all of the areas have radioactive releases in to the atmosphere from normal operation. Table M.2.9.1-1 presents the estimated annual atmospheric radioactive releases.

Tables M.2.9–3 and M.2.9–4 include the atmospheric radiological impacts to the maximally exposed member of the public and the offsite population within 80 km (50 mi), respectively. The MEI would receive an annual dose of 0.42 mrem. An estimated fatal cancer risk of 1.1×10^{-5} would result from 50 years of operation. The population within 80 km (50 mi) would receive a dose of 40 person-rem in 2030 (midlife of operation). An estimated 1.0 fatal cancers would result from 50 years of operation.

Liquid Releases and Resulting Impacts to the Public. For No Action, some areas may have radioactive releases to the offsite surface water from normal operation. Table M.2.9.1–2 presents the estimated annual liquid radioactive releases.

Tables M.2.9–5 and M.2.9–6 include the radiological impacts to the MEI and the offsite populations using water from the Savannah River downstream of SRS to the Atlantic Ocean. The maximally exposed member of the public would receive an annual dose of 0.37 mrem. An estimated fatal cancer risk of 9.3×10^{-6} would result from 50 years of operation. The population would receive a dose of 3.6 person-rem in 2030. An estimated 0.09 fatal cancers would result from 50 years of operation.

Worker Doses. It is projected that in 2005 and beyond, there would be 7,069 badged workers involved in No Action activities. The annual average dose among these workers would be 36 mrem and the annual dose among all these workers would equal 259 person-rem. From 50 years of operation, an estimated fatal cancer risk of 7.2×10^{-4} would result to the average worker and 5.2 fatal cancers could result among all workers.

······································				F	-Area	Н	-Area		
Isotope	SRTC	K-Reactor	L-Reactor	Canyon Releases	Waste Management	Canyon Releases	Waste Management	RBOF	Tritium Facilities
H-3	0	3.5×10^3	1.9×10^2	0	0	0	1.7x10 ⁰	0	2.2x10 ⁴
C-14	0	0	0	7.4x10 ⁻³	0	1.1x10 ⁻³	0	0	0
S-35	0	0	0	0	0	0	0	0	0
[Text deleted.]								-	-
Cr-51	0	0	0	0	0	0	0	0	0
Co-60	0	0	0	0	5.9x10 ⁻⁹	0	0	0	0
Ni-63	0	0	0	0	0	0	0	0	0
Se-79	0	0	0	0	0	0	0	0	0
Sr-89	0	0	0	0	0	0	0	0	0
Sr-90	1.2x10 ⁻⁵	1.9x10 ⁻⁷	1.8x10 ⁻⁵	8.1x10 ⁻⁴	0	1.3x10 ⁻⁴	0	0	0
Y-90	0	0	0	0	0	0	0	0	0
Y-91	0	0	0	0	0	0	0	0	0
Zr-95	0	0	0	0	0	0	0	0	0
Nb-95	0	0	0	0	- 0	0	0	0	ů 0
Tc-99	0	0	0	0	0	0	0	0	0
Ru-106	0	0	4.0x10 ⁻⁷	0	0	0	5.8x10 ⁻⁹	0	0 0
Sn-126	0	0	0	0	0	0	0	0	0
Sb-125	0	0	0	0	0	0	0	ů 0	ů 0
Te-125m	0	0	0	0	0	0	0	0	0 0
Te-127m	0	0	0	0	0	0	0	0	0
Te-127	0	0	0	0	0	0	0	0	Õ
I-129	0	0	0	1.3x10 ⁻³	0	1.2x10 ⁻³	0	0 0	0 0

 Table M.2.9.1–1.
 Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Savannah River Site (curies)

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				F.	Area	H	Area		
Isotope	SRTC	K-Reactor	L-Reactor	Canyon Releases	Waste Management	Canyon Releases	Waste Management	RBOF	Tritium Facilitie
I-131	5.9x10 ⁻⁵	0	0	1.5x10 ⁻⁶	0	4.3x10 ⁻⁵	0	0	0
I-133	2.0×10^{-3}	0	0	0	0	0	0	0	0
[Text deleted.]	2.0/10	-							
Xe-135	3.2×10^{-2}	0	0	0	0	0	0	0	0
Cs-134	0	ů 0	0	6.9x10 ⁻⁷	0	0	1.1x10 ⁻⁷	0	0
Cs-134 Cs-135	0 Ŭ	ů 0	0	0	0	0	0	0	0
	1.5x10 ⁻⁶	1.1x10 ⁻⁷	1.0x10 ⁻⁵	2.3x10 ⁻⁴	3.8x10 ⁻⁶	2.0x10 ⁻⁵	2.2x10 ⁻⁵	2.1×10^{-7}	0
Cs-137	0	0	0	0	0	0	0	0	0
Ce-144	0	0	0	0	0	0	0	0	0
Pr-144	0	0	0	0	0	0	0	0	0
Pm-147	0	0	Ő	0	0	0	0	0	0
Sm-151	0	0	ů 0	0	0	0	0	0	0
Eu-152	0	0	0	0	0	0	0	0	0
Eu-154	0	0	0	Õ	0	- 0	0	0	0
Eu-155	0	0	0	8.8x10 ⁻⁴	2.1x10 ⁻⁶	4.7x10 ⁻⁵	0	0	0
U-235	2.9x10 ⁻⁸	0	0	1.6x10 ⁻⁴	3.2×10^{-7}	4.4x10 ⁻⁴	0	0	0
Pu-238	1.0x10 ⁻⁸	0	4.1x10 ⁻⁷	4.3×10^{-4}	2.6×10^{-7}	1.0x10 ⁻⁴	0	0	0
Pu-239	9.4x10 ⁻⁶	4.4x10 ⁻⁹		4.5210	0	0	0	0	0
Pu-240	0	0	0	-	0	0 0	0	0	0
Pu-241	0	0	0	0	v	4.0x10 ⁻⁵	0	ů 0	0
Am-241	1.3x10 ⁻⁶	0	0	3.1x10 ⁻⁵	1.0x10 ⁻⁷		-	0	0
Cm-244	6.8x10 ⁻⁶	0	0	2.2×10^{-5}	0	3.3x10 ⁻⁶	0	U	0

Table M.2.9.1–1. Annual Atmospheric Radioactive Releases From Normal Operation of No Action at Savannah River Site (curies)—Continued

Isotope	DWPF	M-Area	CIF	P-Reactor	C-Reactor	D-Area	Diffuse Area
H-3	2.0x10 ¹	0	1.2×10^3	1.3×10^3	1.5x10 ²	4.5×10^2	4.3x10 ¹
C-14	2.1×10^{-2}	0	0	0	0	0	4.0×10^{-6}
S-35	0	0	0	0	0	0	$4.0x10^{-6}$
[Text deleted.]					Ū	0	2.0x10 -
Cr-51	0	0	1.5×10^{-2}	0	0	0	0
Co-60	6.1x10 ⁻⁸	0	1.4×10^{-4}	0	0	0	0 3.3x10 ⁻¹⁷
Ni-63	0	0	0	0	0	0	
Se-79	8.8x10 ⁻⁹	0	0	0	ů	0	2.0x10 ⁻⁷
Sr-89	0	0	6.0x10 ⁻⁴	ů 0	0	0	0
Sr-90	2.3x10 ⁻⁵	8.3x10 ⁻⁵	2.2×10^{-2}	ů 0	0	7.2x10 ⁻⁶	0
Y-90	2.4x10 ⁻⁵	0	7.6x10 ⁻⁵	Õ	0	7.2x10 -	1.1x10 ⁻⁴
Y-91	0	0	4.5x10 ⁻⁴	0 0	0	0	0
Zr-95	0	0	4.7x10 ⁻⁴	0	0	0	0
Nb-95	0	0	1.5×10^{-3}	0	0	0	2.4x10 ⁻¹⁴
Tc-99	3.8x10 ⁻⁷	0	0	0	0	0	0
Ru-106	3.2x10 ⁻⁵	0	1.8x10 ⁻⁴	0	0	0	0
Sn-126	6.9x10 ⁻⁸	0	0	0	0	0	5.0x10 ⁻¹²
Sb-125	6.7x10 ⁻⁷	0	0	0	0	0	0
Te-125m	1.0x10 ⁻⁵	0	0 0	0	0	0	7.3x10 ⁻¹⁵
Te-127m	4.5x10 ⁻⁹	0	ů 0	0	0	0	0
Te-127	4.4x10 ⁻⁹	ů 0	0	0	0	0	0
I-129	8.2x10 ⁻⁵	0 Û	0	0	0	0	0
I-131	0	Ő	0	0	U	0	6.9x10 ⁻⁷
I-133	ů 0	õ	0	0	Û	0	0
[Text deleted.]	Ŭ	v	U	0	0	0	0

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Table M.2.9.1–1. Annual Atmospheric Radioactive Releases from Normal Operation of No Action at Savannah River Site (curies)—Continued

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Isotope	DWPF	M–Area	CIF	P-Reactor	C-Reactor	D-Area	Diffuse Area
Xe-135	0	0	0	0	0	0	0
Cs-134	2.9x10 ⁻⁵	0	0	0	0	0	1.4x10 ⁻¹⁷
Cs-135	9.4x10 ⁻⁷	0	0	0	0	0	0
Cs-137	4.1×10^{-3}	0	2.4×10^{-4}	0	0	0	4.3x10 ⁻¹¹
Ce-144	3.0x10 ⁻⁶	0	2.3x10 ⁻⁴	0	0	0	1.1x10 ⁻¹³
Pr-144	3.1x10 ⁻⁶	0	2.3x10 ⁻⁴	0	0	0	0
Pm-147	7.6x10 ⁻⁶	0	9.1x10 ⁻⁴	0	0	0	0
Sm-151	1.6x10 ⁻⁷	0	0	0	0	0	0
Eu-152	1.4x10 ⁻⁹	0	0	0	0	0	0
Eu-154	2.3×10^{-7}	0	0	0	0	0	3.4x10 ⁻¹³
Eu-155	1.6x10 ⁻⁷	0	0	0	0	0	1.6x10 ⁻¹³
U-235	0	1.6x10 ⁻⁵	0	0	0	0	4.7x10 ⁻⁵
Pu-238	7.9x10 ⁻⁷	0	1.4x10 ⁻⁴	0	0	0	4.6x10 ⁻¹²
Pu-239	7.1x10 ⁻⁹	3.5x10 ⁻⁶	5.2×10^{-7}	0	0	8.4x10 ⁻⁷	4.7x10 ⁻⁷
Pu-240	4.8x10 ⁻⁹	0	0	0	0	0	0
Pu-241	7.7x10 ⁻⁷	0	0	0	0	0	- 0
Am-241	8.6x10 ⁻⁹	0	0	0	0	0	8.9x10 ⁻¹³
Cm-244	2.7×10^{-8}	0	0	0	0	0	7.3x10 ⁻¹²

 Table M.2.9.1–1.
 Annual Atmospheric Radioactive Releases from Normal Operation of No Action at Savannah River Site (curies)—Continued

Note: SRTC=Savannah River Technology Center; RBOF=Receiving Basin Offsite Fuel. Source: WSRC 1994d.

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Isotope	Release
H-3	1.3x10 ⁴
Sr-90	0.48
I-129	0.022
Cs-137	0.25
Pm-147	7.0x10 ⁻³
U-235	1.1x10 ⁻⁵
Pu-239	9.6x10 ⁻³

Table M.2.9.1-2.	Annual Liquid Releases From Normal Operation of
No	Action at Savannah River Site (curies)

tal Site release.

Source: WSRC 1994d.

M.2.9.2 **Storage and Disposition**

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Atmospheric Releases and Resulting Impacts to the Public. Total site radiological impacts during operation of storage or disposition facilities can be found by adding the impacts resulting from No Action facilities to the changes in impacts resulting from storage or disposition facilities. For example, to determine the radiological impact for the addition of an AP600 reactor at SRS, the No Action facilities doses would be summed with the AP600 reactor doses. Estimated annual atmospheric radioactive releases for the storage and disposition facilities are given in Section M.2.3. Tables M.2.9-3 and M.2.9-4 include the atmospheric radiological impacts by alternative facility.

Virtually, no change is anticipated in radioactive releases due to the upgraded or new Pu storage facilities for continued Pu storage at SRS above those radioactive releases already included in No Action. Therefore, there are no changes in dose to the public from the upgraded or new Pu storage facilities at SRS.

The annual doses from total site operations associated with the different alternative facilities range from 0 to 0.26 mrem to the maximally exposed member of the public and from 0 to 32 person-rem to the 80-km (50-mi) population in 2030. The associated health effects from annual operations are included in both tables.

Liquid Releases and Resulting Impacts to the Public. There are two disposition technologies that would release liquid discharges to the surface water surrounding SRS. These are the large and small evolutionary LWRs. The liquid releases for these two technologies are given in Section M.2.3. As an example of determining the total site liquid radiological impact associated with the addition of an AP600 reactor at SRS, the No Action liquid doses must be summed with the AP600 reactor liquid doses. Table M.2.9-5 and M.2.9-6 present the liquid radiological impacts for the applicable alternative facilities.

The annual doses associated with the different LWRs that have liquid releases range from 0.015 to 0.067 mrem to the maximally exposed member of the public, and range from 0.096 to 0.39 person-rem to the downstream population in 2030. The associated health effects from annual operations are included in both tables.

Worker Doses and Health Effects. For the storage and disposition alternatives, the impacts from the No Action facilities need to be added to the changes in impacts from the storage or disposition facilities to determine the impacts from total site operations (refer to the worker discussion under No Action, above, and to Table M.2.3.2-1).

M.2.10 RADIOLOGICAL IMPACT AT ROCKY FLATS ENVIRONMENTAL TECHNOLOGY SITE

The results of the radiological consequence assessments for the RFETS and the sources of data used in the assessments are given in Section 4.2.7.9.

M.2.11 RADIOLOGICAL IMPACT AT LOS ALAMOS NATIONAL LABORATORY

The results of the radiological consequence assessments the LANL and the source of data used in the assessments are given in Section 4.2.8.9.

M.2.12 DEEP BOREHOLE DISPOSITION GENERIC SITE

The results of the radiological consequence assessments for the generic borehole site are given in Sections 4.3.3.1.9 and 4.3.3.2.2.9. The sources of data used in the assessments are given in Health Physics Data, October 1996.

M.2.13 GENERIC MIXED OXIDE FUEL FABRICATION SITE

The results of the radiological consequence assessments for the generic MOX fuel fabrication facility are given in Section 4.3.5.1.9. The sources of data used in the assessments are given in Health Physics Data, October 1996.

M.2.14 EXISTING LIGHT-WATER REACTOR GENERIC SITE

The results of the radiological consequence assessments for the generic existing light-water reactor facility are given in Section 4.3.5.2.9. The sources of data used in the assessments are given in Health Physics Data, October 1996.

M.2.15 PARTIALLY COMPLETED REACTOR GENERIC SITE

The results of the radiological consequence assessments for the generic partially completed reactor site are given in Section 4.3.5.3.9. The sources of data used in the assessments are given in Health Physics Data, October 1996.

M.3 HAZARDOUS CHEMICAL IMPACTS TO HUMAN HEALTH

M.3.1 BACKGROUND

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Two general types of adverse human health effects are assessed for hazardous chemical exposure in this PEIS. These are carcinogenic and non-carcinogenic effects. For this reason, two tables were developed to assist the risk assessor in the evaluation process. Table M.3.2-1, the Table of Chemical Toxicity Profiles, characterizes each chemical in terms of physical properties, potential exposure routes, and the effects on target tissues/organs that might be expected. The risk assessor will use it qualitatively to determine how exposure might occur (exposure route), what tissue or organ system might be affected (for example, central nervous system dysfunction or liver cancer) and whether the chemical might possess other properties affecting its bioavailability in a given matrix (that is, air, water, or soil). Table M.3.3-1, the Table of Exposure Limits, provides the risk assessor with the necessary information to calculate risk or expected effects should an individual be exposed to a hazardous chemical for a long time at low levels (chronic exposure) or to higher concentrations for a short time (acute). Where a dose effect calculation is required (milligram [mg]/kilogram [kg]/day), the Reference Dose (RfD) is applicable, and where an inhalation concentration effect is required, the Reference Concentration (for example, Reference Concentration [RfC] in mg/cubic meter) is applicable for chronic exposures. The Permissible Exposure Limit (PEL) value, which regulates worker's exposures over 8-hour (hr) periods, determines the concentration allowed for occupational exposures that would be without adverse acute effects. Other values, such as the Threshold Limit Value, are presented for the reader's information, because they are prepared by the American Conference of Governmental Industrial Hygienists (ACGIH) for guidance on exposures of 8-hr periods, and can be used to augment PELs or serve as exposure levels in the absence of a PEL. All currently regulated chemicals associated with each site and every hazardous chemical are presented in Table M.3.2-1 and Table M.3.3-1.

It was assumed that under normal operation conditions, members of the public would only receive chronic exposures at low levels in the form of air emissions from a centrally located source term at each site; since hazardous chemicals are not released into surface or ground waters or into soil, inhalation is assumed to be the only route of exposure. However, all chemical quantities are accounted for as air emissions, which are several orders of magnitude greater than by all other possible routes combined. It was further assumed that the MEI member of the public would be at the site boundary and this assumption was used when calculating all public exposures, which under normal operating conditions are expected to be chronic and at very low levels. For worker exposures to hazardous chemicals, it was assumed that individuals were exposed only to low air emission concentrations during an 8-hr day for a 40-hr week for a maximum working lifetime of 40 years. The point of exposure chosen was 100 m (328 ft) from a centrally located source term, since the precise placement of source terms onsite could not be made. Further, it could not be determined where the involved and non-involved workers would be relative to the emission sources.

For every site involved in the analysis, Hazard Indexes (HIs) were calculated for every alternative action relative to the site. The exposure concentrations of hazardous chemicals for the public and the onsite workers were developed using the Industrial Source Complex Short Term Model for point, area, and volume sources. This model, which estimates dispersion of emissions from these sources, has been field tested and recommended by the EPA. The modeled concentrations were compared to the unique RfC and PEL values unique to each chemical to yield Hazard Quotients (HQs) for the public and onsite workers, respectively. The HQs were summed to give the HIs for each alternative action at each site, as well as total HIs (that is, No Action HI + alternative-incremental HI). For cancer risk estimation, the inhaled concentrations were converted to doses in mg/kg/day, which were then multiplied by the slope factors unique to each identified carcinogen. The risks for all carcinogens associated with each alternative (incremental risk) at each site were summed, and the No Action cancer risk for each site was added in order to show the total risk should that alternative action be implemented at a given site. We apply this conservative approach to all sites using the guidance under the *Comprehensive Environmental Response Compensation and Liability Act*, which applies to Superfund sites. The first

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assessment in risk analysis is considered a screening step. Under this guidance, if the HI is less than, or equal to 1.0, all non-cancer exposure values meet Occupational Safety and Health Administration (OSHA) standards; if the cancer risk is less than or equal to 1.0×10^{-6} , no further analysis is done. A cancer risk of 1.0×10^{-6} from other sources cannot be distinguished from the cancer risk for an individual member of the general population.

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Storage and Disposition of Weapons-Usable Fissile Materials Final PEIS

M.3.2 CHEMICAL TOXICITY PROFILES

Table M.3.2–1 provides the reader with pertinent facts about each chemical that is included in this PEIS's human health risk assessment. This includes the Chemical Abstracts Service number, which aids in a search for information available on any specific chemical and ensures a positive identity regardless of which name or synonym is used. It also contains physical information (that is, solubility, vapor pressure, and flammability) as well as presenting incompatibility data that is useful in determining whether a hazard might exist and the nature of the hazard. The route of exposure, target organs/tissues, and carcinogenicity provide an abbreviated summary on how individuals might get exposed, what body functions could be affected, and whether chronic exposure could lead to increased cancer incidence in an exposed population.

	CASN	Colubilit-	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity
Compound Acetaldehyde	<u>CAS No.</u> 75-07-0	Solubility Miscible ^d	740 mm ^d	Class IA Flammable liquid ^d	Strong oxidizers, acids, bases, alcohols, ammonia, amines, phenols, ketones, HCN, H ₂ S (Prolonged contact with air may form peroxides that may explode: Easily polymerizes) ^d	Inh, ing con ^d	Eyes, skin, resp sys, kidneys, repro sys (In animals: nasal cancer) ^d	EPA Group B2 ^e
Acetic acid	64-19-7	Miscible ^d	11 mm ^d	Class II Combustible liquid ^d	Strong oxidizers, strong caustics, corrosive to metals ^d	Inh, con ^d	Eyes, skin, resp sys, teeth ^d	Not Classified
Acetone	67-64-1	Miscible ^d	180 mm ^d	Class IB ^d	Oxidizers, acids ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	EPA Group D ^e
Acetonitrile	75-05-8	Miscible ^d	73 mm ^d	Class IB ^d	Strong oxidizers ^d	Inh, abs, ing, con ^d	Resp sys, CVS, CNS, liver, kidneys ^d	Not Classified
Acetylene	74-86-2	2% ^d	44.2 atm ^d	Flammable gas ^d	Zinc; Oxygen and other oxidizing agents such as halogens ^d	Inh, con (liq) ^d	CNS, resp sys ^d	Not Classified
Aluminum	7429-90-5	Insoluble ^d	0 mm (approx) ^d	Combustible solid, finely divided dust is easily ignited ^d	Strong oxidizers, acids, halogenated hydrocarbons ^d	Inh, con ^d	Eyes, skin, resp sys ^d	Not Classified
Aluminum welding fumes	None	Insoluble ^d	0 mm (approx) ^d	Noncombus- tible solid, but dust may form explosive mixtures in air ^d	Chlorine, trifluoride, hot chlorinated rubber, acids, oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^d	Not Classified

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability^a	Incompatibilities	Route of Exposure ^b	Target	Construction for
Ammonia	7664-41-7	34% ^d	8.5 atm ^d	Treat as a flammable gas ^d	Strong oxidizers, acids, halogens, salts of Ag and Zn ^d	Inh, ing (soln), con (soln/liq) ^d	Organs Eyes, skin, resp sys ^d	Carcinogenicity ^c EPA Group D ^e
Ammonium hydroxide	1336-21-6	Soluble ^f	None Found	None Found	Strong oxidizers, acids, halogens, salts of Ag ¹	Inh, abs, ing con ^f	Eyes, skin, resp sys ^f	Not Classified
Antimony (Nonradionuclide)	7440-36-0	Insoluble ^d	0 mm (approx) ^d	Noncombust- ible as solid bulk; moderate explosion hazard as dust exposed to flame ^d	Strong oxidizers, acids, halogenated acids ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CVS ^d	EPA Group D ^e
Arsenic (Insol cmpds/metal)	7440-38-2	Insoluble ^d	0 mm (approx) ^d	Metal: Noncom- bustible as solid bulk; slight explosion hazard as dust exposed to flame ^d	Strong oxidizers, bromine azide ^d	Inh, abs, con, ing ^d	Liver, kidneys, skin, lungs, lymphatic sys (lung and lymphatic cancer) ^d	EPA Group A ^e
Barium	7440-39-3	None Found	10 mm (1049 °C) ^f	Flammable solid spontan- eously combustible; dangerous when wet ^f	Water, acids, carbon tetrachloride, fluorotrichloro- methane, trichloro- ethylene, and tetrachloro- ethylene ^f	None Found	None Found	Not Classified
Benzene	71-43-2	0.07% ^d	75 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, many fluorides and perchlorates, nitric acid ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, blood, CNS, bone marrow (leukemia) ^d	EPA Group A ^e

Table M.3.2-1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Beryllium (metal)	7440-41-7	Insoluble ^d	0 mm ^d	Metal: Noncom- bustible as solid bulk; slight explosion as dust ^d	Acids, caustics, chlorinated hydrocarbons, oxidizers, molten Li ^d	Inh, con ^d	Eyes, skin, resp sys, (lung cancer) ^d	EPA Group B ^e
Bismuth	7440-69-9	Insoluble ^g	1 mm (1021 °C) ^f	Flammable when exposed to flame ^f	Incompatible with Al, BrF ₃ , acids, NOF, ammonium nitrate, perchloric acid, chloride, IF ₅ , nitric acid ^f	None Found	None Found	Not Classifi e d
Boric acid	10043-35-3	1g in 18 ml cold water ^g	Volatile with steam ^g	None Found	K, acetic anhydride ^f	Inh, abs, inq, con ^f	Eyes, skin, resp sys, GI ^f	Not Classified
1,3-Butadiene	106-99-0	Insoluble ^d	2.4 atm ^d	Class I Flammable liquid ^d	Phenol, chlorine dioxide, Cu, crotonaldehyde ^d	Inh, con (liq) ^d	Eyes, skin, resp sys, CNS, repro sys (Hemato cancer) ^d	EPA Group B2 ^e
n-Butane	106-97-8	Slightly soluble ^d	2.05 atm ^d	Class IA Flammable liquid ^d	Strong oxidizers, chlorine, fluorine, (Ni carbonyl+O ₂) ^d	Inh, con (liq) ^d	CNS ^d	Not Classified
1-Butene (butylene)	106-98-9	None Found	3,480 mm ^f	Flammable gas ^f	Aluminium hydroborate ^f	Simple asphyxiant ^f	None Found	Not Classified
2-Butoxyethanol	111-76-2	Miscible ^d	0.8 mm ^d	Class IIIA Combustible liquid ^d	Strong oxidizers, strong caustics ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, blood, kidneys, liver, lymphoid sys ^d	Not Classified
n-Butyl alcohol (1-butanol)	71-36-3	9% ^d	6 mm ^d	Class IC Flammable liquid ^d	Strong oxidizers, strong mineral acids, alkali metals, halogens ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, CNS ^d	EPA Group D ^e
Butyl lactate	138-22-7	Slightly soluble ^d	0.4 mm ^d	Class IIIA Combustible liquid ^d		Inh, ing, con ^d	Eyes, skin, resp. sys, CNS ^d	Not Classified

Health and Safety

Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Cadmium dust (Nonradionuclide)	7440-43-9	Insoluble ^d	0 mm (approx) ^d	Noncombust- ible as solid bulk; will burn as powder ^d	Strong oxidizers, elemental S, Se, and Te ^d	Inh, ing ^d	Resp sys, kidneys, prostate, blood (prostatic and lung cancer) ^d	EPA Group B1 ^e
Cadmium oxide (fume)	1306-19-0	Insoluble ^d	0 mm (approx) ^d	Noncombust- ible solid ^d	Not applicable ^d	Inh ^d	Resp sys, kidneys, prostate, blood (prostatic and lung cancer) ^d	Not Classified
Calcium	7440-70-2	Reacts with water ^g	10 mm (983 °C) ^h	Flammable solid, Spontaneous- ly combustible ^f	Strong oxidizing agents, acids, water, alkali metal hydroxides or carbonates, halogens, Ph, Si, Hg ^f	None Found	None Found	Not Classified
Carbon dioxide	124-38-9	Sublimes ^d	56.5 atm ^d	Non- flammable gas ^d	Dusts of metals (eg. Mg, Zr, Ti, Al, Cr, and Mn) are ignitable and explosive when suspended in CO ₂ ^d	Inh, con (liq/soln) ^d	Resp sys, CVS ^d	Not Classified
Carbon disulfide	75-15-0	0.3% ^d	297 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers; chemically-active metals (eg. Na, K, and Zn); azides; rust; halogens; amines ^d	Inh, abs, ing, con ^d	CNS, PNS, CVS, eyes, kidneys, liver, skin, repro sys ^d	Not Classified
Carbon monoxide	630-08-0	2% ^d	>35 atm ^d	Flammable gas ^d	Strong oxidizers, bromine trifluoride, chlorine trifluoride, Li ^d	Inh, con (liq) ^d	CVS, lungs, blood, CNS ^d	Not Classified

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Carbon tetrachloride	56-23-5	0.05% ^d	91 mm ^d	Non- combustible liquid ^d	Chemically-active metals (eg. Na, K, and Mg), F ₂ , Al ^d	Inh, abs, ing, con ^d	CNS, eyes, lungs, liver, kidneys, skin (In animals: liver cancer) ^d	EPA Group B2 ^e
Chloride (Sodium chloride)	77647-14-5	Soluble ^f	1 mm (865 °C) ^f	Decomposes ^f	Potentially explosive reaction with dichloromaleic anhydride+urea; Violent reaction with BrF ₃ ^f	Inh, abs, ing, con, ipr, sca ^f	Eyes, skin, Gl tract, repro sys, resp sys ^f	Not Classifi c d
Chlorine	7782-50-5	0.7% ^d	6.8 atm ^d	Non- flammable gas ^d	Reacts explosively or forms explosive cmpds with many common substances (e.g., acetylene, ether, turpentine, ammonia, fuel gas, hydrogen and finely divided metals) ^d	Inh, con ^đ	Eyes, skin, resp sys ^d	EPA Group D ^e
Chlorobenzene	108-90-7	0.05% ^d	9 mm ^d	Class IC Flammable liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, liver ^d	Not Classified
Chloroform	67-66-3	0.5% (77 °F) ^d	160 mm ^d	Non- combustible liquid ^d	Strong caustics, chemically active metals (e.g., Al or Mg powder, Na, and K), strong oxidizers ^d	Inh, abs, ing, con ^d	Liver, kidneys, heart, skin, CNS (In animals: liver and kidney cancer) ^d	EPA Group B2 ^c
bis-Chloromethyl ether	542-88-1	Reacts in water (68 °F) ^d	30 mm (72 °F) ^d	Class IB Flammable liquid ^d	Acids, water ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys ^d	EPA Group A ^e

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	In composibilities	Route of	Target	a .
· ` · ` ·					Incompatibilities	Exposure ^b	Organs	Carcinogenicity
Chromium (Hexavalent); Chromium[VI] oxide	18540-29-9; 1333-82-0 (CrO ₃ ; acid)	None Found	Decomp, 61.7 g/100cc (0 °C) ^f	Corrosive crystal ^f	N,N-Dimethylform- amide, explosive reaction or ignites with organic materials and solvents, alcohols and alkali metals (e.g., acetaldehyde, benzene, ethyl acetate, and heat + acetic acid or acetic anhydride, acetone, methanol, butanol, Na, and K) ^f	Inh, abs, ing, con, ipr ^f	Eyes, skin, resp sys, repro sys (Human cancer of nasal cavity and lungs) ^f	EPA Group A ^e
Chromium (Trivalent)	16065-83-1	Varies with cmpd ^d	Varies with cmpd ^d	Varies with cmpd ^d	Varies with cmpd ^d	Inh, ing, con ^d	Eyes, skin ^d	Not Classified
Cobalt (Metal dust and fume)	7440-48-4	Insoluble ^d	0 mm (approx) ^d	Noncombust- ible solid in bulk form; fine dust burns at high temp ^d	Strong oxidizers, ammonium nitrate ^d	Inh, ing, con ^d	Skin, resp sys ^d	Not Classified
Copper (Dusts and mists)	7440-50-8	Insoluble ^d	0 mm (approx) ^d	Noncombust- ible solid in bulk; powder may ignite ^d	Oxidizers, alkalis, sodium azide, acetylene ^d	Inh, ing, con ^d	Eyes, skin, resp sys, liver, kidneys (Increase risk with Wilson's disease) ^d	EPA Group D ^e
Cresol (<i>m</i> -cresol, cresylic acid)	108-39-4	2% ^d	0.14 mm (77 °F) ^d	Class IIIA Combustible liquid ^d	Strong oxidizers, acids ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys, CNS, pancreas, CVS ^d	Not Classified

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			Vapor	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Compound	CAS No.	Solubility	Pressure 78 mm ^d		Oxidizers ^d	Inh, ing, con ^d	Eyes, skin,	Not Classified
Cyclohexane	110-82-7	Insoluble ^d	/8 mm ⁻	Flammable liquid ^d	O A IGIZOIO		resp sys, CNS ^d	
Cyclohexanone	108-94-1	15% ^d	5 mm ^d		Oxidizers, nitric acid ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, CNS, liver, kidneys ^d	Not Classified
Cyclopentane	287-92-3	Insoluble ^d	400 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers (e.g., chlorine, bromine, fluorine) ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	Not Classified
Diacetone alcohol	123-42-2	Miscible ^d	1 mm ^d	Class II Combustible liquid ^d	Strong oxidizers, strong alkalis ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, liver ^d	Not Classified
_	132-64-9	None Found	None Found	None Found	None Found	None Found	None Found	None Found
Dibenzofuran Dibutyl phosphate	107-66-4	Insoluble ^d	1 mm (approx) ^d	Class IIIB Combustible liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	resp sys"	Not Classified
o-Dichlorobenzene	95-50-1	0.01% ^d	1 mm ^d	Class IIIA Combustible liquid ^d	Strong oxidizers, Al, chlorides, acids, acid fumes ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys ^d	Not Classified
3,3-Dichlorobenzidine	91-94-1	7% (59 °F) ^d	None Found	None Found	None Found	Inh, abs, ing, con ^d	Bladder, liver, lung, GI tract (In animals: liver and bladder	EPA Group B2 ^e
Dichlorodifluoromethane	75-71-8	0.03% (77 °F) ^d	5.7 atm ^d	Nonflammable gas ^d	e Chemically active metals (e.g., Na, K, Ca, powdered Al, Zn, and Mg) ^d	Inh, con (liq)	cancer) ^d CVS, PNS	EPA Group D ^e

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure		Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Dichloromethane (Methylene chloride)		2% ^d	350 mm ^d	Combustible liquid ^d	Strong oxidizers, caustics, chemically active metals (eg., Al, Mg powders, K and Na), conc nitric acid ^d	Inh, abs, ing, con ^d	Eyes, skin, CVS, CNS, (In animals: lung, liver, salivary and mammary gland tumors) ^d	EPA Group B2 ^e
Dimethylformamide (DMF)	68-12-2	Miscible ^d	3 mm ^d	Class II Combustible liquid ^d	Carbon tetra chloride, other halogenated cmpds when in contact with iron; strong oxidizers, alkyl aluminums, inorganic nitrates ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys, CVS ^d	Not Classified
2,4-Dinitrotoluene	25321-14-6	Insoluble ^d	1 mm ^d	Combustible solid, but difficult to ignite ^d	Strong oxidizers, caustics, metals such as tin and zinc ^d	Inh, abs, ing, con ^d	Blood, liver, CVS, repro sys (In animals: liver, skin, and kidney tumors) ^d	Not Classified
1,4-Dioxane	123-91-1	Miscible ^d	29 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, decaborane, triethynyl aluminum	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys (In animals: lung, liver and nasal cavity tumors) ^d	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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	CAC N-	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Compound Di-sec octyl phthalate	<u>CAS No.</u> 117-81-7	0.00003% (75 °F) ^d	<0.01 mm ^d	Class IIIB Combustible liquid ^d	Nitrates, strong oxidizers, acids and alkalis ^d	Inh, ing, con ^d	Eyes, resp sys, CNS, liver, repro sys, GI tract (In animals: liver tumors) ^d	Not Classified
Dodecane	112-40-3	None Found	None Found	None Found	None Found	None Found	None Found	Not Classified
Ethane	74-84-0	Soluble (20 °C) ^d	None Found	Flammable asphyxiant ^f	None Found	Simple asphyxiant ^f	None Found	Not Classified
Ethanol (ethyl alcohol)	64-17-5	(20°C) Miscible ^d	44 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, potassium dioxide, bromine pentafluoride, acetyl bromide, acetyl chloride, Pt, Na ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, liver, blood, repro sys ^d	Not Classified
Ethyl acetate	141-78-6	10% (77 °F) ^d	73 mm ^d	Class IB Flammable liquid ^d	Nitrates, strong oxidizers, alkalis and acids ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^d	Not Classified
Ethyl benzene	100-41-4	0.01% ^d	7 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	EPA Group D ^e
Ethylene	74-85-1	20% (0 °C) ^g	None Found		None Found	Simple asphyxiant ^f	None Found	Not Classified
Ethylene dichloride	107-06-2	0.9% ^d	64 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers & caustics; chemically-active metals (e.g., Mg or Al powder), Na and K; liquid ammonia ^d	Inh, abs, ing, con ^d	Eyes, skin, kidneys, liver CNS, CVS (In animals: fore- stomach, mammary gland & circulatory sys cancer) ^d	
Ethyl ether	60-29-7	8% ^d	440 mm ^d	Class IA Flammable liquid ^d	Strong oxidizers, halogens, sulfur, sulfur cmpds ^d	Inh, ing, con		Not Classified

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Table M.3.2-1.	Chemical Toxicity Profiles—Continued
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			Vapor			Route of	Target	
Compound	CAS No.	Solubility	Pressure	Flammability ^a	Incompatibilities	Exposure ^b	Organs	Carcinogenicity ^c
Ethylene glycol	107-21-1	Miscible ^d	0.06 mm ^d	Class III B Combustible liquid ^d	Strong oxidizers, chromium trioxide, potassium permanganate, sodium peroxide (hygroscopic) ^d	Inh, ing, con ^d		EPA Group D ^e
Ethylene glycol monoethyl ether (2-ethoxyethanol, Cellosolve [®])	110-80-5	Miscible ^d	4 mm ^d	Class II Combustible liquid ^a	Strong oxidizers ^d	Inh, abs, ing, con ^d	Eyes, resp, kidneys, liver, repro sys, hemato sys ^d	Not Classified
Ethyltriacetyoxysilane	None Found	None Found	None Found	None Found	None Found	None Found	None Found	Not Classified
Fluoride	None Found	cmpd ^d	Varies with cmpd ^d	Varies with cmpd ^d	Varies with cmpd ^d	None Found	None Found	Not Classified
Formaldehyde	50-00-0	Miscible ^d	>1 atm ^d	Flammable gas ^d	Strong oxidizers, alkalis; acids; phenols, urea, (Tends to polymerize; Reacts with HCl to form bis- chloromethyl ether) ^d	Inh, con ^a	Eyes, resp sys, (nasal cancer) ^d	EPA Group B1 ^e
Formic acid	64-18-6	Miscible ^d	35 mm ^d	Class II Combustible liquid ^d	Strong oxidizers, strong caustics, concentrated sulfuric acid ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^d	Not Classified
n-Heptane	142-82-5	0.0003 <i>%</i> ^d	40 mm (72 °F) ^d	Class 1B Flammable liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Skin, resp sys, CNS ^d	EPA Group D
n-Hexane	110-54-3	0.002% ^d	124 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Skin, eyes, CNS, PNS, resp sys ^d	EPA Group D ⁱ

Table M.3.2–1. Chemical Toxicity Profiles—Continued

			Vapor	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity
Compound Hydrazine	CAS No. 302-01-2	Solubility Miscible ^d	Pressure 10 mm ^d	Class IC Flammable liquid ^d	Oxidizers, hydrogen peroxide, nitric acid, metallic oxides, acids (Can ignite spontaneously on contact with oxidizers or porous materials such as earth, wood and cloth) ^d		Eyes, skin, resp sys, CNS, liver, kidneys (In animals: tumors of lung, liver, blood vessels and intestines) ^d	EPA Group B2 ^e
Hydrogen chloride (hydrochloric acid)	7647-01-0	67% (86 °F) ^d	40.5 atm ^d	Non- flammable gas ^d	Hydroxides, amines, alkalis, Cu, brass, Zn (Highly corrosive to metals) ^d	Inh, ing (soln), con ^d	Resp sys, skin, eyes ^d	Not Classified
Hydrogen cyanide	74-90-8	Miscible ^d	630 mm ^d	Class IA Flammable gas ^d	Amines, oxidizers, acids, sodium hydroxide, calcium hydroxide, sodium carbonate, water, caustics, ammonia (Can polymerize at 122-140 °F) ^d	Inh, abs, ing, con ^d	CNS, CVS, thyroid, blood ^d	Not Classified
Hydrogen fluoride (hydrofluoric acid)	7664-39-3	Miscible ^d	783 mm ^d	Non- flammable gas ^d	Metals, water or steam (Corrosive to metals. Will attack glass and concrete) ^d	Inh, abs(liq), ing (soln), con ^d	Eyes, skin, resp sys, bones ^d	Not Classified
Hydrogen peroxide	7722-84-1	Miscible ^d	5 mm (86 °F) ^d	Noncombus- tible liquid, but a powerful oxidizer ^d	Oxidizable materials, Fe, Cu, brass, bronze, Cr, Zn, Pb, Ag, Mn ^d	Inh, ing, con ^d		Not Classified
Hydrogen sulfide	7783-06-4	0.4% ^d	17.6 atm ^d	Flammable gas ^d	Strong oxidizers, strong nitric acid, metals ^d	Inh, con ^d	Resp sys, CNS, cyes ^d	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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	<u> </u>	~	Vapor	1 1 11 9	•	Route of	Target	Construction in the C
Compound	CAS No.	Solubility	Pressure	Flammability ^a	Incompatibilities	Exposure ^b	Organs	Carcinogenicity ^c
Iron salts (Soluble as Fe)	None	Varies with cmpd ^d	Varies with cmpd ^d	Non- combustible solids ^d	Varies with cmpd ^d	Ing, ing, con ^d	Eyes, skin, liver, resp sys, GI tract ^d	Not Classified
Isobutane	75-28-5	Slight ^d	3.1 atm (70 °F) ^d	Class IA Flammable gas ^d	Strong oxidizers (e.g., nitrates and perchlorates), chlorine, fluorine (Ni carbonyl + O_2) ^d	Inh, con (liq) ^d	CNS ^d	Not Classified
Isobutanol (Isobutyl alcohol)	78-83-1	10% ^d	9 mm ^d	Flammable ^g	Strong oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	Not Classified
Isobutyl acetate	110-19-0	0.6% (77 °F) ^d	13 mm ^d	Class 1B Flammable liquid	Nitrates, strong oxidizers, alkalis, acids ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	Not Classified
Isopropyl alcohol	67-63-0	Miscible ^d	33 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, acetaldehyde, chlorine, ethylene oxide, acids, isocyanates	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	Not Classified
Kerosene	8008-20-6	Insoluble ^d	5 mm (100 °F) ^d	Class II Combustible liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	IARC Group 2A (suspect carcinogen)
Lead	7439-92-1	Insoluble ^d	0 mm (approx) ^d	Non- combustible solid in bulk form ^d	Strong oxidizers, hydrogen peroxide, acids ^d	Inh, ing, con ^d	Eyes, GI tract, CNS, kidneys, blood, gingival tissue ^d	EPA Group B2 ^e
Lead chromate	7758-97-6 ^f	0.000007 (20 °C) ^h	Not Applicable	None Found	Potentially explosive reactions with azo-dye stuffs; Violent reaction with Al + dinitro- naphthalene + heat ^f	Con, ing, scu, ipr ^f	GI tract ^f	Confirmed carcinogen
	7439-93-2	None Found		None Found	None Found	None Found		Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Lithium hydride	7580-67-8	Reacts with water ^d	0 mm (approx) ^d	Combustible solid that can form airborne dust clouds which may explode on contact with flame heat, or oxidizers ^d	Strong oxidizers, halogenated hydrocarbons, acids, water (May ignite spontaneously in air) ^d	Inh, ing, con ^d	Skin, eyes, resp sys, CNS ^d	Not Classified
Magnesium (oxide fume)	1309-48-4	0.009% (86 °F) ^d	0 mm (approx) ^d	Non- combustible solid ^d	Chlorine trifluoride, phosphorus pentachloride ^d	Inh, con ^d	Eyes, resp sys ^d	Not Classified
Manganese (cmpds as fume; as Mn)	7439-96-5	Insoluble ^d	0 mm (approx) ^d	Metal: Combustible solid ^d	Oxidizers (Will react with water to produce H_2) ^d	Inh, ing ^d	Resp sys, blood, CNS, kidneys ^d	EPA Group D ^e
Mercury (Cmpds except organo alkyls; as Hg)	7439-97-6	Insoluble ^d	0.0012 mm ^d	Metal: Non- combustible liquid ^d	Acetylene, ammonia, chlorine dioxide, azides, calcium, sodium carbide, Li, Rb, Cu ^d	Inh, abs, inh, con ^d	Eyes, skin, resp sys, CNS, kidneys ^d	EPA Group D ^e
Methanol (methyl alcohol)	67-56-1	Miscible ^d	96 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, CNS, GI tract ^d	Not Classified
Methyl chloride	74-87-3	0.5% ^d	5.0 atm ^d	Flammable gas ^d	Chemically-active metals (e.g., Al, Zn, and Mg), water ^d	Inh, con (liq) ^d	CNS, liver, kidneys, repro sys ^d	Not Classified
Methylene chloride (dichloromethane)	75-09-2	2% ^d	350 mm ^d	Combustible liquid ^d	Strong oxidizers; caustics; chemically active metals (e.g. Al, Mg powders, K and Na), conc nitric acid ^d	Inh, abs, ing, con ^d	Skin, CVS, eyes, CNS (In animals: lung, liver, salivary gland, and mammary gland tumors) ^d	EPA Group B2 ^e

Table M.3.2–1.	Chemical Toxicity Profiles—Continued
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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
Methyl ethyl ketone (MEK; 2-butanone)	78-93-3	28% ^d	78 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, amines, ammonia, inorganic acids, caustics, Cu, isocyanates, pyridines ^d	Inh, ing, con ^d		EPA Group D ^e
Methyl isobutyl ketone (MIBK; Hexone)	108-10-1	2% ^d	16 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, potassium tert- butoxide ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, liver, kidneys ^d	Not Classified
Naphthalene (naphthene)	91-20-3	0.003% ^d	0.08 mm ^d	Combustible solid, but will take some effort to ignite ^d	Strong oxidizers, chromic anhydride ^d	Inh, abs, ing, con ^d	Eyes, skin, blood, liver, kidneys, CNS ^d	EPA Group D ^e
Nickel (refinery dust)	7440-02-0	Insoluble ^d	0 mm (approx) ^d	Metal: Combustible solid; Ni sponge catalyst ignites spontaneous- ly in air ^d	Strong acids, S, Se, wood and other combustibles, nickel nitrate ^d	Inh, ing, con ^d	Lungs, skin, nasal cavities (lung and nasal cancer) ^d	EPA Group A ^e
Nitric acid	7697-37-2	Miscible ^d	48 mm ^d	Noncombus- tible liquid, but increases flammability of combustible materials	Combustible materials; metallic powders; hydrogen sulfide; carbides; alcohols (Corrosive to metals) ^d	Inh, ing, con ^d	Eyes, resp sys, skin, teeth ^d	Not Classified
Nitrobenzene	98-95-3	0.2% ^d	0.3 mm (77 °F) ^d	Class IIIA Combustible liquid ^d	Conc. nitric acid, nitrogen tetroxide, caustics, phosphorous pentachloride, chemically-active metals (e.g., Sn, Zn) ^d	Inh, abs, ing, con ^d	Eyes, skin, blood, liver, kidneys, CVS, repro sys ^d	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

		C - I - 1 - 11	Vapor	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity
Compound 2-Nitropropane	CAS No. 74-46-9	Solubility 2% ^d	Pressure 13 mm ^d	Flammability Class IC Flammable liquid ^d	Amines; strong acids, alkalis & oxidizers; metal oxides; combustible materials ^d		Eyes, skin, resp sys, CNS, liver, kidneys (In animals: liver tumors) ^d	EPA Group B2 ^d
Octanol	None Found	None Found	None Found	None Found	None Found	None Found	None Found	Not Classified
Oxalic acid	144-62-7	14% ^d	<0.001 mm ^d	Combustible solid ^d	Strong oxidizers, silver cmpds, strong alkalis, chlorites ^d	Inh, ing, con ^d	Eyes, skin, resp sys, kidneys ^d	Not Classified
Ozone	10028-15-6	0.001% (32 °F) ^d	>1 atm ^d	Nonflammable gas, but a powerful oxidizer ^d	All oxidizable materials (inorganic and organic) ^d	Inh, con ^d	Eyes, resp sys ^d	Not Classifi e d
Phenol	108-95-2	9% (77 °F) ^d	0.4 mm	Sp. Gr: 1.06 Combustible solid	Strong oxidizer, calcium hypochlorite, aluminum, chloride, acids)	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys	EPA Group D ^e
Phosphoric acid	7664-38-2	Miscible ^d	0.03 mm ^d	Noncombus- tible liquid ^d	Strong caustics, most metals (Do not mix with solutions containing bleach or ammonia) ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^d	Not Classified
Phosphorous (yellow)	7723-14-0	0.0003% ^d	0.03 mm ^d	Flammable solid ^d	Air, oxidizers (Including elemental S and strong caustics), halogens (Ignites spontaneously in moist air) ^d	Inh, ing, con ^d	Eyes, skin, resp sys, liver, kidneys, jaw, teeth, blood ^d	Not Classified
Phosgene	75-44-5	Slight ^d	1.6 atm ^d	Non- flammable gas ^d	Moisture, alkalis, ammonia, alcohols, Cu ^d	Inh, con (liq) ^d	Eyes, skin, resp sys ^d	Not Classified
							None Found	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity
Potassium chromate (VI)	7789-00-6	Soluble in 1.6 parts cold water ^g	None Found	None Found	None Found	None Found	None Found	Confirmed carcinogen ^f
Potassium hydroxide	1310-58-10	107% (59 °F) ^d	1 mm (1317 °F) ^d	Noncombus- tible solid, may react with H ₂ 0 and other substances and generate sufficient heat to ignite combustible materials ^d	Acids, water, metals (When wet), halogenated hydrocarbons, maleic anhydride ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^a (throat, esophagus, mucous mem- branes) ^f	Not Classified
Propane	74-98-6	0.01% ^d	8.4 atm (70 °F) ^d	Flammable gas ^d	Strong oxidizers ^d	Inh, con (liq) ^d	CNS ^d	Not Classified
Propene (propylene)	115-07-1	None Found	7-8 atm ^g	Flammable gas ^g	None Found	Simple ashyxiant ^g	None Found	Not Classified
Propionaldehyde	123-38-6	Miscible ^f	None Found	Flammable liquid ^f	Oxidizers, vigorous polymerization reaction with methyl methacrylate ^f	Inh, ing, abs, con ^f	Skin, GI tract, resp sys ^f	Not Classified
Propylene oxide	75-56-9	41% ^d	445 mm ^d	Class 1A Flammable liquid ^d	Anhydrous metal chlorides; Fe; strong acids, caustics and peroxides ^d	Inh, ing, con ^d	Eyes, skin, resp sys ^d	EPA Group B2 ^e
Pyridine	110-86-1	Miscible ^d	16 mm ^d	Class IB combustible liquid ^d	Strong oxidizers, strong acids ^d	Inh, ing, abs, con ^d	Eyes, skin, CNS, liver, kidneys, GI tract ^d	Not Classified
Pyrene	129-00-0	Insoluble ^g	None Found	None Found	None Found	None Found	None Found	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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			Vapor			Route of	Target	
Compound	CAS No.	Solubility	Pressure	Flammability ^a	Incompatibilities	Exposure ^b	Organs	Carcinogenicity
Selenium	7782-49-2	Insoluble ^d	0 mm (approx) ^d	Combustible solid ^d	Acids, strong oxidizers, chromium trioxide, potassium bromate, Cd ^d	Inh, ing, con ^d	Eyes, skin, resp sys, liver, kidneys, blood, spleen ^d	EPA Group D ^e
Silicon	7440-21-3	Insoluble ^d	0 mm (approx) ^d	Combustible solid in powder form ^d	Chlorine, fluorine, oxidizers, calcium, cesium carbide, alkaline carbonates ^d	Inh, ing, con ^d	Skin, eyes, resp sys ^d	Not Classified
Silver	7440-22-4	Insoluble ^d	0 mm (approx) ^d	Metal: Non- combustible solid, but flammable as dust or powder ^d	Acetylene, ammonia, hydrogen peroxide, bromoazide, chlorine, trifluoride, ethyleneimine, oxalic acid, tartaric acid ^d	Inh, ing, con ^d	Nasal septum, skin, eyes ^d	EPA Group D ^e
Stoddard Solvent	8052-41-3	Insoluble	None Found	Class II Combustible liquid ^d	Strong oxidizers ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, kidneys ^d	Not Classified
Styrene	100-42-5	0.03% ^d	5 mm ^d	Class IC Flammable liquid ^d	Oxidizers, catalysts for vinyl polymers, peroxides, strong acids, aluminum chloride (May polymerize if contaminated or subjected to heat) ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, CNS, liver, repro sys ^d	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	I	Route of	Target	
Sulfuric acid	7664-93-9	Miscible ^d	0.001 mm ^d	Noncombus- tible liquid, but capable of igniting finely divided combustible materials ^d	Organic materials, chlorates, carbides,	Exposure ^b Inh, ing, con ^d	Organs Resp sys, eyes, skin, teeth ^d	Carcinogenicity ^c Not Classified
Tetrachloroethylene	127-18-4	0.02% ^d	14 mm ^d	Noncombus- tible liquid, but decomposes in a fire to HCl and phosgene ^d	Strong oxidizers, chemically-active metals (e.g., Li, Be & Ba), caustic soda, sodium hydroxide, potash ^d	Inh, abs, ing, con ^d	Eyes, skin, resp sys, liver, kidneys, CNS (In animals: liver tumors) ^d	Not Classified
Tetrahydrofuran (THF)	109-99-91	Miscible ^d	132 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers, Li-Al alloys ^d	Inh, ing, con ^d	Eyes, resp sys, CNS ^d	Not Classified
Titanium	7440-32-6	None Found	None Found	None Found	None Found	None Found	None Found	Not Classified
Toluene	108-88-3	0.07% (74 °F) ^d	21 mm ^d	Class IB Flammable liquid ^d	Strong oxidizers ^d	Inh, abs, ing, con ^d	CNS, eyes, resp sys, liver, kidneys, skin ^d	EPA Group D ^e
Tributyl phosphate	126-73-8	0.6% ^d	0.004 mm (77 °F) ^d	Class III B ^d	Alkalis, oxidizers, water, moist air	Inh, ing, con ^d	Eyes, skin, resp. sys.	Not Classified
1,1,1-Trichloroethane (TCA; methyl chloroform)	71-55-6	0.4% ^d	100 mm ^d	Combustible liquid, but burns with difficulty ^d	Strong caustics; strong oxidizers; chemically-active metals (e.g., Zn, Al, Mg powders, Na, and K); water ^d	Inh, ing, con ^d	CNS, eyes, skin, liver, CVS ^d	EPA Group D ^e

Table M.3.2-1. Chemical Toxicity Profiles—Continued

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Compound	CAS No.	Solubility	Vapor Pressure	Flammability ^a	Incompatibilities	Route of Exposure ^b	Target Organs	Carcinogenicity ^c
1,1,2-Trichloroethane	79-00-5	0.4% ^d	19 mm ^d	Combustible liquid, forms dense soot ^d	Strong oxidizers & caustics; chemically-active metals (e.g., Al, M powders, Na, and K) ^d	Inh, abs, ing, con ^d	Eyes, resp sys, CNS, liver, kidneys (In animals: liver cancer) ^d	Not Classified
Trichloroethylene (TCE, trichloroethene)	79-01-6	0.0001% (77 °F) ^d	58 mm ^d	Combustible liquid, but burns with difficulty ^d	Strong caustics & alkalis; chemically-active metals (e.g., Ba, Li, Na, Mg, Ti, and Be) ^d	Inh, abs, ing, con ^d	Eyes, resp sys, heart, liver, CNS, skin (In animals: liver and kidney cancer) ^d	EPA Group B2 ⁱ
Trichlorotrifluoroethane (Freon 113)	76-13-1	0.02% (77 °F) ^d	285 mm ^d	Noncombus- tible liquid at ordinary temp, but will ignite and burn weakly at 1256 °F ^d	Chemically-active metals (e.g., Ca, powdered Al, Zn, Mg and Be) ^d	Inh, ing, con ^d	,	Not Classified
1,2,4-Trimethylbenzene	95-63-6	0.006% ^d	1 mm (56 °F) ^d	Class II Flammable liquid ^d	Oxidizers, nitric acid ^d	Inh, ing, con ^d	Eyes, skin, resp sys, CNS, blood ^d	Not Classified
Tungsten (insoluble compounds)	7440-33-7	Insoluble ^d	0 mm (approx) ^d	Combustible as fine powder; may ignite sponta- neously ^d	Bromine trifluoride, chlorine trifluoride, F_2 , I_2 , pentafluoride ^d	Inh, ing, con ^d	Eyes, skin, resp sys, blood ^d	Not Classified
VM&P Naphtha	8032-32-4	Insoluble	2-20 mm	Class IB Flammable liquid ^d	None Found	Inh, ing, con ^d	Eyes, skin, resp sys, CNS ^d	Not Classified

Table M.3.2–1. Chemical Toxicity Profiles—Continued

Flammable liquids are classified by OSHA Class IAflash point below 73 °F and boili Class IBflash point below 73 °F and boili Class ICflash point at or above 73 °F and Class II flash point at or above 100 °F and Class IIIA flash point at or above 140 °F Class IIIBflash point at or above 200 °F. DHHS 1992a. Routes of exposure abbreviated as follows: inh-inhalation	compo- nent of fumes ^d o : 0.02% ^d -3 m: slight ^d -3 p: 0.02% ^d 6 None Found 29 CFR 1910.106 g point below 100 g point at or abov below 140 °F. below 140 °F.) °F.	Flammability ^a Varies with component of fumes ^d o-, m-, p-: Class IC Flammable liquids ^d Combustible solid ^g	Varies with component of fumes ^d o-, m-, p-: Strong oxidizers, strong acids ^d None Found	Inh, con ^d o-, m-, p-: inh, abs, ing, con ^d Inh ^g	Eyes, skin, resp sys, CNS ^d o-, m-, p-: Eyes, skin, resp sys, CNS, GI tract, liver, blood, kidneys ^d None Found	Not Classified Not Classified
m:108-33 p: 106-42 Zinc 7440-66 Flammable liquids are classified by OSHA Class IAflash point below 73 °F and boili Class IBflash point below 73 °F and boili Class IIflash point at or above 73 °F and Class IIflash point at or above 100 °F and Class IIIflash point at or above 100 °F and Class IIIflash point at or above 100 °F. DHHS 1992a. Routes of exposure abbreviated as follows: inh-inhalation	-3 m: slight ^d -3 p: 0.02% ^d 6 None Found 29 CFR 1910.106 19 point below 100 19 point at or abov below 140 °F. below 140 °F.	m 9 mm ^d p: 9 mm ^d d None Found	Class IC Flammable liquids ^d Combustible	Strong oxidizers, strong acids ^d	inh, abs, ing, con ^d	Eyes, skin, resp sys, CNS, GI tract, liver, blood, kidneys ^d	
 Flammable liquids are classified by OSHA Class IAflash point below 73 °F and boili Class IBflash point below 73 °F and boili Class ICflash point at or above 73 °F and Class II flash point at or above 100 °F and Class IIIA flash point at or above 140 °F Class IIIBflash point at or above 200 °F. DHHS 1992a. Routes of exposure abbreviated as follows: inh-inhalation 	29 CFR 1910.106 g point below 100 g point at or abov below 140 °F. below 140 °F.) as follows:) °F.		None Found	Inh ^g	None Found	Not Classified
Class IBflash point below 73 °F and boili Class ICflash point at or above 73 °F and Class II flash point at or above 100 °F and Class IIIA flash point at or above 140 °F Class IIIBflash point at or above 200 °F. DHHS 1992a. ⁹ Routes of exposure abbreviated as follows: inh-inhalation	ng point below 100 ng point at or abov pelow 140 °F. below 140 °F.) °F.	50110*		<u></u>		
abs-skin absorption ing-ingestion con-skin and/or eye contact ipr-intraperitoneal scu-subcutaneous.							
EPA Groups for carcinogenicity are classifi EPA Group A: Human Carcinogen EPA Group B1: Probable Human Carcinog EPA Group B2: Probable Human Carcinoge EPA Group C: Possible Human Carcinoger EPA Group D: Not Classifiable as to Human	n-limited evidenc n-sufficient evider	e in human studie nce from animal s	es studies, inadequate	evidence or no data fror	n human studies		
¹ NIOSH 1994a. ² ORNL 1994b.							
UKINL 19940.							

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ⁱ EPA 1993a.

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M.3.3 REGULATED EXPOSURE LIMITS

Hazardous chemicals are regulated by various agencies in order to provide protection to the public (EPA regulated) and to workers OSHA, while others (National Institute for Occupational Safety and Health [NIOSH] and the ACGIH) provide guidelines. The RfDs and RfCs set by EPA represent exposure limits for long-term (chronic) exposure at low doses and concentrations, respectively, that can be considered safe from non-cancerous effects. The PEL represents concentration levels set by OSHA that are safe for 8-hr exposures without causing non-cancerous effects. The slope factor, or the unit risk, is used to convert the daily uptake of a carcinogenic chemical averaged over a lifetime to the incremental risk of an individual developing cancer. Table M.3.3–1 presents the information on exposure limits used to develop HQs for each of the hazardous chemicals and the HIs derived from their summation and the slope factors used to calculate cancer risk for each chemical at the exposure concentrations identified at the various sites or associated with a proposed alternative action.

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Acetaldehyde	75-07-0	0.003 ^d	9x10 ^{-3e}	EPA Group B2 ^e	none found	OSHA-PEL: 360 mg/m ³ ACGIH-TLV: 180 mg/m ³ , STEL: 270 mg/m ³ NIOSH-REL: 3,660 mg/m ³
Acetic acid	64-19-7	0.175 ^f	0.6125 ^g	not classifi e d	none found	OSHA-PEL: 25 mg/m ³ ACGIH-TLV: 25 mg/m ³ , STEL: 37 mg/m ³ NIOSH-REL: 25 mg/m ³ , STEL: 37 mg/m ³ IDLH: 125 mg/m ³
Acetone	67- 6 4-1	0.1 ^e	0.35 ^g	EPA Group D ^e	none found	OSHA-PEL: 2,400 mg/m ³ ACGIH-TLV: 1,780 mg/m ³ NIOSH-REL: 590 mg/m ³ STEL: 2,380 mg/m ³ IDLH: 6,050 mg/m ³
Acetonitrile	75-05-8	0.006 ^e	0.021 ^g	not classifi c d	none found	OSHA-PEL: 70 mg/m ³ ACGIH-TLV: 67 mg/m ³ STEL: 101 mg/m ³ NIOSH-REL: 34 mg/m ³ IDLH: 855 mg/m ³
Acetylene	74-86-2	18.634 ^h	65.219 ^g	not classified	none found	OSHA-PEL: 2,662 mg/m ³ⁱ NIOSH-REL: 2,662 mg/m ³
Aluminum	7429-90-5	0.105 ^f	0.368 ^g	not classified	none found	OSHA-PEL: 15 mg/m ³ ACGIH-TLV: 10 mg/m ³ , (dust) NIOSH-REL: 10 mg/m ³
Aluminum welding fumes	none	0.035 ^h	0.1225 ^g	not classified	none found	NIOSH-REL: 5 mg/m ³

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Ammonia	7664-41-7	2.86x10 ^{-2d} , (34 mg/L, chronic) ^j	0.1°	EPA Group D ^k	none found	OSHA-PEL: 35 mg/m ³ ACGIH-TLV: 17 mg/m ³ , STEL: 24 mg/m ³ NIOSH-REL: 18 mg/m ³ , STEL: 27 mg/m ³ IDLH: 213 mg/m ³
Ammonium hydroxide	1336-21-6	0.014 ¹	0.049 ^g	not classified	none found	LD50 (oral-rat): 350 mg/kg ⁱ PEL: 2 mg/m ^{3m}
Antimony (nonradionuclide)	7440-36-0	4x10 ^{-4e}	1.4x10 ^{-3g}	EPA Group D ^k	none found	OSHA-PEL: 0.5 mg/m ³ ACGIH-TLV: 0.5 mg/m ³ NIOSH-REL: 0.5 mg/m ³ , IDLH: 50 mg/m ³ , IDLH: 50 mg/m ³
Arsenic	7440-38-2	3x10 ^{-4e}	1.05x10 ^{-3g}	EPA Group A ^e	50 (inhal) ^j	OSHA-PEL: 0.01 mg/m ³ⁱ ACGIH-TLV: 0.2 mg/m ³ NIOSH-REL: 0.002 mg/m ³ (ceiling), IDLH: 5 mg/m ³
Barium	7440-39-3	0.07 ⁿ	0.245 ^g	not classified	none found	OSHA-PEL: 0.5 mg/m ³ⁱ ACGIH-TLV: 0.5 mg/m ³ (sol. cmpds. as Ba)
Benzene	71-43-2	2.28x10 ^{-2f}	0.0798 ^g	EPA Group A	0.029 (oral) ^e 0.029 (inhal) ^j	OSHA-PEL: 3.25 mg/m ³ , STEL: 16.25 mg/m ³ ACGIH-TLV: 32 mg/m ³ NIOSH-REL: 0.325 mg/m ³ , STEL: 3.25 mg/m ³ , IDLH: 5 mg/m ³
Beryllium	7440-41-7	5x10 ^{-3e}	0.0175 ^g	EPA Group B2	4.3 (oral) ^e 8.4 (inhal) ^j	OSHA-PEL: 0.002 mg/m ³ , 0.005 mg/m ³ (ceiling) ACGIH-TLV: 0.002 mg/m ³ NIOSH-REL: 0.0005 mg/m ³ , IDLH: 4 mg/m ³
Bismuth	7440-69-9	none found	none found	not classified	none found	none found
Boric acid	10043-35-3	5.7x10 ^{-3f}	0.02 ^g	not classified	none found	PEL: 0.816 mg/m ^{3m} LD50 (oral, rat): 2,660 mg/kg ⁱ

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,}
1,3-Butadiene	106-99-0	15.4 ^f	53.9 ^g	EPA Group B2 ^e	1.8 (inhal) ^j	OSHA-PEL: 2,200 mg/m ³ ACGIH-TLV: 22 mg/m ³ NIOSH-REL: 4,500 mg/m ³
Butane	106-97-8	13.3 ^h	46.55 ^g	not classified	none found	NIOSH-REL: 1,900 mg/m ³ ACGIH-TLV: 1,900 mg/m ³ PEL: 1900 mg/m ^{3m}
1-Butene	106-98-9	none found	none found	not classified	none found	none found
2-Butoxyethanol	111-76-2	1.68 ^f	5.88 ^g	not classified	none found	OSHA-PEL: 240 mg/m ³ (skin) ACGIH-TLV: 121 mg/m ³ NIOSH-REL: 24 mg/m ³ (skin) IDLH: 3,437 mg/m ³
n-Butyl alcohol (1-Butanol)	71-36-3	0.10 ^e	0.35 ^g	EPA Group D ^e	none found	OSHA-PEL: 300 mg/m ³ ACGIH-TLV: 152 mg/m ³ (skin, ceiling) NIOSH-REL: 150 mg/m ³ (skin), IDLH: 4,312 mg/m ³
Butyl lactate	138-22-7	0.21 ^f	0.74 ^g	not classified	none found	PEL: 30 mg/m ³ ACGIH-TLV: 30.0 mg/m ³ NIOSH-REL: 25.0 mg/m ³
Cadmium oxide (fume, as Cd)	1306-19-0	3.5x10 ^{-5f}	1.23x10 ^{-4g}	not classified	none found	OSHA-PEL: 0.005 mg/m ³ ACGIH-TLV: 0.05 mg/m ³ (ceiling) NIOSH-STEL: 9 mg/m ³
Cadmium dust (nonradionuclide)	7440-43-9	5x10 ^{-4e}	1.75x10 ^{-3g}	EPA Group B1 ^e	6.3x10 ⁻³⁰ (Unit Risk: 1.8x10 ⁻⁶ mg/m ³) ⁿ	OSHA-PEL: 0.005 mg/m ³ ACGIH-TLV: 0.05 mg/m ³
Calcium	7440-70-2	none found	none found	not classified	none found	none found
Carbon dioxide	124-38-9	63 ^f	221 ^g	not classified	none found	OSHA-PEL: 9,000 mg/m ³ ACGIH-TLV: 9,000 mg/m ³ , STEL: 54,000 mg/m ³ NIOSH-REL: 9,000 mg/m ³ , STEL: 54,000 mg/m ³ , IDLH: 7.3x10 ⁴ mg/m ³

Table M.3.3-1. Table of Exposure Limits—Continued

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Carbon disulfide	75-15-0	0.1 ^e	0.35 ^g	not classifi c d	none found	OSHA-PEL: 63.2 mg/m ³ ACGIH-TLV: 31 mg/m ³ (skin) NIOSH-REL: 3 mg/m ³ , STEL: 30 mg/m ³ , IDLH: 1,580 mg/m ³
Carbon monoxide	630-08-0	0.385 ^f	1.35 ^g	not classified	none found	OSHA-PEL: 55 mg/m ³ ACGIH-TLV: 29 mg/m ³ NIOSH-REL: 40 mg/m ³ , IDLH: 1,392 mg/m ³
Carbon tetrachloride	56-23-5	7x10 ^{-4e}	2.45x10 ^{-3g}	EPA Group B2 ^e	0.13 (oral) 0.053 (inhal) ^j	OSHA-PEL: 63.9 mg/m ³ , 160 mg/m ³ (ceiling) ACGIH-TLV: 31 mg/m ³ (skin) NIOSH-STEL: 12.6 mg/m ³ IDLH: 1,278 mg/m ³
Chloride (Sodium chloride)	77647-14-5	0.12 ¹	0.42 ^g	not classified	none found	LD50: 3,000 mg/m ³ⁱ PEL: 17.1 mg/m ^{3m}
Chlorine	7782-50-5	0.1 ^e	0.35 ^g	EPA Group D ^k	none found	OSHA-PEL: 3 mg/m ³ (ceiling) ACGIH-TLV: 1.5 mg/m ³ , STEL: 2.9 mg/m ³ NIOSH-REL: 1.45 mg/m ³ (ceiling, 15 min.), IDLH: 29.5 mg/m ³
Chlorobenzene	108-90 - 7	0.02 ^e	0.07 ^g	EPA Group D ^e	none found	OSHA-PEL: 350 mg/m ³ ACGIH-TLV: 46 mg/m ³ NIOSH-REL: 4,680 mg/m ³
Chloroform	67-66-3	0.01 ^e	0.035 ^g	EPA Group B2 ^e	6.1x10 ⁻³ (oral) ^e 0.081 (inhal) ^j	OSHA-PEL: 240 mg/m ³ (ceiling) ACGIH-TLV: 49 mg/m ³ NIOSH-REL: 9.78 mg/m ³ (60 min IDLH: 2,480 mg/m ³
bis-Chloromethyl ether	542-88-1	3.29x10 ^{-5r}	1.15x10 ^{-4g}	EPA Group A ^e	220 ^j	PEL: 0.0047 mg/m ^{3m} ACGIH-TLV: 0.0047 mg/m ³

Table M.3.3–1. Table of Exposure Limits—Continued

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,}
Chromium (Hexavalent)	18540-29-9	5x10 ⁻³	0.0175 ^g	EPA Group A	41 (inhal) ^j	OSHA-PEL: 1 mg/m ³ⁱ ACGIH-TLV: 0.05 mg/m ³ (water soluble)
Chromium (Trivalent)	16065-83-1	1¢	3.5 ^g	not classified	none found	OSHA-PEL: 0.5 mg/m ³ ACGIH-TLV: 0.5 mg/m ³ NIOSH-REL: 0.5 mg/m IDLH: 25 mg/m ³
Cobalt (metal dust and fume)	7440-48-4	7x10 ^{-4f}	2.45x10 ^{-3g}	not classified	none found	OSHA-PEL: 0.1 mg/m ³ ACGIH-TLV: 0.05 mg/m ³ NIOSH-REL: 0.05 mg/m ³ , IDLH: 20 mg/m ³
Copper (dusts and mists)	7440-50-8	0.007 ^f	0.0245 ^g	EPA Group D ^e	non e found	OSHA-PEL: 1 mg/m ³ ACGIH-TLV: 1 mg/m ³ NIOSH-REL: 1 mg/m ³ , IDLH: 100 mg/m ³
Cresol (m-cresol, cresylic acid) ^p	108-39-4	0.154 ^f	0.539 ^g	not classified	none found	OSHA-PEL: 22 mg/m ³ (skin) ACGIH-TLV: 22 mg/m ³ (skin, all isomers) NIOSH-REL: 10 mg/m ³ , IDLH: 1,125 mg/m ³
Cyclohexane	110-82-7	7.35 ^f	25.725 ^g	not classified	none found	OSHA-PEL: 1,050 mg/m ³ ACGIH-TLV: 1,030 mg/m ³ NIOSH-REL: 1,050 mg/m ³ , IDLH: 4,550 mg/m ³
Cyclohexanone	108-94-1	5 ^e	17.5 ^g	not classifi c d	none found	OSHA-PEL: 200 mg/m ³ ACGIH-TLV: 100 mg/m ³ (skin) NIOSH-REL: 100 mg/m ³ (skin)
Cyclopentane	287-92-3	12.05 ^f	42,18 ^g	not classifi e d	none found	OSHA-PEL: 1,721 mg/m ³ⁱ ACGIH-TLV: 1,720 mg/m ³ NIOSH-REL: 1,720 mg/m ³
Dibenzofuran	132-64-9	none found	none found	EPA Group D ^e	none found	none found

Table M.3.3-1. Table of Exposure Limits—Continued

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Dibutyl phosphate	107-66-4	0.035 ^f	0.1225 ^g	not classified	none found	OSHA-PEL: 5 mg/m ³ⁱ ACGIH-TLV: 8.6 mg/m ³ , STEL: 17 mg/m ³ NIOSH-REL: 5 mg/m ³ STEL: 10 mg/m ³
o-Dichlorobenzene (1,2-Dichlorobenzene)	95-50-1	0.09 ^e	0.315 ^g	EPA Group D	none found	OSHA-PEL: 300 mg/m ³ (ceiling) ACGIH-TLV: 150 mg/m ³ , STEL: 301 mg/m ³ NIOSH-REL: 300 mg/m ³ , IDLH: 1,222 mg/m ³
3,3-Dichlorobenzidine	91-94-1	none found	none found	B2°	0.45 ^e	none found
(and salts) Dichlorodifluoromethane	75-71-8	0.2 ^e	0.7 ^g	EPA Group D ^k	none found	OSHA-PEL: 4,950 mg/m ³ ACGIH-TLV: 4,950 mg/m ³ NIOSH-REL: 4,950 mg/m ³ IDLH: 75,450 mg/m ³
Dichloromethane (Methylene chloride)	75-09-2	0.06 ^e	3.0 ^g	EPA Group B2 ^e	7.5x10 ⁻³ (oral) ^e	OSHA-PEL: 1,765 mg/m ³ , 3,530 mg/m ³ (ceiling) ACGIH-TLV: 174 mg/m ³ NIOSH-REL: 8,119 mg/m ³
Dimethylformamide (DMF)	68-12-2	8.58x10 ^{-3d}	0.03 ^e	not classified	none found	OSHA-PEL: 30 mg/m ³ (skin) ACGIH-TLV: 30 mg/m ³ (skin) NIOSH-REL: 30 mg/m ³ (skin), IDLH: 1,520 mg/m ³
2,4,-Dinitrotoluene	25321-14-6	0.002 ^e	0.007 ^g	not classified	none found	OSHA-PEL: 1.5 mg/m ³ (skin) ACGIH-TLV: 0.15 mg/m ³ (skin) NIOSH-REL: 1.5 mg/m ³ (skin), IDLH: 50 mg/m ³

Table M.3.3-1. Table of Exposure Limits—Continued

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Table M.3.3–1. Table of Exposure Limits—Continued

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Di-sec octyl phthalate	117-81-7	0.02 ^q	0.07 ^g	not classifi e d	none	OSHA-PEL: 5 mg/m ³ ACGIH-TLV: 5 mg/m ³ , STEL: 10 mg/m ³ NIOSH-REL: 5 mg/m ³ , STEL: 10 mg/m ³ IDLH: 5,000 mg/m ³
1,4-Dioxane	123-91-1	2.52 ^f	8.82 ^g	EPA Group B2	0.011 ^e	OSHA-PEL: 360 mg/m ³ (skin) ACGIH-TLV: 90 mg/m ³ (skin) NIOSH-REL: 3.6 mg/m ³ (ceiling, 30-min), IDLH: 1,830 mg/m ³
Dodecane	112-40-3	none found	none found	not classified	none found	none found
Ethane	74-84-0	none found	none found	not classified	none found	none found
Ethyl acetate	141-78-6	0.9 ^e	3.15 ^g	not classifi c d	none found	OSHA-PEL: 1,400 mg/m ³ ACGIH-TLV: 1,440 mg/m ³ NIOSH-REL: 1,400 mg/m ³ , IDLH: 7,320 mg/m ³
Ethyl alcohol	64-17-5	13.3 ^f	46.55 ^g	not classified	non e found	OSHA-PEL: 1,900 mg/m ³ ACGIH-TLV: 1,880 mg/m ³ NIOSH-REL 1,900 mg/m ³ IDLH: 6,336 mg/m ³
Ethyl benzene	100-41-4	0.1 ^e	1.0 ^e	EPA Group D ^e	none found	OSHA-PEL: 435 mg/m ³ ACGIH-TLV: 434 mg/m ³ , STEL: 543 mg/m ³ NIOSH-REL: 435 mg/m ³ , STEL: 545 mg/m ³ IDLH: 3,528 mg/m ³
Ethyl ether	60-29-7	0.2 ^e	0.7 ^g	not classifi c d	none found	OSHA-PEL: 1,200 mg/m ³ ACGIH-TLV: 1,210 mg/m ³ , STEL: 1,520 mg/m ³ IDLH: 5,852 mg/m ³
Ethylene	74-85-1	none found	none found	not classified	none found	none found

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Ethylene dichloride (1, 2-Dichloroethane)	107-06-2	1.44 ^f	5.03 ^g	EPA Group B2 ^e	0.091 (oral) ^e 0.091 (inhal) ^j	OSHA-PEL: 205.5 mg/m ³ , 411 mg/m ³ (ceiling) 822 mg/m ³
						(5-min max peak any 3 hrs) ACGIH-TLV: 40 mg/m ³ , NIOSH-REL: 4 mg/m ³ , STEL: 8 mg/m ³ IDLH: 205.5 mg/m ³
Ethylene glycol	107-21-1	2.0 ^e	7.0 ^g	EPA Group D ^k	none found	ACGIH-TLV: 127 mg/m ³ (ceiling) PEL: 127 mg/m ^{3m}
Ethylene glycol monoethyl ether (2-Ethoxyethanol)	110-80-5	5.18 ^f	18.13 ^g	not classified	none found	OSHA-PEL: 740 mg/m ³ (skin) ACGIH-TLV: 18 mg/m ³ (skin) NIOSH-REL: 1.8 mg/m ³ (skin), IDLH: 1,875 mg/m ³
Tel-de-is seture rusilone	none	none	none	not classified	none found	none found
Ethyltriacetyoxysilane Fluoride	16984-48-8	0.0175 ^f	0.061 ^g	not classified	none found	OSHA-PEL: 2.5 mg/m ³ⁱ ACGIH-TLV: 2.5 mg/m ³
Formaldehyde	50-00-0	0.2 ^e	0.7 ^g	EPA Group B1 ^e	0.045 (inhal) ^j	OSHA-PEL: 0.9375 mg/m ³ , STEL: 2.5 mg/m ³ ACGIH-TLV: 0.37 mg/m ³ (ceiling NIOSH-REL: 0.02 mg/m ³ , 0.125 mg/m ³ (ceiling, 15 min.), IDLH: 25 mg/m ³
Formic Acid	64-18-6	0.063 ^f	0.221 ^g	not classified	none found	OSHA-PEL: 9 mg/m ³ ACGIH-TLV: 9.4 mg/m ³ , STEL: 19 mg/m ³ NIOSH-REL: 9 mg/m ³ , IDLH: 57.3 mg/m ³
n-Heptane	142-82-5	14 ^e	49 ^g	EPA Group D	none found	OSHA-PEL: 2,000 mg/m ³ ACGIH-TLV: 1,640 mg/m ³ STEL: 2,050 mg/m ³ NIOSH-REL: 350 mg/m ³ CEILING (15 min): 1,800 mg/m

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Table M.3.3-1. Table of Exposure Limits-Continued

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ^{•1}	Occupational Exposure Levels ^{b,c}
n-Hexane	110-54-3	0.06 ^d	0.2 ^e	EPA Group D ^k	none found	OSHA-PEL: 1800 mg/m ³ ACGIH-TLV: 176 mg/m ³ NIOSH-REL: 180 mg/m ³ , IDLH: 3,938 mg/m ³
Hydrazine	302-01-2	9.31x10 ^{-3f}	3.26x10 ^{-2g}	EPA Group B2 ^e	3.0 (oral) ^e 17 (inhal) ^j	OSHA-PEL: 1.33 mg/m ³ (skin) ACGIH-TLV: 0.13 mg/m ³ (skin) NIOSH-REL: 0.04 mg/m ³ (ceiling, 2 hr.), IDLH: 66.5 mg/m ³
Hydrochloric acid	7647-01-0	2x10 ^{-3d}	7x10 ^{-3e}	not classified	non e found	OSHA-PEL: 7 mg/m ³ ACGIH-TLV: 7.5 mg/m ³ (ceiling) NIOSH-REL: 7 mg/m ³ IDLH: 76 mg/m ³
Hydrogen chloride	7647-01-0	2x10 ^{-3d}	7x10 ^{-3e}	not classified	non e found	OSHA-PEL: 7 mg/m ³ ACGIH-TLV: 7.5 mg/m ³ (ceiling) NIOSH-REL: 7 mg/m ³ IDLH: 76 mg/m ³
Hydrogen cyanide	74-90-8	0.02 ^e	0.07 ^g	not classified	none found	OSHA-PEL: 11 mg/m ³ (skin) ACGIH-TLV: 11 mg/m ³ (skin, ceiling) NIOSH-REL: 5 mg/m ³ (skin), IDLH: 56 mg/m ³
Hydrogen fluoride	7664-39-3	0.06 ^q	0.21 ^g	not classifi e d	none found	OSHA-PEL: 2.49 mg/m ³ ACGIH-TLV: 2.6 mg/m ³ (ceiling) NIOSH-REL: 2.5 mg/m ³ , 5 mg/m ³ (ceiling, 15 min), IDLH: 24.9 mg/m ³
Hydrogen peroxide	7722-84-1	0.0098 ^f	0.0343 ^g	not classifi e d	none found	OSHA-PEL: 1.4 mg/m ³ ACGIH-TLV: 1.4 mg/m ³ NIOSH-REL 1.4 mg/m ³ IDLH: 105.75 mg/m ³

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Hydrogen sulfide	7783-06-4	3x10 ^{-3e}	9x10 ^{-4e}	not classified	none found	OSHA-PEL: 28.4 mg/m ³ (ceiling) ACGIH-TLV: 14 mg/m ³ , STEL: 21 mg/m ³ NIOSH-REL: 15 mg/m ³ (ceiling, 10 min), IDLH: 142 mg/m ³
Iron (salts)	none found	0.007 ^r	0.0245 ^g	not classified	none found	PEL: 1 mg/m ^{3m} ACGIH-TLV: 1 mg/m ³ NIOSH-REL: 1 mg/m ³
Isobutane	75-28-5	13.3 ^h	46.55 ^g	not classified	none found	PEL: 1900 mg/m ^{3m} NIOSH-REL: 1900 mg/m ³
Isobutyl acetate	110-19-0	4.9 ^f	17.15 ^g	not classified	none found	OSHA-PEL: 700 mg/m ³ ACGIH-TLV: 713 mg/m ³ NIOSH-REL: 700 mg/m ³ IDLH: 6,279 mg/m ³
Isobutyl alcohol (isobutanol)	78-83-1	0.3 ^e	1.05 ^g	not classified	none found	OSHA-PEL: 300 mg/m ³ ACGIH-TLV: 152 mg/m ³ NIOSH-REL: 150 mg/m ³ , IDLH: 4,928 mg/m ³
Isopropyl alcohol	67-63-0	6.9 ^q	24.15 ^g	not classified	none found	OSHA-PEL: 980 mg/m ³ ACGIH-TLV: 983 mg/m ³ STEL: 1,230 mg/m ³ NIOSH-REL: 980 mg/m ³ STEL: 1,225 mg/m ³ IDLH: 5,000 mg/m ³
Kerosene	8008-20-6	0.7 ^h	2.45 ^g	IARC Group 2A ⁱ (suspect carcinogen)	none found	NIOSH-REL: 100 mg/m ³
Lead	7439-92-1	3.5x10 ^{-4f}	1.225x10 ^{-3g}	EPA Group B2 ^e	none found	OSHA-PEL: 0.05 mg/m ³ ACGIH-TLV: 0.15 mg/m ³ NIOSH-REL: 0.1 mg/m ³ , IDLH: 100 mg/m ³

Table M.3.3–1. Table of Exposure Limits—Continued

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Lead chromate	7758-97-6	0.00035 ^r	0.001225 ^g	ACGIH Group A2 ⁱ (suspect human carcinogen)	none found	OSHA-PEL: 0.05 mg/m ³ⁱ ACGIH-TLV (as Pb): 0.05 mg/m ³
Lithium	7439-93-2	none found	none found	not classified	none found	none found
Lithium hydride	7580-67-8	1.75x10 ^{-4f}	6.125x10 ^{-4g}	not classified	none found	OSHA-PEL: 0.025 mg/m ³ ACGIH-TLV: 0.025 mg/m ³ NIOSH-REL: 0.025 mg/m ³ , IDLH: 0.5 mg/m ³
Magnesium (oxide fume)	1309-48-4	0.105 ^f	0.368 ^g	not classified	none found	OSHA-PEL: 15 mg/m ³ ACGIH-TLV: 10 mg/m ³ NIOSH-REL: 750 mg/m ³
Manganese	7439-96-5	1.43x10 ^{-5d}	5x10 ^{-5e}	EPA Group D ^e	none found	OSHA-PEL: 5 mg/m ³ (ceiling) ACGIH-TLV: 5 mg/m ³ (dust and compounds) NIOSH-REL: 1 mg/m ³ , STEL: 3 mg/m ³ , IDLH: 500 mg/m ³
Mercury (vapor)	7439-97-6	3x 10 ⁻⁴ (inorganic, chronic) ^j	3x10 ^{-4q}	EPA Group D ^e	none found	OSHA-PEL: 0.1 mg/m ³ (ceiling) ACGIH-TLV: 0.05 mg/m ³ NIOSH-REL: 0.05 mg/m ³ (skin), IDLH: 10 mg/m ³
Methanoi (methyl alcohol)	67-56-1	0.5 ^e	1.75 ^g	not classified	none found	OSHA-PEL: 260 mg/m ³ ACGIH-TLV: 262 mg/m ³ (skin), STEL: 328 mg/m ³ NIOSH-REL: 260 mg/m ³ , STEL: 325 mg/m ³ (skin), IDLH: 7,980 mg/m ³
Methyl chloride	74-87-3	1.47 ^f	5.145 ^g	not classified	none found	OSHA-PEL: 210 mg/m ³ ACGIH-TLV: 103 mg/m ³ STEL: 207 mg/m ³

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Methylene chloride (dichloromethane)	75-09-2	0.06 ^e	3.0 ^q	EPA Group B2 ^c	7.5x10 ⁻³ (oral) ^e	OSHA-PEL: 1,765 mg/m ³ , 3,530 (ceiling) ACGIH-TLV: 174 mg/m ³ NIOSH-IDLH: 8,119 mg/m ³
Methyl ethyl ketone (MEK; 2-Butanone)	78-93-3	1.0 ^e	1.0 ^e	EPA Group D ^e	none found	OSHA-PEL: 590 mg/m ³ ACGIH-TLV: 590 mg/m ³ , STEL: 885 mg/m ³ NIOSH-REL: 590 mg/m ³ , STEL: 885 mg/m ³ , IDLH: 9,000 mg/m ³
Methyl isobutyl ketone (hexone)	108-10-1	0.08 (chronic) ^j	0.28 ^g	not classified	none found	OSHA-PEL: 410 mg/m ³ ACGIH-TLV: 205 mg/m ³ , STEL: 307 mg/m ³ NIOSH-REL: 205 mg/m ³ , STEL: 300 mg/m ³ , IDLH: 2,085 mg/m ³
Naphthalene (napthene)	91-20-3	0.35 ^f	1.225 ^g	EPA Group D ^e	none found	OSHA-PEL: 50 mg/m ³ ACGIH-TLV: 52 mg/m ³ , STEL: 79 mg/m ³ NIOSH-REL: 50 mg/m ³ , STEL: 75 mg/m ³ , IDLH: 1,333 mg/m ³
Nickel (refinery dust)	7440-02-0	0.007 ^f	0.0245 ^g	EPA Group A ^e	0.84 (inhal) ^j	OSHA-PEL: 1.0 mg/m ³ (metal and other compds.) ACGIH-TLV: 1 mg/m ³ NIOSH-REL: 0.015 mg/m ³
Nitric acid	7697-37-2	0.035 ^q	0.1225 ^g	not classified	none found	OSHA-PEL: 5 mg/m ³ ACGIH-TLV: 5.2 mg/m ³ , STEL: 10 mg/m ³ NIOSH-REL: 5 mg/m ³ , STEL: 10 mg/m ³ , IDLH: 65.5 mg/m ³

Table M.3.3–1. Table of Exposure Limits—Continued

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Nitrobenzene	98-95-3	5x10 ^{-4e}	1.75x10 ^{-3g}	EPA Group D ^e	none found	OSHA-PEL: 5 mg/m ³ (skin) ACGIH-TLV: 5 mg/m ³ (skin) NIOSH-REL: 5.0 mg/m ³ (skin), IDLH: 1,024 mg/m ³
2-Nitropropane	79-46-9	5.72x10 ^{-3d}	0.02 ^e	EPA Group B2 ^e	9.4 ^j	OSHA-PEL: 90 mg/m ³ ACGIH-TLV: 36 mg/m ³ IDLH: 370 mg/m ³
Octanol	111-87-5	0.72 ¹	2.52 ^g	not classified	none found	PEL: 102.8 LD50 (oral-rat) 18,000 mg/kg ⁱ
Oxalic acid	144-62-7	0.007 ^f	0.0245 ^g	not classified	none found	OSHA-PEL: 1 mg/m ³ ACGIH-TLV: 1 mg/m ³ , STEL: 2 mg/m ³ NIOSH-REL: 1 mg/m ³ , STEL: 2 mg/m ³ , IDLH: 500 mg/m ³
Ozone	10028-15-6	1.4x10 ^{-4f}	4.9x10 ^{-4g}	not classified	none found	OSHA-PEL: 0.2 mg/m ³ ACGIH-TLV: 0.2 mg/m ³ NIOSH-REL: 0.2 mg/m ³ IDLH: 10 mg/m ³
Phenol	108-95-2	0.6 ^k	2.1 ^g	EPA Group D ^e	none found	OSHA-PEL: 19 mg/m ³ (skin) NIOSH-REL: 19 mg/m ³ (skin, ceiling, 15 min.)
Phosphoric acid	7664-38-2	0.007 ^f	0.0245 ^g	not classifi ed	none found	OSHA-PEL: 1 mg/m ³ ACGIH-TLV: 1 mg/m ³ , STEL: 3 mg/m ³ NIOSH-REL: 1 mg/m ³ , STEL: 3 mg/m ³ , IDLH: 1,000 mg/m ³
Phosphorus (yellow)	7723-14-0	7x10 ^{-4f}	2.45x10 ^{-3g}	not classified	none found	OSHA-PEL: 0.1 mg/m ³ ACGIH-TLV: 0.1 mg/m ³ NIOSH-REL: 0.1 mg/m ³ , IDLH: 5 mg/m ³

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Phosgene	75-44-5	2.8x10 ^{-3f}	9.8x10 ^{-3g}	not classified	non e found	OSHA-PEL: 0.4 mg/m ³ ACGIH-TLV: 0.4 mg/m ³ NIOSH-REL: 0.4 mg/m ³ , 0.8 mg/m ³ (ceiling, 15 min.) IDLH: 8.22 mg/m ³
Plutonium oxide (plutonium cmpds)	none found	none found	none found	not classified	none found	none found
Potassium chromate (VI)	7789-00-6	0.007 ^f	0.0245 ^g	not classified	none found	OSHA-PEL: 1.0 mg/m ³ⁱ NIOSH-REL: 0.05 mg/m ³
Potassium hydroxide	1310-58-03	0.014 ^f	0.049 ^g	not classified	none found	OSHA-PEL: 2 mg/m ³ⁱ ACGIH-TLV: 2 mg/m ³ (ceiling) NIOSH-REL: 2 mg/m ³
Propane	74-98-6	12.6 ^f	44.1 ^g	not classified	none found	OSHA-PEL: 1,800 mg/m ³ NIOSH-REL: 1,800 mg/m ³ , IDLH: 3,843 mg/m ³
Propene	115-07-1	⁻ none found	none found	not classified	none found	none found
Propionaldehyde	123-38-6	0.056 ¹	0.197 ^g	not classified	none found	LD50 (oral, rat): 1,410 mg/kg ⁱ PEL: 8.06 mg/m ^{3m}
Propylene oxide	75-56-9	1.68 ^f	0.03 ^e	EPA Group B2 ^c	0.24 ^e	OSHA-PEL: 240 mg/m ³ ACGIH-TLV: 48 mg/m ³
Pyrene	129-00-0	0.03 ^e	0.105 ^g	EPA Group D ^e	none found	OSHA-PEL: 0.2 mg/m ³ⁱ
Pyridine	110-86-1	0.001 ^e	0.0035 ^g	not classified	non e found	OSHA-PEL: 15 mg/m ³ ACGIH-TLV: 16 mg/m ³ NIOSH-REL: 15 mg/m ³ , IDLH: 3,290 mg/m ³
Selenium	7782-49-2	5x10 ^{-3e}	0.0175 ^g	EPA Group D ^e	none found	OSHA-PEL: 0.2 mg/m ³ ACGIH-TLV: 0.2 mg/m ³ NIOSH-REL: 0.2 mg/m ³ , IDLH: 1 mg/m ³

Table M.3.3-1. Table of Exposure Limits—Continued

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Silicon	7440-21-3	0.035 ^f	0.1225 ^g	not classified	non e found	OSHA-PEL: 15 mg/m ³ (total), 5 mg/m ³ (resp) ACGIH-TLV: 10 mg/m ³ NIOSH-REL: 10 mg/m ³ (total), 5 mg/m ³ (resp)
Silver	7440-22-4	5x10 ^{-3e}	0.0175 ^g	EPA Group D ^e	none found	OSHA-PEL: 0.01 mg/m ³ ACGIH-TLV: 0.1 mg/m ³ NIOSH-REL: 0.01 mg/m ³ , IDLH: 10 mg/m ³
Stoddard solvent	8052-41-3	20.3 ^d	71.05 ^f	not classified	none found	OSHA-PEL: 2,900 mg/m ³ ACGIH-TLV: 525 mg/m ³ NIOSH-REL: 1,800 mg/m ³ (ceiling, 15-min) IDLH: 20,000 mg/m ³
Styrene	100-42-5	0.2 ^e	۱ [¢]	EPA Group C ^k	none found	OSHA-PEL: 433 mg/m ³ , 866 mg/m ³ (ceiling) ACGIH-TLV: 213 mg/m ³ (skin), STEL: 426 mg/m ³ (skin) NIOSH-REL: 215 mg/m ³ , STEL: 425 mg/m ³ , IDLH: 3,031 mg/m ³
Sulfuric acid	7664-93-9	0.007 ^f	0.0245 ^g	not classified	none found	OSHA-PEL: 1 mg/m ³ ACGIH-TLV: 1 mg/m ³ , STEL: 3 mg/m ³ NIOSH-REL: 1 mg/m ³ , IDLH: 15 mg/m ³
Tetrachloroethylene	127-18-4	0.01 ^e	0.035 ^g	EPA Group C-B2 ^j	0.002 ^j	OSHA-PEL: 689 mg/m ³ , 1,378 mg/m ³ (ceiling) ACGIH-TLV: 339 mg/m ³ , STEL: 1,357 mg/m ³ NIOSH-STEL: 1,034 mg/m ³

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Tetrahydrofuran (THF)	109-99-9	4.13 ^f	14.455 ^g	not classified	none found	OSHA-PEL: 590 mg/m ³ ACGIH-TLV: 590 mg/m ³ , STEL: 737 mg/m ³ NIOSH-REL: 590 mg/m ³ , STEL: 735 mg/m ³ , IDLH: 6,000 mg/m ³
Titanium	7440-32-6	none found	none found	not classified	none found	none found
Toluene	108-88-3	0.2 ^e	0.4 ^e	EPA Group D ^e	none found	OSHA-PEL: 766 mg/m ³ , 1,149 mg/m ³ (ceiling) ACGIH-TLV: 188 mg/m ³ (skin) NIOSH-REL: 375 mg/m ³ , STEL: 560 mg/m ³ , IDLH: 1,915 mg/m ³
Tributyl phosphate	126-73-8	0.035 ^d	0.1225 ^g	not classified	none found	OSHA-PEL: 5 mg/m ³ ACGIH-TLV: 2.2 mg/m ³ NIOSH-REL: 2.5 mg/m ³ IDLH: 332.1 mg/m ³
1,1,1-Trichloroethane (TCA; methyl chloroform)	71-55-6	0.035 ^k	1.0	EPA Group D ^e	none found	OSHA-PEL: 1,900 mg/m ³ ACGIH-TLV: 1,910 mg/m ³ , STEL: 2,460 mg/m ³ NIOSH-REL: 1,900 mg/m ³ (ceiling, 15 min.), IDLH: 3,885 mg/m ³
1,1,2-Trichloroethane	79-00-5	0.004 ^e	0.014 ^g	EPA Group C ^e	0.057 (oral) ^e 0.057 (inhal) ^j	OSHA-PEL: 45 mg/m ³ (skin) ACGIH-TLV: 55 mg/m ³ NIOSH-REL: 375 mg/m ³ (skin), IDLH: 555 mg/m ³
Trichloroethylene (TCE)	79-01-6	3.82 ^f	13.377 ^g	EPA Group B2 ^k	6.0x10 ⁻³ (inhal) ^j	OSHA-PEL: 546 mg/m ³ , 1,092 mg/m ³ (ceiling) ACGIH-TLV: 269 mg/m ³ , STEL: 1,070 mg/m ³ NIOSH-IDLH: 5,460 mg/m ³

Table M.3.3–1. Table of Exposure Limits—Continued

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Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Trichlorotrifluoroethane (Freon 113)	76-13-1	30.0 ^e	105.0 ^g	not classified	none found	OSHA-PEL: 7,600 mg/m ³ ACGIH-TLV: 7,670 mg/m ³ , STEL: 9,590 mg/m ³ NIOSH-REL: 7,600 mg/m ³ , STEL: 9,500 mg/m ³ , IDLH: 15,580 mg/m ³
1,2,4-Trimethylbenzene	95-63-6	0.875 ^h	3.06 ^g	not classified	none found	PEL: 125 mg/m ^{3m} NIOSH-REL: 125 mg/m ³
Tungsten (insoluble compds)	7440-33-7	0.035 ^h	0.1225 ^g	not classified	none found	ACGIH-TLV: 5 mg/m ³ STEL: 10 mg/m ³ NIOSH-REL: 5 mg/m ³ STEL: 10 mg/m ³
VM&P naphtha	8032-32-4	2.45 ^h	8.575 ^g	not classified	none found	PEL: 245 mg/m ^{3m} ACGIH-TLV: 1,370 mg/m ³ NIOSH-REL: 350 mg/m ³ , 1,800 mg/m ³ (ceiling, 15 min)
Welding fumes	ZC2550000	0.035 ^r	0.1225 ^g	not classified	none found	TLV ⁱ : 5 mg/m ³

Table M.3.3-1. Table of Exposure Limits—Continued

Compound	Chemical Abstracts Service No.	Reference Dose (oral) (mg/kg/day)	Reference Concentration (inhalation) (mg/m ³)	Cancer Class ^a	Slope Factor (mg/kg/day) ⁻¹	Occupational Exposure Levels ^{b,c}
Kylene (mixture)	1330-20-7	2.0 ^e	7.0 ^g	EPA Group D ^e	non e found	OSHA-PEL: 435 mg/m ³ ACGIH-TLV: 435 mg/m ³ , STEL: 651 mg/m ³ NIOSH-REL: 435 mg/m ³ , STEL: 655 mg/m ³ , IDLH: 3,969 mg/m ³
Zinc	7440-66-6	0.3 ^e	1.05 ^g	EPA Group D ^e	none found	PEL: 42.9 mg/m ^{3m}
EPA Group C: Possible Hu EPA Group D: Not Classifi OSHA and NIOSH exposur ACGIH exposure levels we RfD calculated from RfC, f ORNL 1994a. RfD calculated from OSHA RfC calculated from RfD, f RfD calculated from NIOS Lewis 1992a. EPA 1994b. EPA 1993a. RfD calculated from LD50 PEL calculated from RfD. EPA 1993c.	able as to Human Ca re levels were taken f re taken from ACGII formula from the Cer A-PEL, formula from formula from the Cer H-REL.	from NIOSH 1994a. H nda. hter for Risk Manage the Center for Risk hter for Risk Manage 0 ⁻⁵).	ement, ORNL (ORNL) Management, ORNL (ement, ORNL (ORNL	(ORNL 1992d).		·

Table M.3.3-1. Table of Exposure Limits—Continued

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M.3.4 HAZARDOUS CHEMICAL RISK/EFFECTS CALCULATIONS

Tables M.3.4–1 through M.3.4–79 show the human health risk increment from exposure to hazardous chemicals associated with the various alternative activities. The terms associated with calculations are given in the footnotes for each table so that each calculated value can be verified.

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	Regulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Chemical			ſ	1.27×10^{-8}	4.22x10 ⁻⁵	1.27x10 ⁻⁷	1.20 x 10 ⁻⁶	0	0
Ammonia	0.10	35	f	6.58 x10 ⁻⁵	2.18×10^{-1}	4.87x10 ⁻⁵	3.96x10 ⁻³	0	0
Carbon monoxide	1.35	55	1			_	2.26x10 ⁻⁵	0	0
VOC (toluene)	0.40	766	f	5.23 x10 ⁻⁶	1.73×10^{-2}	1.30 x10 ⁻⁵	2.20210	0	Ū
Health Risk						6.20x10 ⁻⁵	3.99x10 ⁻³		
НІ ^g						0.20110	5.77810	0	0
Cancer risk ^h				<u> </u>					

Table M.3.4–1. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—No Action

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: HF 1995a:2.

Table M.3.4–2. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Upgrade Plutonium Storage Facility (Both Options)

		Regulated E	Exposure Lir	nits/Risk Factors	Emissions Inventory		НQ		Cancer Risk	
	Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
	Carbon monoxide	1.35	55	f	2.92x10 ⁻⁷	9.69x10 ⁻⁴	2.17x10 ⁻⁷	1.76x10 ⁻⁵	0	0
	VOC (toluene) Health Risk	0.4	766	f	2.87x10 ⁻⁷	9.52x10 ⁻⁴	7.18x10 ⁻⁷	1.24×10^{-6}	0	0
ł	HI ^g Cancer risk ^h						9.35x10 ⁻⁷	1.89x10 ⁻⁵	0	0

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: HF DOE 1996a.

	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.93x10 ⁻⁶	9.72x10 ⁻³	2.17x10 ⁻⁶	1.76x10 ⁻⁴	0	0
Chlorine	0.35	3	f	2.82x10 ⁻⁸	9.35x10 ⁻⁵	8.06x10 ⁻⁸	3.11x10 ⁻⁵	0	0
Hydrazine	0.0326	1.33	17	5.64 x 10 ⁻⁹	1.87x10 ⁻⁵	1.73x10 ⁻⁷	1.43x10 ⁻⁵	2.74x10 ⁻⁸	1.23x10 ⁻⁵
Nitric acid	0.1225	5	f	3.38x10 ⁻⁸	1.12x10 ⁻⁴	2.76x10 ⁻⁷	2.24x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x 10 ⁻⁷	1.87x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x10 ⁻⁷	1.87x10 ⁻⁵	0	0
VOC (toluene)	0.4	766	f	3.27x10 ⁻⁷	1.08x10 ⁻³	8.18x10 ⁻⁷	1.41x10 ⁻⁶	0	0
Health Risk HI ^g						3.98x10 ⁻⁶	2.84x10 ⁻⁴		
Cancer risk ^h								2.74x10 ⁻⁸	1.23x10 ⁻⁵

Table M.3.4–3. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site— Consolidate Plutonium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

Table M.3.4–4. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site— Collocate Plutonium and Highly Enriched Uranium Storage Facility

	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary · MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.93x10 ⁻⁶	9.72x10 ⁻³	2.17x10 ⁻⁶	1.76x10 ⁻⁴	0	0
Chlorine	0.35	3	f	3.38x10 ⁻⁸	1.12×10^{-4}	9.67x10 ⁻⁸	3.74x10 ⁻⁵	0	0
Hydrazine	0.0326	1.3	17	5.64x10 ⁻⁹	1.87x10 ⁻⁵	1.73x10 ⁻⁷	1.43x10 ⁻⁵	2.74x10 ⁻⁸	1.23x10 ⁻⁵
Hydrogen chloride	0.007	7	f	5.07x10 ⁻⁸	1.68x10 ⁻⁴	7.25x10 ⁻⁶	2.40x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	5.07x10 ⁻⁸	1.68x10 ⁻⁴	2.41x10 ⁻⁷	6.75x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	5.36x10 ⁻⁷	1.77x10 ⁻³	4.37x10 ⁻⁶	3.55x10 ⁻⁴	0	0
Phosphoric acid	0.0245	1	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x10 ⁻⁷	1.87x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x10 ⁻⁷	1.87x10 ⁻⁵	0	0
VOC (toluene)	0.4	766	f	3.27x10 ⁻⁷	1.08x10 ⁻³	8.18x10 ⁻⁷	1.41x10 ⁻⁶	0	0
Health Risk HI ^g Cancer risk ^h						1.56x10 ⁻⁵	7.14x10 ⁻⁴	2.74x10 ⁻⁸	1.23x10 ⁻⁵

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

	Regulated Exposure Limits/Risk Factors		Emissions	Emissions Inventory		HQ		Cancer Risk	
	RfC	PEL ^a	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Chemical	(mg/m ³)	(mg/m ³)	(IIIg/Kg/Uay)			0	0	0	0
No chemical emissions [Text deleted.]	-								

Table M.3.4–5. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site—No Action

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

• HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

[Text deleted.]

Source: NTS 1993a:4.

	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	(Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.93x10 ⁻⁶	2.23x10 ⁻²	1.43x10 ⁻⁶	4.05x10 ⁻⁴	0	0
Chlorine	0.35	3	f	4.20x10 ⁻⁹	4.85x10 ⁻⁵	1.20x10 ⁻⁸	1.61x10 ⁻⁵	0	0
Hydrazine	0.0326	1.33	17	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	2.57x10 ⁻⁸	7.46x10 ⁻⁶	4.09x10 ⁻⁹	6.38x10 ⁻⁶
Nitric acid	0.1225	5	f	5.04x10 ⁻⁹	5.82x10 ⁻⁵	4.11x10 ⁻⁸	1.16x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	f	2.77x10 ⁻⁷	3.20x10 ⁻³	6.93x10 ⁻⁷	4.18x10 ⁻⁶	0	0
Health Risk									
НI ^g						2.27x10 ⁻⁶	4.65x10 ⁻⁴		
Cancer risk ^h								4.09x10 ⁻⁹	6.38x10 ⁻⁶

Table M.3.4-6. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site-Consolidate Plutonium Storage Facility

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

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	Regulated E	xposure Lir	nits/Risk Factors	Emissions	Inventory	H	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.10x10 ⁻⁶	2.42x10 ⁻²	1.55x10 ⁻⁶	4.41x10 ⁻⁴	0	0
Chlorine	0.35	3	f	6.72x10 ⁻⁹	7.76x10 ⁻⁵	1.92x10 ⁻⁸	2.58x10 ⁻⁵	0	0
Hydrazine	0.0326	1.3	17	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	2.57x10 ⁻⁸	7.46x10 ⁻⁶	4.09x10 ⁻⁹	6.38x10 ⁻⁶
Nitric acid	0.1225	5	f	4.20x 10 ⁻⁹	4.85x10 ⁻⁵	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Phosphoric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	8.40x 10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	f	3.10x10 ⁻⁷	3.59x10 ⁻³	7.77x10 ⁻⁷	4.68x10 ⁻⁶	0	0
Health Risk HI ^g						2.48x10 ⁻⁶	5.08x10 ⁻⁴		,
Cancer risk ^h								4.09x10 ⁻⁹	6.38x10 ⁻⁶

Table M.3.4–7. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site— Consolidate Plutonium Storage Facility at P-Tunnel

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: NT DOE 1996a.

	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.10x10 ⁻⁶	2.42x10 ⁻²	1.55x10 ⁻⁶	4.41x10 ⁻⁴	0	0
Chlorine	0.35	3	f	5.04x10 ⁻⁹	5.82x10 ⁻⁵	1.44x10 ⁻⁸	1.94x10 ⁻⁵	0	0 .
Hydrazine	0.0326	1.3	17	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	2.57x10 ⁻⁸	7.46x10 ⁻⁶	4.09x10 ⁻⁹	6.38x10 ⁻⁶
Hydrogen chloride	0.007	7	f	7.56x10 ⁻⁹	8.73x10 ⁻⁵	1.08x10 ⁻⁶	1.24x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	7.56x10 ⁻⁹	8.73x10 ⁻⁵	3.60x10 ⁻⁸	3.50x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	7.98x10 ⁻⁸	9.21x10 ⁻⁴	6.51x10 ⁻⁷	1.84x10 ⁻⁴	0	0
Phosphoric acid	0.0245	1	ſ	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	f	3.10x10 ⁻⁷	3.59x10 ⁻³	7.77x10 ⁻⁷	4.68x10 ⁻⁶	0	0
Health Risk HI ^g						4.21x10 ⁻⁶	7.24x10 ⁻⁴	-	-
Cancer risk ^h								4.09x10 ⁻⁹	6.38x10 ⁻⁶

Table M.3.4-8. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site-Collocate Plutonium and Highly Enriched Uranium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

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	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.35x10 ⁻⁶	2.71x10 ⁻²	1.74x10 ⁻⁶	4.93x10 ⁻⁴	0	0
Chlorine	0.35	3	f	6.72x10 ⁻⁹	7.76x10 ⁻⁵	1.92x10 ⁻⁸	2.58x10 ⁻⁵	0	0
Hydrazine	0.0326	1.3	17	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	2.57x10 ⁻⁸	7.46x10 ⁻⁶	4.09x10 ⁻⁹	6.38x10 ⁻⁶
Nitric acid	0.1225	5	f	4.20x10 ⁻⁹	4.85x10 ⁻⁵	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Phosphoric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x 10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	f	3.53x10 ⁻⁷	4.07x10 ⁻³	8.82x10 ⁻⁷	5.32x10 ⁻⁶	0	0
Health Risk HI ^g						2.77x10 ⁻⁶	5.62x10 ⁻⁴	Page 10-9	(ap. 10-6
Cancer risk ^h								4.09x10 ⁻⁹	6.38x10 ⁻⁶

Table M.3.4–9. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site— Collocate Plutonium and Highly Enriched Uranium Storage Facility at P-Tunnel

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: NT DOE 1996a.

	Regulated I	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ.	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
1,2,4-Trimethylbenzene	3.06	125	f	1.05x10 ⁻⁶	1.64x10 ⁻³	3.43x10 ⁻⁷	1.31x10 ⁻⁵	0	0
1,3-Butadiene	53.9	2200	1.8	2.66x10 ⁻⁶	4.15x10 ⁻³	4.93x10 ⁻⁸	1.88x10 ⁻⁶	1.37x10 ⁻⁶	2.89x10 ⁻⁴
Acetaldehyde	0.009	360	f	3.74x10 ⁻⁷	5.85x10 ⁻⁴	4.16x10 ⁻⁵	1.62x10 ⁻⁶	0	0
Ammonium hydroxide	0.0499	2	f	1.93x10 ⁻⁵	3.02x10 ⁻²	3.87x10 ⁻⁴	1.51x10 ⁻²	0	0
Arsenic	0.00105	0.01	50	5.08x10 ⁻⁸	7.93x10 ⁻⁵	4.83x10 ⁻⁵	7.93x10 ⁻³	7.26x10 ⁻⁷	1.53x10 ⁻⁴
Benzene	0.0796	3.25	0.029	4.47x10 ⁻⁶	6.98x10 ⁻³	5.62x10 ⁻⁵	2.14×10^{-3}	3.71x10 ⁻⁸	7.84x10 ⁻⁶
Carbon monoxide	1.35	55	f	3.64x10 ⁻³	5.68	2.69x10 ⁻³	1.03x10 ⁻¹	0	0
Carbon tetrachloride	0.00245	63.9	0.053	3.38x10 ⁻⁷	5.28x10 ⁻⁴	1.38x10 ⁻⁴	8.27x10 ⁻⁶	5.13x10 ⁻⁹	1.08x10 ⁻⁶
Chloroform	0.035	240	0.081	2.29x10 ⁻⁸	3.58x10 ⁻⁵	6.56x10 ⁻⁷	1.49x10 ⁻⁷	5.32x10 ⁻¹⁰	1.12x10 ⁻⁷
Chromium-hexavalent	0.0175	1	41	4.83x10 ⁻⁹	7.55x10 ⁻⁶	2.76x10 ⁻⁷	7.5537x10 ⁻⁶	5.67x10 ⁻⁸	1.20x10 ⁻⁵
Chromium-trivalent	3.5	0.5	f	3.74x10 ⁻⁸	5.85x10 ⁻⁵	1.07x10 ⁻⁸	1.17x10 ⁻⁴	0	0
Cyclopentane	42.18	1,720	f	4.23x10 ⁻⁶	6.60x10 ⁻³	1.00x10 ⁻⁷	3.84x10 ⁻⁶	Q	0
Dichloromethane	3.0	1,765	0.0075	7.49x10 ⁻⁶	1.17x10 ⁻²	2.49x10 ⁻⁶	6.63x10 ⁻⁶	1.61x10 ⁻⁸	3.40x10 ⁻⁶
Formaldehyde	0.7	0.9375	0.045	1.16x10 ⁻⁵	1.81x10 ⁻²	1.65x10 ⁻⁵	1.93x10 ⁻²	1.49x10 ⁻⁷	3.16x10 ⁻⁵
Hydrazine	0.0326	1.33	17	1.00x10 ⁻⁷	1.56x10 ⁻⁴	3.07x10 ⁻⁶	1.17x10 ⁻⁴	4.88x10 ⁻⁷	1.03x10 ⁻⁴
Hydrochloric acid	0.007	7	f	1.81x10 ⁻⁵	2.83x10 ⁻²	2.59x10 ⁻³	4.04x10 ⁻³	0	0
Lead	0.001225	0.05	f	5.08x10 ⁻⁸	7.93x10 ⁻⁵	4.14x10 ⁻⁵	1.58x10 ⁻³	0	0
Mercury	0.0003	0.1	f	2.41x10 ⁻⁶	3.77x10 ⁻³	8.06x10 ⁻³	3.77x10 ⁻²	0	0
Naphthalene	1.225	50	f	1.93x10 ⁻⁷	3.02x10 ⁻⁴	1.57x10 ⁻⁷	6.04x10 ⁻⁶	0	0
Nickel	0.0245	1	0.84	3.27x10 ⁻⁶	5.10x10 ⁻³	1.33x10 ⁻⁴	5.10x10 ⁻³	7.86x10 ⁻⁷	1.66x10 ⁻⁴

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Table M.3.4–10. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—No Action

	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Nitric acid	0.1225	5	f	1.81x10 ⁻⁵	2.83x10 ⁻²	1.48x10 ⁻⁴	5.66x10 ⁻³	0	0
Phosphorus	0.00245	0.1	f	6.77x10 ⁻⁷	1.05x10 ⁻³	2.76x10 ⁻⁴	1.05x10 ⁻²	0	0
Potassium hydroxide	0.049	2	f	1.19x10 ⁻⁵	1.86x10 ⁻²	2.44x10 ⁻⁴	9.34x10 ⁻³	0	0
Propionaldehyde	0.197	8.06	f	7.49x10 ⁻⁷	1.17x10 ⁻³	3.80x10 ⁻⁶	1.45x10 ⁻⁴	0	0
Styrene	1	433	f	5.68x10 ⁻⁸	8.87x10 ⁻⁵	5.68x10 ⁻⁸	2.04x10 ⁻⁷	0	0
Tetrachloroethylene	0.035	689	0.002	1.18x10 ⁻⁵	1.85x10 ⁻²	3.38x10 ⁻⁴	2.68x10 ⁻⁵	6.78x10 ⁻⁹	1.43x10 ⁻⁶
Toluene	0.4	766	f	7.01x10 ⁻⁶	1.09x10 ⁻²	1.75x10 ⁻⁵	1.42x10 ⁻⁵	0	0
Trichloroethylene	13.377	546	0.006	5.66x10 ⁻⁸	8.83x10 ⁻⁵	4.23x10 ⁻⁹	1.61x10 ⁻⁷	9.71x10 ⁻¹¹	2.05x10 ⁻⁸
Health Risk						1.53x10 ⁻²	2.23x10 ⁻¹		
Cancer risk ^h								3.64x10 ⁻⁶	7.70x10 ⁻⁴

Table M.3.4–10. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—No Action—Continued

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: INEL 1995a:1.

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	Regulated E	xposure Lin	nits/Risk Factors	Emission	s Inventory	H	IQ.	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.08x10 ⁻⁵	1.68x10 ⁻²	7.97x10 ⁻⁶	3.06x10 ⁻⁴	0	
Chlorine	0.35	3	f	1.21x10 ⁻⁸	1.89x10 ⁻⁵	3.46x10 ⁻⁸	6.29x10 ⁻⁶	0	0
Hydrazine	0.0326	1.33	17	1.21x10 ⁻⁸	1.89x10 ⁻⁵	3.71×10^{-7}	1.45x10 ⁻⁵	5.88x10 ⁻⁸	0
Phosphoric acid	0.0245	1	f	1.21x10 ⁻⁸	1.89x10 ⁻⁵	4.94x10 ⁻⁷	1.45x10 ⁻⁵		1.24x10 ⁻⁵
Sulfuric acid	0.0245	1	f	1.21x10 ⁻⁸	1.89x10 ⁻⁵	4.94×10^{-7}	1.89x10 ⁻⁵	0	0
VOCs (toluene) Health Risk	0.4	766	f	1.00x10 ⁻⁶	1.57x10 ⁻³	4.94x10 ⁻⁶	2.05x10 ⁻⁶	0 0	0 0
HI ^g Cancer risk ^h						1.19x10 ⁻⁵	3.66x10 ⁻⁴	5.88x10 ⁻⁸	1.24x 10 ⁻⁵

Table M.3.4–11. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—Upgrade Plutonium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

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⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: IN DOE 1996a.

	Dominted F	mosure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Churches	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Chemical	<u> </u>	55	f	4.11x10 ⁻⁵	6.42x10 ⁻²	3.04x10 ⁻⁵	1.16x10 ⁻³	0	0
Carbon monoxide			f	3.62x10 ⁻⁸	5.66x10 ⁻⁵	1.03x10 ⁻⁷	1.88x10 ⁻⁵	0	0
Chlorine	0.35	3			1.88x10 ⁻⁵	3.71x10 ⁻⁷	1.45x10 ⁻⁵	5.88x10 ⁻⁸	1.24x10 ⁻⁵
Hydrazine	0.0326	1.33	17	1.21x10 ⁻⁸		_		0	0
Nitric acid	0.1225	5	t	7.25x10 ⁻⁸	1.13x10 ⁻⁴	5.92×10^{-7}	2.26x10 ⁻⁵	-	
Phosphoric acid	0.0245	1	f	1.21x10 ⁻⁸	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
=	0.0245	- 1	f	1.21x10 ⁻⁸	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
Sulfuric acid		1	f	4.83x10 ⁻⁶	7.55x10 ⁻³	1.21x10 ⁻⁵	9.86x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	•	4.85810	7.55X10	1.21X10	2100/10		
Health Risk						4.46x10 ⁻⁵	1.27x10 ⁻³		

Table M.3.4–12. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—Consolidate Plutonium Storage Facility

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HO for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for ME =(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions Concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

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Cancer risk^h

1.24x10⁻⁵

5.88x10⁻⁸

	Regulated E	xposure Li	nits/Risk Factors	Emission	s Inventory	H	IQ	Салс	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	4.83x10 ⁻⁵	7.55x10 ⁻²	3.58x10 ⁻⁵	1.27 10-3		
Chlorine	0.35	3	f	4.83x10 ⁻⁸	7.55x10 ⁻⁵		1.37x10 ⁻³	0	0
Hydrazine	0.0326	1.3	17	1.21x10 ⁻⁸		1.38x10 ⁻⁷	2.51x10 ⁻⁵	0	0
Hydrogen chloride	0.007	7	f		1.88x10 ⁻⁵	3.71x10 ⁻⁷	1.45x10 ⁻⁵	5.88x10 ⁻⁸	1.24x10 ⁻⁵
Hydrogen fluoride	0.21	, 2.49	f	1.08x10 ⁻⁷ 1.08x10 ⁻⁷	1.70x10 ⁻⁴	1.55x10 ⁻⁵	2.42x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	1.08×10^{-6}	1.70×10^{-4}	5.18x10 ⁻⁷	6.82x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	1.14×10^{-8}	1.79x10 ⁻³	9.38x10 ⁻⁶	3.58x10 ⁻⁴	0	0
Sulfuric acid	0.0245	1	f	_	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
VOC (toluene)	0.4	766	f	1.21x10 ⁻⁸	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
Health Risk	0.4	/00		5.68x10 ⁻⁶	8.87x10 ⁻³	1.42×10^{-5}	1.15x10 ⁻⁵	0	0
HIg									
Cancer risk ^h						7.70x10 ⁻⁵	1.91x10 ⁻³		
See Table M 2.2.1.6								5.88x10 ⁻⁸	1.24x10 ⁻⁵

Table M.3.4–13. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory-Collocate Plutonium and Highly Enriched Uranium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

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^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

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	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	(Q	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meter 8 Hours ^e
1,1,1-Trichloroethane	1.0	1,900	f	7.17x10 ⁻⁷	2.15x10 ⁻⁵	7.17x10 ⁻⁶	1.13x10 ⁻⁸	0	0
(TCA)			c	7	5	7	7	0	0
Carbon disulfide	0.35	63.2	f	8.53×10^{-7}	2.56x10 ⁻⁵	2.44×10^{-7}	4.05x10 ⁻⁷	0	0
Carbon tetrachloride	0.00245	63.9	0.53	4.91x10 ⁻⁷	1.48×10^{-4}	2.01×10^{-4}	2.31x10 ⁻⁶	7.45x10 ⁻⁹	3.03x10 ⁻⁷
Chlorobenzene	0.07	350	f	5.63x10 ⁻⁸	1.69x10 ⁻⁵	8.05x10 ⁻⁷	4.83x10 ⁻⁹	0	0
Cresol	0.539	22	f	1.57x10 ⁻⁹	4.72x10 ⁻⁷	2.92x10 ⁻⁹	2.15x10 ⁻⁸	0	0
Cresylic acid	0.539	22	f	1.57x10 ⁻⁹	4.72×10^{-7}	2.92x10 ⁻⁹	2.15x10 ⁻⁸	0	0
Dibenzofuran			f	2.29x10 ⁻⁹	6.87x10 ⁻⁷		_	0	0
Ester glycol ethers (2-Ethoxyethanol)	0.2	740	f	2.70x10 ⁻⁸	8.12x10 ⁻⁶	1.35x10 ⁻⁷	1.10x10 ⁻⁸	0	0
Ethene, Trichloro	13.377	546	0.006	4.98x10 ⁻⁸	1.49x10 ⁻⁵	3.72x10 ⁻⁹	2.74x10 ⁻⁸	8.54x10 ⁻¹¹	3.47x10 ⁻⁹
Ethyl benzene	1	435	f	4.78x10 ⁻⁸	1.43x10 ⁻⁵	4.76x10 ⁻⁸	3.29x10 ⁻⁸	0	0
Ethylene dichloride	5.03	411	0.091	4.19x10 ⁻⁸	1.26x10 ⁻⁵	8.33x10 ⁻⁹	6.12x10 ⁻⁸	1.09x10 ⁻⁹	4.43x10 ⁻⁸
Hydrogen chloride	0.007	7	f	3.49x10 ⁻⁵	1.05x10 ⁻²	4.98x10 ⁻³	1.50x10 ⁻³	0	0
Hydrogen fluoride	0.21	2.49	f	3.71x10 ⁻⁵	1.11x10 ⁻²	1.77x10 ⁻⁴	4.47x10 ⁻³	0	0
Ketones (acetone)	0.35	2,400	f	8.72x10 ⁻⁹	2.62x10 ⁻⁶	2.49x10 ⁻⁸	1.09x10 ⁻⁹	0	0
Methyl alcohol	1.75	260	f	3.45x10 ⁻⁵	1.04x10 ⁻²	1.97x10 ⁻⁵	3.98x10 ⁻⁵	0	0
Methyl ethyl ketone (MEK)	1	590	f	2.23x10 ⁻⁴	6.69x10 ⁻²	2.23x10 ⁻⁴	1.13x10 ⁻⁴	0	0
Methyl isobutyl ketone (MIBK)	0.28	410	f	1.94x10 ⁻⁸	5.84x10 ⁻⁶	6.94x10 ⁻⁸	1.42x10 ⁻⁸	0	0
Naphtalene	0.014	50	f	1.29x10 ⁻⁸	3.87x10 ⁻⁶	9.19x10 ⁻⁷	7.73x10 ⁻⁸	0	0
Nickel	0.0245	1	0.84	5.15x10 ⁻⁹	1.55x10 ⁻⁶	2.10x10 ⁻⁷	1.55x10 ⁻⁶	1.24x10 ⁻⁹	5.03x10 ⁻⁸
Nitrobenzene	0.00175	5	f	1.57x10 ⁻⁹	4.72x10 ⁻⁷	8.99x10 ⁻⁷	9.45x10 ⁻⁸	0	0
Phenol	2.1	19	f	7.03x10 ⁻⁸	2.11x10 ⁻⁵	3.35x10 ⁻⁸	1.11x10 ⁻⁶	0	0
Tetrachloroethylene	0.035	689	0.002	2.03x10 ⁻⁷	6.09x10 ⁻⁵	5.80x10 ⁻⁶	8.85x10 ⁻⁸	1.16x10 ⁻¹⁰	4.72x10 ⁻⁹
Toluene	0.055	766	f	1.47x10 ⁻⁵	4.41x10 ⁻³	3.67x10 ⁻⁵	5.76x10 ⁻⁶	0	0
Trichloroethene	13.377	700 546	0.006	6.15x10 ⁻⁷	1.85x10 ⁻⁴	4.60x 10 ⁻⁸	3.38x10 ⁻⁷	1.06x10 ⁻⁹	4.29x10 ⁻⁸
Xylene	7	435	0.000 f	7.00x10 ⁻⁶	2.10x10 ⁻³	1.00x10 ⁻⁶	4.84x10 ⁻⁶	0	0

Table M.3.4–14. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—No Action

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Table M.3.4–14. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—No Action—Continued

	Regulated E	Regulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	
Health Risk		-,								
НI ^g						5.65x10 ⁻³	6.14×10^{-3}			
Cancer risk ^h						0.00/10	0.1	1.10x10 ⁻⁸	4.48x10 ⁻⁷	

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions Concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

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⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: Chemicals deleted from the emissions list per memorandum are 1,1,2-Trichloroethane, 2-Nitropropane, Benzene, Chromium (VI), Formaldehyde, and Methylene chloride. Source: PX 1995a:4; PX DOE 1996b.

	Romlated F	Regulated Exposure Limits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk		
Chemical ^b	RfC (mg/m ³)	PEL ^c (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	Boundary MEI ^f	Worker 100 Meters 8 Hours ^g
Health Risk HI ^h						i	i	÷	i
Cancer risk ^j						,			•

Table M.3.4–15. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Upgrade Plutonium Storage Facility^a

^a Chemical impacts are the same for all three upgrade subalternatives.

^b No hazardous chemical emissions are associated with the Upgrade Alternative at Pantex.

^c See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^d HQ for MEI=boundary annual emissions/RfC.

^c HO for workers=100-m, 8-hr emissions/PEL.

^f Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

⁸ Cancer risk for workers=(emissions Concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^h HI=sum of individual HQs.

ⁱ Health risks at Pantex are the same for both the Upgrade With RFETS Pits Subalternative and the Upgrade Without RFETS Pu and LANL Subalternative. The health risks for the Upgrade With RFETS Pu and LANL Pu Subaltemative are bounded by the results presented in Table M.3.4-16 for the Consolidated Pu Storage Facility Alternative.

^j Total cancer risk=sum of individual cancer risks.

Source: PX MH 1994a.

	Regulated E	xposure Lir	nits/Risk Factors	Emissions	5 Inventory	H	IQ	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.16x10 ⁻⁴	3.49x10 ⁻²	8.62x10 ⁻⁵	6.35x10 ⁻⁴	0	
Chlorine	0.35	3	f	1.57x10 ⁻⁷	4.72×10^{-5}	4.49x10 ⁻⁷	1.57×10^{-5}	v	0
Hydrazine	0.0326	1.33	17	3.14x10 ⁻⁸	9.44×10^{-6}	9.64x10 ⁻⁷		0	0
Nitric acid	0.1225	5	f	1.88x10 ⁻⁷	5.66x10 ⁻⁵	1.54×10^{-6}	7.26×10^{-6}	1.53×10^{-7}	6.22x10 ⁻⁶
Phosphoric acid	0.0245	1	f	3.14×10^{-8}	9.44×10^{-6}		1.13x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	3.14×10^{-8}	· · · ·	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	f	-	9.44x10 ⁻⁶	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
Health Risk	0.4	700		1.73×10^{-5}	5.19x10 ⁻³	4.32x10 ⁻⁵	6.78x10 ⁻⁶	0	0
HI ^g Cancer risk ^h						1.35x10 ⁻⁴	6.96x10 ⁻⁴		
1 0								1.53x10 ⁻⁷	6.22x10 ⁻⁶

Table M.3.4–16. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Consolidate Plutonium Storage Facility

^a See Table M.3.3–1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HOs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

	Regulated F	xposure Lin	nits/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.19x10 ⁻⁴	3.59x10 ⁻²	8.85x10 ⁻⁵	6.52x10 ⁻⁴	0	0
Chlorine	0.35	3	f	1.57x10 ⁻⁷	4.72x10 ⁻⁵	4.49x10 ⁻⁷	1.57x10 ⁻⁵	0	0
Hydrazine	0.0326	1.33	17	3.14x10 ⁻⁸	9.44x10 ⁻⁶	9.64x10 ⁻⁷	7.26x10 ⁻⁶	1.53x10 ⁻⁷	6.22x10 ⁻⁶
Hydrogen chloride	0.007	7	f	2.83x10 ⁻⁷	8.50x10 ⁻⁵	4.04x10 ⁻⁵	1.21x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	2.83x10 ⁻⁷	8.50x10 ⁻⁵	1.34x10 ⁻⁶	3.41x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	2.98x10 ⁻⁶	8.97x10 ⁻⁴	2.43x10 ⁻⁵	1.79x10 ⁻⁴	0	0
Phosphoric acid	0.0245	1	f	3.14x10 ⁻⁸	9.44x10 ⁻⁶	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	3.14x10 ⁻⁸	9.44x10 ⁻⁶	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
VOC (toluene)	0.4	- 766	f	1.79x10 ⁻⁵	5.38x10 ⁻³	4.48x10 ⁻⁵	7.03x10 ⁻⁶	0	0
Health Risk HI ^g						2.04x10 ⁻⁴	9.28x10 ⁻⁴		
Cancer risk ^h								1.53x10 ⁻⁷	6.22x10 ⁻⁶

Table M.3.4–17. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Collocate Plutonium and Highly Enriched Uranium Storage Facility

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^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions Concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

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	Regulated I	Exposure Lir	nits/Risk Factors	Emissions	Inventory	H	IQ.	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
1,1,1-Trichloroethane (TCA)	1.0	1,900	f	7.26x10 ⁻⁶	4.36x10 ⁻³	7.26x10 ⁻⁶	2.29x10 ⁻⁶	0	0
Acetic acid	0.6125	25	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	5.39x10 ⁻⁸	7.93x10 ⁻⁷	0	0
Carbon monoxide	1.35	55	f	3.14x10 ⁻³	1.88x10 ⁰	2.32×10^{-3}	3.42×10^{-2}	ů 0	0
Chlorine	0.35	3	f	5.78x10 ⁻⁵	3.47x10 ⁻²	1.65x10 ⁻⁴	1.16x10 ⁻²	ů 0	ů 0
Hydrogen chloride	0.007	7.0	f	2.12x10 ⁻⁴	1.27x10 ⁻¹	3.03x10 ⁻²	1.82x10 ⁻²	0	0
Hydrogen fluoride	0.21	2.49	f	2.31x10 ⁻⁶	1.39x10 ⁻³	1.10x10 ⁻⁵	5.57x10 ⁻⁴	0	ů 0
Methyl alcohol	1.75	260	f	8.72x10 ⁻⁴	5.23x10 ⁻¹	4.98x10 ⁻⁴	2.01×10^{-3}	Õ	0
Nitric acid	0.1225	5	f	3.14x10 ⁻⁴	1.88x10 ⁻¹	2.56x10 ⁻³	3.76x10 ⁻²	ů 0	0
Sulfuric acid	0.0245	1	f	8.25x10 ⁻⁵	4.95x10 ⁻²	3.37x10 ⁻³	4.95x10 ⁻²	0	0
VOC (toluene)	0.4	766	f	1.22x10 ⁻⁴	7.33x10 ⁻²	3.05x10 ⁻⁴	9.57x10 ⁻⁵	0	0 0
Health Risk HI ^g Cancer risk ^h						3.95x10 ⁻²	1.54x10 ⁻¹	0	0

Table M.3.4–18. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—No Action

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-h Concentrations)x(0.237[fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: OR LMES 1996i.

······································	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Hydrogen chloride	0.007	7.0	f	3.75x10 ⁻⁷	2.25x10 ⁻⁴	5.36x10 ⁻⁵	3.22x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	3.75x10 ⁻⁷	2.25×10^{-4}	1.79x10 ⁻⁶	9.04x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	3.75x10 ⁻⁶	2.25x10 ⁻³	3.06x10 ⁻⁵	4.50x10 ⁻⁴	0	0
Health Risk HI ^g						8.60x10 ⁻⁵	5.73x10 ⁻⁴		
Cancer risk ^h								0	0

Table M.3.4–19. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Upgrade Highly Enriched Uranium Storage Facility

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^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: OR MMES 1996a.

Table M.3.4–20. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Collocate Plutonium Storage Facilities; Maintain Highly Enriched Uranium Facility

	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	6.27x10 ⁻⁵	3.76x10 ⁻²	4.64x10 ⁻⁵	6.84x10 ⁻⁴	0	0
Chlorine	0.35	3	f	1.98x10 ⁻⁷	1.18x10 ⁻⁴	5.66x10 ⁻⁷	3.96x10 ⁻⁵	0	0
Hydrazine	0.0326	1.33	17	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.01x10 ⁻⁶	1.52x10 ⁻⁵	1.61x10 ⁻⁷	1.30x10 ⁻⁵
Nitric acid	0.1225	5	f	1.98x10 ⁻⁷	1.18x10 ⁻⁴	1.61x10 ⁻⁶	2.37x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.34x10 ⁻⁶	1.98x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.34x10 ⁻⁶	1.98x10 ⁻⁵	0	0
VOC (toluene)	0.4	766	f	7.26x10 ⁻⁶	4.35x10 ⁻³	1.81x10 ⁻⁵	5.69x10 ⁻⁶	0	0
Health Risk									
HIg						7.05x10 ⁻⁵	8.08x10 ⁻⁴		
Cancer risk ^h								1.61x10 ⁻⁷	1.30x10 ⁻⁵

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

	Domilated F	vnosure I in	nits/Risk Factors	Emissions	Inventory	Н	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
	1.35	55	f	6.93x10 ⁻⁵	4.16x10 ⁻²	5.13x10 ⁻⁵	7.56x10 ⁻⁴	0	0
Carbon monoxide	0.35	3	f	2.64x10 ⁻⁷	1.58x10 ⁻⁴	7.54x10 ⁻⁷	5.28x10 ⁻⁵	0	0
Chlorine	0.33	1.33	17	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.01x10 ⁻⁶	1.52x10 ⁻⁵	1.61x10 ⁻⁷	1.30x10 ⁻⁵
Hydrazine		1.55 7	f	2.97x10 ⁻⁷	1.78x10 ⁻⁴	4.24x10 ⁻⁵	2.54x10 ⁻⁵	0	0
Hydrogen chloride	0.007	•	f	2.97x10 ⁻⁷	1.78×10^{-4}	1.41x10 ⁻⁶	7.16x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	3.13x10 ⁻⁶	1.88x10 ⁻³	2.56x10 ⁻⁵	3.76x10 ⁻⁴	0	0
Nitric acid	0.1225	5	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.34x10 ⁻⁶	1.98x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	3.30×10^{-8}	1.98x10 ⁻⁵	1.34×10^{-6}	1.98x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	ſ		4.95×10^{-3}	2.06×10^{-5}	6.46x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	1	8.25x10 ⁻⁶	4.95X10°	2.00210	0.40/10	Ū	· ·
Health Risk HI ^g						1.46x10 ⁻⁴	1.34x10 ⁻³	_	
Cancer risk ^h								1.61x10 ⁻⁷	1.30x10 ⁻

Table M.3.4–21. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Collocate Plutonium and Highly Enriched Uranium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

	Regulate	d Exposure	e Limits/Risk						
-		Factors	3	Emissions	Inventory	H	IQ	Canc	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Benzene	0.0796	3.25	0.029	1.25x10 ⁻⁵	1.37x10 ⁻²	1.57x10 ⁻⁵	4.20x10 ⁻³	1.04x10 ⁻⁸	1.53x10 ⁻⁵
Benzene (DWPF) ^f	0.0796	3.25	0.029	1.23x10 ⁻⁶	1.35x10 ⁻¹	1.55x10 ⁻⁴	4.15x10 ⁻²	1.02x10 ⁻⁷	1.51x10 ⁻⁴
Carbon monoxide	1.35	55	g	5.41x10 ⁻³	59.1	4.01x10 ⁻³	1.07	0	0
Chlorine	0.35	3	g	9.27x10 ⁻⁹	1.01x10 ⁻⁴	2.65x10 ⁻⁸	3.37x10 ⁻⁵	0	0
Chloroform	0.035	240	0.0061	4.79x10 ⁻⁶	5.24x10 ⁻²	1.37x10 ⁻⁴	2.18x10 ⁻⁴	8.36x10 ⁻⁹	1.24x10 ⁻⁵
Cobalt	0.00245	0.1	g	7.46x10 ⁻⁹	8.15x10 ⁻⁵	3.05x10 ⁻⁶	8.15x10 ⁻⁴	0	0
Hydrogen flouride	0.21	2.49	g	4.29x10 ⁻⁸	4.69x10 ⁻⁴	2.04x10 ⁻⁷	1.88x10 ⁻⁴	0	0
Hydrogen fluoride (DWPF) ^f	0.21	2.49	g	8.39x10 ⁻¹²	9.16x10 ⁻⁸	3.99x10 ⁻¹¹	3.68x10 ⁻⁸	0	0
Mercury (vapor)	0.0003	0.1	g	1.89x10 ⁻⁷	2.06x10 ⁻³	6.29x10 ⁻⁴	2.06x10 ⁻²	0	0
Mercury (DWPF) ^f	0.0003	0.1	g	5.17x10 ⁻⁸	5.65x10 ⁻⁴	1.72x10 ⁻⁴	5.65x10 ⁻³	0	0
Mercury oxide (DWPF) ^f	0.0003	0.1	g	6.36x10 ⁻¹⁸	6.95x10 ⁻¹⁴	2.12x10 ⁻¹⁴	6.95x10 ⁻¹³	0	0
Nickel (vapor and compounds)	0.0245	1	0.84	4.31x10 ⁻⁸	4.70x10 ⁻⁴	1.76x10 ⁻⁶	4.70x10 ⁻⁴	1.03x10 ⁻³	1.53x10 ⁻⁵
Nickel compounds (DWPF) ^f	0.0245	1	0.84	3.16x10 ⁻¹⁶	3.45x10 ⁻¹²	1.29x10 ⁻¹⁴	3.45x10 ⁻¹²	7.60x10 ⁻¹⁷	1.12x10 ⁻¹³
Nitric acid	0.1225	5	g	3.73x10 ⁻⁶	4.07×10^{-2}	3.04x10 ⁻⁵	8.15x10 ⁻³	0	0
Phosphoric acid	0.0245	1	g	1.50x10 ⁻⁷	1.63x10 ⁻³	6.11x10 ⁻⁶	1.63x10 ⁻³	0	0
<mark>Health Ri</mark> sk HI ^h						5.16x10 ⁻³	1.16		
Cancer risk ⁱ						2.10/10	1.10	1.31x10 ⁻⁷	1.94x10 ⁻⁴

Table M.3.4-22. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site-

No Action

^a See Table M.3.3-1 for the OSHA-PEL, ACGIH-TLV, NIOSH-REL, and other exposure limit values.

^b HQ for MEI=Boundary Annual Emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f The Defense Waste Process Facility (DWPF), In-Tank Precipitation (ITP) facility, and Consolidation Incineration Facility (CIF) were not in operation during 1994, but potential emissions from DWPF based on limited trials were used to generate DWPF potential emissions. The ITP and CIF data were not included because only the inventory of chemicals to be processed through these facilities was available.

^g There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^h HI=sum of individual HQs.

ⁱ Total cancer risk=sum of individual cancer risks.

Source: SRS 1995a:2; SRS 1996a:1.

	Regulated E	Exposure Lir	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
With All or Some RFETS and LANL Material					······································		944		
Carbon dioxide	211	9,000	f	8.39x10 ⁻⁶	9.16x10 ⁻²	3.98x10 ⁻⁸	1.02x10 ⁻⁵	0	0
Carbon monoxide	1.35	55	f	1.06x10 ⁻⁶	1.15x10 ⁻²	7.82x10 ⁻⁷	2.10x10 ⁻⁴	0	0
VOC (toluene)	0.4	766	f	2.92x10 ⁻⁷	3.19x10 ⁻³	7.30x10 ⁻⁷	4.17x10 ⁻⁶	0	0
Health Risk									
HIg						1.6x10 ⁻⁶	2.24x10 ⁻⁴		
Cancer risk ^h								0	0
With RFETS Non-Pit Pu Material	-								
Carbon monoxide	1.35	55	f	1.06x10 ⁻⁶	1.15x10 ⁻²	7.82x10 ⁻⁷	2.10x10 ⁻⁴	0	0
VOC (toluene)	0.4	766	f	2.80x10 ⁻⁷	3.05x10 ⁻³	6.99x10 ⁻⁷	3.99x10 ⁻⁶	0	0
Health Risk								· ,	-
HI ^g						1.48x10 ⁻⁶	2.14x10 ⁻⁴		
Cancer risk ^h								0	0

Table M.3.4–23. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site— Upgrade Plutonium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions concentrations)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286[converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: SR DOE 1994e; WSRC 1995e.

	Regulated E	xposure Lir	nits/Risk Factors	Emissions	Inventory	Н	(Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.46x10 ⁻⁶	2.68x10 ⁻²	1.82x10 ⁻⁶	4.88x10 ⁻⁴	0	0
Chlorine	0.35	3	f	1.23x10 ⁻⁸	1.34x10 ⁻⁴	3.51x10 ⁻⁸	4.48x10 ⁻⁵	0	0
Hydrazine	0.0326	1.3	17	1.53x10 ⁻⁹	1.68x10 ⁻⁵	4.71x10 ⁻⁸	1.29x10 ⁻⁵	7.48x10 ⁻⁹	1.11x10 ⁻⁵
Nitric acid	0.1225	5	f	9.22x10 ⁻⁹	1.00x10 ⁻⁴	7.53x10 ⁻⁸	2.01x10 ⁻⁵	0	0
Phosphoric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.68x10 ⁻⁵	0	0.
Sulfuric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.68x10 ⁻⁵	0	0
VOC (toluene)	0.4	766	f	2.92x10 ⁻⁷	3.19x10 ⁻³	7.30x 10 ⁻⁷	4.16x10 ⁻⁶	0	0
Health Risk								Ū	v
НI ^g						2.84x10 ⁻⁶	6.04x10 ⁻⁴		
Cancer risk ^h								7.48x10 ⁻⁹	1.11x10 ⁻⁵

Table M.3.4–24.Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—
Consolidate Plutonium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996e.

	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	H	(Q	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	2.61x10 ⁻⁶	2.85x10 ⁻²	1.93x10 ⁻⁶	5.19x10 ⁻⁴	0	0
Chlorine	0.35	3	f	1.53x10 ⁻⁸	1.68x10 ⁻⁴	4.39x10 ⁻⁸	5.60x10 ⁻⁵	0	0
	0.0326	1.33	17	1.53x10 ⁻⁹	1.68x10 ⁻⁵	4.71x10 ⁻⁸	1.29x10 ⁻⁵	7.48x10 ⁻⁹	1.11x10 ⁻⁵
Hydrazine	0.007	7	f	1.38x10 ⁻⁸	1.51x10 ⁻⁴	1.99x10 ⁻⁶	2.16x10 ⁻⁵	0	0
Hydrogen chloride Hydrogen fluoride	0.007	2.49	f	1.38x10 ⁻⁸	1.51x10 ⁻⁴	6.59x10 ⁻⁸	6.07x10 ⁻⁵	0	0
Nitric acid	0.1225	5	f	1.46x10 ⁻⁷	1.59x10 ⁻³	1.19x10 ⁻⁶	3.19x10 ⁻⁴	0	0
Phosphoric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.68x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.68x10 ⁻⁵	0	0
VOC (toluene)	0.0245	766	f	3.07x10 ⁻⁷	3.36x10 ⁻³	7.68x10 ⁻⁷	4.38x10 ⁻⁶	0	0
Health Risk						6.16x10 ⁻⁶	1.03x10 ⁻³	7 49-10-9	1.11x10 ⁻⁵
Cancer risk ^h						-		7.48x10 ⁻⁹	1.11X10

Table M.3.4–25. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site— Collocate Plutonium and Highly Enriched Uranium Storage Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: DOE 1996f.

Sector States

	Regulated	l Exposure Factors	Limits/Risk	Emissions	Inventory	Ĩ	Q	Canc	er Risk
Chemical	RfC (mg/m ³)	PELª	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Beryllium	0.0175	0.002	8.4	1.04x10 ⁻¹²	8.61x10 ⁻¹⁰	5.97x10 ⁻¹¹	4.30x10 ⁻⁷	2.51x10 ⁻¹²	2.80x10 ⁻¹⁰
Carbon monoxide	1.35	55	f	8.85x10 ⁻⁴	7.20x10 ⁻¹	6.562x10 ⁻⁴	1.32x10 ⁻²	0	0
Carbon tetrachloride	0.00245	63.9	0.053	1.21x10 ⁻⁶	9.96x10 ⁻⁴	4.93x10 ⁻⁴	1.55x10 ⁻⁵	1.83x10 ⁻⁸	2.04x10 ⁻⁶
Dioctyl phthalate	0.07	5	f	1.43x10 ⁻⁷	1.18x10 ⁻⁴	2.05x10 ⁻⁶	2.36x10 ⁻⁵	0	0
Freon 113	105	7,600	f	2.46x10 ⁻⁶	2.02×10^{-3}	2.34x10 ⁻⁸	2.66x10 ⁻⁷	0	0
Hydrogen sulfide	0.0009	28.4	f	1.05x10 ⁻⁸	8.69x10 ⁻⁶	1.17x10 ⁻⁵	3.06x1 ⁻⁷	0	0
Lead	0.001225	0.05	f	1.74x10 ⁻²⁰	1.43x10 ⁻¹⁷	1.42x10 ⁻¹⁷	2.87x10 ⁻¹⁶	0	0
Methylene chloride	3	1,765	0.0075	1.25x10 ⁻⁶	1.03x10 ⁻³	4.17x10 ⁻⁷	5.83x10 ⁻⁷	2.68x10 ⁻⁹	2.99x10 ⁻⁷
Nitric acid	0.1225	5	f	1.38x10 ⁻¹⁰	1.13x10 ⁻⁷	1.13x10 ⁻⁹	2.27x10 ⁻⁸	0	0
Trichloroethane	1	1,900	f	3.07x10 ⁻⁶	2.53x10 ⁻³	3.07x10 ⁻⁶	1.33x10 ⁻⁶	0	0
Health Risk HI ^g						1.17x10 ⁻³	1.33x10 ⁻²		
Cancer risk ^h								2.10x10 ⁻⁸	2.34x10 ⁻⁶

Table M.3.4-26. Risk Assessments From Exposure to Hazardous Chemicals at

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: RFETS 1995a:1.

	Regulated	Exposure Factors	Limits/Risk	Emissions	Inventory	н	IQ	Cance	er Risk
	<u></u>	Factors	· · · · · · · · · · · · · · · · · · ·	Emissions	Worker		Worker		Worker
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	100 Meters 8 Hours ^c	MEId	8 Hours ^e
1, 1, 2-Trichloroethane	0.014	45	0.057	1.03x10 ⁻⁴	2.27x10 ⁻²	7.33x10 ⁻³	5.04x10 ⁻⁴	1.67x10 ⁻⁶	5.01x10 ⁻⁵
2-Butoxyethanol	5.88	240	f	1.36x10 ⁻⁵	3.01x10 ⁻³	2.32x10 ⁻⁶	1.25x10 ⁻⁵	0	0
Acetic acid	0.6125	25	f	5.95x10 ⁻⁵	1.31x10 ⁻²	9.71x10 ⁻⁵	5.26x10 ⁻⁴	0	0
Aluminum (metal & oxide)	0.368	15	f	4.43x10 ⁻⁶	9.79x10 ⁻⁴	1.20x10 ⁻⁵	6.53x10 ⁻⁵	0	0
Aluminum welding fumes	0.1225	5	f	1.36x10 ⁻⁵	3.01x10 ⁻³	1.11x10 ⁻⁴	6.02×10^{-4}	0	0
Ammonia	0.1	35	f	8.85x10 ⁻⁵	1.96x10 ⁻²	8.85x10 ⁻⁴	5.59x10 ⁻⁴	0	0
Carbon monoxide	1.35	55	f	2.39x 10 ⁻³	5.28x10 ⁻¹	1.77x10 ⁻³	9.61x10 ⁻³	0	0
Chlorine	0.35	3	f	1.44×10^{-6}	3.18x10 ⁻⁴	4.11x10 ⁻⁶	1.06x10 ⁻⁴	0	0
Chloroform	0.035	240	0.081	5.90x10 ⁻⁵	1.31x10 ⁻²	1.69x10 ⁻³	5.44x10 ⁻⁵	1.37x10 ⁻⁶	4.09x10 ⁻⁵
	25.725	1050	f	3.10x10 ⁻⁶	6.86x10 ⁻⁴	1.21x10 ⁻⁷	6.53x10 ⁻⁷	0	0
Cyclohexane Dichlorodifluoromethane (Freon 12)	0.7	4950	f	1.88x10 ⁻⁶	4.16x10 ⁻⁴	2.69x10 ⁻⁶	8.41x10 ⁻⁸	0	0
Ethyl acetate	3.15	1400	f	9.85x10 ⁻⁶	2.18x10 ⁻³	3.13x10 ⁻⁶	1.56x10 ⁻⁶	0	0
Ethylene glycol	7	127	f	7.97x10 ⁻⁶	1.76x10 ⁻³	1.14x10 ⁻⁶	1.39x10 ⁻⁵	0	0
Ethyl ether	0.7	1200	f	1.88x10 ⁻⁶	4.16x10 ⁻⁴	2.69x10 ⁻⁶	3.47x10 ⁻⁷	0	0
Formaldehyde	0.7	0.9375	0.045	5.43x10 ⁻⁶	1.20x10 ⁻³	7.75x10 ⁻⁶	1.28×10^{-3}	6.98x10 ⁻⁸	2.09x10 ⁻⁶
Heavy metals (zinc)	1.05	42.9	f	1.26x10 ⁻⁵	2.79x10 ⁻³	1.20x10 ⁻⁵	6.51x10 ⁻⁵	0	0
Heptane	49	2000	f	2.05×10^{-4}	4.53×10^{-2}	4.18x10 ⁻⁶	2.26x10 ⁻⁵	0	0
Hexane	0.2	1800	f	8.53x10 ⁻⁶	1.89x10 ⁻³	4.26x10 ⁻⁵	1.05x10 ⁻⁶	0	0
Hexane (other isomers)	0.2	1800	f	1.33x10 ⁻⁶	2.94x10 ⁻⁴	6.64x10 ⁻⁶	1.63x10 ⁻⁷	0	0
Hydrocarbons (hexane)	0.2	1800	f	3.20x 10 ⁻⁴	7.08x10 ⁻²	1.60x10 ⁻³	3.94x10 ⁻⁵	0	0
Hydrogen chloride	0.007	7	f	7.06x10 ⁻⁵	1.56x10 ⁻²	1.01x10 ⁻²	2.23x10 ⁻³	0	0
Hydrogen fluoride	0.007	, 2.49	f	2.68x10 ⁻⁵	5.93x10 ⁻³	1.28x10 ⁻⁴	2.38x10 ⁻³	0	0
Hydrogen peroxide	0.0343	1.4	f	2.21x10 ⁻⁶	4.90x10 ⁻⁴	6.46x10 ⁻⁵	3.50x10 ⁻⁴	0	0
Isobutyl acetate	17.15	700	f	1.99x10 ⁻⁶	4.41x10 ⁻⁴	1.16x10 ⁻⁷	6.30x10 ⁻⁷	0	0
	24.15	980	f	5.97x10 ⁻⁵	1.32×10^{-2}	2.47x10 ⁻⁶	1.35x10 ⁻⁵	0	0
Isopropyl alcohol	24.13	100	f	2.88x10 ⁻⁵	6.37x10 ⁻³	1.18x10 ⁻⁵	6.37x10 ⁻⁵	0	0
Kerosene Lead	0.001225		f	2.88x10 ⁻⁶	6.37x10 ⁻⁴	2.35x10 ⁻³	1.27×10^{-2}	0	0

Table M.3.4–27. Risk Assessments From Exposure to Hazardous Chemicals at Los Alamos National Laboratory-No Action

Health and Safety

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	Regulated	-	e Limits/Risk						
	<u> </u>	Factors	S	Emissions	Inventory	F	IQ	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Lead chromate	0.001225	0.05	f	1.77x10 ⁻⁶	3.92x10 ⁻⁴	1.45x10 ⁻³	7.84x10 ⁻³	0	0 ·
Methanol	1.75	260	f	6.52x10 ⁻⁵	1.44x10 ⁻²	3.73x10 ⁻⁵	5.55x10 ⁻⁵	0	0
Methyl chloride	5.145	210	f	2.88x10 ⁻⁶	6.37x10 ⁻⁴	5.60x10 ⁻⁷	3.03x10 ⁻⁶	0	0
Methyl ethyl ketone (MEK)	1	590	f	2.06x10 ⁻⁴	4.56x10 ⁻²	2.06x10 ⁻⁴	7.74x10 ⁻⁵	0	0
Methylene chloride	3	1765	0.0075	1.22×10^{-4}	2.70x10 ⁻²	4.07x10 ⁻⁵	1.53x10 ⁻⁵	2.62x10 ⁻⁷	7.85x10 ⁻⁶
Nickel (metal)	0.0245	1	0.84	6.09x10 ⁻⁶	1.35x10 ⁻³	2.49x10 ⁻⁴	1.35x10 ⁻³	1.46x10 ⁻⁶	4.38x10 ⁻⁵
Nitric acid	0.1225	5	f	7.32x10 ⁻⁵	1.62x10 ⁻²	5.97x10 ⁻⁴	3.24x10 ⁻³	0	0
Phosgene	0.0098	0.4	f	2.55x10 ⁻⁶	5.63x10 ⁻⁴	2.60x10 ⁻⁴	1.41x10 ⁻³	0	0
Propylene oxide	0.03	240	0.24	3.99x10 ⁻⁶	8.81x10 ⁻⁴	1.33x10 ⁻⁴	3.67x10 ⁻⁶	2.74x10 ⁻⁷	8.19x10 ⁻⁶
Stoddard solvent	71.05	2900	f	2.92x10 ⁻⁵	6.46x10 ⁻³	4.11x10 ⁻⁷	2.23x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	2.44x10 ⁻⁶	5.39x10 ⁻⁴	9.94x10 ⁻⁵	5.39x10 ⁻⁴	0	0
Tetrahydrofuran (THF)	14.455	590	f	1.88x10 ⁻⁶	4.16x10 ⁻⁴	1.30x10 ⁻⁷	7.06x10 ⁻⁷	0	0
Toluene	0.4	766	f	2.75x10 ⁻⁴	6.08x10 ⁻²	6.87x10 ⁻⁴	7.94x10 ⁻⁵	0	0
Tricloroethylene (TCE)	13.377	546	0.006	2.33x10 ⁻⁵	5.14x10 ⁻³	1.74x10 ⁻⁶	9.42x10 ⁻⁶	3.99x10 ⁻⁸	1.19x10 ⁻⁶
Tungsten	0.1225	5	f	1.21x10 ⁻⁵	2.67x10 ⁻³	9.85x10 ⁻⁵	5.34x10 ⁻⁴	0	0
VM&P Naphtha	8.575	245	f	6.79x10 ⁻⁵	1.50x10 ⁻²	7.92x10 ⁻⁶	6.13x10 ⁻⁵	0	0
Welding fumes (acetylene)	65.219	2662	f	5.66x10 ⁻⁵	1.25x10 ⁻²	8.68x10 ⁻⁷	4.70x10 ⁻⁶	0	0
Xylene	7	435	f	1.95x10 ⁻⁴	4.31x10 ⁻²	2.79x10 ⁻⁵	9.92x10 ⁻⁵	0	0
Health Risk HI ^g						3.01x10 ⁻²	4.65x10 ⁻²		
Cancer risk ^h								5.15x10 ⁻⁶	1.54x10 ⁻⁴

Table M.3.4–27. Risk Assessments From Exposure to Hazardous Chemicals at Los Alamos National Laboratory—No Action—Continued

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: LANL1994a.

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	Regulated E	xposure Lin	nits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent	0.74	30	f	4.23x10 ⁻⁶	1.40x10 ⁻²	5.72x10 ⁻⁶	4.67x10 ⁻⁴	0	0
(butyl lactate) VOC (toluene)	0.40	766	f	8.46x10 ⁻⁶	2.80x10 ⁻²	2.12x10 ⁻⁵	3.66x10 ⁻⁵	0	0
Health Risk HI ^g						2.69x10 ⁻⁵	5.04x10 ⁻⁴		
Cancer risk ^h								0	0

Table M.3.4–28. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Pit Disassembly/Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

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	Regulated E	Exposure Lir	nits/Risk Factors	Emissions	s Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	6.30x10 ⁻⁷	7.28x10 ⁻³	8.52x10 ⁻⁷	2.43x10 ⁻⁴	0	0
VOC (toluene) Health Risk	0.40	766	f	1.26x10 ⁻⁶	1.46x10 ⁻²	3.15x10 ⁻⁶	1.90x10 ⁻⁵	0	0
HI ^g Cancer risk ^h						4.00x10 ⁻⁶	2.62x10 ⁻⁴	0	0

Table M.3.4–29. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site—Pit Disassembly/Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HOs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

Regulated E	Aboonic Tun	Regulated Exposure Limits/Risk Factors			HQ		Cancer Risk	
RfC (mg/m ³)	PELª	Slope Factor	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
		1	9.07x10 ⁻⁶	1.42×10^{-2}	1.23x10 ⁻⁵	4.72x10 ⁻⁴	0	0
0.74	766	f	1.81x10 ⁻⁵	2.83x10 ⁻²	2.54x10 ⁻⁵	3.70x10 ⁻⁵	0	0
						- 4		
					5.76x10 ⁻⁵	5.09×10^{-4}		<u>^</u>
						_	0	0
	(mg/m³) 0.74	(mg/m ³) (mg/m ³) 0.74 30	RfC PEL ^a Factor (mg/m ³) (mg/m ³) (mg/kg/day) ⁻¹ 0.74 30 f	RfC PEL ^a Factor MEI (mg/m ³) (mg/m ³) (mg/kg/day) ⁻¹ (mg/m ³) 0.74 30 f 9.07x10 ⁻⁶	RfCPELaFactorMEI8 Hours (mg/m^3) (mg/m^3) (mg/m^3) (mg/m^3) (mg/m^3) 0.7430f $9.07x10^{-6}$ $1.42x10^{-2}$	RfC PEL ^a Factor MEI 8 Hours MEI ^b (mg/m ³) (mg/m ³) (mg/kg/day) ⁻¹ (mg/m ³) (mg/m ³) 0.74 30 f 9.07x10 ⁻⁶ 1.42x10 ⁻² 1.23x10 ⁻⁵	RfC (mg/m³)PEL ^a (mg/m³)Factor (mg/kg/day)^1MEI (mg/m³)8 Hours (mg/m³)MEI ^b 8 Hours ^c 8 Hours ^c 0.7430f $9.07x10^{-6}$ $1.42x10^{-2}$ $1.23x10^{-5}$ $4.72x10^{-4}$ 0.40766f $1.81x10^{-5}$ $2.83x10^{-2}$ $2.54x10^{-5}$ $3.70x10^{-5}$	RfC PEL ^a Factor MEI 8 Hours MEI ^b 8 Hours ^c MEI ^d (mg/m ³) (mg/m ³) (mg/kg/day) ⁻¹ (mg/m ³) 0

Table M.3.4-30. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory-Pit Disassembly/Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HO for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

8 HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

	Regulated E	xposure Lir	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	2.36x10 ⁻⁵	7.09x10 ⁻³	3.19x10 ⁻⁵	2.36x10 ⁻⁴	0	0
VOC (toluene) Health Risk	0.40	766	f	4.72x10 ⁻⁵	1.42×10^{-2}	1.18x10 ⁻⁴	1.85x10 ⁻⁵	0	0
HI ^g Cancer risk ^h						1.50x10 ⁻⁴	2.55x10 ⁻⁴	0	0

Table M.3.4-31. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant-Pit Disassembly/Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)*(0.286 [converts concentration to dose])*(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)*(0.237 [fraction of year exposed])*(0.571 [fraction of lifetime working])*(0.286 [converts concentration to dose])*(SF).

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^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	2.48x10 ⁻⁵	1.49x10 ⁻²	3.35x10 ⁻⁵	4.95x10 ⁻⁴	0	0
VOC (toluene)	0.40	766	f	4.95x10 ⁻⁵	2.97x10 ⁻²	1.24×10^{-4}	3.88x10 ⁻⁵	0	0
Health Risk HI ^g						1.57x10 ⁻⁴	5.34x10 ⁻⁴		
Cancer risk ^h								0	0

Table M.3.4–32. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Pit Disassembly/Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

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	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	1.15x10 ⁻⁶	1.26x10 ⁻²	1. 56 x10 ⁻⁶	4.20x10 ⁻⁴	0	0
VOC (toluene)	0.40	766	f	2.31x10 ⁻⁶	2.52×10^{-2}	5.77x10 ⁻⁶	3.29x10 ⁻⁵	0	0
Health Risk HI ^g						7.33x10 ⁻⁶	4.53x10 ⁻⁴		
Cancer risk ^h								0	0

Table M.3.4–33.Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Pit Disassembly/Conversion Facility

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

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^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEl=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996d.

	Remisted E	xposure Limit	s/Risk Factors	Emissions	Inventory	Н	Q	Cance	r Risk
- Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Ammonia	0.1	35	f	5.64x10 ⁻⁸	1.87x10 ⁻⁴	5.64x10 ⁻⁷	5.3x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	2.25x10 ⁻⁵	7.48x10 ⁻²	1.67x10 ⁻⁵	1.3x10 ⁻³	0	0
Chlorine	0.35	3	f	4.23x10 ⁻⁸	1.40x10 ⁻⁴	1.20x10 ⁻⁷	4.67x10 ⁻⁵	0	0
Chorne Cleaning solvent (butyl lactate)	0.74	30	f	5.64x10 ⁻⁷	1.87x10 ⁻³	7.62x10 ⁻⁷	6.23x10 ⁻⁵	0	0
Ethanol	46.55	1,900	f	1.12x10 ⁻⁷	3.74x10 ⁻⁴	2.42x10 ⁻⁹	1.96x10 ⁻⁷	0	0
Hydrazine	0.0326	1,200	17	5.64x10 ⁻⁹	1.87x10 ⁻⁵	1.73x10 ⁻⁷	1.43x10 ⁻⁵	2.74x10 ⁻⁸	1.23x10 ⁻⁵
Hydrogen chloride	0.007	7	f	6.77x10 ⁻⁸	2.24x10 ⁻⁴	9.67x10 ⁻⁶	3.20x10 ⁻⁵	0	0
Hydrogen fluoride	0.007	2.49	f	4.51x10 ⁻⁹	1.49x10 ⁻⁵	2.15x10 ⁻⁸	6.00x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	1.69x10 ⁻⁸	5.61x10 ⁻⁵	1.38x10 ⁻⁷	1.12x10 ⁻⁵	0	0
Octanol	2.52	102.8	f	6.32x10 ⁻¹⁰	2.09x10 ⁻⁶	2.51x10 ⁻¹⁰	2.03x10 ⁻⁸	0	0
Phosphoric acid	0.0245	102.0	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x10 ⁻⁷	1.87x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	5.64x10 ⁻⁹	1.87x10 ⁻⁵	2.30x10 ⁻⁷	1.87x10 ⁻⁵	0	0
	0.1225	5	f	5.64x10 ⁻¹⁰	1.87x10 ⁻⁶	4.60x10 ⁻⁹	3.74x10 ⁻⁷	0	0
Tributyl phosphate Trichloroethylene	13.377	546	0.006	2.53×10^{-6}	8.41x10 ⁻³	1.89x10 ⁻⁷	1.54x10 ⁻⁵	4.36x10 ⁻⁹	1.95x10 ⁻⁶
Health Risk HI ^g Cancer risk ^h						2.88x10 ⁻⁵	1.59x10 ⁻³	3.18x10 ⁻⁸	1.43x10 ⁻⁵

Table M.3.4–34. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site Plutonium Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: LANL 1996c.

	Regulated E	xposure Lim	its/Risk Factors	Emissions	s Inventory	H	(Q	Cance	r Risk
	RfC	PELª	Slope Factor	Boundary MEI	Worker 100 Meters 8 Hours	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meter 8 Hours ^e
Chemical	(mg/m ³)	(mg/m³)	(mg/kg/day) ⁻¹	(mg/m ³)	(mg/m^3)				U MUULU
Ammonia	0.1	35	f	8.40x10 ⁻⁹	9.70x10 ⁻⁵	8.40x10 ⁻⁹	2.77x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	3.36x10 ⁻⁶	3.88x10 ⁻²	2.49x10 ⁻⁶	7.05x10 ⁻⁴	0	0
Chlorine	0.35	3	f	6.30x10 ⁻⁹	7.27x10 ⁻⁵	1.80x10 ⁻⁸	2.42x10 ⁻⁵	0	0
Cleaning solvent (butyl lactate)	0.74	30	f	8.40x10 ⁻⁸	9.70x10 ⁻⁴	1.13x10 ⁻⁷	3.2x10 ⁻⁵	0	0
[Text deleted.]									
Ethanol	46.55	1,900	f	1.68x10 ⁻⁸	1.94x10 ⁻⁴	3.61x10 ⁻¹⁰	1.02x10 ⁻⁷	0	0
Hydrazine	0.0326	1.3	17	8.40x10 ⁻¹⁰	9.70x 10 ⁻⁶	2.57x10 ⁻⁸	7.46x10 ⁻⁶	4.09x10 ⁻⁹	6.38x10
Hydrogen chloride	0.007	7	f	1.00x10 ⁻⁸	1.16x10 ⁻⁴	1.44x10 ⁻⁶	1.66x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	6.72x10 ⁻¹⁰	7.76x10 ⁻⁵	3.20x10 ⁻⁹	3.11x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	2.52x10 ⁻⁹	2.91x10 ⁻⁵	2.05x10 ⁻⁸	5.82x10 ⁻⁶	0	ů 0
Octanol	2.52	102.8	f	9.42x10 ⁻¹¹	1.08x10 ⁻⁶	3.73x10 ⁻¹¹	1.05x10 ⁻⁸	0	ů 0
Phosphoric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	8.40x10 ⁻¹⁰	9.70x 10 ⁻⁶	3.43x10 ⁻⁸	9.70x10 ⁻⁶	0	0
Tributyl phosphate	0.1225	5	f	8.40x10 ⁻¹¹	9.70x 10 ⁻⁷	6.86x10 ⁻¹⁰	1.94x10 ⁻⁷	0	0
Trichloroethylene	13.377	546	0.006	3.78x10 ⁻⁷	4.36x10 ⁻³	2.82x10 ⁻⁸	7.99x10 ⁻⁶	6.49x10 ⁻¹⁰	1.01x10 ⁻
Health Risk HI ^g						4.29x10 ⁻⁶	8.26x10 ⁻⁴		
Cancer risk ^h								4.73x10 ⁻⁹	7.40x10⁻

Table M.3.4–35. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site— Plutonium Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

<u></u>	Regulated E	xposure Limit	s/Risk Factors	Emissions	Inventory	Н	Q	Cance	r Risk
- Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meter 8 Hours ^e
Ammonia	0.1	35	f	1.21x10 ⁻⁷	1.88x10 ⁻⁴	1.21x10 ⁻⁶	5.39x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	4.83x10 ⁻⁵	7.55x10 ⁻²	3.58x10 ⁻⁵	1.37x10 ⁻³	0	0
Chlorine	0.35	3	f	9.07x10 ⁻⁸	1.41x10 ⁻⁴	2.59x10 ⁻⁷	4.72x10 ⁻⁵	0	0
Cleaning solvent (butyl lactate)	0.74	30	f	1.21x10 ⁻⁶	1.88x10 ⁻³	1.63x10 ⁻⁶	6.29x10 ⁻⁵	0	0
[Text deleted.]						_	_		
Ethanol	46.55	1,900	f	2.41x10 ⁻⁷	3.77x10 ⁻⁴	5.19x10 ⁻⁹	1.98x10 ⁻⁷	0	0
Hydrazine	0.0326	1.3	17	1.21x10 ⁻⁸	1.88x10 ⁻⁵	3.71x10 ⁻⁷	1.45x10 ⁻⁵	5.88x10 ⁻⁸	1.24x10 ⁻⁵
Hydrogen chloride	0.007	7	f	1.45x10 ⁻⁷	2.26x10 ⁻⁴	2.07x10 ⁻⁵	3.23x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	9.67x10 ⁻⁹	1.51x10 ⁻⁵	4.60x10 ⁻⁸	6.06x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	3.62x10 ⁻⁸	5.66x10 ⁻⁵	2.96x10 ⁻⁷	1.13x10 ⁻⁵	0	0
Octanol	2.52	102.8	f	1.35x10 ⁻⁹	2.11x10 ⁻⁶	5.38x10 ⁻¹⁰	2.05x10 ⁻⁸	0	0
Phosphoric acid	0.0245	1	f	1.21x10 ⁻⁸	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	1.21x10 ⁻⁸	1.88x10 ⁻⁵	4.93x10 ⁻⁷	1.88x10 ⁻⁵	0	0
Tributyl phosphate	0.1225	5	f	1.21x10 ⁻⁹	1.88x10 ⁻⁶	9.87x10 ⁻⁹	3.77x10 ⁻⁷	0	0
Trichloroethylene	13.377	546	0.006	5.44x10 ⁻⁶	8.49x10 ⁻³	4.06x10 ⁻⁷	1.55x10 ⁻⁵	9.34x10 ⁻⁹	1.97x10 ⁻⁶
Health Risk HI ^g Cancer risk ^h						6.18x10 ⁻⁵	1.61x10 ⁻³	6.81x10 ⁻⁸	1.44x10 ⁻⁵

Table M.3.4–36. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory— Plutonium Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

-	Regulated E	Exposure Limi	its/Risk Factors	Emissions	Inventory	H	Q	Cance	er Risk
			Slope	Boundary	Worker 100 Meters	Dawadami	Worker	D	Worker
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Factor (mg/kg/day) ⁻¹	MEI (mg/m ³)	8 Hours (mg/m ³)	Boundary MEI ^b	100 Meters 8 Hours ^c	Boundary MEI ^d	100 Meter 8 Hours ^e
Ammonia	0.1	35	f	3.14x10 ⁻⁷	9.44x10 ⁻⁵	3.14x10 ⁻⁶	2.70x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	1.25x10 ⁻⁴	3.78x10 ⁻²	9.35x10 ⁻⁵	6.87x10 ⁻⁴	0	0
Chlorine	0.35	3	f	2.35x10 ⁻⁷	7.08x10 ⁻⁵	6.74x10 ⁻⁷	2.36x10 ⁻⁵	0	0
Cleaning solvent (butyl lactate)	0.74	30	f	3.14x10 ⁻⁶	9.44x10 ⁻⁴	4.25x10 ⁻⁶	3.15x10 ⁻⁵	0	0
[Text deleted.]									
Ethanol	46.55	1,900	f	6.29x10 ⁻⁷	1.89x10 ⁻⁴	1.35x10 ⁻⁸	9.94x10 ⁻⁸	0	0
Hydrazine	0.0326	1.33	17	3.14x10 ⁻⁸	9.44x10 ⁻⁶	9.64x10 ⁻⁷	7.26x10 ⁻⁶	1.53x10 ⁻⁷	6.22x10 ⁻⁶
Hydrogen chloride	0.007	7	f	3.77x10 ⁻⁷	1.13x10 ⁻⁴	5.39x10 ⁻⁵	1.62x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	2.51x10 ⁻⁸	7. 55x10⁻⁶	1.19x10 ⁻⁷	3.03x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	9.43x10 ⁻⁸	2.83x10 ⁻⁵	7.70x10 ⁻⁷	5.66x10 ⁻⁶	0	0
Octanol	2.52	102.8	f	3.52x10 ⁻⁹	1.05x10 ⁻⁶	1.39x10 ⁻⁹	1.03x10 ⁻⁸	0	0
Phosphoric acid	0.0245	1	f	3.14x10 ⁻⁸	9.44x10 ⁻⁶	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
Sulfuric acid	0.0245	1	f	3.14x10 ⁻⁸	9.44x10 ⁻⁶	1.28x10 ⁻⁶	9.44x10 ⁻⁶	0	0
Tributyl phosphate	0.1225	5	f	3.14x10 ⁻⁹	9.44x10 ⁻⁷	2.56x10 ⁻⁸	1.89x10 ⁻⁷	0	0
Trichloroethylene	13.377	546	0.006	1.41x10 ⁻⁵	4.25x10 ⁻³	1.05x10 ⁻⁶	7.78x10 ⁻⁶	2.43x10 ⁻⁸	9.87x10 ⁻⁷
Health Risk HI ^g						1.61x10 ⁻⁴	8.01x10 ⁻⁴		
Cancer risk ^h								1.77x10 ⁻⁷	7.20x10 ⁻⁶

Table M.3.4–37. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant— Plutonium Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

	Regulated H	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ.	Cancer Risk	
			· <u>-</u> ···		Worker		Worker		Worker
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	100 Meters 8 Hours ^c	Boundary MEI ^d	100 Meter 8 Hours ^e
Ammonia	0.1	35	f	3.30x10 ⁻⁷	1.98x10 ⁻⁴	3.30x10 ⁻⁶	5.66x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	1.32x10 ⁻⁴	7.92x10 ⁻²	9.78x10 ⁻⁵	1.44x10 ⁻³	0	0
Chlorine	0.35	3	f	2.47x10 ⁻⁷	1.48x10 ⁻⁴	7.07x10 ⁻⁷	4.95x10 ⁻⁵	0	0
Cleaning solvent (butyl lactate)	0.74	30	f	3.30x10 ⁻⁶	1.98x10 ⁻³	4.46x10 ⁻⁶	6.60x10 ⁻⁵	0	0
[Text deleted.]									
Ethanol	46.55	1,900	f	6.06x10 ⁻⁷	3.96x10 ⁻⁴	1.41x10 ⁻⁸	2.08x10 ⁻⁷	0	0
Hydrazine	0.0326	1.33	17	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.01x10 ⁻⁶	1.52x10 ⁻⁵	1.61x10 ⁻⁷	1.30x10 ⁻⁵
Hydrogen chloride	0.007	7	f	3. 96x 10 ⁻⁷	2.37x10 ⁻⁴	5.66x10 ⁻⁵	3.39x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	2.64x10 ⁻⁸	1.58x10 ⁻⁵	1.25x10 ⁻⁷	6.36x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	9.90x10 ⁻⁸	5.94x10 ⁻⁵	8.08x10 ⁻⁷	1.18x10 ⁻⁵	0	0
Octanol	2.52	102.8	f	3.70x10 ⁻⁹	2.22x10 ⁻⁶	1.46x10 ⁻⁹	2.16x10 ⁻⁸	0	0
Phosphoric acid	0.0245	1	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.34x10 ⁻⁶	1.98x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	3.30x10 ⁻⁸	1.98x10 ⁻⁵	1.34x10 ⁻⁶	1.98x10 ⁻⁵	0	0
Tributyl phosphate	0.1225	5	f	3.30x10 ⁻⁹	1.98x10 ⁻⁶	2.69x10 ⁻⁸	3. 96x10 -7	0	0
Trichloroethylene	13.377	546	0.006	1.48x10 ⁻⁵	8.91x10 ⁻³	1.11x10 ⁻⁶	1.63x10 ⁻⁵	2.55x10 ⁻⁸	2.07x10 ⁻⁶
Health Risk HI ^g Cancer risk ^h						1.69x10 ⁻⁴	1.69x10 ⁻³	1.86x10 ⁻⁷	1.51x10 ⁻⁵

Table M.3.4–38. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Plutonium Conversion Facility

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: LANL 1996c.

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	Regulated E	xposure Lim	its/Risk Factors	Emissions	Inventory	H	Q	Cance	er Risk
			Slope	Boundary	Worker 100 Meters	Boundary	Worker 100 Meters	Boundary	Worker 100 Meters
	RfC	PELª	Factor	MEI	8 Hours	MEI ^b	8 Hours ^c	MEI ^d	8 Hours ^e
Chemical	(mg/m ³)	(mg/m^3)	(mg/kg/day) ⁻¹	(mg/m ³)	(mg/m ³)				
Ammonia	0.1	35	f	1.53x10 ⁻⁸	1.68x10 ⁻⁴	1.53x10 ⁻⁷	4.80x10 ⁻⁶	0	0
Carbon monoxide	1.35	55	f	6.15x10 ⁻⁶	6.7×10^{-2}	4.55x10 ⁻⁶	1.22×10^{-3}	0	0
Chlorine	0.35	3	f	1.15x10 ⁻⁸	1.26x10 ⁻⁴	3.29x10 ⁻⁸	4.20x10 ⁻⁵	0	0
Cleaning solvent (butyl lactate)	0.74	30	f	1.53x10 ⁻⁷	1.68x10 ⁻³	2.07x10 ⁻⁷	5.60x10 ⁻⁵	0	0
[Text deleted.]									
Ethanol	46.55	1,900	f	3.07x10 ⁻⁸	3.36x10 ⁻⁴	6.60x10 ⁻¹⁰	1.76x10 ⁻⁷	0	0
Hydrazine	0.0326	1.33	17	1.53x10 ⁻⁹	1.68x10 ⁻⁵	4.71x10 ⁻⁸	1.29x10 ⁻⁵	7.48x10 ⁻⁹	1.11x10 ⁻⁵
Hydrogen chloride	0.007	7	f	1.84x10 ⁻⁸	2.01x10 ⁻⁴	2.63x 10 ⁻⁶	2.88x10 ⁻⁵	0	0
Hydrogen fluoride	0.21	2.49	f	1.23x10 ⁻⁹	1.34x10 ⁻⁵	5.85x10 ⁻⁹	5.39x10 ⁻⁶	0	0
Nitric acid	0.1225	5	f	4.61x10 ⁻⁹	5.04x10 ⁻⁵	3.76x10 ⁻⁸	1.00x10 ⁻⁵	0	0
Octanol	2.52	102.8	f	1.72x10 ⁻¹⁰	1.88x10 ⁻⁶	6.84x10 ⁻¹¹	1.83x10 ⁻⁸	0	0
Phosphoric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.00x10 ⁻⁵	0	0
Sulfuric acid	0.0245	1	f	1.53x10 ⁻⁹	1.68x10 ⁻⁵	6.27x10 ⁻⁸	1.68x10 ⁻⁵	0	0
Tributyl phospate	0.1225	5	f	1.53x10 ⁻¹⁰	1.68x10 ⁻⁶	1.25x10 ⁻⁹	3.36x10 ⁻⁷	0	0
Trichloroethylene	13.377	546	0.006	6.92x10 ⁻⁷	7.56x10 ⁻³	5.17x10 ⁻⁸	1.38x10 ⁻⁵	1.19x10 ⁻⁹	1.76x10 ⁻⁶
Health Risk HI ^g						7.86x 10 ⁻⁶	1.43x10 ⁻³		
Cancer risk ^h								8.66x10 ⁻⁹	1.28x10 ⁻⁵

Table M.3.4-39.Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site-Plutonium Conversion Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

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^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

	Regulated E	xposure Lir	nits/Risk Factors	Emissions	s Inventory	Н	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	3.71x10 ⁻⁴	2.23x10 ⁻¹	2.75x10 ⁻⁴	4.05x10 ⁻³	0	0
Hydrocarbons (pyrene)	0.105	0.2	f	9.34x10 ⁻⁵	5.60x10 ⁻²	8.90x10 ⁻⁴	2.80x10 ⁻¹	0	0
Health Risk HI ^g						1.17x10 ⁻³	2.85x10 ⁻¹		
Cancer risk ^h								0	0

Table M.3.4-40. Risk Assessments From Exposure to Hazardous Chemicals at the Deep Borehole Complex—Direct Disposition Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HQ=sum of individual HQs.

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^h Total cancer risk=sum of individual cancer risks.

Note: Emissions in the source document are for 10 years; therefore, emissions are divided by 10 for the annual emissions used in the HI and cancer risk calculations. Source: LLNL 1996a.

-	Regulated Exp	osure Limits/Risk	Factors	Emissions	Inventory	H	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/ day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meter 8 Hours ^e
[Text deleted.]					······································				
Carbon dioxide	221	9,000	f	5.13x10 ⁻²	1.70x10 ²	2.32x10 ⁻⁴	1.89x10 ⁻²	0	0
Carbon monoxide	1.35	55	f	1.80x10 ⁻⁴	5.95x10 ⁻¹	1.33x10 ⁻⁴	1.08x10 ⁻²	ů 0	0
Chloride (HCI)	0.007	7	f	6.16x10 ⁻⁶	2.04x10 ⁻²	8.79x10 ⁻⁴	2.91x10 ⁻³	0	0
Fluoride (HF)	0.21	2.49	f	7.70x10 ⁻⁸	2.55x10 ⁻⁴	3.66x10 ⁻⁷	1.02x10 ⁻⁴	0	ů 0
Hydrocarbons (pyrene)	0.105	0.2	f	5.39x10 ⁻⁶	1.78x10 ⁻²	5.13x10 ⁻⁵	8.92x10 ⁻²	0	0
Iron (salts)	0.0245	1	f	7.70x10 ⁻⁸	2.55x10 ⁻⁴	3.14x10 ⁻⁶	2.55x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	7.70x10 ⁻⁶	2.55x10 ⁻²	2.09x10 ⁻⁵	1.70x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	3.85x10 ⁻⁶	1.27x10 ⁻²	1.57x10 ⁻⁴	1.27x10 ⁻²	0	0
Phosphonates (phosgene)	0.0098	0.4	f	7.70x10 ⁻⁷	2.55x10 ⁻³	7.85x10 ⁻⁵	6.37x10 ⁻³	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	2.57x10 ⁻¹⁵	8.50x10 ⁻¹²	1.47x10 ⁻¹³	8.50x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	5.39x10 ⁻⁷	1.78x10 ⁻³	1.35x10 ⁻⁶	2.33x10 ⁻⁶	0	0
Health Risk HI ^g						1.56x10 ⁻³	1.43×10^{-1}	Ū	0
Cancer risk ^h						1.JUATU	1.43810	0	0

Table M.3.4–41. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Ceramic Immobilization Facility for the Immobilized Disposition Alternative

Storage and Disposition of Weapons-Usable Fissile Materials Final PEIS

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/ day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
221	9,000	f	7.64x10 ⁻³	8.82x10 ¹	3.46x10 ⁻⁵	9.80x10 ⁻³	0	0
1.35	55	f	2.67×10^{-5}	3.09x10 ⁻¹	1.98x10 ⁻⁵	5.61x10 ⁻³	0	0
0.007	7	f	9.17x10 ⁻⁷	1.06x10 ⁻²	1.31x10 ⁻⁴	1.51x10 ⁻³	0	0
0.21	2.49	f	1.15x10 ⁻⁸	1.32x10 ⁻⁴	5.46x10 ⁻⁸	5.31x10 ⁻⁵	ů 0	0
0.105	0.2	f	8.02×10^{-7}	9.26x10 ⁻³	7.64x10 ⁻⁶	4.63×10^{-2}	0	0
0.0245	1	f	1.15x10 ⁻⁸	1.32x10 ⁻⁴	4.68x10 ⁻⁷	1.32x10 ⁻⁴	0	0
0.368	15	f	1.15x10 ⁻⁶	1.32x10 ⁻²	3.11x10 ⁻⁶	8.82x10 ⁻⁴	0	0
0.0245	1	f	5.73x10 ⁻⁷	6.61x10 ⁻³	2.34x10 ⁻⁵	6.61x10 ⁻³	0	0
0.0098	0.4	f	1.15x10 ⁻⁷	1.32x10 ⁻³	1.17x10 ⁻⁵	3.31x10 ⁻³	0	0
0.0175	0.01	- f	3.82x10 ⁻¹⁶	4.41x10 ⁻¹²	2.18x10 ⁻¹⁴	4.41x10 ⁻¹⁰	0	0
0.4	766	f	8.02x10 ⁻⁸	9.26x10 ⁻⁴	2.01×10^{-7}	1.21x10 ⁻⁶	0	0 0
					2.32x10 ⁻⁴	7.42x10 ⁻²		
							0	0

HO

Cancer Risk

Table M.3.4-42. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site-Ceramic Immobilization Facility for the Immobilized Disposition Alternative

Emissions Inventory

^a See Table M.3.3-1 for the OSHA-PEL ^b HO for MEI=boundary annual emissio

Regulated Exposure Limits/Risk Factors

 (mg/m^3)

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HOs.

Chemical

Carbon monoxide

Chloride (HCI)

Fluoride (HF)

Hydrocarbons

(pyrene) Iron (salts)

Magnesium

Phosphonates

(phosgene) [Text deleted.]

VOC (toluene)

Cancer risk^h

Health Risk НIg

Silver

(oxide fume) **Phosphates**

(phosphoric acid)

[Text deleted.] Carbon dioxide

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

	Regulated E	Exposure Lim	its/Risk Factors	Emission	s Inventory	H	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	1.10x10 ⁻¹	1.72×10^2	4.98x10 ⁻⁴	1.91x10 ⁻²	0	0
Carbon monoxide	1.35	55	f	3.85x10 ⁻⁴	6.01x10 ⁻¹	2.85x10 ⁻⁴	1.09×10^{-2}	õ	0
Chloride (HCI)	0.007	7	f	1.32x10 ⁻⁵	2.06x10 ⁻²	1.89x10 ⁻³	2.94×10^{-3}	0 0	0
Fluoride (HF)	0.21	2.49	f	1.65x10 ⁻⁷	2.58x10 ⁻⁴	7.85x10 ⁻⁷	1.03×10^{-4}	Õ	0
Hydrocarbons (pyrene)	0.105	0.2	f	1.15x10 ⁻⁵	1.80x10 ⁻²	1.10x10 ⁻⁴	9.01x10 ⁻²	0	0
Iron (salts)	0.0245	1	f	1.65x10 ⁻⁷	2.58x10 ⁻⁴	6.73x10 ⁻⁶	2.58x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	1.65x10 ⁻⁵	2.58x10 ⁻²	4.48x10 ⁻⁵	1.72×10^{-3}	0	0
Phosphates (phosphoric acid)	0.0245	1	f	8.25x10 ⁻⁶	1.29x10 ⁻²	3.37x10 ⁻⁴	1.29x10 ⁻²	0	0
Phosphonates (phosgene)	0.0098	0.4	f	1.65x10 ⁻⁶	2.58x10 ⁻³	1.68x10 ⁻⁴	6.44x10 ⁻³	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	5.50x10 ⁻¹⁵	8.58x10 ⁻¹²	3.14x10 ⁻¹³	8.58x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	1.15x10 ⁻⁶	1.80×10^{-3}	2.89x10 ⁻⁶	2.35×10^{-6}	0	0 0
Health Risk						07/10	2.33710	v	U
Ш ^g						3.34x10 ⁻³	1.44x10 ⁻¹		
Cancer risk ^h						2.0 1/10	1.77810	0	0

Table M.3.4–43. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory— Ceramic Immobilization Facility for the Immobilized Disposition Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

	Degulated E	Typogura I imi	ts/Risk Factors	Emissions	Inventory	H	2	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]			_		,		0.54.10-3	0	0
Carbon dioxide	221	9,000	f	2.86×10^{-1}	8.59x10 ¹	1.29x10 ⁻³	9.54×10^{-3}	0	
Carbon monoxide	1.35	55	f	1.00x10 ⁻³	3.01×10^{-1}	7.41x10 ⁻⁴	5.47×10^{-3}	0	0
Chloride (HCI)	0.007	7	f	3.43x10 ⁻⁵	1.03x10 ⁻²	4.90x10 ⁻³	1.47×10^{-3}	0	0
Fluoride (HF)	0.21	2.49	f	4.29x10 ⁻⁷	1.29x10 ⁻⁴	2.04x10 ⁻⁶	5.17x10 ⁻⁵	0	0
Hydrocarbons	0.105	0.2	f	3.00x10 ⁻⁵	9.02×10^{-3}	2.86x10 ⁻⁴	4.51x10 ⁻²	0	0
(pyrene)	0.0245	1	f	4.29x10 ⁻⁷	1.29x10 ⁻⁴	1.75x10 ⁻⁵	1.29x10 ⁻⁴	0	0
Iron (salts) Magnesium (oxide fume)	0.368	15	f	4.29x10 ⁻⁵	1.29x10 ⁻²	1.17x10 ⁻⁴	8.59x10 ⁻⁴	0	0
Phosphates (phosphoric acid)	0.0245	1	f	2.14×10^{-5}	6.44x10 ⁻³	8.75x10 ⁻⁴	6.44x10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	4.29x10 ⁻⁶	1.29x10 ⁻³	4.38x10 ⁻⁴	3.22×10^{-3}	0	0
[Text deleted.]		0.01	f	1.43x10 ⁻¹⁴	4.29x10 ⁻¹²	8.17x10 ⁻¹³	4.29x10 ⁻¹⁰	0.	0
Silver	0.0175	0.01	f		4.23×10^{-4}	7.51x10 ⁻⁶	1.18x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	1	3.00x10 ⁻⁶	9.02810	7.51810	1.10/10	Ŭ	•
Health Risk						8.68x10 ⁻³	7.23x10 ⁻²		
HI ^g Cancer risk ^h								0	0

Table M.3.4–44. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Ceramic Immobilization Facility for the Immobilized Disposition Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

_	Regulated Exp	osure Limits/	Risk Factors	Emissions	Inventory	Н	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	3.00x10 ⁻¹	1.80x10 ²	1.36x10 ⁻³	2.00x10 ⁻²	0	0
Carbon monoxide	1.35	55	f	1.05×10^{-3}	6.30x10 ⁻¹	7.78x10 ⁻⁴	1.15x10 ⁻²	0	0
Chloride (HCI)	0.007	7	f	3.60x10 ⁻⁵	2.16x10 ⁻²	5.15x10 ⁻³	3.09x10 ⁻³	0	0
Fluoride (HF)	0.21	2.49	f	4.50x 10 ⁻⁷	2.70x10 ⁻⁴	2.14x10 ⁻⁶	1.09x10 ⁻⁴	0	0
Hydrocarbons (pyrene)	0.105	0.2	f	3.15x10 ⁻⁵	1.89x10 ⁻²	3.00x10 ⁻⁴	9.46x10 ⁻²	0	0
Iron (salts)	0.0245	1	f	4.50x 10 ⁻⁷	2.70x10 ⁻⁴	1.84x10 ⁻⁵	2.70x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	4.50x 10 ⁻⁵	2.70x10 ⁻²	1.22×10^{-4}	1.80x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	2.25×10^{-5}	1.35x10 ⁻²	9.19x10 ⁻⁴	1.35x10 ⁻²	0	0
Phosphonates (phosgene)	0.0098	0.4	f	4.50x10 ⁻⁶	2.70x10 ⁻³	4.59x10 ⁻⁴	6.75x10 ⁻³	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	1.50x 10 ⁻¹⁴	9.01x10 ⁻¹²	8.58x10 ⁻¹³	9.01x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	3.15x10 ⁻⁶	1.89x10 ⁻³	7.88x10 ⁻⁶	2.47x10 ⁻⁶	0	0
Health Risk									
HI ^g						9.11x10 ⁻³	1.52×10^{-1}		
Cancer risk ^h								0	0

Table M.3.4–45. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation— Ceramic Immobilization Facility for the Immobilized Disposition Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

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^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

	D		its/Risk Factors	Emissions	Inventory	H	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meter 8 Hours ^e
[Text deleted.]				2	2		1 70 10-2	0	0
Carbon dioxide	221	9,000	f	1.40×10^{-2}	1.53×10^2	6.33x10 ⁻⁵	1.70×10^{-2}		0
Carbon monoxide	1.35	55	f	4.89x10 ⁻⁵	5.35×10^{-1}	3.62×10^{-5}	9.72×10^{-3}	0	
Chloride (HCI)	0.007	7	f	1.68x10 ⁻⁶	1.83x10 ⁻²	2.40x10 ⁻⁴	2.62×10^{-3}	0	0
Fluoride (HF)	0.21	2.49	f	2.10x10 ⁻⁸	2.29x10 ⁻⁴	9.99x10 ⁻⁸	9.20x10 ⁻⁵	0	0
Hydrocarbons	0.105	0.2	f	1.47x10 ⁻⁶	1.60x10 ⁻²	1.40x10 ⁻⁵	8.02x10 ⁻²	0	0
(pyrene)			f	2.10x10 ⁻⁸	2.29x10 ⁻⁴	8.56x10 ⁻⁷	2.29x10 ⁻⁴	0	0
Iron (salts)	0.0245	1	f		2.29×10^{-2}	5.70x10 ⁻⁶	1.53×10^{-3}	0	0
Magnesium (oxide fume)	0.368	15		2.10x10 ⁻⁶			1.15x10 ⁻²	0	0
Phosphates (phosphoric acid)	0.0245	1	f	1.05×10^{-6}	1.15x10 ⁻²	4.28x10 ⁻⁵			
Phosphonates (phosgene)	0.0098	0.4	f	2.10x10 ⁻⁷	2.29x10 ⁻³	2.14x10 ⁻⁵	5.73x10 ⁻³	0	0
[Text deleted.]		0.01	f	6.99x10 ⁻¹⁶	7.64x10 ⁻¹²	3.99x10 ⁻¹⁴	7.64x10 ⁻¹⁰	0	0
Silver	0.0175	0.01	f	1.47×10^{-7}	1.60×10^{-3}	3.67x10 ⁻⁷	2.09x10 ⁻⁶	0	0
VOC (toluene)	0.4	766	·	1.4/X10	1.00x10	5.07 410	2.07.110		
Health Risk						4.24x10 ⁻⁴	1.29x 10 ⁻¹		
HI ^g Cancer risk ^h								0	0

Table M.3.4-46. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Ceramic Immobilization Facility for the Immobilized Disposition Alternative

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996e.

	Regulated E	xposure Lin	nits/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b (mg/m ³)	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	3.71x10 ⁻⁴	2.23x10 ⁻¹	2.75x10 ⁻⁴	4.05x10 ⁻³	0	0
Hydrocarbons (pyrene)	0.105	0.2	f	9.264x10 ⁻⁵	5.56x10 ⁻²	8.82x10 ⁻⁴	2.78x10 ⁻¹	0	ů 0
Health Risk HI ^g Cancer risk ^h						1.16x10 ⁻³	2.82x10 ⁻¹	0	•

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Source: LLNL 1996h.

<u> </u>	Populated F	vnosure Limit	s/Risk Factors	Emissions	Inventory	Н	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]							0 44 10-4	0	0
Carbon dioxide	221	9000	t	2.57×10^{-3}	8.5	1.16x10 ⁻⁵	9.44×10^{-4}	0	
Carbon monoxide	1.35	55	f	4.10x10 ⁻⁴	1.36	3.04×10^{-4}	2.47×10^{-2}	0	0
Chloride	0.42	17.1	f	4.62×10^{-6}	1.53x10 ⁻²	1.1x10 ⁻⁵	8.95x10 ⁻⁴	0	0
(sodium chloride) Fluoride (HF)	0.061	2.5	f	1.13x10 ⁻⁷	3.74x10 ⁻⁴	1.85x10 ⁻⁶	1.50x10 ⁻⁴	0	0
[Text deleted.] Iron (salts)	0.0245	1	f	5.64x10 ⁻⁸	1.87x10 ⁻⁴	2.30x10 ⁻⁶	1.87x10 ⁻⁴	0	0
Magnesium	0.368	15	f	6.16x10 ⁻⁶	2.04x10 ⁻²	1.67x10 ⁻⁵	1.36x10 ⁻³	0	0
(oxide fume) Phosphates	0.0245	1	f	2.82x10 ⁻⁶	9.35x10 ⁻³	1.15x10 ⁻⁴	9.35x10 ⁻³	0	0
(phosphoric acid) VOC (toluene)	0.4	766	f	8.21x10 ⁻⁵	2.72x10 ⁻¹	2.05x10 ⁻⁴	3.55x10 ⁻⁴	0	0
Health Risk						6.68x10 ⁻⁴	3.80x 10 ⁻²		
HI ^g Cancer risk ^h								0	0

Table M.3.4–48. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Vitrification Alternative

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

	Regulated Ex	posure Limi	ts/Risk Factors	Emissions	Inventory	Н	[Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]						······································			
Carbon dioxide	221	9000	f	3.82x10 ⁻⁴	4.41	1.73x10 ⁻⁶	4.90x10 ⁻⁴	0	0
Carbon monoxide	1.35	55	f	6.11x10 ⁻⁵	7.06x10 ⁻¹	4.53x10 ⁻⁵	1.28×10^{-2}	0	-
Chloride (sodium chloride)	0.42	17.1	f	6.88x10 ⁻⁷	7.94x10 ⁻³	1.64×10^{-6}	4.64×10^{-4}	0	0 0
Fluoride (HF) [Text deleted.]	0.061	2.5	f	1.68x10 ⁻⁸	1.94x10 ⁻⁴	2.76x10 ⁻⁷	7.76x10 ⁻⁵	0	0
Iron (salts)	0.0245	1	f	8.4x10 ⁻⁹	9.7x10 ⁻⁵	3.43x10 ⁻⁷	9.7x10 ⁻⁵		
Magnesium (oxide fume)	0.368	15	f	9.17x10 ⁻⁷	1.06×10^{-2}	2.49x10 ⁻⁶	9.7x10 ⁻⁵ 7.06x10 ⁻⁴	0 0	0 0
Phosphates (phosphoric acid)	0.0245	1	f	4.2×10^{-7}	4.85x10 ⁻³	1.72x10 ⁻⁵	4.85x10 ⁻³	0	0
VOC (toluene) Health Risk	0.4	766	f	1.22x10 ⁻⁵	1.41x10 ⁻¹	3.06x10 ⁻⁵	1.84x10 ⁻⁴	0	0
HI ^g						9.95x10 ⁻⁵	1.97x10 ⁻²		
Cancer risk ^h						J.JJXIU	1.9/X10 -	0	0

Table M.3.4-49. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site-Vitrification Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996c.

	Regulated H	Exposure Lir	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]				-					
Carbon dioxide	221	9,000	f	5.50x10 ⁻³	8.58	2.49x10 ⁻⁵	9.54x10 ⁻⁴	0	0
Carbon monoxide	1.35	55	f	8.80x10 ⁻⁴	1.37	6.52x10 ⁻⁴	2.50x 10 ⁻²	0	0
Chloride (sodium chloride)	0.42	17.1	f	9.90x10 ⁻⁶	1.55x10 ⁻²	2.36x10 ⁻⁵	9.04x10 ⁻⁴	0	0
Fluoride	0.061	2.5	f	2.42×10^{-7}	3.78x10 ⁻⁴	3.97x10 ⁻⁶	1.51x10 ⁻⁴	0	0
[Text deleted.]									
Iron (salts)	0.0245	1	f	1.21x10 ⁻⁷	1.89x10 ⁻⁴	4.94x10 ⁻⁶	1.89x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	1.32x10 ⁻⁵	2.06x10 ⁻²	3.59x10 ⁻⁵	1.37x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	6.05x10 ⁻⁶	9.44x10 ⁻³	2.47x10 ⁻⁴	9.44x10 ⁻³	0	0
VOC (toluene)	0.4	766	f	1.76x10 ⁻⁴	2.75x10 ⁻¹	4.40x10 ⁻⁴	3.59x10 ⁻⁴	0	0
Health Risk HI ^g						1.43x10 ⁻³	3.83x10 ⁻²		
Cancer risk ^h								0	0

Table M.3.4–50. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—Vitrification Alternative

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* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

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^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds. Source: LLNL 1996c.

	Regulated I	Exposure Lir	nits/Risk Factors	Emission	s Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]						···			······
Carbon dioxide	221	9,000	f	1.43x10 ⁻²	4.29	6.47x10 ⁻⁵	4 77-10-4		
Carbon monoxide	1.35	55	f	2.29x10 ⁻³	6.87x10 ⁻¹		4.77x10 ⁻⁴	0	0
Chloride	0.42	17.1	f	2.57×10^{-5}	7.73×10^{-3}	1.69×10^{-3}	1.25x10 ⁻²	0	0
(sodium chloride)				2.37810	7.73X10 ⁻⁵	6.13x10 ⁻⁵	4.52×10^{-4}	0	0
Fluoride	0.061	2.5	f	6.29x10 ⁻⁷	1.89x10 ⁻⁴	1.02.10-5	5 5 6 5		
[Text deleted.]				0.29810	1.09710	1.03x10 ⁻⁵	7.56x10 ⁻⁵	0	0
fron (salts)	0.0245	1	f	3.15x10 ⁻⁶	9.45x10 ⁻⁵	1.00 10-5	0.10.105		
Magnesium	0.368	15	f	3.43x10 ⁻⁵		1.28x10 ⁻⁵	9.45x10 ⁻⁵	0	0
(oxide fume)	2.000	1.5		3.43X10°	1.03×10^{-2}	9.32x10 ⁻⁵	6.87x10 ⁻⁴	0	0
Phosphates (phosphoric acid)	0.0245	1	f	1.57x10 ⁻⁵	4.72×10^{-3}	6.42x10 ⁻⁴	4.72x10 ⁻³	0	0
VOC (toluene)	0.4	766	f	4.58x10 ⁻⁴	1.27.10-1	1 1 4 4 9 3			
Health Risk HI ^g				4.JOX IV	1.37x10 ⁻¹	1.14x10 ⁻³	1.79x10 ⁻⁴	0	0
Cancer risk ^h						3.72x10 ⁻³	1.92x10 ⁻²		
See Table M 2 2 1 1 front								0	0

Table M.3.4-51. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant-Vitrification Alternative

See Table M.3.3.1-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HQs.

h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996c.

	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	1.50x10 ⁻²	9.01	6.79x10 ⁻⁵	1.0x10 ⁻³	0	0
Carbon monoxide	1.35	55	f	2.40×10^{-3}	1.44	1.78x10 ⁻³	2.62x10 ⁻²	0	0
Chloride (sodium chloride)	0.42	17.1	f	2.70x10 ⁻⁵	1.62×10^{-2}	6.43x10 ⁻⁵	9.48x10 ⁻⁴	0	0
Fluoride	0.061	2.5	f	6.60x10 ⁻⁷	3.96x10 ⁻⁴	1.08x10 ⁻⁵	1.59x10 ⁻⁴	0	0
[Text deleted.]									
Iron (salts)	0.0245	1	f	3.30x10 ⁻⁷	1.98x10 ⁻⁴	1.35x10 ⁻⁵	1.98x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	3.60x10 ⁻⁵	2.16x10 ⁻²	9.79x10 ⁻⁵	1.44x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	1.65x10 ⁻⁵	9.91x10 ⁻³	6.74x10 ⁻⁴	9.91x10 ⁻³	0	0
VOC (toluene)	0.4	766	f	4.80x10 ⁻⁴	2.88x10 ⁻¹	1.20×10^{-3}	3.76x10 ⁻⁴	0	0
Health Risk HI ^g						3.91x10 ⁻³	4.02x10 ⁻²		
Cancer risk ^h -			-					0	0

Table M.3.4–52. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—Vitrification Alternative

^a See Table M.3.3.1-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996c.

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	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	6.99x10 ⁻⁴	7.64	3.16x10 ⁻⁶	8.48x10 ⁻⁴	0	0
Carbon monoxide	1.35	55	f	1.12x10 ⁻⁴	1.22	8.28x10 ⁻⁵	2.22x10 ⁻²	0	0 ·
Chloride (sodium chloride)	0.42	17.1	f	1.26x10 ⁻⁶	1.37x10 ⁻²	3.00x10 ⁻⁶	8.04x10 ⁻⁴	0	0
Fluoride	0.061	2.5	f	3.08x10 ⁻⁸	3.36x10 ⁻⁴	5.04x10 ⁻⁷	1.34x10 ⁻⁴	0	0
[Text deleted.]								Ū	0
Iron (salts)	0.0245	1	f	1.54x10 ⁻⁸	1.68x10 ⁻⁴	6.28x10 ⁻⁷	1.68x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	1.68x10 ⁻⁶	1.83x10 ⁻²	4.56x10 ⁻⁶	1.22×10^{-3}	0	Ő
Phosphates (phosphoric acid)	0.0245	1	f	7.69x 10 ⁻⁷	8.40x10 ⁻³	3.14x10 ⁻⁵	8.40x10 ⁻³	0	0
VOC (toluene)	0.4	766	f	2.24x10 ⁻⁵	2.44×10^{-1}	5.59x10 ⁻⁵	3.19x10 ⁻⁴	0	0
Health Risk						0.07/10	5.15410	0	U
НI ^g						1.82x10 ⁻⁴	3.41x10 ⁻²		
Cancer risk ^h							5.71410	0	0

Table M.3.4-53. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Vitrification Alternative

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

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	Regulated E	Exposure Lir	nits/Risk Factors	Emissions	Inventory	Н	Q	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]			······						
Carbon dioxide	221	9,000	f	4.10×10^{-2}	1.36x10 ²	1.86x10 ⁻⁴	1.51x10 ⁻²	0	0
Carbon monoxide	1.35	55	f	1.41x10 ⁻³	4.67	1.05x10 ⁻³	8.50x10 ⁻²	0	0
Chloride (HCl)	0.007	7	f	4.62x10 ⁻⁶	1.53x10 ⁻²	6.60x10 ⁻⁴	2.19x10 ⁻³	0	0
Fluoride (HF)	0.21	2.49	f	6.16x10 ⁻⁸	2.04x10 ⁻⁴	2.93x10 ⁻⁷	8.19x10 ⁻⁵	0	0
Iron (salts)	0.0245	1	f	5.64x10 ⁻⁸	1.87x10 ⁻⁴	2.30x10 ⁻⁶	1.87x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	6.16x10 ⁻⁶	2.04x10 ⁻²	1.67x10 ⁻⁵	1.36x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	2.82x10 ⁻⁶	9.35x10 ⁻³	1.15x10 ⁻⁴	9.35x10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	5.64x10 ⁻⁶	1.87x10 ⁻²	5.76x10 ⁻⁴	4.67x10 ⁻²	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	2.57x10 ⁻¹⁵	8.50x10 ⁻¹²	1.47x10 ⁻¹³	8.50x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	4.62x10 ⁻⁷	1.53x10 ⁻³	1.15x10 ⁻⁶	2.00x10 ⁻⁶	0	0
Health Risk HI ^g						2.60x10 ⁻³	1.60x10 ⁻¹	,	
Cancer risk ^h								0	0

Table M.3.4–54. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Ceramic Immobilization Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

Chemical	Regulated I	Exposure Lir	nits/Risk Factors	Emissions	s Inventory	HQ		Cancer Risk	
	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary Annual MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]							· · · · · · · · · · · · · · · · · · ·		
Carbon dioxide	221	9,000	f	6.11x10 ⁻³	7.06x10 ¹	2.77x10 ⁻⁵	7.84x10 ⁻³	0	0
Carbon monoxide	1.35	55	f	2.10x10 ⁻⁴	2.43	1.56×10^{-4}	4.41×10^{-2}	0	0
Chloride (HCl)	0.007	7	f	6.88x10 ⁻⁷	7.94x10 ⁻³	9.82×10^{-5}	1.13×10^{-3}	0	0
Fluoride (HF)	0.21	2.49	f	9.17x10 ⁻⁹	1.06x10 ⁻⁴	4.37×10^{-8}	4.25x10 ⁻⁵	0 0	0
Iron (salts)	0.0245	1	f	8.40x10 ⁻⁹	9.70x10 ⁻⁵	3.43x10 ⁻⁷	9.70x10 ⁻⁵	0	0
Magnesium (oxide fume)	0.368	15	f	9.17x10 ⁻⁷	1.06x10 ⁻²	2.49×10^{-6}	7.06x10 ⁻⁴	0	0
Phosphates (phosphoric acid)	0.0245	1	f	4.20x10 ⁻⁷	4.85x10 ⁻³	1.72x10 ⁻⁵	4.85x10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	8.40x10 ⁻⁷	9.70x10 ⁻³	8.58x10 ⁻⁵	2.43x10 ⁻²	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	3.82x10 ⁻¹⁶	4.41x10 ⁻¹²	2.18x10 ⁻¹⁴	4.41x10 ⁻¹⁰	0	٥
VOC (toluene)	0.4	766	f	6.88x10 ⁻⁸	7.94x10 ⁻⁴	1.72×10^{-7}	1.04x10 ⁻⁶	0	0 0
Health Risk						1.7 2710	1.04410	U	U
HI ^g						3.87x10 ⁻⁴	8.30x10 ⁻²		
Cancer risk ^h						2.07410	0.50710	0	0

Table M.3.4-55. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site—Ceramic Immobilization Alternative

* See Table M.3.3.1-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(Emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

	Regulated H	Exposure Lir	nits/Risk Factors	Emission	s Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									·····
Carbon dioxide	221	9,000	f	8.80x10 ⁻²	1.37×10^2	3.98x10 ⁻²	1.53x10 ⁻³	0	0
Carbon monoxide	1.35	55	f	3.02x10 ⁻³	4.72	2.24x10 ⁻²	8.58x10 ⁻²	0	0
Chloride (HCl)	0.007	7	f	9.90x10 ⁻⁶	1.55x10 ⁻²	1.41x10 ⁻³	2.21x10 ⁻³	0	0
Fluoride (HF)	0.21	2.49	f	1.32x10 ⁻⁷	2.06x10 ⁻⁴	6.28x10 ⁻⁵	8.27x10 ⁻⁵	0	0
Iron (salts)	0.0245	1	f	1.21x10 ⁻⁷	1.89x10 ⁻⁴	4.94x10 ⁻⁴	1.89x10 ⁻⁵	0	0
Magnesium (oxide fume)	0.368	15	f	1.32x10 ⁻⁵	2.06x10 ⁻²	3.59x10 ⁻⁶	1.37x10 ⁻⁴	0	ů 0
Phosphates (phosphoric acid)	0.0245	1	f	6.05x10 ⁻⁶	9.44x10 ⁻³	2.47x10 ⁻³	9.44x10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	1.21x10 ⁻⁵	1.89x10 ⁻²	1.23x10 ⁻²	4.72x10 ⁻²	0	0
[Text deleted.]								Ŭ	U
Silver	0.0175	0.01	f	5.50x10 ⁻¹⁵	8.58x10 ⁻¹²	3.14x10 ⁻¹⁰	8.58x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	9.90x10 ⁻⁷	1.55x10 ⁻³	2.47x10 ⁻⁶	2.02×10^{-6}	0	0
Health Risk								-	Ū
HI ^g						5.58x10 ⁻³	1.62×10^{-1}		
Cancer risk ^h								0	0

Table M.3.4–56.	Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—
	Ceramic Immobilization Alternative

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

	Regulated E	Exposure Lin	nits/Risk Factors	Emissions	s Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	2.29x10 ⁻¹	6.87x10 ⁻¹	1.04x10 ⁻²	7.64x10 ⁻³	0	0
Carbon monoxide	1.35	55	f	7.86x10 ⁻³	2.36	5.83x10 ⁻²	4.29x 10 ⁻²	0	0
Chloride (HCl)	0.007	7	f	2.57x10 ⁻⁵	7.73x 10 ⁻³	3.68x10 ⁻³	1.10×10^{-3}	0	0
Fluoride (HF)	0.21	2.49	f	3.43x10 ⁻⁷	1.03x10 ⁻⁴	1.63x10 ⁻⁶	4.14x10 ⁻⁵	0	ů 0
Iron (salts)	0.0245	1	f	3.15x10 ⁻⁷	9.45x10 ⁻⁵	1.28x10 ⁻⁴	9.45x10 ⁻⁵	0	ů 0
Magnesium (oxide fume)	0.368	15	f	3.43x10 ⁻⁵	1.03×10^{-2}	9.32x10 ⁻⁶	6.87x10 ⁻⁴	0	0
Phosphates (phosphoric acid)	0.0245	1	f	1.57x10 ⁻⁵	4.72x10 ⁻³	6.42x10 ⁻³	4.72×10^{-3}	0	0
Phosphonates (phosgene)	0.0098	0.4	f	3.15x10 ⁻⁵	9.45x10 ⁻³	3.21x10 ⁻²	2.36x10 ⁻²	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	1.43x10 ⁻¹⁴	4.29x10 ⁻¹²	8.17x10 ⁻¹⁰	4.29x10 ⁻¹⁰	0	0
VOC (toluene)	0.4	766	f	2.57x10 ⁻⁶	7.73x10 ⁻⁴	6.43x10 ⁻⁶	1.01x10 ⁻⁶	0	0
Health Risk HI ^g						1.45x10 ⁻²	8.09x10 ⁻²	U	0
Cancer risk ^h								0	0

Table M.3.4–57. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Ceramic Immobilization Alternative

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

- <u> </u>	Regulated I	Exposure Lin	nits/Risk Factors	Emissions	Inventory	Н	Q	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
[Text deleted.]					- 1			0	
Carbon dioxide	221	9,000	t	2.40×10^{-1}	1.44×10^2	1.09×10^{-3}	1.60×10^{-2}	0	0
Carbon monoxide	1.35	55	f	8.25x10 ⁻³	4.95	6.11x10 ⁻³	9.01x10 ⁻²	0	0
Chloride (HCl)	0.007	7	f	2.70x10 ⁻⁵	1.62x10 ⁻²	3.86x10 ⁻³	2.32×10^{-3}	0	0
Fluoride (HF)	0.21	2.49	f	3.60x10 ⁻⁷	2.16x10 ⁻⁴	1.72x10 ⁻⁶	8.68x10 ⁻⁵	0	0
Iron (salts)	0.0245	1	f	3.30x10 ⁻⁷	1.98x10 ⁻⁴	1.35x10 ⁻⁵	1.98x10 ⁻⁴	0	0
Magnesium (oxide fume)	0.368	15	f	3.60x10 ⁻⁵	2.16x10 ⁻²	9.79x10 ⁻⁵	1.44x10 ⁻³	0	0
Phosphates (phosphoric acid)	0.0245	1	f	1.65x10 ⁻⁵	9.91x10 ⁻³	6.74x10 ⁻⁴	9.91x10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	3.30x10 ⁻⁵	1.98x10 ⁻²	3.37x10 ⁻³	4.95x10 ⁻²	0	0
[Text deleted.]			r.	14	. 12		0.01.10-10	0	0
Silver	0.0175	0.01	t	1.50x10 ⁻¹⁴	9.01x10 ⁻¹²	8.58x10 ⁻¹³	9.01x10 ⁻¹⁰	0	
VOC (toluene)	0.4	766	f	2.70x10 ⁻⁶	1.62×10^{-3}	6.75x10 ⁻⁶	2.12×10^{-6}	0	0
Health Risk						2			

T-11- M 2 / 58	Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—Ceramic Immobilization Alternative
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^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

1.70x10⁻¹

0

0

 1.52×10^{-2}

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

Шg

Cancer risk^h

	Regulated I	Exposure Li	mits/Risk Factors	Emission	s Inventory	 F	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL* (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^{a,b}	Worker 100 Meters 8 Hours ^c	Boundary MEI ^{a,d}	Worker 100 Meter 8 Hours ^e
[Text deleted.]									
Carbon dioxide	221	9,000	f	1.12x10 ⁻²	1.22×10^2	5.06x10 ⁻⁵	1.36x10 ⁻²	0	0
Carbon monoxide	1.35	55	f	3.84x10 ⁻⁴	4.20	2.85x10 ⁻⁴	7.64x10 ⁻²	0	
Chloride (HCl)	0.007	7	f	1.26x10 ⁻⁶	1.37x10 ⁻²	1.80x10 ⁻⁴	1.96x10 ⁻³	0	0
Fluoride (HF)	0.21	2.49	f	1.68x10 ⁻⁸	1.83x10 ⁻⁴	7.99x10 ⁻⁸	7.36x10 ⁻⁵		0
Iron (salts)	0.0245	1	f	1.54x10 ⁻⁸	1.68x10 ⁻⁴	6.28x10 ⁻⁷	1.68×10^{-4}	0	0
Magnesium (oxide fume)	0.368	15	f	1.68x10 ⁻⁶	1.83x10 ⁻²	4.56x10 ⁻⁶	1.22×10^{-3}	0 0	0 0
Phosphates (phosphoric acid)	0.0245	1	f	7.69x10 ⁻⁷	8.40x10 ⁻³	3.14x10 ⁻⁵	8.40x 10 ⁻³	0	0
Phosphonates (phosgene)	0.0098	0.4	f	1.54x10 ⁻⁶	1.68x10 ⁻²	1.57x10 ⁻⁴	4.20x 10 ⁻²	0	0
[Text deleted.]									
Silver	0.0175	0.01	f	6.99x10 ⁻¹⁶	7.64x10 ⁻¹²	2.00.10-14	R () () ()		
VOC (toluene)	0.4	766	f	1.26×10^{-7}		3.99x10 ⁻¹⁴	7.64x10 ⁻¹⁰	0	0
Health Risk	5.1	,		1.20710	1.37x10 ⁻³	3.15x10 ⁻⁷	1.79x10 ⁻⁶	0	0
HIg									
Cancer risk ^h						7.09x10 ⁻⁴	1.44x10 ⁻¹		
								0	0

Table M.3.4–59. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Ceramic Immobilization Alternative

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HOs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996d.

	Domilated F	vposure Lin	nits/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Chemical			(ing/kg/ddy)	5.06x10 ⁻⁷	7.90x10 ⁻⁴	3.75x10 ⁻⁷	1.44x10 ⁻⁵	0	0
Carbon monoxide	1.35	55	f		8.58x10 ⁻⁴	1.37x10 ⁻⁶	1.12x10 ⁻⁶	0	0
VOCs (toluene)	0.4	766	I	5.50	8.38210	1.57×10	1.12×10	-	
Health Risk						1.75x10 ⁻⁶	1.55x10 ⁻⁵		
HI ^g						1.75810	1.55810	0	0
Cancer risk ^h									

Table M.3.4–60. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—Electrometallurgical Treatment Alternative

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LLNL 1996b.

	Regulated E	Regulated Exposure Limits/Risk Factors			Emissions Inventory		НQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	
Cleaning solvent (butyl lactate)	0.74	30	f	1.41×10^{-5}	4.67x10 ⁻²	1.91x10 ⁻⁵	1.56x10 ⁻³	0	0	
[Text deleted.]										
VOC (toluene) Health Risk	0.40	766	f	5.64x10 ⁻⁶	1.87x10 ⁻²	1.41x10 ⁻⁵	2.44x10 ⁻⁵	0	0	
HI ^g Cancer risk ^h						3.32x10 ⁻⁵	1.58x10 ⁻³	0	0	

Table M.3.4–61. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Mixed Oxide Fuel Fabrication Facility

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

	Regulated Exposure Limits/Risk Factors		Emissions	Inventory	HQ		Cancer Risk		
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	2.1x10 ⁻⁶	2.43x10 ⁻²	2.84x10 ⁻⁶	8.08x10 ⁻⁴	0	0
[Text deleted.] VOC (toluene)	0.40	766	f	8.40x10 ⁻⁷	9.70x10 ⁻³	2.10x10 ⁻⁶	1.27x 10 ⁻⁵	0	0
Health Risk HI ^g Cancer risk ^h						4.94x10 ⁻⁶	8.21x10 ⁻⁴	0	0

Table M.3.4–62. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site— Mixed Oxide Fuel Fabrication Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HO for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996b.

	Regulated	Regulated Exposure Limits/Risk Factors			Emissions Inventory		IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	ſ	3.02x10 ⁻⁵	4.72×10^{-2}	4.09x10 ⁻⁵	1.57x10 ⁻³	0	0
[Text deleted.]									
VOC (toluene) Health Risk	0.40	766	f	1.21x10 ⁻⁵	1.89x10 ⁻²	3.02x10 ⁻⁵	2.47x10 ⁻⁵	0	0
HI ^g						7.11x10 ⁻⁵	1.60x10 ⁻³		
Cancer risk ^h								0	0

Table M.3.4-63. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory-Mixed Oxide Fuel Fabrication Facility

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HOs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996b.

	Regulated E	Exposure Lim	its/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day)	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	Ŧ	7.86x10 ⁻⁵	2.36x10 ⁻²	1.06x10 ⁻⁴	7.87x10 ⁻⁴	0	0
[Text deleted.] VOC (toluene)	0.40	766	f	3.15x10 ⁻⁵	9.45x10 ⁻³	7.86x10 ⁻⁵	1.23x 10 ⁻⁵	0	0
Health Risk HI ^g Cancer risk ^h						1.85x10 ⁻⁴	8.00x 10 ⁻⁴	0	0

Table M.3.4–64. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Mixed Oxide Fuel Fabrication Facility

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor (SF)).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Source: LANL 1996b.

Section.

	Regulated H	Regulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	
Cleaning solvent (butyl latate) [Text deleted.]	0.74	30	f	8.25x10 ⁻⁵	4.95x10 ⁻²	1.12x10 ⁻⁴	1.65x10 ⁻³	0	0	
VOC (toluene) Health Risk	0.40	766	f	3.30x10 ⁻⁵	1.98x10 ⁻²	8.25x10 ⁻⁵	2.59x10 ⁻⁵	0	0	
HI ^g Cancer risk ^h						1.94x10 ⁻⁴	1.68x10 ⁻³			
								0	0	

Table M.3.4-65. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—Mixed Oxide Fuel Fabrication Facility

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions Concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

• ;

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

	Regulated I	Exposure Lin	nits/Risk Factors	Emissions	Inventory	H	IQ	Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	3.84x10 ⁻⁶	4.20x10 ⁻²	5.20x10 ⁻⁶	1.40x10 ⁻³	0	0
[Text deleted.] VOC (toluene)	0.40	766	f	1.54x10 ⁻⁶	1.68x10 ⁻²	3.84x10 ⁻⁶	2.19x10 ⁻⁵	0	0
Health Risk HI ^g						9.04x10 ⁻⁶	1.42x10 ⁻³	-	0
Cancer risk ^h								0	0

Table M.3.4–66. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Mixed Oxide Fuel Fabrication Facility

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^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

• Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds.

Table M.3.4-67. Risk Assessments From Exposure to Hazardous Chemicals at Generic Site—Mixed Oxide Fuel Fabrication Facility

	Regulate	ed Exposure	e Limits/Risk						
	Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Cleaning solvent (butyl lactate)	0.74	30	f	8.25x10 ⁻⁵	4.95x10 ⁻²	1.12x10 ⁻⁴	1.65x10 ⁻³	0	0
[Text deleted.]									
VOC (toluene)	0.40	766	f	3.30x10 ⁻⁵	1.98x10 ⁻²	8.25x10 ⁻⁵	2.59x10 ⁻⁵	0	0
Health Risk									
НI ^g						1.94x10 ⁻⁴	1.68x10 ⁻³		
Cancer risk ^h								0	0

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: VOC=volatile organic compounds. Incremental HIs and cancer risks calculated with ORR dispersion factors. Total HIs and cancer risks calculated with 0 No Action. Source: LANL 1996b.

Regulate		ulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	
Carbon monoxide	1.35	55	f	2.57x10 ⁻⁷	8.50x10 ⁻⁴	1.90x10 ⁻⁷	1.55x10 ⁻⁵	0	0	
Health Risk HI ^g Cancer risk ^h						1.90x10 ⁻⁷	1.55x10 ⁻⁵	0	0	

Table M.3.4–68. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Large Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: HIs and cancer risks are based on air emissions of the criteria pollutant, carbon monoxide. Other pollutants, potential hazardous chemicals, are water releases. Source: LLNL 1996g.

	Regulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	3.82x10 ⁻⁸	4.41x10 ⁻⁴	2.83x10 ⁻⁸	8.02x10 ⁻⁶	0	0
Health Risk HI ^g Cancer risk ^h						2.83x10 ⁻⁸	8.02x10 ⁻⁶	0	0

Table M.3.4–69. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site—Large Evolutionary Light Water Reactor

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: HIs and cancer risks are based on air emissions of the criteria pollutant, carbon monoxide. Other pollutants, potential hazardous chemicals, are water releases. Source: LLNL 1996g.

	Regulated Exposure Limits/Risk Factors			Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	5.50x10 ⁻⁷	8.58x10 ⁻⁴	4.07x10 ⁻⁷	1.56x10 ⁻⁵	0	0
Health Risk HI ^g Cancer risk ^h						4.07x10 ⁻⁷	1.56x10 ⁻⁵	0	0

Table M.3.4–70. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory— Large Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-PEL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: HIs and cancer risks are based on air emissions of the criteria pollutant, carbon monoxide. Other pollutants, potential hazardous chemicals, are water releases. Source: LLNL 1996g.

	Regulated Exposure Limits/Risk Factors		Emission	Emissions Inventory		НQ		er Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide Health Risk	1.35	55	f	1.43x10 ⁻⁶	4.29x10 ⁻⁴	1.06x10 ⁻⁶	7.81x10 ⁻⁶	0	0
HI ^g Cancer risk ^h						1.06x10 ⁻⁶	7.81x10 ⁻⁶		
								0	0

Table M.3.4–71. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Large Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^B HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Regulated Exposure Limits/Risk Factors		Emissions	Emissions Inventory		HQ		Cancer Risk		
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.50x10 ⁻⁶	9.01x10 ⁻⁴	1.11x10 ⁻⁶	1.64x10 ⁻⁵	0	0
Health Risk HI ^g						1.11x10 ⁻⁶	1.64x10 ⁻⁵		
Cancer risk ^h								0	0

Table M.3.4-72. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—Large Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

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	Regulated H	Exposure Lin	nits/Risk Factors	Emission	s Inventory	E	IQ	Cance	er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	6.99x10 ⁻⁸	7.64x10 ⁻⁴	5.18x10 ⁻⁸	1.39x10 ⁻⁵	0	0
Health Risk HI ^g						5.18x10 ⁻⁸	1.39x10 ⁻⁵		
Cancer risk ^h								0	0

Table M.3.4-73. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site—Large Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

	Regulated H	Regulated Exposure Limits/Risk Factors		Emissions	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e	
Carbon monoxide	1.35	55	f	2.57x10 ⁻⁷	8.50x10 ⁻⁴	1.90x10 ⁻⁷	1.55x10 ⁻⁵	0	0	
Health Risk HI ^g Cancer risk ^h						1.90x10 ⁻⁷	1.55x10 ⁻⁵	0	0	

Table M.3.4–74. Risk Assessments From Exposure to Hazardous Chemicals at Hanford Site—Small Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hrs)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Regulated Exposure Limits/Risk Factors			Emission	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide Health Risk	1.35	55	f	3.82x10 ⁻⁸	4.41x10 ⁻⁴	2.83x10 ⁻⁸	8.02x10 ⁻⁶	0	0
HI ^g Cancer risk ^h						2.83x10 ⁻⁸	8.02x10 ⁻⁶		
	······							0	0

Table M.3.4–75. Risk Assessments From Exposure to Hazardous Chemicals at Nevada Test Site—Small Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^c Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen. ^g HI=sum of individual HOs.

^h Total cancer risk=sum of individual cancer risks.

	Regulated Exposure Limits/Risk Factors		Emission	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	5.50x10 ⁻⁷	8.58x10 ⁻⁴	4.07x10 ⁻⁷	1.56x10 ⁻⁵	0	0
Health Risk						4.07x10 ⁻⁷	1.56x10 ⁻⁵		
Cancer risk ^h								0	0

Table M.3.4–76. Risk Assessments From Exposure to Hazardous Chemicals at Idaho National Engineering Laboratory—Small Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HO for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

⁸ HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Note: HIs and cancer risks are based on air emissions of the criteria pollutant, carbon monoxide. Other pollutants, potential hazardous chemicals, are water releases. Source: LLNL 1996g.

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Regulated Exposu		Exposure Lin	nits/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.43x10 ⁻⁶	4.29x10 ⁻⁴	1.06x10 ⁻⁶	7.81x10 ⁻⁶	0	0
Health Risk								-	U U
HI ^g						1.06x10 ⁻⁶	7.81x10 ⁻⁶		
Cancer risk ^h								0	0

Table M.3.4–77. Risk Assessments From Exposure to Hazardous Chemicals at Pantex Plant—Small Evolutionary Light Water Reactor

* See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

	Regulated F	Exposure Lir	nits/Risk Factors	Emission	Emissions Inventory		HQ		er Risk
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary ME	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide	1.35	55	f	1.50x10 ⁻⁶	9.01x10 ⁻⁴	1.11x10 ⁻⁶	1.64x10 ⁻⁵	0	0
Health Risk HI ^g						1.11x10 ⁻⁶	1.64x10 ⁻⁵	0	0
Cancer risk ^h								0	0

Table M.3.4–78. Risk Assessments From Exposure to Hazardous Chemicals at Oak Ridge Reservation—Small Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HO for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

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^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

8 HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

Regulated Expos		Exposure Lir	mits/Risk Factors	Emissions Inventory		HQ		Cancer Risk	
Chemical	RfC (mg/m ³)	PEL ^a (mg/m ³)	Slope Factor (mg/kg/day) ⁻¹	Boundary MEI (mg/m ³)	Worker 100 Meters 8 Hours (mg/m ³)	Boundary MEI ^b	Worker 100 Meters 8 Hours ^c	Boundary MEI ^d	Worker 100 Meters 8 Hours ^e
Carbon monoxide Health Risk	1.35	55	f	6.99x10 ⁻⁸	7.64x10 ⁻⁴	5.18x10 ⁻⁸	1.39x10 ⁻⁵	0	0
HI ^g Cancer risk ^h						5.18x10 ⁻⁸	1.39x10 ⁻⁵	_	
		····						0	0

Table M.3.4-79. Risk Assessments From Exposure to Hazardous Chemicals at Savannah River Site-Small Evolutionary Light Water Reactor

^a See Table M.3.3-1 for the OSHA-PEL, NIOSH-REL, ACGIH-TLV, and other exposure limit values.

^b HQ for MEI=boundary annual emissions/RfC.

^c HQ for workers=100-m, 8-hr emissions/PEL.

^d Cancer risk for MEI=(emissions concentrations)x(0.286 [converts concentration to dose])x(slope factor [SF]).

^e Cancer risk for workers=(emissions for 8-hr)x(0.237 [fraction of year exposed])x(0.571 [fraction of lifetime working])x(0.286 [converts concentration to dose])x(SF).

^f There is no slope factor when the data show the chemical is not a carcinogen, or alternatively when there are either no data or insufficient data to suggest the chemical is a carcinogen.

^g HI=sum of individual HQs.

^h Total cancer risk=sum of individual cancer risks.

M.4 HUMAN HEALTH STUDIES: EPIDEMIOLOGY

Various epidemiologic studies have been conducted at some of the sites evaluated in this PEIS because of the concern for potential health effects (that is, premature fatalities) associated with the manufacture and testing of nuclear weapons. These studies focus on the DOE workforce and residents of communities, surrounding DOE sites.

M.4.1 BACKGROUND

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The health effects associated with ionizing radiation exposure were first published about 60 years ago. Studies published in the 1930s first documented cancer among painters who used radium to paint watch dials back in 1910-1920. Radiation therapy for disease was used since the 1930s, and studies have shown that the risk of cancer was related to the amounts of radiation received. Nuclear weapons research and manufacture and consequent exposure to radiation occurred beginning in the late 1930's. Exposure to radionuclides has changed over time with higher levels occurring in the early days of research and production. Numerous epidemiologic studies have been conducted among workers who manufactured and tested nuclear weapons due to the concern with potential adverse health effects. More recently, concerns about radiologic contaminants off-site have resulted in health studies among communities that surround DOE facilities. The following section briefly gives an overview of epidemiology followed by a review of epidemiologic studies of sites evaluated in the PEIS.

Epidemiology is the study of the distribution and determinants of disease in human populations. The distribution of disease is considered in relation to time, place, and person. Relevant population characteristics should include the age, race and sex distribution of a population, as well as other characteristics related to health, such as social characteristics (for example, income and education), occupation, susceptibility to disease, and exposure to specific agents. Determinants of disease include the causes of disease, as well as factors that influence the risk of disease.

M.4.1.1 Study Designs

Ecologic Studies. Ecologic studies compare the frequency of a disease in groups of people in conjunction with simple descriptive studies of geographical information in, an attempt to determine how health events among populations vary with levels of exposure. These groups may be identified as the residents of a neighborhood, a city, or a county where demographic information and disease or mortality data are available. Exposure to specific agents may be defined in terms of residential location or proximity to a particular area, such as distance from a waste disposal site. An example of an ecologic study is a comparison of the rate of heart disease among community residents by drinking water quality.

The major disadvantage of ecologic studies is that the measure of exposure is based on the average level of exposure in the community, when we are really interested in the individual's exposure. Ecologic studies do not take into account other factors, such as age and race that may also be related to disease. These types of studies may lead to incorrect conclusions, an "ecologic fallacy." For the above example, it would be incorrect to assume that the level of water hardness influences the risk of getting heart disease. Despite the obvious problems with ecologic studies, they can be a useful first step in identifying possible associations between risk of disease and environmental exposures. However, because of their potential for bias they should never be considered more than an initial step in investigation of disease causation.

Cohort Studies. The cohort study design is a type of epidemiologic study frequently used to examine occupational exposures within a defined workforce. A cohort study requires a defined population that can be classified as being exposed or not exposed to an agent of interest, such as radiation or chemicals that influence the probability of occurrence of a given disease. Characterization of the exposure may be qualitative (for example, high, low, or no exposure) or very quantitative (for example, radiation measured in Sieverts [Sv] and

chemicals in parts per million). Surrogates for exposure, such as job titles, are frequently used in the absence of quantitative exposure data.

Individuals enumerated in the study population are followed for a period of time to observe who died. In general overall rates of death and cause-specific rates of death have been assessed for workers at the PEIS sites. Death rates for the exposed worker population are compared with death rates of workers who did not have the exposure (internal comparison), or compared with expected death rates based on the U.S. population or State death rates (external comparison). If the rates of death differ from what is expected, an association is said to exist between the disease and exposure. In cohorts where the exposure has not been characterized, excess mortality can be identified, but these deaths cannot be attributed to a specific exposure, and additional studies may be warranted. More recent studies have looked at other disease endpoints, such as overall and cause-specific cancer incidence (newly diagnosed) rates.

Most cohort studies at PEIS sites have been historical cohort studies, that is, the exposure occurred some time in the distant past. These studies rely on past records to document exposure. This type of study can be problematic if exposure records are incomplete or were destroyed. Cohort studies require extremely large populations that have been followed for many (20-30) years. They are generally difficult to conduct and are very expensive. These studies are not well suited to studying diseases that are rare. Cohort studies do, however, provide a direct estimate of the risk of death from a specific disease, and allow an investigator to look at many disease endpoints.

Case-Control Studies. The case-control study design starts with the identification of persons with the disease of interest (case) and a suitable comparison (control population of persons without the disease). Controls must be persons who are at risk for the disease and are representative of the population that generated the cases. The selection of an appropriate control group is often quite problematic. Cases and controls are then compared with respect to the proportion of individuals exposed to the agent of interest. Case-control studies require fewer persons than cohort studies, and therefore, are usually less costly and less time consuming, but are limited to the study of one disease (or cause of death). These types of studies are well suited for the study of rare diseases and are generally used to examine the relationship between a specific disease and exposure.

M.4.1.2 Definitions

Unfamiliar terms frequently used in epidemiologic studies, including those used in this document, are defined below.

Age, gender, and cigarette smoking are the principal determinants of mortality. Standardization is a statistical method used to control for the effects of age, gender, or other characteristics so that death may be compare among different population groups. There are two ways to standardize rates, the indirect or direct methods. In general the indirect method of standardization is most frequently used.

Indirect standardization: The disease rates in the reference (comparison) population are multiplied by the number of individuals in the same age and gender group in the study population to obtain the expected rate of disease for the study population.

Direct standardization: The disease rates in the study population are multiplied by the number of individuals in the same age and gender group in the reference (comparison) population. This gives the expected rates of disease for the reference population if these rates had prevailed in that group.

Standardized mortality ratio (SMR): The SMR is the ratio of the number of deaths observed in the study population to the number of expected deaths. The expected number of deaths is based on a reference (or comparison population). Death rates for the U.S. population (or State) are most frequently used as the comparison to obtain expected rates. An SMR of 1 indicates a similar risk of disease in the study population

compared with the reference population. An SMR greater than 1 indicates excess risk of disease in the study population compared with the reference group, and an SMR less than 1 indicates a deficit of disease.

[Text deleted.]

Relative risk: The ratio of the risk of disease among the exposed population to the risk of disease in the unexposed population. Relative risks are estimated from cohort studies.

Odds ratio: The ratio of the odds of disease if exposed, to the odds of disease if not exposed. Under certain conditions, the odds ratio approaches the relative risk. Odds ratios are estimated from case-control studies.

Excess Relative Risk (ERR): Per SV is based on a regression model in which the relative risk is assumed to be of the form $1 + \beta Z$, where Z is the cumulative dose in SV.

Standardized Rate Ratio (SRR): A rate ratio in which the numerator and the denominator have been standardized to the same (standard) population distribution.

[Text deleted.]

Healthy Worker Effect: A phenomenon observed in studies of occupational diseases. Workers usually exhibit lower overall death or disease rates compared to the general population, due to the fact that the severely ill and disabled are excluded from employment. Rates from the general population may be inappropriate for comparison if this effect is not taken into consideration.

Confidence Interval (CI): A range of values for a variable of interest, for example, a rate, constructed so that this range has a specified probability of including the true value of the variable. The specified probability is called the confidence level, and the end points of the confidence interval are called the confidence limits.

P, **P** (**Probability**) **Value:** The probability that a test statistic would be as extreme as or more extreme than observed if the null hypothesis were true. The letter P, followed by the abbreviation n.s. (not significant) or by the symbol < (less than) and a decimal notation such as 0.01, 0.05, is a statement of the probability that the difference observed could have occurred by chance, if the groups are really alike, that is, under the *Null Hypothesis*. Investigators may arbitrarily set their own significance levels, but in most biomedical and epidemiologic work, a study result whose probability value is less than 5 percent (P < 0.05) or 1 percent (P < 0.01) is considered sufficiently unlikely to have occurred by chance to justify the designation "statistically significant."

Multivariate Analysis: A set of techniques used when the variation in several variables has to be studied simultaneously. In statistics, any analytic method that allows the simultaneous study of two or more *Dependent Variables*.

Incidence: (*Syn:incident number*) The number of instances of illness commencing, or of persons falling ill, during a given period in a specified population. More generally, the number of new cases of a disease in a defined population, within a specified period of time. The term incidence is sometimes used to denote *Incidence Rate*.

Incidence Rate: The rate at which new events occur in a population. The numerator is the number of new events that occur in a defined period; the denominator if the population at risk of experiencing the event during this period, sometimes expressed as person-time. The incidence rate most often used in public health practice is calculated by the formula

Number of new events in specified period x10ⁿ Number of persons exposed to risk during this period

In a dynamic population, the denominator is the average size of the population, often the estimated population at the mid-period. If the period is a year, this is the annual incidence rate. This rate is an estimate of the persontime incidence rate, that is, the rate per 10ⁿ person-years. If the rate is low, as with many chronic diseases, it is also a good estimate of the cumulative incidence rate. In follow-up studies with no censoring, the incidence rate is calculated by dividing the number of new cases in a specified period by the initial size of the cohort of persons being followed; this is equivalent to the cumulative incidence rate during the period. If the number of new cases during a specified period is divided by the sum of the person-time units at risk for all persons during the period, the result is the person-time incidence rate.

[Text deleted.]

M.4.2 HANFORD SITE

Surrounding Community

Sever et al. published two studies in 1988 of birth defects in Benton and Franklin Counties in which Hanford is located (AJE 1988a:226-242, 243-254). The prevalence of births of congenital malformed infants for the study period from 1968 to 1980 was the focus of one of the two studies (AJE 1988a:243-254). The congenital malformation rate in the newborn population of 19.6/1000 was not elevated compared with the rates for the States of Washington, Idaho, and Oregon (12.2 / 1000). Neural tube defects were more common than expected in the comparison area (Prevalence=1.72; 95% CI=1.22-2.34). The companion case-control study investigated whether there was any association of parental occupational exposure to external radiation and the risk of congenital malformations among births occurring from 1957 to 1980 (AJE 1988a:226-242). Two defects, congenital dislocation of the hip (12 observed, 7.1 expected, p<0.025) and tracheoesophageal fistula (4 observed, 1.4 expected, p<0.05), showed statistically significant association with parental employment at Hanford but not with parental radiation exposure.

Neural tube defects showed a significant association with parental preconception external radiation exposure. Other defects studied, including Down's Syndrome, showed no evidence of such an association with parental external radiation exposure.

Jablon et al. examined cancer mortality in populations living near nuclear facilities in the U.S., including Hanford (JAMA 1991a:1403-1408). The study compared cancer mortality in 107 counties with or near 62 nuclear facilities to those in comparison counties with similar demographic characteristics but without nuclear facilities. For Hanford, Benton, Franklin, and Grant Counties were studied. The authors concluded that no general association was detected between residents in a county with a nuclear facility and death attributable to leukemia or any other form of cancer. The authors also noted that interpretation of the study results is limited by the study's ecological approach in which the exposures of individuals are not known.

Worker Studies

Mancuso and Sanders Era

Studies of the Hanford workers began in 1969. Initially, the study of Hanford workers conducted by the University of Pittsburgh was designed to evaluate longevity and disability in workers (HP 1978a:521-538). Hanford workers were compared with their brothers or sisters and to a national sample of employed people from the Social Security Administration continuous work history files. The study included 17,600 males and 3,900

females hired from 1944 through 1971, and considered deaths that occurred from 1944 to October 1972. Workers were categorized as "radiation exposed workers" and "nonexposed workers." In general, the longevity for both males and females within each category were similar, with the largest difference for exposed men who had a nonsignificantly reduced longevity relative to their sibling controls. A second analysis included about 1,800 Hanford workers; 1,800 matched Social Security Administration continuous work history controls; and 3,055 "identified siblings." The disability claim rate for all Hanford workers was significantly lower than the matched Social Security Administration controls, as was the rate for radiation-exposed workers.

Analyses were expanded to examine specific causes of death (HP 1977a:369-385). In these analyses, the average cumulative radiation dose for workers dying of a site specific cancer, or group of cancers, was compared with the average radiation dose for all workers dying from all causes.

For deaths from 1944-1972, the following cancer types were reported with higher radiation doses: multiple myeloma, pancreas, brain, kidney, lung, colon, myeloid leukemia, and lymphomas. When the comparison was made against the average dose for all noncancer deaths rather than for all deaths, excess deaths were attributed to radiation for all cancers combined, multiple myeloma, myeloid leukemia, pancreas, and lung.

The authors examined the amount of radiation necessary to double the risk of death for specific cancers. Five cancer categories were concluded to have significant doubling doses: bone marrow cancers, pancreatic cancer, lung cancer, reticuloendothelial neoplasms, and all cancers combined. Next, the authors explored whether the doses received at some specific ages were more important than at other ages, and they concluded that sensitivity to radiation carcinogenesis was high before age 25 years and after age 45 years.

As the analytic methods used in the study were controversial, the Hanford data were re-analyzed by other investigators in 1979, and the analytic methods were reassessed. Hutchinson et al. concluded that analyses of the Hanford data, adjusted for age and calendar year of death, reduced the number of cancer sites for which a radiation dose relationship could be suggested to two: cancer of the pancreas (p=0.011 for trend test) and multiple myeloma (p=0.009 for trend test) (HP 1979a:207-220). For both of these sites, more deaths were observed than expected only among those with doses exceeding 10 rad. The authors also considered the issue of sensitive ages for radiation exposure and concluded such ages could not be identified without considering lifetime patterns of exposure ages.

In a separate independent analysis, Gofman et al. considered these issues using a different methodological approach (HP 1979a:617-639). The authors reported, consistent with the finding of Hutchinson et al., that differences in radiation dose between those dying of cancer compared with other diseases are found primarily in those receiving 10 rad or more exposure. The authors estimated that radiation caused a 3.5 percent increment in cancer deaths. The doubling dose for cancers overall was estimated at 43.5 rad, consistent with the Mancuso estimate previously reported. The authors did not concur with Mancuso et al. on the suggestion of variation in sensitivity to radiation by age at exposure.

Other methodological problems in the original analyses were identified by Anderson who concluded that the estimate of excess deaths was "implausible," but did agree that the analyses were consistent with some excess deaths from multiple myeloma, cancer of the pancreas, and possibly lung cancer (HP 1978b:743-750). A deficit in leukemia deaths was noted. The Mancuso study was also reviewed by the National Radiological Protection Board (NRPB) in the United Kingdom. This report concluded that the only excess fatal malignancies at Hanford that may be associated with radiation are cancer of the pancreas and multiple myeloma (NRPB 1978a). The report indicated that further investigation was necessary, as the effect could have been due to other carcinogens.

In 1978, Kneale, Stewart, and Mancuso updated the Hanford study with death information to 1977 (IAEA 1978a:387-412). The authors concluded that approximately 5 percent of the cancer deaths at Hanford were

radiation- induced and that these extra deaths were probably concentrated among cancers of the bone marrow, lung, and pancreas.

In 1981, Kneale et al. again reported on the Hanford data, using a different analytic technique (BJIM 1981a:156-166). The cohort included radiation-monitored employees up to 1975 and deaths through 1977. The authors estimated a linear model doubling dose at 15 rads, estimated the latency to be 25 years, and rejected the hypothesis that all age at exposure groups are equally sensitive to radiation.

In 1993, Kneale and Stewart published a re-analysis of the Hanford data (AJIM 1993a:371-389). The study included 27,395 male and 8,473 female workers who worked between 1944 and 1978 and had been monitored for radiation. Deaths were determined through 1986. In this analysis, all cancers listed on the death certificate were included in the study. The authors concluded that the Hanford data supported a doubling dose from 8.6 to 44.8 mSv, with a nonlinear dose response, in contrast to the prior study. The estimated proportion of radiation-caused cancers ranged from 12.5 percent to 50.9 percent, the cancer latency period was estimated to be 14-17 years, and the most radiosensitive ages for exposure were over 58 years of age.

In 1996, Stewart and Kneale again investigated the relationship between age at exposure and cancer risk in the Hanford data using monitored workers described in the 1993 analysis (OEM 1996a:225-230). The data were adjusted to account for the effects of date of birth and date of death. The workers were grouped by average doses into intervals of when dose was received to allow for cancer latency and age groups to isolate the most sensitive age at exposure.

The authors concluded that sensitivity to carcinogenic effects of radiation increase progressively with age during adult life and providing that the dose is too small to produce many cell deaths, the ratio of leukemias to solid tumors is no different for radiogenic and idiopathic tumors in contrast with the atomic bomb survivor data, which found a strong association with leukemia.

Simultaneously, other researchers were reporting the results of studies of the Hanford workers. In 1979, Gilbert and Marks reported the results of analyses of the mortality experience of Hanford workers from the time the plant was built through April 1974 (RR 1979a:122-148). The cohort consisted of 20,842 white males hired before 1966 with a focus on 13,075 employed at least 2 years. Mortality rates were not higher than expected among workers for all causes of death, all malignant neoplasms, diseases of the circulatory system, accidents, or other causes. When individual cancer sites were considered, only malignant neoplasm of the pancreas (SMR=130, p<0.05) among individuals who had worked less than 2 years at Hanford was significantly elevated.

To determine if there was an association with external radiation exposure, the mortality experience of workers who had been monitored for radiation was compared with all workers in the study. Among white males monitored for radiation, there was a statistically significant trend between mortality and increasing radiation dose for pancreatic cancer (4 observed, 2.5 expected; p=0.07 for trend test) and multiple myeloma (12.4 observed, 3.6 expected; p=0.006 for trend test) when lagged 2 years for cancer development. When exposures were lagged for 10 years, only deaths due to multiple myeloma (6.2 observed, 1.5 expected; p=0.006 for trend test) showed a trend with cumulative occupational exposure to ionizing radiation.

The mortality experience of the Hanford cohort was updated the following year (RR 1980a:740-741). Three hundred and ninety additional deaths among white males, occurring to May 1977, were included in the study. Results were similar to those previously reported.

The cohort was again updated in 1983 (RR 1983a:211-213). This analysis was expanded to include workers hired during and after 1965 and employed 2 or more years. In this analysis, the significant positive trend between increasing dose and pancreatic cancer disappeared. The significant trend for multiple myeloma remained.

The next update of the cohort mortality study for Hanford was published in 1989 (HP 1989a:11-25). The cohort consisted of 31,500 males and 12,600 females first employed through 1978. Deaths from 1944-1981 were analyzed for the entire cohort. Death certificates for radiation-monitored workers who died in the State of Washington between 1982-1985 were also obtained.

Overall, Hanford workers continued to have death rates substantially below the general U.S. population. Among female workers not monitored for external radiation, there were significantly more deaths for the category of accidents, poisonings, and violence than expected (SMR=1.38, p=0.05). Monitored females had a higher rate of death from diseases of the musculoskeletal system and connective tissues than expected (SMR=2.33, p=0.05). When individual cancer sites were considered, males not monitored for radiation were observed to have significantly higher rates of death from pancreatic cancer (SMR=1.69, p=0.01) and solid tumors (SMR=1.56, p=0.05) than expected.

The risk analyses for trends by radiation dose were lagged for 2- and 10-year induction periods, and included deaths from 1947 through 1981. No correlation between mortality and dose was seen when the analyses were lagged for 2 years. When dose was lagged 10 years, there was a suggestive trend between dose and deaths from all cancers, genital cancer among females, and multiple myeloma.

Although the number of workers at Hanford with Pu deposition was limited, data on these workers were analyzed separately to examine major cause of death categories by exposure categories. No trends between increasing death rates and increasing deposition Pu were detected. As cause of death information was available through 1985 for those dying in the State of Washington, additional analyses were conducted. Four additional deaths from multiple myeloma were observed, but the trend with dose was not statistically significant.

The Hanford cohort was once again updated by Gilbert et al. in 1993 (HP 1993a:577-590). This analysis included workers who were employed 6 months or more and were first employed through 1978. Deaths among the entire cohort that occurred from 1944 through 1986, and through 1989 for monitored workers who died in the State of Washington, were analyzed. This data set included 456 workers not previously studied and eliminated 265 individuals who never actually worked at the site. Radiation dose records from construction worker files were also added to the data set.

When the death rates for Hanford workers were compared with the general U.S. population, monitored females continued to have an elevated rate of deaths from musculoskeletal system and connective tissue conditions (SMR=2.06, p=0.05) noted in the 1989 paper. As previously reported, unmonitored males continued to have higher death rates for pancreatic cancer (SMR=1.57, p=0.05) and the category noted as miscellaneous solid tumors (SMR=1.47, p=0.05).

As in previous papers, the data were then analyzed to examine trends between the risk of death and external radiation dose lagged for 2 and 10 years. Statistically significant trends were seen when the dose was lagged 10 years for deaths due to pancreatic cancer (SMR=1.59, p=0.065), Hodgkin's disease (SMR=1.80, p=0.038), and multiple myeloma (SMR=1.54, p=0.10). Deaths due to liver cancer (SMR=1.93, p=0.065) were detected when the exposures were lagged for 2 years. Additional analyses were conducted, which included "all" cancers noted on the death certificate, in addition to those reported on the death certificate as the "underlying cause of death." The investigators concluded that there were no additional cancers that showed significant correlations with dose as compared with the previous analysis that used the underlying cause of death.

Hanford workers have been included in several studies that have examined occupational risks across the nuclear complex, both in the U.S. and internationally. These combined studies have been undertaken in an attempt to increase the statistical power of the studies to detect the effects of low-level chronic radiation exposure.

A combined site mortality study included workers from Hanford, Oak Ridge, and Rocky Flats (RR 1993a:408-421). Earlier analyses of these cohorts indicated that risk estimates calculated through

extrapolation from high-dose data to low-dose data did not seriously underestimate risks of exposure to lowdose radiation (AJE 1990a:917-927; RR 1989a:19-35). The updated analyses were performed in order to determine whether the extrapolated risks represented an over-estimation of the true risk at low doses. The study population consisted of white males employed at one of the three facilities for at least 6 months and monitored for external radiation. The Hanford population also included females and nonwhite workers. The total population dose was 1237 Sv. Analyses included trend tests for site-specific cancer deaths and several broad noncancer categories. Statistically significant trends were noted for cancer of the esophagus (p=0.015 for trend test), cancer of the larynx (p=0.019 for trend test), and Hodgkin's disease (p=0.048 for trend test). These cancers were not related to radiation exposure levels in previously published studies. Excess relative risk models were calculated for the combined DOE populations and for each DOE site separately. Without exception, all risk estimates included the possibility of zero risk (that is, the confidence interval for the risk coefficient went from below zero to above zero). There was evidence of an increase in the excess relative risk for cancer with increasing age in the Hanford and Oak Ridge populations; both populations showed significant correlations of all cancer with radiation dose among those 75 years and older.

Multiple myeloma (p=0.103 for trend test) was the only cancer found to exhibit a statistically significant correlation with radiation exposure that was based on the excess previously reported among Hanford workers.

An international effort to pool data from populations exposed to external radiation included Hanford workers, as well as workers at Rocky Flats and Oak Ridge in the U.S. and other radiation worker populations in Canada and Britain (RR 1995a:117-132). The cohort compared 95,673 workers employed 6 months or longer and the population dose was 3,543.2 Sv. There was no evidence of an association between radiation dose and mortality from all causes or from all cancers. There was a significant dose-response relationship with leukemia, excluding chronic lymphocytic leukemia (ERR=2.18 per Sv; 90 percent CI 0.1-5.7) and multiple myeloma (ERR not computed; 44 observed; p=0.037 for trend test). The study results do not suggest that current radiation risk estimates for cancer at low levels of exposure are appreciable in error.

Epidemiologic Studies

DOE's Office of Epidemiologic Studies has implemented an epidemiologic surveillance program at Hanford to monitor the health of current workers. This program will evaluate the occurrence of illness and injury in the workforce on a continuing basis and the results will be issued in annual reports. The implementation of this program will facilitate an ongoing assessment of the health and safety of Hanford's workforce and will help identify emerging health issues.

Currently operational at a number of DOE sites, including production sites and research and development (R&D) facilities, epidemiologic surveillance uses routinely collected health data including descriptions of illness resulting in absences lasting 5 or more consecutive workdays, disabilities, and OSHA recordable injuries and illnesses abstracted from the OSHA 200 log. These health event data, coupled with demographic data about the active workforce at participating sites, are analyzed to evaluate whether particular occupational groups are at increased risk of disease or injury when compared with other workers at a site. As the program continues and data for an extended period of time become available, time trend analysis will become an increasingly important part of the evaluation of worker health. Monitoring the health of the workforce provides a baseline determination of the illness and injury experience of workers and a tool for monitoring the effects of changes made to improve the safety and health of workers. Noteworthy changes in the health of the workforce may indicate the need for more detailed study or increased health and safety measures to ensure adequate protection for workers.

Memorandum of Understanding

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The Hanford Environmental Dose Reconstruction (Hanford Environmental Dose Reconstruction) Project was undertaken by DOE to estimate the radiation dose that people may have received from nuclear operations at

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Hanford (WA Ecology 1994a). In 1990, DOE entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites. The Centers for Disease Control and Prevention's National Center for Environmental Health is responsible for dose reconstruction studies and has managed the Hanford Environmental Dose Reconstruction Project since that time.

The study determined that the largest doses to offsite populations were from iodine-131 released into the air in large quantities between December 1944 and December 1947. The most important radiation exposure pathway for iodine-131 was the consumption of milk produced by cows grazing on pasture downwind of Hanford. The doses to the thyroid gland of individuals near Hanford were larger than those farther from the site, and depended on the iodine-131 deposition and quantity of milk consumed at each location.

A second pathway of potential importance was the Columbia River. Releases to the river from Hanford were highest in the years 1956-1965, which was the height of reactor operations at Hanford. The most important means of exposure from the river pathway was the consumption of fish by local residents. However, maximum doses for the heaviest consumption of fish were estimated to be about half the dose an individual normally receives each year from all sources of radioactive materials in the U.S. environment. This study is in its final stages and will be completed shortly.

A study in the United Kingdom linked a father's exposure to ionizing radiation in the workplace with the subsequent risk of leukemia in his children (RR=6.42; 95 percent CI=1.57-26.3) (BMJ 1990a:423-434). A study was undertaken to replicate this study in other similar populations. Hanford was one of three sites selected for study by NIOSH. The population under study consists of residents of Benton and Franklin Counties. The study includes leukemia, non-Hodgkin's lymphoma, and central nervous system tumors diagnosed from 1957-1991 in children under the age of 15. The study is expected to be completed in 1996.

A number of studies of the Hanford workforce are underway, directed by NIOSH, under the Memorandum of Understanding.

Researchers at the University of Texas Medical Branch are conducting a cohort mortality study of female nuclear weapons workers exposed to low levels of ionizing radiation and other workplace physical and chemical agents at 12 DOE facilities, including Hanford. The study will be completed in 1997.

A study of multiple myeloma among workers exposed to ionizing radiation and other physical and chemical agents is being conducted by the University of North Carolina at various DOE sites, including Hanford. The study is expected to be completed in 1996-1997.

An epidemiologic evaluation of childhood leukemia and paternal exposure to ionizing radiation is being conducted by Battelle Columbus. The study will collect information of selected childhood cancers, residential history, and the father's exposure to ionizing radiation. Completion of the study is expected in 1996-1997.

An epidemiologic study of leukemia at four DOE sites is being conducted by NIOSH. Sites selected for the study include Hanford.

Boston University is conducting a health-effects-of-job-stress study associated with the redesign and reconfiguration of the nuclear weapons industry. This study will identify how specific practices for managing change affect individual health and work performance and to recommend ways to minimize health effects in the future. Hanford is one of seven DOE facilities included in this multisite study. The study will begin in 1996 and is scheduled for completion in 1999.

A comprehensive occupational health surveillance project at Hanford will design and implement a health surveillance system at the site. The University of Washington and the Hanford Environmental Health Foundation will conduct the study. Completion is expected in 1998.

A study of heat stress among carpenters at Hanford will assess the real effects of heat stress on physiologic functions in a real work situation. The study is being conducted by Michigan State University and the United Brotherhood of Carpenters' Health and Safety Fund. The study is scheduled for completion in 1999.

Other Related Studies

The Hanford Thyroid Disease Study began in 1988 under the management of Centers for Disease Control and will be completed in 1998 (HF FHCRC 1995a). It was initiated based on preliminary information from the Hanford Environmental Dose Reconstruction Project indicating that releases of radioactive iodine-131 from Hanford in its early years may have produced exposures to human thyroids large enough to have affected the gland's functioning. About 3,200 people living at various distances from Hanford have been located and are now being examined for thyroid disease and thyroid function. These people were selected because as infants during the years of peak releases of iodine-131 they were the most sensitive population group. Information gathered from the individuals in the study about their diet, milk consumption, age, sex, and place of residence will be used to calculate individual doses received by the thyroid gland using the models developed in the Hanford Environmental Dose Reconstruction Project. The study results are expected in 1998.

M.4.3 NEVADA TEST SITE

Surrounding Communities. Above ground testing of nuclear weapons at the NTS Test Range Complex in southern Nevada between 1951 and 1958 resulted in the dissemination of radioactive fallout over southeastern Nevada and southwestern Utah through wind dispersion. Several epidemiologic studies have been conducted to investigate possible health effects of low-level radiative fallout on residents of these States. These studies focused on leukemia and thyroid disease in children downwind of NTS.

A series of ecologic studies showed equivocal results in potentially exposed children. A cross sectional review of thyroid modularity among teenage children reported by Weiss et al. found no significant difference in the frequency of nodules among "potentially exposed" and "not exposed" children (AJPH 1971a:241-249). Exposure was defined in terms county of residence. Rallison et al. reported no significant difference in any type of thyroid disease between Utah children exposed to fallout radiation in the 1950s and control groups drawn from Utah and Arizona (AJM 1974a:457-463; JAMA 1975a:1069-1072).

To investigate the possible relationship between childhood leukemia and radioactive fallout, Lyon et al. conducted a mortality study of Utah-children under 15 years old who died in Utah between 1944 and 1975 (NEJM 1979a:397-402). Lyon et al. selected this age group because of the reported increased susceptibility of children to the neoplastic effects of radiation and the lack of a comparison group over 14 years of age with suitable low exposures. Lyon et al. obtained death certificates from the Utah vital statistics registrar and based on year of death, categorized decedents into either high (fallout years of 1951-1958) or low exposure periods (combined pre-fallout years of 1944-1950 and post-fallout years of 1959-1975). From estimated fallout patterns contained in maps of 26 tests, Lyon et al. categorized 17 southern rural counties high fallout area and the remaining northern urban counties as low fallout areas. Age-specific mortality rates derived for deaths which occurred in the combined low exposure periods were compared with those in the high exposure period. For reasons unknown, leukemia mortality during the low exposure periods in high fallout counties was half that of the United States and Utah. A significant excess of leukemia occurred among children statewide who died during the high fallout period compared to those who died during the low fallout periods (SMR=1.40, 95 percent CI=1.08-1.82, p<0.01). This excess was more pronounced among those who resided in the high fallout area (SMR=2.44, 95 percent CI=1.18-5.03). No pattern was found for other childhood cancers in relation to fallout exposure. A radiation dosage was not available, and the effect of migration were not determined for this study.

Beck and Krey reconstructed exposure of Utah residents studied by Lyon et al. (Science 1983a:18-24) to external gamma-radiation from NTS fallout through measurements of residual cesium-137 and Pu in soil

(NEJM 1979a:397-402). Beck and Krey found that residents in southwest Utah closest to NTS received the highest exposures, but noted that residents of urban northern areas received a higher mean dose and a significantly greater population dose than did residents of most counties closer to the test site. Northern Utah residents received higher average bone doses than southern Utah residents; therefore, the distance from NTS should not be the sole criteria for dividing the State into geographic subgroups for the purpose of conducting epidemiologic studies. Beck and Krey concluded that bone doses to southern Utah residents were too low to account for the excess leukemia deaths identified by Lyon et al. They also determined that bone and whole body doses from NTS fallout were small relative to lifetime doses most Utah residents receive from background radiation, and that it was unlikely that these exposures would have resulted in any observed health effects.

Land et al. attempted to confirm the association between leukemia and fallout reported by Lyon et al. using cancer mortality data from the National Center for Health Statistics for the period 1950 through 1978 (NEJM 1979a:397-402); (Science 1984a:139-144). No statistically significant differences in mortality from leukemia or other childhood malignancies between northern (SRR=1.52, 90 percent CI=1.24-1.87) and southern Utah (SRR=1.49, 90 percent CI=0.88-2.51) were observed. The observed difference in leukemia mortality between the border and interior counties was opposite in direction to that reported by Lyon et al. Results indicated a downward trend in childhood leukemia mortality over time. Eastern Oregon and the State of Iowa also were selected for comparison with Utah. The leukemia mortality rate for eastern Oregon was higher (SRR=1.81, 90 percent CI=1.07-3.07), and Iowa lower (SRR=1.16, 90 percent CI=1.02-1.31) than the rate for Utah (SRR=1.49, 90 percent CI=0.88-2.51). Land et al. concluded that these results suggest that the association reported by Lyon et al. merely reflects an unexplained low leukemia rate in southern Utah for the period 1944 to 1949.

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States Streets

Another study that assessed the development of cancer among individuals potentially exposed to radioactive fallout has been reported by Rallison et al. (HP 1990c:739-746). This study examined the thyroid neoplasia risk in a cohort of children born between 1947 and 1954 in two counties near nuclear test sites, one in Utah and one in Nevada. A comparison group of Arizona children presumed to have no fallout exposure was also evaluated. The children (11 to 18 years of age) were examined between 1965 and 1968 for thyroid abnormalities and were reexamined in 1985 and 1986. Children living in the nuclear testing (Utah/Nevada) area had a higher rate of thyroid neoplasia (5.6/1000 for phase 1 and 24.6/1000 for phase 2) than the comparison children in Arizona (3.3/1000 for phase and 20.2/1000 for phase 2), but the differences were not statistically significant (RR=1.2, p=0.65 for phase 2). The authors concluded that living near the NTS in the 1950s has not resulted in a statistically significant increase in thyroid neoplasms.

A study by Johnson examined cancer incidence in a cohort of families that were members of the Church of Jesus Christ of Latter-Day Saints in southwest Utah near the NTS (JAMA 1984b:230-236). The study compared cancer incidence among all Utah members of the Church of Jesus Christ of Latter-Day Saints during the period 1967-1975 with cancer incidence among two exposed populations: persons residing in a "high fallout area" and an "exposure effects group" residing in a broader area that received less intense exposure from radioactive fallout. Limitations of the study include: the inability to locate 40 percent of the defined population; the lack of verifying the reported diagnosis of cancer; and the inability to interview a comparable control group.

Cancer incidence for both exposed groups was compared with that of all Utah members of the Church of Jesus Christ of Latter-Day Saints for two time periods, 1958-1966 and 1972-1980. Johnson found an apparent increased incidence of leukemia (19 cases, 3.6 expected, p=0.01) and cancers of the thyroid (6 observed, 1.4 expected, p=0.01) and bone (3 observed, 0.3 expected, p=0.01) for residents of the high fallout area for both time periods. Additional analyses suggested that a higher proportion of the cancers among exposed groups were in radiosensitive tissues and the proportional excess increased with time compared with all Utah members of the Church of Jesus Christ of Latter-Day Saints. The ratio of radiosensitive cancers to all other cancers from 1959-1966 was 24 percent higher among the "high fallout area" group and 29.6 percent higher among those in the "fallout effects" group. For 1972-1980, the ratio was 53.3 percent higher in the "high fallout area" group and 300 percent higher in the "fallout effects" group.

Machado examined cancer mortality rates of a three-county region in southwestern Utah in comparison to the remainder of Utah (AJE 1987c:44-61). There was no excess risk of cancer mortality in southwest Utah, with the exception of leukemia (OR=1.45, 90 percent CI=1.18-1.79 with Utah controls), which showed a statistically significant excess for all ages combined, and for children age 0-14. In fact, mortality from all cancer sites combined was lower in southwest Utah than the remainder of the State. The authors noted that their findings, including those for leukemia, were inconsistent with the cancer incidence study conducted by Johnson (JAMA 1984b:230-236).

Archer measured soil, milk, and bone strontium-90 levels to identify states with high-, intermediate and low-fallout contamination (AEH 1987a:263-271). He then correlated the deaths from radiogenic and nonradioactive leukemias with the time periods of above ground nuclear testing both in the United States and Asia. The results show that leukemia deaths in children were higher in States with high exposure and lower in States with less exposure. He showed that leukemia deaths in children peaked approximately 5.5 years following nuclear testing peaks. The last leukemia peak in the United States occurred in 1968 to 1969, 5.5 years after the last year of a 3-year period of intensive testing in Asia. The increases were seen in the radiogenic leukemias (myeloid and acute leukemias), and not with "all other leukemias."

Kerber et al. updated a previously identified cohort of children living in portions of Utah, Nevada, and Arizona, to estimate individual radiation doses and determine thyroid disease status through 1985-1986 (JAMA 1993a:2076-2082). Of the 4,818 children originally examined between 1965-1970, 2,473 were included in the followup exam. Outcomes of interest included thyroid cancers, neoplasms, and nodules based on physical examinations of the thyroid. Exposure of the thyroid to radioiodines was based on radionuclide deposition rates provided by DOE and surveys of milk producers. Children with questionable findings were referred to a panel of endocrinologists for further examination. The authors reported an excess number of thyroid neoplasms (combined benign and malignant) and a positive dose-response trend for neoplasms, both of which were statistically significant. The authors also reported a positive dose-response trend for thyroid nodules, not statistically significant, and a positive dose-response trend for thyroid carcinomas with marginal statistical significance. The authors estimated that an excess of between 1 and 12 neoplasms (between 0 to 6 excess malignancies was probably caused by exposure to radioiodines from the nuclear weapons testing. A letter to the editor criticized Kerber et al. for relying on food histories obtained 22 years after the fact to depict radioiodine intake, and for the untested modeling approach-for determining dose to the thyroid (JAMA 1994a:825-826). These concerns were addressed by Kerber et al., which acknowledged the uncertainties in the dose estimates, but concluded that their estimates were conservative (JAMA 1994a:826).

Till et al. estimated doses to the thyroid of 3,545 subjects who were exposed to radioiodine fallout from NTS (HP 1995a:472-483). The U.S. Public Health Service first examined this cohort for thyroid disease between 1965-70 and later in 1985-86. Till et al. assigned individual doses based on age, residence histories, dietary histories, and lifestyle. Individualized dose and uncertainty was combined with the results of clinical examinations to determine the relationship between dose from NTS fallout and thyroid disease incidence.

Workers. Military personnel and civilian employees of the Department of Defense observed and participated in maneuvers at the NTS Test Range Complex during above ground tests. An excess number of leukemia cases was reported (9 cases, 3.5 expected) among the 3,224 men who participated in military maneuvers in August 1957 at the time of the nuclear test explosion "Smoky" (JAMA 1980a:1575-1578). The participants were located and queried on their health status, diseases, or hospitalizations as of December 1981. Various Federal records systems were linked, including clinical files, and next of kin was queried about cause of death for those participants who were deceased. Exposure information was available from film badged records, and the mean gamma dose for the entire cohort was 466.2 mrem. In a later report of the same cohort, the number of incident cases of leukemia had increased to 10 with 4 expected (O/E=2.5, 95 percent CI=1.2-4.6) (JAMA 1983a:620-624). No excess in "total cancers" was observed, however. In addition, four cases of polycythemia vera were reported where 0.2 was expected (JAMA 1984a:662-664). The excess in leukemia cancer incidence and mortality appear to be limited to the soldiers who participated in "Smoky." The leukemia excess was not observed in a National Research Council mortality study of soldiers exposed to five series of tests at two sites: Nevada Test Site (PLUMBBOB) and the Pacific Proving Ground (DOE 1985b). The National Research Council reported that the number of leukemia cases in "Smoky" was greater, but the increase was considered nonsignificant when analyzed with the data from the other four tests. In 1989, however, it was discovered that the roster of the atomic veterans cohort on which the National Research Council based its 1985 study contained misclassification errors. As a result, this study is being reanalyzed, and the National Research Council anticipates publishing the new results by 1997.

M.4.4 IDAHO NATIONAL ENGINEERING LABORATORY

Surrounding Communities

Jablon et al. examined cancer mortality in populations living near nuclear facilities in the U.S., including INEL in Idaho (JAMA 1991a:1403-1408). The study compared cancer mortality from 1950-1984 in 107 counties with or near 62 nuclear facilities with cancer mortality in control counties without nuclear facilities. Cancer mortality for Bingham, Butte, and Jefferson Counties, where INEL is located, was compared with nine control counties in the same region, with similar demographic characteristics. The authors concluded that no general association was detected between residents in a county with a nuclear facility and death attributable to leukemia or any other form of cancer. The authors noted that interpretation of the study results is limited by the study's ecological approach in which the exposures of individuals are not known.

Cancer morbidity and mortality data in two additional counties near INEL, Clark and Minidoka, were reviewed by the Idaho Department of Health and Welfare (ID DHW 1991a; ID DHW 1991b). Clark County lies northeast of INEL and Minidoka County southwest of INEL. Cancer death rates were examined for the years 1950-1989 and cancer incidence rates for the years 1978-1987 to determine if any significant trends in cancer morbidity and mortality could be observed in these counties compared with the entire State. No statistically significant differences in age- and sex-adjusted death rates were observed in either county.

When cancer incidence data were considered, the overall cancer incidence rate in Clark County was higher than expected based on the State of Idaho's experience. When the Clark County data were examined by primary site, only two sites were found to be significantly higher than expected—female breast cancer (8 cases observed vs. 3.2 expected, p=0.05) and lip cancer (3 cases observed vs. 0.4 expected, p=0.05). In Minidoka County, there was no increase in overall cancer incidence rate compared with the entire State. Examination by primary sites in Minidoka County, however, showed three cancer sites were found to be increased—cancer of the stomach (20 cases observed vs. 11.6 expected, p=0.05), lip (23 cases observed vs. 8 expected, p=0.01), and uterus (40 cases observed vs. 24.2 expected, p=0.01). These studies also suffered from the limitations inherent in ecological studies. In addition, the authors noted that too many comparisons were made for "significant" results and that the data for Clark County, with an estimated population of 800, were too small to make meaningful analyses.

State Health Agreement Program

In 1991, INEL completed a historical dose reconstruction study to examine the impact of radioactive materials released to the environment during INEL's past operations. Subsequently, under the State Health Agreement program managed by the DOE Office of Epidemiologic Studies, a grant was awarded to the State of Idaho to convene an expert panel to review the final dose reconstruction report. The State panel evaluated the environmental transport and dose assessment models used for the dose reconstruction and recommended that additional work, involving public participation, be done to more fully examine offsite consequences (ID DHW 1993a).

Workers

No occupational epidemiologic studies have been completed at INEL to date.

Memorandum of Understanding

DOE entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites, and the Centers for Disease Control and Prevention became responsible for conducting dose reconstructions in several host States, including Idaho. Under the Memorandum of Understanding, Centers for Disease Control began a phased approach to determine the need for an expansion of the dose reconstruction work initiated earlier and reviewed by the State of Idaho. The first part, data identification retrieval, was completed in 1995. No decision about the need for additional phases of a dose reconstruction for INEL has yet been reached. NIOSH is responsible for worker studies and is currently conducting a cohort mortality study of the workforce with a projected completion date of September 1997 (IN DOE 1995e).

Epidemiologic Studies

DOE's Office of Epidemiologic Studies has implemented an epidemiologic surveillance program at INEL to monitor the health of current workers. This program will evaluate the occurrence of illness and injury in the workforce on a continuing basis and the results will be issued in annual reports. The implementation of this program will facilitate an ongoing assessment of the health and safety of INEL's workforce and will help identify emerging health issues.

Currently operational at a number of DOE sites, including production sites and R&D facilities, epidemiologic surveillance uses routinely collected health data including descriptions of illness resulting in absences lasting 5 or more consecutive workdays, disabilities, and OSHA recordable injuries and illnesses abstracted from the OSHA 200 log. These health event data, coupled with demographic data about the active workforce at the participating sites, are analyzed to evaluate whether particular occupational groups are at increased risk of disease or injury when compared with other workers at a site. As the program continues and data for an extended period of time become available, time trend analysis will become an increasingly important part of the evaluation of worker health. Monitoring the health of the workforce provides a baseline determination of the illness and injury experience of workers and a tool for monitoring the effects of changes made to improve the safety and health of workers. Noteworthy changes in the health of the workforce may indicate the need for more detailed study or increased health and safety measures to ensure adequate protection for workers.

M.4.5 PANTEX PLANT

Surrounding Communities. A June 1994 study by the Texas Cancer Registry, Texas Department of Health, showed significant increases in prostate cancer mortality among Potter County and Randall County males, and leukemia mortality among Carson County males during the period 1981-1992 (TX DOH 1994a). There were no statistically significant increases observed in site-specific cancer mortality among females during this period. For cancer incidence during the period 1986-1992, no statistically significant excesses in males were seen; however, cancer of the prostate was slightly elevated in Potter/Randall County males. Analysis of the four major cell-specific types of leukemia, showed a significant excess in the incidence of chronic lymphocytic leukemia among Potter/Randall County females. This study was conducted in Carson, Potter, and Randall Counties, which are located near Pantex. This study focused only on cancers of the breast, prostate, brain, thyroid, and leukemia, which were of specific concern to citizens in the area. Other radiation-associated cancers such as bone and lung, were not included in this study. Although prostate cancer and chronic lymphocytic leukemia have not been linked to radiation exposure, further follow-up to this study was recommended.

Workers. An epidemiologic study of Pantex workers was published by Acquavella (HP 1985a:735-746). This study compared total and cause-specific mortality for Pantex workers employed between 1951 and December 31, 1978, with expected cause-specific mortalities based on U.S. death rates. Significantly fewer deaths were observed in the workforce than would be expected based on U.S. death rates for the following causes of death: all cancers (SMR=0.72; 95 percent CI=0.64 0.81), arteriosclerotic heart disease (SMR=0.75; 95 percent CI=0.61-0.91), and digestive diseases (SMR=0.46; 95 percent CI=0.22-0.85). No specific causes of death occurred significantly more frequently than expected. Slightly elevated mortality ratios were observed for brain cancer (SMR=1.36; 95 percent CI=0.37-3.47) and leukemia (SMR=1.28; 95 percent CI=0.35-3.27); neither excess was statistically significant. The four deaths from brain cancer all occurred among those who had worked at the plant less than 5 years. The four deaths from leukemia occurred with equal frequency among those who had worked at the plant a short time and those who had worked more than 15 years.

Memorandum of Understanding. A follow-up of the 1985 mortality study of the Pantex workforce is planned. The update will be conducted by NIOSH as part of a research program funded by DOE under a Memorandum of Understanding with the Department of Health and Human Services. The followup study is scheduled to commence either in late 1996 or early 1997. In addition, female workers at Pantex will be included in a NIOSH funded multisite study of mortality among female nuclear weapons workers.

Epidemiologic Surveillance. DOE's Office of Epidemiologic Studies' Epidemiologic Surveillance Program was implemented at Pantex in 1993 in order to monitor the health of current workers. This program evaluates the occurrence of illness and injury in the workforce on a continuing basis and issues the results of the ongoing surveillance in annual reports. The program facilitates an ongoing assessment of the health and safety of the site's workforce and helps to identify any emerging health issues in a timely manner. Monthly data collection began on January 1, 1994, and the results of the first complete year of epidemiologic surveillance will be presented to workers and other site stakeholder groups in spring 1996.

Currently operational at a number of DOE sites, including production sites and R&D laboratories, epidemiologic surveillance makes use of routinely collected health data including descriptions of illness resulting in absences lasting five or more consecutive workdays, disabilities, and OSHA recordable injuries and illnesses abstracted from the OSHA 200 log. These health event data coupled with demographic data about the active workforce at the participating sites, are analyzed to evaluate whether particular occupational groups are at increased risk of disease or injury when compared with other workers at a site. As the program continues and data become available for an extended period of time, trend analysis will become an increasingly important part of the evaluation of workers health. Monitoring for changes in the health of the work force provides both a baseline determination of the illness and injury experience of workers and a tool for monitoring the effects of changes made to improve the safety and health of workers. Noteworthy changes in the health of the workforce may indicate areas in need of more detailed study or increased health and safety measures to ensure adequate protection for workers.

M.4.6 OAK RIDGE RESERVATION

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Surrounding Communities. The population-based National Cancer Institute mortality survey for selected nuclear facilities (NIH Publication No. 90-874; JAMA 1991a:1403–1408) examined the cancer mortality in communities near several nuclear facilities, including Anderson and Roane counties. No excess cancer mortality was observed in the population living in the exposed counties when compared to the U.S. white male population nor when compared to the population of the control counties (Blount, Bradley, Coffee, Jefferson, Hamblen, TN, and Henderson, NC), nor when time trends were assessed.

Tennessee Medical Management, Inc. used data from the Tennessee Cancer Reporting System to compare mortality and incidence data for counties near Oak Ridge, Tennessee, for the 3-year period 1988-1990 to the U.S. population (TMM 1993a). For Oak Ridge, total deaths from all causes was significantly lower than expected. For Anderson County, the observed number of deaths from uterine cancer and from cancer of respiratory and intrathoracic organs was statistically greater than expected and the number of deaths from brain cancer, breast cancer, and all the other sites category were lower than expected for Anderson County. For Roane County, the number of deaths from cancer of the respiratory and intrathoracic organs was statistically greater than expected; the number of deaths from cancer of the digestive organs and the peritoneum, and from uterine, lip, oral cavity, and pharynx cancer was lower than expected.

Tennessee Medical Management, Inc. examined new (incident) cancer cases and identified the following statistically significant: for Anderson County, the observed numbers of cases of cancer of the prostate and of cancer of the lung and bronchus were greater than expected. Leukemia, stomach and small intestine cancers, and cancers of the colon and intestinal tract were lower than expected. For Roane County, the number of cases of cancer of the lung and bronchus was greater than expected. Non-Hodgkin's lymphoma, female breast cancer, esophageal cancer, cancer of the pancreas, and cancer in all sites were lower than expected. The only consistent excess reported for both cancer mortality and cancer incidence was for cancer of respiratory and intrathoracic organs.

Because of a concern for possible contamination of the population by mercury, the Tennessee Department of Health and Environment conducted a pilot study in 1984 (TN DHE 1984a). The study showed no difference in urine or hair mercury exposures (residence or activity in contaminated areas) compared to those with little potential exposure. Mercury levels in some soils measured as high as 2,000 parts per million. Analysis of a few soil samples showed that most of the mercury in the soil however, was inorganic, thereby lowering the probability of bioaccumulation and health effects. Examination of the long-term effects of exposure to mercury and other chemicals continues.

State Health Agreement Program. Under the State Health Agreement program managed by the DOE's Office of Epidemiologic Studies, a grant was awarded to the Tennessee Department of Health and Environment. The purpose of the grant was to determine the extent of exposure to contaminants among workers and residents of the surrounding community as a result of ORR operations and to assess the current status of health outcomes and determine their potential association with these exposures.

A dose reconstruction feasibility study began in 1992 with the contract awarded by the State of Tennessee to Chemrisk. The contractor performed extensive review of Oak Ridge documents and issued a report which concluded that sufficient information exists to reconstruct past releases and offsite doses caused by radioactive and hazardous materials. The report also concluded that doses from mercury, polychlorinated biphenyls, radioactive iodine, and radioactive cesium may have been great enough to cause harmful health effects in offsite population. Based on this information, a full dose reconstruction study was initiated in August 1994.

Other activities supported under the grant include: development of a birth defects registry; a quality improvement program for the Tennessee cancer registry; a review and evaluation of the DOE occupational medical program; and the implementation of a community participation/public information program.

Technical support to the State health department is provided by a 12 member Oak Ridge Health Agreement Steering Panel. The Health Advisory Panel provides direction and oversight to those working on health studies, ensures public input, and informs the public of activities related to the health studies. A representative of the Centers for Disease Control and Prevention's National Center for Environmental Health is a member of the advisory panel. A representative from DOE serves as an "ex-officio" member.

Workers. Between 1943 and 1985, there were 118,588 male and female individuals of all races who were ever employed in any of the Oak Ridge facilities. These included ORNL for nuclear research (also called the X-10 Facility), Y-12 under management of the Tennessee-Eastman Corporation (1943 to 1947) which produced enriched uranium by the electromagnetic separation process, Y-12 under management of Union Carbide (1948 to 1984) which fabricated and certified nuclear weapons parts, and K-25 (Oak Ridge Gaseous Diffusion Plant) which produced enriched uranium through the gaseous process. Analyses at the Oak Ridge facilities have been

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carried out mostly for white males, and for specific cohorts talking into consideration time-related exposure risks.

Oak Ridge National Laboratory. The mortality experience of 8,375 white males employed at least a month between 1943 and 1972 at ORNL was compared with the U.S. white male population using SMR analyses in a 1985 paper by Checkoway, et al. (BJIM 1985a:525-533). Increases in deaths from leukemia (SMR=1.49, .16 observed; 95 percent Confidence Interval=0.31-4.38), cancer of the prostate (SMR=1.16, 14 observed, 11.9 expected), and Hodgkin's disease (SMR=1.10, 5 observed, 3.7 expected) were observed, although none were statistically significant. Dose response analyses were performed for all causes of death combined, all cancers combined, leukemia, and prostate cancer comparing exposed worker death rates with non-exposed worker death rates. Dosimetry data were available for the entire period of the study with the total population external radiation dose measuring 135 Sv. No dose response gradients were observed. Death rates were calculated for 11 different job categories by length of time in each job in an attempt to determine whether specific work environments were related to cancer and leukemia. Leukemia mortality was observed to be related to length of employment in engineering and maintenance jobs.

Follow-up of this cohort was extended through 1984 in an updated study by Wing et al. (JAMA 1991a:1397-1402). Again, death rates in the worker population were compared with those in the U.S. population. Nonstatistically significant increases were noted for cancers of the pancreas (SMR=1.09, 25 observed, 95 percent CI=0.71-1.61), prostate (SMR=1.05, 26 observed, 95 percent CI=0.68-1.53), brain (SMR=1.04, 15 observed, 95 percent CI=0.58-1.72), and lymphosarcoma and/or reticulosarcoma (SMR=1.05, 9 observed, 95 percent CI=0.48-1.99). There was a significant increase in deaths from leukemia (SMR=1.63, 28 observed, 95 percent confidence, interval 1.08-2.35). The total population external radiation dose was 144 Sv. Dose response analyses performed for all causes except cancer, lung cancer, and leukemia did not demonstrate a relationship between level of external radiation and increased risk of death from these outcomes. There was a significant dose response relationship (4.94 percent per 10 milliSieverts) between cancer deaths and level of external radiation dose using models with a 20-year lag. A subgroup of workers who were monitored for internal contamination had non-statistically elevated SMRs for cancer of the prostate (SMR=1.12 10-observed), 95 percent CI=0.53-2.05 and lymphosarcoma and/or reticulosarcoma (SMR=1.65, 6 observed, 95 percent CI=0.60-3.59). The workers monitored for internal contamination had a statistically significant elevated SMR for leukemia (SMR=2.23, 16 observed, 95 percent CI=1.27-3.62).

A second publication on the above data set (Wing, et al), examined the effect of controlling for a number of possible selection and confounding factors on the risk coefficient for all cancer dose responses (AJIM 1993a:265-279). Models were adjusted for the following variables with little change in the previously reported risk coefficient: employment during the World War II era, short-term employment job category, and exposure to beryllium lead, and mercury. The authors concluded that the previously calculated dose response estimate was fairly stable when adjustments were made for a wide range of potential confounders that were not explored in the earlier study.

Y-12 Plant. Y-12 is a nuclear weapons metals fabrication plant where the radiologic exposure of greatest concern is internal exposure from the inhalation of uranium compounds. The Tennessee Eastman Corporation managed the plant from 1943 to 1947. Polednak and Frome reported a follow-up through 1974 of all 18,869 white male workers employed at Y-12 from 1943 to 1947 (JOM 1981a:169-178). The workers included those exposed to internal ("alpha") and external ("beta") radiation through the inhalation of uranium dusts, electrical workers who performed maintenance in the exposed areas, and other non-exposed workers. Individual measures of exposure were not available for any members of this cohort so exposure levels were inferred from plant areas of work and jobs. High average air levels of uranium dust were documented in departments employing chemical workers. Elevated SMRs were observed for mental, psychoneurotic, personality disorders (SMR=1.36, 33 observed, 24.2 expected), emphysema (SMR 1.16, 100 observed, 85.9 expected), diseases of the bones and organs of movement (SMR=1.22, 11 observed, 8.5 expected), lung cancer (SMR=1.09, 324 observed, 296.5 expected), and external causes of death (SMR=1.09, 623 observed, 571.8 expected). The lung cancer SMR was

greater among workers employed for 1 year or more compared with workers employed less than 1 year and was more pronounced in workers hired at the age of 45 or older (SMR=1.51;95 percent CI 1.01-2.31). Of the workers employed after the age of 44, the SMR for lung cancer was greatest for electrical workers (SMR=1.55, 7 observed, D=1.11), alpha chemistry workers (SMR=3.02, 7 observed, D=2.27) and beta process workers (SMR=1.5, 11 observed, D=1.3).

During the early operation of Y-12, from 1942-1947, a group of male workers was exposed to phosgene gas on a chronic basis (N=694) and a smaller group of males received acute exposures (N=106) along with a small group of females (N=91) (ER 1980a:357-367; TIH 1985a:137-147). A control group of 9,280 workers who also worked at Y-12 during the same era, but who did not have phosgene exposure, was also described. All groups were followed through the end of 1978. The SMRs for the chronically-exposed group and the control group, were similar for all causes examined. There was no evidence for increased mortality from respiratory diseases in this group and the SMR for lung cancer, while elevated, was similar to the lung cancer SMR for workers in the rest of the plant. Among those with acute exposures, the SMR for respiratory diseases was elevated (SMR=2.66, 5 observed). This elevation may be related to residual lung damage from the acute phosgene exposure. It was difficult to trace the vital status of the 91 women; therefore, description of these highly exposed workers was limited to listing the frequency of their initial symptoms after exposure. As expected, nausea, vomiting and cough were the most frequently reported symptoms. Unexpectedly, the women experienced a lower frequency of pneumonitis than their male counterparts.

The portion of the Y-12 cohort employed between 1947 and 1974 was described by Checkoway et al. (AJE 1988a:255-266). This study included 6,781 white male workers first employed at Y-12 between 1947 and 1974 who were employed for at least 30 days. Mortality data were collected for the cohort through the end of 1979 and were used to perform SMR and cause specific dose-response analyses. Non-statistically significant increases were observed for all cancers (SMR=1.01, 196 observed, 95 percent CI=0.88-1.17), diseases of the blood-forming organs (SMR=1.48, 3 observed, 95 percent CI=0.31-4.38), kidney cancer (SMR=I.22, 6 observed, 95 percent CI=0.45-2.66), brain cancer (SMR=1.80, 14 observed, 95 percent CI=0.98 - 3.02), and other lymphatic cancers (SMR=1.86, 9 observed, 95 percent CI=0.85-3.53). A statistically significant increase in deaths from lung cancer (SMR=1.36, 89 observed; 95 percent CI=1.09-1.67) was observed compared with the U.S. lung cancer rates, but not with Tennessee lung cancer rates (SMR=1.18, 95 percent CI=0.95-1.45). Dose-response analyses for lung cancer and internal alpha radiation dose and external gamma radiation dose did not reveal a positive relationship for a 0-year or 10-year lag. Examination of lung cancer rates distributed across both internal and external dose categories suggested a dose-response with external radiation dose among individuals who had 5 or more rems of internal dose. Brain cancer was not related to the level of internal or external radiation dose.

The Y-12 cohort studied by Checkoway was updated through the end of 1990 by Loomis and Wolf and included African-American and white female workers (AJIM 1996a:131-141). The dose-response analyses were not included in the update; therefore, only SMR analyses are reported. For all workers examined as a group, nonstatistically significant elevations were observed for cancer of the pancreas (SMR=1.36, 34 observed, 95 percent CI=0.94-1.90), skin cancer (SMR)=1.07, 11 observed, 95 percent CI=0.54-1.92), breast cancer (females only, SMR=1.21, 11 observed, 95 percent CI=0.60-2.17), prostate cancer (SMR)=1.31, 36 observed, 95 percent CI=0.91-1.81), kidney cancer (SMR=1.30, 16 observed, 95 percent CI=0.74-2.11), brain cancer (SMR=1.29, 20 observed 95 percent CI=0.79-2.00), cancers of other lymphatic tissues (SMR=1.32, 22 observed, 95 percent CI=0.82-1.99) and diseases of the blood forming organs (SMR=1.23, 6 observed, 95 percent CI=0.45-2.68). The SMR for lung cancer was statistically significant (SMR=1.17, 202 observed; 95 percent Cl 1.01-1.34), particularly in the white male segment of the population (SMR=1.20, 194 observed 95 percent CI=1.04-1.38). Examination of the lung cancer mortality by year of hire, latency, duration of employment and calendar year at risk indicated the excess was confined to those who were first hired before 1954 (SMR=1.27, 161 observed), and was greatest in persons employed 5 to 20 years with 10 to 30 years of follow-up. Elevated lung cancer deaths rates were first evident between 1955 and 1964 and continued to increase from 1975 to 1979, followed by a decrease in lung cancer death rates.

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Between 1953 and 1963, Y-12 used mercury in a process to produce large quantities of enriched lithium. Cragle et al. studied all workers employed at Y-12 at least 5 months between January 1, 1953 and April 30, 1958 (N=5,663) (JOM 1984a:817-821). This group was categorized into workers exposed to mercury and workers not exposed to mercury based on results of urinalysis data supplied by the plant. Vital status follow-up was complete through the end of 1978, and SMRs were calculated. Compared with non-exposed workers, there were no differences in the mortality patterns for mercury exposed workers as a whole, workers with the highest mercury exposures, and workers employed more than a year in a mercury process. The authors acknowledge that mortality is not the optimal end point to assess health effects related to mercury exposure.

The mercury workers were involved in a clinical study by Albers et al. who examined 502 Y-12 workers, 247 of whom worked in the mercury process 20 to 35 years prior to the examination (AN 1988a:651-659). Correlations between declining neurological function and increasing exposure were identified. An exposure assessment was determined for each mercury worker during the time of employment in the mercury process. Study subjects who had at least one urinalysis equal or greater than 0.6 mg./L of mercury showed decreased strength, coordination and sensations along with increased tremor, and prevalence of Babinski and snout reflexes when compared with the 255 unexposed workers. Clinical polyneuropathy was associated with the level, of the highest exposure, but not with the duration of exposure.

K-25 Site. The K-25 Site enriched uranium beginning in 1945 using a gaseous diffusion process. There was potential exposure to uranium dust, oxidized uranium compounds, uranium hexafluoride, and a number of chemical compounds used in the process. In later years of operation, the gas centrifuge process was used to enrich uranium. No analyses of death rates for this population have been published; however, health effects have been studied.

Powdered nickel was used at K-25 in the production of the barrier material used to separate and enrich uranium. Workers who fabricated the barrier material were exposed to nickel powder through inhalation. Cragle et al. updated an earlier study by Godbold et al. of 814 workers who were employed in the manufacture of barrier material between 1948 and 1953 (JOM 1979a:799-806); (IARC 1984a:57-63). A comparison group of white males employed at K-25 sometime between 1948 and 1953 (N=7,552) was also selected. The SMRs in the barrier group were similar to those in the non-barrier worker group for most non-cancer outcomes. The nickel workers were noted to have a higher rate of death from cancers of the buccal cavity and pharynx (SMR=2.92, 3 observed, 95 percent CI=0.59-8.54) than the non-nickel workers (SMR=0.23, 3 observed, 95 percent CI=0.05-0.67). When the standardized rates were directly compared, the rate of buccal cavity and pharynx cancer in the nickel workers was approximately 19 times higher than the rate in the non-nickel workers. The authors acknowledge that the number of cases is quite small and recommended additional follow-up to determine if this trend continued. There were no nasal sinus cancers observed in the worker population exposed to metallic nickel in contrast to the results of studies of workers in nickel refineries where the rates of sinus cancer related to nickel compounds are quite high.

K-25 workers employed in the gas centrifuge process were the focus of an interview study by Cragle et al. (AOEH 1992a:826-834). The study was conducted in order to determine the incidence rate for cancer and illness symptoms among workers exposed to epoxy resin and solvents prevalent in the process. A total of 263 workers determined to have worked closest and longest to the process were compared with 271 employees employed at the plant during the same time, but did not work in the centrifuge process. The centrifuge workers and the non-centrifuge workers had similar overall cancer incidence rates. However, the centrifuge workers also reported five incident bladder cancers versus none reported by the non-centrifuge group. The centrifuge workers also reported significantly more rashes, dizziness, and numb or tingling limbs during employment, which are symptoms associated with high solvent exposure. One of the epoxy resins used in the early years of the process was a potential bladder cancingen, but none of the workers with bladder cancer had jobs that required routine, hands-on work with that material. A specific causative agent for the increase in bladder cancer was not identified.

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Combined-Oak Ridge Reservation Facilities. Frome et al. reported on the mortality experience of World War II workers employed at three ORR facilities between 1943 and 1947 (RR 1990a:138-152). Poisson regression analyses were used to control for potential confounders such as facility of employment, socioeconomic status, period of follow-up, and birth year. The cohort included white males employed at any ORR facility at least 30 days between the start of the operation and 1947 and were never employed at an ORR facility after 1947 (N=28,008). Elevated mortality was statistically significant for all causes (SMR=1.11, 11,671 observed, 10,537 expected; standard deviation (sd)=1), tuberculosis (SMR=1.37, 108 observed, 78 expected; sd=10.2), mental, psychoneurotic, and personality disorders (SMR=1.60, 81 observed, 50 expected; sd=10.2), cerebrovascular disease (SMR 1.11, 833 observed, 753 expected; sd=3.9), diseases of the respiratory system (SMR=1.25, 792 observed, 634 expected; sd=4.4), emphysema (SMR=1.24, 209 observed, 168 expected; sd=8.4), all accidents (SMR=1.28, 694 observed, 542 expected, sd=3.8), and motor vehicle accidents (SMR=1.44, 339 observed, 235 expected; sd=5.5). The only elevated site specific cancer that was statistically significant was lung cancer (SMR=1.27, 850 observed, 667 expected, sd=4.4, p<0.01). A surrogate for radiation exposure based on a workers job and department was used to indicate the probability of exposure. This surrogate for actual radiation exposure was not associated with increased rates of cancer.

Carpenter investigated earlier reports of an association between brain cancer and employment at Y-12 by conducting a case-control study of workers employed between 1943 and 1977 at ORNL or Y-12 (JOM 1987a:601-604). Cases consisted of 72 white males and 17 white females with brain cancer. Four controls were selected for each case matched on age, sex, cohort, year of birth, and year of hire. Analyses with respect to internal and external radiation exposures indicated no association with brain cancer. Two companion papers were also published from this case-control study, one examined relationships between brain cancer and chemical exposures, and the other examined non-occupational risk factors (AJIM 1988a:351-362); (AJPH 1987a:1180-1182). No statistically significant association between the use of 26 chemicals evaluated and the risk of brain cancer was observed. The chemicals evaluated included those encountered in welding fumes, beryllium, mercury, 4,4-methylene is 2-chloroaniline or MOCA, cutting oils, thorium, methylene chloride, and other solvents. Excess brain cancer was observed among individuals employed for more than 20 years (odds ratio=7.0, 9 cases; 95 percent Cl 1.2-41.1). Analysis of 82 cases with complete medical records revealed an association with a previous diagnosis of epilepsy (odds ratio=5.7, 4 cases; 95 percent. CI=1.0-32.1) recorded for pre-employment and health status follow-up.

Causes of death among white male welders (N=1,059) employed between 1943 and 1973 at Y-12, the K-25 Site, and ORNL were studied by Polednak (AEH 198la:235-242). Based on deaths reported through 1974, mortality from all causes for welders was slightly lower than that expected based on death rates for U.S. white males (SMR=0.87, 173 observed, 199 expected, 95 percent CI=0.75-1.01). Non-statistically significant decreases in mortality were also observed for all cancers (SMR=0.88, 32 observed, 36.57 expected, 95 percent CI=0.60-1.23), especially digestive cancer (SMR=0.49, 5 observed, 10.3 expected, 95 percent CI=0.16-1.14); diseases of the circulatory system (SMR=0.74, 72 observed, 97.51 expected, 95 percent CI=0.58-0.94); diseases of the digestive system (SMR=0.76, 9 observed, 11.86 expected 95 percent, CI=0.35-1.4), and accidents (SMR=0.89, 16 observed, 17.86 expected, 95 percent CI=0.51-1.44). Non-statistically significant increases were noted for lung cancer (SMR=1.50, 17 observed, 11.37 expected, 95 percent CI=0.87-2.40); diseases of the respiratory system (SMR=1.33, 13 observed, 9.77 expected, 95 percent CI=0.71-2.27), especially emphysema (SMR=2.21, 6 observed, 2.71 expected, 95 percent CI=0.81-4.82); and suicide (SMR=1.64, 10 observed, 6.09 expected; 95 percent CI=0.79 - 3.02). A subgroup of welders (N=536) exposed to nickel oxides (possible respiratory carcinogens) at K-25 were compared with welders at the other two facilities (N=523). The risk of lung cancer and other respiratory diseases did not differ between the two groups.

Combined Nuclear Sites. ORR workers have been included in several studies that have examined occupational risks across the nuclear complex, both in the United States and internationally. These combined studies have been undertaken in an attempt to increase the statistical power of the studies to detect the effects of low-level chronic radiation exposure.

Health and Safety

Y-12 workers were included in a lung cancer case-control study of workers from the Fernald Feed Materials and Production Center cohort and the Mallinckrodt Chemical Works cohort. Dupree et al. conducted a nested case-control study of lung cancer (N=787) to investigate the relationship between lung cancer and uranium dust exposure (Epidemiology 1995a:370-375). Eligible cases were employed at least 183 days in any of the facilities and died before January 1, 1983, with lung cancer listed anywhere on the death certificate. Inclusion of deaths through 1982 allowed over 30 years of observation at each facility. One control was matched to each case on facility, race, gender, and birth and hire dates within three years. Data collected on all study members included smoking history, first pay code (a surrogate for socioeconomic status), complete work histories and occupational radiation monitoring records. Annual radiation lung dose from deposited uranium was estimated for each study member. Annual external whole body doses from gamma radiation were determined for workers who had personal monitoring data available. Potential confounders considered in the analysis were smoking (ever/never used tobacco) and pay code (monthly/non-monthly). With a 10-year lag, cumulative lung doses ranged from 1 to 137 centigays (cGy) for cases and from 0 to.80 cGy for controls. The odds ratios for lung cancer mortality for seven cumulative internal dose groups did not demonstrate increasing risk with increasing dose. An odds ratio of 2.0 was estimated for those exposed to 25 cGy or more, but the 95 percent confidence interval of 0.20 to 20 showed great uncertainty in the estimate. There was a suggestion of an exposure effect for workers hired at age 45 years or older.

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A combined site mortality study included workers from ORNL, the Hanford Site and the Rocky Flats Plant (RR 1993a:408-421). Earlier analyses of these cohorts by Gilbert et al. indicated that risk estimates calculated through extrapolation from high-dose data to low-dose data did not seriously underestimate risks of exposure to low-dose radiation (AJE 1990a:917-927; RR 1989a:19-35). The updated analyses were performed in order to determine whether the extrapolated risks represented an over-estimation of the true risk at low doses. The study population consisted of white males employed at one of the three facilities for at least six months and monitored for external radiation. The Hanford population also included females and non-white workers. The total population dose was 1,237 Sv. Analyses included trend tests for site specific cancer deaths and several broad non-cancer categories. Statistically significant trends were noted for cancer of the esophagus, cancer of the larynx, and Hodgkin's disease. These cancers were not related to radiation exposure levels in previously published studies. Excess relative risk models were calculated for the combined DOE populations and for each DOE site separately. Without exception, all risk estimates included the possibility of zero risk (that is, the confidence interval for the risk coefficient went from below zero to above zero). There was evidence of an increase in the excess relative risk for cancer with increasing age in the Hanford and ORNL population; both populations showed significant correlations of all cancer with radiation dose among those 75 years and older.

An international effort to pool data from populations exposed to external radiation included the ORNL population in addition to other radiation worker populations in the United States, Canada, and Britain (RR 1995a:117-132). The cohort comprised 95,673 workers (85.4 percent men) employed 6 months or longer and the population dose was 3,843.2 Sv. There was no evidence of an association between radiation dose and mortality from all causes or from all cancers. There was a significant dose-response relationship with leukemia, excluding chronic lymphocytic leukemia (excess relative risk=2.18 per SV, 90 percent CI=0.1-5.7) and multiple melanoma (excess relative risk not computed; 44 observed). The study results do not suggest that current radiation risk estimates for cancer at low levels of exposure are appreciable in error.

Memorandum of Understanding. The Department entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites. NIOSH is responsible for the conduct or management of worker studies.

The following studies at the ORR are managed by NIOSH with funding from DOE: a study of multiple myeloma among workers at the K-25 Plant at Oak Ridge (expected completion date 1996); a multisite study to assess the potential association between paternal exposure to ionizing radiation and the risk of leukemia in offspring of exposed male workers; a study of neurologic health outcomes in workers exposed to high levels of mercury between 1953 and 1963; studies of mortality among Oak Ridge workers; a multisite study of mortality

among female nuclear workers; a multi-site exposure assessment of hazardous waste/cleanup workers; a chronic beryllium disease study; and a multi-site study of heat stress and performance among carpenters.

M.4.7 SAVANNAH RIVER SITE

The Savannah River Site, established in 1953 in Aiken, SC, produced Pu, tritium, and other nuclear materials. There are reports that millions of curies of tritium have been released over the years both in plant exhaust plumes and in surface and groundwater streams (ED 1982a:135-152).

Surrounding Communities. In 1984, Sauer and Associates examined mortality rates in Georgia and South Carolina by distance from the Savannah River Plant (now known as the SRS) (SR duPont 1984b). Rates for areas near the plant were compared with U.S. rates and with rates for counties located more than 50 miles away. Breast cancer, respiratory cancer, leukemia, thyroid cancer, bone cancer, malignant melanoma of the skin, non-respiratory cancer, congenital anomalies or birth defects, early infancy death rates, stroke, or cardiovascular disease in the populations living near the plant did not show any excess risk compared with the reference populations.

State Health Agreement Program. Under the State Health Agreement program managed by the DOE Office of Epidemiologic Studies, a grant was awarded to the Medical University of South Carolina in 1991 to develop the Savannah River Region Health Information System. The purpose of the Savannah River Region Health Information System database was to assess the health of populations surrounding SRS by tracking cancer rates and, birth defect rates in the area. Information from the registry is available to public and private health care providers for use in evaluating cancer control efforts. A steering committee provides advice to the Savannah River Region Health Information System and communicates public concerns to Savannah River Region Health Information System. It consists of 12-community members and persons with technical expertise representing South Carolina and Georgia. The meetings are open to the public.

Workers. A descriptive mortality study was conducted that included 9,860 white male workers who had been employed at least 90 days at the Savannah River Plant between 1952 and the end of 1974 (AJIM 1988b:370-401). Vital status was followed through the end of 1980 and mortality was compared with the U.S. population. SMRs were computed separately for hourly and salaried employees. For hourly employees non-statistically significant increases were seen for cancer of the rectum (SMR=1.09, 5 observed, 95 percent CI=0.35-2.54), cancer of the pancreas (SMR=1.08, 10 observed, 95 percent CI=0.59-2.18), leukemia and aleukemia (SMR=1.63, 13 observed, 95 percent CI=0.87-2.80), other lymphatic tissue (SMR=1.06, 5 observed, 95 percent CI=0.34-2.48), benign neoplasms (SMR=1.33, 4 observed, 95 percent CI=0.36-3.40), and motor vehicle accidents (SMR=1.10, 63 observed, 95 percent CI=0.84-1.4). Salaried employees exhibited non-statistically significant increases in cancer of the liver (SMR=1.84, 3 observed, 95 percent CI=0.38-5.38), cancer of the prostate (SMR=1.35, 5 observed, 95 percent CI=0.44-3.16), cancer of the bladder-(SMR=1.87, 4 observed, 95 percent CI=0.51 4.79), brain cancer (SMR=1.06, 4,observed, 95 percent CI=0.29-2.72), leukemia and aleukemia (SMR=1.05, 4 observed, 95 percent CI=0.29-2.69), and other lymphatic tissue (SMR=1.23, 3 observed, 95 percent CI=0.26-3.61). No trends between increasing duration of employment and SMRs were observed. A statistically significant excess of leukemia deaths was observed for hourly workers employed at least 5, but less than 15 years (SMR=2.75, 6 observed, 95 percent CI=1.01-5.99). Review of the plant records and job duties of the workers who died from leukemia indicated that two of the cases had potential routine exposure to solvents, four had potential occasional exposure to solvents and one had potential for minimal exposure. Benzene, a known carcinogen was reportedly not used at the plant.

Epidemiologic Studies. The Department's Office of Epidemiologic Studies has implemented an Epidemiologic Surveillance Program at SRS to monitor the health of current workers. This program will evaluate the occurrence of illness and injury in the workforce on a continuing basis and the results will be issued in annual reports. The implementation of this program will facilitate an ongoing assessment of the health and safety of SRS's workforce and will help identify emerging health issues.

Currently operational at a number of DOE sites, including production sites and R&D facilities, epidemiologic surveillance uses routinely collected health data including descriptions of illness resulting in absences lasting 5 or more consecutive workdays, disabilities, and OSHA recordable injuries and illnesses abstracted from the OSHA 200 log. These health event data, coupled with demographic data about the active workforce at the participating sites, are analyzed to evaluate whether particular occupational groups are at increased risk of disease or injury when compared with other workers at a site. As the program continues and data for an extended period of time become available, time trend analysis will become an increasingly important part of the evaluation of worker health. Monitoring the health of the workforce provides a baseline determination of the illness and injury experience of workers and a tool for monitoring the effects of changes made to improve the safety and health of workers. Noteworthy changes in the health of the workforce may indicate the need for more detailed study or increased health and safety measures to ensure adequate protection for workers.

Memorandum of Understanding. The Department entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites. The Centers for Disease Control and Prevention's National Center for Environmental Health is responsible for dose reconstruction studies and NIOSH is responsible for worker studies. These activities are funded by DOE.

A study of mortality among SRS workers employed from 1952 to 1974 to examine whether risks of death due to selected causes may be related to occupational exposures at SRS is being conducted by NIOSH. SRS is also included in several multisite studies managed by NIOSH. The first study is to assess the potential association between paternal work-related exposure to ionizing radiation and the risk of leukemia in offspring of exposed male workers. The second study is to examine causes of death among female workers at nuclear weapons facilities to develop risk estimates based on exposures to external and internal ionizing radiation and to hazardous chemicals. A third multi-site project is a case-control study of multiple myeloma; a type of blood cell cancer.

A dose reconstruction project around SRS is being conducted by the National Center for Environmental Health to determine the type and amount of contaminants to which people living around the site may have been exposed, to identify exposure pathways of concern and to quantify the doses people may have received as a result of SRS operations. The estimated completion date is 1999 or 2000.

M.4.8 ROCKY FLATS ENVIRONMENTAL TECHNOLOGY SITE

Surrounding Communities

Johnson examined cancer incidence from 1969 to 1971 among non-Hispanic whites in the Denver area to determine if exposure to a small concentration of Pu and other radionuclides had increased the incidence of cancer (Ambio 1981a:176-182). The authors categorized census tracts into four areas based on Pu isotope concentrations in soil from a 1970 Atomic Energy Commission survey. The highest concentrations of Pu were closest to the Rocky Flats Plant (Area I) and decreased with distance from the plant (Areas II & III). Area IV was considered unexposed. The study calculated cancer incidence rates for each of the four areas. To account for confounding factors, median income and education levels of the study and control populations derived from 1970 census data also were considered.

Cancer incidence appeared to be inversely proportional to the distance from Rocky Flats. Among males, total cancer incidence for 1969-1971 was significantly elevated by 24 percent in Area I and by 15 percent in Area II compared with Area IV. Among women, total cancer incidence was 10 percent higher in Area I than Area IV. When specific cancer sites for each area were compared with Area IV, cancer of the lung and bronchus was significantly elevated in men by 33 percent in Area I and by 46 percent in Area II. Cancers of the colon and rectum were significantly elevated in men by 47 percent and in women by 37 percent in Area I. Leukemia was significantly elevated in women in Area III by 58 percent. There were 18 percent fewer leukemia cases than expected among women in Area I. Cancer of the tongue, pharynx, and esophagus was significantly elevated in

men by 139 percent and in women by 257 percent in Area II. Men in Area II also had significant elevations in liver, gallbladder, and testicular cancers. The author concluded that over the study period, cancer incidence increased with increasing Pu soil concentrations and that exposure of the public to low concentrations of Pu and other radionuclides may effect the incidence of total and radiation-related cancer.

To further investigate these reported geographic correlations between Rocky Flats and cancer incidence patterns, Crump et al. re-examined cancer incidence data for the 3-year period studied by Johnson, 1969 to 1971, and also analyzed data from a later period, 1979-1981 (AJE 1987b:127-135). Crump et al. used the same concentration contours for soil Pu contamination as the Johnson study and computed gender- and age-specific cancer rates for each of the four exposure areas. As in the previous study, Area I, with the highest exposure, was closest to Rocky Flats; Area IV was most distant.

The authors' findings paralleled the earlier results of Johnson for 1969-1971. For 1979-1981, significant positive trends were observed in males for total cancer, "radiosensitive cancer," as defined by BEIR III, and respiratory cancer; and in females for total cancer, radiosensitive cancer, and digestive cancer. Whereas cancer incidence tended to decrease with increasing distance from the Rocky Flats Plant for the 1969-1971 study period, cancer incidence in the 1979-1981 time period was lower in Area I, closer to Rocky Flats, than for Area II in six of the cancer sites studied.

To examine the possible effects of urbanization on cancer incidence, census tracts were then grouped by distance from the State Capitol Building. Total cancer incidence was higher nearer to downtown Denver and the State Capitol for males in the earlier period, and for both sexes in the later period. Cancer incidence rates were found to decrease in all directions as one moved away from the State Capitol, including the direction of Rocky Flats. After controlling for distance from the Capitol, the statistically significant association of increases in various cancers among those living near Rocky Flats disappeared.

The authors then compared that part of Area I closest to Rocky Flats (within 16 km) to the whole Denver metropolitan area. No excess was seen for either males or females during either study period for total cancer, radiosensitive cancer, or respiratory cancer.

Crump et al. concluded that this study did not support a correlation between environmental exposure to Pu from Rocky Flats and cancer incidence; correlations of cancer incidence with proximity to Rocky Flats disappeared for both time periods when analyses took into account the levels of urbanization.

Jablon et al. analyzed cause, gender, race, and age-specific cancer mortality from 1950 through 1984 in residents who lived near 62 nuclear facilities throughout the United States (JAMA 1991a:1403-1408). Each of 107 counties with or near nuclear installations, including Jefferson County, where Rocky Flats is located, was matched to three control counties in the same region, without nuclear facilities. The matching criteria included population size, age, race, urban/rural differences, manufacturing, education, income, migration, and infant death rates. The authors concluded that the survey did not detect any general association between residence in a county with a nuclear facility and death attributable to leukemia or any other form of cancer. The authors noted that interpretation of the study results is limited by its ecologic approach in which the exposures of individuals are not known.

State Health Agreement Program

Under the State Health Agreement program managed by the DOE Office of Epidemiologic Studies, a grant was awarded to the Colorado Department of Public Health and Environment for the performance of an offsite historical dose reconstruction. Due to be completed by 1998, this study includes a thorough examination of major fires and other events releasing Pu from Rocky Flats, estimates of resulting risks due to exposure to Pu and other environmental releases, as well as extensive support of cancer and birth defects registries and public involvement activities.

Workers

Voelz et al. conducted a historical cohort mortality study of 7,112 white male workers ever employed at Rocky Flats between 1952 and 1979 (HP 1983b:493-503).

Cause specific death rates in workers were compared with those in the U.S. white male population adjusted for age and calendar year. Statistically significant fewer deaths were observed than expected based on rates in the U.S. population from all causes (SMR=0.54, 95 percent CI=0.49-0.60), all cancers (SMR=0.64, 95 percent CI=0.52 0.77), digestive organs/peritoneal cancer (SMR=0.66, 95 percent CI=0.44-0.95), and respiratory cancer (SMR=0.58, 95 percent CI=0.40-0.80). Benign and unspecified neoplasms were the only cause of death significantly elevated in these workers with eight cases observed, (SMR=3.32, 95 percent CI=1.43-6.53). All eight tumors were intracranial. The cohort was then stratified by exposure to Pu. Among Pu exposed workers, significantly fewer deaths than expected were observed from all causes of death (SMR=0.38, 95 percent CI=0.31-0.46), all malignant neoplasms (SMR=0.41, 95 percent CI=0.23-0.59), and respiratory cancer (SMR=0.20, 95 percent CI=0.05-0.52). No cases of bone cancer were observed. Workers not exposed to Pu also showed significantly fewer deaths from all causes and all cancers.

Workers exposed to external radiation had significantly fewer deaths from all causes (SMR=0.49, 95 percent CI=0.43-0.54), all cancers (SMR=0.58, 95 percent CI=0.46-0.73), and respiratory cancer (SMR=0.57, 95 percent CI=0.37-0.83) than expected when compared with U.S. white males. Six of the total eight cases of benign and unspecified neoplasms occurred in workers exposed to external radiation. Two occurred in those exposed to Pu.

To investigate whether brain tumor mortality was associated with exposure to internally deposited Pu or external radiation, Reyes et al. conducted a case-control study of all primary brain tumor deaths that occurred among white males who had been employed at Rocky Flats between 1952 and 1977, and died between 1952 and 1980 (JOM 1984b:721-725). Sixteen cases were identified. Four controls were matched to each case on year of birth and period of employment. Demographic data and detailed work histories were obtained from employment records. Exposure data for internally deposited Pu and external radiation data were obtained from Rocky Flats Plant health physics records.

No statistically significant association was found between brain tumor mortality and exposure to Pu or cumulative external radiation exposure. No significant dose response trends were observed for any job or work area. The authors noted that the study was limited by the small number of cases in the study and the small proportion (10 percent) of the cohort who had died.

Tietjen presented mortality data on all causes of death, all cancer deaths, and lung cancer deaths for Rocky Flats workers with exposures greater than 74 Bq (2.0 nanocuries [nCi]) (HP 1987a:625-628). No excess mortality was observed, with fewer deaths from all causes (SMR=0.70, 95 percent CI=0.54-0.89) and lung cancer (SMR=0.14, 95 percent CI=0.0-0.76) than expected compared with U.S. rates. When compared to an internal comparison group, the Risk Ratio (RR) for mortality from all causes was 1.16 (95 percent CI=0.89-1.52) and for lung cancer it was 0.21 (95 percent CI=0.03-1.26).

To further elucidate the risks from exposure to low levels of Pu and external radiation, Wilkinson et al. studied the cohort of workers employed at Rocky Flats between 1952 and 1979 (AJE 1987d:231-250). The analyses were limited to 5,413 white males who were employed for at least 2 years at Rocky Flats. Workers with cumulative exposures >1 rem were considered exposed to external radiation; those with body burdens ≥ 2 nCi were considered exposed to Pu.

Compared with death rates among white males in the United States, significantly fewer deaths were observed than expected from all causes (SMR=0.62, 90 percent CI=0.57-0.68), all cancers (SMR=0.71, 90 percent CI=0.59-0.84), diseases of the circulatory system (SMR=0.61, 90 percent CI=0.54-0.69), accidents, poisonings,

and violence (SMR=0.65, 90 percent CI=0.51-0.81). As reported earlier by Voelz et al., benign and unspecified neoplasms were the only cause of death significantly elevated (SMR=3.16, 90 percent CI=1.77-7.07). Workers with Pu body burdens of ≥ 2 nCi were then compared with workers with less exposure. As cancers take varying times to develop, analyses were conducted considering induction periods of 2, 5, and 10 years. No significant excesses were seen for a 2-year induction period. After a 5-year induction period, significant increases from all causes (RR=1.33, 90 percent CI=1.05-1.68) and lymphopoietic cancer (RR=9.86, 90 percent CI=1.26-94.03) were observed. After 10 years, the excess in death from leukemia was no longer statistically significant (RR=5.22, 90 percent CI=0.57-38.8).

Similar analyses were conducted for workers who received 1 rem or more of external radiation compared with workers less exposed. Workers with ≥ 1 rem had statistically significant fewer deaths from all cancers, when compared with those with <1 rem. No dose-response relationships for Pu or external radiation were found. The authors noted nonstatistically significant increases in myeloid leukemia, lymphosarcoma and reticulum cell sarcoma, liver neoplasms, and unspecified brain tumors in workers with ≥ 1 rem of external radiation compared with workers with <1 rem.

Gilbert et al. conducted a combined cohort mortality analysis of white male workers employed at Hanford, Oak Ridge, or Rocky Flats for at least 6 months and monitored for exposure to external radiation (RR 1989a:19-35). Analyses for Rocky Flats were based on the same vital status and cause of death information reported by Wilkinson et al. To eliminate overlap, those who worked at multiple facilities were included in the analysis for the facility where they first met eligibility requirements; doses accumulated at other facilities were excluded. To allow for minimum latency (the time between exposure and the diagnosis of cancer), cumulative dose was lagged 2 years for leukemia and 10 years for other cancers. Expected death rates were derived from age and calendar specific death rates for U.S. white males.

In Rocky Flats workers monitored for external radiation, significantly fewer deaths were observed than expected from all causes, lung cancer, circulatory diseases, respiratory diseases excluding pneumonia, cirrhosis, and external causes. Consistent with previous studies of this cohort, benign and unspecified neoplasms of the brain were the only cause of death significantly elevated (SMR=3.84, 95 percent CI=1.5, 7.9). Unmonitored workers had a borderline statistically significant excess mortality from all cancers (SMR=1.6, 90 percent CI=1.0-2.5) but did not differ from monitored workers with respect to site-specific cancer mortality.

Analyses of mortality by cumulative radiation dose found no indication of increased cancer deaths with increased radiation dose, but a significant positive association was observed between noncancer mortality and radiation exposure. The authors indicated that mortality from circulatory diseases and external causes were contributors to this correlation with noncancer mortality. The authors concluded that there was no evidence of a correlation between chronic low-dose radiation exposure and mortality from all cancer or from leukemia.

In 1993, Gilbert et al. published an update of their previous analyses of data from Hanford, Oak Ridge, and Rocky Flats (RR 1993a:408-421). Four additional years of mortality data for the Rocky Flats cohort were included in this later analysis. As in the previous analysis, the study was limited to white males employed for at least 6 months and monitored for external radiation. All analyses were based on internal comparisons of death rates by level of radiation dose, as internal comparisons were considered by the authors to be less subject to bias and more likely to detect risks resulting from radiation exposure than were comparisons to external populations. Workers were included in the analyses beginning with the year after initial employment plus 5 years, or the first year of monitoring, whichever occurred later.

The previously observed correlation between noncancer mortality and external radiation exposure in Rocky Flats workers was no longer statistically significant, and external causes of death were now negatively correlated with radiation dose. Benign and unspecified neoplasms of the brain, which had been shown to be elevated in previous papers by Voelz and Wilkinson, remained elevated and showed no evidence of any dose response relationship with external radiation. Deaths from leukemia indicated a positive correlation with external radiation exposure at Rocky Flats, but not at two other facilities considered in the analyses.

An international effort to pool data from populations exposed to external radiation included Rocky Flats workers, as well as workers at Hanford and Oak Ridge in the United States and other radiation worker populations in Canada and Britain (RR 1995a:117-132). The cohort was comprised of 95,673 workers employed 6 months or longer and the population dose was 3,543.2 Sv. There was no evidence of an association between radiation dose and mortality from all causes or from all cancers. There was a significant dose-response relationship with leukemia, excluding chronic lymphocytic leukemia (ERR=2.18 per Sv; 90 percent CI 0.1-5.7) and multiple myeloma (excess relative risk not computed; 44 observed). The authors concluded that the study results did not suggest that current radiation risk estimates for cancer at low levels of exposure are appreciable in error.

Epidemiologic Studies

The Department's Office of Epidemiologic Studies has implemented an epidemiologic surveillance program at Rocky Flats to monitor the health of current workers. This program will evaluate the occurrence of illness and injury in the workforce on a continuing basis and the results will be issued in annual reports. The implementation of this program will facilitate an ongoing assessment of the health and safety of Rocky Flats' workforce and will help identify emerging health issues.

Currently operational at a number of DOE sites, including production sites and R&D facilities, epidemiologic surveillance uses routinely collected health data including descriptions of illness resulting in absences lasting 5 or more consecutive workdays, disabilities, and OSHA recordable injuries and illnesses abstracted from the OSHA 200 log. These health event data, coupled with demographic data about the active workforce at the participating sites, are analyzed to evaluate whether particular occupational groups are at increased risk of disease or injury when compared with other workers at a site. As the program continues and data for an extended period of time become available, time trend analysis will become an increasingly important part of the evaluation of worker health. Monitoring the health of the workforce provides a baseline determination of the illness and injury experience of workers and a tool for monitoring the effects of changes made to improve the safety and health of workers. Noteworthy changes in the health of the workforce may indicate the need for more detailed study or increased health and safety measures to ensure adequate protection for workers.

Memorandum of Understanding

The Department entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites, and the Centers for Disease Control and Prevention became responsible for conducting dose reconstructions in several host States, including Idaho. NIOSH is responsible for worker studies. These activities are funded by DOE. A number of studies of the Rocky Flats' workforce are ongoing under the Memorandum of Understanding.

A study is currently underway by NIOSH, under a cooperative agreement with the State of Colorado, to update the Rocky Flats cohort mortality and cancer incidence among Rocky Flats workers. This study should be completed in 1997.

The University of Colorado is conducting a sentinel exposure event surveillance/evaluation at DOE sites. This study will develop a sentinel exposure event surveillance and evaluation system for exposures to chemicals and both ionizing and non-ionizing radiation in the defense nuclear industry. The pilot will start at Rocky Flats in 1997.

The National Jewish Center for Immunology and Respiratory Medicine is conducting a study of lung fibrosis in Pu workers at Rocky Flats. The goal of the study is to confirm that Pu workers are at increased risk for developing radiographic abnormalities consistent with fibrosis; to determine the relationship between Pu-239 and other radionuclide and chemical exposures and the development of lung fibrosis; and to determine the frequency of fibrosis on lung biopsies of Rocky Flats workers compared to biopsies from nonexposed individuals and to relate the clinical, physiologic, and pathologic severity to radionuclide dose.

Under a cooperative agreement with the State of Colorado, a study is being conducted of the relationship between the different types of leukemia commonly diagnosed in children and parental exposure to ionizing radiation used in medical procedures and received through occupational exposure.

M.4.9 LOS ALAMOS NATIONAL LABORATORY

Los Alamos and adjacent counties comprise a unique setting and history. The LANL for much of its existence was a closed community where most of the residents had direct economic ties to the Laboratory. Nearly all male residents and some of the female residents are employed at LANL. Medical care in Los Alamos County had been centralized at the Laboratory and a single community hospital. This is a unique, highly educated community situated adjacent to lands populated by Native Americans.

Surrounding Communities. Selected cancer mortality and incidence (newly diagnosed cancer) rates between 1950-1969, for eleven selected cancers among white males in Los Alamos County were compared with rates for the State of New Mexico, the United States five "socioeconomic and occupational" control counties, and five high education Western counties," based on U.S. Bureau of the Census information (ER 1981a:86-105). The comparisons were made to identify cancer types that were greater than expected while taking into account important factors, such as income and education, associated with cancer patterns. Six cancer types were identified that had rates greater than cancer rates for one or more of the four comparison groups; they are: cancer of the bile ducts and liver; bladder; prostate; brain and nervous system; lympho- and reticulo-sarcoma; and leukemia. Cancer rates of the prostate, bladder, and leukemia were also greater than expected.

Compared with New Mexico white males, Los Alamos County Anglo-white males show non-statistically significant excesses in cancer incidence from 1969-1974 for the stomach colon, rectum, pancreas, lung, and bladder (ER 1981a:86-105). All cancers combined show a 35 percent statistically significant excess. Los Alamos County white females show non-statistically significant excesses for cancer of the stomach, large intestine, lymphosarcoma and reticulosarcoma, and leukemia. All cancers combined show a statistically significant 40 percent excess.

In 1991, the New Mexico Department of Health initiated epidemiologic studies in response to citizen concerns about an apparent excess of brain tumors among residents of the western area neighborhood of Los Alamos County as a result of historical LANL nuclear operations. The New Mexico Department of Health conducted a descriptive study of brain cancer incidence in Los Alamos County and for 22 other sites (NM DOH 1993a). The study showed that during the mid- to late- 1980s an approximate 80 percent excess of brain cancer had occurred in Los Alamos County compared with a New Mexico reference population and national statistics. The excess incidence had disproportionately occurred among persons who were residents of the western area at the time of diagnosis or death; however, there were only three cases and they were confined to the 2-year time period, 1986 and 1987. Additional descriptive studies showed that the brain cancer rates for Los Alamos County were within the range of rates observed across New Mexico counties from 1983-1987 and 1988-1991. A review of mortality statistics for benign or unspecified neoplasms of the brain and nervous system showed no deaths from these causes in Western Area residents during 1984-1990.

Los Alamos County breast cancer incidence rates remained level but higher than New Mexico rates from 1970-1990. Reproductive and demographic factors associated with the risk of breast cancer were thought to account for the higher rates. A special study was conducted to examine the recent increase in breast cancer since 1988 (NM DOH 1994a). The New Mexico Tumor Registry concluded that the increase seen in 1988-1992 was primarily due to increased detection of early stage disease. The incidence of ovarian cancer in Los Alamos County women was elevated from the mid- 1970s to 1990. From 1986 through 1990, ovarian cancer incidence in Los Alamos County was roughly two-fold higher compared with New Mexico reference population rates. The excess ovarian cancer rate was confined to a census tract corresponding to two neighborhoods and was four to six-fold higher than that observed in the remaining Los Alamos County census tracts.

The incidence rates for melanoma (cancer of the skin) in Los Alamos County workers elevated from 1970 through 1990, with peak elevations occurring from the mid- to late-1980's. There was approximately a two-fold excess risk compared with a New Mexico State reference population. The excess melanoma incidence observed in Los Alamos County was thought to be related to the high ambient solar ultraviolet radiation intensity due to its high altitude.

A four-fold increase in thyroid cancer incidence during the late 1980s was noted in a study by Athas (NM DOH 1996a). A case-series records review was initiated to examine data relating to the detection, diagnosis, and known risk factors for thyroid cancer. All cases of thyroid cancer diagnosed among Los Alamos County residents between 1970 and 1995 were identified through the New Mexico Tumor Registry. The incidence rate for thyroid cancer in Los Alamos County was slightly higher than New Mexico rates between 1970 and the mid-1980's. There was a statistically-significant four-fold increase during the late-1980s and early 1990s compared with the State, but the rate began to decline in 1994 and 1995.

The higher than expected number of thyroid cancer cases could not be explained by changes in diagnosis of thyroid cancer among Los Alamos County residents. Additional analyses suggested that increased medical surveillance and greater access-to medical care were responsible for the recent excess in Los Alamos County. Potential risk factors for thyroid cancer including therapeutic irradiation, genetic susceptibility, occupational radiation exposure, and weight were also examined. However, the investigation did not identify a specific cause for the elevated rate of thyroid cancer in Los Alamos County.

Male Workers. A mortality study of 224 white males with the highest internal depositions of Pu 239 (10 nanocuries or more) at LANL were examined by Voelz, et al. (LANL 1985a). Followup was through April 1980; SMRs were low for all cause of death (SNM=0.56, 95 percent CI=0.40-0.75), all malignant neoplasms (SMR=0.54, 95 percent CI=0.23-1.06), compared with U.S. white males and lung cancer (SMR=20, 95 percent CI=0-110).

A cohort mortality study by Wiggs et al. examined the causes of death among 15,727 white males hired at LANL between 1943 and 1977 (HP 1994a:577-588). The purpose of the study was to determine if Pu deposition and external ionizing radiation were related to worker mortality. After nearly 30 years of followup, the LANL workforce experienced 37 percent fewer deaths from all causes, and 36 percent fewer deaths due to cancer than expected when compared with death rates for the U.S. population.

The researchers identified a subset of 3,775 workers who had been monitored for Pu exposure; of these, 303 workers were categorized as "exposed" based on a urine bioassay for Pu; the remainder were "non-exposed." One case of rare bone cancer, osteogenic sarcoma, a type of cancer related to Pu exposure in animal studies, was noted among the Pu exposed group. The overall mortality and site-specific rates of cancer did not differ significantly between the two groups of workers. A non-statistically significant increase in lung cancer among the exposed group was noted, but there was no information on cigarette use among the workers.

When researchers examined data for the 10,182 workers who were monitored for exposure to external ionizing radiation (including 245 workers exposed to Pu) they observed a dose-response relationship for cancers of the brain/central nervous system, cancer of the esophagus, and Hodgkin's disease. When the 225 Pu-exposed workers were excluded from the analysis, there was a statistically significant dose response between external ionizing radiation and kidney cancer and lymphocytic leukemia.

A special lifetime medical study was conducted on 26 of the workers who have the largest internal depositions of Pu at LANL. Voelz and Lawrence reported on the 42-year follow-up of the 26 white males who designed and built the first atomic bomb and were determined to have had a significant deposition of Pu-239 sometime in 1944 or 1945 based on job assignment, working conditions, and urine levels of Pu (HP 1991a:181-190). Their mortality experience was compared to U.S. white males adjusted for age and calendar time. The mortality rates were also compared with rates for a cohort of Los Alamos workers hired at the same time and born between the same years; no significant differences were observed for all cause mortality and all cancer mortality. One of the seven reported deaths was due to bone sarcoma, the most frequent radiation-induced cancer observed in persons with radium depositions.

Wiggs reported on 6,970 women employed at LANL at least 6 months from 1943 through 1979, with deaths determined through 1981 (LA Wiggs 1987a). The mortality rates for all causes of death combined and all cancers combined were 24 percent and 22 percent below the rate for the U.S. population. Although the overall rates are low, women occupationally exposed to ionizing radiation have elevated rates for cancer of the ovary and of the pancreas relative to those not exposed. An unusual finding was that female radiation workers experienced a statistically significant excess of death from suicide. In a special in-depth study, the suicides were compared to two control groups, deaths from other injuries and deaths from non-injuries. History of employment as a radiation worker was significantly associated with death from suicide for both comparison groups. No significant associations for duration of employment, Pu exposure, or marital status were seen (APHA 1988a).

As a result of a reported three-fold excess of malignant melanoma among laboratory workers at LLNL in California and similarities between occupational exposures and prevailing sunshine conditions at LANL and LLNL, an investigation was undertaken to assess the risk of melanoma at LANL (Lancet 1981a:712-716). Incidence data were obtained from the New Mexico Tumor Registry. No excess risk for melanoma was detected at LANL among 11,308 laboratory workers between 1969 and 1978. Six cases were identified where about 5.7 were expected (Lancet 1982a:883-884). The rate for the total cohort, Hispanic males and females, non-Hispanic males and females were not significantly different from the corresponding New Mexico rates.

A special in-depth, study of fifteen cases diagnosed through 1982 did not detect an association between melanoma and exposure to any type of external radiation as measured by film badges, neutron exposures, Pu body burden based on urine samples, or employment as a chemist or physicist (HP 1983c:587-592). However, the melanoma cases were more educated than the comparison group using the college and graduate degree as a measure of education; a finding consistent with other reports of malignant melanoma according to the authors. The numbers in this study are too small to detect any but large excesses.

Memorandum of Understanding. The Department entered into a Memorandum of Understanding with the Department of Health and Human Services to conduct health studies at DOE sites. NIOSH is responsible for managing or conducting the worker studies. The following multi-site studies that include LANL are currently underway: a study of mortality among female nuclear weapons workers; a case-control study of multiple myeloma; a leukemia study; and an exposure assessment of hazardous waste/cleanup workers.

M.5 FACILITY ACCIDENTS

M.5.1 EVALUATION METHODOLOGIES AND ASSUMPTIONS

M.5.1.1 Introduction

The potential for facility accidents and the magnitudes of their consequences are important factors in the evaluation of the storage and disposition alternatives addressed in the PEIS. The health risk issues are twofold:

- Whether accidents at any of the individual storage and disposition facilities (or reasonable combinations thereof) pose unacceptable health risks to workers or the general public.
- Whether alternative locations for storage and disposition facilities (or reasonable combinations thereof) can provide lesser public or worker health risks. These lesser risks may arise either from a greater isolation of the site from the public, or from a reduced frequency of such external accident initiators as seismic events, aircraft crashes, and so forth.

Guidance for implementation of Council on Environmental Quality regulation 40 CFR 1502.22, as amended (51 FR 15618) requires the evaluation of impacts which have low probability of occurrence but high consequences if they do occur; thus facility accidents must be addressed to the extent feasible in the PEIS. Further, public comments received during the scoping process clearly indicated the public concern with facility safety and consequent health risks, and the need to address these concerns in the decisionmaking process.

For the No Action case, potential accidents are defined in existing facility documentation, such as safety analysis reports, hazards assessment documents, NEPA documents and probabilistic risk assessments. The accidents include radiological and chemical accidents that produce high consequences but have a low likelihood of occurrence, and a spectrum of other accidents that have a higher likelihood of occurrence and lesser consequences than the high consequence accidents. The data in these documents includes accident scenarios, materials at risk, source terms (quantities of hazardous materials released to the environment) and consequences.

For new storage and disposition facilities, the identification of accident scenarios and associated data would normally be a product of safety analysis reports performed on completed facility designs. However, the conceptual design information available during the PEIS preparation is not useful for quantitative safety analyses. Accordingly, for each of the storage and disposition facilities, the accident information developed for similar existing facilities is used as a surrogate and the likelihood and consequences (which are site dependent) are recomputed for each of the storage and disposition proposed sites where a facility may be located. This calculation reflects the effects of such site parameters as population size and distribution, meteorology and distance to the site boundary.

This analysis also acknowledges, semi-quantitatively, the differences in likelihood of accident initiators at specific sites (for example, aircraft impacts, beyond evaluation basis seismic events) as well as qualitatively discussing the opportunities for risk reduction afforded by the potential incorporation of new technologies, processes or protective features in the storage and disposition facilities that will enhance public health and safety over the existing facilities. Subsequent to the PEIS, evaluation of the specific benefits achieved by such measures would be presented in the tiered, project-specific environmental impact statement for each facility. Also, for each facility, a *Hazards Analysis Document* that identifies and estimates the effects of all major hazards that have the potential to affect the environment, workers and the public would be issued in conjunction with the Conceptual Design Package. Additional accident analyses for identified major hazards would be provided in a *Preliminary Safety Analysis Report* to be issued during the period of Definitive Design (Title II) Review. A *Final Safety Analysis Report* would be prepared during the construction period and issued before testing begins

as final documented evidence that the new facility can be operated in a manner that does not present any undo risk to the health and safety of workers and the public.

In determining the potential for facility accidents and the magnitudes of their consequences, this PEIS incorporates two important concepts to the presentation of results: risk and uncertainties and conservatism.

M.5.1.1.1 Risk

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Risk is most important when presenting accident analysis results. The chance that an accident might occur during the conduct of an operation is called the probability of occurrence. An event that is certain to occur has a probability of 1 (as in 100 percent certainty). The probability of occurrence of an accident is less than one because accidents, by definition, are not certain to occur. If an accident is expected to happen once every 5 years, the frequency (and probability) of occurrence is 0.2/yr (1 occurrence + 5 years = 0.2 occurrences/yr).

Once the frequency (occurrences per year) and the consequences (for radiation effects, measured in terms of the number of latent cancer fatalities caused by the radiation exposure) of an accident are known, the risk can be determined. The risk of latent cancer fatalities per year is the product of the annual frequency of occurrence times the number of latent cancer fatalities that would result if the accident occurred. This annual risk expresses the expected number of latent cancer fatalities per year, taking account of both the annual chance that an accident might occur and the estimated consequences if it does occur.

For example, if the frequency of an accident were 0.2 occurrences/yr and the number of latent cancer fatalities resulting from the accident were 0.05, the risk would be 0.01 latent cancer fatalities/yr (0.2 occurrences/yr x 0.05 latent cancer fatalities per occurrence = 0.01 latent cancer fatalities/yr). Another way to express this risk (0.01 latent cancer fatalities/yr) is to note that if the operation subject to the accident continued for 100 years, one latent cancer fatality would be likely to occur because of accidents during that period. This is equivalent to 1 chance in 100 that a single latent cancer fatality would be caused by the accident source for each year of operation.

A frame of reference for the risks from accidents associated with storage and disposition alternatives can be developed in the same way. As an example, the risk of a latent cancer fatality from a beyond evaluation basis earthquake (the maximum radiation exposure consequence) for a hypothetical individual at the INEL site boundary from the consolidation of Pu would be approximately 2.7×10^{-11} $(1.0 \times 10^{-7} \times 2.7 \times 10^{-4})/yr$ (Table M.5.2.1.2–3). This risk can be compared with the lifetime risks of death from other accidental causes to gain a perspective. For example, the risk of dying from a motor vehicle accident is about 1 in 80. Similarly, the risk of death for the average American from fires is approximately 1 in 500, and for death from accidental poisoning, the risk is about 1 in 1,000. These comparisons are not meant to imply that risks of a latent cancer fatality caused by DOE operations are trivial, only to show how they compare with other, more common risks. Radiological risks to the general public from DOE operations are considered to be involuntary risks, as opposed to voluntary risks such as operating a motor vehicle.

M.5.1.1.2 Uncertainties and Conservatism

[Text deleted.] For routine operations, the results of monitoring actual operations provide realistic estimates of source terms, which when combined with conservative estimates of the effects of radiation, produce estimates of risk that are very unlikely to be exceeded. The effects for all alternatives have been calculated using uniform source terms and other factors, so this PEIS provides an appropriate means of comparing potential impacts on human health and the environment.

The analyses of hypothetical accidents are based on calculations that in turn are based on sequences of events and models of effects that have not occurred. The models provide estimates of the probabilities, source terms, pathways for dispersion and exposure, and the effects on human health and the environment that are as realistic

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as possible. In many cases, the probability of the accidents postulated is very low and little experience is available; thus, the consequences are uncertain. This has required the use of models or values for input that produce estimates of consequences and risks that are higher than would actually occur in order to provide conservative results. All the alternatives have been evaluated using uniform methods and data, allowing a fair comparison of all the alternatives on the same basis. [Text deleted.]

M.5.1.2 Safety Design Process

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One of the major design goals for storage and disposition facilities is to achieve a reduced risk to worker and public health and safety relative to that associated with similar operations at the existing nuclear weapons complex. Significant changes exist between storage and disposition facilities and the current facilities design criteria and safety standards, which would reduce total risk to the public. These changes include: design to current DOE structural and safety criteria; smaller throughput, batch size and inventories of certain hazardous materials; and elimination of the same hazardous materials. This would reduce potential offsite health effects if a significant accidental release were to occur.

Storage and disposition facilities would be designed to comply with current Federal, State and local laws, DOE Orders, and industrial codes and standards. This would provide a plant that is highly resistant to the effects of natural phenomena, including earthquake, flood, tornado, high wind, as well as credible events as appropriate to the site, such as fire and explosions, and man-made threats to its continuing structural integrity for containing hazardous materials. The facilities would be designed to maintain their continuing structural integrity in the event of any credible accident or event, including an aircraft crash.

The design process for the storage and disposition facilities would comply with the requirements for safety analysis and evaluation in DOE O 430.1 and Order 5480.23. These require that the safety assessment be an integral part of the design process to ensure compliance with all DOE safety criteria by the time that the facilities are constructed and in operation.

The safety analysis process begins early in conceptual design with identification of hazards having potential to produce unacceptable safety consequences to workers or the public. As the design develops, failure mode and effects analyses are performed to identify events which have the potential to release hazardous material. The kinds of events considered include equipment failure, spills, human error, fire, explosions, criticality, earthquake, electrical storms, tornado, flood, and aircraft crash. These postulated events become focal points for design changes or improvements to prevent unacceptable accidents. These analyses continue as the design progresses to assess the need for safety equipment and to assess the performance of this equipment in accident mitigation. Eventually, the safety analyses are formally documented in a safety analysis report (SAR) and, if appropriate, in a probabilistic risk assessment (PRA). The PRA documents the estimated frequency and consequence for a complete spectrum of accidents and helps to identify where design improvements could make meaningful safety improvements.

The first SAR is completed at the conclusion of conceptual design and includes identification of hazards and some limited assessment of a few enveloping evaluation basis accidents. This analysis includes deterministic safety analysis and failure modes and effects analysis of major systems. A detailed comprehensive preliminary SAR is completed by the completion of preliminary design and provides a broad assessment of the range of evaluation basis accident scenarios and the performance of equipment provided in the facility specifically for accident consequence mitigation. A limited PRA may be included in that analysis.

The SAR continues to be developed during detailed design. The safety review of this report and any supporting PRA is completed and safety issues resolved before initiation of construction of the facility. There is also a final SAR produced that includes documentation of safety-related design changes during construction and the impact of those changes on the safety assessment. It also includes the results of any safety-related research and

development that has been performed to support the safety assessment of the facility. Final approval of the final SAR is required before the facility is allowed to commence operation.

M.5.1.3 Analysis Methodology

M.5.1.3.1 Introduction

The MELCOR Accident Consequence Code System (MACCS) was used to estimate the consequences of all storage and disposition facilities for all accidents. A discussion of the MACCS computer code is provided in Section M.5.1.3.2. A detailed description of the MACCS model is available in NUREG/CR-6059, SAND92-2146. The MACCS computer code has been used for the analysis of accidents for many environmental impact statements and other safety documentations and is considered applicable for analyzing potential accidents associated with the storage and disposition of Pu and HEU.

M.5.1.3.2 MACCS Overview

MACCS models the offsite consequences of an accident that releases a plume of radioactive materials to the atmosphere. Should such an accidental release occur, the radioactive gases and aerosols in the plume would be transported by the prevailing wind while dispersing in the atmosphere. The environment would be contaminated by radioactive materials deposited from the plume and the population would be exposed to radiation. An estimation of the range and probability of the health effects induced by the radiation exposures not avoided by protective actions and the economic costs and losses that would result from the contamination of the environment are the objectives of a MACCS calculation.

There are two fundamental aspects of the organization of MACCS which are basic to its understanding: the time scale after the accident is divided into various "phases;" and the region surrounding the reactor is divided into a polar-coordinate grid.

The time scale after the accident is divided into three phases: emergency phase, intermediate phase, and longterm phase. The emergency phase begins immediately after the accident and could last up to seven days following the accident. In this period, the exposure of population to both radioactive clouds and contaminated ground is modeled. Various protective measures can be specified for this phase, including evacuation, sheltering, and dose-dependent relocation.

The intermediate phase can be used to represent a period in which evaluations are performed and decisions are made regarding the type of protective measure actions which need to be taken. In this period, the radioactive clouds are assumed to be gone and the only exposure pathways are those from the contaminated ground. The protective measure which can be taken during this period is temporary relocation.

The long-term phase represents all time subsequent to the intermediate phase. The only exposure pathways considered here are those resulting from the contaminated ground. A variety of protective measures can be taken in the long-term phase in order to reduce doses to acceptable levels: decontamination, interdiction, and condemnation of property.

The spatial grid used to represent the region is centered on the facility itself. The user specifies the number of radial divisions as well as their endpoint distances. Up to 35 of these divisions may be defined, extending out to a maximum distance of 9,999 km (6,213 mi). The angular divisions used to define the spatial grid correspond to the sixteen directions of the compass.

Since the emergency phase calculations utilize dose-response models for early fatality and early injury which are highly non-linear, it is necessary for those calculations to be performed on a finer grid than the calculations

of the intermediate and long-term phases. For this reason, the sixteen compass sectors are divided into three, five, or seven user-specified subdivisions in the calculations of the emergency phase.

The dose-to-risk conversion factors (0.0005 latent cancer fatalities/person-rem for the public and 0.0004 for the worker) used in this PEIS to relate radiation exposures to latent cancer fatalities are based on the 1990 Recommendations of the International Commission on Radiation Protection (ICRP Publication 60). These conversion factors are consistent with those used by the U.S. NRC in its rulemaking "Standards for Protection Against Radiation" (10 CFR 20). In developing these conversion factors, the International Commission on Radiological Protection reviewed many studies, including Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIR V) and Sources, Effects and Risks of Ionizing Radiation. These conversion factors fall within the range of uncertainty associated with the conversion factors that are discussed in the National Academy of Sciences NAS/NRC (1990). The conversion factors apply where the dose to an individual is less than 20 rem (20,000 millirem [mrem]) and the dose rate is less than 10 rem (10,000 mrem) per hour. At doses greater than 20 rem (20,000 mrem), the conversion factors used to relate radiation doses to latent cancer fatalities are doubled. At much higher doses, prompt effects, rather than latent cancer fatalities, may be the primary concern. Unusual accident situations that may result in high radiation doses to individuals are considered special cases.

The MACCS code was applied in a probabilistic manner using a weather bin sampling technique. Centerline doses as a function of distance were calculated for each of 150 meteorological sequence samples; the mean value of these doses and increased likelihoods of cancer fatality for the distance corresponding to the location of the maximum offsite individual (sometimes referred to as the "maximum exposed individual") at each site were reported for that individual. Doses to an uninvolved worker were calculated similarly, except that the worker would experience an increased likelihood of cancer fatality of 4.0×10^{-4} times the dose in rem for doses less than 20 rem or exposure rates less than 10 rem/hr. For larger doses, when the rate of exposure is greater than 10 rem/hr, the increased likelihood of latent cancer fatality is doubled. The estimated dose to a worker was based on a location 1,000 m (3,280 ft) from the release point.

Offsite population doses and latent cancer fatalities are calculated by MACCS using a similar methodology to that described for the maximum offsite individual. In the case of the population, each of the sampled meteorological sequences was applied to each of the 16 sectors (accounting for the frequency of occurrence of the wind blowing in that direction). Population doses are the sum of the individual doses in each sector. Once again, the mean value of the calculated population doses and latent cancer fatalities for each of these trials is reported.

M.5.1.3.3 Methodology and Techniques

The relative consequences of postulated accidents in the evaluation of each alternative are assessed in the Public and Occupational Health and Safety Sections of Section 4.2 for the storage alternatives and Section 4.3 for the disposition alternatives. The accident analysis involves less detail than a formal probabilistic risk assessment and facility safety analysis by addressing bounding accidents (relatively low probability of occurrence and high consequence) and a representative spectrum of possible operational accidents (relatively high probability of occurrence and low consequence). The technical approach for the selection of accidents is consistent with the DOE Office of NEPA Oversight *Recommendation for the Preparation of Environmental Assessments and Environmental Impact Statement* guidance, which recommends consideration of two major categories of accidents: within design basis accidents and beyond design basis accidents.

The preliminary accident analyses (conducted during the feasibility design) were performed primarily to identify those systems and structures which should be categorized as "safety class." This determination, for a particular system or structure, involves assessing whether the consequence of an accident in which that system

or structure fails exceeds some threshold exposure value. In general, the consequence assessments are very conservative to ensure that cost estimates which result from the feasibility design have a conservative basis.

In developing a range of accidents to consider, it is common to consider only those accidents that have a probability of occurrence equal to or greater than 10^{-7} per year. The accidents evaluated were selected to represent a spectrum of accident probabilities and consequences ranging from low-probability/high-consequence to high-probability/low-consequence events. However, because of the preliminary nature of the designs under consideration here, it has not been possible to assess quantitatively the probability of occurrence of all of the events addressed. The information provided does not indicate the total risk of operating the facility but does provide information identifying high risk events that could be used to differentiate safety risks among alternatives if an accident were to occur. The probabilities for the accidents described have been estimated by considering qualitatively accident probabilities from other facilities and locations. It is possible that the beyond design basis accidents included for consideration here will later be shown in tiered NEPA documentation to have probabilities of occurrence much less than 10^{-7} .

For each potential accident, information is provided on the risk and consequences to three types of receptors: (1) a worker, (2) a maximally exposed individual member of the public, and (3) the offsite population. The first receptor, a worker, is a hypothetical individual working on the site but not involved in the proposed action. This worker is assumed to be located at a point 1,000 m (3,280 ft)from the location of the accident. Although other distances closer to the accident could have been assumed, the results would be less accurate because of limitations of the MACCS computer code in modeling the effects of building and local terrain on the dispersion of the released radioactive substances. A worker that is closer than 1,000 m (3,280 ft) from the accident will generally receive a higher dose, while a worker further away would generally receive a lower dose. At some sites where the distance from the accident to the nearest site boundary is less than 1,000 m (3,280 ft), the worker is assumed to be located at the site boundary. The second receptor, a member of the public, is a hypothetical individual are intended to represent the highest risks to a member of the public. The third receptor, the offsite population, represents all members of the public located within 80 km (50 mi) of the location of the accident. The choice of 80 km (50 mi) is a common practice, although other distances could have been used.

The consequences of an accident for a worker or individual at the site boundary are expressed in terms of dose (rem) and probability of a cancer fatality if the individual is exposed to the dose. The risk of cancer fatality to the individual is the mathematical product of the probability of the accident and the consequence (probability of a cancer fatality). The consequences for the offsite population are expressed in terms of population dose (person-rem) and the number of cancer fatalities in the population within 80 km (50 mi) of the site boundary. The risk of the estimated number of cancer fatalities is the mathematical product of the number of cancer fatalities and the probability of the accident. The estimated risks are expressed either on an annual basis or on the basis of the operational campaign proposed or assumed for a storage or disposition facility, depending on the context of the information.

The MACCS model is one of a number of models that could be used for accident evaluations. The models will generally differ in their results because of the many differences in their assumptions and techniques. The MACCS model was selected because it is commonly used for SAR and EIS accident analyses, particularly for severe accident analyses. For each of the accidents selected for evaluation of an alternative, information is provided on the accident probability, dose, cancer fatalities, and risk.

M.5.1.3.4 Isotopic Spectra Used in the Storage and Disposition Accident Analyses

For each of the accidents selected for evaluation of an alternative, source term information (radionuclide release) is generated based on the total Pu release using the pertinent radionuclide spectrum for that alternative. A mixed Pu spectrum presented in Table M.5.1.3.4–1 is used for Pu storage and disposition alternatives. A

weapons grade Pu spectrum presented in Table M.5.1.3.4-2 is used for Pit Disassembly and Conversion operations. The Pu spectrum presented in Table M.5.1.3.4-3 is used for Pu conversion process operations.

Isotope	Isotopic Content (g/g Pu)	Specific Activity of Isotope (Ci/g Isotope)	Specific Activity (Ci/g Pu)
Pu-238	9.21x10 ⁻⁵	17.1	1.58x10 ⁻³
Pu-238 Pu-239	0.921	0.0621	0.0572
	0.0666	0.228	0.0152
Pu-240	5.23x10 ⁻⁴	103	0.0539
Pu-241	5.69x10 ⁻⁴	3.93x10 ⁻³	2.23x10 ⁻⁶
Pu-242		3.43	2.84x10 ⁻⁴
Am-241	8.28x10 ⁻⁵		2,04710

Table M.5.1.3.4-1.	Isotopic Distribution for a	Mixed Plutonium Release
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^a Isotopic distribution for mixed Pu aged for 60 years. Used for calculating the source terms for the accidents evaluated for Pu storage and disposition alternatives.

Note: Am=Americium.

Source: HNUS 1996a.

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Table M.5.1.3.4-2.	Isotopic Distribution for a Weapons-Grade Plutonium Release ^a

Isotope	Isotopic Content (g/g Pu)	Specific Activity of Isotope (Ci/g Isotope)	Specific Activity (Ci/g Pu)
Botope	3.29x10 ⁻⁵	17.1	5.63x10 ⁻⁴
Pu-239	0.930	0.0621	0.0578
	0.0596	0.228	0.0136
Pu-240	4.19×10^{-4}	103	0.0430
Pu-241	1.0×10^{-4}	3.93x10 ⁻³	3.93×10^{-7}
Pu-242	6.63×10^{-3}	3.43	0.0227
Am-241	0.03X10 *		510227

^a Isotopic distribution for weapons-grade Pu aged for 60 years. Used for calculating the source terms for the potential accidents evaluated for pit disassembly process operations.

Note: Am=Americium.

Source: HNUS 1996a.

Table M.5.1.3.4-3.	Isotopic Distribution for a Non-Pit (Pu Conversion) Plutonium Release
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Isotope	Isotopic Content (g/g Pu)	Specific Activity of Isotope (Ci/g Isotope)	Specific Activity (Ci/g Pu)
Pu-238	2.12x10 ⁻⁴	17.1	3.62×10^{-3}
Pu-239	0.902	0.0621	0.0560
Pu-239 Pu-240	0.0807	0.228	0.0184
	7.35×10^{-4}	103	0.0757
Pu-241	1.51x10 ⁻³	3.93x10 ⁻³	5.94x10 ⁻⁶
Pu-242	1.16x10 ⁻⁴	3.43	3.99x10 ⁻⁴
Am-241	1.16×10	3.45	

^a Isotopic distribution for non-pit (Pu conversion) Pu aged for 60 years. Used for calculating the source terms for the potential accidents evaluated for Pu conversion process operations.

Note: Am=Americium.

Source: HNUS 1996a.

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