394

Vol. 3
Appendices A-S

Chemical Stockpile Disposal Program Final Programmatic Environmental Impact Statement

January 1988

Program Executive Officer—Program Manager for
Chemical Demilitarization
Aberdeen Proving Ground, Md. 21010-5401

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
JAN 1988		**00-00-1988 to 00-00-1988**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Chemical Stockpile Disposal Program Final Programmatic Environmental Impact Statement Vol. 3	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES) **U.S. Army Chemical Materials Agency ,Aberdeen Proving Ground,MD,21010-5424**	8. PERFORMING ORGANIZATION REPORT NUMBER
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release; distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			
unclassified	**unclassified**	**unclassified**	**Same as Report (SAR)**	**731**	

ORNL DWG 86- 11962

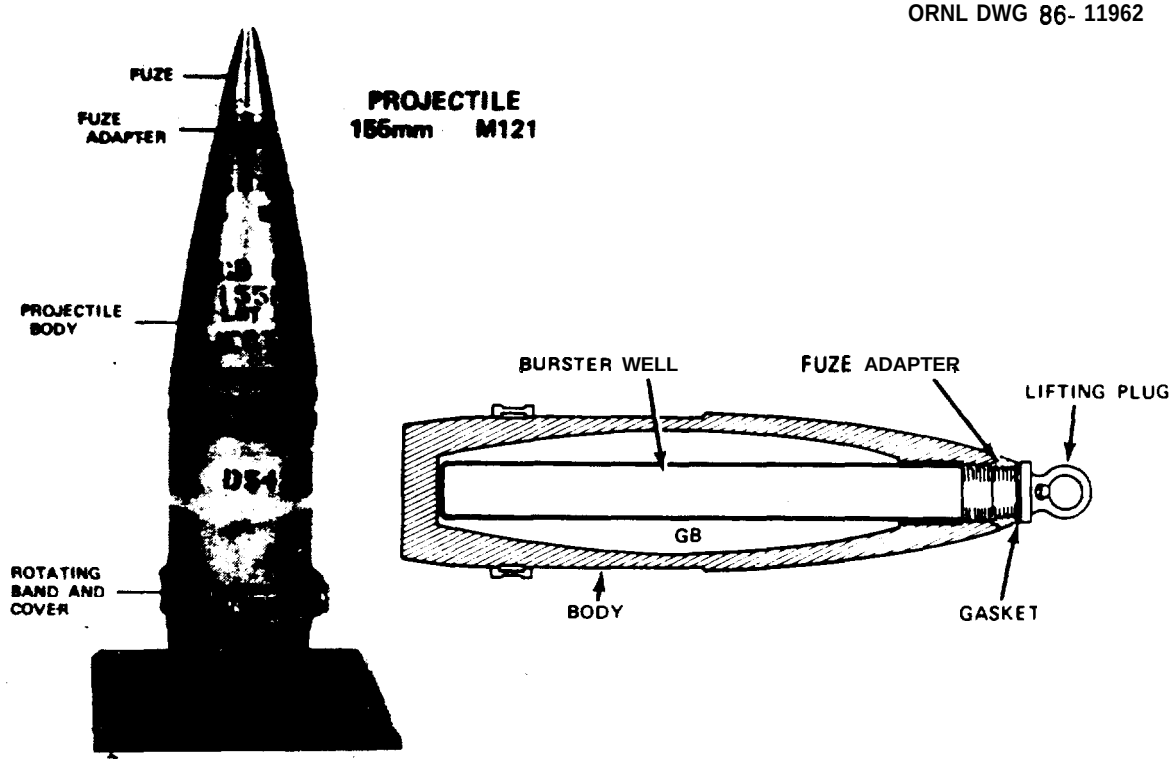


Fig. A.3. The 155-mm artillery projectiles are filled with mustard agent, GB, or VX.

ORNL DWG 86- 11963

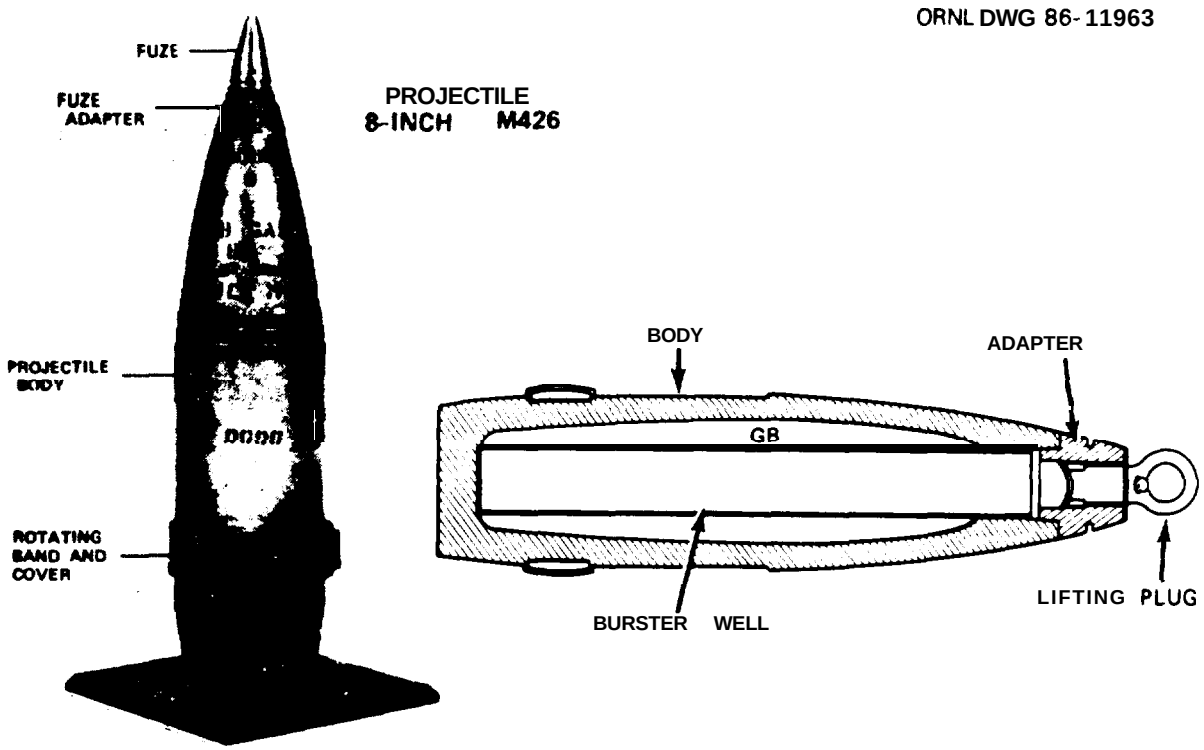


Fig. A.4. The 8-in. artillery projectiles are filled with GB or VX.

CONTENTS

Page

Appendix A. CHARACTERIZATION OF CHEMICAL STOCKPILE . . . . . A-l

Appendix B. TOXICITY OF WARFARE AGENTS AND THEIR BREAKDOWN PRODUCTS . . . . . B-l

Appendix C. INCINERATION PLANT DESIGN AND EFFLUENTS . . . . . C-l

Appendix D. BACKGROUND OF THE CHEMICAL STOCKPILE DISPOSAL PROGRAM . . . . . D-l

Appendix E. LEAKING MUNITIONS AND THE DRILL AND TRANSFER SYSTEM . . . . . E-l

Appendix F. COMPLIANCE WITH THE ENDANGERED SPECIES ACT . . . . . F-l

Appendix G. TRANSPORTATION CONCEPTS . . . . . G-l

Appendix H. SEISMICITY . . . . . H-l

Appendix I. COORDINATION WITH THE PRESIDENT'S COUNCIL ON ENVIRONMENTAL QUALITY . . . . . I-l

Appendix J. RISK ANALYSIS SUPPORTING THE FINAL ENVIRONMENTAL IMPACT STATEMENT FOR THE CHEMICAL STOCKPILE DISPOSAL PROGRAM . . . . . J-l

Appendix K. ATMOSPHERIC DISPERSION . . . . . K-l

Appendix L. GENERIC EMERGENCY RESPONSE CONCEPT PLAN . . . . . L-l

Appendix M. APPROACH FOR SELECTING THE ENVIRONMENTALLY PREFERRED ALTERNATIVE FOR THE CHEMICAL STOCKPILE DISPOSAL PROGRAM FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT . . . . . M-l

Appendix N. AQUATIC SPILL MODELING AND ANALYSIS . . . . . N-l

Appendix 0. TOXICOLOGY AND POTENTIAL IMPACTS OF CHEMICAL AGENTS TO ECOLOGICAL RESOURCES . . . . . O-l

Appendix P. CULTURAL AND SOCIOECONOMIC IMPACTS . . . . . P-l

Appendix Q. SENSITIVITY OF FATALITY ESTIMATES TO DOSE-RESPONSE VALUES, METEOROLOGICAL CONDITIONS, AND DISTRIBUTION

OF SENSTITIVE POPULATIONS IN AN AFFECTED
ENVIRONMENT . . . . . Q-l

Appendix R. EVALUATION OF COMMUNITY GROUPS' INPUT . . . . . R-l

Appendix S. OCEAN TRANSPORT OF ABERDEEN PROVING GROUND STOCKPILE:
BASIS FOR ELIMINATING THIS ALTERNATIVE . . . . . S-l

Appendix A

CHARACTERIZATION OF CHEMICAL STOCKPILE

A.1. INTRODUCTION

The chemical munitions stockpile contains a variety of chemical agents in several munitions. The exact number of munitions containing chemical agents is classified because of defense considerations, but the stockpile distribution by percent of agent tonnage is shown in Fig. A.l.

In addition, the types of chemicals and munitions stored at each location in the United States can be given. The attached map (Fig. A.2) shows this distribution throughout the country.

The lethal chemicals stored are basically three types: the persistent nerve agent VX, the nonpersistent nerve agent sarin (GB), and the persistent mustard agents H, HD, and HT. Additionally, there are very small quantities of Lewisite (L), a blister agent similar to mustard, and of GA, the first nerve agent developed, at Tooele Army Depot (TEAD).

The lethal chemical munitions in storage consist of cartridges, projectiles, land mines, and rockets. Bulk agent is maintained in a limited quantity of bombs and airborne spray tanks and in a large number of bulk one-ton containers.

The size and composition of the stockpile vary greatly at each of the storage sites. Appendix B discusses in more depth the toxicity of agents slated for disposal.

The inventories of munitions and chemical agents differ greatly from site to site. Since no two storage-site inventories are alike, the optimal disposal program must address the unique inventory at each.

A.2. TYPES OF MUNITIONS

The lethal chemical munitions that the United States now has in storage consist of the following items:

1. 155-mm artillery projectiles (see Fig. A.3). The 155-mm stock is filled with mustard, GB, or VX.

2. 8-in. artillery projectiles (see Fig. A.4). The 8-in. projectiles are filled with GB or VX.

3. 105-mm artillery projectiles and cartridges (see Fig. A.5). These rounds are filled with mustard agent or GB.

4. 4.2-in. mortar cartridges (see Fig. A.6). All are filled with mustard agent.

5. M23 land mines (see Fig. A.7). All the mines are filled with VX.

6. M55 115-mm rockets (see Fig. A.8). The rockets are filled with GB ox VX.

These items contain propellant and/or explosive components.

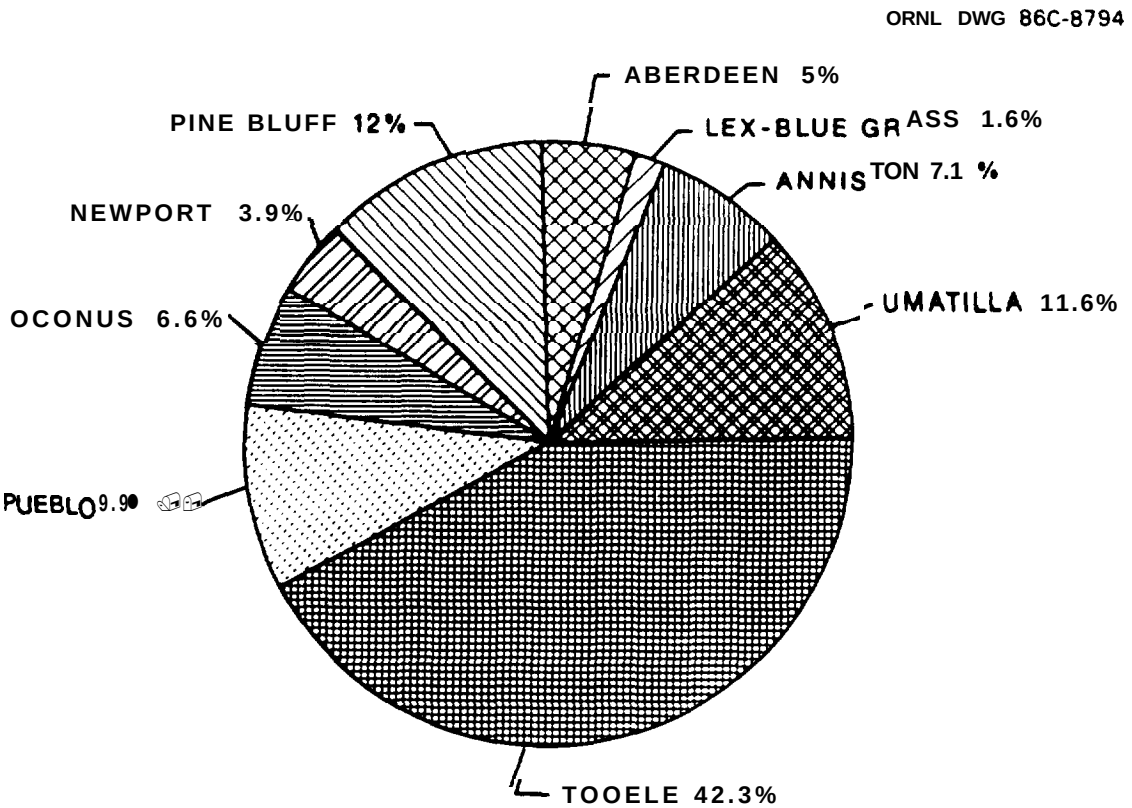


Fig. A.I. Chemical munitions stockpile distribution by percent of agent tonnage.

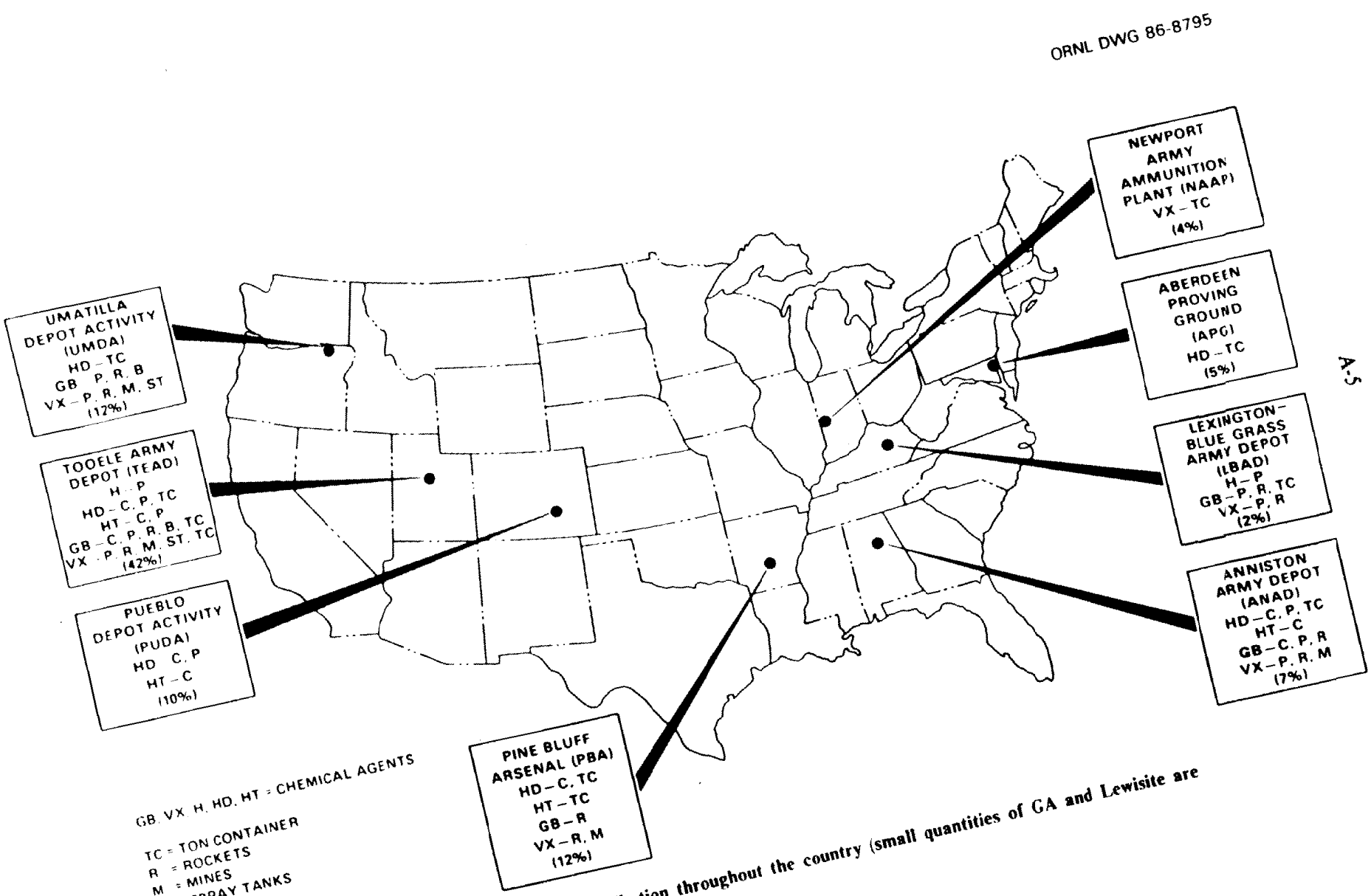


Fig. A.2. Stockpile distribution throughout the country (small quantities of GA and Lewisite are also stored at TEAD).

ORNL DWG 86-I 1962

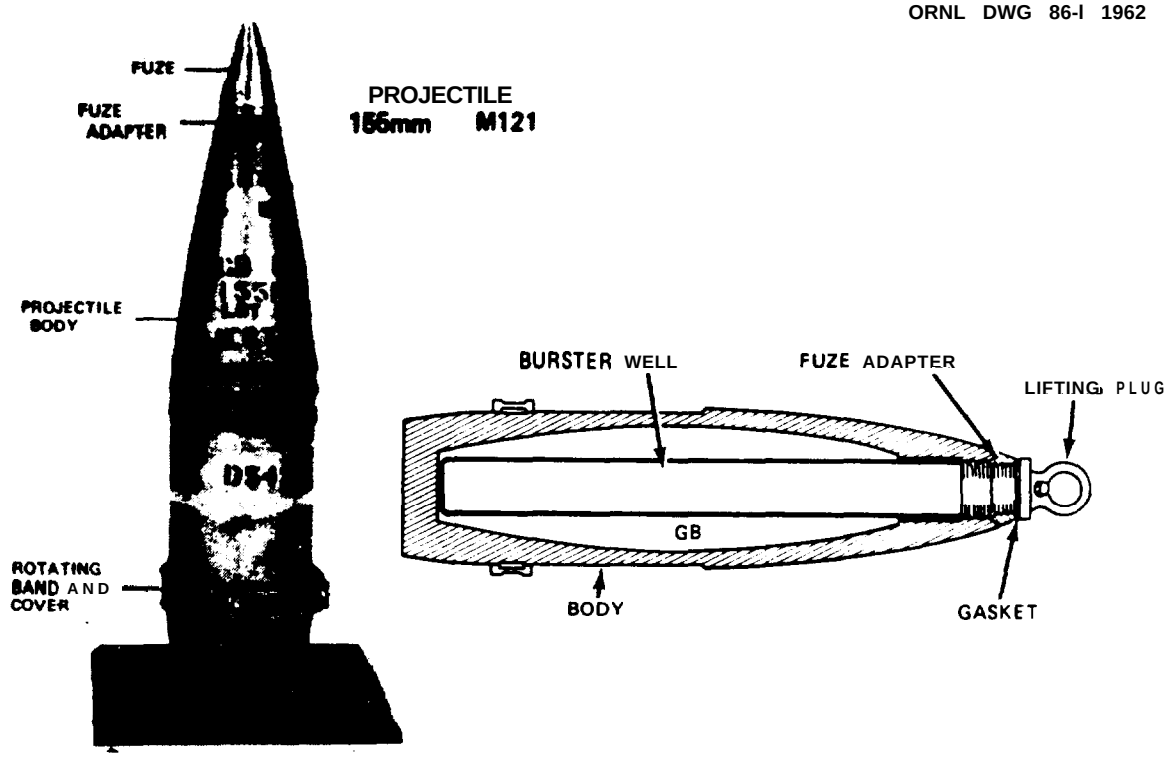


Fig. A.3. The 155-mm artillery projectiles are tilled with mustard agent, GB, or VX.

ORNL DWG 86- 11963

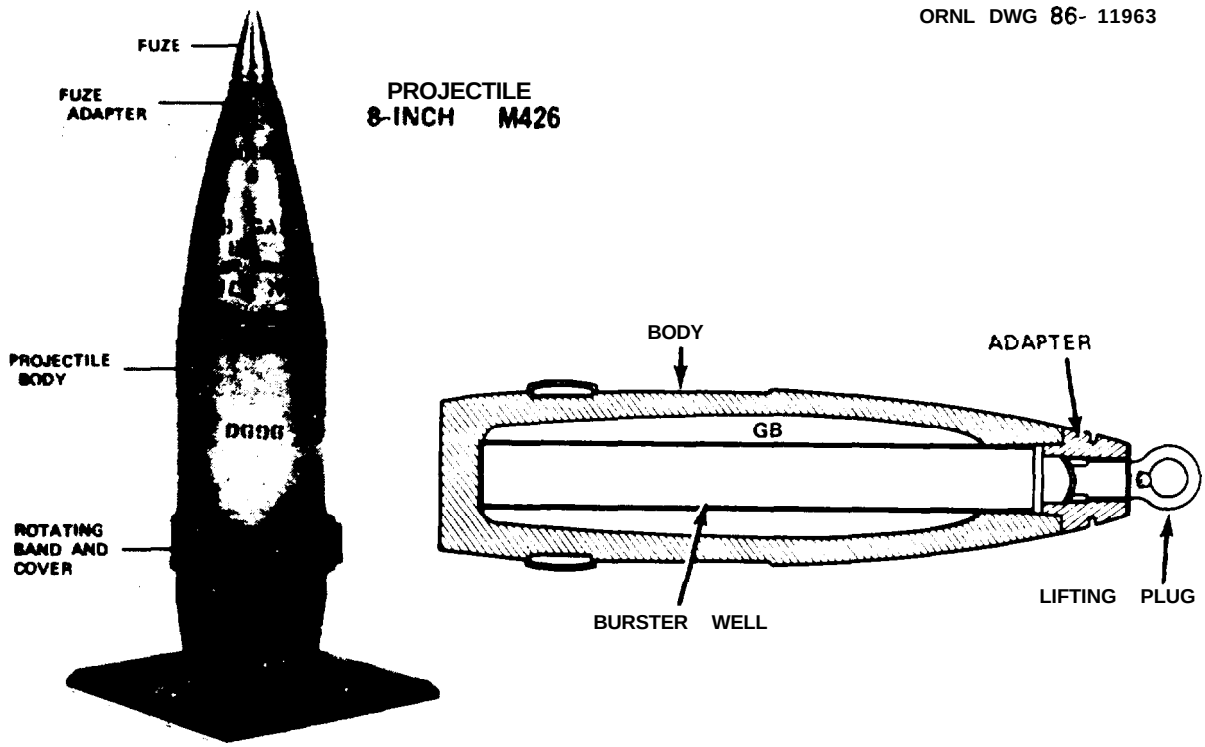


Fig. A.4. The 8-in. artillery projectiles are filled with CB or VX.

ORNL DWG 86-11964

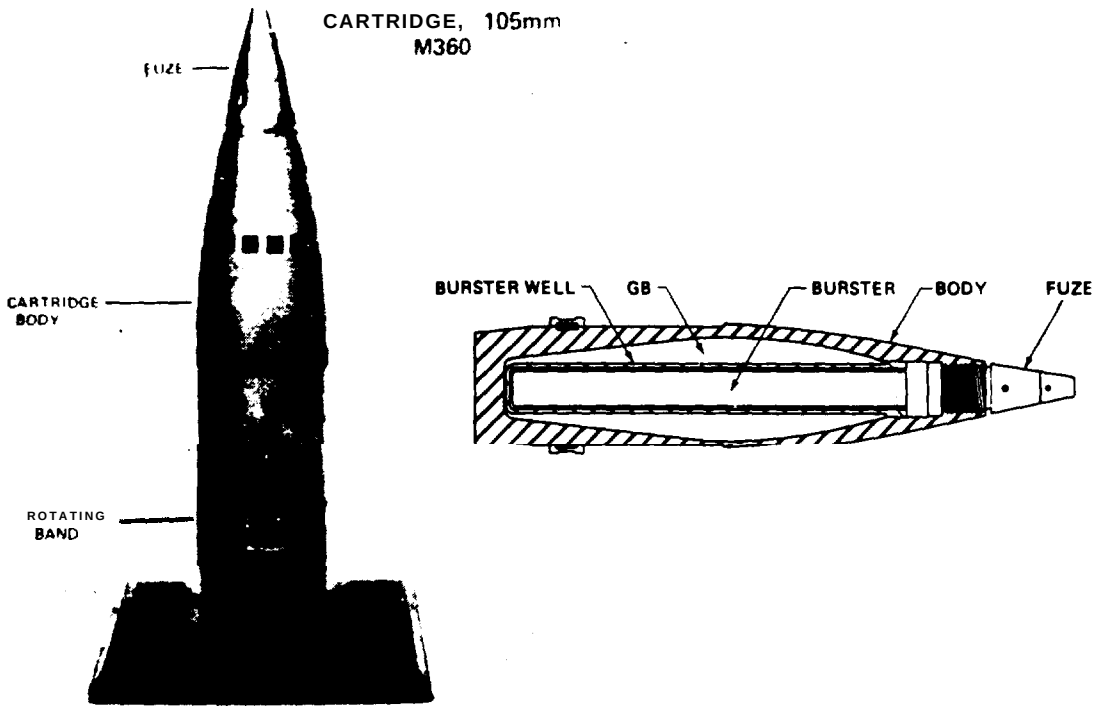


Fig. A.5. The 105-mm artillery projectiles are filled with mustard agent or GB.

ORNL DWG 86-11965

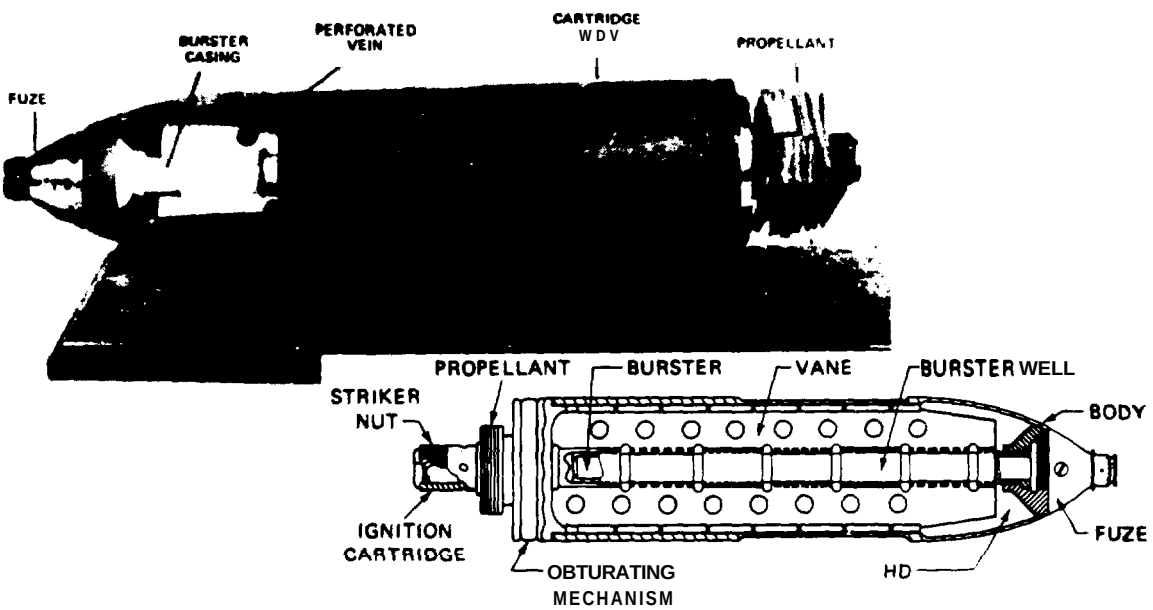


Fig. A.6. The 4.2-in. mortar projectiles are filled with mustard agent and GB.

ORNL DWG 86- 11966

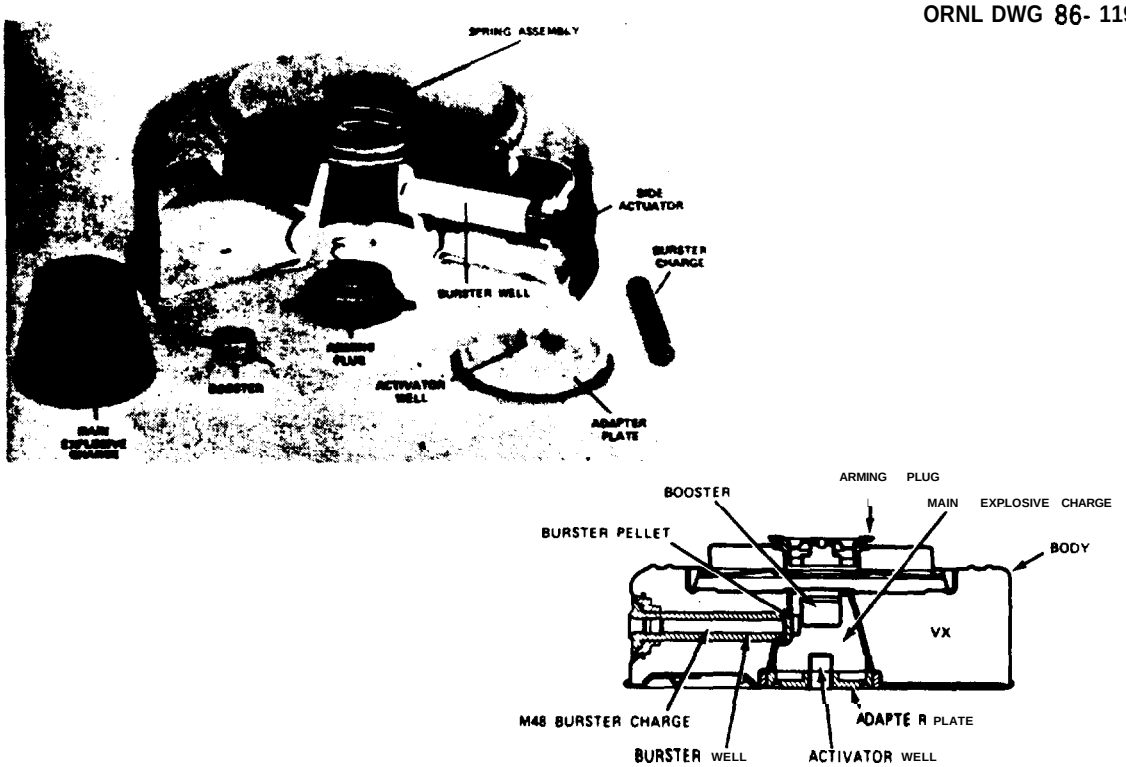


Fig. A.7. M23 land mines are filled with VX.

OANL DWG 86-I 1967

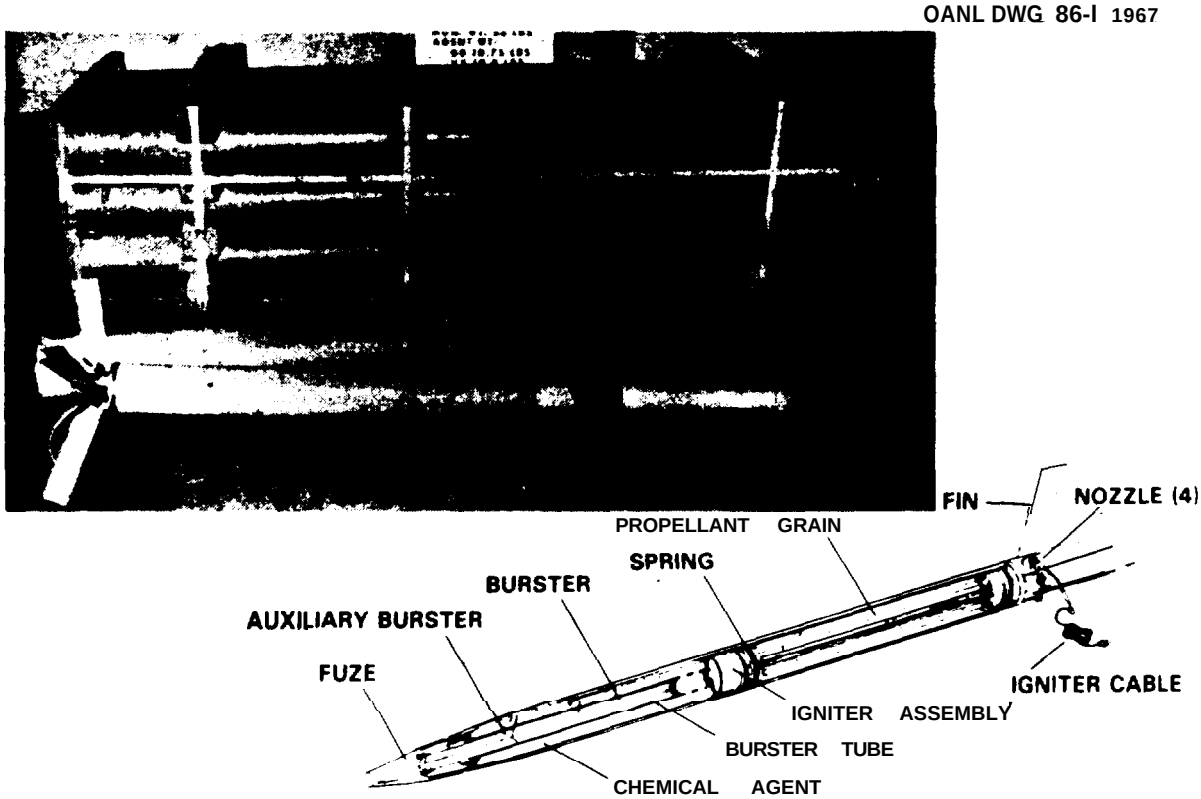


Fig. A.8. M55 115-mm rockets are filled with GB or VX.

The stockpile also contains the following aircraft-delivered munitions, which do not contain explosive components,

1. MC-1 750-lb Air Force bombs (Fig. A.9), filled with chemical agent GB.

2. MK-94 500-lb Navy bombs (Fig. A.10), filled with chemical agent GB.

3. MK-116 ("weteye") 525-lb Navy bombs (Fig. A.11), filled with chemical agent GB.

4. TMU-28/B aerial spray tanks (Fig. A.12), filled with chemical agent VX.

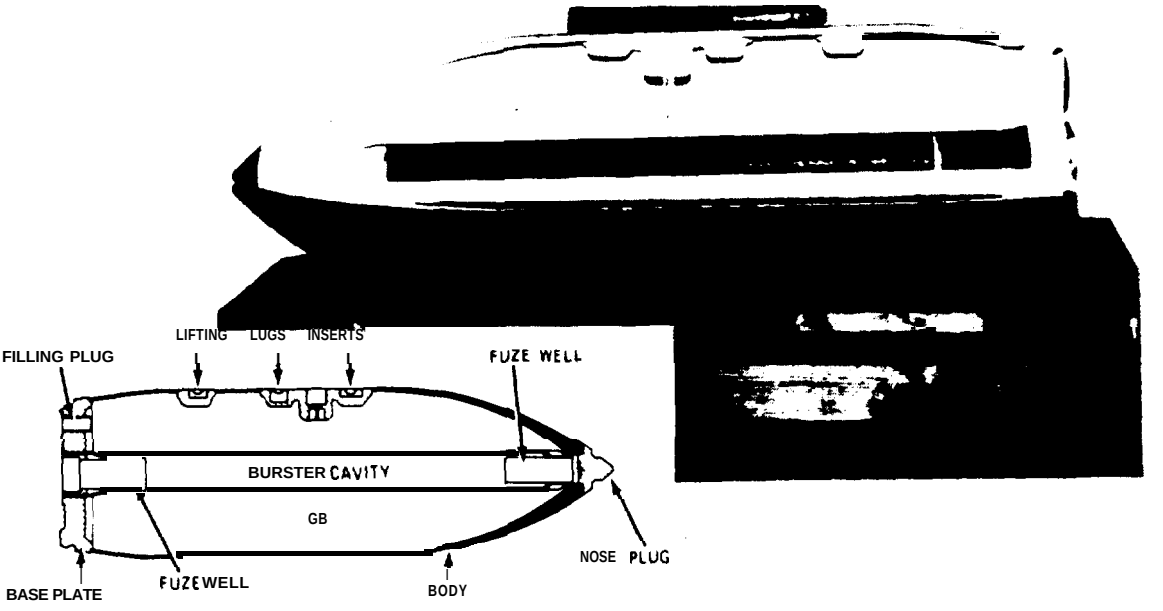
In addition, the United States has a large quantity of bulk chemical agent--about 61% of the total chemical inventory. This bulk agent includes the agents GA, GB, VX, mustard agent, and Lewisite. GA and Lewisite are stored only at TEAD in relatively small quantities. The bulk agent is stored in liquid form in steel ton containers (see Fig. A.13).

The makeup of the CSDP stockpile of munitions and bulk items containing lethal chamical agents to be destroyed is presented in Table A.l. The composition of each type of munition and bulk item, including type of agent explosive, propellant, and fuze, is shown in Table A.2.

Many of the munitions that contain chemical agents also include fuses, bursters, or propellants (see Table A.1). Fuzes are mechanical detonation devices for setting off the bursting charge of a projectile, rocket, or land mine. They are composed of a variety of compounds, including cyclonite, lead styphnate, lead oxide, barium nitrate, antimony sulfide, tetracine, and potassium chlorate. Bursters are explosive charges of a projectile, rocket, or land mine that, when detonated by a fuze, disperse agent and metal fragments into the surrounding area. Bursters are composed of various ingredients, including tetryl, tetrytol (tetryl plus TNT), or Composition B (cyclonite plus TNT). Propellants, the energetic materials that power a projectile or rocket, are composed of several compounds, including nitrocellulose, dinitrotoluene, lead stearate, triacetin, dibutylphthalate, and diphenylamine. The fiberglass shipping and firing tubes for M55 rockets have been found to contain small quantities of PCBs (polychlorinated biphenyls); they will be processed with the rockets rather than removed.

Chemical munitions (other than ton containers) are stored in configurations generally suitable for transport during wartime (e.g., in boxes, protective tubes, or metal overpacks on pallets). Dunnage includes any material, container, and padding used in packaging the munitions for storage and handling. Dunnage also includes wood pallets, some treated with PCP (pentachlorophenol); metal fasteners; steel drums; and polystyrene packing.

ORNL DWG 86- 11968



Fig, A.9. The MC-1 750-lb Air Force bombs are filled with GB.

ORNL DWG 86-I 1969

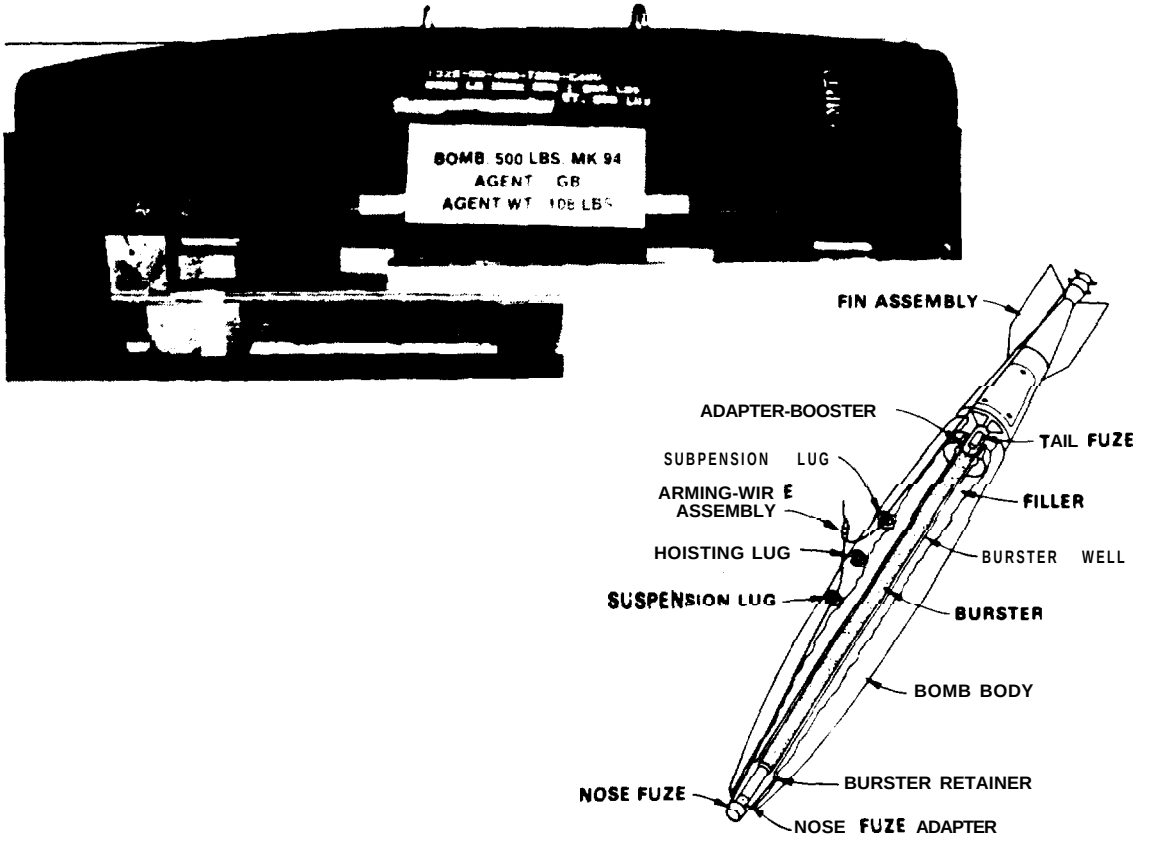


Fig. A.lO. The MK-94 500-lb Navy bombs are filled with GB.

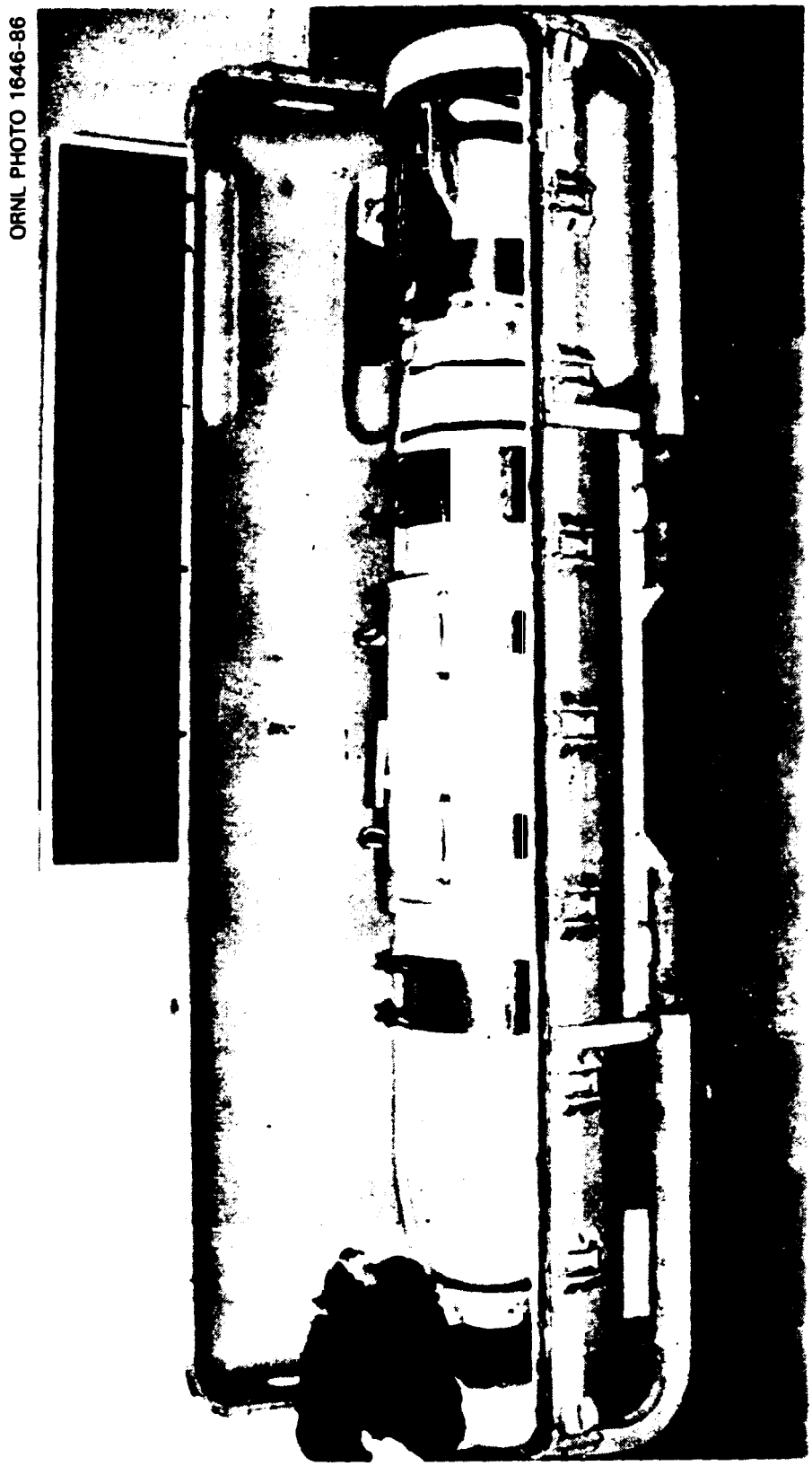


Fig. A.11. The MK-116 (weteye) 525-lb Navy bombs are filled with GB.

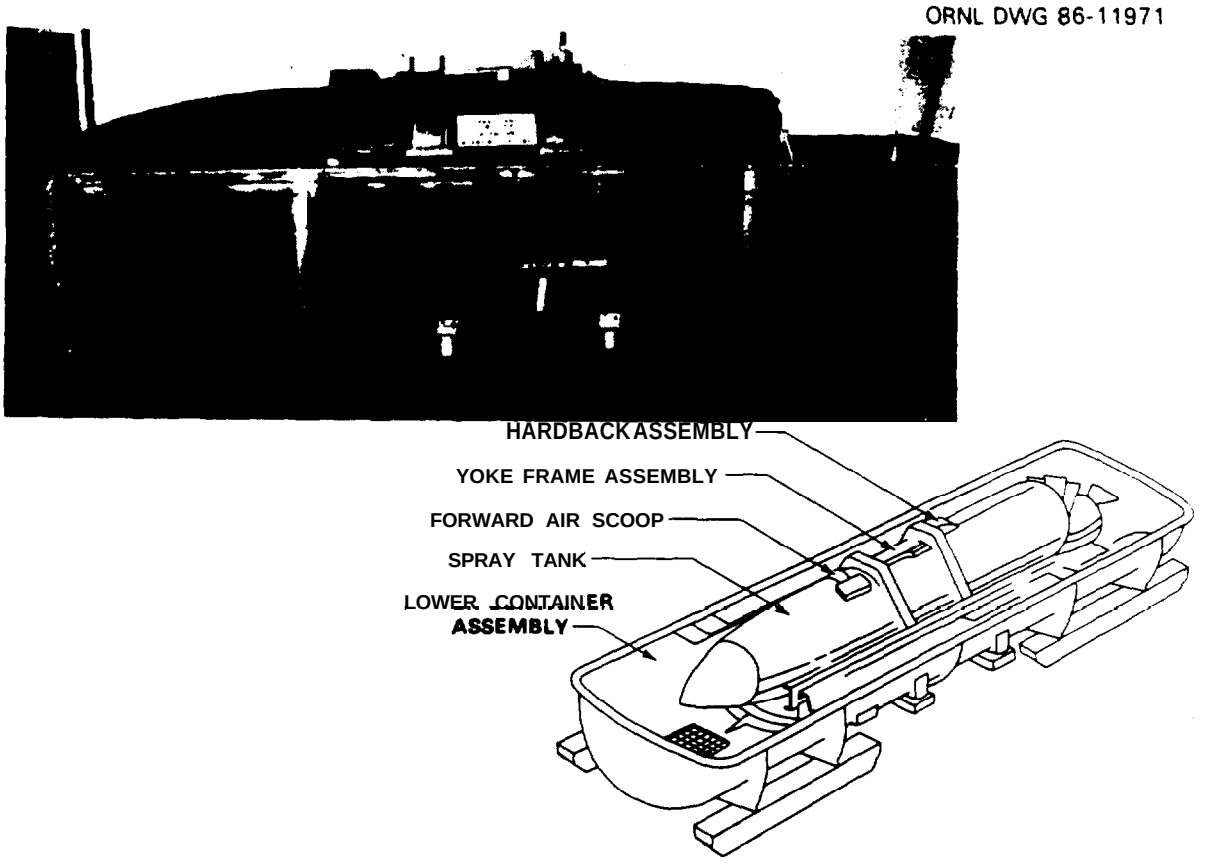


Fig. A.12. The TMU-28/B spray tanks are filled with VX.

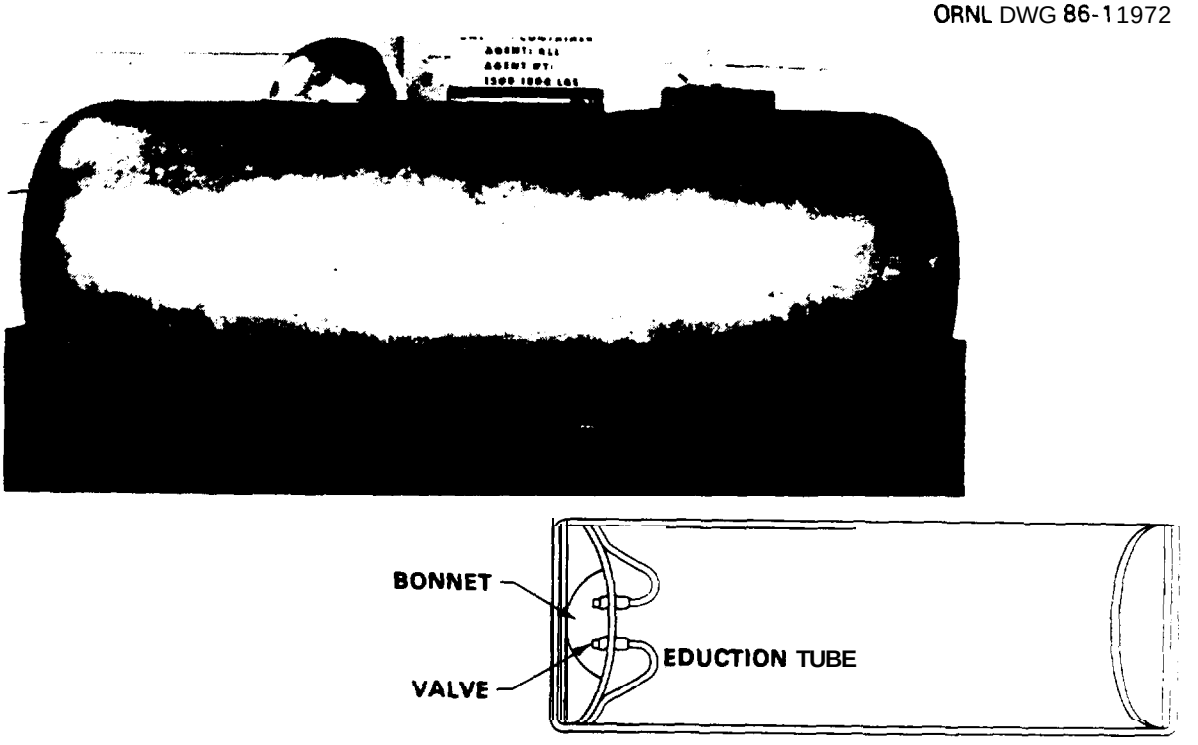


Fig. A.13. The bulk agent is stored in liquid form in these steel ton containers.

Table A.1. Composition of munitions

Munition	Agent	Fuze	Bunter	Propellant	Dunnage
M55 115-mm rockets[a]	GB, VX	Yes	Yes	Yes	Yes
M23 land mines	v x	Yes[b]	Yes	No	Yes
4.2-h mortars	Mustard	Yes	Yes	Yes	Yes
105-mm cartridges	GB, Mustard	Yes	Yes	Yes	Yes
105-mm projectiles	GB, Mustard	Yes[c]	Yes[c]	No	Yes
155-mm projectiles	GB, VX, Mustard	No	Yes[c]	No	Yes
8-in. projectiles	GB, VX	No	Yes[c]	No	Yes
Bombs (500-750 lb)	GB	No	No	No	Yes
Weteye bomb	GB	No	No	No	NO
Spray tanks	v x	No	No	No	No
Ton containers (bulk agents -68% of stockpile)	GB, VX, GA, Mustard, Lewisite	No	No	No	No

[a]M55 rockets arc processed in individual fiberglass hipping containers.
[b]Fuze and land mines are stored together but not assembled.
[c]Some projectiles have not been put into explosive configuration.

Table A.2. Description of lethal chemical agent munitions

Munition	Model/agent	Dimensions		Weight[a] (lb)	Agent		Burster		Propellant			Fuze model	Other energetic components
		Diameter	Length[a] (in.)		Type	Weight[a] (lb)	Model	Explosive	Weight[c] (lb)	Model	Weight[a] (lb)		
105-mm cartridge	M60	105 mm	31.1	42.92	HD[d]	2.97	M5	Tetrytol	0.3	Mb7	2.83	M57	M28B2 primer
		105 mm	31.1	42.92	HD	2.97	M5	Tetrytol	0.3	Mb7	2.83	M51A5	M22 booster
	M360	105 mm	31.1	43.86	GB	1.63	M40	Tetrytol	1.12	Mb7	2.83	M508	M28B2 primer
		105 mm	31.1	43.86	GB	1.63	M40A	Comp B	1.12	Mb7	2.83	M508	M28B2 primer
		105 mm	31.1	43.86	GB	1.63	NA[b]	NA	1.12	Mb7	2.83	M557	NA
105-mm projectile	M360	105 mm	16.0	32	GB	1.63						0	
4.2-in mortar	M2	4.2 in.	210	24.67	HD	6.0	M14	Tetryl	0.14	Mb	0.4	M8	M2 primer
	M2	4.2 in.	210	24.47	HT[d]	5.8	M14	Tetryl	014	Mb	0.4	M8	M2 primer
	M2A1	4.2 in.	210	24.67	HD	6.0	M14	Tetryl	0.14	Mb	0.4	M8	M2 primer
155-mm projectile	M104	155 mm	26.A	98.9	HD	11.7	NA	NA	0.41				
		155 mm	26.8	98.9	HD	11.7	Mb	Tetrytol	0.41				
		155 mm	26.8	98.9	H[d]	11.7	Mb	Tetrytol	041				
	M110	155 mm	26.8	98.9	HD	11.7	NA	NA	041				
		155 mm	26.8	98.9	H	11.7	Mb	Tetrytol	0.41				
		155 mm	26.8	98.9	HD	11.7	Mb	Tetrytol	0.41				
		155 mm	26.8	98.9	H	11.7	Mb	Tetrytol	0.41				
	M121	155 mm	26.8	96.4	GB	6.5							
	M121A1	155 mm	26.8	98.9	VX	6.3	M71	Comp B	2.45				
	M121A1	155 mm	26.8	98.9	GB	6.5	M71	camp B	2.45				
	M122	155 mm	26.8	98.9	GB	6.5	M37	Tetrytol	2.45				
8-in projectile	M426	8 in.	35.1	203	VX	14.5	M83	Comp B	7.0				
		8 in.	35.1	203	GB	14.5	M83	Camp B	7.0				
		8 in.	35.1	203	GB	14.5	NA	NA	7.0				
Land mine	M23	13.5 in.	5	23	VX	10.5	M38	Comp B	0.8			M603	
Rocket	M55	115 mm	7R.0	57	GB	10.7	M34	Comp B	3.2	M28	19.3	M417	Mb2 primer
		115 mm	7R.0	57	GB	10.7	M36	Comp B	32	M28	19.3	M417	Mb2 primer
		115 mm	7R.0	57	VX	10.0	M34	Comp B	32	Mb7	19.3	M417	Mb2 primer
		115 mm	78.0	57	VX	10.0	M36	Comp B	3.2	Mb7	19.3	M417	Mb2 primer
500-lb bomb	MK94.0	10.8	60.0	441	GB	108							
525-lb weteye	MK11b-0	14 in.	86.0	525	GB	347							
750-lb bomb	MC.1	16.0	500	725	GB	220							
Spray tank	TMU-28/B	22.5	185.5	1.935	VX	1.356							
Bulk containers	Agent GB	30.1	85.1	2.900	GB	1.500							
	Agent H	30.1	85.1	3.100	H	1,700							
	Agent HD	30.1	85.1	3.100	HD	1.700							
	Agent HT	30.1	85.1	3.100	HT	1.700							
	Agent L	30.1	85.1	3.100	L	1,700							
	Agent VX	30.1	85.1	3.000	VX	1,600							
	Agent GA	30.1	85.1	NA	GA	NA							

[d]HD, HT, H are referred to as Mustard.
[b]NA = information not available.
[a]For conversion of the English units to metric units 1 in = 2.54 cm and 1 lb = 0.454 kg

A.3. LETHAL CHEMICAL AGENTS

The munitions and bulk containers are each filled with one of the following lethal chemical agents: GB, VX, or mustard (and, only at TEAD, very small quantities of Lewisite and GA). The lethal chemical agents are liquids at room temperature and are not corrosive. The agents do not exist in the munitions or bulk containers as pure compounds, and they are stabilized (to prevent acid formation) with various compounds. Information about the chemical composition and the physical characteristics of the agents GB, VX, and mustard is presented in Tables A.3-A.5. Toxicity is discussed in Appendix B.

The expected agent decomposition products due to incineration or decontamination, as well as degradation due to aging, are listed in Table A.6. The Army believes it is reasonable to presume at this point that none of the agent decomposition products will be more difficult to destroy by incineration than the agents and that the decomposition products are less hazardous than pure agents.

Table A.3. Chemical agent CB characteristics[a]

Physical properties	Composition	
	Component	Percent[b]
Chemical name: isopropylmethylphosphonofluoridate; sarin	Isopropylmcthylphosphonofluoridatc (GB)	93
Chemical formula: $C_4H_{10}FO_2P$	N,N'-diisopropylcarbodimide (DICDI)[c]	4
Molecular weight: 140.10	Tributylaminc (TBA)	1.95
Vapor density (air = 1.00): 4.86	Methyl dilluoride	0.5
Liquid density at 25°C: 1.0887 g/cc	HF	0.2
Freezing point: -56°C	Aluminum	0.2
Boiling point: 158°C	HCI	0.1
Vapor pressure: 2.9 mm Hg at 25°C	Iron	0.05
Flash point: does not flash	Nickel	0.0025
Viscosity (ccntistokes at 25°C): 1.28	Copper	0.0004
Color: clear to straw to amber		
Odor: none		
Special properties: none		
Solubility properties: miscible with water and readily soluble in all organic solvents		
AH combustion: 10,000 Btu/lb		
Physical state: viscous liquid		

[a]GB is a rapid-acting lethal nerve agent. The action within the body is the inactivation of cholinesterase. The hazard from GB is from vapor absorption through the respiratory tract, although it can be absorbed through any part of the skin, through the eyes, and through the gastrointestinal tract by ingestion. The agent absorption rate is accelerated through cuts and abrasions in the skin. When dispersed as large droplets, GB is moderately persistent; it is nonpersistent when disseminated as a cloud of very line particles.

[b]These numbers are given as a guide only and do not represent product specifications or the exact constituency of the agent.

[c]These constituents are not both in all GB.

Table A.4. Chemical agent VX characteristics[a]

Physical properties	Composition	
	Component	Percent[b]
Chemical name: o-ethyl S-(2-diisopropylaminoethyl) mcthylphosphonothiolatc	o-ethyl *S*-(2-diisopropylaminoethyl) mcthylphosphonothiolate (VX)	93
Chemical formula: $C_{11}H_{26}NO_2PS$	Pyrodiestcr	3
Molecular weight: 267.37	*N,N'*diisopropylcarbodiimide (DICDI) or dicyciohcxylcarbodiimidc	2.5
Vapor density (air = 1.00): 9.2	Free mcrcaptan	
Liquid density at 25°C: 1.008 g/cc	H_2SO_4	0.3
Freezing point: below -39°C	Free sulphur	0.14
Boiling point: 300°C	Iron	0.05
Vapor pressure at 20°C: 0.00066 mm Hg	Aluminum	0.01
Flash point: 159°C	Nickel	0.0025
Viscosity (ccntistokcs at 25°C): 9.96	Copper	0.0004
Color: clear to straw		
Odor: none		
Special properties: none		
Solubility properties: best solvents are dilute mineral acids		
ΔH combustion: 15,000 Btu/lb		
Physical state: viscous liquid		

'VX is a rapid-acting lethal nerve agent. The action within the body is the inactivation of cholinestcrase. The hazard from **VX** is primarily from liquid absorption through the skin. although it can be absorbed through the respiratory tract as a vapor or aerosol and through the gastrointestinal tract by ingestion. **VX** is slow to evaporate and may persist as **a** liquid for several days.

[b]These numbers are given as a guide only and do not represent product specifications or the exact constituency of the agent.

Table A.5. Chemical agent mustard characteristics'

Physical properties	Composition	
	Component	Percent[c]
Chemical name: bis(2-chlorethyl)sulfide 2,2'-dichlorodiethyl sulfide-sulfur mustard	bis(2-chloroethyl)sulfide (HD-mustard)	92
Chemical formula: $(ClCH_2CH_2)_2S$	Free sulphur	7.38
Molecular weight: 175.00[b]	$FeCl_2$	0.5
Vapor density (air = 1.00): 5.4[b]	HCl	0.1
Liquid density at 20°C (g/cc): 1.27'	Aluminum	0.01
Freezing point: 5-14°C[b]	Nickel	0.0025
Boiling point: 225.4°C[b]	Copper	0.0004
Vapor pressure at 20°C (mm Hg): 0.0596		
Flash point: 100°C[b]		
Viscosity (centistokes at 20°C): 3.95[b]		
Color: amber to dark brown liquid		
Odor: garlic		
Special properties: permeates ordinary rubber		
Solubility properties: water (distilled)—0.092 at 22°C; completely soluble in acetone, CCl_4, CH_3Cl, tetrachloroethane, ethyl benzoate, ether		
AH combustion: 8.100" Btu/lb		
Physical state: viscous liquid		

'Mustard is a persistent and powerful blistering agent. It acts principally by poisoning the cells in the surfaces contacted. Both liquid and vapor cause intense inflammation and may cause severe blistering of both the skin and mucous membranes. Mustard is only moderately volatile. Mustard is designated H. HD, and HT. H is mustard made by the Levinstein process. It contains up to 25% by weight of impurities, chiefly sulfur, organosulfur chlorides, and polysulfides. HD (distilled mustard) is mustard purified by washing and vacuum distillation, which reduces impurities to about 5%. HT is a 60:40 mixture by weight of HD and T. T is an abbreviation for bis[2(2-chloroethylthio)ethyl] ether.

[b]Applies only to H.

'These numbers are given as a guide and do not represent product specifications or the exact constituency of the agent.

Table A.6. Decomposition products from incineration, decontamination, and aging of chemical agents'

Process reaction	Products of decomposition
Thermal degradation (incineration) of GB	CO_2, H_2O, P_2O_5, and HF
Thermal degradation (incineration) of VX	CO_2, H_2O, NO_x, P_2O_5, and SO_2
Thermal degradation (incineration) of mustard	CO_2, H_2O, HCl, and SO_2
Reaction of decontaminant (Na_2CO_3) and GB	NaF, H_2O, and monosodium salt of isopropyl methyl phosphoric acid
Reaction of decontaminant (NaOCl) and VX	Na_2SO_4, NaCl, H_2O, Na_2CO_3, diisopropylamine and sodium o-ethyl methyl phosphonatc
Reaction of decontaminant (NaOCl) and mustard	Na_2SO_4, NaCl, CO_2, and H_2O
Degradation of GB due to age	HF; isopropyl methylphosphonic acid; isopropanol; propene; methyl phosphono fluoridate; diisopropyl methyl phosphonatc (DIMP); and methylphosphonic acid
Degradation of VX due to age	Thiolaminc Ethylmethylphosphonic acid Ethanol bis(2-diisopropylaminoethyl) thioether bis(2-diisopropylaminoethyl) disulfidc *o,o'*-diethylmethyl phosphonlate *o,o'*-diethylmethyl phosphonothiolate Diisopropylaminoethyl mercaptan *o,S*-diethyl methyl phosphonothioate Diisopropylaminoethylethyl sulfide *o,S*-diethyl methyl phosphonate *S,S'*-diisopropylamino ethyl methyl phosphonate S-ethyl, S-diispropyl amino ethyl methyl phosphonothiolate o-ethyl, S-diisopropyl amino ethyl methyl phosphonothiolate *S'*-diisopropyl amino ethyl methyl phosphonothioate Diispropyl amine
Degradation of mustard due to age	HCl; ethylene; ethylene dichloride; 2,2'-dichlorodiethyl disulfide; vinyl chloride; and hydrogen sulfide

'It is assumed that none of the agent products is more difficult to destroy by incineration than the agents themselves and that the agent products are also less hazardous than the agents.

Appendix B

TOXICITY OF WARFARE AGENTS AND THEIR BREAKDOWN PRODUCTS

CONTENTS

Page

EXECUTIVE SUMMARY B-5

B.l INTRODUCTION B-14
B.l.l NERVE AGENTS (GA, GB, and VX) B-14
B.1.2 VESICANT (BLISTER) AGENTS (H, HD, HT, and Lewisite) B-15

8.2 NERVE AGENT TOXICITY B-18
B.2.1 SUMMARY B-18
B.2.2 ACUTE TOXICITY B-19
B.2.2.1 Agent GA B-26
B.2.2.2 Agent GB B-27
B.2.2.3 Agent VX B-32
B.2.2.4 Mixtures B-36
B.2.3 CHRONIC TOXICITY B-37
B.2.3.1 Agent GA B-42
B.2.3.2 Agent GB B-42
B.2.3.3 Agent VX B-46
B.2.4 ANTIDOTES AND PERSONAL DECONTAMINATION B-48
B.2.4.1 Antidote Treatment Regime, Dosage, Toxicity, Aging, and Cautions B-48
B.2.4.2 New Developments, Particularly with Oximes B-58
B.2.4.3 Evaluation B-64
B.2.4.4 Personal Decontamination B-66
B.2.5 D2PC CODE CONSIDERATION B-67

B.3 VESICANT AGENT TOXICITY B-68
B.3.1 SUMMARY B-68
B.3.2 ACUTE TOXICITY B-69
B.3.2.1 Agents H/HD B-70
B.3.2.2 Agents HT/T B-81
B.3.2.3 Agent L (Lewisite) B-82
B.3.3 CHRONIC TOXICITY B-83
B.3.3.1 Agents H/HD B-83
B.3.3.2 Agent HT/T B-104
B.3.3.3 Agent L (Lewisite) B-105
B.3.4 ANTIDOTES AND PERSONAL DECONTAMINATION B-107
B.3.4.1 Agents H/HD B-107
B.3.4.2 Agent HT B-108
B.3.4.3 Agent L (Lewisite) B-109
B.3.5 D2PC CODE CONSIDERATIONS B-112

B.4 AGENT ADDITIVES (STABILIZERS) AND DECONTAMINATION TOXICITY B-113

B.5 BREAKDOWN PRODUCT TOXICITY B-116

B.5.1 HYDROLYSIS PRODUCTS B-117
B.5.1.1 Formation B-117
B.5.1.2 Hydrolysis Product Description B-122
B.5.1.3 Chronic and Acute Toxicity B-122
B.5.2 DECONTAMINATION PRODUCTS B-124
B.5.2.1 Formation B-124
B.5.2.2 Decontamination B-125
B.5.2.3 Chronic and Acute Toxicity B-126
B.5.3 COMBUSTION PRODUCTS B-152
B.5.3.1 Formation B-152
B.5.3.2 Combustion Product Description B-153
B.5.3.3 Chronic and Acute Toxicity B-154

B.6 REENTRY CRITERIA FOR AGENT CONTAMINATION OF FOOD, WATER, AND SURFACES B-157
B.6.1 DISPOSITION OF LIVESTOCK AND FOOD PRODUCTS B-158
B.6.2 CRITERIA FOR REENTRY BY THE GENERAL PUBLIC B-159
B.6.2.1 Drinking Water Criteria B-160
B.6.2.2 Air Criteria B-165
B.6.2.3 Garden Produce Criteria B-165
B.6.2.4 Food + Water Ingestion Criteria B-166
B.6.2.5 Contaminated Surfaces Criteria B-168

DEFINITION OF TERMS B-169

REFERENCES FOR APPENDIX B B-174

EXECUTIVE SUMMARY

The text of this appendix is a comprehensive evaluation of chemical agent toxicity and related issues, such as antidote use and reentry criteria. Four objectives are met:

1. To provide, in a single location of the EIS, the toxicity information needed for an informed decision;

2. To generate a resource document for the impact assessment, the U.S. Army, and cooperating agencies;

3. To address health-related concerns raised during the public comment period; and

4. To synthesize existing information in developing new analyses.

The first two objectives are satisfied by the discussions of acute and chronic toxicity for each compound or mixture classified as either a nerve agent (acetylcholinesterase inhibitor) or vesicant (induces skin blisters and generates chemical burns of mucous membranes and/or eyes). The interested reader is referred to Sects. B.2 and B.3 for substantial detail.

The third objective is addressed by several new sections added since release of the DPEIS in July 1986. They are:

1. an evaluation of currently available and experimental agent antidotes, their treatment protocols, and toxicity (Sect. B.2.4),

2. agent-specific personal decontamination (see Sects, B.2 and B.3),

3. toxicological assessment of stabilizers and decontaminants (Sect. B.4),

4. toxicological assessment of the known products of agent/stabilizer hydrolysis, combustion, decontamination, and microbial decomposition (Sect. B.5), and

5. development of proposed decision criteria for use in determining safe entry and use of foodstuffs, water, forage crops, etc., in the event of an unplanned release with off-site consequences (Sect. 8.6).

Resulting new insight, which simultaneously fulfills the fourth objective, includes a semiquantitative assessment of mustard agent carcinogenicity relative to other better-characterized carcinogens (Sect. B.3), the generation of recommended-as-safe maximum permissible concentrations (MPCs) for agent in or on drinking water and foodstuffs, and recommended decision criteria for disposition of potentially

contaminated livestock and agricultural crops (Sect. B.6). These latter analyses are particularly pertinent when considering the nonlethal risks of various transport options assessed in the EIS.

Highlights and major findings follow.

NERVE AGENT TOXICITY

The three nerve agents considered in the current assessment are

1. GA [Chemical Abstracts Service (CAS) # 77-81-6, ethyl-N,N-dimethyl phosphoramidocyanidate],

2. GB (CAS # 107-44-8, isopropyl methyl phosphonofluoridate), and

3. VX ["V" for venom, CAS # 50782-69-9, 0-ethyl-S-(2-diisopropyl aminoethyl)methyl phosphonothiolate] .

The fact that nerve agents are acutely lethal at relatively low concentrations (e.g., human skin LD_{50} of 0.04 mg/kg for agent VX) is the paramount factor in the consideration of nerve agent toxicity in the EIS process. For the inhalation route, differences in individual agent volatility generate agent-specific considerations in the evaluation of human health effects. Because agent GB is highly volatile, an unplanned release could disperse toxic concentrations over a large area. With the less-volatile agent VX, toxic concentrations would not disperse widely but could persist in the environment long after an unplanned release.

Although existing information on the chronic health effects in animals is meager, all information gathered so far indicates that nerve agents do not cause mutations or cancer and do not damage the fetus or cause reproductive problems. Agents GA and GB have the potential to cause a delayed neuropathic condition, but only days after exposure to concentrations many times the lethal dose. Delayed neuropathy is not a relevant issue in comparison to concerns of lethality. The possibility of long-term brain dysfunction has been raised; however, the only human cases displaying functional changes in brain activity are weapons factory workers who had experienced at least one (and often more) toxic exposures sufficient to produce pallor, muscle weakness, and red blood cell cholinesterase depression. There is no evidence to suggest that there would be delayed or latent human health effects upon exposure to very low nerve agent concentrations comparable to those expected during normal incineration plant operation.

Immediate personal skin decontamination measures using readily available household materials have been tested in guinea pigs. Absorption by flour followed by washing with water and wiping with wet tissue paper is most effective. Washing with soapy water gives significant protection if nothing else is at hand; in either case, speed is of the essence.

VESICANT AGENT TOXICITY

The vesicant agents in the unitary stockpile scheduled for destruction are:

1. H [Levinstein mustard, CAS # 505-60-2, bis(2-chloroethyl) sulfide],

2. HD (distilled mustard),

3. HT [a plant-run mixture containing about 60% HD and <40% agent "T," CAS # 63918-89-8, bis-(2-chloroethylthioethyl)ether, which add:. stability and persistence to the blend by lowering the freezing point; agent T also has considerable toxic properties of its own and is highly mutagenic in fruit flies], and

4. Lewisite (Agent L, an organic arsenical, CAS # 541-25-3, 2-chlorovinyl dichloroarsine).

Following human exposure to any formulation of mustard agent, there is usually a latency period of several hours before signs of toxicity begin to appear. These signs include eye inflammation (which *occurs* at lower doses than any other effect, and is therefore the most sensitive indicator of mustard agent exposure), skin irritation (which can produce large blisters), and irritation of the respiratory tract. Recovery from these toxic effects can take days or weeks. Mustard gas is not as potent in producing immediate death following exposure as are the nerve agents. Military hospital records from World War I and the present-day Iran-Iraq conflict indicate 3 to 4% acute lethality from battlefield exposures to mustard agent. Acute effects are certainly disabling in the short term and require special care and resources to prevent subsequent infection of skin, respiratory tract, and eyes.

There is no specific antidote for mustard agent poisoning. The best way to minimize the effects of mustard agent exposure is very rapid removal of the agent from all body surfaces by thorough washing and absorption of the agent by various chloramine powders or even household flour. Mustard is very persistent in the environment (on the order of years) and can adsorb onto soils and vegetation.

In addition to the toxic signs mentioned above, mustard agent exposure can also produce delayed effects (i.e., toxic manifestations that appear years after initial exposure). Apparent healing of eye damage after acute, high-level exposure can be followed by permanent vision impairment over the course of years, although this effect is infrequent. Respiratory tract damage can result in chronic bronchitis, given conditions of sufficiently severe exposure. Epidemiological evidence and results of animal studies both indicate that mustard agent can induce cancer. The human evidence arises from workers in poison gas factories under wartime conditions who were exposed to irritating levels of this and other warfare agents for years. The cancers produced in these exposed individuals were in the respiratory tract and on the skin.

Mustard agent, because of its highly reactive chemical nature, can react with DNA to produce mutations. Evidence from experimental animal studies and from epidemiological studies of poison gas factory workers has so far not reliably demonstrated.. that mustard agent exposure produces reproductive effects. Because of the ability of mustard agent to modify genetic material, delayed effects at the cellular level are a possibility.

Agents HT and Lewisite are not as well characterized as sulfur mustard but possess generally similar vesicant properties. Agent HT is less volatile, more stable, and more lethal than HD and is considered carcinogenic because of the presence of HD and mutagenic because of HD and T. Lewisite does not exhibit the latency period displayed by mustard agent and is noted for causing immediate severe pain upon contact with skin and eyes. It is also known to be a systemic poison (liver and kidneys) at sufficiently large doses and has induced Bowen's disease, a relatively slow-growing and usually nonfatal form of skin cancer, among exposed soldiers and poison gas factory workers. It is the only vesicant for which there are specific antidotes (for both tissue and systemic effects).

AVAILABLE AND EXPERIMENTAL NERVE AGENT ANTIDOTES

Available antidotes to GA, GB, and VX can be lifesaving if administered within minutes of significant exposure and when used together with decontamination and intensive medical support. In the United States, the only clinically available antidotes are atropine and pralidoxime (2-PAM-Cl). Even with their use, artificial respiration and other support is likely to be needed in cases of severe exposure. Both atropine and 2-PAM-Cl can have severe or life-threatening side effects and must be used under trained medical supervision with certain knowledge that exposure has occurred. Other countries use different oximes and may use centrally acting drugs in addition to atropine-oxime therapy. Research is under way to find better antidotes, but human testing in the United States has not yet been undertaken with the most recent oxime of promise, HI-6.

A significant increase in survival rates has been observed if experimental animals are pretreated with pyridostigmine before nerve agent exposure, followed by the usual antidote protocol. This finding has led to the recommended prophylactic use of pyridostigmine among military services in the United Kingdom. Work with other pretreatment regimens in animals is promising in terms of increased survival and in maintenance of normal breathing rates and performance. However, these regimens must undergo much further evaluation and clinical testing before they can be considered for general human use.

STABILIZER AND DECONTAMINANT TOXICITY

Tributylamine, diisopropyl methyl phosphonate, diisopropyl carbodiimide, and diethyl dimethylpyrophosphonate are found in nerve agent (GB and VX) formulations as either stabilizers or chemical contaminants arising during manufacture. Very little toxicological information could be found regarding these chemicals, but current information suggests their toxic effects are insignificant in comparison *to* nerve agent potency.

Treatment of agent-contaminated surfaces with decontaminating chemicals [supertropical bleach (STB) or decontaminating solution No. 2 (DS-2), which contains diethylene triamine] could potentially result in public exposure to decontaminants in the event of an unplanned release with off-site consequences. Although some decontaminants have toxic properties at high doses (rabbit skin LD_{50} of diethylene triamine is 1090 mg/kg), their toxicity is much less than that of the agents themselves. The prudent decision would be to decontaminate using all precaution to contain the solution and avoid contact.

BREAKDOWN PRODUCT TOXICITY

Unintended release of chemical agents into the environment could result in human exposure to a variety of their breakdown products. Leakage into surface or groundwater would lead to the generation of hydrolysis products via interaction of the chemical agents with water. The chemical agents could also interact with chemicals used in decontamination and produce decontamination products. Combustion products, both complete and incomplete, would be released in the event of an accidental fire *or* explosion involving the chemical agents. To the extent possible, these breakdown products have been identified and their toxicities evaluated in Sect. B.5.

An extensive listing of known products of agent hydrolysis, decontamination, and combustion is included in this section; but it is by no means complete to the authors' satisfaction. The principal reasons are that each breakdown reaction has not always been completely evaluated, and sufficient biological effects data have not been generated. This latter situation is particularly true for the more exotic products.

As expected, none of the possible breakdown products are as acutely toxic as the agents from which they would be derived. Of the hydrolysis products, hydrogen cyanide (HCN), from GA, and arsenite, from Lewisite, present the greatest health hazard. The acute toxic effects from HCN would be confined to the contaminated site and would rapidly dissipate as HCN is oxidized to the nontoxic cyanate. Arsenicals, including arsenite, are persistent in the environment. Because they are carcinogenic, *even* chronic exposure to low levels should be minimized.

Contaminated sites should be confined to the extent possible and access to sites should be restricted until monitoring data indicate that reentry is safe.

The two most likely decontaminants, STB and DS-2, have as principal by-products the compounds chloroform, acetic acid, ammonia, cyanogen chloride, and butyric acid. Of these, chloroform is the only suspect human carcinogen. It is thought to exhibit rather modest potency for lifetime oral exposure; potency information for dermal or inhalation exposure is not available. It is fetotoxic and teratogenic in rats and produces sperm abnormalities in mice; it may be mutagenic in vivo, although most in vitro tests have been negative. It is a skin and eye irritant and can cause anesthesia and death (1000 ppm) upon inhalation at high concentrations. Acetic acid, ammonia, and butyric (N-butanoic) acid are well-known eye and skin irritants; inhalation of ammonia at high concentrations (35 mg/m^3) can cause death. Cyanogen chloride is a highly toxic military poison gas with effects very similar to hydrogen cyanide. Note that usual precautions of protective clothing and containment used during agent spill cleanup should be more than adequate to protect operations personnel and the general public from these products of decontamination.

In the event of an accidental fire or explosion involving chemical agents, several toxic gases could be generated. They include hydrogen cyanide, from GA; hydrogen fluoride, from GB; nitrogen dioxide, from GA and VX; sulfur dioxide, from VX and HD; and chlorine, from Lewisite. These gases could disperse from the area at a rate determined by the prevailing wind conditions and would not present a persistent hazard. Arsenic compounds, from Lewisite, could deposit at and around the combustion site and would present a more long-term exposure problem. It should be noted that there is only a small stockpile of Lewisite, the only agent from which the arsenic compounds would be produced, and it is confined to a single storage site (Tooele Army Depot). Thus, on-site incineration would virtually eliminate the possibility of exposure to arsenicals.

PROPOSED REENTRY DECISION CRITERIA

In the event of an unplanned release of agent during handling or transport, a potential exists for contamination of drinking water, forage crops, grains, garden produce, and livestock (either by surface contact or ingestion of contaminated food or water). Persistent agents, such as VX or the mustards, pose the greatest human health concern for reentry.

Several general guidelines for safe use of agricultural products and water have been developed in cooperation with the Centers for Disease Control (DHHS) and civilian agencies having regulatory responsibilities for monitoring crops, livestock and water [e.g., U.S. Department of Agriculture (USDA), U.S. Environmental Protection Agency (EPA), Food and Drug Administration (FDA)]. These guidelines are

1. Meat and milk from animals not actually killed by nerve agents could be used without concern provided external contamination could be eliminated. (The logic here is that an animal survivor of nerve agent exposure would contain no unreacted agent of sufficient concentration to be toxic in its muscle tissues. In contrast, the muscle tissue and blood of dead animals could contain unreacted agent, depending on the dose received.) The general public is often especially concerned about dairy products. If there is reluctance to consume or market milk from potentially contaminated dairy herds immediately after a major release, discarding milk produced during the first three to seven days following release should provide adequate assurance of safety. Note, however, that lambs suckling VX-poisoned ewes with clinical manifestations of organophosphorous toxicity demonstrated no signs of illness during the Skull Valley incident of 1968 (Van Kampen et al. 1969). This is strong evidence that VX is not secreted in milk.

2. Special precautions to prevent surface contamination would be required if residually contaminated animals without signs of toxicity were slaughtered or milked.

3. Meat and milk from animals killed by agent exposure or exhibiting severe toxic response should not be salvaged for hides, meat, or other animal products. These animals and their products should be destroyed. Destruction criteria will be developed in conjunction with appropriate federal agencies on a site-specific basis.

4. If more stringent disposition criteria are desired and testing facilities are readily available, animal cholinesterase could be tested; a depression of 50% or more below normal is the suggested action level for quarantine (baseline levels for various livestock species would require characterization).

5. Forage, grains, and garden produce could be quarantined until tested.

In Sect. B.6, quarantine, or action levels, have been developed to designate when unprotected members of the general public should either be denied entry or provided alternate sources of water or food. Protective assumptions of threshold effects as well as ingestion rates were used to derive recommended MPCs for drinking water and garden produce. MPCs in air are those previously recommended as safe by Surgeon General's Panels convened by DHHS (see Table B.21). Existing detection capability is sufficient for atmospheric MPCs but not for drinking water or garden produce values. Although theoretically plausible, research and development will be necessary before concentrations of agent in water or produce can be determined at recommended MPC levels with any consistency.

A similar approach was attempted for evaluating surface contamination of porous media such as wood, masonry, and textiles. Decision criteria for these materials are of particular concern in the event of an off-site transportation accident. However, there are no data available to use in correlating the amount of agent on or absorbed into any given surface with the concentration observed in off-gases. Therefore, we consider it prudent to prohibit access to contaminated buildings and their contents by the general public until the area can be determined to be acceptably safe. This time period is currently indefinite, given that the most sensitive form of commercially available off-gas analysis is unable to detect when porous surfaces such as fabric or plastic are free of agent. Desorption of agent from porous or plastic surfaces could continue over time by means of elevated temperature or passive diffusion. This is an obvious area for further investigation.

MAJOR INSIGHTS

Restricted Access

In the event of an unplanned agent release with atmospheric dispersion off-site, the area potentially contaminated will be restricted from public use until the area can be determined to be acceptably safe. This decision will be necessary regardless of the presence or absence of associated fatalities. At present,

1. there is no detection capability to measure agent concentrations that are considered safe for unrestricted access to food and/or water by the general public, and

2. there are no criteria or monitoring equipment for designating potentially contaminated masonry, wood, or fabric as free of hazardous concentrations.

Because fresh food and water brought in from uncontaminated areas and stored reserves could be substituted for potentially contaminated supplies, the consequences of the first finding above are mitigable. Of more significance is the finding that buildings and personal effects could not now be readily declared free of hazardous concentrations if off-site agent contamination occurs. As a result, the period of restricted access to homes and other personal property would be indefinite, particularly for persistent agents such as VX or sulfur mustard. The economic implications of such private property restrictions are great.

Mustard Carcinogenicity

The tumorigenic potential of sulfur mustard was compared with that of other, better-characterized carcinogens such as benzo(a)pyrene [B(a)P] and N-nitrosodimethylamine (DMNA) to develop an estimate of

relative (carcinogenic) potency. The -limited animal data available indicate that sulfur mustard is likely to approximate B(a)P in its ability to produce malignant tumors in man [i.e., a mustard relative potency factor (RPF) of between 1 and 3 when compared to B(a)P].

With an RPF, it is possible to calculate upper-limit estimates of excess lifetime cancer risk assuming the linear nonthreshold dose-response relationship of Risk = $(Q^*)(D)$, where Risk is the additional individual lifetime risk of developing cancer based on a lifetime of continuous exposure to dose D of a compound with the potency factor Q*. Reasonable assumptions of adult body weight, respiratory volume, and incineration plant operation were made to estimate individual excess lifetime cancer risk to a member of the general public at the site boundary of the Aberdeen Proving Ground during incinerator operation. The calculated upper-limit risk ranged between 8 x 10^{-8} and 2 x 10^{-7}. For comparison, the limit of regulatory acceptability for many drinking water pollutants is a lifetime risk of 1 x 10^{-5}, and the annual fatal accident rate of nominally safe industries in the United States is 1 x 10^{-4}. Excess lifetime cancer risk estimates $<10^{-6}$ are acceptable to most of the population and are not regulated by EPA or FDA.

Antidote Use

Assessment of antidote treatment protocols and toxicity leads us to recommend:

1. Treatment with antidotes should be performed by trained individuals, and only when agent contamination is relatively certain.

2. To prevent substance abuse and poisoning, antidotes should not be distributed to the general public.

B.l. INTRODUCTION

The nerve and blister agents evaluated in this appendix have been especially formulated to cause -major injuries or death to enemy forces in wartime. Agents GA, GB, VX, H, HD, HT, and Lewisite were created to be a significant portion of the U.S. retaliatory stockpile when chemical warfare agents were considered acceptable military weapons. Now, they must be treated in a manner that neutralizes their potential to cause harm to nearby populations and facility workers. This appendix has been composed to provide toxicity information needed for an informed public decision.

The toxicity evaluation addresses the spectrum of health effects that each agent could produce. A finite possibility of a release exists for any disposal alternative (i.e., continued storage, on-site disposal, regional or national disposal installations with their attendant array of transportation options for munitions and storage containers). Unplanned release of chemical agents could result in exposures of Army personnel and the general population in the vicinity of the accident. Effects on individuals in the exposed population could range from transient to life-threatening, depending on such factors as the type and concentration of agent released, the duration of exposure, the availability of antidotes, and individual variations in susceptibility. Existing historical data on chronic subacute exposures to munitions workers have also been compiled,

Appendix B has been revised and updated to address concerns raised during the public comment period concerning variable agent sensitivity among the general population; toxicity of the products of agent hydrolysis, combustion, decontamination and soil microbial activity; threshold vs no-threshold response for cancer induction; noncholinergic response to nerve agents; potential reproductive effects; recommended antidotes, and their dose rates; and expedient personal decontamination procedures.

A number of specialized terms have been used to provide descriptive precision to the biological endpoints observed in standard toxicological tests for each agent. The following text and data tables will make free use of these terms; a glossary is included for the reader's convenience in the Definition of Terms section at the end of Appendix B.

B.l.l NERVE AGENTS (GA, GB, AND VX)

The three nerve agents considered in this analysis are all organophosphorus compounds that affect the nervous system. They are known as GA, or Tabun (ethyl-N,N-dimethyl phosphoramidocyanidate, CAS # 77-81-6); GB, or Sarin (isopropyl methyl phosphonofluoridate, CAS # 107-44-E); and vx (0-ethyl-S-[2-diisopropyl aminoethyl]methyl phosphonothiolate, CAS # 50782-69-g). Usually odorless, colorless, and tasteless, the nerve agents are highly toxic in both liquid and vapor forms.

Physical properties are described- in Table B.l; a complete description of acute and chronic toxicity, antidote use, and recommended decontamination procedures begins in Sect. B.2.

B.1.2 VESICANT (BLISTER) AGENTS (H, HD, HT, AND LEWISITE)

The active ingredient in H and HD and a major component (60%) of HT is the same chemical substance, bis(2-chloroethyl)sulfide (CAS # 505-60-2). Many names, such as mustard gas, sulfur mustard, mustard, yperite, etc., have been applied to this agent. Throughout this analysis we will use the terms mustard or mustard agent (or sulfur mustard to distinguish it from a different compound, nitrogen mustard). Mustard gas is a misnomer because the chemical is a liquid at ordinary environmental temperatures. The chemical warfare agent H is an agent containing 70% mustard and 30% sulfur impurities manufactured by the unstable Levinstein process. The chemical warfare agent HD is purified mustard, from which impurities have been removed by distillation and washing. Agents H and HD will be considered equivalent in the ensuing toxicological discussion and will not be considered separately. Some physical properties of mustard are listed in Table B.l.

Mustard has a garlic-like odor. It has a significant volatility at ordinary temperatures, so that mustard vapor would be in the air immediately surrounding droplets of liquid mustard. Thus, the hazard of human contact is not only with droplets of the liquid mustard agent, but with agent vapors. Because of its low aqueous solubility, mustard agent is very persistent (i.e., remains a hazard for an extended time) in the environment.

HT is a product of a reaction that yields about 60% HD (described above) and <40% T (bis[2(2-chloroethylthio)ethyl]ether, CAS # 63918-89-8). It is a lethal vesicant mixture of greater toxicity and stability than HD. It is very similar in appearance and biological activity to H/HD mustard, but HT is considered to be the more active agent. This agent is also liquid at room temperature but is soluble only in organic solvents. Its poor water solubility makes it a persistent contaminant of soils and surfaces other than rubber, which it readily permeates. Hydrolysis in water occurs only after prolonged boiling, while caustic alkalies hydrolyze HT readily. See Table B.l for a more complete physical description of HT.

Lewisite (Agent L) is an arsenical vesicant (dichloro[2-chlorovinyl]arsine, CAS # 541-25-3) (Table B.l). This agent is approximately 10 times more volatile than HD and can be used as a "moderate irritant" vapor over great distances. Lewisite is liquid at room temperature and is only slightly soluble in water. It is considered of intermediate persistency in soils because of its low water solubility (U.S. Army 1974). It decomposes upon application of heat and may degrade considerably upon shell detonation; it is reasonably stable when stored free of water contamination (U.S. Army 1974).

Table B.l. Chemical and physical properties of chemical munitions

	GA[a,b]	*GB*[a,b]	*VX*[a,b]
Chemical name	Ethyl-N,N-dimcthyl phosphoramidocyanidate	Isopropyl methyl phosphonofluoridatc	O-ethyl-S-(2-diisopropyl aminoethyl) methyl phosphonothiolate
Chemical formula	$C_5H_{11}N_2O_2P$	$C_4H_{10}FO_2P$	$C_{11}H_{26}NO_2PS$
Chemical abstract No.	77-81-6	107-44-8	50782-69-9
Molecular weight	162.1	140.1	267.4
Description	Colorless, odorless liquid	Colorless, odorless liquid	Colorless, odorless liquid
Melting point	− 50°C	− 56°C	− 39°C (calculated)
Boiling point	*245°C*	158°C	298°C
Density (liquid)	1.08 g/mL (25°C)	1.09 g/mL (25°C)	1.0083 g/mL (25°C)
Volatility	610 mg/m^3 (25°C)	22 x 10^4 mg/m^3 (25°C)	10.5 mg/m^3 (25°C)
Solubility. water	98 g/L (25°C) [miscible]	Miscible	30 g/L (25°C) 75 g/L (15°C) miscible <9.4°C
Solubility, other	Very soluble in most organic solvents	Readily soluble in organic solvents	Readily soluble in organic solvents
Biological activity	Lethal anticholincsterasc agent	Lethal anticholinesterase agent	Lethal anticholinesterase agent
Storage location	TEAD	ANAD, LBAD. PBA, TEAD UMDA	ANAD, LBAD, NAAP, PBA TEAD, UMDA
Munition typed	TC	P, R, B. C, TC	P, R, M, ST, TC
	H, HP'	*HT*[a,b]	*Lewisite (L)*[a,b]
Chemical name	Bis(2-chloroethyl)sulfide	Plant-run mixture containing about 60% HD and <40% "T" or Bis-[2-chloroethyl thioethyl]ether	2-chlorovinyl dichloroarsine
Chemical formula	$C_4H_8Cl_2S$	"T" = $C_8H_{16}Cl_2OS_2$	$C_2H_2AsCl_3$
Chemical abstract No.	505-60-2	"T" = 63918-89-8 541-25-3	

Table B.1 (continued)

	GA[a,b]	*GB*[a,b]	*VX*[a,b]
Molecular weight	159.1	"T" = 263.26	207.31
Description	Oily, pale yellow liquid	Clear, yellowish liquid	Liquid with faint odor of geranium
Melting point	13–15°C	0–1.3°C	0.1°C (purified form) (-18.0 to 0.1°C, depending on purity and isomers present)'
Boiling point	215217°C	>228°C (not constant)	190°C
Density (liquid)	1.27 g/mL (25°C)	1.27 g/mL (25°C)	1.89 g/mL (20°C)
Volatility	920 mg/m^3 (25°C)	831 mg/m^3 (25°C)	6.5 x 10^3 mg/m^3 (25°C)
Solubility. water	0.68–0.92 g/L (25°C)	Insoluble	Insoluble (slightly soluble in distilled water)
Solubility, other	Very soluble in organic solvents	Soluble in organic solvents	Soluble in ordinary organic solvents
Biological activity	Blister agent	Lethal blister agent	Lethal blister agent
Storage location	APG, ANAD, LBAD, PBA, PUDA,	ANAD, TEAD. PUDA. PBA TEAD. UMDA	TEAD
Munition type[c]	TC. P. C	TC, S	T C

[a]U.S. Army 1974.
[b]Windholz et al. 1983.
[c]Fox and Scott 1980.
[d]U.S. Army and U.S. Air Force 1975.
[e]B = bombs, C = cartridges, M = mines, P = projectiles, R = rockets, S = shells. ST = spray tanks, TC = ton containers.

A complete description of vesicant agent toxicity, recommended treatment and decontamination procedures is found in Sect. B.3. The breakdown products of these agents are listed and their toxicity described in Sect. B.5.

B.2. NERVE AGENT TOXICITY

B.2.1 SUMMARY

- The nerve agents (GA, GB, and VX) are organophosphorus esters that are usually odorless, colorless, and tasteless. VX is less volatile than the agents GA and GB (Table B.l).

- Nerve agents, toxic in both liquid and vapor form, directly affect the nervous system by inhibiting acetylcholinesterase (AChE), an enzyme that prevents the accumulation of the neurotransmitter acetylcholine (ACh). Additionally, there are other, noncholinergic effects that contribute to the toxicity of nerve agents.

- The clinical signs of nerve agent poisoning result from effects on smooth muscle and glands (drooling, excessive sweating, vomiting, diarrhea, involuntary urination), effects on skeletal muscles (easy fatigue, weakness, muscle twitching), and effects on the central nervous system (headache, confusion, convulsions, and coma). Respiratory failure, the immediate cause of death, appears to result primarily from depression of the brain's respiratory center.

- In terms of acute toxicity to humans by inhalation, the relative potency of nerve agents can be ranked as follows: VX is more toxic than GB, which is more toxic than GA (i.e., VX > GB > GA).

- The current treatment in the United States for acute nerve agent exposure involves the use of atropine sulfate (CAS # 55-48-l) and an oxime , 2-PAM-Cl (CAS # 51-15-O). Research is currently under way to find other oximes or oxime combinations that afford better protection against central nervous system effects.

- Information is meager on the possible chronic health effects from prolonged low-level exposure to nerve agents. Animal and in vitro studies recently completed or currently under way are designed to determine whether these nerve agents can cause mutations, damage fetuses, cause reproductive problems, or generate neurotoxic syndromes such as delayed neuropathy. It is unclear as to whether brain dysfunction (i.e., impairment in memory and ability to concentrate) is a chronic health effect of either acute high-level or prolonged mid-level exposure to nerve agents.

B.2.2 ACUTE TOXICITY

The nerve agents are organophosphorus esters that are among the most potent of the chemical warfare agents. Nerve agents can be absorbed by many routes, and because they are odorless, tasteless, and nonirritating to the skin, their entry into the body may not be perceived by the individual until grave symptoms appear. In vapor or aerosol form, nerve agents can be inhaled or absorbed through the skin or the conjunctiva of the eye; as a liquid, they can be absorbed through the skin, conjunctiva, and upper digestive or gastrointestinal tract (Grob and Harvey 1953). As will be discussed later, the efficacy of the individual agents in skin penetration (percutaneous) varies; however, all nerve agents present a significant contact hazard.

Within seconds of exposure to nerve agent vapor and well before any systemic effects develop, local effects may be observed in the eyes and the respiratory system. Depending on the agent and the dosage, these local effects may be a constriction of the pupils of the eye (miosis) lasting only several hours or a prolonged miosis persisting for many weeks (Rengstorff 1985; Sidell 1974; Taylor 1980). The onset of moderate systemic effects depends in part on the route of exposure: within 30 min following inhalation, within 45 min after ingestion (oral intake), and within 2 to 3 h after application on the skin (Grob and Harvey 1953). Exposure to lethal doses, however, can lead to death within 10 min after a single deep inhalation (Robinson 1967). Following short term or acute exposure, mild systemic effects may last for several hours to days, whereas moderately severe symptoms can last for 1 to 6 d. During the recovery period, symptoms may recur intermittently, particularly following physical exertion (Grob and Harvey 1953). In a study of organophosphate insecticide poisonings, often certain clinical symptoms such as cardiac problems showed a delay in their onset (Hirshberg and Lerman 1984). Delayed or chronic health effects that may result from acute exposure to nerve agents will be discussed in Sect. B.2.3 (Chronic Toxicity).

The toxic effects of nerve agents are due both to their ability to irreversibly inhibit acetylcholinesterase (AChE), an enzyme responsible for the breakdown of the neurotransmitter acetylcholine (ACh), and to other biological effects that appear to have no relationship to AChE inhibition (i.e., noncholinergic effects). The classical description of the mode of action of nerve agents involves the consequences of excessive ACh accumulation at nerve junctions where only minute quantities of ACh are needed for transmission. When ACh accumulates within the portions of the nervous system that control smooth muscle, cardiac muscle, and endocrine-exocrine glandular function, the following symptoms can result: drooling, increased bronchial (lung) secretions, bronchoconstriction, miosis, excessive sweating, vomiting, diarrhea, abdominal cramping, involuntary urination, and heartbeat irregularities (arrhythmias). In addition, ACh accumulation can affect the central nervous system (CNS; the brain and spinal cord), resulting in headache,

anxiety, confusion, restlessness, giddiness, electroencephalographic (EEG) changes, or even convulsions and coma, depending on the agent and the dosage (Grob and Harvey 1953). The third area affected by ACh accumulation is a portion of the.. nervous system controlling skeletal muscles. Thus, acute exposure to nerve agents can also result in a generalized weakness that increases with exertion, as well as muscle twitching and cramping. Examination of selected muscles in rats following single sublethal i.v. or subcutaneous injections of nerve agents has shown that muscle fiber death (necrosis) and marked damage to the nerve-muscle junction develops within 12 to 24 h after injection with regeneration of the muscle tissue beginning at 3 to 4 d (Ariens et al. 1969; Meshul et al. 1985). Respiratory failure, the immediate cause of death in lethal nerve agent exposure, is an example of an effect that occurs as the result of ACh accumulation at several sites in the nervous system, Depression of the brain's respiratory center, neuromuscular block of the respiratory muscles, airway constriction, and increased lung secretions are all recognized as contributing factors to nerve agent-induced respiratory failure, though there is debate as to the relative importance of each factor (Adams, Yamamura, and O'Leary 1976; Karczmar 1985; Krop and Kunkel 1954; Meeter and Wolthius 1968b; Rickett, Glenn, and Beers 1986; Rickett, Glenn, and Houston 1987).

Recent research interest has developed as to the acute behavioral toxicity of nerve agents. In this relatively new field of investigation, animals are tested for changes in motor behavior and learning behavior following exposure to the compound of interest. (The tests designed to measure behavioral effects are many, and the descriptions are too lengthy for detailed discussion here, so this analysis will simply give the results of these tests in general terms.) Gerber and O'Shaughnessy (1986) have demonstrated that certain behavioral tests can be affected by factors such as the systemic toxicity of a chemical (e.g., animals with impaired liver function may "perform" poorly) or by reduction of food and water intake in an animal. Particularly in regard to nerve agents, care must be taken to distinguish between changes in motor behavior that originate from actual CNS effects and changes in motor activity that arise from peripheral actions on muscle function. Also, because tests designed to assess effects on learning behavior often use food as a reward, the possibility of appetite suppression with nerve agent exposure should be investigated before conclusions are drawn.

In a recent review, Karczmar (1984) has listed the CNS effects, including behavioral and mental health effects, that have been observed with anticholinesterase chemicals, including nerve agents. To date, the number of these effects that can be ascribed to nerve agent exposure is limited. The motor effects observed in rats, mice, or guinea pigs following exposure to GA or GB include tremors, hind-limb abduction (extension away from the body), rearing activity, chewing movements, both low-dose increases and higher-dose decreases in spontaneous activity, and decreased coordination (Hoskins et al. 1986; Landauer and Romano 1984; Little et al. 1986; Lynch, Rice, and Robinson 1986; Romano

and Landauer 1986; Rylands 1982). In most cases, these motor effects appeared at levels of nerve agent exposure that caused effects ranging from mild (some salivation, fine tremors) to moderate (excessive salivation and weeping, generalized tremors) in the animals. On the other hand, animal tests for acute effects on learning behavior have shown positive results at exposure levels below those that cause symptoms of nerve agent poisoning (Landauer and Romano 1984; Romano and Landauer 1986). Details of the behavioral toxicity of the individual nerve agents will be discussed in the following sections.

There are "side effects" of the nerve agent-induced ACh accumulation that involve action on other CNS neurotransmitter systems (e.g., norepinephrine, dopamine, gamma-aminobutyric acid), resulting in numerous biological effects (Fernando, Hoskins, and Ho 1984; Hoskins, Liu, and Ho 1986; Sevaljevic et al. 1981; Sivam, Hoskins, and Ho 1984). One such effect is hypothermia, which has been described in agent-exposed rats (Meeter and Wolthius 1968a; Sivam, Hoskins, and Ho 1984). Another is a long-lasting insensitivity to pain (analgesia), observed in mice exposed to sublethal doses of nerve agents (Clement and Copeman 1984). Brain and cardiac lesions found in animals that survive high doses of nerve agents are also thought to result from the release of transmitters other than ACh (McLeod 1985; Singer et al. 1987). These side effects are thought to result from the interplay of the various neurotransmitters within the nervous system (Karczmar 1985; Marquis 1986, pp. 67-68); however, there still exists the possibility that nerve agents exert direct effects on these same systems (Dasheiff, Einberg, and Grenell 1977; Idriss et al. 1986; O'Neill 1981). A second category of potential "side effects" of AChE inhibition arises from the recognition that AChE has enzymatic functions in addition to the breakdown of ACh. Thus, inhibition of the AChE enzyme may produce changes other than simply increased ACh levels (Lohs 1975; Marquis 1986, p. 68). Much of the current research on nerve agents is exploring these ramifications of AChE inhibition (Karczmar 1985).

In addition to the irreversible inhibition of AChE, with its many possible ramifications, there still remain many biological effects of nerve agent exposure in animals that seem to be noncholinergic in origin. These noncholinergic effects are currently being investigated as to their importance in the toxicity of nerve agents, particularly because "...no direct evidence has been produced which unequivocally demonstrates a causal relationship between nerve agent toxicity and AChE inhibition" (Rickett, Glenn, and Houston 1987). In particular, CNS effects (including behavioral toxicity, discussed previously) appear to be independent of either brain AChE inhibition or ACh accumulation (Lynch, Rice, and Robinson 1986; Van Meter, Karczmar, and Ficus 1978). Studies in rats on the temporal relationship between brain AChE inhibition and the presence of certain neurotoxic symptoms (tremors, chewing movements, hind-limb abduction, and convulsions) showed a lack of correlation, which led Hoskins and his colleagues to conclude that other neurotransmitters were involved in these particular neurotoxic

symptoms (Hoskins et al. 1986). Further discussion on the noncholinergic effects of nerve agent exposure can be found in the later section on chronic toxicity of nerve agents (Sect. B.2.3), as well as in recent reviews (Clement 1985; Karczmar 1985; Marquis 1986, pp. 53-71)

Despite new knowledge derived in animals as to the novel cholinergic and noncholinergic effects of nerve agents and related organophosphates, it is still widely accepted that inhibition of AChE is the primary cause in humans of acute toxic responses to nerve agent exposure. For this reason, there have been attempts to measure blood cholinesterase (ChE) activity as an indicator of the magnitude of nerve agent exposure and/or the severity of clinical symptoms, or to monitor the return of blood ChE function as an index of recovery. In none of the above cited cases is there a good correlation. Within the blood, there are two types of ChE activity that can be measured. Estimation of red blood cell (RBC) AChE activity is theoretically preferred because this cholinesterase is similar to the AChE found at the nerve synapses. RBC AChE, however, is replenished only with the formation of new RBCs, and, thus, recovery of RBC AChE activity does not reflect the recovery of AChE activity in the tissues. Measurement of ChE activity in the plasma (pseudocholinesterase) is easier and more accurate than estimation of RBC AChE activity (Namba et al. 1971), but it is less relevant because the inhibition of pseudocholinesterase, which has no known biological function, may not reflect actual AChE inhibition (Daniels 1987). For example, agent vx demonstrates only minor inhibition of pseudocholinesterase, preferentially inhibiting AChE (Goudou and Rieger 1983; Sidell and Groff 1974).

The range of blood ChE activity (both plasma and RBC) in unexposed individuals is so wide that it is not possible to determine from a single test whether a person has had a recent exposure to a cholinesterase inhibitor, especially if the exposure is minor (Wolfsie and Winter 1952). When individual baseline (prior to exposure) blood AChE activity is known and is compared with the AChE activity following nerve agent exposure, there does appear to be a relationship between AChE inhibition and dosage at a limited range of acute sublethal doses. There appears to be a better correlation between brain AChE inhibition and the degree of toxicity, in that GA and GB injected into rats produced a dose-dependent inhibition of brain AChE, with lethal doses producing >90% inhibition (Sivam, Hoskins, and Ho 1984). Although brain AChE activity may reflect more closely than blood AChE the dose response to nerve agent exposure, brain AChE monitoring has no practical application in situations of human exposure, owing to the difficulty and hazards of sampling.

Because experimental human exposure data are not available on the lethal dosage of the nerve agents discussed here, animal toxicity data must instead be considered. The dosage levels-of GA, GB, and VX that result in 50% lethality (LD_{50}, LCt_{50}) in animal populations given an acute exposure by a variety of routes [inhalation, skin, intravenous (i.v.)] are presented in Table B.2. The designation of the route of

Table B.2. Acute toxk levels of nerve agents

Exposure route	GA	GB	v x	vx (aerosol)
		Inhalation. LCt_{50} (mg-min/m³)		
Human, mild activity (estimated)	135–150[a,b]	70[a]	30[a]	20–50[c]
Human, resting (estimated)	200–400[a,d]	,00[a,d,e]	36'	
Monkey	187'	74[a]		~50[a]
Dog	320[a]	60[a]		15[a,f]
Rabbit	960[a]	120[a]		25[a,f,g]
Guinea Pig		180[a]		8, 30[a,f,h]
Rat	450[a]	220[a]		17[a,f]
Mouse		240, 310[a,i]	40[a]	7'
		Percutaneous. LCt_{50} (mg-min/m³)		
Human, clothed (estimated)		15,000[a]	60. 3,600[a,j]	75, 300[c,k]
Human, bare skin (estimated)	~20,000–40,000[a,d]	12,000[a]	6. 360[a,j]	
Dog, clipped			4.6, 89[a,l]	3.5, 31.8[a,m]
Rabbit, clipped		2,000[a]	8.3, 28[a,n]	124. 180[a]
Rabbit, clipped and clothed			539[o]	
		Skin, LD_{50} (mg/kg)		
Human (estimated)	14–21[p]	24[a]	*0.04'*	
Monkey, shaved	9.3.		-0.065'	
Pig, clipped		115.9[a]	<0.40[a]	
Dog, depilated	~45[a]	10.8"	0.054.	
Cat, depilated		6.2[a]	0.012[a]	
Rabbit, depilated	3[a]	4.4[a]	0.025, 0.205'	
Rat, depilated	12.6"	2.5'	0.10[a]	
Mouse. depilated		1–9.2[a,s]	0.046'	
		Intravenous. LD_{50} (mg/kg)		
Human (estimated)	0.014[b]	0.014.	0.008'	
Monkey	-0.05.	0.020[a]	0.0084'	
Goat		0.015[a]	<0.005[a]	
Dog	0.084.	0.010[a]	0.0063~	
Cat		0.015–0.018[a,t]	- 0 0025[a]	
Rabbit	0.063[a]	0.0147'	0.0084[a,q]	

Table B.2 (continued)

Exposure route	GA	GB	VX	VX (aerosol)
Rat	0.07[a]	0.045[a]	0.0079[a]	
Mouse	0.311[v]	0.07–0.113[v,w,x]	0.0141[a]	
		Oral, LD_{50} (mg/kg)		
Rat		1.06	0.10[a]	
		Incapacitating dose, ICt_{50} (mg-min/m^3)		
Human, inhalation, mild activity (estimated)	100[d]	35–72[a,d,p]	24[a]	5–15[c]
Human, inhalation, resting (estimated)	300[o]	75[p]		
Human, percutaneous, clothed (estimated)		8,000[a]		30–150[c,k]
		Minimum effective dose—miosis, ECt_{50} (mg-min/m^3)		
Human, inhalation (estimated)	0.9[a]	2–4[e]	0.09[y]	
		Minimum effect dose—tremors, ECt_{50} (mg-min/m^3)		
Human, inhalation (estimated)		4[e]	1.6[y]	
		Minimum effective dose—miosis, ED_{50} (mg/kg)		
Human, skin dose (estimated)			0.0314[a]	

Table B.2 (continued)

Exposure route	GA	GB	VX	VX (aerosol)
	No-effects dose-miosis ($mg\text{-}min/m^3$)			
Human, inhalation (estimated)		0.5[i]	0.02[y]	

[a]U.S. Army 1974.
[b]Robinson 1967.
[c]Krachow 1956.
[d]Dick 198 I.
[e]McNamara and Leitnakcr 197 1.
[f]Only head exposed.
[g]Wind speed 0.01 mph.
[h]Wind speed 15 and 0 mph, respectively.
[i]Active, resting, respectively.
[j]Masked, wind speed 15 and 1 mph. respectively.
[k]Masked. wind speed 10 and 0 mph and particle size 15, 5μm, respectively.
[l]Wind speed 20 and 0 mph, respectively.
[m]Wind speed 15 and 5 mph, respectively
[n]Wind speed 8 and 0 mph, respectively.
[o]Musselman et al. 1967, clothed in cotton sateen over cotton t-shirt.
[p]U.S. Army 1975.
[q]Fielding 1960.
[r]Wiles and Alexander 1960; bare, clipped; and clothed, unclipped, respectively.
[s]Loomis and Salafsky 1963.
[t]Murtha and Harris 1980.
[u]O'Leary, Kunkel, and Jones 1961.
[v]Schoene and Oldiges 1973.
[w]Sammet 1983.
[x]Little et al. 1986.
[y]McNamara, Vocci, and Leitnaker 1973.

exposure is important, for there are differences in absorption and/or degradation with different avenues of entry into the body. Although the i.v. route does not pose a relevant route of accidental exposure of man, inclusion of these data in Table B.2 serves to establish the intrinsic toxicity of an agent without species or individual variations in absorption. Also included in Table B.2 are estimates of dosages, based on animal data, that could cause 50% mortality in human populations exposed by some of these same routes. The LD_{50} values (or the estimated values) are given on a milligram of agent per kilogram of body weight (mg/kg) basis, so that comparisons between species (particularly comparisons of animal data with human estimates) are possible. Table B.2 also contains information on the estimated median incapacitating dose, the minimum effective dose, and the no-effects dose for each nerve agent (see Definition of Terms).

B.2.2.1 Agent GA

According to Robinson (1967), GA, or Tabun, was the first nerve agent developed for chemical warfare. Its toxicity has been surpassed by the toxicity of newer G agents such as GB, which are also more resistant to hydrolysis. GA contains cyanide instead of fluoride in its structure. Agent GA is stored at only one CONUS site (TEAD), and its contribution to the total stockpile is quite small. The concerns about human health hazards presented by GA, therefore, are relatively minor compared with those of GB and VX.

In Table B.2 the lower animal toxicity of GA compared with GB is evidenced by the higher LCt_{50} and LD_{50} values for GA, meaning that more GA is necessary to cause 50% lethality in an exposed animal population. Within certain animal species and with certain exposure routes, GA and GB appear to be equally toxic; however, this may be because of the method of determining the LD_{50} values or the species variation in susceptibility, GA differs from other G nerve agents in some of its biochemical effects on the brains of exposed animals and also in the rarity of GA-induced convulsions, even at lethal doses (Liu et al. 1986). GA has been found to cause acute motor and learning behavior effects in rats at doses between 54 and 71% of the LD_{50}, with certain behavioral changes occurring in the absence of obvious symptoms of nerve agent exposure (Romano and Landauer 1986).

The detoxification or breakdown of GA within the body proceeds at a low rate (U.S. Army 1975), by way of an enzyme designated tabunase, which has been identified in several species, including man (Augustinsson and Heimburger 1954).

As mentioned in the introduction to this section, the human doses for lethality (LD_{50}) or incapacitation (ICt_{50}) provided in Table B.2 are merely estimates (U.S. Army 1974; Dick 1981). This is evident in a comparison of the inhalation incapacitating dose (ICt_{50}) and the range given for the lethal dose (LCt_{50}) for GA in resting man (breathing 10 L/min); the incapacitating dose falls within the range for the lethal

"dose." The human toxicity by inhalation of GA vapors is approximately half that of GB (Table B.2); this difference is well supported by the animal data. GA appears to be more toxic to the ciliary muscles of the eyes than GB because constriction of pupils occurs at a lower concentration of GA [i.e., minimum effective doses of 0.9 and 2 to 4-mg min/m^3, respectively (U.S. Army 1974)]. The higher estimated LD_{50} value for GA toxicity for humans by skin absorption is roughly equivalent to the estimate for GB, and the i.v. human LD_{50} estimate for GA is equal to that for GB. The equivalencies of these estimates are not necessarily supported by the animal data, but no discussions of the bases for the human estimates are given in the source document (U.S. Army 1974).

GA is less volatile than GB (Table B.l) and would be expected to remain on the skin and in the environment somewhat longer. Although it is not as persistent as VX, under certain weather conditions (light breeze, 20°C or 66°F) GA can remain in the environment from 1 to 4 days (Dick 1981), so that a temporary problem of persistence could occur following an unplanned release.

B.2.2.2 Agent GB

Agent GB, or Sarin, is the most studied of the three nerve agents considered in this analysis. Because of the high volatilit and expected rapid dispersion of this highly toxic agent, GB is also the agent of greatest concern in an unplanned release. Compared with the other nerve agents considered in this analysis, GB is more toxic than GA and less toxic than VX. GB is very effective as a toxic inhalant but somewhat less effective as a skin penetrant, because it evaporates so rapidly from the skin. Because the general acute toxicity of nerve agents has been described above, this section will discuss only the specific features of GB toxicity.

GB is a very rapidly acting toxicant; there is little difference between the 15-min and the 24-h lethal dose for animals by i.v. injection (Fielding 1960). Although GB is less toxic than VX by a variety of exposure routes (see Table B.2), GB may actually be more toxic than VX at the neuromuscular junctions. When GB or any one of several V agents related to VX was applied directly to the isc'ated rat diaphragm at the junction of nerve and muscle (thereby eliminating factors such as absorption efficiencies and attenuation differences), GB was found to be twice as potent as the V agents (Stewart 1956). Under conditions of i.v. infusion of GA, GB, and VX at the rate of one LD_{50} per 15 min in cats, the equivalent amount of each agent sufficient to induce respiratory arrest was 0.5 LD_{50} for GB whereas 1.25 and 15 LD_{50}s were needed for GA and VX, respectively (Rickett, Glenn, and Beers 1986). These differences reflect the rapidity of the toxic action of GB compared with VX and the higher toxicity compared with GA. GB is thought to act primarily on the peripheral nervous system; however, the respiratory arrest described previously was mediated through effects on the central nervous system. GB is very efficient at producing central

respiratory arrest in guinea pigs and cats at i.v. doses too low to cause an effect on the respiratory muscles (Murtha and Harris 1980).

Compared with VX, GB is less stable once it enters the body, and, thus, less of the agent actually reaches the target tissues. One source of this attenuation or detoxification in certain animal species is an enzyme in the plasma called aliesterase (AE). AE combines rapidly with GB and prevents it from interacting with AChE. In rats, 10 min after i.v. injection of radiolabeled GB, approximately 70% of the activity of the plasma is bound to large protein molecules identical to AE (Polak and Cohen 1970). Pretreatment of rats with triorthocresylphosphate, a chemical that irreversibly blocks AE, resulted in enhancement of GB toxicity 6 to 8 times (Polak and Cohen 1969). Moreover, less GB was found in the plasma and more in the brain, muscles, kidneys, and lungs of pretreated rats as compared to rats that received no pretreatment. Similar AE modification of GB toxicity has been observed in guinea pigs and mice, although guinea pig plasma AE binding capacity for GB is lower than that of rat plasma (Christen and Cohen 1969; Fonnurn and Sterri 1981). The presence of AE in rodent plasma may by itself account for the relative resistance of mice and rats to GB toxicity compared with other animal species (see Table B.2 for LD_{50} values). Human plasma does not contain AE. Grob and Harvey (1958) calculated from human studies with GB that there is very little detoxification when GB is injected into the bloods. eam. With this major difference between rodents and humans as to the (. ification of GB, the uncertainty of estimating human LD_{50} value: om data obtained in rodents is apparent.

Metabolism studies of GB have been carried out in dogs and mice. In mice, there was a rapid hydrolysis of the intravenously injected GB, so that within 1 min, less than 10% of the GB found in the tissues was nonhydrolyzed GB (Little et al. 1986). This rapid hydrolysis of GB may be due again to the plasma aliesterase, but this has not been established. There is still a question of the relevancy of metabolism studies done in an animal species that is relatively resistant to GB toxicity. Metabolism studies in dogs demonstrated that the main product of GB detoxification is isopropyl methylphosphonic acid and that this compound accounts for the majority of GB activity found in brain tissue (Fleisher, Harris, and Berkowitz 1969).

Like all other nerve agents, GB combines with and inhibits AChE, resulting in the accumulation of ACh. GB has been used, however, in many studies designed to determine how great a role AChE inhibition plays in nerve agent toxicity. From human studies in which small quantities of GB were injected directly into the bloodstream, Grob and Harvey (1958) calculated that about 75% of GB combines with AChE in the muscle, about 22% with blood ChE, and about 3% with AChE in brain and liver. The inhibition of blood ChE has no resulting toxicity (i.e., there are no neurotransmitter receptor sites in the blood). It is the GB in muscle and brain that causes the symptoms of nerve agent exposure. Within the muscles of cats, GB was found to cause a dose-dependent inhibition of AChE activity; however, there was not a simple relationship between AChE

inhibition and alteration of muscle function (Brimblecombe, French, and Webb 1979).

Once GB is in the blood, it is capable of permeating the brain's natural protective barrier to foreign substances (the blood-brain barrier). Cholinesterase inhibitors vary in their ability to pass through this barrier, a property that has been related to the lipid solubility of the compound (Holmstedt 1959, p. 603). Within the brains of mice injected intramuscularly with GB, regional differences in AChE inhibition were observed (Bajgar 1971, 1972). Bajgar concluded that the differences in AChE inhibition were due to regional differences in GB penetration rather than a selectivity of GB for AChE in specific parts of the brain. Regional differences in AChE activity have also been observed in isolated, perfused dog brains administered GB (Singh, Zelezniker, and Drewes 1986) and in the brains of dogs following i.v. injection of GB (Fleischer, Harris, and Berkowitz 1969). Studies in rats demonstrated that over 94% of GB bound to AChE in the brains 30 min after injection is actually isopropyl ❑ ethylphosphonic acid, a metabolite of GB (Fleisher, Harris, and Berkowitz 1970).

There appear to be mechanisms other than (or in addition to) AChE inhibition responsible for the observed toxicity of GB to the brain. In rats, Harris et al. (1967) reported that 51% of the GB found in the brain was bound to sites other than AChE. In studies of spontaneous recovery from central respiratory failure in guinea pigs, there was no association of respiratory recovery with recovery of brainstem AChE levels (Adams, Yamamura, and O'Leary 1976). Adams and his colleagues concluded that the recovery occurred through a desensitization of the ACh receptors to the excess ACh, but it is also possible that AChE inhibition was not actually responsible for the initial respiratory failure. GB has been shown to cause a number of noncholinergic effects in the brain, including effects on other neurotransmitters and enzymes. Most effects are too detailed to discuss individually in this analysis, but all serve to make the point that GB does much more than simply inhibit AChE in the brain (Dashieff, Einberg, and Grenell 1977; Fernando, Hoskins, and Ho 1984; Flynn and Wecker 1986; Hoskins, Liu, and Ho 1986; Liu et al. 1986; Sivam, Hoskins, and Ho 1984; Zhao et al. 1983).

GB has been tested in several animal species for behavioral toxicity. In these studies, the animals were given low doses of GB, so that the general signs of nerve agent poisoning would not mask the subtle behavioral changes. In mice and rats, many of the motor behavioral effects, such as hypoactivity (decreased spontaneous activity), rearing movements, and loss of coordination, were manifest at doses that produced other mild effects such as temporary tremors; whereas in conditioned learning tests, behavioral effects appeared in the absence of other toxic symptoms. Wolthuis and Vanwersch (1984) tested several anticholinesterase compounds at low doses for acute effects in rats, using six behavioral tests. GB was without effect at doses up to 30% LD_{50}; however, several other organophosphates and

organophosphorus compounds produced behavioral effects within the 3% to 10% LD_{50} range. Guinea pigs showed impaired performance in a swimming test with GB administration at a dose causing excessive salivation and general tremors in some animals (Rylands 1982). Studies in marmosets showed that in the dose range of 33% to 55% of the LD_{50}, there were significant decreases in a food-reinforced visually guided reaching response (D'Mello and Duffy 1985).

Data on human responses to GB come from accidental exposures and limited studies on low doses of GB given to volunteers. In one incident of accidental exposure to GB vapors, two men had significant blood ChE lowering for 90 d and extreme miosis (pupillary constriction) that persisted for 30 to 40 d but no other signs or symptoms of nerve agent poisoning (Rengstorff 1985). Other accidental exposures to GB were described by Sidell (1974): in one, the victim manifested severe symptoms and required respiratory assistance and extended hospitalization; in the other, three victims suffered some temporary symptoms, such as transient mild respiratory distress, together with marked miosis and blood ChE depression that required several months for full recovery.

Grob and Harvey (1958) reported on the effects in man of the administration of low doses of GB. When either 0.003 or 0.005 mg/kg of GB was injected directly into an artery in the arm, Grob and Harvey observed some initial local effects (reduction in grip strength, tremors following exercise) followed by systemic effects, including many of the symptoms listed earlier in Sect. B.2. These doses, which correspond to 21% and 36% of the estimated human i.v. LD_{50}, resulted in RBC AChE reduction to 52% and 28% of original activity and a plasma ChE reduction to 61% and 42%, respectively. When GB was given orally to the volunteers, approximately 3.5 times as much GB was needed to produce the same degree of blood ChE depression as observed with injection into the artery. With the oral administration, Grob and Harvey noted a very narrow margin between the doses that produce mild symptoms and those that produce moderately severe symptoms. They also noted that following the disappearance of symptoms, there remains an increased susceptibility to further exposure to GB within 24 h of the first exposure. The possibility of oral exposure of the population to GB is remote because GB persists as a ground contaminant only in very cold conditions (Dick 1981).

As mentioned previously, GB is less effective as a toxic skin penetrant than as an inhalant. The estimated LCt_{50} for percutaneous toxicity is 158 times higher than the estimated LCt_{50} for inhalation (Table B.2). Rapid evaporation from the skin is the primary factor in the relatively low percutaneous toxicity observed; with the prevention of evaporation (i.e., covering the exposed skin), the toxicity of GB increases almost a hundredfold (Ainsworth 1954). Another factor limiting the percutaneous toxicity is the reaction of GB with skin constituents, thereby attenuating the amount of GB that reaches target tissues (Freeman et al. 1953). Fats such as lanolin and lard have been shown to

enhance the skin penetration of GB, probably by dissolving the agent and by preventing evaporation (Marzulli et al. 1957). Mechanical abrasion of rabbit skin increased GB percutaneous toxicity a hundredfold (Marzulli and Williams 1953). Fielding (1960) relates an unfortunate incident that exemplifies the individual variability in percutaneous absorption of GB. Seventeen of 18 men exposed to 200 mg of GB through two layers of clothing showed no symptoms of GB poisoning; the other man died, despite treatment when symptoms of nerve agent poisoning appeared (Cullumbine et al. 1954).

In a review of GB toxicity, McNamara and Leitnaker (1971) state:- "Absorption through the conjunctiva causes local effects but negligible systemic effects." Grob and Harvey (1958) instilled 0.0003 mg GB in the eyes (conjunctival sacs) of volunteers and noted a marked miosis that began at 10 min and slowly diminished over the period of 60 h. At a dose of 0.0009 mg, the pupillary constriction that occurred was near maximal for 72 h and did not disappear until after 90 h. No depression of blood ChE activity was noticed at either dose level, In studies on GB applied to the eyes of guinea pigs, a rapid depression of AChE activity in the iris and cornea was noted with a lesser inhibition of AChE in the retina, but no mention was made of RBC AChE depression or other systemic effects in the treated guinea pigs (Lund-Karlsen and Fonnum 1976). There are, however, ocular LD_{50} values available for several animal species that are equivalent to the LD_{50} values for subcutaneous injection (U.S. Army 1974). This suggests that systemic effects are possible with GB absorption through the conjunctiva and possibly the cornea of the eye.

The estimated dose level of GB that results in incapacitation rather than death (Table B.2) shows the same potency relationship to GA and VX as observed for the lethal dose (i.e., VX > GB > GA, where > means more toxic). Studies of the retention and absorption of GB vapors by men either resting or exercising demonstrated that there was a higher percentage of the inhaled GB retained by the inactive men (Oberst et al. 1968). Under similar exposure conditions of time and concentration, however, a larger dose of GB was received by the active men because of their faster breathing rate.

In determining the lowest concentration of GB that produces a biological effect, miosis (constriction of the pupils) is used as a sensitive indicator of nerve agent exposure. There are several questions surrounding the validity of the derived no-effects and minimum effective concentrations for miosis by GB. The basis for these determinations by McNamara and Leitnaker (1971) are to be found in a report by Johns (1952) in which volunteers were exposed to low concentrations of GB (maximum = 6 $mg\text{-}min/m^3$), and the effects on pupil diameter were measured. The design of the exposure studies was faulty and the data obtained were insufficient to confidently predict concentrations of GB that would cause miosis in half of the exposed population (ECt_{50}) or in none of the population (no-effects level).

B.2.2.3 Agent VX

Agent VX is the most lethal of all the nerve agents discussed in this document. As a group, the V agents (V for venom) are more stable than the G agents, more resistant to detoxification, less volatile, more efficient at skin penetration, and more environmentally persistent. Because of these characteristics, VX is most effective as a skin penetrant and lethal contact agent. Under conditions favorable to skin penetration, VX can be about 1000 times as toxic as GB in rabbits (Fielding 1960). The evaporation of VX from the skin is almost negligible, whereas GB evaporates in a matter of minutes (see Table B.l for comparative volatilities). GB penetrates the skin more rapidly than VX, but VX suffers virtually no degradation as it slowly penetrates the skin; thus, more of this compound is able to reach the bloodstream (van Hooidonk et al. 1980). Whereas GB skin penetration in rabbits appeared to be complete by 30 min (Freeman et al. 1953), with a penetration efficiency of nonevaporating GB calculated to be 0.04% (Fielding 1960), complete penetration by VX with essentially 100% of the skin dose reaching the circulatory system required about 4.5 h (Marzulli and Wiles 1957). In vitro studies suggest that VX can penetrate in unaltered form through the epidermis and dermis of the skin, penetrate through the nerve membranes, and accumulate within the nerve cells (Farquharson et al. 1980).

Once inside the body, VX not only inhibits AChE (resulting in accumulated ACh, which then reacts with the ACh receptors) but also reacts directly with the ACh receptors and other neurotransmitter receptors (Chen and Chi 1986; Idriss et al. 1986; Zhao et al. 1983). Although the other nerve agents (GA and GB) react with the ACh receptor in a manner similar to ACh itself, VX counteracts the effects of ACh (Rickett, Glenn, and Houston 1987). As stated in the introduction to this section, the interaction of all three nerve agents with components of the nervous system is complex and multifaceted and cannot be described as simply the inhibition of AChE.

Another contributing factor to the high toxicity of VX is its very specific reaction with, and subsequent inhibition of, AChE. Unlike the other nerve agents, it fails to show significant reaction with pseudocholinesterases or other enzymes in the body (Goudou and Rieger 1983; Sidell and Groff 1974); the result is that more VX is available to react specifically with the target enzyme, AChE.

A number of investigators have reported the distinctly slower toxic action of VX as compared with the G agents, both in terms of onset of symptoms and in recovery (Rickett, Glenn, and Houston 1987; Weger and Szinicz 1981). This delay cannot be attributed completely to slower skin penetration, because delayed toxicity is also observed when VX is administered intravenously (Rickett, Glenn, and Beers 1986). With GB there is essentially no difference between the 15-min and 24-h lethal intravenous doses; with VX there is approximately a twofold difference (Fielding 1960). It is important, therefore, in determining LD_{50} and

LCt_{50} levels for VX to allow enough time to accurately assess the toxicity. Although the basis for this delay is not explained fully at this time, Fielding (1960) conjectured that its larger molecular size and different solubility characteristics may cause VX to be slower in its diffusion through tissues and cell membranes to the target tissues.

Two other features of VX toxicity are worthy of mention. In a review of VX toxicity, Ross et al. (1982) noted that VX has been shown to have a direct effect on the heart, independent of its AChE-inhibiting potency. Direct effects of other organophosphorus compounds on the heart had been observed earlier by Wolthius and Meeter (1968). Recently, Robineau and Guittin (1987) have investigated the cardiac toxicity of VX injected subcutaneously into dogs (1.5, 3.0, or 6.0 $\mu g/kg$). In addition to the expected cardiac effects resulting from AChE inhibition, there was a significant effect of VX on ventricular function through an unknown mechanism. Also in the same review by Ross et al. (1982) and in a study by Sidell and Grof (1974), the authors described another feature of VX toxicity--a spontaneous reactivation of a portion of the VX-inhibited AChE, meaning that, with time, there is dissociation of some of the agent and the enzyme, allowing the AChE enzyme to function again. In the study by Sidell and Groff (1974), spontaneous reactivation of RBC AChE proceeded at a rate of about 1%/h over the first 70 h. In a recent study in mice by Goudou and Rieger (1983), the authors failed to find any spontaneous reactivation under a *variety* of *test* conditions. Their results suggested that the recovery of muscle AChE function after inhibition by VX was actually due to an initial rapid synthesis of AChE (3 to 15 h) followed by a slow phase of AChE return.

According to Ross et al. (1982), the LD_{50} values for humans have been derived from mathematical models, extrapolations from animal data, and estimations from sublethal experimentation in humans. Many of the reports in which these human values are derived are confidential reports and unavailable for review. Ross et al. (1982) note that AChE levels have been used as indicators of VX toxicity in humans and indicate that extrapolations to LD_{50} values based on AChE levels should be interpreted with caution because of the poor correlation of AChE levels and toxicity in animals.

In comparison with GB human exposure estimates, VX is estimated to be approximately twice as toxic by inhalation, 10 times as toxic by oral administration, and approximately 170 times as toxic following skin exposure (National Research Council 1984). Estimates are available for human lethal inhalation doses of VX in both aerosol (small particles) and vapor (gas) phases (Table B.2). Animal inhalation data are available primarily for VX aerosol. In most cases, only the animals' heads and not their total bodies were exposed, so as to limit the skin absorption of VX. The mouse LCt_{50}s for both vapor and aerosol were obtained with total-body exposure; in the case of VX aerosol, skin absorption appears to contribute to the total toxicity. The estimated human LCt_{50} values are equivalent for vapor and aerosol. However, it would probably be difficult to achieve a high vapor concentration of VX because of its low

volatility; therefore, it is likely that a greater time of exposure to VX vapor would be necessary to achieve the same toxicity.

Percutaneous (skin) absorption is a more likely route of VX exposure than inhalation; moreover, percutaneous toxicity is more likely to occur from the absorption of VX aerosol or liquid than from the vapor. The LCt_{50} estimates for percutaneous absorption are established by exposure of animals to VX vapor or aerosol in a special chamber in which only the body is exposed. The animals are often shaved, clipped, or depilated prior to exposure in order to approximate human skin exposure, and the wind speed within the chamber is varied to simulate a range of meteorological conditions. The effect of wind speed on the percutaneous LCt_{50} values is quite large, in that under windy conditions, lethal effects are detected at much lower concentrations of VX. In the dog, a 20-mph wind speed compared with still air results in a 20-fold reduction in the LCt_{50} values; in the rabbit, the LCt_{50} determined with an 8-mph wind speed is 3.4 times lower than that obtained in still air (Table B.2). Although wind speeds of 20-mph may never be encountered in an unplanned release of VX, it is important to realize that wind speed can significantly increase the percutaneous toxicity of VX. Another way of determining VX percutaneous toxicity in animals is to apply liquid VX directly to the bare skin. The LD_{50}s for skin absorption are quite similar regardless of species (Table B.2).

The range in the estimated human percutaneous LCt_{50} values listed in Table B.2 encompasses the influence not only of wind speed but also of particle size and degree of skin exposure (clothed or bare). Animal data primarily show the influence of wind speed. Krackow (1956) has calculated percutaneous LCt_{50}s for men wearing gas masks so that only the neck, ears, hands, and wrists are exposed to aerosol particles ranging in size from 5 to 15 μm at wind velocities from 0 to 10 mph. [The micrometer (μm), often referred to as micron (μ) in the literature, is equal to 10^{-6} m or 10^{-3} μm.] Under these conditions, the lower LCt_{50} (75 mg-min/m^3) is estimated for the larger particles with the higher wind velocity, while 300 mg-min/m^3 is estimated for the smaller particles at the lowest wind speed. Other data of Krackow (1956) suggest that, with 10-μm particles and 20-mph winds (or 20-μm particles and 10-mph winds) the LCt_{50} might be as low as 10 mg-min/m^3.

The human percutaneous LCt_{50} values listed in Table B.2 for VX vapor are actually estimates for lethality for VX vapor containing 2-μm particles (U.S. Army 1974). These exposure conditions represent a hybrid vapor/aerosol condition. Nonetheless, Fielding (1960) considers that the percutaneous LCt_{50}s for VX vapor and VX aerosol would be similar under conditions of high concentrations in air and short exposure times. With lower vapor concentrations, Fielding (1960) notes that "doubt has been expressed regarding the percutaneous lethality of VX vapor to humans in view of the possibility that the excretion rate may be greater than the skin-absorption rate."

The amount of skin surface exposed to VX vapor or aerosol is of obvious importance. Studies in rabbits with VX vapor or with direct application of liquid VX have shown the protective effect of clothing (see Table B.2). Clothing is estimated to reduce by tenfold the percutaneous vapor toxicity of VX. Not all body areas, however, are equal in their permeability to VX. The doses of VX necessary to cause 70% inhibition of AChE when applied to equal areas of the cheek, forehead, abdomen, and volar surface (i.e., palm side) of the forearm have been estimated to be 5.1, 11.2, 31.8, and 40.0 mg/kg, respectively (Sim 1962). The differences in absorption are important to remember in evaluating studies in which the forearm is exposed and extrapolation; are made to total-body exposure. Craig, Cummings, and Sim (1977) have measured the percutaneous absorption of liquid VX through the cheek and the forearm of men at environmental temperatures ranging from -18 to 46°C. The fraction of the applied dose that penetrated in 3 h ranged from 3.5% at -18°C to 31.9% at 46°C for the cheek and from 0.6% at +18°C to 2.9% at 46°C for the forearm. Because the skin can act as a storage depot for VX with movement from this depot promoted by increased temperature, the authors suggest that cooling of the skin surface following percutaneous exposure to VX can delay absorption until treatment is possible. It should also be noted that decontamination is a particularly important treatment for percutaneous VX exposure because of the slowness of its skin penetration. Further discussion of appropriate decontamination following VX exposure can be found in Sect. B.2.4.4.

The estimated i.v. LD50 for humans (0.008 mg/kg) is similar to that determined for many animal species, with the major exception of the mouse (Table B.2). Low doses of VX (.001 mg/kg) have been injected intravenously into Army volunteers to try to correlate the dose of VX received with the amount of AChE inhibition or the presence of clinical symptoms. By injecting VX directly into the bloodstream, the wide differences in individual skin absorption that have been observed in other studies (Craig, Cummings, and Sim 1977) were bypassed. In four men who received 0.001 mg/kg VX in a 4-h infusion, there was good agreement as to the individual percent decrease (50%) in RBC AChE levels compared with preinfusion AChE levels (Kimura, McNamara, and Sim 1960). (It is not possible to compare the closeness of the absolute AChE values because these were not given). In another study, reported by Side11 and Groff (1974), a group of 34 men were given doses ranging from 0.0012 to 0.0017 mg/kg in an attempt to find an i.v. dose of VX that would cause 75% inhibition of RBC AChE levels. In this dosage range, most subjects had transient symptoms of lightheadedness and some had nausea and vomiting, with the most prominent symptoms occurring 1 h after injection, when the RBC AChE inhibition was maximal. A dose of 0.0015 mg/kg was found to produce 75% inhibition of the baseline AChE levels.

There is a paucity of data on the oral toxicity of VX, despite the fact that the environmental persistence of this agent makes this a relevant route of human exposure. VX can persist on leaf surfaces in an undegraded form, so that animals grazing on the contaminated vegetation

ingest VX (Ross et al. 1982). In a report on VX persistence in soil following shell bursts, at 46 d after contamination, there was still sufficient VX to kill 4 of 10 guinea pigs fed grass from the contaminated area (Dewey and Fish undated, as cited in Fielding 1960). Four months were required for the AChE levels in sheep accidentally exposed to WI-contaminated vegetation to return to normal (van Kampen et al. 1970). Almost a year elapsed before normal AChE levels were observed in swine that foraged on the contaminated ground (Ross et al. 1982). The only animal oral LD_{50} value available is 0.1 mg/kg for rats, 10 times the oral toxicity observed for GB (Table B.2). In human volunteers given oral doses of VX in water, a dose of 0.004 mg/kg was necessary to cause a 70% reduction in RBC AChE levels (Side11 and Groff 1974). This oral dose is about three times the i.v. dose needed to cause a similar level of AChE inhibition. At oral doses ranging from 0.002 to 0.0045 mg/kg, only a few subjects suffered any gastrointestinal symptoms, and there were no changes in heart rate, blood pressure, or pupil size in any of the subjects. Eating prior to drinking the VX solution appeared to enhance the AChE inhibition; tap water (as compared with a 5% dextrose solution) seemed to retard the anti-AChE activity.

Estimates have also been made for incapacitating doses (ICt_{50}) for humans either by inhalation or by skin absorption of VX aerosol or vapor (Table B.2). As explained in the footnotes to Table B.2, the ranges in these various estimates encompass different particle sizes (in the case of aerosols) and different wind velocities. Fielding (1960) notes that these ranges may also depend on what is meant by incapacitation. Estimates have been made for the lowest air concentration of VX that produces miosis, one of the more-sensitive indices of exposure to the vapors of anticholinesterase compounds. The estimated ECt_{50} found in Table B.2 for pupillary constriction by VX is an extrapolation from the derived value for GB in humans (which may not itself be sound). To obtain the VX ECt_{50} for humans, the GB "dose" for miosis in humans was first compared with the minimum dose of GB that causes miosis in rabbits, and the assumption was made that man is twice as sensitive as the rabbit. This factor of two was then applied to the concentration of VX that produces pupillary constriction in rabbits, to arrive at the minimum concentration of VX that would be expected to cause miosis in humans (McNamara, Vocci, and Leitnaker 1973). Similarly, the VX no-effects doses for ❑ iosis and for tremors are based on extrapolations from derived values for GB. Because. these VX "doses" are used to determine presumed safe levels for human exposure to VX, there should be more research to determine whether these minimum and no-effects values are truly credible.

B.2.2.4 Mixtures

At several of the storage depots (LBAD, ANAD, PBA, TEAD, and UMDA) both GB and VX are in the stockpile. So the question arises as to toxic effects of a GB-VX mixture if they were simultaneously released. In the

only report found to date that addresses this issue, GB and VX were administered simultaneously and sequentially to mice (Boskovic et al. 1981). When the agents were administered as a mixture of 0.5 LD_{50} of each agent, the resulting mixture had an LD_{50} lower than either agent alone, meaning that the total effect was more than additive. When an LD_{50} dose of VX was administered 1 h prior to an LD_{50} dose of GB, the VX had a protective effect on the inhibition of brain and blood AChE by GB. However, when the nerve agents were administered in reverse order, there was potentiation of AChE inhibition (i.e., the inhibition was more than additive). Potentiation of toxicity with the administration of certain combinations of organophosphate insecticide has been described (Frawley et al. 1957); however, with other insecticide combinations, there was no potentiation in the AChE inhibition (Vestweber and Kruckenberg 1972). Further investigation is needed on the possible potentiation of toxic mixtures of nerve agents or combinations of nerve agents and pesticides.

B.2.3 CHRONIC TOXICITY

In addition to consideration of the acute toxic effects of the nerve agents, there is public interest in the possibility of chronic disease states or delayed effects that might develop following exposure to an agent. Areas of chronic health concern include cancer (carcinogenesis), genetic effects such as mutations, developmental effects on unborn children (teratogenesis), and effects on reproductive ability (fertility). Additionally, there is public concern about possible chronic health problems such as delayed neuropathy or brain dysfunction that could develop as consequences of the neurotoxicity of the nerve agents.

For the sake of discussion and data organization, we shall use the term "chronic effects" to encompass biological endpoints that are not acute. A rigorous application of "chronic" would include only those effects that persist over a long period of time. However, we shall include in this category some effects that are more precisely defined as delayed (manifest at some time after exposure), which may or may not persist for a long period of time. Additionally, we shall make a distinction between the chronic effects that arise from a single exposure to nerve agents and those that arise from long-term, low-level exposures.

Although the data are often sparse on the chronic health effects of nerve agents per se, there is an expanding field of literature on chronic health effects of organophosphates, particularly insecticides (Karczmar 1984; Marquis 1986, pp. 62-71). The nerve agents so closely resemble many of these insecticides, both structurally and functionally, that it is appropriate to review this literature with regard to potential health problems, particularly where there are data gaps for nerve agents.

Among the list of possible delayed abnormalities that can result from a single exposure to organophosphates insecticides are serious and often fatal cardiac complications, which develop after apparent recovery from acute toxic effects (Hirshberg and Lerman 1984; Kiss and Fazekas 1979; Ludomirsky et al. 1982). The cardiac complications presented most often are heartbeat irregularities (arrhythmias).

Electrocardiogram (EKG) abnormalities in a human that persisted for four weeks have been described in a single case of acute exposure to a nerve agent (Side11 1974); additionally, cardiac lesions have been found in animals surviving high doses of nerve agents (McLeod 1985; Singer et al. 1987). All the aforementioned cardiac effects (arrhythmias, EKG changes, and lesions) may be secondary to primary effects on the brain following nerve agent and pesticide exposures (McLeod et al. 1982 ; Weidler 1974). Recently Arsura et al. (1987) described complications that can result from the use of anticholinesterase medication in patients who already have cardiac problems. A possibility exists, therefore, for the exacerbation of preexisting cardiac problems with nerve agent exposure, although there is no direct evidence for this.

There has been growing evidence that both acute and chronic exposure to organophosphates may cause delayed psychological and electroencephalogram (EEG) changes that can persist for days, months, or even years following exposure. There have been observations of schizophrenic and depressive reactions with severe memory impairment and concentration difficulties (Gershon and Shaw 1961) and elevated levels of anxiety (Levin, Rodnitzky and Mick 1976) in persons chronically exposed to pesticides. When schizophrenic and manic-depressive patients were given daily doses of an organophosphate, there was exacerbation of their psychiatric states (Rowntree, Nevin, and Wilson 1950). On the other hand, an epidemiological survey failed to find any association between total pesticide sales in a geographical area and the number of hospital admissions for mental disorders in the same area (Stoller et al. 1965) ; however, it is difficult to draw conclusions from such a survey.

In addition to the psychological effects produced by chronic pesticide exposure, there are data on brain dysfunction following acute nerve agent exposure, specifically agent GB (Duffy et al. 1979, Duffy and Burchfiel 1980). In these studies, changes in EEG patterns and increases in rapid eye movement (REM) sleep were observed at 1 year or longer following accidental exposure to GB. Statistically significant EEG changes were observed only in a group comparison of exposed workers with control subjects; it was not possible to make a diagnosis of exposure based on visual inspection of an individual EEG. Studies in monkeys, where the exposure conditions could be controlled, substantiate the human EEG changes observed with GB exposure (Burchfiel, Duffy, and Sim 1976). Metcalf and Holmes (1969) also reported on EEG, psychological, and neurological changes in persons exposed to organophosphates. [Al though the source of the organophosphate exposure was not specified in the paper, Karczmar (1984) states that this study

included industrial workers exposed to GB.] Certain psychological effects, including impairment in memory and ability to concentrate as well as some EEG abnormalities, were noted in the workers exposed to nerve agents. These studies will be discussed in further detail in Sect. B.2.3.2. Among volunteers exposed to nerve agents as part of an Army program, there were no records indicating that these individuals experienced any changes in brain function that persisted long after initial exposure (National Research Council 1982). At this point, some 30 to 40 years later, detection of any current brain dysfunctions resulting from those exposures would be very unlikely.

The significance of altered EEGs is not known. They may bear no relationship to either psychological or neurological functioning; however, other chemicals such as LSD and amphetamines that cause brain wave alterations and sleep pattern changes similar to those observed with GB are among the most psychoactive drugs known. Thus, these persistent EEG changes induced by GB exposure may reflect subtle changes in brain function (Duffy and rurchfiel 1980). Some of the GB-exposed personnel in the study by Duffy and Burchfiel (1980) had EEGs similar to those of the control group, suggesting that not all brains manifest EEG changes following exposure. Because of the conflicting results, it is uncertain at this point whether exposure to nerve agents in an accidental release could result in permanent changes in brain function.

A third possibility of a delayed effect that can follow either acute or chronic exposures to nerve agents is a type of progressive, irreversible neurotoxicity called delayed neuropathy. This syndrome first received widespread attention in the 1920s, when some 20,000 cases developed in the southern United States among persons who drank "Jamaica Ginger" that was adulterated with an organophosphate ester TOCP (Abou-Donia 1981). In delayed neuropathy, there is a symptomless period of 5 to 30 d followed by some initially mild symptoms, such as weakness, tingling, and muscle twitching in the legs. A flaccid paralysis eventually develops, first in the legs and then progressing to the hands and the thighs. Not all animal species are susceptible to delayed neuropathy. Mice and rats are resistant, whereas humans, chickens, cats, and sheep are susceptible to this effect of organophosphates (Abou-Donia 1981). The mechanism of action that leads to this syndrome is still not fully known; however, the inhibition of AChE appears to play no part in this delayed effect. Many investigators believe that inhibition of an enzyme designated neurotoxic esterase (NTE) is responsible for delayed neuropathy because of correlations between inhibition of this enzyme in vitro and induction of delayed neuropathy in vivo (Correll and Ehrich 1987; Gordon et al. 1983; Vranken, DeBisschop, and Willems 1982). In a review on delayed neuropathy, however, Abou-Donia (1981) raises several reasons why this explanation for the mode of action may not be correct. There do appear to be certain chemical structural requirements for an organophosphate to cause delayed neuropathy, but the situation is not quite as simple as a fluorine-phosphorus bond's being necessary and sufficient, as was once proposed by Davies, Holland, and Rumens (1960).

Because the nerve agents are the most toxic of organophosphate compounds, the test animals (usually chickens) must be protected against the lethal effects by pretreatment with various antidotes in order to test for delayed neuropathy. The ability of the nerve agents to induce delayed neuropathy will be discussed on an individual basis in Sects. B.2.3.1 through B.2.3.3.

One further comment on delayed neurotoxic effects: an intermediate syndrome of neurotoxic effects has been described recently in several cases of insecticide exposure (Senanayake and Karalliedde 1987). The onset of the intermediate syndrome paralysis was 24 to 96 h after poisoning, well after the acute cholinergic crisis had ended and before the expected onset of delayed neuropathy. The muscles involved in the intermediate syndrome were different from those that are involved in delayed neuropathy and unfortunately included the respiratory muscles. Nothing is known about the ability of nerve agents to cause this intermediate neurotoxic syndrome.

There are a number of tests that are designed to determine whether a chemical can damage deoxyribonucleic acid (DNA). Because DNA provides the fundamental code for normal cell function, permanent changes in DNA, called mutations, can result in cell death and permanent changes in cell function. If these mutational events occur in germ cells in the ovaries or testes, the results might be passed on to the next generation as inherited abnormalities. Damage to the DNA of other cells can result in the transformation of a normal cell into a malignant or cancerous cell (carcinogenesis). Damage to the DNA of cells in a developing fetus can result in death or transformation of a cell leading to abnormal development (teratogenesis). For these reasons, the tests for DNA damage by nerve agents are important in assessing the possible human health hazards presented by nerve agents (Kimball and Munro 1981, pp. 3-5).

In the attached Definition of Terms, brief descriptions are given of the assays for DNA damage or mutagenesis that have been or will be applied to nerve agents. In the studies completed to date (Table B.3), there was no evidence of DNA damage caused by the nerve agents tested. In the in vitro assays, the nerve agents showed low toxicity for the target cells, and, thus, it was possible to test for mutagenicity at concentrations many times higher than the estimated human lethal doses. Organophosphate compounds related to nerve agents have given positive results in certain tests for DNA damage (Malhi and Grover 1987; Nishio and Uyeki 1981); however, with other assays and other compounds, there have been negative results (Velazquez et al. 1987) or evidence of only weak mutagenicity (Velazquez et al. 1986). It is important that the nerve agents be submitted to a variety of assays before conclusions are drawn as to their ability to damage DNA.

Thus far, nerve agents have also given negative results in tests to determine whether fetal exposure to nerve agents could cause developmental abnormalities. Rabbits and rats have been used for these assays in which the nerve agents were administered during the time of major fetal organ development. In examination of agent-exposed rodent

fetuses for skeletal or organ malformations, no defects were found that could be linked to nerve agent exposure (LaBorde and Bates 1986; Schreider et al. 1985). A well-known organophosphate insecticide, malathion, has been shown to cause developmental abnormalities in chickens (Gill and La Ham 1972; Greenberg and La Ham 1969). The lower toxicity of malathion may allow testing for teratogenic effects at much higher doses than is possible with the nerve agents.

A major deficit in the chronic studies either completed or planned for nerve agents is the absence of long-term tests of the carcinogenic (cancer-causing) potential of nerve agents. It is not enough to rely on the tests for DNA damage to determine whether nerve agents can cause cancer because (1) these tests are not always valid predictors (Rousseaux 1987) and (2) it is possible that chronic exposure to nerve agents might result in cancer through indirect means. There have been several recent reports, for example, on the imrnunotoxicity of organophosphates (Casale, Cohen, and Dicapua 1983; Rodgers et al. 1986; Wysocki, Kalina, and Owczarzy 1987). The immune system is an important defense against cancers arising either spontaneously or from environmental carcinogens. Thus, if nerve agents can suppress the immune system (there is no evidence to support this supposition as yet), long-term exposure to nerve agents could result in altered *tumor* patterns within the exposed population.

Continual exposure to low levels of anticholinesterase compounds can induce a decrease in sensitivity to their cholinergic effects. This adaptive process, called tolerance, can be measured in two ways: changes in the effects caused by the same dosage level or changes in the dosage level needed to cause the same effects (Russell and Overstreet 1987). Many times, animals receiving doses of organophosphates that initially produced symptoms show a decrease in symptoms or actual absence of symptoms with prolonged dosing (Costa, Schwab, and Murphy 1982). Many investigators consider the biological mechanism underlying tolerance to be an actual decrease in the density of ACh receptors in the brain, organs, and muscles so that, despite the continuing accumulation of ACh, there is a decrease in the stimulation by ACh (Blanchet et al. 1986; Cverstreet et al. 1974; Volpe, Biagioni, and Marquis 1985). The development of tolerance to certain behavioral effects seems to parallel a decrease in ACh receptors in the brain. Another possible mechanism for tolerance is the fast recovery of certain AChE species (see review by Russell and Cverstreet 1987). It is important to remember that tolerance has only been linked to the cholinergic effects of anticholinesterase compounds; there appears to be no such adaptive process for the noncholinergic effects.

Studies in rats to assess the development of tolerance to nerve agents following chronic exposure have yielded some interesting results. In one study, the animals were injected for 85 d with 15 to 25% LD_{50} of nerve agents. The animals showed a reduced growth rate for the first 30 to 38 d, but after this period, the treated rats grew at rates similar to those of controls (Dulaney, Hoskins, and Ho 1985). Effects of chronic

exposure to anticholinesterase compounds on weight gain had been reported previously (Glow, Richardson, and Rose 1966); however, in the nerve agent study, tolerance to the decreased weight gain effect was observed. In other studies where higher doses of nerve agent (50 to 60% LD_{50}) were given to rats for a shorter period of time, no tolerance to neurotoxic effects was observed (Fernando et al. 1985). Fernando and his colleagues concluded that there was a lack of tolerance in rats to doses of nerve agent that produce severe neurotoxic symptoms and, in fact, there was a cumulative toxicity of these very potent anticholinesterase compounds.

B.2.3.1 Agent GA

At present, the chronic health effects of GA are unknown. The tests for genetic effects, effects on the fetus, and reproductive effects designed by the U.S. Army Medical Bioengineering Research and Development Laboratory will be initiated in 1987 and 1988 (Table B.3), so it will be a while before the results are available. The main deficit in these planned studies is the absence of any long-term assay for carcinogenicity of GA.

In delayed peripheral neuropathy studies, it appears that, under appropriate conditions, exposure to GA could result in this neurotoxic syndrome. Despite the fact that GA contains a cyanide group instead of fluorine, GA does show in vitro inhibition of the NTE enzyme (Vranken, DeBisschop, and Willems 1982). The prediction was made, however, that doses 100 to 150 times the LD_{50} dose would be needed to cause delayed neuropathy in chickens (Gordon et al. 1983). Tests of GA in chickens at 120 times the LD_{50} dose elicited mild neuropathic symptoms in one of two hens that survived the dosage divided into two daily injections, but no delayed neuropathy was observed in survivors of a single dose (Willems, Nicaise and DeBisschop 1984). The authors concluded that even higher doses of GA would be needed to produce the clinical signs of delayed peripheral neuropathy. If human populations were ever exposed to these massive doses (greater than 120 times the LD_{50} value), the likelihood of death is very high, so the possibility of delayed neuropathy is not a relevant concern with GA exposure.

B.2.3.2 Agent GB

Somewhat more is known about the chronic health effects of agent GB compared with GA, although the studies on GB reproductive effects, subchronic effects, and delayed neuropathy are still to be completed (Table B.3). The only data describing the carcinogenic (cancer-causing) potential of GB comes from a study by Weimer et al. (1979) in which rats (Fischer 344 strain) and mice ("A" strain) were chosen for exposure to airborne GB because of these rodent species' susceptibility to certain types of tumors. Exposure of these rodent strains to low doses of GB for 6 h/d, 5 d/week, for up to 52 weeks (maximum cumulative exposure of 10.5 $mg\text{-}min/m^3$) did not result in an increase of tumors in either rodent

Table B.3. Chronic effects of nerve agents

Effect	GA	GB	VX
Carcinogenicity			
Rat		Negative[a,b]	
Mouse		Negative[a,b]	
Mutagenicity			
S. typhimurium (Ames)'	(Start 6/87)[d]	Negative"'	Negative[f,g]
Saccharomyces cerevisiae[c]			Negative'
Mouse lymphoma mutation'	(Start 6/87)[d]	Negative[d,e]	Negative'
Sister chromatid exchange	(Start 12/87)[d]	Negative[d,e]	
Unscheduled DNA synthesis	(Start 8/87)[d]	Negative[d,e]	
Drosophila melanogaster			Negative[j]
Teratogenicity			
Sheep			Negative[k]
Rabbit	(Start 12/87)[d]	Negative'	Negative[m,n]
Rat	(Start 8/87)[d]	Negative'	Negative[m,o]
Reproductive effects			
Sheep. 1 generation, females			Negative'
Rat, 3 generations	(Start 8/88)[d]	(Start 8/88)[d]	Negative"
Rat, dominant lethal	(Start 4/88)[d]	(Start 4/87)[d]	(In progress)[d,p]
Delayed neuropathy			
Chicken. single dose	(Start 6/87)[d]	Positive[q,r]	Negative'
Chicken, multiple doses	(Start 12/87)[d]	(In progress)[d]	Negative[d,t]
Subchronic effects			
Rat, 90 days	(Start 4/88)[d]	(Start 10/87)[d]	Negative[d,u]

[a]Weimer et al. 1979. Results negative, but test not definitive.
[b]Low dose (0.0001 to 0.001 mg/m^3) exposure up to 52 weeks.
'Tested with and without activation.
[d]Dacre 1987.
[e]Two forms of GB tested (Type I and 11).
[f]Crook et al. 1983, tested in five strains at concentration range of 2.7 x 10^{-6} to 1093 μg/plate.
[g]Remsen et al. 1984, tested in five strains at concentration range of 0.01 to 10μg/plate.
[h]Shifine, Schreider, and Rosenblatt 1983, tested at concentration range of 25 to 100 μg/ml.
[i]Kawakami. Schrieder, and Rosenblatt 1984. tested at concentration range of 1 to 100 μg/ml.
'Crook et al. 1983. sex-linked recessive lethal test, tested at concentrations of 0.000005 and 0.004 mg/m^3.
'Van Kampen et al. 1970. accidental exposure of sheep.
[l]LaBorde and Bates 1986. Two forms of GB tested: **Type** I (tributylamine stablizer) **and Type II** (dicyclohexylcarbodiimide stabilizer) at concentrations from 100 to 380 &kg/day for gestational days 6 to I5 **in rats,** and concentrations of 5 to 15 $\mu g/kg/day$ for gestational days 6 to 19 in rabbits.
"'Tested at concentration range of 0.25 to 4.0 $\mu g/kg/day$ for gestational days 6 to 15 in rats and 6 to 19 in rabbits
"Schreider et al. 1985.
'Hartley 1987.
[p]Tested at concentration range of 0.25 to 4.0 $\mu g/kg$.
[q]Davies, Holland and Rumen 1960.
'Gordon et al. 1983.
[s]Wilson et al. 1984, tested subcutaneously in atropine/2-PAM-treated chickens at concentration range of IO to 150 μg kg
'Atropinized chickens given subcutaneous injections of 40 $\mu g/kg$ for 90 days.
[u]Tested orally at concentration range of 0.25 to 4.0 &kg/day.

strain within the 1.5 years of study. Although the results suggest that GB is not carcinogenic, this study was not designed to be a definitive study of the carcinogenic activity of GB, particularly with the limited doses employed.

Studies designed by the U.S. Army to test the ability of GB to cause DNA damage and/or induce mutations have recently been completed, and the results of all assays have been reported to be negative (Dacre 1987). The reports on these genetic effects assays are not available for review at present.

Agent GB has been tested for effects on the fetus or embryo by giving pregnant New Zealand White rabbits and CD rats oral doses of GB during the period of major fetal organ development. Two forms of GB with either the tributylamine stabilizer (Type I) or the dicyclohexylcarbodiimide stabilizer (Type II) were tested. The pregnant animals were sacrificed on day 20 of their pregnancy, and the litters were examined for a number of biological effects: number and status of the fetal implants, individual fetal weight, and fetal malformations. No evidence of developmental toxicity was found with either Type I or Type II GB in either species, even at doses of GB that resulted in maternal toxicity or mortality.

Although definitive tests of the effects of GB exposure on reproduction have yet to be finished (Table B.3), some data exist on the effects of chronic exposure to very low levels of GB. In a review of a study by Denk (1975), Weimer et al. (1979) reported that rats exposed to airborne GB at concentrations of 0.001 or 0.0001 mg/m^3 for up to 12 months had no dominant lethal mutations (see Definition of Terms) nor adverse effect on reproductive performance through three generations. Note, however, that these doses were very low and did not produce any overt signs of toxicity. Another study evaluated testicular atrophy in Fischer rats after a six-month exposure to low doses of GB; no differences were found between treated and nontreated animals (Morin and McKinley 1976). The details of the studies planned by the U.S. Army on the reproductive effects of GB exposure (Table B.3) are not known; these studies may include higher doses to rats than were used in the earlier studies. Such data are needed to more accurately ascertain the risk of GB exposure on human reproductive parameters. Furthermore, because rats (and mice) show a relative resistance to the acute toxicity of GB (a factor that does allow testing at higher concentrations), it is also important to test an animal species that does not show the rapid detoxification of GB that the rats and mice do.

Animal studies using chickens (because of their sensitivity to delayed neuropathy) have shown that high-dose exposure to GB can cause delayed neuropathy (Davies, Holland, and Rumen 1960; Gordon et al. 1983). Because GB can cause this neurotoxic syndrome in chickens, the possibility also exists that this syndrome could occur in humans surviving exposures to lethal concentrations of GB. In the studies in chickens, the doses used to induce delayed neuropathy were from 20 to 30 times the LD_{50} for GB in chickens. In order to protect the birds from

the acute lethality of this agent, the chickens were given protective doses of antidotes prior to the injection of GB. Gordon et al. (1983) feel that, because it is possible to successfully treat the acute toxicity of GB, the potential exists for the development of neuropathic symptoms in the survivors of severe GB poisoning. Further studies are planned to determine the ability of GB (Types I and II) to produce delayed neuropathy in antidote-protected chickens with either a single dose or one-third the single dose given once a week for three weeks. In the hypothetical event of accidental exposure of humans to high concentrations of GB, the obvious overwhelming concern would be the protection from acute lethality. If these emergency measures were indeed successful, then the exposed persons should be monitored for the late development of neuropathic symptoms. In any case, the concern about this potential chronic health effect would be relegated to a lower priority in the face of the likely acute lethality that would result from exposure to doses of this magnitude.

Studies of industrial workers accidentally exposed to agent GB were carried out by Duffy et al. (1979), who analyzed EEG abnormalities one year following the exposure, and also by Metcalf and Holmes (1969), who studied psychological and neurological alterations as well as EEG changes. When the EEG patterns of the exposed workers were compared with the EEGs of other workers who had no exposure or access to GB, several differences were observed (for details of EEG spectra differences see Duffy et al. 1979); furthermore, the amount of REM sleep was increased in the GB-exposed workers. An important point to remember is that these EEG changes were evident a year after exposure--during this time the workers had no other known organophosphate exposure and showed no reduction in blood ChE activity. In comparisons of a group of "highly exposed" workers to a group of "minimally exposed" workers, Metcalf and Holmes (1969) found disturbances in memory and in the ability to concentrate, as well as EEG changes and minor motor coordination deficits. It is not clear in the Metcalf and Holmes report whether the GB exposures of the subjects were recent or not. It is also not clear from these two studies whether the persistent EEG changes could be correlated with the persistent psychological changes observed.

Following the observation of EEG changes in workers exposed to GB, a study was carried out in monkeys to substantiate these long-term effects of GB on the human EEG (Burchfiel, Duffy, and Sim 1976). The monkeys were given either a single dose of GB that produced overt toxic symptoms or 10 smaller doses at weekly intervals, which did not result in any clinical symptoms. In both the acute and chronic exposure groups, there were alterations in the EEG patterns up to 1 year after exposure. Some of the brain wave changes observed in the monkeys were similar to those observed in the EEGs from GB-exposed workers. Another important finding from this study was that, at one year, there were greater differences in the EEG patterns of the animals that received the series of smaller doses (with no resulting clinical symptoms) than there were in the animals that received the single dose. Because the total dosage in the

series was twice that of the single dose, this suggests it is the total amount of GB received and not the presence of clinical symptoms that determined the degree of EEG alteration. As mentioned previously in the general discussion of chronic effects, the meaning of altered EEGs is not clear, and at this point it is not certain whether long-term exposure to GB could result in permanent brain dysfunction.

B.2.3.3 Agent VX

As with the other nerve agents, the majority of animal studies on VX have dealt with the acute toxicity of this chemical agent. However, with the potential for chronic exposure of personnel during VX research or its transport or disposal, the possibility of adverse health effects induced by chronic VX exposure must be addressed. To date, most of the chronic health effects studies designed by the U.S. Army Biomedical Research and Development Laboratory (USABRDL) have been completed, and in most cases the reports are available for review. As with the other nerve agents, no tests for carcinogenicity were included in these studies. In a review on the health effects of VX, Ross et al. (1982) found no available literature as to the carcinogenic potential of VX. McNamara, Vocci, and Leitnaker (1973) reported that there was no association of increased cancer in personnel working daily with this agent; however, as with the other nerve agents, definitive studies on the carcinogenic potential are needed.

The potential of VX to cause genetic effects has been addressed in several studies supported by USABRDL (see Table B.3). These studies include mutagenicity in bacteria (Salmonella typhimurium, Ames assay), mutagenicity in yeast *(Saccharomyces cerevisiae),* mutagenicity in fruit flies *(Drosophila melanogaster),* and mutagenicity in a mammalian cell line (mouse lymphoma L5178Y). In the studies in the bacteria and yeast, VX was tested with and without enzyme activation because some compounds need to be metabolized before they can show their mutagenic properties. Additionally, the range of concentrations in the *Salmonella typhimurium* assay included concentrations that corresponded to approximately 40,000 times the estimated intravenous LD_{50} for humans (Crook et al. 1983). Results were negative in both the *Salmonella typhimurium* and the *Saccharomyces cerevisiae* assays (Crook et al. 1983; Remsen et al. 1984; Shifrine, Schreider, and Rosenblatt 1983). In the *Drosophilia melanogaster* sex-linked, recessive lethal mutation test (see Definition of Terms), only one mutation was observed at the higher VX concentration (0.004 mg/m^3), for a mutation percentage of 0.5% (Crook et al. 1983). A repeat test at the same concentration yielded no mutations. Thus, results were also negative for VX in this mutagenicity assay. The fourth assay for mutagenic activity involved the use of mammalian cells, which may provide better health risk estimates for humans than bacteria or yeast. Again in this assay, VX was tested with and without metabolic activation. At lower concentrations (1 to 20 $\mu g/mL$), there was no statistically significant increase in the mutation frequency; at the

higher test concentrations (50 to 100 μg/mL), there was a small increase in the number of mutants that appeared to be related to dose but not to activation (Kawakami, Schreider, and Rosenblatt 1984). This increase in mutations compared with controls was not above the twofold increase set as a criterion for a positive mutagen (Amacher and Paillet 1979; Clive et al. 1979), thus VX was considered by the authors to be a nonmutagen. One assay for genetic effects that is lacking from the VX series is the sister chromatid exchange (SCE) assay, which tests for chromosomal damage rather than mutations. Nishio and Uyeki (1981) found nine out of ten organophosphates to be positive in the SCE assay. The SCE assay is being used in the assessment of the genetic effects of the other nerve agents, GA and GB.

Data on the potential of VX to affect fetal development (teratogenesis) come from the accidental exposure of sheep to lethal concentrations of VX and from controlled studies in rats and rabbits. Van Kampen et al. (1970) reported studies on 79 surviving ewes in the accidental lethal VX exposure in Skull Valley, Utah, in 1968 in which 4500 of the 6300 affected sheep died or were killed. The dose that the accidentally exposed pregnant ewes received is not known, and the dosage given another group of purposely dosed pregnant ewes is classified, making it difficult to evaluate this study. The accidentally exposed animals demonstrated depressed RBC AChE activities up to four months after the initial intoxication, suggesting significant VX exposure. Under the conditions of both accidental and intentional exposure, no evidence of any significant developmental effects were noted in the offspring of the ewes.

The teratogenic potential of VX in rats and rabbits was tested by subcutaneous (s.c.) injection of 0.25 to 4.0 μg/(kg•d) for gestational days 6 through 15 in rats and days 6 through 19 in rabbits. The pregnant animals were killed on day 20; the fetuses were removed and examined for body weight and for skeletal and organ abnormalities. Results of the studies in rats showed no statistically significant relationship between the dose of VX and any of the parameters studied (Schreider et al. 1985). Results of the teratogenic studies in rabbits were also reported to be negative (Hartley 1987), but at present the report on this study is unavailable for review.

In addition to the teratogenic potential, information is also needed on the effects of chronic exposure of VX on reproductive potential. The only data available come from the acute accidental exposure of sheep to unknown levels of VX (Van Kampen et al. 1970). The exposed ewes were evaluated for their reproductive capacity by breeding them five to six months after exposure, No effects on reproductive capacity were found in these animals; however, the dose of VX received by these animals is unknown. Reproductive studies in rats are still in progress, and the results of the three-generation assay for reproductive potential are reported to be negative (Dacre 1987); however, the reports are not available for review or for discussion of the details of the study. It is not known, for example, whether the dosage levels of VX (0.25 to 4.0

μg/kg) were administered in an acute, subchronic, or chronic exposure mode.

Agent VX has given negative results in all tests for delayed neuropathy. In tests of the ability of nerve agents to inhibit the NTE enzyme, VX was at least 1000-fold less active than agent GB (Gordon et al. 1983; Vranken, DeBisschop, and Willems 1982). With a single injection of VX into antidote-protected chickens at doses up to 12 mg/kg (40 times the LD50 in unprotected chickens), no neuropathic signs have been observed (Davies, Holland, and Rumen 1960; Wilson et al. 1984). Tests have also been recently completed on the ability of VX to induce delayed neuropathy in antidote-protected chickens when injected s.c. at 40 μg/(kg•d) for 90 days. The results of this subchronic exposure test have been reported to be negative, although the report is not yet available for review (Dacre 1987).

There have been no specific reports on brain dysfunction following exposure to VX; most studies related to nerve agents specify agent GB. Because at this point the question of long-term or permanent alterations in psychological parameters or EEG patterns relates to all organophosphates, there is concern that brain dysfunction could occur following either acute or chronic exposure to VX. More information is needed in this area before a decision can be made about the possibility of this chronic health effect.

B.2.4 ANTIDOTES AND PERSONAL DECONTAHINATION

B.2.4.1 Antidote Treatment Regime, Dosage, Toxicity, Aging, and Cautions

The choice of appropriate treatment for nerve agent poisoning depends on the agent and the extent and route(s) of exposure. Very mild exposure to nerve agent vapor may necessitate only decontamination and observation; severe exposure to vapor or liquid requires immediate decontamination, administration of antidotes, establishment of artificial respiration if necessary (Stares 1976; Sidell 1976), continuous monitoring for at least 24 h, and intensive supportive therapy over days or weeks to maintain life. Immediate care is vital, primarily to prevent death from respiratory failure, which can occur within minutes or seconds in cases of massive inhalation exposure (Side11 1987). Prompt attention is also necessary because the agent-AChE complex becomes resistant degrees to reactivation by oxime-type antidotes within minutes to a few hours, depending on the agent,

The standard antidotes available clinically in the United States are atropine (an anticholinergic drug) and pralidoxime (2-PAM-Cl). Atropine is used to antagonize the excess ACh that accumulates at nerve endings in the absence of functional AChE. It counters the airway tightening (bronchoconstriction) and lung secretions (bronchosecretions) that interfere with breathing or support by a respirator. Atropine also opposes the agent's toxic effects of nausea, vomiting, and diarrhea. It partially relieves the CNS respiratory depression but cannot reverse the paralysis of the respiratory muscles (Taylor 1980; Stares 1976). The

recommended approach for organophosphate (OP) pesticide poisoning is to give 1 to 4 mg intramuscularly (i.m.) or i.v. initially, and to maintain with 2-mg doses every 10 to 60 min for several hours, then to adjust additional doses according to the individual's responses thereafter (Gosselin et al. 1984, p. 111-340; Side11 1974; Taylor 1980). Massive amounts of atropine often must be used over a period of days or weeks in cases of severe organophosphate poisoning compared with the usual therapeutic dosages. The total dose may reach as much as 600 to 800 mg or more of atropine in severe cases because of the need to counteract the organophosphate pesticide continuously mobilized from body stores (Gosselin et al. 1984, pp. III-336 to 111-343; Sidell 1974; Hirshberg and Lerman 1984). In one exceptional case, a total of 30 g was given over 5 weeks (LeBlanc, Benson, and Gilg 1986). The usual therapeutic dose of atropine for nonorganophosphate poisoning applications is 0.4 to 0.6 mg (Side11 1974, 1987).

Therapy for nerve agents is similar to that for organophosphate pesticides but does not usually require such prolonged administration of atropine (see Table 8.4); Sidell (1987) recommends 2 to 4 mg atropine i.v. every 3 to 8 min until signs of its effects, such as a decrease in lung secretions and pulmonary resistance, are seen. In one case of Sarin inhalation, 4 mg atropine (2 mg i.v. + 2 mg i.m.) was given initially, followed by several 2-mg (i.v. except for one i.m.) doses over the next 2 h. Pralidoxime was also required (Side11 1974). For organophosphate poisoning in children, Haddad and Winchester (1983) recommend 0.05 mg/kg atropine i.v., followed by maintenance doses of 0.02 to 0.05 mg/kg as needed. In cases of nerve agent exposure by vapor or on the skin, atropine can be administered quickly to adults from the armed forces MARK I nerve agent antidote kit if this is available. One kit provides an injector containing 2 mg of atropine sulfate and one containing 600 mg of pralidoxime chloride (Side11 1987).

Atropine itself is highly toxic, with a rating of about 5 on a scale of 1 to 6, 6 being most toxic; the probable oral lethal dose for humans at this rating is given as 5 to 50 mg/kg [7 drops to 1 teaspoon for a 150-lb (70-kg) person] by Gosselin et al. (1984, II-236 and I-2). The major consequences of overdosage with atropine are listed in Table B.4. This antidote must therefore be used with caution and only with sure knowledge that nerve agent exposure has occurred. This is particularly important with regard to children, the elderly, or debilitated individuals. In the absence of exposure, a 2-mg dose to an older person might result in "a greater degree of mental disturbance, including delirium, and the rapid heart rate might cause heart damage or precipitate an arrhythmia" (Side11 1987). In cases of severe organophosphate exposure, however, by far the main treatment error has been underdosage, rather than overdosage, with atropine because of the large doses required (Gosselin et al. 1984, p. 111-339). Some sources caution that atropine is dangerous in cases of inadequate oxygen supply (anoxia) and that the consequent bluish color of the victim (cyanosis) must be corrected before atropinization. However, in nerve agent

Table B.4. Nerve agent antidote summary: Actions, dosages, side effects/overdose symptom

Antidote/action	Dose	Overdose symptoms, management
Atropine (dl-hyoscyrmine)		Symptoms (Gosselin et al. 1984, pp. III-47 to III-50)
Anticholinergic alkaloid; used to block effects of parasympathetic nerve stimulation. Prepared from powdered roots of *Atropa belladonna* and *Datura stramontum*. In massive doses, used to treat AChE poisoning and to manage certain psychiatric states (Gosselin et al. 1984, pp. III-47 to III-50)	Adult: 2-4 or more mg of atropine sulfate i.m. or i.v. Full atropinization maintained at 2-mg doses every 10 to 60 min for several hours Mean lethal dose unknown (recovery after ingestion of 1000 mg documented); lethal estimate of 10 mg, although recovery documented at IOO-mg dose in many adults; children more susceptible (Gosselin et al. 1984, p. 111-47)	• Dryness of mucous membranes, burning pain in throat, difficulty in swallowing, and intense thirst • Skin hot, dry, flushed. Rash over face. neck, and upper trunk, especially in infants and children. Peeling of skin may follow • Exceptionally high fever
Relieves smooth muscle constriction in lung and GI tract and reduces glandular paralysis (cleans up respiratory tract secretions) (Sidell 1986)	Children: initial dose = 0.05 mg/kg Maintenance doses for children range from 0.02 to 0.05 mg/kg For all: provide atropine until signs of 'atropinization" occur (dry mouth and dry lungs); use until signs of improvement are seen; taper off dose (Sidell 1987)	• Sinus tachycardia (rapid heart beat), palpitations, elevated blood pressure • Uncommonly: nausea, vomiting, and abdominal distension in infants, urinary urgency and hesitancy; inability to void • Restlessness. fatigue, excitement and confusion, progressing to mania and delirium, which may persist for hours or days. Hallucinations, particularly of visual type. Patients may exhibit selfdestructive acts • Seizures may occur, but true convulsions arc rare • Rare: coma, depression of medullary centers, circulatory collapse and death from respiratory failure

Table B.4 (continued)

Antidote/action	Dose	Overdose symptoms, management
		Management (Gosselin et al. 1984, pp. III-47 to III-50): Toxicity rating = 5, EXTREMELY TOXIC. [Probable oral lethal dose in humans of 5–50 mg/kg, or 7 drops to 1 teaspoon for 150-lb (70-kg) person] Treat symptoms with physostigmine salicylate. Onset of symptoms within 15–30 min, maximum effects at 2-3 h and recovery within 12 h
2-PAM-Cl (Protopam chloride; 2-pyridine aldoxime methyl chloride; pralidoxime): Treat poisoning due to OP insecticides and nerve gases; anticholincsterase antagonist. Effective when given with atropine (Gosselin et al. 1984, p. H-382) Acts by removing OP from cholinesterase and restoring normal control of skeletal muscle contraction (relieves twitching and convulsions) (Sidell 1986)	Adult: 1-2 gm in 100 mL saline i.v. over 15–30 min for initial dose. Second dose after 1 h if symptoms indicate Children: initial dose of 15-25 mg/kg, followed by second . after 1 h if symptoms indicate" Infants: try 15 mg/kg[a]	**Symptoms (Gosselin et al. 1984, p. II-382 where not specified):** • Less effective after aging (when bond between OP and cholinesterase becomes irreversible). Substantial aging occurs within 5 h for GB (Sidell 1986). • Rapid and possibly dangerous rise in blood pressure' • Temporary rapid heart beat (tachycardia) • Mild weakness, dizziness • Blurred or double vision

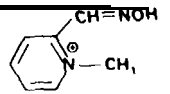
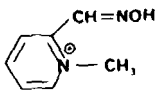
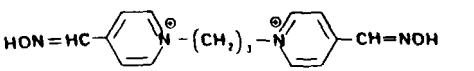
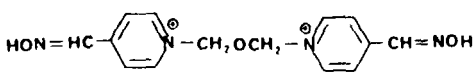
'Sidell. F. R. Planning, Operations and Training Branch, USAMRICD, Aberdeen Proving Ground, Md. Personal communication to N. B. Munro. Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., June 15, 1987.

poisoning this preliminary treatment may not be possible because of the intense bronchoconstriction, heavy bronchosecretion, and even lockjaw (trismus) that are characteristic of severe poisoning (Gosselin et al. 1984, p. III-340). In such- cases, atropine administration may be necessary to relieve these symptoms so that anoxia may be corrected.

Several other possible complications of atropine administration have been mentioned based on observations in animals. Krop and Kunkel (1954) occasionally observed fatal ventricular fibrillation in dogs and cats that they attributed to atropine given late in the course of GB poisoning. Fernando, Hoskins, and Ho (1985, 1986) have described a behavioral supersensitivity reaction to atropine in rats exposed to sublethal single doses of GB or other nerve agents. It consists of an exacerbation of limb shakes (myoclonus) and, at higher atropine doses, of certain stereotyped behaviors--sniffing, turning, and head movements. Whether this might ever pose analogous problems in humans is very questionable, but Fernando, Hoskins, and Ho (1986) suggest the need to monitor closely any victim of nerve agent exposure treated with atropine, especially if multiple doses are given during the first few hours or up to two to three days after exposure. Monitoring of body temperature may also be indicated, particularly in infants and children, because atropine can cause exceptionally high fever (Gosselin et al. 1984, p. 111-49). This may not be a problem in cases of nerve agent exposure because the nerve agents cause hypothermia in rodents (Meeter and Wolthuis, 1968a). They may do so in humans, although there is little or no mention in the literature of body temperature effects in cases of organophosphate poisoning. Hypothermia has apparently been seen in humans in a few cases of severe organophosphate exposure (Meeter and Wolthuis 1968a; J. G. Clement, Biomedical Section, Defence Research Establishment, Ralston, Alberta, Canada, personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., May 12, 1987).

In a clear case of nerve agent exposure and with established symptoms, a second antidote may be administered to complement the actions of atropine. Pralidoxime chloride (2-PAM-Cl) is a compound with an active oxime group, RCH-NOH; it splits off the nerve agent from AChE, reactivating the enzyme and gradually restoring normal muscle function. It is currently the only oxime antidote approved by the U.S. Food and Drug Administration for clinical use in organophosphate poisoning. Table B.5 gives common names, abbreviations, chemical names, and structures of pralidoxime and two other oximes. As shown in Table B.4, the usual adult dosage of 2-PAM-Cl consists of 1 to 2 g i.v. in 100 mL of saline over 15 to 30 min (Gosselin et al. 1984, p. 111-340; Barr 1985). A suggested initial dose for children is 15 to 25 mg/kg according to F. R. Side11 (F. R. Sidell, Planning, Operations, and Training Branch, USAMRICD, Aberdeen Proving Ground, Md., personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., June 15, 1987). Barr (1985) recommends 25 mg/kg. No data are available on which to base a dose for infants, but Sidell recommends using 15 mg/kg

Table B.5. Older oximes—nomenclature and abbreviations

Trivial name	Abbreviation or trade name	Chemical name	Structure[a]
Pralidoxime chloride	2-PAM-Cl	pyridine-2-aldoxime methyl chloride	 Cl^-
Pralidoxime methane sulfonate	P_2S	*N*-methyl-2-pyridiniumaldoxime methanesulfonate	 $CH_3SO_4^-$
Obidoxime	Toxogonin	bis(4-hydroxyiminomethyl-pyridinium (1)methyl)-ether dichloride	 $2Cl^-$
Trimedoxime	TMB_4	*N,N'*-trimethylene bis(pyridium-4-aldoxime)dichloride (or dibromide)	 $2Cl^-$

[a]From Stares 1976.

as a starting point (F. R. Sidell, Planning, Operations, and Training Branch, USAMRICD, Aberdeen Proving Ground, Md., personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., June 15, 1987). The initial dose may be followed by two or three periodic maintenance doses over a period of not more than 48 h if muscle weakness persists and if the initial dose gives beneficial results (Erdmann 1976) or for as long as it appears to be of help (F. R. Sidell, Planning, Operations, and Training Branch, USAMRICD, Aberdeen Proving Ground, Md., personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., August 27, 1987). The limits on oxime use result both from the aging of poisoned AChE and from the toxicity of the oxime (Erdmann 1976). The major effect of oximes is to restore skeletal muscle function; 2-PAM-Cl has little or no CNS effect, possibly because it does not seem to cross the blood-brain barrier readily (Taylor 1980). Because of the poisoned AChE aging problem, prompt administration of an oxime antidote is essential and can be facilitated if nerve agent antidote kits are available to emergency medical personnel and to the workers involved in decommissioning nerve agent stockpiles. They can be used for self-administration or administration by another.

"Aging" of phosphorylated enzyme is thought to result from the loss of an alkyl or alkoxy group from the nerve agent moiety (Taylor 1980). The remaining enzyme-modified nerve agent complex is more stable and is resistant to reactivation by oximes or similar antidotes. The rate and extent of aging varies with the nerve agent in question. In humans, aging is substantial (50 to 60%) within 5 to 6 h after GB exposure but less extensive (approximately 40%) even 48 h after VX exposure (Side11 and Groff 1974).

Because 2-PAM-Cl itself is toxic, it must be used with caution. The patient must be monitored closely for hypertension when the drug is given i.v. because rapid infusion will produce a marked and potentially dangerous rise in blood pressure, as well as double vision, nausea, and vomiting (Side11 1987). Mild weakness, dizziness, blurred vision, double vision (diplopia), and temporary rapid heartbeat have often been seen in normal subjects given doses as low as 0.5 g (F. R. Sidell, Planning, Operations, and Training Branch, USAMRICD, Aberdeen Proving Ground, Md., personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., August 27, 1987). At higher than therapeutic doses, 2-PAM-Cl can inhibit AChE and block neuromuscular transmission (Taylor 1980; Erdmann 1976; Oldiges 1976).

According to F. R. Sidell (1987), there has been little use of this drug in children or the elderly. Because these groups are generally more sensitive to drugs, it may be expected that these side effects might be more severe or be seen at lower doses among children or elderly.

A commonly used measure of toxicity is the median lethal dose or LD_{50}. This is the dose of a substance causing death in 50% of the animals tested under specified and controlled conditions with defined species, age, gender, etc. The LD_{50} values for a given chemical usually

differ depending on the route of administration (in part because the extent of absorption differs). Therefore, it is common to refer to an oral LD_{50} as opposed to a dermal LD_{50}, for example. The lower the LD_{50} value, the more toxic the substance. The median lethal doses (LD_{50}s) are given for 2-PAM-Cl and several other oximes in Table B.6. These values are for mice, rats, and guinea pigs. LD_{50} values for 2-PAM-Cl in -four species are shown in Table B.7. The oxime is substantially more toxic to dogs than to any of the three rodent species tested. Calesnick, Christensen and Richter (1967) studied the toxicity of 2-PAM-Cl, its methane sulfonate salt (P_2S), and trimedoxime (TMB4) in human subjects. Of the three, 2-PAM-Cl was least toxic, P_2S somewhat more toxic, and TMB4 most toxic, especially when given orally over extended periods of time (chronic exposure) rather than in a single dose (acute exposure).

Additional therapy for CNS effects may be needed. McLeod (1985) cites evidence for extensive brain lesions produced in experimental animals by GB and interprets the damage as caused by convulsions or seizure activity that kill neurons (nerve cells) because of excessive demands on energy metabolism in the neurons and also probably from secondary anoxia and acidosis. Anticonvulsants such as diazepam (Valium@) have been used effectively in some experimental animals (Boskovic 1981; Martin *et* al. 1985; Inns and Leadbeater 1983). On the basis of increased animal survival, Valium@ is a part of the treatment regime adopted by the United Kingdom for use by its military services against nerve agent poisoning (Gall 1981).

Other CNS effects have been noted in clinical situations. For example, a compound related to atropine (scopolamine) has been useful in relieving nonlethal but disagreeable and potentially disabling psychiatric side effects of nerve agent exposure. A patient who had experienced a severe exposure to GD (a nerve agent chemically related to GA and GB, but not part of the stockpile of unitary weapons) suffered from temporary depression, bad dreams, decreased alertness, and disturbed sleep patterns during the recovery period after atropine and pralidoxime treatment. Scopolamine hydrobromide was given and produced an improvement in the mental functioning of the patient (Side11 1974). Very severe acute OP pesticide exposure or chronic exposures have resulted in abnormal EEG patterns and a variety of neuropsychiatric symptoms varying in severity from anxiety to hallucinations have been reported (Gosselin et al. 1984, p. 1x1-338). This body of experience suggests that in GA, GB, or VX poisoning it may be necessary to identify treatment agents such as scopolamine whose CNS activity can counteract some nerve agent effects not adequately opposed by atropine and pralidoxime.

Although 2-PAM-Cl is the only oxime available for use in the United States, some European countries have used Toxogonin to a considerable extent (Gosselin et al. 1984, p. 111-339). It was thought at one time that the USSR relied on "TAB" autoinjectors containing Toxogonin, atropine and benactyzine for troop protection in the field (Koelle 1981). Toxogonin (obidoxime), a compound having two oxime groups, ***is***

Table B.6. Toxicity (LD_{50}) of newer compared to older oximes in three species

Antidote	mice, i.p. (mg/kg)[a]	mice, i.m. (mg/kg)[b]	rats. i.v. (mg/kg)[c]	rats, i.p. (mg/kg)[d]	guinea pig, i.m. (mg/kg)[e]
			Old		
2-PAM-Cl	120	143	101		
P_2S					302
Toxogonin	145	147			93
TMB,	70	47			34
			New		
HI-6	514	450		324	
HS-6	316	345			
HGG-12	136	518			
HGG-42		431			

"Clement (198 I); 24 h LD_{50}.
[b]Adapted from Oldiges and Schoene (1970). Lenz and Maxwell (1981).
'Adapted from Simeon et al. (1979).
'Adapted from Kepncr and Wolthius (1978).
'Adapted from Inns and Leadbeater (1983).

Table B.7. Acute toxicity of 2-PAM-Cl and two H-oximes in four species[a]

Species	Route of injection	LD_{50} (mg/kg ± standard error)		
		2-PAM-Cl	HI-6	HGG-12
Mouse	i.p.	162 ± 4	670 ± 44	177 ± 9
Rat	i.m.	206 ± 3	860 ± 28	1179 ± 36
Guinea pig	i.m.	184 ± 2	500 ± 14	281 ± 4
Dog	i.m.	75 ± 2	350 ± 10	60 ± 4

[a]Adapted from Table 2 of Boskovic, Kovacevic, and Jovanovic (1984).

Appendix C

DEMILITARIZATION PLANT DESIGN

Table B.8. ED_{50} values and safety ratios for antidotes against GA and GB in mice[a]

Antidote	mg/kg			
	GAGB			
2-PAM-Cl			26	(5.5)[b]
Toxogonin	9	(4.3)	3	(47.0)
TMB,	1.6	(29.0)	3	(15.0)
HI-6			1	(404.8)
HS-6			8	(44.5)
HGG- 12			10.6	(49. I)
HGG-42			4	(109.2)

'Adapted from Table 1, Lenz and Maxwell (1981); work from Schoene's laboratory (Schoene and Oldiges 1973; Oldiges and Schoene 1970; Schoene, Steinhanses, and Oldiges 1976; Schoene 1981); therapy given 1 min after agent; atropine 10 mg/kg; GA 3 LD_{50}; GB 2 LD_{50}.

[b]Values in parentheses are safety ratios (LD_{50}/ED_{50}).

Table B.9. ED_{50} values and safety ratios of oximes in mice, rats, and guinea pigs exposed to GA[a]

Antidote	ED_{50}, mg/kg (LD_{50}/ED_{50})					
	Mice		Rats		Guinea pigs	
2-PAM-Cl	7 . 2	(17)	16	(16)	7.6	(23)
HI-6	3.0	(191)	11	(89)	0.75	(533)
HGG-12	0.08	(2109)	2.5	(456)	0.12	(1874)

"Adapted from Boskovic, Kovacevic, and Jovanovic (1984); mice and rats were given 1.3 LD_{50}s of GA; guinea pigs were given 2.6 LD_{50}s of GA. Oximts and atropine (4.2 mg/kg) were given i.p. to mice immediately after s.c. dose of nerve agent. Oximes were given i.m. to rats and guinea pigs.

Table B.10. Therapeutic value of antidotes against GA, GB, and VX[a]

	Protective ratios*								
	GA		GB			VX			
	Mouse	Rat	Guinea pig	Mouse	Rat	Guinea pig	Mouse	Rat	Guinea pig
A[c] + 2-PAM-Cl		1.6[d,e]				47[f]			
A + P_2S[g]	1.5"		2.5'	8.7'		38'			25'
A + Toxogonin[g]	12.0[h]		19'	33.0[h]		59'			58'
A + TMB_4[g]	14.0"		13'	27.0[h]		46'			40[i]
A + Toxogonin							40[h]		
A + HI-6		2.w	2.2'			76'70'	70[j]		66'
A + HGG-42 (30 μmol)					5.0[k] (dog)		15' (dog)		
A + HGG-12		2.6'	3.7'			12'	5[k] (dog)		5.1'

[a] Adapted largely from Table V, Lenz and Maxwell (1981) that used data from references f, h, j. and k below.

[b] Protective ratio = LD_{50} with treatment/LD_{50} with no treatment.

[c] A = atropinc.

'Antidotes given i.m., immediately after agent given s.c. in reference e.

'Cetkovic et al. 1984.

[f] Simeon et al. 1979.

'Antidote given 10 min before challenge with agent in reference h; half given 10 min before agent, half with atropinc 1 min after agent challenge for all antidotes tested in reference i.

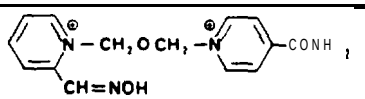
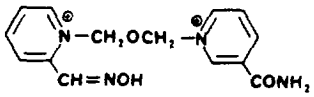
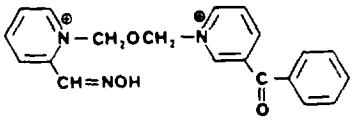
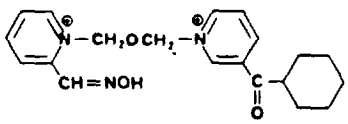
"Heilbron and Tolagen 1965.

'Inns and Leadbeater 1983.

'Wilhelm et al. 1979.

[k] Hauser and Wegcr 1979.

Table **B.11.** Newer **oximes—nomenclature and structure**[a]

Abbreviation	Structure	
HI-6		$2Cl^-$
HS-6		2Cl
HGG-12		$2H_2O$
HGG-42		$2H_2O$

'Adapted from **Lenz** and Maxwell (198 1).

given in Table B.11 for GA-exposed mice, rats, and guinea pigs (Boskovic, Kovacevic, and Jovanovic 1984). Overall, where data permit comparisons, the H-oximes appear to be more potent and less toxic antidotes than the older oximes.

Another basis for comparison of antidotes is the protective ratio, that is, the ratio of LD_{50} with treatment to LD_{50} without treatment. The higher the value, the more protection is afforded by the antidote. This is a measure of efficacy rather than potency, and, according to Lenz and Maxwell (1981), it indicates "the maximal response obtained with a saturating concentration." The most potent substance is not necessarily the most efficacious. The higher the value, the more protection is afforded by the antidote. Protective ratio values for several older and newer oximes are given in Table B.8 against GA, GB, and VX. Note that protective ratios depend on the amount of oxime administered. It is difficult to compare values from different experiments or laboratories if the experimental protocols vary. HI-6 seems to give the best protection against GB and VX, especially when its lower toxicity and greater stability are kept in mind. Toxogonin appears more promising than other oximes against GA in guinea pigs, which are thought to be a better model for human antidote effects than mice. The improvement in protection afforded guinea pigs by adding pyridostigmine pretreatment and Valium@ as supportive therapy in addition to atropine and oximes is shown in Table B.9 and can be compared with the values for atropine and oxime alone given in Table B.8 (Inns and Leadbeater 1983). In this system, Toxogonin gives the best protection against GB and VX, and TMB4 and HS_6 are best against GA.

All of these comparisons suffer from difficulties inherent in using LD_{50} or ED_{50} values that, strictly speaking, may not be comparable or may be misleading (Doull, Klaassen, and Amdur 1980, p. 2) even given equal LD_{50} or ED50 values, The discussion following the paper by Lenz and Maxwell (1981) contains warnings about this problem in addition to a discussion of the difference between efficacy and potency as it pertains to selecting the most appropriate compound(s) for protection against nerve agents. Investigators have developed new mathematical methods to model the results of various atropine/2-PAM-Cl treatment regimens after nerve agent poisoning in guinea pigs and to assess the benefits of pyridostigmine pretreatment in addition to the atropine/2-PAM-Cl regimen (Carter, Jones, and Carchman 1985; Jones, Carter, and Carchman 1985). Such methods should be exceedingly useful if they are widely applied in future research on improved oximes or other protective compounds or combinations of compounds.

Extrapolating from animal data to human effectiveness is a problem basic to evaluating therapies for nerve agent poisoning. Several approaches have been taken to deal with this obstacle. Some studies have been made on primates. Other studies have used human tissue, cells, or enzymes in vitro (in the test tube or artificial environment). For example, van Helden, van der Wiel, and Wolthuis (1983) used human respiratory and marmoset diaphragm muscle preparations as well as in

vivo (in the intact body) tests with marmosets. They concluded that marmoset muscle is a good model for human muscle when studying the effects of nerve agent therapy. In their study, HI-6 was very effective against VX and GB in both human and marmoset muscle, while Toxogonin was most effective against GA.

Measurement of the extent of in vitro reactivation of AChE has been used to assess the protective potential of antidotes fairly frequently, but results do not necessarily parallel those obtained in other in vitro systems or in vivo. One of the difficulties in using in vitro tests such as this to predict effects in humans is that the nerve agents have multiple effects other than inactivating AChE, so that enzyme reactivation may not be the key to antidote effectiveness (Rickett, Glenn, and Houston 1987). Some of the antidotes seem to have pharmacological activities other than enzyme reactivating ability that enhance their ability to oppose nerve agent effects, and these activities may vary in extent or type from species to species. These other pharmacological effects are not well understood. Thus, choosing tests or species that are good models for antidote activity in humans is a complex problem.

It should be mentioned that the search for more effective oximes or other antidotal compounds is continuing (Kiffer and Minard 1986; Len.? and Maxwell 1981). Modifications of existing oximes to reduce their toxicity and lower their rate of excretion or inactivation are also being attempted (Heldman et al. 1986). To date, there are some substances of promise, but most are far from ready for clinical application. In Canada, permission will soon be sought to begin clinical testing of HI-6, but even if permission is obtained, it will be some time before testing is completed and it becomes clinically available (should it pass the tests).

B.2.4.2.2 Pretreatment

In addition to the development of new oximes, combinations of other therapeutic agents with atropine and oximes have been examined. A promising approach has been to pretreat with carbamate compounds that are, themselves, anticholinesterases. The carbamate protects a fraction of AChE that thus cannot be blocked by nerve agent; the carbamate then spontaneously hydrolyzes off of the AChE within a matter of hours.

Pretreatment is a part of the approach adopted recently by the United Kingdom for its military establishment. It plans to use a combination of pralidoxime mesylate (P_2S), atropine, and Valium® together with pyridostigmine pretreatment (Leadbeater, Inns, and Rylands 1985; Gall 1981). The dosage to be used for pyridostigmine pretreatment is 30 mg orally every 8 h (Gall 1981). Pretreatment with the carbamate pyridostigmine substantially increases the protective effects of atropine and P_2S against the lethal effects of GB and VX in guinea pigs, and the addition of Valium® further enhances protection (Gall 1981). For military purposes, the P_2S would be given with atropine i.m. via

autoinjector in a dosage of 500 mg, to be administered up to three times at 15-min intervals; 5 mg of Valium* would be taken orally with each self-injection (Gall 1981). Pyridostigmine is an anticholinesterase that has frequently been used clinically for the long-term treatment of myasthenia gravis (Taylor 1980).

The improvement in protection afforded guinea pigs by adding pyridostigmine pretreatment and Valium* as supportive therapy in addition to atropine and oximes is shown in Table 8.12 and may be compared with the values for atropine and oxime alone given in Table B.8 (Inns and Leadbeater 1983). In this system, Toxogonin gives the best protection against GB and VX, and TMB4 and HS-6 do best against GA.

A related carbamate, physostigmine, has been shown to protect animals against not only nerve agent lethality but also incapacitation. Physostigmine, a compound that enters the CNS more readily than pyridostigmine, when used together with aprophen (an anticholinergic) as a pretreatment, gave better protection in guinea pigs than did pyridostigmine alone or with aprophen against both death and incapacitation by GB (Leadbeater, Inns, and Rylands 1985). In both cases, postpoisoning therapy consisted of atropine, P_2S, and Valium@. Physostigmine, like pyridostigmine, is widely used clinically for the treatment of myasthenia gravis (Taylor 1980). However, it has not been approved for use in humans as a pretreatment against nerve agent exposure. Furthermore, in a civilian population, extended pretreatment (prophylaxis) is probably not feasible or desirable.

B.2.4.3 Evaluation

Antidotes or combinations of antidotes are available to counteract, at least partially, the effects of exposure to GA, GB, and VX. The antidotes must be used in adequate dosage and in conjunction with prompt decontamination, intensive monitoring, and individualized supportive therapy, especially in cases of severe exposure, because incapacitation and respiratory failure can occur even with prompt antidote administration. The latter effects can recur after apparent improvement, so repeated administration of antidotes and artificial respiration may be required, especially in the first 24 h after exposure. Another problem pertinent tc the present assessment is that there is no single treatment that is equally appropriate for the three nerve agents. It appears that 2-PAM-Cl is the treatment of choice for GB or VX exposure, while Toxogonin appears better suited to treat GA poisoning. Protection against lethality can be increased beyond a few LD_{50}s of nerve agent by giving pretreatment with pyridostigmine and adding Valium@ to the treatment regimen as an anticonvulsant. These options have been adopted by the armed forces of the United Kingdom. At present however, it must be remembered that in the United States, only 2-PAM-Cl is available to augment atropine therapy.

Table 8.12. Therapeutic value of antidotes against CA, GB, and VX with pyridostigmine pretreatment in guinea pigs[a,b]

Antidote	Dose mg/kg	Protective ratio		
		GA	GB	VX
		11	11	15
P_2S	30	34	45	69
HS-6	47	73	57	82
HI-6	49	34	110	68
HGG-12	81	68	54	55
HGG-42	82	12	40	31
TM B,	46	76	220	310
Toxogonin	47		380	410

[a]Adapted from Inns and Leadbeater (1983). Table 4.

[b]Pyridostigmine (0.084 mg/kg i.m.) given 30 min before nerve agent (s.c.). One min after nerve agent, antidote given i.m. together with 14 mg/kg atropine (A); Valium* (1.8 mg/kg) given i.m. immediately after atropinc plus antidote mixture.

It does not appear that precautionary administration of protective drugs to the general population would be feasible or desirable. Even the therapeutic administration of atropine and pralidoxime in the event of civilian exposure poses significant legal and logistical problems. The requirement for treatment within a short time, even minutes, of exposure would probably necessitate the involvement of trained nonmedical personnel. Information from the states in which depots of lethal nerve agents are located indicate that, in most, at least some legislative or regulatory changes would be required to permit nonmedical personnel to administer treatment without on-site medical supervision (Leffingwell 1987).

To date, the only treatment combination that has avoided incapacitation as well as lethality after severe nerve agent exposure in animals requires pretreatment with an anticholinesterase carbamate (physostigmine) together with an anticholinergic (aprophen). Pretreatment was coupled with atropine, oxime, and Valium@ therapy following the subsequent nerve agent exposure (Inns and Leadbeater 1983). Whether such a regimen would be effective in humans as well as guinea pigs is not known, and much work with humans will be required to establish its efficacy, safety, and feasibility (L. Leadbeater, Chemical Defence Establishment, Porton Down, England, personal communication to N. B. Munro, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., May 5, 1987).

Additional protection may be available in the next several years against GB and VX exposure if HI-6 passes clinical trials and is approved for human use. Unfortunately, research to date in animals does not indicate that HI-6 will be useful against GA exposure unless a pretreatment regimen is used, When it is coupled with HI-6, pretreatment with pyridostigmine substantially increases protection against GA, GB, and VX in guinea pigs, although other oximes gave still more protection against GA at the doses tested (Inns and Leadbeater 1983). Much work will have to be done to determine whether HI-6 can be substituted successfully for P_2S or 2-PAM-Cl in a pretreatment/postpoisoning therapy regimen in humans.

B.2.4.4 Personal Decontamination

Under conditions of accidental release of the nerve agents and exposure of the civilian population, personal decontamination procedures can be helpful in reducing the percutaneous (skin) toxicity of these chemical agents. Trapp (1985) reviewed a study by van Hooidonk et al. (1983) in which household products were evaluated as skin decontaminants for selected nerve agents (VX and a G agent related to GA and GB) and also for a vesicant agent. This research involved in vitro studies with the abdominal skin from guinea pigs and in vivo studies with the same animal species. The household products tested included (1) tissue paper as a dry absorptive material; (2) washing powder, abrasive, flour, and talcum powder as adsorbing powders; (3) water, salad oil, bleaching

water, soapy water, and methylated spirits as wet decontaminants; and (4) combinations of absorbing powders and wet decontaminants. In vivo studies with nerve agents indicate that the best decontamination was achieved with absorption of the agent with flour followed by washing with water and wiping with wet tissue paper. However, merely washing the skin with soapy water gave significant results and is recommended if no other method is available. Speed is very important in the decontamination process. A time delay of 30 min significantly reduced the efficacy of all the decontamination procedures tested.

B.2.5 D2PC CODE CONSIDERATIONS

The Army Dispersion Code (D2PC) is used to calculate the toxic effects of chemical agent dispersal following an accidental release. The D2PC code was developed for estimating combatant lethalities and is, therefore, based on healthy *young* males dressed *in* combatant clothing under battlefield conditions. This code was not intended for the general population, which includes infants, children, the elderly, and the debilitated. It is important to determine whether the biological endpoints (e.g., 1% lethality) in the D2PC code appear appropriate for healthy young adults (70-kg man) and whether they are protective enough to be extended to the general population.

For agent GA, the 1% lethality value of 20 mg-min/m^3 and the no-deaths concentration of 12 mg-min/m^3 in the D2PC code appear to be reasonable, perhaps even overestimating risk for healthy young adults when these values are compared with the estimates for the incapacitating concentration (100 mg-min/m^3) and the minimum effective dose concentration for pupillary constriction (0.9 mg-min/m^3) (Table B.2).

For agent GB, which is more toxic than GA, the D2PC code values for 1% lethality and no deaths are 10 mg-min/m^3 and 6 mg-min/m^3, respectively. These values appear to be appropriate compared with the LCt_{50} by inhalation (70 mg-min/m^3), the ICt_{50} (35 to 55 mg-min/m^3), and the ED50 (2 to 4 mg-min/m^3) for agent GB found in Table B.2. These estimates may be inappropriate for infants and other sensitive members of the general population.

The D2PC code has the following values for VX doses: 1% lethality = 4 mg-min/m^3 and no deaths = 2 mg-min/m^3. If these dose-rate values are applied to healthy adults wearing combatant clothing and receiving the majority of their total VX exposure by inhalation, these dose rates are probably conservative from a health risk standpoint. However, the lethality estimates for VX are dependent on the amount of clothing worn, the wind speed and the size of the particles (for VX in aerosol form). With a wind speed of 20 mph and 10-μ aerosol particles, the percutaneous LCt_{50} might be as low as 10 mg-min/m^3 among clothed individuals (soldiers) (Krackow 1956). In unclothed (naked) persons exposed to 2-p particles at a wind speed of 15 mph, the estimated percutaneous LCt_{50} is 6 mg-min/m^3. These estimated LCt_{50} values are very close to the D2PC code's 1% lethality estimate for VX of 4 mg-min/m^3. Although it is not

anticipated that the population exposed to VX will be unclothed, the toxicity data suggest that percutaneous absorption may contribute greatly to the total dosage received, especially under high wind speeds. Thus, under unusual (and perhaps unlikely) conditions of high wind speeds with either large-particle VX aerosols or large amounts of skin exposure, the D2PC values may not be protective enough for either healthy adults or the general population. The dose-rate numbers in the D2PC code for the various levels of lethality should actually be lower under these unusual conditions. Accordingly, under these unusual conditions, the "zones" of lethality (no death, etc.) could extend further than is currently estimated by the D2PC code. End-result values for the D2PC code were selected at the 95% confidence level. That is, there is less than 1 chance in 20 that the true no-deaths dose is lower than that used in the D2PC model. This provides a margin of safety that may be wide enough to protect even the most vulnerable members of the population.

Identification of these concerns with respect to applying the D2PC code fatality dose estimates to a general public composed of individuals in various age classes and states of health led to an 80% downscaling of the no-deaths dose for use in emergency response planning (see Appendix L). The logic is that differences in body mass, inhalation rates, and potential sensitivity indicate that some individuals may be killed by doses that would not induce fatalities among healthy young males. However, it is also clear that the entire general population is not at the same risk as its most vulnerable members. Thus, the boundary of the emergency planning zones for each host site will be established by the location of the distance at which 0.2 times the D2PC no-deaths dose is attained under various accident assumptions (see Sect. 3 and Appendix J). The authors of this Appendix and the staff of the Centers for Disease Control (DHHS, Atlanta, Ga.) consider that this concept defines zones beyond which the most susceptible members of the population would experience no deaths. Further details describing the development of this concept can be found in Appendix L (Generic Emergency Response Concept Plan).

B.3 VESICANT AGENT TOXICITY

B.3.1 SUMMARY

The vesicant or blister agents to be discussed include H, HD, HT, and L (Lewisite). These blister agents are capable of producing severe chemical burns upon direct contact with human tissues, especially the eyes, skin, and upper respiratory tract.

- Vesicant agents interact with important cellular constituents and are capable of producing delayed and/or chronic health effects, as well as acute effects, under appropriate conditions of exposure and dose. Delayed effects may be manifested years after exposure ends.

- Vesicants are cellular poisons and produce death of individual cells in target tissues. The biological mode of action of mustard agent is that of an alkylating agent. By chemically reacting with cellular proteins, enzymes, RNA, and DNA, mustard alters these components in such a manner that individual cells are killed.

- Because mustard can react with DNA, it is capable of producing genetic effects (e.g., mutation).

- There is no specific antidote known for mustard poisoning. Once the chemical reaction of mustard with cellular constituents occurs, the process is not reversible. Therefore, immediate and thorough removal of the agent from contaminated tissues is essential.

B.3.2 ACUTE TOXICITY

The vesicant or blister chemical warfare agents present a quite different picture of acute clinical signs as compared to the nerve agents. While the nerve agents produce lethality at very low doses, the vesicants are not acutely lethal at similar low doses. In fact, mustard gas has been estimated to have caused only one-half of one percent of World War I battlefield deaths (Blewett 1986). More recent experience of soldiers in the Iran-Iraq War has been 3 to 4% fatalities following exposures to high battlefield concentrations of mustard agent (Dunn 1986). The actual battlefield concentrations are unreported. [Exposure *estimates* from a WWII Japanese poison gas factory have suggested 50 to 70 mg/m^3 air concentrations of mustard are acutely irritating and can produce most of the acute signs of mustard poisoning (Inada et al. 1978)]. Vesicants are usually classified as incapacitating agents, which can be lethal, given the proper circumstances of exposure. Their function in warfare is to decrease the opponents' ability to fight, a function that they achieve in a highly effective manner. Vesicant exposure produces chemical burns on tissues that come into contact with either vapors or liquid droplets/aerosols. Exposed skin surfaces, eyes, nose and throat, the bronchial tubes, and upper gastrointestinal tract (exposed from swallowed agent) are all at risk of developing chemical burns. Following initial tissue damage, various unpleasant and debilitating effects follow, such as the development of large, painful blisters that arise on skin exposed to mustard agent. An individual exposed to blistering concentrations of agent is certainly "incapacitated,* often for weeks before returning to normal activity. A description of acute clinical signs produced by individual vesicants follows.

B.3.2.1 Agents H/HD

B.3.2.1.1 Toxic effects

Sulfur mustard (see Sect. B.1 for physical properties and chemical structure) is the active ingredient in H and HD and a major component of HT (60% is HD). Because H does not contain as much active ingredient as HD (see Sect. B.1), it is expected to have slightly less blistering power than HD (U.S. Army 1975) but otherwise to have the same biological properties. A summary of acute effects (Blewett 1986; Medema 1986; Sollmann 1957a) after exposure to toxicologically active concentrations (50 to > 100 $mg\text{-}min/m^3$) of mustard includes the following:

1. A latency period of several hours before chemical burns become manifest (although at very high concentrations of mustard gas, immediate irritation may be produced). Because agent exposure does not produce immediate symptoms, exposed individuals do not promptly seek to decontaminate themselves or to request medical assistance.

2. Eyes are inflamed, eyelids become swollen, there is pain in the eyes and temporary blindness. The eyes are adversely affected at lower vapor concentrations than other tissues.

3. A variety of dose-dependent effects on the respiratory tract, including discomfort in the throat, continuous hoarse coughing, nasal discharges, copious mucus production, inflammation of respiratory passages (e.g., the bronchi), which can lead to secondary infection and a possible subsequent bronchopneumonia, which can be fatal.

4. A general irritation of the skin, first manifested as a rash with itching, which then develops (at higher exposures) into large blisters that are painful and may require weeks to heal.

5. At high doses, mustard can have deleterious effects upon the immune system, thus rendering the individual more susceptible to infections.

6. In terms of acute lethality, mustard is not as hazardous as the nerve agents. For example, its lethal potency is approximately 1 to 10% that of GB in dogs and rabbits. (These potencies are based on intravenous dosages, which provide possibly the best comparison of a chemical's inherent lethal toxicological potential.)

The toxicological effects of mustard agent are many and varied. A detailed discussion of them could fill many pages but would be inappropriate for this document. Table B.13 presents information regarding the acute toxic signs experienced upon receiving certain doses of HD. It is important to recognize that the various lethal doses of HD

Table B.13. Acute toxic effects of vesicant agents H/HD, HT, Lewisite, and T

Exposure route	H/HD	HT	Lewisite	T
		Inhalation LCt_{50}(mg-min/m^3)		
Human (estimated)	1,500[a,b]		1,200–1,500[b]	~400[c]
Monkey	800[b]			
Goat	1,900[b]			
Dog	600[b,c]	100–200[b]		
Cat	700[b,c]			
Rabbit	1,025[b]	3,000–6,000[b]		
Guinea pig	1.7006	3,000–6,000[b]		
Rat	800–1,512[b,d]		1,500(9 min)	
Mouse	860–1,380[b,d]	1,100(10 d)[b]	150(10 min)	
		820(15 d)[b]		
		Inhalation LCt_{50}(mg-min/m^3) (Head exposed, body protected)		
Mouse			1,400–1,600(10 min)[b]	
		Percutaneous LCt_{50} (vapor) (mg-min/m^3) (Head protected, body exposed)		
Human (estimated)	10,000[b]		100.000[b]	
Monkey	13,000[b]			
Dog	7,700[b]		40,000(10 min)[b]	
			30,000(30–60 min)[b]	
Cat	8,700[b]		30,000(30-45 min)[b]	
Rabbit	5,000[b]		15,000(10 min)[b]	
Guinea pig	~20,000[b]		20,000–25,000(10–40 min)[b]	
Rat	~3,000[b]		20,000(9–25 min)[b]	
Mouse	3,400[b]		300–7,000(10 min)[b]	
		Percutaneous LD_{50} (liquid)(mg/kg body weight) (Head protected, body exposed)		
Mouse			15[b]	
Rat			15–24[b]	
Rabbit			5–6[b]	
Guinea pip			12[b]	
Dog			-70, 38[b]	
Goat			10–24[b]	
		Percutaneous + Inhahation LCt_{50}(mg-min/m^3) (No protection)		
Mouse			900–2800(10 min)[b]	
			500(9–14 min)[b]	
Rat			1,500(9–25 min)[b]	
			580(60–180 min)[b]	
			20,000[e]	
Guinea pig			1,000(9–14 min)[b]	
			470(60–180 min)[b]	
Rabbit			1,200(7.5–13 min)[b]	
			1,500(60–310 min)[b]	
Goat			1,250(100–255 min)[b]	
Dog			1,400(7.5–15 min)[b]	

Table B.13 (continued)

Exposure route	H/HD	HT	Lewisite	T
		LD_{50}(mg/kg body weight) (Applied as liquid)		
Human (estimated)	100[b]			
Farm animals (unspecified)			10[f], IS'	
Dog			15[g], 38[e]	
		Skin LD_{50}(mg/kg body weight) (continued) (Applied as liquid)		
Rabbit	100[b]		4[h], 6[g]	
Guinea pig			12[f]	
Rat	9[i], 18[b]		15–24[e,g]	
	15 (96 h mortality)'			
	194 (24 h mortality)'			
Mouse	92[b]		15[f]	
Goat			15[g]	
		Intravenous LD_{50}(mg/kg body weight)		
Dog	0.2[b]		2.0[b,f]	
Rabbit	~1.1–4.5[b]		0.5'. 2.d	
Rat	0.7–3.3[k]			
Mouse	3.3, 8.6"			
		Oral LD_{50}(mg/kg body weight)		
Rat	17[b]		50[e]	
		Incapacitating dose ICt_{50}(mg-min/m³)		
Human, percutaneous (masked)	2,000(70–80°F)[b,l] 1,000(90°F)[m]	None established'	>1,500[b]	
Human, eyes	200[b]	None established[b]	<300[b]	
		Minimum effective dose, ED		
Human skin (irritation)			95 μg/man[f]	
Human skin (blisters)	50 mg-min/m³ [b,n] 32 mg/man[c]	-3.5 mg/man[b] 4 mg/man[c]	-200 mg-min/m³(30 min)[b]	4 mg/man[c]
Human eyes (marginal)	12–70 mg-min/m³ [b]			
Conjunctivitis	30 mg-min/m³ [n] (60 min)			
(Reddening, no incapacitation)	70 mg-min/m³ [b]			
(Reddening, mild incapacitation)	90 mg-min/m³ [b]			
Rabbit skin			-25 mg-min/m³(30 min)[b]	
Rabbit eyes		Similar to HD[b]	~1 mg-min/m³(30 min)[b]	
Dog skin			-50 mg-min/m³(30 min)[b]	
Dog eyes			-20 mg-min/m³(30 min)[b]	

Table B.13 (continue&

Exposure route	H/HD	HT	Lewisite	T
	No effect dose (mg-min/m³)			
Human eyes	<12[m]			
Human (estimated)	2 (≥90°F)[m]			
	Severe systemic effects (mg/kg body weight)			
Human (skin) (estimated)			13.4[p] 8.1[q]	
	Inhalation. lowest *lethal dose (mg/m³)*			
Human	150 (10 min) 70 (30 min)		48(30 min)'	
	Skin absorption, lethal (mg/kg body weight)			
Human	64'		37.6[q] 53.7[p]	

"Because HD exposures are cumulative, the lethal dose is not changed with variations in time of exposure, within reasonable limits.

[b]U.S. Army, 1974.

Robinson 1967.

"Ranges of LCt_{50}s arc summarized for all exposure times reported.

[e]U.S. Army 1984.

National Technical Information Service 1946.

'Cameron, Carleton and Short 1946.

"Danielli et al. 1947.

'Young 1947.

[k]Vojvodic et al. 1985.

[k]Anslow et al. 1948.

'Incapacitating dose varies significantly with amount of perspiration on skin surface, which is, in turn, dependent on ambient temperature and humidity levels.

[m]McNamara et al. 1975.

"Mild to moderate erythema is produced at ambient temperature of 90°F.

[o]Dahl et al. 1985.

[p]Windholz et al. 1983.

[q]Sollman 1957b.

'Back, Thomas, and MacEwen 1972.

[s]Inada et al. 1978.

'WHO Technical Report 1970.

listed for humans are *estimates* based upon extrapolations from other species. As previously described, even under wartime exposure conditions HD is not notable as a lethal agent. Significant human exposures to HD during the chemical stockpile disposal operations are conceivable only during an unplanned release. Exposures expected during such circumstances would occur by inhalation of HD vapors or skin contact with vapors or liquid. Any form of injected (i.e., subcutaneous) dose is probably irrelevant to expected exposures during stockpile disposal. Dermal exposure to liquid HD droplets (e.g., see skin LD_{50} values) is also unlikely for the general public; an explosive accidental release would be required, but no munition in the stockpile contains both enough explosives and mustard to create a hazardous mist more than a few hundred yards away. Oral ingestion of HD agent is not considered likely either; the possible effect of contamination of food and water supplies is discussed in Sect. B.6.

The intravenous LD_{50} values indicate -that variation among species spans a range of about fortyfold from least to greatest, in terms of this toxicity parameter. The intravenous LD_{50}s appear to be in the range of 1 to 4 mg/kg body weight for rabbits and rats (mice seem somewhat more resistant while dogs are more sensitive). Comparison of these mustard values with intravenous LD_{50}s for other agents can also provide an estimate of the relative lethal potency of various agents. In Figs. B.l and B.2, the intravenous and inhalation lethal dosages of various agents for two animal species are compared. The nerve agents are all much more potent in producing acute lethality than mustard by intravenous dose. Comparison of lethal inhalation doses demonstrates that mustard is only approximately 25 to 50% as potent as GA, 5 to 10% as potent as GB, and 1 to 3% as potent as VX. Comparative potency of the various agents by differing routes of exposure is, of course, not only dependent upon their differing toxicologic potentials, but upon differing absorptive properties (i.e., effectiveness of skin penetration, amount of agent that reacts with skin and respiratory tissues in comparison with amount absorbed into blood, etc.).

The LCt_{50} dosage by inhalation is similar for a number of animal species (i.e., approximately 1000 to 1500 mg-min/m^3). The human LCt_{50} is estimated on the basis of animal toxicity data. The document providing the estimate of the human LCt_{50} for inhalation (U.S. Army 1974) also notes that because the effects of HD are cumulative (i.e., very limited detoxification), the lethal dosage is not significantly changed, within reasonable limits, with variations in exposure time. What these time limits are (minutes or hours) is not stated. McNamara et al. (1975), however, note that the effect of the same dose of HD is lessened when given over a longer period of time, which suggests that some biological detoxification takes place. A value for a lethal inhalation dose of HD for a human is also given in Table B.13, Essentially no details about this exposure (whether it is again an estimate or based on an actual exposure) are given in the reference (Back, Thomas, and MacEwen 1972).

Two types of lethal skin exposures to HD are denoted in Table B.13. In one type, animals are exposed in a special chamber in which only the body is in contact with the HD vapor, while the head is in uncontaminated air. It seems possible that, because of the low volatility of HD, condensation of HD vapors onto the animal's body might occur, thus rendering at least some of the total dose as a liquid application upon skin. The LD_{50} dosages for liquid HD applied directly to the skin are also indicated. It can be seen that in all species studied, the LD_{50} dose (on a mg/kg basis) for skin application is considerably larger than that for intravenous injection (e.g., 20 mg/kg skin LD_{50} vs 0.2 mg/kg intravenous LD_{50} for dogs). This would be expected because much of the highly reactive HD would react with dead cells, which form the outer layer of skin, leaving only a fraction of the total applied dose to reach living skin cells or be absorbed by the blood.

Before a discussion of the incapacitating dosages of HD listed in Table B.13 is undertaken, the clinical signs (Blewett 1986; Lohs 1975; Medema 1986; Sollmann 1957a) associated with nonlethal HD poisoning should be described. The following descriptions of toxic effects should not be construed as all-encompassing or definitive with regard to timing of manifestations, etc. Rather, the description should be viewed as representative of the constellation of effects that can arise.

The biological activity of mustard agent is characterized by a latent period followed by severe inflammation, blistering, and local necrosis (cell death). Medema (1986) states that there is an observed effect within one hour of exposure only when gross contact with liquid agent occurs.

Toxicological effects are usually local at the point of agent contact with skin, eyes, or respiratory tract. The first effect to manifest itself is usually eye irritation (watering, reddening, pain, swelling of the eyelids, etc.), taking place 2' to 3 h following exposure. In the period 4 to 16 h following exposure, eye effects become more severe, nasal discharge occurs, nausea and vomiting may begin and recur for several hours, and diarrhea may develop. Skin rashes also begin to emerge at this time. Twenty-four hours after the exposure, the eyes are swollen almost shut and very painful, the skin is swollen and reddened, there is hoarse coughing, and the throat is raw and irritated. During the next 24 h, skin erythema may progress into (sometimes large) blisters, and eye irritation begins to subside (but the inflammation persists for several days). In cases of severe exposure, the damage to the respiratory tract becomes evident; expectoration (phlegm discharge and spitting) is copious, with mucus and occasional sloughing off of tracheal mucosa. Secondary infection of the respiratory tract can occur (e.g., bronchopneumonia), with attendant fever, and potentially can be followed, if severe and untreated, by death. In less severe exposures, involvement of the respiratory tract may be manifested by any or all of the following: rhinitis, laryngitis, tracheitis, and bronchitis (e.g., inflammation of the nasal passages, larynx, trachea, and bronchial

tubes). From the experience of World War I soldiers exposed to mustard, damage to the throat and other portions of the upper respiratory tract presented the greatest potential for lethal consequences (Haber 1986; Sollmann 1957a). Recovery of individuals with respiratory damage to a state where moderate activity was possible could take from 4 to 8 weeks (Haber 1986). Of course, these exposures occurred before development of antibiotic therapy, and antibiotics would be of value in dealing with such secondary infections.

In cases of high-level exposure, eye inflammation with severe pain may persist for several days, with the irritation continuing at a lesser level for several weeks. The severity of skin lesions experienced by exposed persons is influenced by a number of factors besides extent of exposure, including individual differences in skin sensitivity, ambient temperature, amount of sweat on the skin, etc. (U.S. Army 1974; Sollmann 1957a). With milder exposures or as the first stage of a severe exposure, skin damage takes the form of an erythema (reddening) or rash, which may itch. If the exposure is not severe, erythema may be the only skin effect. With more severe exposures, erythematous areas begin to fill with fluid, and a blister arises (sometimes 3 to 4 in. in diameter), reaching its maximum size in approximately 24 h. After several days, the blisters usually break. Blisters are relatively painless for several days, but after five to six days the pain becomes severe upon exposure to air or on contact; sensitivity of the blistered area can persist for two to three weeks. Ulceration of the blister may or may not develop; in the most severe burns, a blister does not develop but the initial burn progresses to an ulcer, which may take 5 to 7 weeks to heal. As might be expected, mustard agent burns are susceptible to infection, and boils can develop in and around the affected area, although the fluid from the blister itself does not cause a secondary blister to develop.

Table B.13 presents mustard dosages that can produce the described symptoms. The eye is apparently one of the most sensitive organs to the toxic action of mustard vapors; concentrations of mustard barely perceptible by odor can produce eye damage while not affecting the skin or the respiratory tract (U.S. Army 1974). Dahl et al. (1985) note that vapor concentrations of 0.5 μg of mustard per liter of air (e.g., 0.5 mg/m^3) for exposure periods of 1 h (i.e., 30 $mg\text{-}min/m^3$) are sufficient to produce conjunctivitis in man. Mustard agent also penetrates the cornea very rapidly; 10 min is cited in Dahl et al. (1985) and Geeraets, Abedi, and Blanke (1977). Because of this rapid penetration and subsequent disappearance from the cornea1 surface, attempts to wash the agent from the eye must take place very quickly after exposure. Compounding this difficulty is the fact that there is a latent period before eye effects begin to appear, so that eye irrigation at the time such effects appear, is futile in removing the agent (Dahl et al. 1985). Minimal effective doses for various toxic eye manifestations can be delineated (Table B.13). Mild exposures (20 to 70 $mg\text{-}min/m^3$) to HD vapor may produce lacrimation (tearing) and swelling, while more severe

exposures (100 mg-min/m^3) can produce blepharospasm (blinking), blurring of vision, edema of the conjunctiva and eyelids, iritis, and a mucous discharge (Dahl et al. 1985; Geeraets, Abedi, and Blanke 1977; Sollmann 1957a). Pain is associated with these ocular effects. Following this acute phase, there occurs a gradual regeneration of the damaged tissues, so that the eyes may become normal within weeks following the exposure (Dahl et al. 1985). After severe injury, however, heavy cornea1 vascularization may occur, and cornea1 erosion and ulceration may develop over several months (Geeraets, Abedi, and Blanke 1977). A late phase of mustard-induced keratitis or keratopathy, which essentially results in loss of vision, may occur 8 to 40 years after exposure (Dahl et al. 1985; Grant 1986). This point will be further discussed in Sect. B.3.3.2, chronic effects of mustard.

Because the eyes are so sensitive and because eye effects can appear more rapidly than other toxic signs (Sollmann 1957a), the eye can serve as a useful indicator of a no-acute-effect level of mustard (see Table B.13) (i.e., a dose of agent that does not produce detectable eye effects). The nose might also serve as a useful monitor for low levels of mustard vapor, because the odor is distinctive, but individual variation in sense of smell, nasal congestion, etc., may render this rather subjective. The minimum concentration of mustard normally detectable by smell is stated to be between 0.7 and 1.3 mg/m^3 (Thorpe and Whiteley 1939). The no-effect dose for the eyes of 12 mg-min/m^3 (Table B.13) seems reasonably consistent with the literature information available. The incapacitating eye dose (200 mg-min/m^3) in Table B.13 is probably somewhat subjective, depending on the individual.

Vapor, mist, and/or liquid droplets of mustard can produce skin damage as well as eye damage. At the cellular level, the effects on skin can be described as follows: basal cells of the epidermis (i.e., those cells that regenerate the outer skin surface) are quite rapidly killed by the agent; there is separation of the epidermis from the underlying dermis and increased vascular permeability (i.e., fluids from the blood leak into the damaged area), producing a dermal edema; and some degree of inflammatory response occurs, in which white blood cells invade the damaged area (Vogt et al. 1984; Papirmeister et al. 1984). Vascular leakage accounts for the large blisters produced by mustard poisoning. The doses of HD that can produce various degrees of human skin damage are: 0.1 *to* 1.0 $\mu g/cm^2$ for erythema/edema; 1 *to* 2.5 $\mu g/cm^2$ for edema, vesication; >2.5 $\mu g/cm^2$ for central necrosis and vesication on the circumference of the necrotic area (Papirmeister et al. 1984). Nagy et al. (1946) estimate that 6 $\mu g/cm^2$ of mustard agent is effective in producing vesication in 50% of exposed sites. Thus, small amounts of liquid mustard applied to human skin *can* produce damage.

In their studies of mustard agent effects on skin using human volunteers, Nagy et al. (1946) reported that there was a linear relationship between the amount of mustard that penetrated the skin and the time of exposure. In other words, the longer the time of exposure, the more mustard that penetrates the skin, and the more severe the

ensuing damage. No difference in rate of penetration of mustard between black and white skin was seen in these studies (Nagy et al. 1946). Nagy et al. (1946) also compared the penetration of mustard at two ambient temperatures (22°C and 31°C was higher at 31°C, but no difference in the ability of mustard to produce blistering was observed at the two temperatures. The amount of moisture on the skin surface also has a decided effect upon the degree of damage produced by HD (U.S. Army 1974, 1975; Nagy et al. 1946). U.S. Army reports (U.S. Army 1974, 1975) indicate that increasing damage occurs with increasing wetness of skin, so that ambient temperature, relative humidity, and degree of physical activity can all be important variables. Therefore, the median skin incapacitating doses in Table B.13 are given for different ambient temperatures.

The minimal effective dose (50 mg-min/m^3) for human skin in Table 8.13 refers to development of a mild to moderate erythema or rash. The time to appearance of this rash varies depending on extent of exposure and skin wetness; in general, mild skin damage appears relatively late after exposure and heals earlier than the more extensive damage from blistering concentrations (Sollmann 1957a). Following healing, areas of skin affected by mustard are usually altered in pigmentation (i.e., sometimes tanned or blackened), may be hypersusceptible to irritation, and can be permanently scarred (Sollmann 1957a). There are apparently large individual differences regarding susceptibility to skin damage (100-fold greater dose necessary to produce a reaction in a resistant vs sensitive person); racial differences may also exist (Sollmann 1957a). The level of skin damage that becomes incapacitating is, as with the eyes, somewhat subjective; the incapacitating skin dose in Table B.13 is 1000 to 2000 mg-min/m^3, for an individual with a protective mask. The comments previously made with regard to skin should be understood to apply to regions of skin that are normally exposed to the air (e.g., arms and neck). Medema (1986) states that the groin region is 10 times more sensitive to the skin-damaging effects of mustard than other body regions.

Blewett's (1986) article has a table that shows the distribution of mustard gas wounds to various body parts in World War I soldiers (each victim had an average of 3.5 separate mustard wounds). The data in this table indicate that the regions affected in the highest percentage of victims were the eyes (86%), the respiratory tract (75%), the scrotum (42%), the face (27%), and the anus (24%). These data indicate the most vulnerable areas of the human body to the noxious activity of mustard vapors or mists (moist body parts in general).

Acute systemic reactions to mustard are only likely to occur with severe exposures (i.e., 1000 mg-min/m^3) (U.S. Army 1974). Some of the toxic signs might include loss of appetite, malaise, nausea, vomiting, depression, and fever (U.S. Army 1974, 1975; Lohs 1975). These effects may appear before or concurrently with skin manifestations. Recovery from the vomiting may occur within 24 to 36 h, although the other manifestations may continue for longer periods (U.S. Army 1974). The

extensive skin burns from a severe mustard exposure apparently do not share the toxic properties associated with thermal burns (Sollmann 1957a). An incident occurred in World War II in which personnel had to swim through a mixture of mustard agent and fuel oil to escape sinking ships. Some of these individuals experienced a systemic shock-like syndrome that was not amenable to the usual medical therapy for shock (Bodansky 1945). A form of systemic poisoning was also noted in mustard workers in poison gas factories during World War I (Haber 1986). The symptoms included listlessness, depression, headaches, indigestion, and breathlessness. The concentration of mustard these individuals were exposed to is unknown, but was high enough to produce acute toxic effects.

Mustard agent at sufficiently high doses can also produce acute injury to the hematopoietic system (e.g., bone marrow, thymus, lymph nodes, and spleen) thereby causing a depression in circulating white cells and a consequent increased susceptibility to infection from pathogenic microorganisms (U.S. Army 1975; Bodansky 1945). Friedberg, Mengel, and Schlick (1983) administered 15 mg/kg of body weight of mustard agent by intraperitoneal injection to mice and noted that this dose significantly depressed the number of bone-marrow cells/femur at 3 and 8 days following treatment. The white blood cell count was also depressed at 3 days after treatment but rose to control levels by 8 days. The dose given to these mice was definitely high, as the LD50 for mustard by this same route of injection was 19 mg/kg. Catalini et al. (1971) injected 0.625 mg/kg body weight (approximately 50% of the LD50 dose) of mustard subcutaneously into young rats and found evidence of injury to the thymus, with a maximal inhibition of cell division in the thymus occurring the fifth day after injection, Recovery of the thymus from this injury was gradual and slow. What doses might produce damage to the bone marrow or white cells in humans is not available from the literature. How long this lowered white cell count might persist in humans following exposure is also unclear.

If H or HD were released to the environment during disposal operations, an obvious concern is the potential exposure of humans to residues of the agent that could be deposited by atmospheric dispersion over some defined area. Of course, an evacuation of the area would be expected, and criteria for safe reentry (Sect. B.6) would need to be established. The persistence of the agent in the environment is also of concern for ecological systems and is discussed in that regard in appropriate sections of the PEIS (Sects. 3 and 4). Only a very brief summary of the persistence of H/HD as this relates to human health is presented here.

One of mustard's most useful properties, from a military standpoint, is its persistence on surfaces (Blewett 1986; U.S. Army 1975; Medema 1986). Mustard is not really a gas at ordinary temperatures, and droplets of the agent released, for example, in an explosive accident would deposit on surfaces and slowly evaporate, thus posing a risk from inhalation of the agent as well as a hazard of direct contact with

agent-contaminated surfaces. Indeed, this very set of conditions subsequent to mustard shelling was observed in World War I (Blewett 1986). The important question is how long does this hazard persist? In extreme cases, mustard can apparently persist for decades, as indicated by anecdotal accounts of individuals receiving burns on World War I battlefield sites contaminated with mustard many years after the end of World War I (Blewett 1986). The usual time frame given for the persistence of contact-hazard mustard is approximately one week, (Blewett 1986; Dick 1981; Trapp 1985) under average weather (nonwinter) conditions in open terrain. An additional item of concern with regard to mustard is its persistence and/or degradation in water. This is of importance in establishing when potentially contaminated surface water supplies may be deemed safe to use (Sect. B.6).

B.3.2.1.2 Mode of action

In terms of chemical reactivity, sulfur mustard is a classic example of an alkylating agent. In basic terms, this means that the chemical structure of mustard is such that it can easily rearrange to a chemically reactive form, which can readily interact with a variety of molecular structures. In living cells, there are a variety of "target" molecules with which mustard can readily combine. Thus it can react with components of DNA, RNA, and proteins (Dixon and Needham 1946; Grant 1986; McNamara et al. 1975; Papirmeister et al. 1985; Trapp 1985). This chemical interaction results in transfer and attachment of part of the mustard molecule (an alkyl group, $-CH_2-CH_2-S-CH_2-CH_2-Cl$) to specific locations on biological molecules (McNamara et al. 1975). Hence the name, alkylating agent, has been applied to this compound and others that behave in a similar manner. The chemical modifications that various biological molecules undergo through alkylation can result in severe disorganization of normal biological function of these molecules. This, in turn, can lead to disruption of cell function, which can cause cell death. Thus, at the cellular level, mustard is considered a cell poison (Medema 1986), and it is particularly toxic to dividing cells. Cytostasis (i.e., blockage of the normal cell cycle progression), mutation (i.e., genetic damage to DNA), and slow cell death can also result (Vogt et al. 1984). Indeed, from early biological studies of mustard, it was known that this agent produced many cytological abnormalities in dividing cells, with particular effects upon mitosis (Fell and Allsopp 1948). Because of the similarity of the lesions produced by mustard and X rays, the mustards and other similarly acting chemicals are sometimes termed "radiomimetic," that is, imitating the effect of radiation (Gross et al. 1985; Lohs 1975). Thus, intestinal epithelial damage leading to diarrhea, depression of proliferation of white cell precursors in the bone marrow leading to depressed white blood cell counts and injury to respiratory epithelium can all be features of mustard poisoning (Vogt et al. 1984). The skin epithelium is an important target because of its proliferating basal cell layer (Vogt

et al. 1984). It should not be concluded, however, that mustard is only effective against proliferating cells; at sufficient concentration, it produces cellular necrosis (i.e., death) in any exposed cells (Grant 1986; Vogt et al. 1984).

From a biochemical point of view, it would be important to know the exact molecular sites that are attacked by mustard, as this might allow development of rational therapy at the molecular level. Unfortunately, because such a wide variety of cellular targets are available for reaction with mustard, the problem becomes very complex. Damage to DNA, for instance, a cross-linking of two adjacent strands of DNA, can account for many of the deleterious cellular effects (Grant 1986; Gross et al. 1985 ; Lohs 1975). There are many other cellular macromolecules besides DNA that are susceptible to mustard attacks. However, Levy (1965) found evidence of cell membrane modification by doses of mustard too small to effectively alter DNA, and Dixon and Needham (1946) found certain enzymes to be inhibited, while others were not. An interesting biochemical hypothesis to account for the generation of skin lesions by mustard has been proposed by workers at the U.S. Army Medical Research Institute of Chemical Defense (Papirmeister et al. 1985; Gross et al. 1985). This hypothesis links initial chemical binding of mustard to DNA, through a complex series of steps, to release of intracellular enzymes that are responsible for the skin damage and blistering produced by the agent. As the authors themselves point out, however, further work is needed to validate this hypothesis and devise procedures for attenuating mustard damage.

B.3.2.2 Agents HT/T

Agent HT is a product of one process for making mustard. HT contains about 60% sulfur mustard and <40% agent T, and a variety of sulfur contaminants and impurities. The acute effects of HD. are described in Sect. B.3.2.1. Agent T (bis[2(2-chloroethythio)ethyl]ether, see Table B.l) lowers the freezing point and thereby increases stability. It also possesses significant toxic properties of its own.

Agent T has been considered a mustard with relatively weak vesicant action because of observed delay in symptom onset (Auerbach and Robson 1947a). However, the human doses for skin blister development (4 mg/man) and inhalation lethality (LCt_{50} of 400 mg-min/m^3) from exposure to T are much less than for the mustard agent HD (percutaneous blisters at 32 mg/man; inhalation LCt_{50} of 1500 mg-min/m^3) (Robinson 1967; see Table B.13). Thus, agent T, far from being a mere additive, contributes considerable biological activity to the HT mixture for direct tissue exposure.

Available data characterizing HT toxicity have been confined to studies performed by the U.S. Army, which are summarized in chemical agent data sheets (U.S. Army 1974). Results of animal and human acute toxicity testing are presented in Table B.13.

Median incapacitating and lethal doses to man and experimental animals have not been established, although lethal exposures may be obtained through the eye, skin, inhalation, and ingestion routes (U.S. Army 1974). No data have been identified that would allow characterization of time of toxic effect onset or tolerable environmental concentrations to unprotected populations. Most biological effects observed after animal exposure to HT are similar to those induced by HD, although HT is more rapid and/or seve're. This greater activity is a result of the presence of stable agent T in the mixture; the more volatile HD dissipates and leaves a reactive blend containing a higher concentration of T. The increased toxicity of HT is reflected in the agent-specific estimates for one- percent-lethality distance, calculated to be that distance at which a given dosage would be lethal to 1% of an unprotected population (U.S. Army 1979). The critical dosage is 150 mg-min/m^3 for HD, but 75 mg-min/m^3 for HT.

B.3.2.3 Agent L (Lewisite)

Lewisite (dichloro (2-chlorovinyl) arsine] is considered not only a lethal vesicant but also a systemic poison when absorbed into the bloodstream (Table B.l.). The liver, gall bladder, and bile duct are particularly vulnerable, although damage to the kidneys and urinary tract is also possible at high skin doses (Cameron, Carleton, and Short 1946). Lewisite inhalation and ingestion severely damage the mucous membranes of the airways, mouth, stomach, and intestine (Cameron, Carleton, and Short 1946). Like the mustards, Lewisite is also a cellular poison, but in a somewhat different manner. Rather than indiscriminantly destroying proteins, Lewisite directly affects cellular enzyme systems.

Lethal exposures in humans and experimental animals can occur via inhalation, skin or eye contact, or ingestion (Table B.13). Mustard and Lewisite exhibit approximately equal human inhalation toxicity (1200-1500 mg-min/m^3); however, Lewisite is faster acting and more toxic via direct skin contact. According to some estimates, a 2-mL skin dose of liquid Lewisite in an adult (i.e., 37.6 mg/kg) can be fatal (Sollmann 1957b; Windholz et al. 1983). Without treatment, death from such a dose can occur in a matter of hours. One hypothesis contends that immediate death is due to "Lewisite shock," or loss of blood plasma resulting from the increased permeability of capillaries damaged by circulating Lewisite (Cameron, Carleton, and Short 1946). Severely burned victims of house fires, vehicular accidents, etc., suffer similar loss of blood plasma ("burn shock"). Smaller, but still lethal, doses of Lewisite reduce liver function and result in death among experimental animal populations after some delay, but usually within a week after exposure (Cameron, Carleton, and Short 1946). The threshold for onset of severe systemic effects in humans is approximately 10 mg/kg to the skin (range of 9.1 to 13.4 mg/kg) (Sollman 1957b; Windholz et al, 1983).

Lewisite exposure is further characterized by immediate onset of pain, in direct contrast to the delayed action of mustard agents (Cogan 1943; Sim 1971). So it is likely that, unless unconscious, anyone exposed to Agent L would seek and receive some degree of decontamination and/or treatment. Decontamination by copious flushing with water or mild solutions of sodium bicarbonate or detergent needs to be particularly swift in the case of ocular exposure, in which permanent blindness from cornea1 necrosis and secondary scarring may result if decontamination is not accomplished within 60 s (U.S. Army 1974). Inflammation of the iris can also result from sublethal exposures to the eye. Information and treatment protocols for specific Lewisite antidotes and decontamination procedures are provided in Sect. B.3.4.3.

It is not known if Lewisite per se is persistent. However, arsenic is an elemental poison and any group of residual hydrolysis, combustion or decontamination products is likely to contain an arsenical compound. See Table B.18 and Sect. B.5 for an analysis of Lewisite breakdown product toxicities.

B.3.3 CHRONIC TOXICITY

In addition to the acute effects produced by exposures to vesicant agents, there is also the possibility of chronic effects arising after exposure to these agents. For the sake of discussion and data organization, we will use the term "chronic toxicity or effects" generically to encompass any adverse biological effects that are not acute. The reader should understand that such categorization includes effects that might be more precisely defined as "delayed or late" (i.e., manifested at some time after exposure, sometimes after a period of seeming inactivity), or effects that arise following long-term exposures to low concentrations or amounts of agent. A rigorous application of "chronic" would include only those adverse biological effects that persist over a long period of time. The exposures that produce these "chronic effects" could be (and from epidemiological evidence, have been) either brief or long term.

B.3.3.1 Agents H/HD

Because of the ability of mustard to react irreversibly with a variety of biological molecules, including DNA, RNA, and proteins, the resultant biological damage could have immediate consequences, or might not manifest itself for a considerable interval. The late or delayed effects we consider for HD include eye toxicity; respiratory diseases (excluding respiratory cancer); carcinogenesis (i.e., development of cancer); mutation, particularly in relation to reproductive effects; and a category that includes other, less well-documented or defined effects. Before more in-depth discussions of these points, a brief summary of major findings in regard to chronic effects of mustard will be presented.

1. In some of the individuals who received eye damage from mustard in World War I, the lesions apparently healed. Decades later, eye deteriorations occurred, in some cases leading to blindness.

2. An increased risk of developing chronic bronchitis has been observed among individuals poisoned by mustard in World War I.

3. Cancer of the respiratory tract (e.g., lung, larynx, and pharynx) has been associated with occupational mustard gas exposures in epidemiological studies. Note that these workers were exposed in wartime, under less than optimal industrial hygiene conditions, to relatively high levels of mustard (high enough to cause acute toxic symptoms) over an extended period of time (often years). Furthermore, these exposures were not to mustard only, but simultaneously to other chemical warfare agents. World War I soldiers who were poisoned by mustard showed some increased risk of lung cancer, but this increase was neither large nor statistically significant (see following discussion).

4 Evidence exists that these same groups of wartime poison-gas factory workers are also at increased risk of developing skin cancer. In addition, individuals whose skin has been blistered by mustard are vulnerable to subsequent injury at the same sites.

5 Mustard can produce cancer in experimental animals, and a variety of biological test systems have found it to be mutagenic. In addition, some biological assays have demonstrated heritable genetic effects, implying that mustard produces damage in parental germ cells (i.e., reproductive effects).

6 The evidence for human reproductive effects following mustard exposure is equivocal. Some investigators have reported evidence for such effects in workers occupationally exposed to mustard, while others, studying another occupational group, have not seen such effects. Again, note that these wartime occupational groups probably received long-term exposures to appreciable doses.

B.3.3.1.1 Chronic eye toxicity

Eye damage suffered by most soldiers exposed to mustard in World War I was temporary in nature, and no permanent effects were observed (Grant 1986). In a smaller number of soldiers, where the eye was probably exposed to higher vapor concentrations or liquid droplets, a permanent, relapsing condition developed (Geeraets, Abedi, and Blanke 1977; Grant 1986). This chronic condition, called keratitis or delayed keratopathy, is characterized by recurring erosion and ulceration of the cornea, which can eventually lead to vision impairment or blindness (Geeraets, Abedi, and Blanke 1977; Grant 1986). This delayed effect can have a

latent period (a state of seeming inactivity) of 8 to 40 years after apparent recovery from the acute injury (Dahl et al. 1985), and in some cases has been found to relapse (alternating ulceration and healing) for decades (Grant 1986). A single acute injury in which the adult victim received immediate medical attention after direct eye exposure to mustard (unreported whether vapor or liquid droplets) illustrates the potential for a delayed type of eye damage (Geeraets, Abedi, and Blanke 1977). Initially the individual seemed to exhibit normal recovery. Two years after the event, however, the cornea was opaque, and vision was reduced to light perception only. This may be an exceptional case, as the dose to the eye could have been very large, or the individual could have been hypersensitive or experienced an infection or unrelated injury. Balanced against this must be the experience of most World War I soldiers, many of whom experienced severe acute eye effects but did not develop any permanent eye damage.

B.3.3.1.2 Respiratory diseases

Evidence from occupational and wartime exposures indicates that mustard agent can produce chronic respiratory damage given appropriate conditions of exposure. The respiratory conditions that can arise include a range of ailments from asthma-like conditions to a severe chronic emphysematous bronchitis, and secondary infections such as bronchopneumonia and tuberculosis (Lohs 1975; Sollmann 1957a). Among groups of World War I soldiers exposed to mustard, subsequent epidemiological studies have indicated the following results:

1. Among a group of British pensioners, almost all in the group exposed to mustard gas were diagnosed as having chronic bronchitis (but the pensions had been granted because of chronic bronchitis, so this does not necessarily mean all mustard-gas-exposed soldiers developed chronic bronchitis) and displayed an excess mortality from chronic bronchitis deaths as compared to the general male population. (Case and Lea 1955).

2. In a group of U.S. World War I veterans, the incidence of chronic bronchitis was much higher (65%) in mustard-gas-exposed men than in control groups (i.e., 35% in nongassed veterans who had pneumonia during the flu epidemic of 1918 and 20% in veterans who were only wounded in the extremities) (Beebe 1960). The study by Beebe also demonstrated that the mustard-gas-exposed men had a higher incidence of death from tuberculosis and pneumonia than the control groups.

In groups of workers occupationally exposed to mustard, evidence of respiratory damage was also found. Increased evidence of death from respiratory diseases (including cancer) was observed in a group of mustard gas workers in a poison gas factory that operated in Japan during World War II (Nishimoto et al. 1983), and a high proportion of these workers also had chronic bronchitis (Wada et al. 1968). A

confounding variable of their exposure was that they were also exposed to Lewisite and other gases, but the amount of L manufactured was small in relation to mustard. Further provided in Wada et al. (1962a), who noted that productive cough, irregular fevers, long-standing chronic bronchitis, emphysematous changes, and pleural adhesions were common among these individuals.

It seems clear from these studies that exposures to mustard gas can produce permanent respiratory damage, which can take the form of a chronic bronchitis and which can also predispose affected individuals to other respiratory infections (e.g., pneumonia, tuberculosis). It must also be noted, however, that dosages of agent capable of producing these respiratory effects are not well defined. An estimate of mustard concentration in the atmosphere in areas of falling poison gas shells in World War I is 19 to 33 mg/m^3 (Thorpe and Whiteley 1939). In the case of the Japanese workers, although some occupational safety measures were employed during factory operation, wartime worker safety provisions were minimal at best, and acute toxic effects from exposure to mustard and other chemical warfare agents were frequently reported among the workers (Wada et al. 1962b). In fact, some workers even died as a result of acute gas poisoning during plant operation. An estimate of airborne mustard concentration in this factory is 50 to 70 mg/m^3 (Inada et al. 1978). Another factor to be considered is that occupationally exposed individuals experienced daily mustard doses over a period of years.

Appropriate enforcement of industrial hygiene practices can make a significant difference in the delayed respiratory effects seen among war gas factory workers. Manning et al. (1981) retrospectively studied a group of workers from a British mustard gas factory active in World War II and found that the only significantly increased respiratory mortality compared to controls was that resulting from pneumonia. The pneumonia mortality incidence was questionable because of assumptions made about untraced members of the studied group. Chronic bronchitis was not significantly elevated among the workers. The authors attribute the differences between the Japanese and British workers to differences in protective industrial hygiene measures between the two plants.

We would conclude from these studies that respiratory injury with delayed effects is very unlikely for the general public except in the case of an accidental release of mustard; even then, the possibility of such effects is very much dependent upon concentration of agent and duration of exposure. Epidemiological evidence suggests that exposures associated with eventual development of significant respiratory effects were either occupational exposures of long duration to (at least) irritating levels of mustard, or exposures of unprotected soldiers in battlefield situations.

B.3.3.1.3 Carcinogenesis

Mustard has been shown to be carcinogenic in tests in experimental animals, and high-level exposures to mustard agent in wartime have been associated with increased cancer risk in humans. The human evidence will be discussed first, because it is probably more relevant to concerns regarding human health effects.

In soldiers exposed to mustard in World War I, there is evidence of a positive association between mustard exposure and increased risk of developing lung cancer 10 to 40 years after exposure, but the increased risk is not large (Beebe 1960; Case and Lea 1955; IARC 1975; Norman 1975). British pensioners exposed to mustard agent were compared to (1) war pensioners who had chronic bronchitis and (2) a group of amputees. Neither of the latter two groups had been exposed to mustard. Mortality from lung cancer was elevated in both the mustard-gas group and the chronic bronchitis group, compared to the amputee group. On the basis of this study, the increase in lung cancer could not be attributed to mustard agent exposures alone (Case and Lea 1955). Apparently, lung tissue damaged by either chronic bronchitis ***(or*** by the etiologic agent that produced chronic bronchitis) or battlefield mustard agent exposures was more likely to become malignant than nondamaged tissue.

American soldiers who experienced mustard agent exposures were also compared to two other groups: (1) soldiers who had pneumonia during the influenza outbreak of 1918, but who were not exposed to mustard and (2) wounded soldiers, who had neither pneumonia nor mustard exposures. Cases of respiratory cancer in the mustard-exposed group were somewhat elevated, compared to the other two groups [ratio of observed to expected cases: mustard gas, 1.47; pneumonia, 0.81; wounded, 1.15 (Beebe 1960)]. A further study of this same group of American soldiers, involving an additional 10 years of follow-up (Norman 1975), did not change the conclusions of the previous study, The risk of death from lung cancer among those poisoned by mustard was 1.3 compared to controls, with 95% confidence limits of 0.9 to 1.9. Norman (1975) concludes that this increased risk does not make a strong case for a carcinogenic effect of mustard, probably because an insufficient dose was received.

More definitive evidence of the carcinogenic action of mustard in humans comes from occupational exposures experienced in poison gas factories active in World War II. The Japanese factory on Okuno-jima island has already been mentioned (Sect. B.3.3.1.2). Japanese workers experienced exposures to multiple poisonous agents besides mustard (i.e., Lewisite, hydrocyanic acid, diphenylcyanarsine, and chloracetophenone), although mustard was produced in much larger quantities than the other agents (Wada et al. 1962b). The concentrations of mustard in factory atmospheres (estimated to be 50 to 70 mg/m^3) was great enough to produce most of the acute manifestations of mustard poisoning. Some protective clothing was worn, but it was not available in adequate amounts and was not in the best of repair. Industrial

hygiene measures were haphazard at best. All grasses and trees around the laboratories, particularly the mustard gas factory, died (Wada et al. 1962a; Inada et al. 1978). Retrospective studies of these workers have shown definite increases-in respiratory cancer among workers who produced mustard agent compared with office workers at the same factory (IARC 1975; Nishimoto et al. 1983; Tokuoka et al. 1986; Wada et al. 1962a; Wada et al. 1968;). Some highlights from these studies include:

1. increased mortality due to respiratory cancer in mustard gas production workers [i.e., 33 deaths in a group of 495 workers compared to 0.9 deaths expected in a group of this size (Wada et al. 1968)];

2. evidence of a dose-response relationship between mustard exposure and subsequent development of respiratory cancer [i.e., production workers and other workers whose jobs directly related to poison gas experienced higher respiratory cancer risk, compared to workers in the same factory not involved at all with the poison gases (Nishimoto et al. 1983; Yamakido et al. 1985b)];

3. a standardized mortality ratio of lung cancer for workers directly involved with poison gas production slightly larger than three, indicating that the number of deaths from lung cancer in this occupational population was more than three times that of males living in the surrounding area (Nishimoto et al. 1983); and

4. histological evidence that most of the respiratory tract cancers arose in the proximal part of the respiratory tract (e.g., pharynx, larynx, trachea) rather than the periphery, as would be expected from inhalation of a carcinogenic gas (Nishimoto et al. 1983; Tokuoka et al. 1986).

The cancers occurred in the tongue, pharynx, sinus, larynx, trachea, and bronchi. In a group of mustard-gas-exposed Japanese war factory workers who developed lung cancer, the average length of employment was 5.7 years (Tokuoka et al. 1986).

Besides the observed respiratory tract cancers, a recent report indicates an increase in digestive tract neoplasms in these Japanese workers, although no further details are indicated (Yamakido et al. 1985b). The mustard production workers also experienced chronic skin damage (pigmented and depigmented spots) and skin cancers (basal cell carcinoma and Bowen's disease; Inada et al. 1978). These lesions were not observed among employees in the same factory who were not engaged in mustard production. In addition, a correlation between length of employment in mustard gas production and severity of skin lesions was noted. These results provide strong evidence for an association between mustard agent exposure and certain skin cancers; again it should be noted that doses were large, and the duration of exposure was long term

[mean of nine years' employment for those with skin lesions studied by Inada et al. (1978)].

Epidemiological studies of German poison gas factory workers have also indicated increased incidence of malignant respiratory cancers (IARC 1975; Lohs 1975). Klehr (1984) reported multiple skin tumors (i.e., basal cell carcinoma, Bowen's disease) in German workers who were exposed to sulfur mustard and nitrogen mustard without adequate protection during the dismantling of a poison gas factory. These individuals also experienced necrotic and gangrenous skin ulcerations, which had a tendency to spread and to be resistive to therapy.

In contrast to the occupational experiences reported above, a study of British workers employed in a mustard gas factory during World War II did not reveal a significant increase in cancer deaths (Manning et al. 1981). However, a significant increase in diagnosed cases of laryngeal carcinoma among these factory workers *when* compared to a control population was noted. These individuals with laryngeal cancer had worked in the mustard gas manufacturing process for four to five years. Although lung cancer deaths were elevated in the occupational group when compared to the appropriate control group, the increase was not statistically significant. The authors suggest that differences in the cancer risk of the British and Japanese groups could have been attributable to better industrial hygiene practices in wartime England.

Based upon available data, the IARC report (1975) concludes that: "There is evidence for an increased incidence of cancers of the respiratory tract in men exposed to mustard gas." Thus, mustard is included in a group of chemicals that are carcinogenic for humans, based upon sufficient evidence to support a causal relation between exposure to the chemical and cancer (Saracci 1981; Nelson 1981).

The foregoing studies, although important, do not provide useful source term information for estimating the carcinogenic activity of mustard agents. For wartime inhalation exposures, only surmises as to air concentration of mustard can be made. Animal carcinogenesis studies can provide more carefully defined doses and dose rates than most epidemiological studies although the problem of species extrapolation exists when experimental animal studies are considered. Evidence for mustard carcinogenesis in mice and *rats* is shown in Table B.14.

The airborne concentration of 1590 mg/m^3 for the mouse inhalation study was estimated by Heston and Levillain (1953) and is probably considerably larger than the actual concentration inhaled by the mice. Strain A mice, which have a genetic susceptibility to pulmonary tumors, were used in this study. Animals were sacrificed at 4 and 11 months after exposure. The pulmonary tumor incidence in the exposed animals (combining all mice examined), was 49%) compared to 27% in control animals. This result was statistically significant. It is interesting that this very short exposure time (15 min) was effective in producing tumorigenesis.

Table B.14. Chronic effects observed **for** the **vesicant agents H/HD, HT, Lewisite, and T**

Effect	H/HD Response (dose)	Lewisite response	T response
	Carcinogenicity		
Mouse-inhalation (1 5-min exposure)	Pulmonary tumors' (~1590 mg/m³)[b]		
Mouse-subcutaneous injection (6 weeks)	Fibrosarcomas at injection' site (-6 mg/kg)[d]		
Mouse-intravenous (6 d)	Pulmonary tumors' (~3–4 mg/kg)[d]		
Rat-inhalation (≥3-month exposure)	Skin tumors' (0.1 mg/m³)[g]		
Human-inhalation and skin deposition	Respiratory tract tumors, skin cancers[h] (unknown)'		
Mouse-skin (278 d)	Negative' (2 mg total)		
Mouse-skin	Negative' (dose unknown)		
	Mutagenicity		
S. typhimurium (Ames)	Positive'	Negative'	
Neurospora crassa (mold) (30-min exposure)	Specific locus mutation[m] (200 μmol/L)[n]		
Saccharomyces cerevisiae (yeast)	DNA damage[0] (500 μmol/L)		
Drosophila melanogasrer (fruit fly) (Parcntcral injection)	Specific locus mutation[p] (45 pmol/fly)		
Drosophila melanogaster (Vapor, 5-min exposure)			Chromosome breakage[q] (production of sex-linked lethal mutations)
Drosophila melanogaster (Vapor-6 to 30 min)		Negative[q]	
Drosophila melanogasrer (Vapor-l 5-min exposure)	Cytogenctic damage[q]— visible mutations, deletions, inversions (dose unknown)'		
Drosophila melanogasrer (Vapor-15-min exposure)	Sex chromosome loss and nondisjunction4 (dose unknown)		
Drosophila melanogasrer (Parcntcral injection)	Sex chromosome loss' and nondisjunction (75 pmol/fly)		
Drosophila melanogasrer (Vapor-l 5-min exposure)	Heritable translocation[q] (dose unknown)'		
Mouse-ascites cells (lntraperitoneal injection-l h)[u]	DNA damage' (5 mg/kg)[d]		
Mouse-L cells (10 min to 24 h)	DNA damage' (1 mg/L)[n]		

Table B.14 **(continued)**

Effect	H/HD Response (dose)	Lewisitc response	T response
Mouse leukocyte (Subcutaneous injection)[w]	Somatic cell mutation[y] (100 mg/kg)[d]		
Mouse leukocyte[v]	Somatic cell mutation[y] (1 μg/L)		
Mouse leukocyte[v]	Chromosomal aberrations[y] (20 μg/L)		
Hamster fibroblasts (20 min)	Chromosomal aberrations[v] (8 μg/L)		
Human cells (HeLa)[x]	DNA damage[z] (2 mg/L)		
Vicia faba (broad bean) (root meristem)		Negative?	
CHO cells--forward mutation assay	In progress[v]	In progress[v]	
Alkaline elution assay (DNA strand breaks)	Planned[v]	Planned[v]	
In vitro sister chromatid exchange assay	In progress[v]	Planned[v]	
	Teratogenicity		
Rat (intragastric)[dd]	Negative[cc]	Negative[v]	
Rabbit (intragastric)[dd]	Negative[cc]	Negative[v]	
Rat (inhalation-l to 52 weeks)	Negative[v]		
	Reproductive effects		
(Vapor--l 5-min exposure)	Dominant lethal mutation[q] (dose unknown)		
Rat (inhalation. ≥2 weeks)	Dominant lethal mutation[ee] (0.1 mg/m^3)[ff]		
Rat (inhalation)	Negative[f] (0.1 mg/m^3)[g]		
Rat, dominant lethal mutation	In progress[v]	Planned[v]	
Rat, three generations	In progress[v]	In progress[v]	

Table B.14 (continued)

Effect	H/HD Response (dose)	Lewisitc response	T response
		effects	
Rat, 90 d	In progress'	In progress'	

[a]Heston and Levillain 1953.
*Dose is estimated, assuming complete volatilization of HD.
[c]Heston 1953.
[d]Dose is expressed as mg/kg body weight.
'Hcston 1950.
[f]McNamara et al. 1975.
'Exposure to 0.1 mg/m^3 of HD was for 6.5 h/d, 5 d/week; for the remainder of each 24-h day, animals were exposed to 0.0025 mg/m^3 of HD vapor.
[h]Inada et al. 1978; International Agency for Research on Cancer 1975.
'Exposures were either in war situations or to workers in a mustard gas manufacturing plant during wartime production. The duration of exposure for these workers was years.
[j]Fell and Allsopp 1948.
'Berenblum and Shubik 1949.
[l]Dacre (personal communication) 1987.
[m]Dickey et al. 1949.
"Concentration of HD in culture fluid was as shown.
"Kirchner and Brcndcl 1983.
[p]Fahmy and Fahmy 1971.
[q]Auerbach and Robson 1947b.
[r]Very difficult to estimate dose received. A 1:10 mixture of mustard gas in cyclohcxanc was sprayed by an atomizer at IO-set intervals in an air stream that flowed at 2 L/min. This air stream flowed through the exposure chamber.
[s]Fahmy and Fahmy 1972.
'Brookes and Lawley 196 1.
"Host-mediated assay
"Reid and Walker 1969.
'"Host-mediated assay. Murinc leukemia L5 178Y cells were grown as ascitcs in mice.
'Duration of exposure was not given.
[y]Capizzi et al. 1973.
'Savage and Brcckon 198 1.
[aa]Ball and Roberts 1972.
[bb]Loveless 195 1.
[cc]Rommerein and Hackett 1986.
[dd]HD was tested at concentrations of 0.5 to 2.0 mg/kg body weight for gestational days 6 through 15 in rats; for rabbits. tested at concentrations of 0.4 to 0.8 mg/kg hody weight for gestational days 6 through 19.
[ee]Rozimarek et al. 1973.
[ff]Estimated total dose was 630 µg/kg body weight.

Tumors were also induced in mice (Table B.14 for amount administered and duration of exposure) by subcutaneous or intravenous injections (Heston 1950, 1953), although the relevance of these routes of administration to the anticipated human exposures that might result from accidental mustard exposures is questionable. Nevertheless, these studies do provide evidence of the carcinogenic potential of mustard.

Studies performed by U.S. Army investigators had as their goal the development of data regarding the effect of low-level mustard vapor exposures upon the carcinogenic activity of mustard (McNamara et al. 1975) (Table B.14). McNamara et al. (1975) reported on a series of experiments in which groups of animals (dogs, rabbits, guinea pigs, rats, and mice) were exposed either to 0.001 mg/m^3 of HD vapor continuously 5 d/week or to 0.1 mg/m^3 of HD vapor 6.5 h/d plus 0.0025 mg/m^3 for the remainder of the 24-h day (again, 5 d/week). The duration of exposure varied from 1 to 52 weeks. No acute toxicity signs were seen in any of the animals exposed, except in the case of dogs exposed to the higher dose, who developed eye effects. The carcinogenicity associated with exposure to these two concentrations of HD was examined. Few lesions that could be directly attributed to agent exposure were seen in most of the animal species studied. However, the period of observation (one year) for rabbits, guinea pigs, and dogs was not sufficient for a modern carcinogenicity study. The duration of the experiment was *not* sufficient for a definitive carcinogenesis study in rats and mice, either. Particularly in the case of very low-level exposures to presumed carcinogens, the period of observation should approach the animal's lifespan and experimental group sizes should be large enough for statistical treatment. Of 79 rats exposed at the lower concentration, five had chronic keratitis (inflammation of the cornea) possibly related to agent exposure. Of 79 rats exposed at the higher concentration, four had skin squamous cell carcinomas that were definitely attributed to HD, and five had squamous or basal cell carcinomas of the skin that were probably the result of agent exposure. All the animals with carcinomas had been exposed to HD for 12 months.

In a separate carcinogenicity study (McNamara et al. 1975), rats were exposed to air concentrations of 0.001 or 0.1 mg/m^3 HD (same conditions as previously stated) from 1 to 52 weeks and were sacrificed at certain times after the end of the exposure. The total tumor incidence (number of animals with tumors, expressed as a percentage of total animals in the experimental group) was 7% for controls, 10% for animals exposed to 0.001 mg/m^3 HD, and 44% for animals exposed to 0.1 mg/m^3 HD. Agent-related tumors were seen in animals exposed to 0.1 mg/m^3 for 12 weeks or longer; shorter times of exposure were not effective in producing agent-related tumors. The tumors (both malignant and benign) observed in the high-dose group were various skin lesions, consisting of carcinomas and other types of lesions involving various skin structures. The conclusion of this study was that exposures to 0.1 mg/m^3 of HD could be expected to produce a high frequency of skin cancer, given appropriate duration of exposure. The lower concentration (0.001 mg/m^3)

was not effective in producing tumors. Mice were also exposed to the two concentrations of HD for various time periods. No increase in tumor incidence in the exposed mouse groups was seen, and no agent-related tumors were observed.

It is difficult to conclude from these studies that the lower airborne concentration of mustard used (0.001 mg/m^3) is without carcinogenic activity and hence can serve as a "benchmark" dose from which noncarcinogenic exposure standards can be calculated for humans. The duration of exposure was not sufficient to firmly establish this dose as a noncarcinogenic dose. Two-year bioassays in rodents would provide a firmer scientific basis for calculating acceptable human exposure guidelines on the basis of the dose response in animals.

Two other animal studies of mustard carcinogenicity are of relevance to the problem at hand. Fell and Allsopp (1948) treated mice with repeated applications of mustard (five times per week, for as iong as 278 d) by applying liquid solutions to the skin of the back. The amount of mustard per application was either 2.5 or 12.5 μg, and animals in the high-dose group received an estimated maximum cumulative dose of 2.0 mg. The animals were kept for slightly more than a year from the start of the experiment before they were sacrificed. No evidence of gross or microscopic tumor formation was seen in any of the animals tested. The authors correctly noted that too few animals were tested to consider this a definitive carcinogenicity study, but they do note that their results agree with an earlier study (Berenblum 1929) in which similar repeated applications of small amounts of mustard on mouse skin did not produce tumors.

Berenblum and Shubik (1949) tested mustard in an initiation-promotion study and found it was not active as an initiator. In these types of studies, a suspect chemical is applied as a single dose or a few repeated doses onto mouse skin, and subsequently a standard promoting agent (i.e., a substance that promotes or facilities the production of a tumor, in this case croton oil) is applied over a long period of time. Appearance of tumors at the site of application demonstrates that the test compound has initiating activity. In a sense, the promoting agent hastens the inherent carcinogenic activity of the initiating agent. The doses of mustard the mice actually received in this study were not well defined, because the mustard was applied as a droplet on the end of a glass rod that had been dipped into a solution of 0.1% mustard in paraffin oil. This experiment is interesting, however, in that it showed that a low concentration of mustard was not an initiator of carcinogenesis, while various components of coal tar, applied at similar doses, were active in this assay system.

It is important, because of the carcinogenicity of mustard for humans under some exposure conditions, to consider the hazards associated with inadvertent mustard exposures under the disposal program for these agents. Concentrations of mustard to which the general population would be exposed during an unplanned release might be large enough to produce acute health effects. The most relevant exposure

experience with which to compare is that of World War I soldiers. In these veterans, there was some suggestion of increased respiratory cancer mortality, but the increased risk over the control population was not large and not statistically significant. We do not know what the battlefield doses were, what the extent of secondary respiratory infection in these individuals was, and what effect their subsequent life (e.g., the Great Depression with its attendant malnutrition, etc., must **have** been a major stressful event for some of these veterans) had on the eventual development of cancer. Limited information on smoking was available in **one** study; none was available in the others. The occupational exposures to mustard agent that have produced carcinogenic responses seem quite irrelevant to a single accidental release, because the doses the workers received were clearly large enough to provoke acute effects, and they experienced these exposures for periods of, typically, years. In the case of an unplanned release of mustard, then, it is possible that there would be no detectable effect upon cancer incidence in the exposed population. If an effect is seen, it might be a small increase in the incidence of respiratory tract *tumors* in the exposed group. Detectable increases of malignant skin tumors from single accidental exposures are not expected, as these occurred only among occupational populations.

If certain of the alternative methods of disposal of the chemical stockpile are selected (i.e., incineration at each site or regional centers), then the general public around these sites might be exposed to exceedingly low **(submicrogram/m^3)** concentrations of mustard agent that escape the process of complete combustion. The question that then arises is whether this minuscule concentration of mustard might, if inhaled over an extended period of time, produce an increased risk of cancer in humans. To rephrase this query, will the amount of mustard released from the incinerator stack (again, it must be emphasized that this is speculation--perhaps incineration conditions will be such that all mustard molecules, if not completely burned, will at least be significantly altered in chemical structure) be such that no detectable increase in risk of cancer would be produced in the exposed population, in comparison to the (background) incidence of cancer in human populations not so exposed? Evidence from occupational exposures to mustard cannot be directly compared to the incineration scenario, as the occupational exposures were literally many orders of magnitude (i.e., 10^5 to 10^6 times) greater than in the hypothetical case of unburned mustard releases from incinerators.

Various regulatory agencies, particularly EPA, have adopted the view that there is no threshold dose for carcinogenic chemicals (U.S. EPA 1986b). What this means is that exposure to any concentration of a carcinogenic chemical is assumed to carry some finite risk of cancer. Whether this has practical utility is another matter, because if the increased cancer incidence from exposure to low doses of a chemical is small, this increase cannot be detected in comparison with the normal "background" incidence for cancer. [Thus, the present U.S. *cancer*

incidence is approximately 25,000 out of 100,000 people at risk (Norman, 1987).] If exposure to some dose of some chemical produces a risk of 1 in 100,000, then in a population of 100,000 exposed persons, there could be one ***additional*** cancer case (i.e., 25,001, as a result of the chemical exposure). Statistical considerations make it extremely difficult or practically impossible to definitely attribute such small incremental increases to any given factor. As a practical solution, some regulatory agencies have considered taking the approach of a "permissible" risk [i.e., to regulate chemical carcinogens so that the additional risk from exposure is no more than some small number (e.g., 1 in 100,000 or 1 in 1,000,000)]. The designation of a "permissible concentration," then, for exposure to a chemical carcinogen, refers to a concentration to which an individual can be exposed and not experience an additional risk of cancer above the designated level (i in 100,000 or 1 in 1,000,000). By this approach a carcinogenic chemical is regulated from the standpoint of what concentration is judged, on the basis of scientific evidence, to produce an undetectably increased risk of cancer. This evidence commonly involves extrapolations of dose-response data from animal tumorigenesis studies using appropriate or selected mathematical models (U.S. EPA 1986a). Various other appropriate factors may also be included to modify dose estimates depending on the quality and extent of the data available. Using such procedures, the Department of Health and Human Services has considered the carcinogenic risk from exposure to mustard. The exposure limit to the general population proposed as being without health risks by Department of Health and Human Services is 1×10^{-4} mg/m^3, 72-h time-weighted average. This concentration has been judged not to produce a significantly increased risk of cancer. Humans are exposed to a variety of natural carcinogens (cosmic rays, natural radiation, UV irradiation from sunlight, carcinogenic chemicals produced by cooking foods, etc.) that are impossible to escape. The safest procedure then, is to attempt to restrain all exposures to chemical carcinogens to levels where the chemical carcinogen exposure does not provide an ***additional*** cancer risk, over and above the daily risk of simply living.

B.3.3.1.3.1 Relative potency of sulfur mustard

Although review committees at IARC have concluded that sulfur mustard is a human carcinogen (IARC 1975), they do not present an estimate of how potent a carcinogen H/HD might be. "Mustard gas" is grouped by IARC with arsenic, asbestos, diethylstibestrol, and vinyl chloride in a category that also includes industrial processes (such as nickel refining) considered carcinogenic to humans (IARC "Group 1," the most serious category; Saracci 1981). It would be useful to be able to compare the activity of mustard agent with that of other human carcinogens such as benzo(a)pyrene [B(a)P], cadmium, benzene, or *n*-nitrosodimethylamine (DMNA). Investigators at NIOSH, EPA, and ORNL have considered similar problems in attempting to screen and rank the

toxicity and carcinogenic potential of each of the thousands of compounds present at chemical waste sites (Jones, Walsh, and Zeighami 1985). One promising method evaluates the toxicity of a compound to be assayed (i.e., the "interviewing" compound) by comparing the results of similar biological tests between a well-characterized compound and the interviewing compound (i.e., dose of reference chemical in given test divided by dose of interviewing chemical in same test equals relative potency). The resulting ratios of the test results are considered a measure of relative potency for the interviewing chemical (Jones, Walsh, and Zeighami 1985; Jones et al. in press), and the sequential array of results provides a toxicological profile for the relatively untested interviewing compound. To provide an estimate of statistical uncertainty, the median value (the value that falls midway in the range, with 50% of the results above and 50% below) is reported, along with the upper and lower values of the central 50% (e.g., the interquartile range) of the array (Jones, Walsh, and Zeighami 1985; Jones et al. in press). Details of the approach are presented in Watson, Jones, and Griffin (in review).

As previously discussed in Sect. B.3.3.1.3, there are very few published studies that include specific, controlled examination of sulfur mustard carcinogenicity. The few that are available describe production of malignant tumors in populations of laboratory mice (Heston 1950, 1953) and rats (McNamara et al. 1975).

To perform a relative potency comparison, the experimental tumorigenic doses from Heston (1950, 1953) and McNamara et al. (1975) have been recently compared with doses found to produce tumors under similar experimental conditions in the same *or* closely related species of laboratory animals exposed to the occupational carcinogen B(a)P. Benzo(a)pyrene is a compound produced in the off-gases from coke ovens, tar distillation factories, street paving and roof pitching, creosote production, etc. (Gammage 1983). Toxicological interest in this compound revived in the last decade when startup of a domestic synfuel (from coal) industry was considered a viable alternative to imported oil. As a result, there are many toxicological data on B(a)P.

The resulting median carcinogenic potency value for sulfur mustard relative to B(a)P ("relative potency") is between 1 and 3, with a maximum interquartile range of 0.6 to 11.1. In other words, the limited animal data available indicate that sulfur mustard is likely to approximate B(a)P in its ability to produce malignant tumors in man (assuming that *mouse* and rat data are indicative of human response). The upper value of the range (i.e., 11.1) indicates that our current estimate of relative potency contains an approximate order of magnitude (factor of 10) uncertainty. Only more and better data could refine this estimate.

With a relative potency factor, it is possible to calculate estimates of excess lifetime cancer risk assuming the linear nonthreshold dose-response relationship of

$$\text{Risk} = (Q^*)(D),$$

where Risk is the additional individual lifetime risk of developing cancer based on a lifetime of continuous exposure to dose D of a compound with the potency factor Q^*. Reasonable assumptions of adult body weight, respiratory volume and incineration plant operation were made. The resulting estimate of individual excess lifetime cancer risk to a member of the general public at the site boundary of the Aberdeen Proving Ground (APG) during incinerator operation ranged between 8 x 10-8 and 2 $\times 10^{-7}$. [For calculation details, see Watson, Jones, and Griffin (in review).] Lifetime risk estimates $<10^{-6}$ are acceptable to most of the population and are not regulated by EPA or FDA. For comparison, the limit of regulatory acceptability for many drinking water pollutants is a lifetime risk of 1×10^{-5} (Munro and Travis 1986).

A recent examination of the last decade's federal regulatory decisions (in which some measure of risk was estimated prior to promulgation) demonstrates that protective action for carcinogen exposure was always invoked if the maximum estimated individual lifetime risk exceeded 4×10^{-3} (Travis et al. 1987). There has been only one instance (Food and Drug Administration control of DMNA in baby bottle manufacture, $R = 4 \times 10^{-8}$; FDA Docket No. 83D-0414) where protective action was taken to reduce estimated lifetime cancer risk at levels below 1×10^{-6} (Travis et al. 1987).

It is useful here to point out that EPA currently accepts maximum contaminant levels (MCLs) of several water pollutants that are estimated to generate excess lifetime cancer risks greater than that of mustard at the APG site boundary (assuming continuous consumption of 2 L/d). Examples are l,l-dichloroethylene ($R = 1 \times 10^{-4}$), toxaphene ($R = 2 \times 10^{-4}$), lindane ($R = 4 \times 10^{-5}$), and 1,2-dichloroethane ($R = 1 \times 10^{-5}$) (Munro and Travis 1986). In addition, the estimated individual lifetime cancer risk at the current MCL for arsenic is 2×10^{-2} (Munro and Travis 1986). These estimates are illustrative of many enforcement standards established prior to application of quantitative risk analysis methodologies. Similar findings have been identified upon systematic evaluation of Delaney Clause standards governing the pesticide content of processed foods (Norman 1987).

Additional perspective can be obtained by considering other industrial risks. In August of 1987, the National Council of Radiation Protection (NCRP) expanded the concept of comparable risk as a basis for occupational radiation exposure limits in that "the level of protection provided for radiation workers should, as far as possible, be comparable with that in other 'safe' industries" (NCRP 1987, p. 6). Examination of historical data maintained by the National Safety Council (NSC) defined safe industries "as those having an associated annual fatality accident rate of 1 or less per 10,000 workers, i.e., an average annual risk of

10^{-4}" (NCRP 1987, p. 8). For comparison, a decidedly unsafe industry such as mining or quarrying exhibits an annual accidental fatality rate of 6 x 10^{-4} in the United States (NSC 1985, as cited in NCRP 1987). There is still room for improvement, but the annual risks of accidental death associated with these safe and unsafe industries illustrate rates that have previously been considered acceptable (1 x 10^{-4} per year) or not (6 x 10^{-4} per year). The estimated excess risk (3.6 x 10^{-4}) of developing a malignancy (not necessarily fatal) after five years of continuous occupational exposure to currently allowable quantities of sulfur mustard (i.e., 3 x 10^{-3} mg/m^3 8-h TWA) is numerically comparable to that of accidental death risk in a nominally safe or moderately safe industry for a one-year work period. However, the human health consequences of the two risks (i.e., case of malignancy vs accidental death) are strikingly different.

B.3.3.1.4 Mutagenesis

Mustard agent has been found to produce various mutagenic and genetic effects in a wide variety of test organisms (see Table B.14). The list included in the table is not meant to be encyclopedic; many other studies could be cited. Our purpose is to present a summary of known biological activity. A review article summarizing many of the pertinent findings appeared in Nucation *Research* (Fox and Scott 1980); a few general conclusions from this article follow. Mustard is capable of reacting chemically with DNA (the carrier of genetic information for all organisms except some viruses), and DNA from a wide variety of species has been shown to be modified by the chemical. Mustard has also been found to be mutagenic in a wide variety of animal species, ranging from microorganisms to mammals. In fruit flies, mutations have included dominant lethal mutations and visible (phenotypic) mutations as well as recessive sex-linked, autosomal, and visible lethal mutations. Chromosomal aberrations following mustard exposure include deletions, inversions, duplications, and translocations. Mustard agent has also been demonstrated to produce various kinds of chromosomal structure damage in plant and animal cells; these are structural aberrations, including chromosome stickiness and chromosomal breakage. Fox and Scott (1980) summarize their conclusions by the statement, "There can be no doubt as to the mutagenicity of. . .sulfur mustard. . . ." Ongoing studies initiated by the U.S. Army Bioengineering Research and Development Laboratory (USABRDL), Health Effects Research Division, will add to the mustard agent mutagenesis data base. Work in progress includes mutagenesis in the Ames assay (the result was positive), the CHO (Chinese hamster ovary cells) assay, a sister chromatid exchange assay, and an alkaline elution assay (which measures DNA strand breakage) (Table B.14). Evidence of elevated sister chromatid exchanges (compared to controls) has been seen in lymphocytes taken from fishermen inadvertently exposed to mustard (Wulf et al. 1985) when they dredged up old, leaking mustard shells dumped in the North Sea after World War II.

The specific dose of mustard that individuals received cannot be quantified, but each person experienced acute toxicity (skin and eye effects), suggesting a rather high dose.

What health effects, if any, somatic cell mutations may produce in humans is unclear. A correlation between mutagenic activity of a chemical and that chemical's ability to produce cancer has been observed in a number of experimental assays. In the case of mustard agent, sufficient evidence already exists that it is carcinogenic, given appropriate circumstances of exposure. Therefore, the fact that it is a mutagen does not contribute materially to its assessment as a carcinogen. Possibly the most relevant health concern relating to its ability to damage genetic material is the potential for damage to reproductive cells (i.e., eggs or sperm). The next section will discuss pertinent evidence in regard to reproductive effects of mustard agent.

B.3.3.1.5 Reproductive effects and teratogenesis

In attempting to ascertain if humans exposed to mustard agent experience reproductive effects, attention has been focused upon groups of individuals occupationally exposed to mustard in poison gas factories. This population is an obvious one to study, as these individuals certainly were exposed to the highest dosages of agent for the longest duration of any group of people who have ever had contact with mustard. Furthermore, the exigencies of wartime resulted in the presence of male and female workers in the factories, although most of the workers were males. Analysis of data for both sexes might identify potential deleterious effects of mustard on either male or female reproductive performance.

Yamakido et al. (1985a) studied a group (325) of former workers from a World War II Japanese poison gas factory, their spouses (226), and their offspring (a total of 456) for possible genetic effects. The workers and their families were divided into three groups: (1) poison gas production workers who were exposed primarily to mustard and Lewisite and who were felt to have received the largest exposures; (2) other factory workers, not engaged in gas production, who probably received an exposure to medium concentrations [compared to groups (1) and (3)]; and (3) workers not directly involved with mustard or Lewisite (e.g., office workers, transportation, etc.) who were felt to have received the lowest exposures. In the first group, 18% were females, while there were 17% and 64% females in groups (2) and (3), respectively. The sex distribution in the offspring studied was about equally divided between male and female. A general health examination was carried out on all individuals under study, as well as some biochemical analyses of blood samples carried out on children of the workers. The purpose of these analyses was to look for evidence of specific gene mutation, presumably caused by mustard exposure of the parents. The general health examination did not reveal evidence of any diseases in the children that could be ascribed to genetic effects, or

any abnormality that was significantly different compared to the appropriate parental group. Biochemical analyses detected evidence of genetic variants (i.e., different from the majority of the population) in a small number of the children examined. Examination of their parents showed that the genetic variation was also in one or both of the parents, so that it could not be ascribed to a mutation induced *in* a parental germ cell by mustard exposure. Yamakido et al. (1985a) concluded that no evidence of mustard agent-induced mutations could be detected in their study group.

Lohs (1975) provides a brief summary of a study of reproductive effects in German poison gas factory workers (Hellmann 1970). These male workers were stated to be exposed to sulfur and nitrogen mustard under wartime conditions in World War II, although no details of possible exposure parameters, length of employment, manufacture of other poison gases, etc., are given. Evidence for dominant, sex-linked, lethal mutations was seen in descendants of 134 former poison gas factory workers. This was manifested as an increase in the sex ratio (i.e., greater than normal number of female births, which indicates higher than usual mortality of male fetuses) among the offspring of fathers employed in poison gas production. Impairment of various stages of spermatogenesis was also seen. It is difficult to know how much importance is to be attached to these observations. The confounding fact that the workers were apparently exposed to nitrogen mustard (a potent mutagen) as well as sulfur mustard makes interpretation of the results very uncertain, as far as an effect of sulfur mustard is concerned. The lack of exposure information lends further uncertainty.

Relatively few animal studies have directly investigated the potential of mustard gas to adversely affect the reproductive process. The potential reproductive-fetotoxic activity of low-level exposures to HD was studied in rats (McNamara et al. 1975). In one experimental protocol, male rats were exposed to atmospheres containing either 0.001 mg/m^3 or 0.1 mg/m^3 mustard (see carcinogenesis Sect. B.3.3.1.3 for details) for varying intervals (range from 1 *to* 52 weeks) and then bred to unexposed female rats. At the end of gestation, the percentage of dead fetuses was determined as an index of dominant lethal mutagenesis. The percentage of fetal death in the controls (12 month) and the ranges in the two exposed groups (animals were tested at 1, 2, 4, 8, 12, 24, 36, and 52 weeks) were as follows: 4.12% (control); 1.18 to 8.60% (low dose); 1.72 to 21.05% (high dose). The highest percentage of fetal deaths (21.05%) seen in the high-dose group occurred after 12 weeks of exposure. Other relatively high values *were* seen after 4 weeks (10.1%), 24 weeks (10.3%), and 52 weeks (12.5%). McNamara et al. (1975) concluded that there was no evidence for mutagenesis and that no differences between control and experimental groups were observed. Perhaps these conclusions were based on statistical analyses that were not explicitly stated; the elevated fetal mortality in the high-dose group suggests a possible connection between agent exposure and the fetal death effects.

Somewhat different conclusions about the same data set (or subset thereof) were reached by Rozimarek et al. (1973). They concluded that significant dominant lethal mutagenesis effects were observed in the high-dose (0.1 mg/m^3) group of animals. The dominant lethal mutation rate was stated to reach a maximum of 9.4% after 12 weeks of exposure and remained at this level for longer time periods tested. The reason for this difference in interpretation is unclear. The experimental results seem to at least suggest some effect of HD exposure on mammalian male fertility, although a dose-response relationship appears to be missing.

Dominant lethal effects have also been observed in fruit flies (Auerbach and Robson 1947b) (see Table B.14). The dosage necessary to produce this effect is not clearly stated in Auerbach and Robson (1947b), and the extrapolation between insects and mammalian species is not straightforward.

Further studies by McNamara et al. (1975) investigated the effect of low-level HD exposures on fetal toxicity and teratogenesis when pregnant female rats were directly exposed to HD. Animals were exposed to the same two HD air concentrations (i.e., 0.001 and 0.1 mg/m^3) during the first, second, or third weeks of gestation, or during the entire pregnancy. No change in fetal mortality or increase in gross fetal abnormalities was observed when the exposed group was compared to control groups.

The USABRDL extramural program on toxicology of chemical agents has several ongoing (or recently completed) studies that will be relevant to assessing the reproductive hazards of HD exposure (Table B.14). Teratology studies in rats and rabbits have been conducted (Rommerein and Hackett 1986). Pregnant rats were exposed to doses of HD of 0.5 to 2.0 mg/kg body weight by gastric intubation from day 6 to day 15 of gestation and were then sacrificed on day 20. There was some evidence of maternal toxicity at all dose levels. Some evidence for fetotoxicity as demonstrated by depression of fetal body weights and retardation of fetal growth occurred at the highest HD dose. Skeletal abnormalities were also seen in rat fetuses in this highest dose group.

Pregnant rabbits were treated (orally) with doses of 0.4 to 0.8 mg/kg body weight on days 6 through 19 of gestation. No significant effects on intrauterine death or fetal development were observed, although there was some evidence of maternal toxicity at doses of 0.8 mg/kg (diminished weight gain). At this dose, body weights of fetuses were also reduced from normal. Rommerein and Hackett (1986) concluded that HD was not teratogenic because fetal effects were only observed when the mothers exhibited toxic responses.

In addition to these teratogenesis studies, the U.S. Army is also sponsoring a three-generation rat reproduction study and a dominant lethal mutation study in rats, both of which are currently in progress (Table B.14). These studies, when completed, should add significantly to the data base regarding the reproductive toxicity of mustard.

In summary, evidence from both human and animal studies regarding the reproductive toxicity of mustard is equivocal. In some cases, effects are noted, but other studies are negative. The evidence from the German poison gas workers suggests an adverse effect, but it is not clear that this is due to sulfur mustard, as the workers were also exposed to nitrogen mustard (a compound significantly different in structure from the mustard agent with which we are concerned). If this positive result is of more importance than the negative findings in the Japanese worker population (and it is not clear this is so), the fact remains that these occupational exposures were almost certainly to high levels of poisonous agents for long durations and the workers were essentially without protective equipment. Any of the unplanned release scenarios that might occur during chemical stockpile disposal would produce only a "pulse" of possibly high exposure of very short duration. Exposure to agents during routine disposal operations, if it occurs at all, would only be to very low levels. From these considerations, we would conclude that reproductive effects from mustard exposure are unlikely for the general population during sulfur mustard disposal.

B.3.3.1.6 Other effects

A variety of other delayed effects, some rather ill-defined, are presented in the literature as the result of mustard poisoning (Weiss 1958, as cited in Lohs 1975). Among workers (presumably German, although not explicitly stated) in mustard gas factories before and during World War II, the following delayed effects were enumerated: periodontitis (inflammation of the gums), leading to tooth decay; osteoporosis (increased fragility of bones); premature aging; stomach disorders (decreased stomach acidity); liver injury (rare); and unspecified injuries of the central nervous system. The types of exposures (multiple poison agents? concentration? protective measures?) and durations that produced these effects are not stated and have not been documented elsewhere.

Lohs (1975) also notes that various psychopathological effects have been reported in former poison gas factory workers. The manifestations included impaired concentration, diminished libido and potency, and stomach and neuralgia-like complaints. How many, if any, of these rather vague manifestations are results of mustard exposure is unclear.

Skin lesions that are produced by mustard burns, even though they may heal, may exhibit certain evidences of permanent damage (Sollmann 1957a). There is often some alteration of skin pigmentation at the sites of skin damage. Furthermore, the affected areas of skin are unusually sensitive to subsequent mechanical injury, so that a mild contusion or abrasion may produce a blister.

McNamara et al. (1975) studied a number of physiological parameters in laboratory animals during their chronic exposure experiments. As mentioned previously, exposures were to air concentrations of 0.1 mg/m^3 or 0.001 mg/m^3 of HD for 5 d/week, up to 52 weeks. The animals were

monitored for toxic signs and effects resulting from the exposure. No overt toxic signs were detected in animals exposed to 0.001 mg/m^3 for up to 52 weeks; ocular effects (cornea1 opacity, chronic keratitis, vascularization, pigmentation, and granulation) were noted in dogs exposed for more than 16 weeks'to 0.1 mg/m^3, although no other animal species showed overt toxic signs on exposure to this concentration. Hematologic parameters (red blood cell count, hematocrit, white blood cell count, serum enzymes, etc.) in exposed rabbits and dogs were not significantly different from those of control animals, except for elevated concentrations of the blood enzyme serum glutamic-oxalic transaminase (SGOT) in dogs after 12 to 18 weeks of exposure to 0.1 mg/m^3 of HD. Elevated quantities of SGOT in blood serum are an indicator of tissue injury in the liver and/or heart. McNamara et al. (1975) also observed that the albumin/globulin ratios in serum of dogs exposed to either concentration of HD for 52 weeks was unchanged compared to control values.

Animals (rabbits, guinea pigs, and dogs) that had been exposed to the two vapor concentrations of HD for various times were also tested for sensitization to HD, particularly with regard to the skin, eyes, and respiratory tract. No evidence of sensitization was found (McNamara et al. 1975).

An antigen challenge in a rabbit exposed to 0.1 mg/m^3 of HD for one year produced an essentially normal response. Work in progress, sponsored by USABRDL, includes a 90-d subchronic test with mustard in rats. Results from this experiment may provide further information regarding other potential delayed or chronic effects.

B.3.3.2 Agents HT/T

No data specific to chronic toxicity of Agent HT were identified during preparation of this document. Because HT is a mixture of about 60% distilled mustard (HD) and up to 40% agent T (bis-[2-chloroethylthioethyl]ether, described in Table B.l and Sect. B.3.2.2), it is expected that chronic effects of HT would encompass those of both mustard agent (Sect. B.3.3.1) and T.

Agent T has been demonstrated to be highly mutagenic (Auerbach 1950), with an ability to produce sex-linked lethal mutations in *Drosophila melanogaster* (fruit fly) on an order comparable to that of mustard agent and some forms of radiation (X rays). Adult males were exposed to fine sprays of agent T, after which they were mated to untreated adult females. Differences' from normal hatch rates in the first (Fl) generation were considered evidence of lethal mutation in the chromosomes of the treated males' spermatozoa (Auerbach and Robson 1944) (Table B.14). Further experiments by Auerbach and Robson (1947a) suggest that agent T may have the capacity to induce chromosomal rearrangements in *D. melanogaster.* However, the corroborative data for this effect were considered suggestive rather than conclusive. Evidence of other chronic effects has not been published. Although these findings with a single

insect species cannot be considered an absolute indication of mutagenic activity in human systems, they represent reason for caution. We presume that agent HT possesses a capacity for chronic toxicity approximately equal to that of mustard agent.

B.3.3.2.1 Relative potency of HT

As stated in Sect. B.3.3.2, there are data describing mutagenic effects of Agent T but no data on carcinogenicity of T or HT. In the absence of any additional information, we have assumed that the relative carcinogenic potency of HT approximates that of sulfur mustard, which comprises 60% of the HT formulation. See Sect. B.3.3.1.3.1 for a description of HD potency.

B.3.3.3 Agent L (Levisite)

Very little evaluation of potential chronic effects of Lewisite exposure in isolation has been performed; what exists is based on anecdotal, wartime exposure evidence and 30- to 40-year-old studies of chromosomal changes in one insect species and one plant species.

The ability of Lewisite to produce sex-linked lethal mutations and chromosomal rearrangements among the first (F1) generation of exposed adult *D. melanogaster* (fruit fly) was tested in the Pharmacology Department of the University of Edinburgh during World War II and reported in the late 1940s and early 1950s (Auerbach and Robson 1946, 1947a; Auerbach 1950). *Vicia faba* L. (broad bean) root tip meristems were exposed to aqueous solutions of Lewisite at the Royal Cancer Hospital in London and then microscopically examined for changes in normal cellular division (Loveless 1951). In both sets of experiments, Lewisite did not exhibit any mutagenic properties.

Historically, the few cases of long-term follow-up of human exposure indicate that carcinogenicity may be an effect of acute exposures. A former infantryman in the German Occupation Army of France during World War II received an accidental exposure to liquid Lewisite on the skin of his lower right leg in 1940 (Krause and Grussendorf 1978). Intense pain ensued and a blistered lesion formed; the wound never healed. In 1948, the ulcerated lesion was diagnosed as malignant, surgically removed, and later treated with X rays. By 1978, the ulcerated area involved the inner third of the victim's lower leg and was histologically diagnosed as Bowen's disease, an intraepidermal squamous cell carcinoma. At the time of last report (Krause and Grussendorf 1978), the patient was 77 years old, in otherwise good health, and receiving palliative treatment (i.e., relieving symptoms but not curative) 38 years after a single exposure. No metastasis was noted.

Other human evidence is less direct. From 1929 through 1945, the Japanese Army operated a poison gas factory on Okuno-jima, an island in the Inland Sea and south of the Hiroshima Prefecture (Wada et al. 1962a, 1962b; Nishimoto et al. 1983, 1986). This factory produced Lewisite, mustard gas, hydrocyanic acid, diphenylcyanarsine (sneezing gas),

chloracetophenone (MACE@ or tear gas), and phosgene for use in the invasion and occupation of mainland China. At peak capacity, this facility produced 450 tons/month of mustard gas; 50 tons/month each of Lewisite, diphenylcyanarsine, and hydrocyanic acid; and 25 tons/month of chloracetophenone. Approximately 1000 individuals were employed in the manufacture of these war materials during the period of maximum production (1937-1942) (Wada et al. 1962b). As discussed in Sect. B.3.3.1.3, protective clothing, ventilation, monitoring, and sanitation were poor or nonexistent, and many workers suffered severe exposures. Study of worker death certificates through 1962 revealed a high incidence of respiratory tract cancer (14%) and digestive tract cancer (9.6%) (Wada et al. 1962a). The remaining deaths were largely caused (39.7%) by respiratory disease (tuberculous or other pulmonary infections) thought to be secondary to epithelial damage induced by vesicant gas inhalation.

Later follow-up divided the worker population into exposure groups based on job title (Nishimoto et al. 1983). Examination of death certificates and autopsy reports through 1979 found that "Those. . .who were engaged in manufacture of yperite [mustard] and Lewisite gases had a high mortality due to diseases of the respiratory tract, particularly malignant tumors" (Nishimoto et al. 1983). Smoking had been previously ruled out as a factor. Retired workers are also observed to exhibit impaired immunity (Nishimoto et al. 1986). Because each worker was exposed to both agents, it is not possible to implicate Lewisite as opposed to mustard agent as a carcinogen in this study. Furthermore, there are no quantitative estimates of dose or exposure rates, although they must have been high under the wartime conditions described. However, there are sufficient data to implicate Lewisite as a carcinogen at elevated, sublethal exposures that cause blistering and pain.

Teratogenic properties and reproductive toxicity of high-level Lewisite exposure are suspected but have not been substantiated. Zoological examination of Okuno-jima Island in 1947 identified "abnormalities" in the fauna (Wada et al. 1962b). These abnormalities were not further described. The active arsenical group in Lewisite is thought to alter metabolic function and thereby affect developing offspring (Sasser 1985a,b). Several experiments designed to test this theory have been recently completed by the U.S. Army Biomedical Engineering Research and Development Laboratory (USABRDL) (Dacre 1987). Examination of maternal and fetal effects was accomplished by exposing pregnant rats and rabbits to Lewisite by gastric intubation at a daily dose range of 0 to 1.5 mg/kg for rats and 0 to 0.6 mg/kg for rabbits. Results were negative, indicating that Lewisite is not likely to be a mammalian teratogen. Work in progress will examine fetal abnormalities in three generations of rats. Future plans include an evaluation of dominant lethal mutations in offspring of Lewisite-treated rats (Dacre 1987).

Recent work to determine in vitro mutagenicity with cell lines at USABRDL has obtained negative results for the *Ames S. typhimurium* assay (Dacre 1987). Experiments are in progress testing Lewisite in the CHO forward mutation assay. Planned work includes (1) an alkaline elution assay to measure DNA strand breaks and (2) an in vitro sister-chromatid exchange assay. These studies are not scheduled for completion in time to be included in the current analysis.

B.3.3.3.1 Relative potency of Lewisite

In an extensive literature search for this document, no Lewisite data were found that could be used in an estimate of relative carcinogenic potency. All available data from laboratory animal studies of Lewisite describe acute toxicity (LD_{50} and LD_{Lo}). Thus, an estimate of relative acute potency was performed by means of the methodology described in Sect. B.3.3.1.3.1. Lewisite is approximately four times as toxic as B(a)P in producing acute lethality in laboratory rats, rabbits and guinea pigs; the interquartile range is 0.4 to 27.6.

B.3.4 ANTIDOTES AND PERSONAL DECONTAMINATION

B-3.4.1 Agents H/HD

There is no specific antidote known for mustard poisoning. (Lohs 1975; Dahl et al. 1985; Sollmann 1957b). As explained in Sect. B.3.2 (Mode of Action), mustard is highly reactive chemically and forms a chemical bond with many biological molecules. Once this bond is formed, the reaction for all practical purposes. is irreversible. Attempts to remove the mustard residue from biological molecules have been unsuccessful, except with drastic chemical procedures that are in themselves injurious (Sollmann 1957a; Grant 1986). U.S. Army manuals that discuss therapy for various chemical warfare agents correctly emphasize that instantaneous removal of mustard from body surfaces is the best form of protection (U.S. Army 1974). One way to accomplish this is by. washing with soap and water. A study of the use of various household products to remove mustard from guinea pig skin (van Hooidonk et al. 1983) indicated that most such products (tissue paper, flour, talcum powder, washing abrasive, salad oil, etc.) were effective in reducing skin damage if applied within 4 min of contamination. The most effective treatment was sprinkling flour on the contaminated skin, followed by removal of the flour with wet tissue paper. Interestingly, wet tissue paper alone simply spread the mustard over a larger skin surface, suggesting that washing with water needs to be combined with detergent use or some other solubilizer or adsorber of mustard.

Attempts at therapy of mustard poisoning in humans have generally been aimed at rapid decontamination and symptomatic (i.e., treatment of mustard-induced symptoms) therapy (Vojvodic et al. 1985). Some attention has been focused on use of agents that might react with mustard and thereby act as a mustard "scavenger" (Vojvodic et al. 1985). Work on

rats has indicated that in animals dosed with three times the LD_{50} dose of mustard and injected with various drugs 30 min later, the best protective effects (decreased lethality, fewer pathological organ changes, less loss of body weight) were obtained with a combination of sodium thiosulfate, vitamin E, and dexamethasone (Vojvodic et al. 1985). The dosages of drugs used were: 3000 mg/kg of sodium thiosulfate, 8 mg/kg of dexamethasone, and 20 mg/kg of vitamin E. The sodium thiosulfate was thought to act as a mustard scavenger (i.e., simply reacted with mustard); vitamin E is considered an antioxidant and free-radical scavenger. Dexamethasone is a corticosteroid, an antiinflammatory agent. Among rabbits receiving a dermal dose of mustard, use of steroid (cortisone) injections 15 min after treatment, or cortisone injections plus hydrocortisone ointment at the site of mustard application, produced a beneficial effect on skin swelling and decreased the thickness of the skin lesion produced by mustard (Vogt et al. 1984). This therapy did not hasten the rate of healing, however. The preceding suggests that symptomatic therapy could have beneficial effects and that mustard scavengers may also be useful, but that such treatment must be initiated relatively quickly after exposure. It is to be emphasized that the previously described treatments are, to our knowledge, untested for their efficacy in humans.

U.S. Army documents (U.S. Army 1974, 1975) that discuss these chemical warfare agents also provide information regarding decontamination and first aid in the case of battlefield exposures. In the case of H and HD, emphasis is placed upon immediate decontamination following exposure. Copious flushing with water is recommended for eye contamination. Fuller's earth powder (which is used to adsorb liquid agent droplets) and chloramine powder (which reacts chemically with mustard) are effective skin decontaminants and are supplied to military personnel in field kits. (Trapp 1985; Sollmann 1957a). The chloramine reaction alters mustard's chemical structure so that it no longer reacts with biological molecules. A protective ointment, known as "M5" and supplied to field personnel, contains chloramide S-330, which can function both as a decontaminant and a protective barrier (Koslow 1987). The chloramines, along with effective equipment decontaminants such as hypochlorite, operate by producing "free" chlorine, which chemically reacts with mustard (Trapp 1985; Sollmann 1957a). Such chemicals could be useful in reducing skin damage in accidentally exposed populations (if applied quickly enough after exposure), but these chemicals cannot be used in the eye, because of their own deleterious properties, and they cannot be used to alleviate damage to the respiratory tract.

B.3.4.2 Agent HT

Treatment and decontamination procedures described for mustard agent in Sect. B-3.4.1. are effective for HT exposure as well. Because HT is considered to be more toxic than HD, swift removal of the agent and treatment of symptoms is imperative. Decontamination of personnel can be

accomplished by washing with copious quantities of either water (HT is poorly soluble in aqueous solutions) or soap and water. Chlorine bleach solutions will neutralize HT, as will the reagents contained in the Army medical "M13" kits and M5 ointment.

B.3.4.3 Agent L (Lewisite)

Historically, the antidote of choice for treating Lewisite and arsenical poisoning in general is the synthetic dithiol 2,3-dimercaptopropanol developed by British toxicologists in the years immediately prior to World War II (Oehme 1979). Known as British antiLewisite (BAL), this compound prevents skin vesication from Lewisite exposure after an untreated time lapse of as much as 1 h. Intravenous or intramuscular administration of BAL will also provide protection from topical and systemic Lewisite effects by binding arsenic to the BAL molecule, thus permitting transport of the arsenic complex to the excretory system and removal through the urine. Nevertheless, therapeutic administration must be closely monitored because BAL possesses considerable toxic properties of its own and will interfere with cellular respiration (Oehme 1979). Recommended treatment and precautions are summarized in Table B.15.

In the absence of BAL for treating exposed skin, decontamination may be accomplished by washing with water, soap and water, solutions of chlorine bleach or soda, and use of absorbent powders such as Fuller's earth (U.S. Army 1974; Trapp 1985). As previously discussed, exposed eyes should be immediately flushed with large amounts of water or weak solutions of soda or detergent (U.S. Army 1974). Following World War II, BAL use was expanded to include civilian medicine, where it has been an effective treatment in cases of lead, mercury, copper, and arsenic poisoning (Klaasen 1980, as cited in Aposhian et al. 1984). In the late 1950s, scientists in the Soviet Union and the People's Republic of China reported success with new, water-soluble BAL analogues in treating occupational victims of heavy-metal poisoning and as a preventive occupational therapy among workers in heavy metals industries (Petrunkin 1956; Klimova 1958; and Liang *et* al. 1957, all as cited in Aposhian 1984). These new BAL analogues are the sodium salt of 2,3-dimercapto-1-propanesulfonic acid (DMPS) and meso-dimercaptosuccinic acid (DMSA). In addition to being water soluble and exhibiting less toxicity than BAL, they possess the added advantage of oral administration (not a recommended route for BAL treatment). Recent work in the United States demonstrates that both DMPS and DMSA protect rabbits receiving oral or subcutaneous doses of Lewisite from developing lethal systemic effects (Aposhian et al. 1984). Even when treatment was delayed for 90 min after Lewisite exposure, all exposed animals survived; between 83 and 100% of exposed animals receiving no treatment died (Aposhian et al. 1984).

Preliminary experiments on the skin of swine indicate that pretreatment with DMPS or DMSA in a thin collagen film may also be protective against the blistering reaction of Lewisite application

Table B.15. Treatment protocol for British ● tilewisitc (BAL) (2,3-dimercapto-1-propanol) (dimercaprol)

Effectiveness	Dose	Precautions
Lifesaving in acute poisoning of **arsenicals** (except amine) and **solutions** of organic Hg compounds. Also for chronic As or **Au** poisoning. Action by displacing the metal from its combination with SH groups of enzyme proteins.'	I.M. injection only (10% preparation in peanut oil). Dose of **5 mg/kg** repeated every 3 h for 24 h (produces no significant cumulative toxicity due to rapid breakdown of **BAL)** but leaves granulomas.[a] I.M. into buttocks in dosage of 0.5 **mL/25** lb body weight up to maximum of 4.0 **mL**. Repeat in 4, 8, 12 **h**. For severe cases, interval shortened to **h**.[b]	Consistent objective response is rise in systolic and diastolic blood pressure **plus tachycardia (rapid** heartbeat). 1. Nausea and sometimes vomiting. 2. Headache. 3. **Burning** sensation of lips, mouth. 4. Feeling of constriction in throat, chest. hands. 5. Conjunctivitis, tearing, salivation. 6. Hand tingling. 7. Burning sensation in penis. 8. Forehead and hands sweating. 9. Abdominal pain. IO. Tremors. 11. **Lower back** pain. 12. Anxiety, weakness, and restlessness. 13. Tachycardia (rapid heartbeat) and elevated arterial **blood** pressure. 14. Persistent fever in children. 15. Occasional painful sterile **abcesses** at injection **sites**. 16. Coma and convulsions at high dose (in children, this occurs at 10, 25. and 40.5 **mg/kg) (recovery** prompt).' Toxicity Rating = 4 **(?),** VERY TOXIC [Probable oral lethal dose = **50–500 mg/kg** or I tsp to 1 oz for ***150*** lb (70 kg) person].' To treat BAL overdose: Symptoms usually subside in 30-90 min. **I.M.** use of **1:100** solution epinephrine HCI (0.1 to 0.5 ml) or oral epintphrine sulfate (25-50 **mg**).[a]

[a]**Gosselin,** R. E., R. P. Smith, H. C. Hodge, and J. E. **Braddock** 1984, ***Clinical Toxicology of*** Commercial ***Products*** **(5th** Ed.), Williams and Wilkins (Baltimore/London), pp. III-50 to **III-52**.

[b]**Sidell,** F. R. 1986. ***General*** Guidelines **for *Therapy of Acetylcholinesterase Intoxication,*** Medical Research Division, Biomedical Laboratory, **Edgewood** Arsenal, Md. (Handout).

(Aposhian et al. 1984). This technique would have utility for emergency workers and rescue teams who may need to enter contaminated areas or handle/treat contaminated individuals. Further studies on swine skin have demonstrated the effectiveness of another Soviet compound (Mercaptid; 3-(*p*-tolythio)-1,2-propanethiol) in dealing with postexposure treatment of Lewisite-contaminated skin; topical application of Mercaptid "diminished the severity of the burns" as observed in a nonquantitative visual assay of exposed swine skin (Aposhian et al. 1984). Note that swine skin is considered by many investigators to be reasonably similar to human skin in its reaction to certain surface-active chemicals.

Dosage and treatment protocols for antidote use of DMPS and DMSA use in Lewisite poisoning have not yet been developed because these compounds are considered "orphan drugs." However, DMPS and DMSA have been used to treat several isolated cases of arsenical and methylmercury poisoning (Lenz et al. 1981; Clarkson et al. 1981; all as cited in Aposhian et al. 1984) and to reduce the body burden of lead among smelter workers (Friedham et al. 1978, as cited in Aposhian et al. 1984). Treatment for the arsenic-poisoned individual mentioned was 300 mg DMSA by mouth every 6 h for three days; the victim recovered.

An additional Lewisite antidote that has not seen much use since the end of World War II is 2,3-dimercaptopropanol glucoside (BAL-INTRAV), another BAL analogue developed for intravenous and subcutaneous treatment of topical Lewisite exposure and systemic effects (Danielli et al. 1947). BAL-INTRAV also demonstrates a lower mammalian toxicity than BAL (LD_{50} in rabbits for BAL-INTRAV is 5000 mg/kg as compared to 50 mg/kg for BAL) (Danielli et al. 1947). A further advantage of BAL-INTRAV therapy is the relatively lengthy time delay that can occur between exposure and treatment with no observed change in successful outcome, at least for laboratory rabbits. With a delay of 4 h, treated rabbits exhibited lesions at the site of Lewisite application, "but made an uneventful recovery" (Danielli et al. 1947). A delay of 6.5-h permitted eventual full recovery after one to three days of poor appetite and illness. A delay of 12 h was fatal to all exposed rabbits (Danielli et al. 1947). The above values were observed in test populations of adult rabbits; younger animals (with thinner skins) were more sensitive to Lewisite exposure. At the same dose as the adults (1.5 mg/kg), none of the young rabbits survived a 6.5-h delay in treatment with BAL-INTRAV; all survived a 4.0 h delay.

Although wartime conditions prevented larger populations of experimental animals from being tested, the investigations performed by Danielli and his colleagues (1947) demonstrate that BAL-INTRAV has merit in Lewisite therapy, that time before treatment should be kept at a minimum, and that young individuals should be treated sooner after Lewisite contamination than adults.

B.3.5 D2PC CODE CONSIDERATIONS

A few comments will be made about the appropriateness of the various effect-level dosages delineated in the D2PC code, which was used to calculate the extent of atmospheric dispersal of various agents over the surrounding area in case of an unplanned release (see Sects. 3 and 4 of PEIS). [It is important to note that the D2PC code was developed for military applications (i.e., for use in estimating results of a gas attack on the battlefield). Application to accidents during disposal operations is far afield from its original intended purpose.] The no-death dosage for H and HD in the code is 100 mg-min/m^3. This value is only 5 to 7% of the lowest dose reported to be lethal to humans (Table B.13). Using some assumptions (i.e., an adult inhalation rate of 15 L/min, all of inhaled agent reaches the bloodstream, equivalent toxic effect of intravenous HD in animals and humans), it is possible to calculate an "intravenous" HD dose resulting from this no-death air concentration, and compare it with intravenous LD_{50} doses in animals. Making this comparison, the no-deaths dosage in the D2PC code is only 2 to 11% of the LD_{50} dose for a 70-kg adult. The actual percentage would be even lower, because not all of the inhalation dose would reach the bloodstream. For an adult, therefore, the no-death dosage in the D2PC code seems reasonable. For infants and small children, this no-death dosage may not be protective. Based upon physiological factors, the appropriate downscaling of the infant no-death dosage might be 20% of the adult dosage (V. Houk, Center for Environmental Health, CDC, DHHS, Atlanta, Ga., letter communication to Brig. Gen. D. Nydam, OPMCM, Aberdeen Proving Ground, Aberdeen, Md., June 12, 1987). Lethalities among infants/children *might* result from exposure to the no-deaths dosage presently in the D2PC code, but the code is not intended to supply extreme toxicological accuracy. In the context of this assessment, it is mainly useful for consistent comparison of various accident scenarios and disposal alternatives.

The 1%-death dose level (i.e., dose-rate at which 1% of the exposed population is expected to die) in the D2PC code is 150 mg-min/m^3 for H and HD, or 1.5 times as large as the no-deaths value. This is the same value cited as the 1% lethality distance in the Safety Regulations Bulletins (U.S. Army 1979) for Chemical Agents H, HD, and HT. The toxicological data and documents that delineate the calculation of this value were not available. The 1% lethality value appears to be protective (i.e., is a lower dose rate than the actual dose rate that would cause 1% lethality).

The LCt_{50} value used in making population lethality estimates for HD (based upon atmospheric dispersion models of an accidental agent release) is 1500 mg-min/m^3. This value is cited in U.S. Army (1974) and McNamara et al. (1975) as the median lethal dose and is apparently an estimate. Using the same type of assumptions as previously listed, one can calculate the intravenous dose received from exposure to this airborne concentration. This total dose, assuming complete absorption

into the blood stream, is within the range of LD_{50} (intravenous) doses for various animal species. For healthy adults, this dose rate seems to be a reasonable estimate. Use of a 20% correction factor, as in the case of no-death estimates, would be inappropriate for 1% and 50% lethality estimates. These latter dosages are used to estimate the response of a population; because the average member of a population will be more like the healthy adult than like an infant, downscaling the 1% and 50% lethality doses would greatly overestimate fatalities.

B.4 AGENT ADDITIVES (STABILIZERS) AND DECONTAMINANT TOXICITY

There are two stabilizers used in nerve agent formulations (tributylamine for GB and diisopropyl-carbodiimide for GB and VX). GB and VX preparations also contain the contaminants diisopropyl methylphosphonate (in GB) and diethyl dimethylpyrophosphonate (in VX). In addition, in this section we include a discussion of the constituents of the DS-2 decontaminant preparation (diethylenetriamine and 2-methoxyethanol). An additional decontaminant preparation that can be used is STB (supertropical bleach). No specific toxicological information on STB was found.

Very little relevant toxicological information was found in relation to the GB and VX stabilizers and contaminants. General population exposure to these compounds would only occur if there were an unplanned release of either GB or VX. In such an accident scenario, the overwhelming concern would be protection from the toxicological activity of the nerve agents themselves. The routes of exposure relevant to such an accidental exposure would be inhalation of vapors and skin deposition of agent. Obviously, stabilizers and contaminants in GB and VX preparations could also be inhaled or deposited on the skin along with the agent. There is almost no information about the toxicity of the contaminants and stabilizers by these routes of exposure (Table B.16). No relevant toxicological information for diisopropylcarbodiimide or diethyl dimethylpyrophosphonate could be found. In the case of the GB contaminant, diisopropyl methylphosphonate, a comparison of the oral toxicities of this compound with GB itself indicates that diisopropyl methylphosphonate is approximately 0.1% as toxic as GB. The GB stabilizer tributylamine is approximately 2% as ***toxic*** as GB, when tested on rabbit skin, while oral exposures indicate it is about 0.2 to 0.4% as toxic as GB. No relevant biological information was found in regard to chronic effects of any of the stabilizers ***or*** contaminants (Table B.17). It appears, on the basis of current information, that the contaminants and stabilizers in the GB and VX preparation will have an insignificant effect on the overall hazard of human exposure to accidental releases of these agents during disposal operations.

General population exposure to the constituents of the DS-2 decontaminant solution is only envisioned to take place after areas contaminated by accidental release of chemical agent have been treated

Table 8.16. **Acute** ● fleets of ● gcat ● dditiw **(stabilizers) and decontaminants**

Product identification	LD_{50}	LC_{50}	$LD_{Lo}(LC_{Lo})$	Other effects
GB stabilizer				
Tributylamine	**Rat, oral, 540 mg/kg**[a] *Mouse, oral,* **114 mg/kg**[c] **Rabbit. oral. 615 mg/kg**[c] Guinea pig, oral, **350 mg/kg**[c] **Rabbit, skin, 250 mg/kg**[a]		**Rat. inhalation. 75 ppm/4 h**[a]	**Human-central nervous** system stimulation. **skin irritation, sensitization**[b] **Human—eye. skin, and** respiratory irritant'
GB contaminant				
Diisopropyl methylphosphonate	**Rat. oral, 826 mg/kg**[e] **Mouse, oral. 1041 mg/kg**[e]			
GB and VX stabilizer				
Diisopropyylcarbodiimide[f]				
VX contaminant				
Diethyldimethylpyrophonsophonate[f]				
DS-2 **decontaminant** constituents				
Diethylene triamine	**Rat. oral, 1080 mg/kg**[g] **Rabbit, skin 1090 mg/kg/**[h] Guinea pig, skin, 162 **mg/kg**[i]			**Rabbit skin-severe irritation, 10 mg/24 h**' **Rabbit eye-severe irritation. 750 μg**[h] Potent primary skin **irritant**[j]
2-Methoxyethanol	**Rat. oral, 2460 mg/kg**[k] **Rabbit, oral. 890 mg/kg**[o] **Rabbit. skin. 1280 mg/kg**[l] Guinea pig, oral, 950 **mg/kg**[k]	Rat. Inhalation. **1500 ppm/7 h**' Mouse. inhalation. **1480 ppm/7 h**[p]	Human, oral, *3380* **mg/kg**[m]	Human--convulsions. 25 ppm" **Rabbit-mild skin irritation, 483 mg/24 h**[q] **Rabbit--lye irritation. 97 mg**[q] **Guinea pig-mild eye irritation. 10 μg**[r] **Human—CNS** symptoms and **encephalopathy**[s]

[a] *Toxicol ond Appl Phorm 28: 313.* 1974.
[b] Windholz et al. 1983. p. 1375
[c] Gigiena i Sanitarya42: 36, 1977.
[d] *NFPA Fire Protect Guide Hozord Matls* 1978, pp. 49-286
[e] Plaa and Duncan 1978
[f] No relevant biological data were found
[g] Hine et al. 1958
[h] *J Indusr. Hyg Toxicol.* 1949.
[i] Grant 1986, p. 336.
[j] *J. Indusr Hyg. Toxicol. 1941.*
[k] *Raw Material Doro Hondbook* 194 1.
[l] *J. Indust. Hyg. Toxicol. 1946.*
[m] *J. Indusr. Hyg Toxicol* 1938.
[o] *AMA Arch Indust Hlth* 1956
[p] *J Indust. Hyg Toxicol.* 1943.
[q] *Toxicol. Appl. Phormocol.* 197 1.
[r] *J Phormocy ond Phormocol 1959*
[s] Gosselin 1984. pp. 111-174–75.

Table B.17. **Chronic** effects of **agent additives** (stabilizers) **and decontaminants**

Product identification	Genetic effects	Reproductive effects
GB stabilizer		
Tributylamine[a]		
GB contaminant		
Diisopropyl methylphosphonatc"		
GB and VX stabilizer		
Diisopropylcarbodiimidc'		
VX contaminant		
Dicthyldimothylpyrophosphonate"		
Decontaminant (DS-2) **constitutents**		
Diethylenetriame	Mutagenicity: **negative**[b]	
2-Methyoxyethanol	Mutation: positive'	**Teratogenesis**[c], **fetotoxicity**[d]
	Sperm morphology: positive'	Male reproductive toxicity'

[a] No relevant biological data were found.

[b] Ames assay results were negative (Murphey-Corb, Kong, and Murray 1983; Anon. 1982).

'Dominant lethal mutations were produced in rats; oral doses were 500 mg/kg (Anderson et al. 1984).

[c] The effect was observed in mice and rats; oral doses were 500 mg/kg (Anderson et al. 1984).

The effect was observed in pregnant rats exposed by inhalation to 50 ppm (or greater) on days 7-l 5 of gestation (Hardin 1983); in pregnant rabbits exposed by inhalation to 50 ppm 6 h/d on days 6-18 of gestation (Hartley et al. 1984).

[d] The effect was observed in pregnant rats exposed by inhalation to 200 ppm on days 7-15 of gestation (Hardin 1983). Pregnant rats exposed by inhalation to 25 ppm 7 h/d on days 7–13 of gestation also showed toxic effects on the newborn (Nelson et al. 1983).

Testicular toxicity was observed in male rats and mice exposed by inhalation to 100 ppm (or greater) for 9 d (Hardin 1983); paternal effects (unspecified) observed in rabbits exposed to 300 ppm, 6 h/d, 5 d/week for 65 d; paternal effects (unspecified) observed in rats exposed to 30 ppm. 6 h/d for 65 d (Miller et al. 1983).

with decontaminant. The actual decontamination procedure is assumed to take place by properly trained personnel wearing protective clothing. Following a suitable time interval for decontamination to take place and for the area to be declared safe for reentry, the general public would again be allowed into the area. The only general population exposures envisioned under this scenario would be to residues of decontaminant solution left on buildings, trees, etc. The routes of exposure might be either skin exposure (i.e., touching a residue-contaminated surface) or inhalation of vapors from evaporating residue. There is little relevant acute toxicological information for diethylenetriamine (Table B.16). It apparently has some activity as an eye and skin irritant. The lethal dose (LD_{50}) of diethylenetriamine by skin absorption is much larger than lethal doses of nerve agents for the same animal species (i.e., approximately 300-fold larger for GB and GA and 40,000-fold larger for VX). No information on the chronic toxicity of diethylenetriamine was available, other than it was negative in a mutagenicity test (Table B.17). More toxicological information could be found in regard to 2-methoxyethanol, the other DS-2 constituent. 2-Methoxyethanol is used as a solvent in a large number of products under a variety of trade names such as methyl cellosolve. It has skin and eye irritant properties and can cause central nervous system disturbances at large enough doses. Its acute toxicity (LC_{50}) by inhalation in rats and mice is approximately 4700 $mg/m^3/7$ h (converted from data in Table B.16). These lethal doses are much larger than the lethal doses of nerve agents themselves (i.e., approximately 200-fold larger for 2-methoxyethanol compared to GA, and approximately 5500-fold larger for 2-methoxyethanol compared to VX). Comparison of rabbit skin lethality data shows similar orders of magnitude difference between 2-methoxyethanol and the nerve agents. Some chronic toxicology data were also found for 2-methoxyethanol (Table B.17). Evidence for various reproductive effects and mutagenic activity has been seen. Existing data relevant to inhalation refer only to reproductive effects and the necessary doses are in the range of tens to hundreds of parts per million. Data indicate that 2-methoxyethanol can produce a variety of reproductive effects, including effects on the male reproductive system and effects on offspring.

In conclusion, the agents themselves pose much more of a health threat than the constituents of the DS-2 solution. Nevertheless, because of the potential reproductive effects from 2-•ethoxyethanol exposures, it would be prudent to ensure that 2-methoxyethanol residues are washed from DS-2-treated surfaces and structures before the return of the general public to a decontaminated area.

B.5 BREAKDOWN PRODUCT TOXICITY

In addition to the chemical agents themselves, it is also important to consider the toxic effects of breakdown products that could be derived from them. Breakdown products addressed include: hydrolysis products that would be generated by introduction of the agent into

water, decontamination products produced in the decontamination of an agent, combustion products that could be produced in the event of an accidental fire or explosion, and expected products of soil microbial activity (Table B.18). Because very little is known about possible products of photodecomposition, there is no discussion of this process in this document.

Note that, at normal incineration operating temperatures, complete combustion is expected. All products of complete incinerator combustion are listed in footnote e of Table B.18.

B.5.1 HYDROLYSIS PRODUCTS

Accidental introduction of chemical agents into surface water or groundwater would generate large numbers of breakdown products via interaction with water (i.e., by hydrolysis). Hydrolysis products are smaller and less complex molecules than the parent agent. The hydrolysis products almost always will have lost the potent toxicity for which the parent agent was designed. Nonetheless, some of these products are toxic, and this toxicity must be considered in assessing the risks involved in disposing of these agents.

B.5.1.1 Formation

The rate and extent to which an agent will be degraded by hydrolysis depends on several factors. Most important is the water solubility of the parent agent (see Table B.l for physical property description of all agents). This ranges from the slightly soluble agents, Lewisite and HD, to the very soluble agent, GB. Generally, the warmer the water the greater is the agent solubility; VX is the exception. For example, GA is about 1.4 times more soluble at 25°C than at 20°C; whereas VX is 2.5 times more soluble at 15°C than at 25°C (U.S. Army 1974, pp. 132 and 177). Of course, the larger the body of water and, thus, the dilution factor, the greater the opportunity to dissolve all the agent. Because mixing increases the rate of solubilization, water turbulence is also an important factor.

Once the agent is solubilized, hydrolysis depends on the chemical structure of the agent and the pH and temperature of the water. Higher temperature and pH generally promote hydrolysis. Multiple products can be formed from a single agent, and pH influences which of these products will predominate (e.g., under acidic conditions Lewisite hydrolyzes to chlorovinyl-arsenous oxide and hydrochloric acid, whereas under basic conditions the chlorovinyl arsenous oxide is further hydrolyzed to acetylene and arsenite).

For purposes of this report, all those hydrolysis products that might reasonably be expected to form and to persist for significant periods of time under the pH and temperature conditions existing in surface water and groundwater sources have been considered. We realize that variations in these parameters between water sources can alter the

Table B.18. Potential products of agent hydrolysis, decontamination, combustion, microbial degradation, and photodecomposition.

Process/agent	Product name	Product formula	CAS No.	Reference
Tabun (GA)	Ethylphosphoryl cyanidatc	$C_3H_6NPO_3$	Not found	*a*
	Dimethylamine	C_2H_7N	124-40-3	*a*
	Hydrogen cyanide	HCN	74-90-8	*a*
	Ethyl, *N*-*N*-dimethyl-amido phosphoric acid	$C_4H_{12}NPO_3$	Not found	*a*
Sarin (GB)	Isopropyl methyl-phosphonic acid	$C_4H_{11}O_3P$	1832-54-8	*b*
	Methyl phosphonic acid	CH_5O_3P	993-13-s	*c*
	Hydrogen fluoride (Hydrofluoric acid)	HF	7664-39-3	*b*
	Isopropyl alcohol	C_3H_8O	67-63-0	*b*
GB Stabilizer DIPC (Diiso-propyl carbo-diimide)	1,3 Diisopropylurea	$C_7H_{16}N_2O$	4128-37-4	*b*
VX	Ethyl methyl phosphonic acid	$C_3H_9PO_3$	1832-53-7	*b*
	Ethyl methyl phos-phonothioic acid	$C_3H_9SPO_2$	18005-40-8	*b*
	Diisopropylamino-ethanol	$C_8H_{19}ON$	96-80-o	*b*
	Bis (2-diisopropyl-aminoethyl) sulfide	$C_{16}H_{36}N_2S$	Not found	*b*
	Hydrolysis (continued)			
VX (continued)	Dicthyl methyl phosphonate	$C_5H_{13}O_3P$	683-08-9	
	Ethanol (ethyl alcohol)	C_2H_5OH	64-17-5	
	Ethyl hydrogen methyl phosphonate		Not found	
	Methyl phosphonic acid	CH_5O_3P	993-13-s	
	S-(Diisopropylamino ethyl) methyl-phosphonothioatc	$C_9H_{22}NSPO_2$	73207-98-4	
VX Stabilizer DIPC (Diiso-propylcarbo-diimide)	1,3 Diisopropyl urea	$C_7H_{16}N_2O$	4128-37-4	
Distilled mustard (HD)	Bis-2(bis(2-hydroxy ethyl)-sulfonium ethyl) sulfide dichloride	$C_{12}H_{28}S_3Cl_2$	64036-79-9	
	Hydrogen chloride (Hydrochloric acid)	HCI	7647-01-o	
	Thiodiglycol	$C_4H_{10}SO_4$	111-48-8	
Lewisite (L)	Chlorovinyl arsenous oxide	C_2H_2ClAsO	3088-37-7	

Table B.18 (continued)

Process/agent	Product name	Product formula	CAS No.	Reference
	Hydrogen chloride (Hydrochloric acid)	HCI	7647-01-o	*b*
	Acetylene	C_2H_2	774-86-2	*b*
	Sodium arsenite	$NaAsO_2$[d]	7784-46-5	*b*
	Deconraminafion			
GA	Cyanogen chloride	CClN	506-77-4	*b*
GB	Isopropyl methyl-phosphonic acid	$C_4H_{11}O_3P$	1832-54-8	*b*
	Sodium fluoride	NaF	7681-49-4	*b*
	Calcium fluoride	CaF_2	7789-75-5	*b*
GB Stabilizer DIPC	N-chloroisopropylaminc	C_3H_8NCl	26245-56-7	*b*
	1,3-Diisopropylurea	$C_7H_{10}N_2O$	4128-37-4	*b*
	Chloroform	$CHCl_3$	67-66-3	*b*
	Acetic acid	$C_2H_4O_2$	64-19-7	*b*
	Ammonia	NH_3	7664-41-7	*b*
GB Stabilizer TBA (Tributylamine)	Dibutylchloraminc	$C_8H_{18}NCl$	999-33-1	*b*
	N-butanoic acid	$C_4H_8O_2$	107-92-6	*b*
v x	N-chlorodiisopropylaminc	$C_6H_{14}ClN$	24948-81-0	*b*
	Chloroform	$CHCL_3$	67-66-3	*b*
	Acetic acid	CH_3CO_2H	64-19-7	*b*
	N-chloroisopropylaminc	C_3H_8NCl	26245-56-7	*b*
	ethyl methyl phosphonic acid	$C_3H_9PO_3$	1832-537	*b*
VX Stabilizer DIPC	N-chloroisopropylaminc	C_3H_8NCl	26245-56-1	*b*
	1,3 diisopropylurea	$C_7H_{16}N_2O$	4128-37-4	*b*
	Chloroform	$CHCl_3$	67-66-3	*b*
	Acetic acid	$C_2H_4O_2$	64-19-1	*b*
	Ammonia	NH_3	7664-41-7	*b*
Distilled mustard (HD)	2-Chloroethyl vinyl sulfide	C_4H_7SCL	81142-02-1	*b*
	Divinyl sulfide	C_4H_6S	627-51-0	*b*
	Mustard sulfoxidc	$C_4H_8SOCl_2$	5819-08-9	*b*
	Mustard sulfone	$C_4H_8SO_2Cl_2$	471-03-4	*b*
	2-Hydroxycthyl vinyl sulfide	C_4H_8SO	3090-56-o	*b*
	Chloroform	$CHCl_3$	67-66-3	*b*
	Thiodyglycol	$C_4H_{10}SO_4$	1 1 1-48-8	*b*
	Carbon dioxide	CO_2	124-38-9	*b*
	Calcium sulfate	$CaSO_4$	7778-18-g	*b*
	Calcium chloride	$CaCl_2$	10043-52-4	*b*
	Combustion'			
Tabun (GA)	Hydrogen cyanide	HCN	74-90-8	*a*
	Phosphorous pentoxide	P_2O_5	1314-56-3	*f*
	Nitrogen dioxide	NO_2	10102-44-0	*e*

Table B.18 (continued)

Process/agent	Product name	Product formula	CAS No.	Reference
Sarin (GB)	Phosphoric acid	H_3PO_4	7664-38-2	a
	Hydrogen fluoride (Hydrofluoric acid)	HF	7664-39-3	a
	Phosphorous pentoxide	P_2O_5	1314-56-3	f
VX	Phosphoric acid	H_3PO_4	7664-38-2	a
	Sulfuric acid	H_2SO_4	7664-93-9	a
	Phosphorous pentoxide	P_2O_5	1314-56-3	g
	Nitrogen	N_2	7727-37-9	a
	Nitrogen dioxide	NO_2	10102-44-O	e
Distilled mustard (HD)	Sulfur	S	7704-34-9	g
	Sulfur dioxide	SO_2	7446-09-s	g
	Hydrogen sulfide	H_2S	7783-06-4	g
	Ethanethiol	C_2H_5SH	75-08-1	g
	Diethyl disulfide	$(C_2H_5)_2S_2$	110-81-6	g
	1,4-Dithiane	$(C_2H_4)_2S_2$	505-29-3	g
	Methylene chloride	CH_2Cl_2	75-09-2	g
	Chloroform	$CHCl_3$	67-66-3	g
	Ethyl chloride	C_2H_5Cl	75-00-3	g
	1,2-dichloroethane	$C_2H_4Cl_2$	107-06-2	b
	1,1,1-trichloroethane	$C_2H_3Cl_3$	71-55-6	g
	1,1,2-trichloroethane	$C_2H_3CL_3$	79-00-5	g
	1,1,1,2-tetrachloroethane	$C_2H_2Cl_4$	630-20-6	g
	1,1,2,2-tetrachloroethane	$C_2H_2CL_4$	79-34-5	g
	Hydrogen chloride	HCI	7647-01-o	g
Lewisite (L)	Acetylene	C_2H_2	74-86-2	g
	Acetylene monochloride	C_2HCl	593-63-5	g
	Arsenic trichloride	$AsCl_3$	7784-34-l	g
	Arsenic trioxidc	As_2O_3	1327-53-3	g
	Chlorine	Cl_2	7782-50-5	g
	Methyl chloride	CH_3Cl	74-87-3	g
	Vinyl chloride	C_2H_3Cl	75-01-4	g
	Acetylene dichloride	$C_2H_2Cl_2$	540-59-O	g
		C_6H_5		g
	Arsenic oxychloride	AsOCl	Not found	g
	Chlorovinyl arsenous oxide	C_2H_2ClAsO	3088-37-7	a
	Microbial Activity			
VX	Ethyl methyl phosphonic acid	$C_3H_9PO_3$	1832-537	h
	Methyl phosphonic acid	CH_5PO_3	993-13-5	h
	Diisopropyl ethyl mercapto amine	$C_8H_{19}SN$	5842-07-9	h

Table B.18 (continued)

Process/agent	Product name	Product formula	CAS No.	Reference
		Photodecompostion		
Influence of photodecomposition for the agents under consideration is unclear. Knowledge is limited and no details are available.				

"U.S. Army 1974.
[b]Small 1984.
[c]Mill and Gould 1979.
'Hydrolysis product probably a mixture of sodium arsenite ($NaAsO_2$) and the unstable intermediate, sodium metaarscnate (Na_3AsO_3).
'Combustion products listing includes many compounds that would be released only by incomplete combustion, such as during a fire or explosion. Under normal temperature and residence times for incinerator operation, the following combustion products are expected:

GA: CO_2, H_2O, NO_2, P_2O_5
GB: CO_2, H_2O, HF, P_2O_5
VX: CO_2, H_2O, NO_2, SO_2, P_2O_5
HD: SO_2, CO_2, H_2O, HCl
L: $AsCl_3$, CO_2, H_2O, Cl_2, As_2O.

[f]Flamm 1986.
[g]Brooks and Parker 1979.
[h]Kaaijk and Frijlink 1977; Verwej and Boter 1976; as cited in Trapp 1985.
'Trapp 1985.

rate and extent to which a particular hydrolysis product will be produced.

B.5.1.2 Hydrolysis Product Description (see Table B.18)

Products expected from hydrolysis of GA include ethylphosphorylcyanidate, dimethylamine, hydrogen cyanide, and ethyl-*N*,*N*-dimethylamidophosphoric acid (U.S. Army 1974, pp. 132 and 177). No information on the stability or persistence of the first and last products is readily available.

GB hydrolyzes to isopropylmethylphosphonic acid, methylphosphonic acid, hydrofluoric acid, and isopropyl alcohol (Small 1984, p. 8; Mill and Gould 1979). The latter three components represent the ultimate products of hydrolysis. Hydrolysis of diisopropylcarbodiimide (DIPC), the GB stabilizer, produces 1,3-diisopropylurea (Small 1984, p. 9).

Because of its complex structure, several hydrolysis products may be derived from VX (U.S. Army 1974, p. 178; Small 1984, pp. 7 and 8). These include ethylmethylphosphonic acid, ethylmethylphosphonothioic acid, diisopropylamino-ethanol, bis(2-diisopropylaminoethyl)sulfide, diethyl-methylphosphonate (although this compound is listed on page 178 in U.S. Army 1974 as a product of VX hydrolysis, we do not believe that it is a legitimate product under conditions likely to occur in nature), ethanol, ethyl hydrogen methyl phosphonate, methyl phosphonic acid, and *S*-(diisopropylaminoethyl)-methylphosphonothioate. VX, like GB, also contains DIPC as a stabilizer with 1,3-diisopropyl urea as its hydrolysis product.

Hydrolysis of HD produces thiodiglycol, bis-2-[bis(2-hydroxyethyl)-sulfonium ethyl]sulfide dichloride (HD-2TDG), and hydrochloric acid (Small 1984, pp. 6 and 7; U.S. Army 1974, p. 47). Thiodigylcol and HCl are the products of complete hydrolysis, although under the conditions that these hydrolyses would be occurring, any acids formed would be converted to their salt forms. HD-2TDG is a rather persistent intermediate that forms as HD dissolves.

Products of Lewisite hydrolysis include chlorovinyl arsenous oxide, hydrochloric acid, acetylene, and sodium arsenite. Chlorovinyl arsenous oxide is produced under acidic conditions and would undergo further hydrolysis to acetylene and arsenite.

B.5.1.3 Chronic and Acute Toxicity

Primary routes of potential exposure to the hydrolysis products of chemical agents are oral and percutaneous absorption through the use of contaminated waters for drinking, bathing, and recreational activities. In considering the potential toxic effects of agent hydrolysis products, it should be kept in mind that, except near the primary contamination site, extensive dilution will have occurred before humans are exposed.

No toxicity data were found for the GA hydrolysis products ethylphosphoryl-cyanidate and ethyl-N,N-dimethylamidophosphoric acid. In the three chronic toxicity tests for which data were available,

dimethylamine is negative. Large oral doses of dimethylamine are required for lethality in rodents and rabbits. The acute toxic effects of hydrogen cyanide are well known and are indicated by the low LD_{50} and LD_{Lo} values listed. Where the comparison can be made (e.g., LD_{50} in humans via percutaneous exposure), HCN (LD_{50} = 100 mg/kg) is approximately 20% as toxic as GA (LD_{50} = 14 to 21 mg/kg) (U.S. Army 1974, pp. 81 and 120). Weakness, fatigue, and partial paralysis have been attributed to chronic exposure to nonlethal quantities of HCN. The potential for chronic exposure to HCN is low because it quickly oxidizes to the nontoxic cyanate in the environment.

The GB hydrolysis product isopropylmethylphosphonate is negative for mutagenicity in the S. *typhimurium* test (Mecler 1981, pp. 31-40). It is only mildly irritating to the skin, and large doses are required to produce lethality in rodents (Mecler 1981, pp. 6-20). Methylphosphonate is reported (Mill and Gould 1979) to be nontoxic (presumably in reference to acute toxicity); no chronic toxicity data were found for this compound. Fluoride is not a hazard except at relatively high doses; the beneficial effects of low levels on preventing tooth decay are well known. Chronic effects of isopropanol (rubbing alcohol) are relatively minor, and large amounts are required to produce acute toxic effects. Diisopropylurea is a hydrolysis product of the GB stabilizer DIPC. No biological effects data were found for this compound.

For most of the hydrolysis products of VX no biological effects data were found. These include ethylmethylphosphonic acid, ethylmethyl phosphonothioic acid, bis(2-diisopropylaminoethyl)disulfide, diethylmethylphosphonate, ethyl-hydrogenmethylphosphonate, and *S*-(diisopropylaminoethyl)methyl phosphothionate. Diisopropylaminoethanol is lethal in rats (oral) and rabbits (percutaneous) at rather high doses. Data for chronic toxicity of this compound were not found. Carcinogenicity, chromosomal damage, and reproductive effects of ethanol are well known and have been reported. Ethanol (common "alcohol") can be lethal; however, both the chronic and acute effects of ethanol are manifested only at very high doses. Methylphosphonic acid is reportedly nontoxic (Mill and Gould 1979).

At high doses, the HD hydrolysis product thiodiglycol is a skin irritant and is lethal in guinea pigs. No chronic effects data were found for thiodiglycol. An LD_{Lo} of 250 mg/kg in the rat has been reported for bis-2[bis-(2-hydroxyethyl)-sulfonium ethyl]sulfide dichloride. No other biological effects data were found. The chronic and acute effects reported for hydrochloric acid result from its acidity. Under the conditions of hydrolysis, neutralization of this acidity is expected.

The hydrolysis product of Lewisite, chlorovinylarsenous oxide, retains vesicant properties similar to those of Lewisite (see Sect. B.3.2.4). No other biological effects data for this compound were found. Hydrochloric acid is discussed under HD. Toxicity data for acetylene are all pertinent to inhalation exposure and will be discussed under Combustion Products. Arsenite is lethal when ingested in small

quantities. Perhaps of more concern, in the context of this document, are the chronic effects, including cancer in humans, that are produced by low levels of this compound.

B.S.2 DECONTAMINATION PRODUCTS

In the event of an unplanned release of chemical warfare agents, affected terrain, buildings, and objects will require decontamination. The Army has developed various methods of physical and chemical decontamination. It is expected that these would be the methods of choice whether the incident took place on or off an Army facility. Chemical safety data sheets prepared by the U.S. Army (1974) contain recommended decontamination procedures for each agent. Trapp (1985) provides extenslve information about many decontaminants and their applications but no information on by-products. Small (1984) presents information about the expected by-products of chemical decontamination for two decontaminants, supertropical bleach (STB; a mixture of chlorinated limes with low water content), and decontamination solution No. 2 (DS-2; a mixture of 2% caustic soda, 28% 2-methoxyethanol, and 70% diethylenetriamine) (Trapp 1985). The present review considers only the by-products for which we have information, although other decontaminants yielding unknown by-products may be used. Small (1984) cautions that the compounds identified as breakdown products in his report have been found in laboratory studies under controlled conditions of temperature, pH, and stoichiometry (defined chemical properties and composition) that may not simulate field conditions. Three N-chloroalkylamines, *N*-chlorodiisopropylamine, dibutylchloramine, and N-chloroisopropylamine (DPCA, DBCA, and IPCA) are included as reasonable products to expect from studies of amine chlorination, but Small (1984) limited the list of other postulated reaction products to better-known compounds.

B.5.2.1 Formation

The rate and extent to which chemical warfare agents will be broken down by chemical decontaminants depends, like hydrolysis, on many factors. One is whether a large excess of decontaminant reaches all the agent. Small (1984) lists by-products of both complete and incomplete reaction with decontaminant. Small also considers two other scenarios that have been eliminated as nonrelevant for purposes of the present analysis. In one, decontaminant does not reach agent-contaminated soil but later alters soil pH, causing. the agent to undergo alkaline hydrolysis; in the other, no chemical decontamination occurs and the agent undergoes reaction in the unaltered soil environment.

Temperature will have a major effect on the rate of decontamination reactions; elevated temperatures favor more rapid reactions (Trapp 1985). Temperature dependence is particularly significant for reaction of STB slurries and other aqueous decontaminants. Under winter conditions, slow (if any) reactions would be expected; heating of STB solutions would be necessary. DS-2 solution is effective over a wider

temperature range (Trapp 1985, Table 11), although it too is affected by temperature (Trapp 1985, Table 12). DS-2 is considered a "universal decontaminant" from a chemical standpoint, but cost considerations restrict its use as a decontaminant to equipment (U.S. Army 1974; Trapp 1985).

STB, which contains calcium hypochlorite as an active species, does not dissolve readily and forms unstable solutions in water (Trapp 1985). Its oxidizing ability is dependent on the available active chlorine content. It is not active against mustards below 10°C (50°F) but otherwise is effective against V and G agents and mustards (Trapp 1985). STB slurry is most active against chemical warfare agents in the midalkaline range; this can be obtained by spraying the solution with hot exhaust gases, which also raises the temperature. These beneficial effects are somewhat offset by carbon monoxide from exhaust gases that competes for the available active chlorine (Trapp 1985).

B.5.2.2 Decontamination Product Description

Table B.18 lists information on expected decontamination products. Almost no information is available on expected products of GA decontamination because Small (1984) did not consider GA. Chemical safety sheets prepared by the U.S. Army (1974) mention that cyanogen chloride may be formed by the use of bleach slurry for decontamination of GA-tainted equipment.

GB decontamination by-products are the same whether from STB or DS-2 decontamination and whether reactions are complete or not. The products include isopropyl methylphosphonic acid and sodium or calcium fluoride (Small 1984). The decontamination products of GB stabilizer diisopropyl carbodiimide (DIPC) differ, depending on the decontaminant used. With DS-2, DIPC forms 1,3-diisopropylurea. With STB, the products may include N-chloroisopropylamine, 1,3-diisopropylurea, chloroform, acetic acid, and ammonia (Small 1984). The other GB stabilizer, TBA, is unreactive in the presence of DS-2; with STB, it produces dibutylchloramine and *N*-butanoic acid (Small 1984).

VX by-products also differ according to the decontaminant used. DS-2 produces ethyl methylphosphonic acid. Possible decomposition products from STB reaction with VX include ethylmethylphosphonic acid, chloroform, acetic acid, ammonia, N-chlorodiisopropylamine, and *N*-chloroisopropylamine (Small 1984). The by-products of the DIPC stabilizer were listed in the preceding paragraph in conjunction with GB decontamination.

HD decontamination may yield divinyl sulfide, 2-chloroethyl vinyl sulfide, thiodiglycol, and 2-hydroxyethyl vinyl sulfide when accomplished with DS-2 (Small 1984). STB decontamination of HD may produce only carbon dioxide, calcium sulfate, and calcium chloride if the reaction is complete; however, incomplete reaction may result in the formation of mustard sulfoxide, mustard sulfone, and chloroform (Small 1984).

B.5.2.3 Chronic and Acute Toxicity

Tables B.19 and B.20 describe the toxicity of decontamination products. The primary routes of potential exposure to decontamination products of chemical warfare agents are percutaneous (dermal) and inhalation through direct contact with decontaminated surfaces or inhalation of volatilized substances from those surfaces. The potential for exposure to these substances, at least for the general population, will be affected by their stability because decontamination would presumably take place after population evacuation. Direct contact could involve either no dilution of the substance or substantial dilution by rain or postdecontamination washing of surfaces. The consequences of dermal exposure would depend in part on the potential for absorption through the skin. The degree of inhalation exposure would depend on the potential dilution in ambient air and this, in turn, would be greatly affected by whether the substance were applied indoors or outdoors.

Cyanogen chloride, the one identified GA by-product, is a highly toxic military poison gas (Windholz et al. 1983, p. 385). Its toxic effects are very similar to hydrogen cyanide.

GB by-products include one organic acid, isopropyl methylphosphonic acid (toxicity is discussed in Sect. B.5.1.3 on hydrolysis), and sodium or calcium fluoride. Sodium fluoride is very toxic at high doses, but calcium fluoride is not (Windholz et al. 1983, pp. 230, 1235). Both are used in the fluoridation of water and are considered quite safe at the low concentrations used for fluoridation (1.4 to 2.4 mg/L) (U.S. EPA 1985). No indication of chronic toxicity is given by Windholz et al. (1983, pp. 230, 1235) for either fluoride.

GB stabilizer DIPC yields a variety of by-products. Of these, the toxicity of 1,3-diisopropylurea is discussed in the hydrolysis section (Sect. B.5.1.3). No relevant biological data were found for *N*-chloroisopropylamine. Chloroform is a suspect human carcinogen of rather modest potency for lifetime oral exposure (IARC 1982; U.S. EPA 1986b; Jorgenson et al. 1985). It may be mutagenic in vivo although most in vitro tests have given negative results (Davidson et al. 1982). It is fetotoxic and teratogenic in rats and produces sperm abnormalities in mice (Davidson et al. 1982). It is a skin and eye irritant (Grant 1986, p. 213; Smyth et al. 1962); inhalation can cause CNS depression and anesthesia at higher concentrations and death from cardiac arrest (IARC 1979a; Torkelson and Rowe 1981, p. 3463; Doull, Klaassen, and Amdur 1980, p. 471). Liver and kidney damage can ensue after acute exposure (IARC 1979a; Torkelson and Rowe 1981, p.3463). Acetic acid is also a skin and eye irritant and can cause severe cornea1 injury in concentrated solution (NIOSH/OSHA 1981, p. 2). Bronchopneumonia and lung edema may appear in humans after acute overexposure (Sittig 1981, pp. 20-21). No chronic toxic effects would be expected from acetic acid exposure. Ammonia is an eye and skin irritant at low concentrations and can cause severe injuries and death to humans from inhalation at high concentrations (Wands 1981, pp. 3048-52).

Table B.19. Acute effects of exposure to products of agent hydrolysis, decontamination, comb&ion, and microbial degradation

Process / product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
		GA hydrolysis'	
Ethyl phosphorylcyanidate[2] ($C_3H_6NPO_3$)			
Dimethylaminc (C_2H_7N)	Rat. oral. 698 mg/kg[3] Mouse, oral. 316 mg/kg[3] Rabbit, oral. 240 mg/kg[3] Guinea pig, oral. 240 mg/kg[3]		Rabbit eye irritant, 50 mg (5 min)'
Hydrogen cyanide (HCN)	Human, skin, 100 mg/kg[5] Mouse, oral, 3.1 mg/kg[7]	Human, oral, 570 μg/kg[6] Dog, oral, 4 mg/kg[8] Rabbit, oral, 4 mg/kg[8] Pig, oral, 2 mg/kg[9]	Respiratory poison'
Ethyl, *N,N*-dimethyl amido phosphoric acid' ($C_4H_{12}NPO_3$)			
		GB hydrolysis'	
Isopropylmethyl phosphonic acid ($C_4H_{11}O_3P$)	Rat, oral, male, 7650 mg/kg[10] Rat, oral, female, 6070 mg/kg[10] Mouse, oral, male, 5620 mg/kg[10] Mouse. oral, female, 6550 mg/kg[10]		Negative, rabbit, eye irritant'" Rabbit, mild skin irritant 2 mg/kg[10]
Methyl phosphonic acid (CH_5O_3P)			Nontoxic"
Hydrogen fluoride (HF)		Human, oral, 0.9 g[12]	
Isopropyl alcohol (C_3H_8O)	Rat, oral 5045 mg/kg[13] Mouse. oral, 3600 mg/kg[13]	Human, oral, 5272 mg/kg[14] Human, oral, 3570 mg/kg[15]	
1,3 Diisopropylurea[2,16] ($C_7H_{16}N_2O$)			

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
		VX *hydrolysis'*	
Ethyl methyl phosphonic acid' ($C_3H_9PO_3$)			
Ethyl methyl phosphonothioic acid' ($C_3H_9SPO_2$)			
Diisopropyl amino ethanol ($C_8H_{19}ON$)	Rat, oral, 1070 mg/kg[17] Rabbit, skin, 450 mg/kg[18]		
Bis (2diisopropyl aminoethyl) disulfide[2] ($C_{16}H_{36}N_2S$)			
Diethyl methyl phosphonate[2]			
Ethanol (C_2H_5OH)	Rat, oral, 10,260 mg/kg[19]	Human, child. oral, 200 mg/kg[20] Human, adult, oral, 1400 mg/kg[22]	CNS depressant."
Ethyl hydrogen methyl phospbonate'			
Methyl phosphonic acid (CH_5O_3P)			Nontoxic"
S-(diisopropylaminc ethyl) methyl phosphothionate' ($C_9H_{22}NSPO_2$)			
1,3 Diisopropylurea[2,16] ($C_7H_{16}N_2O$)			

Table 8.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
		H D hydrolysis	
Bis-2[bis(2-hydrolyethyl) sulfonium ethyl] sulfide dichloride ($C_{12}H_{28}S_3Cl_2$)		Rat, oral, 250 mg/kg[23]	
Hydrochloric acid (HCl)	Rabbit, oral, 900 mg/kg[24]	Human, oral, 202 mg/kg[25]	Rabbit, eye irritant, 100 mg[26]
Thiodiglycol ($C_4H_{10}SO_4$)	Guinea pig. oral, 3960 mg/kg[27]		Rabbit, skin irritant, 500 mg[28]
		Lewisite hydrolysis'	
Chlorovinyl arsenous oxide (C_2H_2CLAsO)			Irritant[29]
Hydrochloric acid[30] (HCl)			
Acetylene (C_2H_2)[31]			
Sodium arsenite ($NaAsO_2$)	Rat, oral, 41 mg/kg[32] Rat, skin, 150 mg/kg[34]	Human, child, oral, 2 mg/kg[33]	
		GA decontamination[35]	
Cyanogen chloride[36] (CClN)		Mouse, inhalation, 780 mg/m^3/7.5 min[37] Dog, inhalation, 800 mg/m^3/7.5 min[37] Rabbit, inhalation, 3000 ppm/2 min[37]	Human, eye, severe, 100 mg/m^3/2 min[38]
		GB decontamination"	
Isopropyl methyl phosphonic acid ($C_4H_{11}O_3P$)			Rabbit, eye irritant" Rabbit, skin irritant, 2 mg/kg (mild)"'

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Sodium fluoride[39] (NaF)			Used for fluoridation of water[39]
Calcium fluoride[40] (CaF_2)			Used for fluoridation of water[40]
N-chloroiso-propylamine[41,2] (C_3H_8NCl)			
1,3-Diisopropylurea[41,2] ($C_7H_{16}N_2O$)			
Acetic acid" ($C_2H_4O_2$)	Rabbit, skin, 1060 mg/kg[42] Guinea pig, inhalation, 5000 ppm/1 h[45] Mouse, inhalation, 5620 ppm/1 h[47] Mouse, inhalation, 5000 ppm/1 h[45]	Human, inhalation 816 ppm/3 min[43] Rat, inhalation, 16,000 ppm/4 h[46]	Human, skin irritant (mild) SO mg/24 h[44] Rabbit, skin irritant (mild) 50 mg/24 h" Rabbit, skin irritant (severe) 325 mg/24 h[42] Human, eye pain and cornea injury, 4–10% solution" Human, extreme eye irritation, >25 ppm[48] Human, conjunctivitis, (eye) > IO ppm[48] Rabbit, eye, open, severe irritation, 50 μg[49] Human, lung, broncho-pneumonia and lung edema may follow acute overexposure[50]
Ammonia (NH_3)[41]	Cat and rabbit, inhalation, 7000 mg/m³/1 h[51] Rat, inhalation, 7.6 mg/L/2 h[51] Mouse, inhalation, 3.3 mg/L/2 h[51]	Human, inhalation, 35 mg/m³/10 min[51]	Severe injuries and death result from exposure at high concentrations"

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Chloroform $(CHCl_3)$[41]		Human, inhalation, 10 ppm/y[52] Human, inhalation, 1000 mg/m³/7 min[54] Human, inhalation, 5000 mg/m³/7 min[55] Dog, inhalation, 100 g/m³[57] Cat, inhalation, 35,000 mg/m³/4 h[55] Guinea pig, inhalation, 20.000 ppm/2 h[58] Rabbit, inhalation, 59 g/m³[57] Rat, inhalation, 8000 ppm/4 h[54] Mouse, inhalation, 28 g/m³[56]	Human. eye. pain, irritation, and corneal injury[53] Anesthesia, CNS depression, death from cardiac or respiratory arrest[56] Human, liver, and kidney damage[56] Rabbit, skin, irritant IO mg/24 h[54] Rabbit, eye, irritant, 148 mg[59]
Carbon dioxide (CO_2)			Nontoxic; constituent of earth's atmosphere
Dibutylchloramine[60,2] $(C_8H_{18}NCl)$			
N-butanoic acid[60] $(C_4H_8O_2)$	Rabbit, skin, 530 mg/kg[61]	Human, skin, 1%/48 h[62]	Human, skin. mild irritant[63] Rabbit, skin, severe irritation, 10 mg/24 h[64] Rabbit, skin, moderate irritation, 500 mg[61] Rabbit, eye, severe irritation, 250 μg[64]
		VX decontamination[35]	
N-chlorodiiso-propylamine[2]			
Chloroform[65] $(CHCl_3)$			

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Acetic acid" (CH_3CO_2H)			
N-chloroisopropyl-amine (C_3H_8NCl)[2]			
Ethyl methyl phosphonic acid' ($C_3H_9CO_3$)			
N-chloroiso-propylamine[66.2] (C_3H_8NCl)			
1,3 Diisopropylurea[66.2] ($C_7H_{16}N_2O$)			
Ammonia (NH_3)[65,66]			
	HD decontamination[35]		
2-Chloroethyl vinyl sulfide' (C_4H_7SCl)			
Divinyl sulfide" (C_4H_6S)			
Mustard sulfoxide ($C_4H_8SO_2Cl_2$)		Rat, 150 mg/kg[68]	
Mustard sulfone ($C_4H_8SO_2Cl_2$)		Cat, inhalation, 1430 mg/m^3/10 min[69] Rahbit. inhalation, 1430 mg/m^3/ IO min[69]	
2-Hydroxyethyl vinyl sulfide' (C_4H_8SO)			
Thiodyglycol ($C_4H_{10}SO_4$)	Guinea pig, oral, 3960 mg/kg[27]	Rabbit, skin irritant.	500 mg[28]
Carbon dioxide (CO_2)			Nontoxic; constituent of earth's atmosphere

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Calcium sulfate ($CaSO_4$)			Nontoxic; plaster of paris[70]
Calcium chloride ($CaCl_2$)	Rat, oral, 4 g/kg[71]		Dessicant, used in therapeutic treatment of hypocalcemia[71]
	GA combustion[72]		
Hydrogen cyanide (HCN)	Rat, inhalation, 484 ppm/5 min[73] Mouse. inhalation, 323 ppm 5 min[73] Human, inhalation, 4000 mg/m^3 (0.5 min)[76] Human, inhalation, 687 mg/m^3 (30 min)[76]	Human, inhalation, 200 ppm/5 min[74] Human, inhalation, 120 mg/m^3/1 h[77] Human, inhalation, 200 mg/m^3/ IO min[78]	
Phosphorous pentoxide[79] (P_2O_5)			
Nitrogen dioxide (NO_2)[80]	Rat, inhalation, 88 ppm/4 h[81] Mouse, inhalation, 1000 ppm/ IO min[83] Rabbit, inhalation, 3 15 ppm/ I5 min[85] Guinea pig, inhalation, 30 ppm/1 h[86]	Human, inhalation, 200 ppm/1 min[82] Dog, inhalation, I23 mg/m^{3}[84]	
	GB combustion[72]		
Phosphoric acid (H_3PO_4)			Human, skin and mucous membranes, irritant"
Hydrogen fluoride (HF)	Rat, inhalation, 1276 ppm/1 h[88] Mouse, inhalation, 342 ppm/1 h[91] Monkey, inhalation, 1774 ppm/1 h[92]	Human, inhalation, 50 ppm/30 min [89]	Human, inhalation, TC_{Lo} = 100 mg/m^3/1 min"
Phosphorous pentoxide[93] (P_2O_5)			

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
		VX combustion"	
Phosphoric acid" (H_3PO_4)			
Sulfuric acid (H_2SO_4)	Rat, inhalation, 510 $mg/m^3/2$ h[95] Mouse, inhalation, 320 $mg/m^3/2$ h[95]		Human, inhalation, severe irritant[96]
Phosphorous pentoxide[93] (P_2O_5)			Human, inhalation, TC_{Lo} = 5 mg/m^3/ 15 min[97]
Nitrogen (N_2)			Nontoxic; constituent of earth's atmosphere (~80%)[98]
Nitrogen dioxide* (NO_2)			
		HD combustion[72]	
Sulfur (S_2)			Human, eye irritation, 8 ppm[100] Human, respiratory tract, inflammation[101]
Sulfur dioxide[102] (SO_2)	Rat, inhalation, 2520 ppm/1 h[103]	Human, inhalation, 3000 ppm/5 min[74]	Human, eye and respiratory tract, irritant'" TC_{Lo} = 3 ppm/5 d and 4 ppm/1 min[105]
Hydrogen sulfide (H_2S)	Rat, 444 ppm[106] Mouse, 673 ppm/1 h[109]	Human, 600 ppm/30 min[107] Human, 800 ppm/5 min[74]	Human, eye irritation, rhinitis, tracheobronchitis, pulmonary edema[108] Dog, cat, rabbit, temporary damage to eye, 50–100 ppm/ several hours to days[110]
Ethancthiol (C_2H_5SH)	Rat, 4420 ppm/4 h[111]		Irritant, mucous membranes"'

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Diethyl disulfide[2] $(C_2H_5)_2S_2$			
1,4-Dithiane[2] $(C_2H_4)_2S_2$			
Methylene chloride (CH_2Cl_2)	Mouse, 14,400 ppm/7 h"' Rat, 88.000 mg/m^3/30 min"'	Dog, 14.000 ppm/7 h[113] Rabbit. 10.000 ppm/7 h[116]	Human, behavioral symptoms, 500 ppm/8 h"' Human, behavioral symptoms, cardiovascular, 500 ppm/1 y"' Human, disturbance of psychomotor performance, 800 ppm; other observable neurological effects, down to 200 ppm[118]
Chloroform"' $(CHCl_3)$			
Ethyl chloride (C_2H_5Cl)	Rat, 160 g/m^3/2 h[95] Mouse, 146 g/m'/2 h[95]		Human, 40,000 ppm (two inhalationations) stupor, eye irritation, stomach cramps[120] Human, 19,000 ppm (1 2 min), weak analgesia[120] Human, 13.000 ppm, CNS effects[121]
1,2-Dichloroethane $(C_2H_4Cl_2)$	Rat, 1000 ppm/7 h[122]	Mouse. 5000 mg/m^3/2 h[123] Rabbit, 3000 ppm/7 h[125] Guinea pig, 1500 ppm/7 h[125]	Human, peripheral nervous system effects, coma, gastrointestinal tract effects, 4000 ppm/1 h[124] Human, eye, nose, throat irritation[126] Many species (cat, rat monkey, rabbit), fatty changes in liver"'

Tsbk B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
1,1,1-Trichloroethane ($C_2H_3Cl_3$)	Rat, 18.000 ppm/4 h'" Mouse, 391 1 ppm/2 h[130]	Cat, 600 mg/m^3/4 h[95] Human, 27 g/m^3/10 min[131]	Human, 200 ppm/4 h, behavioral symptoms[129] Human, 450 ppm/8 h, eye irritation'" Human, >5000 ppm, narcosis, life-threatening'" Human, 1900-2650 ppm, lightheadedness[133] Human, 1000 ppm, disturbance of equilibrium'" Human, 350-500 ppm, slight changes in perception[133]
1,1,2-Trichloroethane ($C_2H_3Cl_3$)	Rat, 2000 ppm/4 h[134]	Cat, 13,100 mg/m^3/4.5 h[135] Rat, 500 ppm/8 h[32]	Human, 2000 ppm/5 min, equilibrium disturbances, CNS depressant effects'"
1,1,1,2-Tetrachloroethane ($C_2H_2Cl_4$)	Rat and rabbit, 2500 mg/m^3[137]		Human, nervous system[138]
1,1,2,2-Tetrachloroethane		Rat, 1000 ppm/4 h[139] Mouse, 9 g/m'/40 min'" Cat, 19 g/m^3/45 min[135]	Human, TC_{Lo} = 1000 mg/m^3 /30 min, produces behavioral symptoms[135]
Hydrogen chloride (HCl)	Mouse, inhalation, 2142 ppm/30 min[140] Rat, inhalation, 5666 ppm/30 min'"	Human, 1300 ppm/30 min"' Human, 3000 ppm/5 min[74] Rabbit, 4416 ppm/30 min[142] Guinea pig, 4416 ppm/30 min[142]	Human, inhalation, inflammation and ulceration of respiratory tract'"
Lewisite combustion"			
Acetylene (C_2H_2)		Human, 50 ppm/5 min[74]	
Acetylene monochloride' (C_2HCl)			
Arsenic trichloridc ($AsCl_3$)		Mouse, 338 ppm/10 min[143] Cat, 200 mg/m^3/20 min[144]	

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Arsenic trioxide (As_2O_3)			145
Chlorine (Cl_2)	Rat. inhalation, 293 ppm/1 h[110] Mouse, inhalation, 137 ppm/1 h[110]	Human, 500 ppm/5 min[146] Human, 2530 mg/m^3/30 min[147] Cat, 660 ppm/4 h[147] Rabbit. 660 ppm/4 h[147] Guinea pig, 330 ppm/7 h[147]	
Methyl chloride (C_2H_3Cl)	Rat, inhalation, 152,000 mg/m^3/30 min'" Mouse, inhalation, 3146 ppm/7 h[148]	Rat, 6500 mg/m^3 [148]	
Vinyl chloride (C_2H_3Cl)			Human, narcotic at high levels[149]
Acetylene dichloride ($C_2H_2Cl_2$)		Rabbit, -200 ppm/1 h[150]	Rabbit, renal and hepatic toxicity, 126–307 ppm/1 h or 17–23 ppm/6 h[150]
C_6H_5[2]			
Arsenic oxychloridc' (AsOCl)			
Chlorovinyl arsenous oxide' (C_2H_2AsO)			
	Microbial activity		
Ethyl methyl phosphonic acid' ($C_3H_9PO_3$)			
Methyl phosphonic acid[151] (CH_5PO_3)			

Table B.19 (continued)

Process/ product identification	LD_{50}/LC_{50}	LD_{Lo}/LC_{Lo}	Other effects
Diisopropyl ethyl mercapto amine[2] ($C_8H_{19}SN$)			

[1]Routes of exposure pertinent to hydrolysis products are oral and percutaneous only.
[2]No relevant biological data were found.
[3]Dzhanashvili 1967.
[4]*Brit. J. Ind. Med. 23:* 153, 1966.
[5]Towill et al. 1978. p. 7.
[6]Pest. Chem. Official Compend.. 1966, p. 596.
[7]Dclga et al. 1961.
[8]*Abdernalden's Handbuch det Biologischen Arbeitsmethods 4: 1340 (* 1935).
[9]Selby et al. 1971.
[10]Mecler 198 1, pp. 6-20.
[11]It was reported that MPA is not toxic; no data were given (Mill and Gould 1979).
[12]A 2.3-g dose of HF is considered lethal, but 0.9 g has been recorded as fatal (Arena and Drew 1986, pp. 192–194).
[13]Antonova and Salmina 1978.
[14]Adelson 1962.
[15]Deichmann 1969. p. 339.
[16]Compound is a hydrolysis product of the GB and VX stabilizer diisopropylcarbodiimide.
[17]Union Carbide Data Sheet 1969.
[18]Smyth et al. 1954.
[19]Windholz et al. 1983, pp. 34–35.
[20]*Archiv. Toxikol.* 17: *183* (1958).
[21]Doull et al. 1980. p. 687.
[22]Raw Material Data Handbook 1974. p. 44.
[23]National Academy of Sciences, National Research Council, Chemical Biological Coordination Center Review 1953.
[24]Biochemischc Zeitschrift 134: 437 (1923).
[25]Arena and Drew 1986. pp. 290–292.
[26]Eye was rinsed after application [*Toxicology* 23: 281 (1982)].
[27]*J. Indust. Hyg. Toxicol. 23: 259* (*I94 I*).
[28]Union Carbide Data Sheet 197 I.
[29]Compound has vcsicant properties (U.S. Army 1974, p. 71).
[30]See H D hydrolysis.

[31]Toxicological data for acetylene are based on exposures to gas or vapor phase; see Lewisite combustion products for vapor phase data.
[32]*Amer. Indust. Hyg. Assoc. J.* **30:** 470 (1969).
[33]Peoples et al. 1977.
[34]*Pharmaceutial J.* **185:** 361 (1960).
[35]Routes of exposure pertinent to decontamination products are percutaneous and inhalation only.
[36]Toxic effects similar to HCN (Windholz et al. 1983, p. 385).
[37]*Abdernalden's Handbuch der Biologischen Arbeitsmetheden* **4:** 1341 (1935).
[38]*Brit. J. Exp. Path.* **33:** 241 (1946).
[39]Toxic via ingestion (oral rat LD_{50} = 0.18 mg/kg; oral human LD_{10} = 5 to 10 g) and aqueous solutions. Would burn moist skin. Not inhalation hazard (Windholz et al. 1983, p. 1235).

[40]Toxic via ingestion (oral guinea pig LD_{50} = 5 g/kg) (Windholz et al. 1983, p. 230).
[41]Decontamination product of GB stabilizer diisopropylcarbodiimide.
[42]Union Carbide Data Sheet (8/7/63).
[43]*Arch. Ind. Health* **21:** 28 (1960).
[44]Nixon, Tyson, and Wertz 1975.
[45]Verschueren 1983, p. 146.
[46]*Am. Indust. Hyg. Assoc.* Qtrly. (1956).
[47]Ghiringhilli and DiFabio 1957.
[48]NIOSH/OSHA 1981, p. 2.
[49]*Arch. Ind. Hyg. and Occup. Med.* **4:** 119 (1951).
[50]Sittig 1981, pp. 20–21.
[51]Wands 1981, pp. 3048–52.
[52]Iti. *Tox. and Hazard Indust. Chem. Safety Man.* 1982, p. 121.
[53]Grant 1986, p. 213.
[54]Smyth et al. 1962.
[55]*Arch. fur Hygiene und Bakteriologie* **116:** 131 (1936).
[56]Torkelson and Rowe 1981, p. 3463; Doull, Klaassen and Amdur 1980, p. 471; IARC 1979a, p. 415.
[57]*Pesticide Chemicals Official Compendium*, Assoc. of the Amer. Pestic. Contr. Officials Inc. 1966, p. 230.
[58]*Fluorine Chemistry Reviews* **1:** 197 (1967).
[59]Torkelson, Oyen, and Rowe 1976.
[60]Decontamination product of GB stabilizer tributylamine.
[61]Union Carbide Data Sheet (4/10/68).
[62]Opdyke 1981.
[63]Guest, Katz, and Astill 1982, p. 4914.
[64]*Arch. Ind. Hygiene and Occup. Med.* **10:** 61 (1954).
[65]See toxicological data under GB decontamination for chloroform, acetic acid, and ammonia.
[66]Decontamination product of VX stabilizer diisopropylcarbodiimide.
[67]Toxicity data only for nonpercutaneous or noninhalation exposures.

[68]Route of administration unknown (Ishidate et al. 1952).
[69]NDRC 1942.
[70]Windholz et al. 1983, p. 234.
[71]Windholz et al. 1983. p. 229.
[72]Route of exposure pertinent to combustion products is inhalation only.
[73]*Toxicol. & Appl. Pharm. 42:* 417 (1977).
[74]*Tabulae Biologicae* 3: *23 I (* 1933).
[75]Toxic hydrogen cyanide exposure can occur via inhalation, ingestion, or percutaneous absorption (Towill et al. 1978, p. 127).
[76]U.S. Army 1974, p. 81.
[77]*J. Indust. Hyg. Toxicol.* 24: *255* (1942).
[78]WHO. Technical Report Series 1970. p. 30.
[79]P_2O_5 is expected to react with water in the atmosphere and be converted to phosphoric acid (H_3PO_4). See GB combustion.
[80]Levels of NO_2 in air regulated by USEPA under provisions of the Clean Air Act. Maximum permissible levels are 100 $\mu g/m^3$. *Code of Federal Regulations* 1986a.
[81]Gray et al. 1954.
[82]Mason 1974.
[83]*J. Combust. Tox. I: 246* (1977).
[84]*Tox. & Appl. Pharm.* 9: 160 (1966).
[85]*Amer. Indust. Hyg. Assoc. J.* 23: *457* (1962).
[86]Buckley and Balchum 1965.
[87]Windholz et al. 1983, p. 1059.
[88]Darmer et al. 1972.
[89]Deichman and Gerarde 1969. pp. 3 17–18.
[90]Effects are to the eye, nasal, and pulmonary systems. *J. Indust. Hyg. 16:* I29 (1934).
[91]*J. Combust. Tox. 3:* 61 (*1976).*
[92]Aerospace Med. Res. Lab. Report No. TR-70-77 (1970).
[93]See GA combustion.
[94]See GB comustion.
[95]Izmerov 1982.
[96]Corrosive to all body tissues; inhalation of concentrated vapors may cause serious lung damage (Windholz et al. 1983, pp. 1288-I 289).
[97]Pulmonary system is affected (Iti. *Tox. and Hazard. Indust. Safety Man. 1982,* p. 499).
[98]Windholz et al. 1983, p. 947.
[99]See GA combustion.
[100]*Anal. Chem. 21:* 141 1 (1949).
[101]No concentrations were provided (*Encyc. Occup. Health and Safety* 1971, p. 1368).
[102]Levels of SO_2 in air regulated by USEPA under provisions of the Clean Air Act. Maximum permissible levels are 80 $\mu g/m^3$ with a once annual 24-h maximum of *365* $\mu g/m^3$. *Code of Federal Regulations* 1986b.
[103]NTIS No. AD-A148-952.
[104]Arena and Drew 1986, pp. 313–314.
[105]*Tox. Appl. Pharm.* 22: 319 (1972) and *J. Appl. Phys.* 17: *252 (1962).*

[106]Exposure time was not specified. Personal communication from H. B. Lackey, Chem. Prod. Div., Crown Zellerback Corp., Camas, Wash., to Dr. H. E. Christensen. NIOSH, 1978.
[107]Lefaux 1968. p. 207.
[108]Gosselin et al. 1976, part III, pp. 169 173.
[109]NTIS, No. PB 214-270.
[110]Grant 1986, pp. 495497.
[111]Amer. *Indust. Hyg. Assoc. J.* 19: 171 (1958).
[112]Windholz et al. 1983. pp. 540541.
[113]*NIH Bull. 191: 1 (1949).*
[114]Stewart et al. 1972.
[115]Bakhishev 1975.
[116]*J. Indust. Hyg. Tox.* 26: *8 (1944).*
[117]Intermittent exposures (Abstracts on *Hygiene* 43: 1123 [1968]).
[118]Winnecke 1981.
[119]See chloroform inhalation toxicity under GB decontamination.
[120]Patty 1962, p. 1275.
[121]No duration of exposure given (ACGIH 1971, p. 106).
[122]Spencer et al. 1951.
[123]Lazarew 1929.
[124]*Pesticide Chemicals Official Compendium.* Assoc. of the Amer. Pestic. Contr. Officials, Inc., 1966, p. 500.
[125]*J. Pharmacol. Exp. Ther.* 84: *53 (1945).*
[126]Exposure was to "high" (undefined) concentration. Menschick 1971, pp. 385-386.
[127]Exposure was to 1000 ppm approx. 7 h/d, 5 d/week. Patty 1962, p. 1282.
[128]Marhold 1972.
[129]Arch. *Toxicol.* (Suppl.) 5: 96 (1982).
[130]*Jap. J. Indust. Health* 13: *226* (1971).
[131]*J. Occup. Med. 8: 358* (1966).
[132]Salvini et al. 1971.
[133]USEPA 1982. Report No. EPA-600/8-82-003, pp. (5)49-(5)50.
[134]Verschueren 1983. p. 606.
[135]*Archiv. fur Hygiene und Bakteriologie 116:* 131 *(1936).*
[136]Arena and Drew 1986, pp. 259-260.
[137]Truhaut et al. 1974.
[138]Dose-dependent symptoms include headache, vertigo, nervousness, numbness, and tremors. National Research Council, *Drinking Water and Health 1977,* p. 768.
[139]*J. Indust. Hyg. Tox. 31: 343* (1949).
[140]Windholz et al. 1983, pp. 692 and 696.
[141]Lefaux 1968, p. 207.
[142]Machle et al. 1942.

[143] *Handbook of Toxicology* 1955, p. 324.
[144] *Handbook of Toxicology* 1955, p. 1.
[145] Adult human fatal dose is 120 mg. Arena and Drew 1986. pp. 188-192.
[146] Prentiss 1937, pp. 148-151.
[147] Lehmann 1887.
[148] Haz. Subst. Data Base 1987 (No. 883).
[149] Windholz et al. 1983. p. 1430.
[150] Estimated from Rcichart et al. 1978.
[151] See entries on methyl phosphonic acid under GB hydrolysis.

Table B.20. Chronic effects of exposure to products of agent hydrolysis, decontamination, combustion, and microbial degradation

Process/product identification	Carcinogenicity	Genetic effects	Reproductive effects	Systemic effects
		GA hydrolysis[1]		
Ethyl phosphorylcyanidate[2] ($C_3H_6NPO_3$)				
Dimethylamine (C_2H_7N)		Mutagenicity negative[3] Cytogenicity negative[5]	Negative[4]	
Hydrogen cyanide (HCN)				Weakness, fatigue[6] paralysis[7]
Ethyl, *N,N*-dimethyl ammo phosphoric acid[2] ($C_4H_{12}NPO_3$)				
		GB hydrolysis[1]		
Isopropylmethyl phosphonic acid ($C_4H_{11}O_3P$)		Mutagenicity negative[8]		Negative[9]
Methyl phosphonic acid[1] (CH_5O_3P)				
Hydrogen fluoride (HF)				*10*
Isopropyl Alcohol (C_3H_8O)		Mutagenicity negative[11] Cytogenicity negative"	Positive[12]	Human (oral) TD_{Lo} = 223 mg/kg[13]
1,3-Diisopropylurea[2,15] ($C_7H_{16}N_2O$)				
		VX hydrolysis[1]		
Ethyl methyl phosphonic acid[1] ($C_3H_9PO_3$)				
Ethyl methyl phosphonothioic acid[1] ($C_3H_9SPO_2$)				
Diisopropylaminoethanol[7] ($C_8H_{19}ON$)				
Bis (2-diisopropylaminoethyl) sulfide[7] ($C_{16}H_{36}N_2S$)				
Diethyl methyl phosphonate[2]				
Ethanol (C_2H_5OH)	Rat. TD_{50} = 9110 mg/kg[16]	Cytogenicity positive[17]	Teratogenic[18] Fetotoxic[19]	

Table B.20 (continued)

Process/product identification	Carcinogenicity	Genetic effects	Reproductive effects	Systemic effects
Ethyl hydrogen methyl phosphonate[2]				
Methyl phosphonic acid[2] (CH_5O_3P)				
S-(diisopropyl aminoethyl) methyl phosphonothioate[1] ($C_9H_{22}NSPO_2$)				
1,3-Diisopropylurea[2,20] ($C_7H_{16}N_2O$)				
		HD hydrolysis[1]		
Bis-2[bis(2-hydroxyethyl)-sulfonium ethyl] sulfide dichloride[2] ($C_{12}H_{28}S_3Cl_2$)				
Hydrogen chloride (HCl)		Cytogenicity positive[21,22]	Positive[23]	
Thiodiglycol ($C_4H_{10}SO_4$)[2]				
		Lewisite hydrolysis[1]		
Chlorovinyl arsenous oxide[2] (C_2H_2ClAsO)				
Hydrogen chloride (HCl)[24]				
Acetylene (C_2H_2)[25]				
Sodium arsenite ($NaAsO_2$)	Human carcinogen[26]	Transformation"[28] Cytogenicity positive[27,30,31] Mutagenicity[31]	Mouse TD_{Lo}: 40-45 mg/kg (10 d pregnant)" Hamster TD_{Lo}: 5 mg/kg (9 d pregnant)" Hamster TD_{Lo}: 25 mg/kg (12 d pregnant)[36]	
		GA decontamination[17]		
Cyanogen chloride[1] (CClN)				
		GB decontamination[17]		
Isopropyl methyl phosphoric acid ($C_4H_{11}O_3P$)		Mutagenicity negative[8]		*Negative*[1]
Sodium fluoride (NaF)[2]				
Calcium fluoride (CaF_2)[2]				
GB stabilizer DIPC decontamination[17]				
N-chloroisopropylamine[2] (C_3H_8NCl)				
1,3-Diisopropyl urea[1] ($C_7H_{16}N_2O$)				

Table B.20 (continued)

Process/product identification	Carcinogenicity	Genetic effects	Reproductive effects	Systemic effects
Chloroform ($CHCl_3$)	Probable human carcinogen[48] Rat positive[42] Mouse positive[42,44] Rat positive, Mouse negative[47]	Mutagenicity negative[39] Mutagenicity positive[43] Chromosome effects negative[45]	Rat fetotoxic, retarded development, teratogenic[40] Mouse sperm abnormalities[46] Mouse negative in sperm morphology assay[a]	Liver (human) hepatomegaly, fatty degeneration, toxic hepatitis[a] CNS (human) psychiatric and neurologic effects[a]
Acetic acid ($C_2H_4O_2$)	Mouse negative[50]	Cell transformation negative[51] Mutagenicity negative[53]	Rat, rabbit, mouse negative[52]	
Ammonia (NH_3)[2]				
		GB stabilizer TBA decontamination[37]		
Dibutylchloramine ($C_8H_{18}NCl$)[2]				
N-butanoic acid ($C_4H_8O_2$)	Negative (as promoter)[54]	Mutagenicity negative[a] Chromosome aberrations negative[55,56] Sister chromatid exchange positive[56]		
		VX decontamination[37]		
N-chlorodiisopropylamine[2]				
Chloroform ($CHCl_3$)[57]				
Acetic acid ($C_2H_4O_2$)[58]				
N-chloroisopropylamine[2] (C_3H_8NCl)				
Ethyl methyl phosphonic acid[2] ($C_3H_9PO_3$)				
		VX *stabilizer DIPC decontamination*		
N-chloroisopropylamine[2] (C_3H_8NCl)				
1, 3 Diisopropylurea[2] ($C_7H_{16}N_2O$)				
Chloroform ($CHCl_3$)[57]				
Acetic acid ($C_2H_4O_2$)[58]				
Ammonia (NH_3)[2]				
		HD decontamination[37]		
2-Chloroethyl vinyl sulfide[2] (C_4H_7SCl)				
Divinyl sulfide (C_4H_6S)			Embryotoxicity negative[59]	
Mustard sulfoxide ($C_4H_8SOCl_2$)[2]				
Mustard sulfone ($C_4H_8SO_2Cl_2$)[2]				

Koike, M., Ito, T., and Yamamoto, M. 1983. "Cytogenetic Effects of Maternal Alcohol Uptake on F1 Mouse Fetuses," *Teratology 28,* 31A.

Koslow, E. 1987. "Decontamination," pp. 28-30 in *Nuclear, Biological and Chemical Defense & Technology International 1987 Yearbook.*

Krackow, E. H. 1956. Toxicology *of V Agents,* Army Chemical Center, Chemical Warfare Labs., CWLR 2065 (Secret Report, Unclassified Title), as quoted in Fielding 1960.

Krause, H., and Grussendorf, E. I. 1978 "Syntopy of Bowen's Disease and Lewisite Scar," Hautarzt **29**(9), 490-3.

Krop, S. and Kunkel, A. M. 1954. "Observations on Pharmacology of the Anticholinesterases Sarin and Tabun," *Proc. Soc. Exp. Biol. Med.* 86, 530-33.

Kusic, R., et al. 1985. "HI-6 in Man: Blood Levels, Urinary Excretion, and Tolerance after Intramuscular Administration of the Oxime to Healthy Volunteers," *Fund. and Appl.* Toxicol. 5, S89-97.

LaBorde, J. B., and Bates, H. K. 1986. *Developmental Toxicity Study of Agent GB-DCSM Types I and II in CD Rats and NZW Rabbits. Final Report,* National Center for Toxicological Research, FDA, Jefferson, Ark.

Land, P. C., et al. 1981. "Morphologic Changes in Mouse Spermatozoa after Exposure to Inhalation Anesthetics during Early Spermatogenesis," *Anesthesiology* **54**(1), 53.

Landauer, M. R. and Romano, J. A. 1984. "Acute Behavioral Toxicity of the Organophosphate Sarin in Rats," *Neurobehav. Toxicol. Teratol.* 6, 239-43.

Landolph, J. R. 1983. "Transformation of C3H/10T1/2 Mouse Fibroblasts by Carcinogenic Metal Salts in the Absence of Base Substitution Mutations," *Proc. Am. Assoc. Cancer Res. 24,* 100.

Larramendy, M. L., Popescu, N. C., and DiPaolo, J. A. 1981. "Induction by Inorganic Metal Salts of Sister Chromatid Exchanges and Chromosome Aberrations in Human and Syrian Hamster Cell Strains," *Environ. Mutagen. 3, 597-606.*

Lazarew, N. W. 1929. "Uber die Marketische Wirkungskraft der Dampfe der Chlorderivaten des Methans, des Athans und des Athylens," *Naunyn-Schmiedeberg's Archiv fur Experimentelle Pathologie und Pharmakologie* 141, 19-24.

Table B.20 (continued)

Process/product identification	Carcinogenicity	Genetic effects	Reproductive effects	Systemic cffatr
Phosphorous pentoxide (P_2O_5)[64]				
Nitrogen (N_2)				Nontoxic: constituent of earth's atmosphere
Nitrogen dioxide (NO_2)[79]				
		H D combustion[82]		
Sulfur (S_2)		Mutagenicity negative"		
Sulfur dioxide (SO_2)		Mutagenicity ambiguous to negative"	Rat. TC_{Lo} = *4970* $\mu g/m^3$[82] mouse, TC_{Lo} = 25 *ppm/7 h*[83]	
Hydrogen sulfide (H_2S)			Human ● mbigttotts for spontaneous abortions at >4 $\mu g/m^3$[84]	
Ethanethii (C_2H_5SH)				Negative"
Diethyldisulfide ($(C_2H_5)_2S_2$[2]				
1.4 Dithiane ($(C_2H_4)_2S_2$[2]				
Methylene chloride (CH_2Cl_2)	Positive endocrine system" Positive- pulmonary system" *Positive—unspecified*[90]	Mutagenicity -positive[87] sister chromatid exchange[89]		
Chloroform ($CHCl_3$)[57]				
Ethyl chloride (C_2H_5Cl)	Currently under test"	Mutagenicity- positive"		
1,2-Dichloroethane ($C_2H_4Cl_2$)	Rat-poaitivc (oral)" *Mouse—positive* (oral)" Rat-negative"	Mutagenicity—positive[97] Mutagenicity —psitive[96] Rat- DNA damage"	Rat, rabbits- negative *teratogenicity*, embryotoxicity. reproductive performance[94] Rat- fertility effects"	
1,1,1-Trichloroethane ($C_2H_3Cl_3$)	Rat—negative[100] Rat and mouse— negative *(oral)*[103]	Mutagenicity positive[101]	Rat—developmental toaicity 2100 ppm/24 h[102] *Rat -embryotoxicity* 2100 ppm/6 h'"	
1,1,2-Trichloroethane ($C_2H_3Cl_3$)	Mouse—positive (oral)'" Rat -negative (oral)'"	Mutagenicity- negative"		
1,1,1,2-Tetrachloroethane ($C_2H_2Cl_4$)	Mouse—positive (oral)[108] Rat-negative (oral)[108]	Mutagenicity-negative in *Salmonella* test[108]		
1,1,2,2-Tetrachloroethane ($C_2H_2Cl_4$)	Mouse—positive (oral)[108] Rat -negative (oral)[108]	Mutagenicity —negative in *Salmonella* test[108]		High concentrations induce narcosis. nephritis. toxic hepatitis, and liver atrophy'.
Hydrogen chloride (HCl)	Rat –negative[110]	Chromosome aberrations"'	Rat. TC, = *450* mg/m^3/1 h[111]	
		Lewisite combustion[62]		
Acctyltnc (C_2H_2)		Mutagenicity negative[113]		Human TC, = 20 pph[114]

ive effects	systemic effects

e

re[116]

s y[121,122]

)84)

of teeth) (Arena and Drew 1986. pp. 192 94).

ts) (Antonova and Salmina 1978)

Radikc et al. 1981

l entry under GB combustion.
lagenic in Ames test at 210 ppm/40 min (Isomura et al. 1984).

lima greater. respectively. than controls (Isomura et al. 1984).
id sister chromatid exchanges (Tsuda et al. 1981).
terial Safety Data Sheet #8 1984).
ial mutations were observed in similarly treated mice (Voroshilin et al. 1975).
h showed impaired fertility and embryotoxicity (*Labor Hyg. and Occ. Dis* 19: 57

on frequency (Gerdes 1971).
wa 1974).
ity and no teratogenic effects (Murray et al. 1979)
ffects (Hoffman and Campbell 1977).

st (Shapiro 1977).
and Tsereteli 197 I).
Cosmetics Toxicol. 18: 743. 1980).
Neimi 1982).
, organ weights, etc. (*ACGIH 4th ed and suppl.* 1980, p. 189).

I Series. NTP-TR-306. 1986)

deaths (Friedlander 1978)

6. 18 of pregnancy) (Rao et al. 1980).
Hammons 1979. pp. 96 104)

,800 and 5600 mg/kg (National Cancer Inst. 1977).

GB stabilizer TBA yields diButylchloramine and *N*-Butanoic acid on reaction with STB. No relevant biological data were found for dibutylchloramine. *N*-Butanoic acid is a mild irritant for human skin (Guest, Katz, and Astill 1982, p. 4914). It can cause mild to severe skin or eye irritation in rabbits depending on the dosage and mode of exposure [Union Carbide Data Sheet 4/10/68; *Arch. Ind. Hygiene and Occ.*

[18]Epidemiological evidence for teratogenic effects in humans and laboratory animals was presented (Streissguth et al. 1980).
"Fading female mice 40% ethanol prior b mating caused increased fetus absorption and increased chromosome aberrations (Koike et al. 1983).
[20]Hydrolysis product of the VX stabilizer diisopropylcarbodiimide.
[21]Produced *nondisjunction* in *Drosophila melanogaster* (fruit fly) fed 100 ppm (*Theor Appl. Genetics 39: 330 1969).*
[22]Chromosome ● berralions were induced in CHO cells with HCl-induced sublethal pH's below 5.5 (Brusick 1986).
[23]Cytogenetic abnormalities and developmental defects were observed in sea urchin embryos. both in exposed embryos and in the offspring of exposed sperm, exposed to HCl-induced sublethal pH's below 6.5 (Cipollaro et al. 1986).
[24]See HD hydrolysis products.
"Data for acetylene arc based on exposures io the gas or vapor phase and are not applicable io oral and percutaneous exposures See Lewisite combustion for gas-phase ● atylene data,
[26]International Agency for Research on Cancer (IARC), 1980 (pp. 39- 141).
"Hamster embryo cells were transformed in vitro with 2 μmol/L (Lee et al. 1985).
"Mouse embryo cells were transformed in vitm with 12 μmol/L (Landolph 1983).
[29]Reduced fertility and embryotoxicity were observed (Baxley et al. 1980; Baxley et al. 198 I).
[30]Chromosome abberations were induced in human lymphocytes in vitro with I mg/L (Larramendy et al. 1981).
"Chromosome aberrations were induced in human leucocytes and fibroblasts in vitro with I nmol/L (*Advances in Exptl Mrd ond Biol* 91: I 17 1978).
[32]SCEs were induced in human lymphocytes in vitro with 3900 nmol/ (*Scand J Work*, Environ *and Health 7: 277* 1981)
"Similar cytogenetic effects were observed in hamster cells in culture (Ohno et al 1982; Wan et al 1982).
"Reduced fertility was observed (Harrison and Hood 198 I).
[35]Arsenite was reported as both positive (Nishioka, 1975) and negative (Rossman et al, 1980) in *E. coli*. Ii was negative in CHO cells (Rossman et al, 1980).
[36]Embryotoxicity was observed (Hood and Harrison 1982).
"The only routes of exposure considered relevant for decontamination products arc inhalation and percutaneous (skin) absorption
"Davidson et al 1982; U.S EPA 1979; and IARC 1982; estimated potency in humans (based on animal oral studies) = 8.1×10^{-2} cancers/mg/kg/d for 70-year exposure (U.S. EPA 1986. pp. 7.77 to 7-80).
[38]"Negative in 5 strains in Ames test (Van Abbe et al 1982); all but one other study gave negative results (Davidson et al. 1982)
[40]Rats were exposed via inhalation to subanesthetic doses (150, 500, and 1500 mg/m^3 or 30. 100. and 300 ppm) for 7 h/d, days 6 I5 of gestation; 100 ppm caused low incidence of acaudate fetuses with imperforate anuses; 100 ppm for 7 h/d in another study resulted in cleft palate (Davidson et al. 1982)
[41]IARC 1979a; ACGIH 1980.
"Kidney epithelial tumors. $CHCl_3$ administered via gavage; IARC 1979a
"Induced mitotic gene convertants. recombinrnts and revertants in *Saccharomyces cerevisiae* (yeast) in culture (Callen et al. 1980): in vivo studies in mice were positive (Davidson et al. 1982).
[44]Hepatocellular carcinomas in mice (IARC 1979a).
"In vitro test with human lymphocytes negative for chromosome breakage or sister chromatid exchange (Kirkland. Smith. and Van Abbe 1981)
[46]Increased abnormal sperm in mice exposed via inhalation 4 h/d for 5 d (Land et al. 1981).
"Positive for renal tubular adcnomas and adenocarcinomas in male rats when given in drinking water; negative for hepatocellular carcinomas in mice when given in drinking water (Jorgenson et al 1985).
[48]Given intraperitoneally 5 times/d at 5 mL/kg/d to mrk (CBA x BALB/C) F I mice (Topham 1981).
[49]NIOSH 1974.
[50]10% solution applied to skin, a few days per week for lifetime (Orr 1938)
"250 1500 μg/ml doses. mouse cell culture (Abernethy et al 1982).
"Rats and mice 16-1600 mg/kg of cider vinegar (5% acetic acid) via oral intubation, days 6 I5 of gestation: rabbits treated days 6 18 with same dosage; no effects on reproductive or teratogenic endpoints (Food and Drug Research Labs 1974).
"Ames test and Chinese hamster lung cells (Ishidate et al 1984).
[54]Trosko et al 1981.
"Negative both in Ames test and in Chinese hamster lung cell assay for chromosome aberrations (Ishidate et al. 1981)
[56]3 4-fold increase in frequency of sister chromatid exchange: reversible inhibition of cell proliferation and differentiation in Chinese hamster lung cells (Tai and Ting 1979)
[57]See chloroform entries under GB stabilizer.
[58]See acetic acid entries under GB stabilizer.
[59]Glukharev et al. 1980.
[60]Ames test (Leopold et al. 1982)
[61]Windholz et al 1983, p. 234.

[62]Only route of exposure considered relevant for exposure to combustion products is inhalation.
[63]Windholz 1983 p. 6%.
[64]P_2O_5 is expected to react with moisture in the atmosphere and produce phosphoric acid (H_3PO_4). See phosphoric acid entry under GB combustion.
[65]Rats exposed to 8 27 ppm NO_2 (3 h) exhibited significant lung cell mutation over range IS 27 ppm. NO_2 is also mutagenic in Ames test at 2 IO ppm/40 min (Isomura et al 1984).
[66]*Gigiena i Sanitarya* 42: 22 (1977).
[67]Teratology 29: 33A (1984).
[68]Doull, Klaassen, and Amdur 1980 (pp. 622 62S).
[69]Rat lung cells exposed to 8 27 ppm NO_2 (3 h) exhibited increases in chromosome aberration that were 2.5 and I I.6 times greater, respectively. than controls (Isomura et al. 1984).
[70]CHO cells exposed in vitro to 10–100 ppm NO_2 (10 min) exhibited increased incidence of chromosome aberrations and sister chromatid exchanges (Tsuda et al. 1981).
"Human, inhalation TC_{Lo} = 100 mg/m³. Effect was not reported. but probably not chronic (General Electric Co. Material Safety Data Sheet #8 1984).
"Rats exposed to I mg/m³ (6 h/d) for I month had increased frequency of chromosome aberrations. No dominant lethal mutations were observed in similarly treated mice (Voroshilin et al. 1975).
"Rats exposed to 470 µg/m³/4 h (I -22 d pregnant) showed unpaired fertility; rats similarly exposed at 49R0 µg/m³/4 h showed impaired fertility and embryotoxicity (*Labor Hyg. and Occ. Dis* 19: 57 1975).
[74]Exposure of *D. melanogaster* (fruit fly) to HF in air increased sterility and sex-linked recessive kthal (SLRL) mutation frequency (Gerdes 1971).
"Rats exposed to 0. I mg/m³ throughout gestation showed embryotoxic and teratogenic effects (Lenchenko and Sharipova 1974).
[76]Inhalation of 2 mg/m³ (7 h/d) by mire (6–15 d pregnant) and rabbits (6- 18 d pregnant) exhibited littk embryotoxicity and no teratogenic effects (Murray et al. 1979).
[77]Exposure of chick embryos from day I to day I4 of development had little effect on survival and no other apparent effects (Hoffman and Campbell 1977).
[78]Malcolm and Paul 1961.
[79]See NO_2 entry under GA combustion.
[80]Ames lest (Moriya et al. 1983).
"Ambiguous mutagenesis responses were obtained in bacterial and fruit fly tests; negative in mouse dominant lethal test (Shapiro 1977).
"Maternal and newborn effects were observed when rats exposed I2 h/d for I2 weeks prior to mating (Shalamberidze and Tsereteli 197 I).
"'Reproductive and embryotoxic effects were observed when mice were exposed during days 6–15 of pregnancy (Food *Cosmetics Toxicol II: 743.* 1980).
"'Trend was observed among women exposed to > 4 µg/m³, but data were not statistically significant (Hemminki and Neimi 1982).
"'Chronic exposure of rabbits. rats. and mice to 100 mg/m³ for S months had minimal effects on cardiovascular system, organ weights. etc. (*ACGIH 4th ed and suppl.* 1980, p. 189)
[86]Rat. inhalation. 3500 ppm/6 h per day for 2 years (intermittent) (Fund. *Appl Toxicol.* 4: *30.* 1984).
"'Ames test. IARC 1979b.
""Mouse. inhalation. 2000 ppm/5 h per day for 2 years (continuous) (National Toxicological Program Technical Report Series. NTP-TR-306. 1986)
@Hamster cells. 5000 ppm/ I h (Jongen et al. 1981).
[90]Human male workers exposed to 104 to *4116* mg/m³ for up to 30 years had an increased incidence of cancer related deaths (Friedlander 1978)
"'Under test for carcinogenicity in the National Toxicology Program.
[92]Ames test (Riccio et al. 1983).
[93]Oral intubation: 47 or 95 mg/d for 78 reeks (Drury and Hammons 1979).
"Rat. inhalation 100 ppm/7 h per day (days 6–15 of pregnancy); rabbit, inhalation 100 or 300 ppm/7 h per day (days 6 18 of pregnancy) (Rao et al 1980).
"Oral intubation, 97 or 195 mg/kg daily for males; 149 or 299 mg/kg daily for females. all for 78 weeks (Drury and Hammons 1979. pp 96 104)
"Human lymphoblastoid cells (Cmp et al. 1985).
[97]Inhalation, S ppm/7 h per day for 78 weeks (intermittent) (Maltoni et al. 1980)
[98]Inhalation, I *SO* ppm/6 h (*Toxicol Appl Pharmacol* 62: 190, 1982).
[99]Inhalation, 14 ppm/4 h per day (6 d/wk for 6 mo) (Vozovaya 1974).
[100]Inhalation exposures, 6 h/d. S d/wk for I y to 875 or 1750 ppm (Rampy et al. 1977).
"'*Ames assay (Developments in Toxicol and Environ. Sciences 2: 249. 1977).*
[102]Animals were treated the I4 days before mating and day I-20th d of pregnancy (*Toxicologist* I: 28. 1981).
'""Animals were exposed by oral gavage S d/week for 78 weeks. Dose levels were: rats-750 and 1500 mg/kg; mice-Z.800 and 5600 mg/kg (National Cancer Inst. 1977).
[104]Animals were treated at days 1–20 of pregnancy (*I Toxicol. Environ Health* 9: 251, 1982).
[105]Oral gavage at 195 and 390 mg/kg/d for 78 weeks (NCI Technical Report Series 1978)
[106]IARC 1979c.
"'Animals were treated via oral gavage at 46 and 96 mg/kg/d for 78 weeks (NCI Technical Report Series 1978).

Table B.20 (continued)

[108] Liver tumors were induced by oral exposure in mice but not in rats (Zeiger 1987).
[109] Arena and Drew 1986 (p. 260).
[110] No tumors were produced in rats exposed to 10 ppm HCl, 6 h/d, 5 d/week for life. Under the same conditions 15 ppm formaldehyde did produce tumors (Sellakumar et al. 1985).
[111] Nondisjunction was produced in *D melanogaster* (fruit fly) exposed to 100 ppm HCl for 24 h (*Theoretical and Applied Genetics* 39: 330, *1969)*
[112] *Akushcherstvo i Ginekologiya* 53: *69. 1977.*
[113] Hughes et al. 1984.
[114] Parts per hundred, effects were not identified; may or may not k chronic (Deichmann 1969. p. 67).
[115] IARC 1980.
[116] Mice, inhalation. 28.500 $\mu g/m^3$ (4 h/d on days 9-12 of pregnancy). Exhibited increased fatal deaths. skeletal defects and chromosome • krralions (Nagymaitenyi et al. 1985).
[117] Ames Test (Andrews et al. 1976).
[118] Rats treated via inhalation 1500 ppm (6 h/d for 50 days) or 2000 ppm (6 h/d for 5 days) before mating. Paternal reproductive effects were observed (HSDB #883, 1987).
[119] IARC 1979d.
[120] Compound is mutagenic in *S. typhimurium, D. melanogaster*, and mammalian cells in vitro IARC 1979d.
[121] *Labor Hyg. Occup. Dis* 24: *2 0* (1980).
[122] Wives of husbands exposed to tkr levels had nearly twice the number of fetal deaths as controls (IARC 1979d).
[123] Workers exposed to occupational levels have increased frequencies of chromosomal aberration (IARC 1979d).
[124] Mice that inhaled 9 ppm (6 h/d. I d/week for I year) exhibited significant increase+ in renal cystademonas (Reichert et al. 1980).
[125] Indirect effect, when acetylene dichloride was buffered or stabilized. no effect was observed in *S. typhimurium* (Reichert et al. 1983).

GB stabilizer TBA yields diButylchloramine and N-Butanoic acid on reaction with STB. No relevant biological data were found for dibutylchloramine. N-Butanoic acid is a mild irritant for human skin (Guest, Katz, and Astill 1982, p. 4914). It can cause mild to severe skin or eye irritation in rabbits depending on the dosage and mode of exposure [Union Carbide Data Sheet 4/10/68; Arch. *Ind. Hygiene and Occ.* Med. 10:61 (1954)).

The toxicity information for VX by-product ethyl methyl phosphonic acid is discussed in Sect. B.5.1.3 on hydrolysis. No relevant biological data were found for N-chlorodiisopropylamine. The toxicities of chloroform, acetic acid, ammonia, and N-chloroisopropylamine are discussed in the preceding paragraphs. The information on the stabilizer DIPC by-products is included in the preceding under GB stabilizers,

Relatively little toxicity information is available for any of the HD by-products other than chloroform, and that is discussed under GB stabilizer DIPC. No relevant biological data were found for 2-chloroethyl vinyl sulfide. No data for relevant routes of exposure were found for divinyl sulfide. In one test for embryotoxicity, divinyl sulfide was reported to have no effect (Glukharev et al. 1980). An LD_{Lo} value of 150 mg/kg in the rat for mustard sulfoxide given by an unreported route of administration is found in Ishidate et al. (1952). No chronic toxicity data were found for this compound. An inhalation LC_{Lo} value of 1430 mg/m^3 over 10 months is found in NDRC (1942) for mustard sulfone in cats and rabbits; no chronic toxicity data were found for this substance. No relevant toxicity data of any kind were found for 2-hydroxyethyl vinyl sulfide.

B.5.3 COMBUSTION PRODUCTS

Products of complete incineration of chemical agents, in addition to carbon dioxide (C02) and water (H_2O), include: for GA, nitrogen dioxide (N02) and phosphorous pentoxide (P_2O_5); for GB, hydrogen fluoride (HF) and P_2O_5; for VX, N02, P_2O_5 and sulfur dioxide (S02); for HD, hydrogen chloride (HCl) and S02; and for Lewisite, chlorine (Cl_2), arsenic trichloride ($AsCl_3$) and arsenic oxides (see footnote e of Table B.18). Elaborate pollution abatement systems are incorporated into the incineration facility design to collect these compounds and to insure that their emissions do not exceed federally regulated standards. However, there are conditions that can result in incomplete agent combustion, with the concomitant generation of intermediate products. The toxicity of all these products is summarized below.

B.5.3.1 Formation

Incomplete combustion of chemical agents can yield products ranging in molecular size and complexity from the parent compound down to the final products indicated above. Insufficient heat and/or air are conditions that will result in incomplete combustion. In the incineration setting, malfunctions such as flameout, temperature

fluctuations, and *emergency* shutdowns could produce such conditions. Pollution abatement systems and safeguards, designed to prevent release of uncombusted agents and intermediate products to the environment, are integral components of the incineration facility. Accidental explosions and/or fires involving chemical agents would also produce conditions favoring incomplete combustion; control of emissions from these causes would be difficult. As in the case of hydrolysis, even incomplete combustion would reduce the agents to less complex and less toxic structures than the parent compounds.

B.5.3.2 Combustion Product Description

Products of complete agent combustion are listed *in* footnote e of Table B.18 and were discussed in Sect. B.5.3. Some of these same products are formed under conditions of incomplete combustion. The compounds most likely to be produced by incomplete combustion are identified in this section. An extensive study of the products of incomplete combustion of the vesicants HD and Lewisite has been made (Brooks and Parker 1979). Similar studies have not been made on the nerve agents; therefore, our list and assessment of the products of incomplete combustion of nerve agents is not as complete. The relative quantities of these products will depend on the temperatures and air availability at the time of combustion, Many will be produced in only trace amounts.

Products of incomplete combustion of GA include hydrogen cyanide (HCN) and P_2O_5. P_2O_5 is a very reactive compound that hydrolyzes upon contact with moisture to form phosphoric acid (H_3PO_4); therefore, under human exposure conditions, P_2O_5 is equivalent to H_3PO_4. Products identified for GB are all equivalent to those that are produced by complete combustion (i.e., H_3PO_4, HF and P_2O_5). The VX products that were identified are essentially equivalent to those of complete combustion. They are nitrogen (N_2), H_3PO_4, sulfuric acid (H_2SO_4) and P_2O_5. Hydration of SO2 in the presence of oxygen produces H_2SO_4.

Several compounds have been identified as products of incomplete combustion of HD (Brooks and Parker 1979). These include sulfur, sulfur dioxide, hydrogen sulfide, ethanethiol, diethyldisulfide, 1,4-dithiane, methylene chloride, chloroform, ethyl chloride, 1,2-dichloroethane, 1,1,1-trichloroethane, 1,1,2-trichloroethane, 1,1,1,2-tetrachloroethane, 1,1,2,2-tetrachloroethane, and hydrogen chloride. The quantity of each compound produced is dependent upon conditions of temperature and air concentration (Brooks and Parker 1979).

Lewisite, like HD, can yield a large number of incomplete combustion products. They include acetylene, acetylene monochloride, arsenic trichloride, arsenic trioxide, chlorine, methyl chloride, vinyl chloride, acetylene dichloride, an unidentified compound with the empirical formula of C_6H_5, arsenic oxychloride (Brooks and Parker 1979), and possibly chlorovinyl arsenous oxide (U.S. Army 1974, p. 71).

Temperature and air content are here again determining 'factors in the proportions of each compound produced (Brooks and Parker 1979).

B.5.3.3 Chronic and Acute Toxicity

The primary route of exposure to agent combustion products is by inhalation. Human exposures would be greatest at the site of combustion and would diminish away from the site in a manner determined primarily by the prevailing atmospheric conditions.

The HCN that would be produced by incomplete combustion of GA is of concern. Its acute toxicity in man is approximately 0.07 times that of GA, the LCt_{50}s being 2000 and 135 mg-min/m^3, respectively (U.S. Army 1974, pp. 81 and 129). No information about the proportion of GA that would be converted to HCN under conditions of incomplete combustion was found. Under usual conditions of humidity, or upon contact with mucous membranes, P_2O_5 would hydrate to form phosphoric acid (see following GB discussion). Nitrogen dioxide is a combustion product of both the nitrogen-containing agents GA and VX. NO2 is lethal to man at an LD_{Lo} of 200 ppm (l-min exposure) (Mason 1974). NO2 is mutagenic in microbial and mammalian cell systems (Isomura et al. 1984). Embryotoxicity was observed when pregnant rats were exposed to NO2 (Gigiena i *Sanitariya* 1977; ***Teratology*** 1984). Human exposure to chronic low levels of NO2 results in pulmonary difficulties associated with emphysema (Doull, Klaassen, and Amdur 1980, pp. 622-625).

Phosphoric acid, via phosphorous pentoxide, is a product of complete combustion of all three nerve agents, including GB. Because it is a strong acid, it can be irritating to the skin and mucous membranes (Windholz et al. 1983, p. 1059). A human inhalation TC_{Lo} of 100 mg/m^3 was reported, but no effects were given (General Electric Company 1984). Inhaled HF is toxic to man and animals. For comparison with the effect of GB, the LC_{50} value for HF in the monkey (Table B.19) converts to an LCt_{50} of about 88,000 mg-min/m^3, whereas the LCt_{50} for GB in the monkey is 74 mg-min/m^3 (U.S. Army 1974, p. 136). Inhaled HF produced chromosome aberrations in rats but did not induce dominant lethal mutations in mice (Voroshilin et al. 1975). HF produced sterility and induced mutations in fruit flies (Gerdes 1971). Reproductive effects, including teratogenesis, were observed in rats exposed to high concentrations of HF.

Nitrogen and sulfuric acid are the only combustion products of VX that are not discussed previously under GA and GB. Air contains about 80% nitrogen (N2). The N2 added to the atmosphere from VX combustion would be inconsequential. As another strong acid, H_2SO_4 can be corrosive to body tissues, and inhalation of high concentrations could produce respiratory problems (Windholz et al. 1983, pp. 1288-1289). Based upon LCt_{50}s, H_2SO_4 is 3000 to 5000 times less acutely toxic to rats and mice than is VX (Izmerov et al. 1982; U.S. Army 1974, p. 168). No effects on reproduction were observed in mice, rabbits, or chicks exposed to H_2SO_4

(Murray et al. 1979; Hoffman and Campbell 1977). NO2 is another product of VX combustion and was previously discussed under GA.

Sulfur, one of the many products of HD combustion, is essentially nontoxic. Sulfur dioxide (SO_2) can be irritating to mucous membranes, in part because it combines with water to produce sulfurous acid. Concentrations of 3000 ppm for brief periods can be fatal to man *(Tabulae Biologicae 1933).* Whether SO2 is mutagenic in common bacterial assay systems has not been settled; it was negative in the mouse dominant lethal test (Shapiro 1977). Reproductive effects were observed in rats and mice exposed to S02. Hydrogen sulfide (H_2S) is a strong irritant (Gosselin 1976, pp. 169-173) and can be lethal to man in the 600 to 800 ppm range (Lefaux 1968, p. *207; Tabulae Biologicae* 1933). In this regard, under certain conditions of incomplete combustion, nearly 15% of the HD was converted to H_2S (Brooks and Parker 1979). No significant chronic toxicity effects were found for H_2S. Ethanethiol is irritating to mucous membranes (Windholz 1983, pp. 540-541). In the rat, an LC_{50} of 4420 ppm/4 h has been reported (Amer. *Indust. Hygiene Assoc. J.* 1958). Chronic exposure to 100 mg/m^3 had no significant systemic effects on rabbits, rats, or mice (ACGIH 1980). No relevant biological data were found for diethyldisulfide or 1,4-dithiane. Large doses (10,000 to 15,000 ppm) of methylene chloride are required to kill experimental animals. Much lower exposures produce behavioral and cardiovascular symptoms in man. Methylene chloride tested positive for carcinogenicity in rats *(Fund. Appl. Toxicol.* 1984) and mice (National Toxicology Program Technical Report Series 1986), and epidemiological data suggest an involvement in human cancer (Friedlander, Hearne, and Hall 1978). However, this compound is probably not of great concern because less than 0.0002% of HD was converted to methylene chloride under the wide range of combustion conditions tested (Brooks and Parker 1979). The toxicity of chloroform is addressed in Sect. 5.2 under Decontamination Products. It should be noted that **maximum** conversion of HD to chloroform was 0.0007% under the combustion conditions tested by Brooks and Parker (1979). The maximum conversion of HD to ethyl chloride was less than 0.008% under the combustion conditions of Brooks and Parker (1979). This, together with the extremely large concentrations required for toxicity, virtually eliminates ethyl chloride as a health hazard associated with HD combustion. Exposures of experimental animals to 1,2-dichloroethane at concentrations from 1000 to 3000 ppm are fatal. Although orally administered 1,2-dichloroethane produces tumors in rats and mice, exposure via inhalation did not (Maltoni et al. 1980). The compound tested positive in both microbial and mammalian cell mutagenesis assays (Riccio et al. 1983; Crespi et al. 1985). Inhalation of 1,2-dichloroethane caused DNA damage *(Toxicol. Appl. Pharmacol.* 1982) and fertility effects in rats (Vozovaya 1974). Inhaled 1,2-dichloroethane had no effect on teratogenicity, embryotoxicity, or reproduction performance in rats or rabbits (Rao et al. 1980). No information about the quantity of 1,2-dichloroethane that might be produced from HD combustion was obtained. 1,1,1-Trichloroethane was not

carcinogenic in rats exposed via inhalation (Rampy et al. 1977) or in rats or mice via oral exposure (National Cancer Institute 1977). It is mutagenic in the Ames assay (Developments in Toxicol. Environ. Sci. 1977). Exposure to 2100 ppm before and/or during pregnancy of rats produced developmental and embryotoxic effects *(Toxicologist* 1981; *J. Toxicol. Environ. Health* 1982). The mouse is more susceptible than the rat to the lethal effects of **1,1,1-trichloroethane** with LC_{50}s of 3911 **ppm/2** h and 18,000 **ppm/4** h, respectively **(***Jap. J. Indust.* Health 1971; **Marhold** 1972). Concentrations greater than **5000** ppm are considered to be life-threatening in humans **(USEPA** 1982). Like other chlorinated hydrocarbons, production of **1,1,1-trichloroethane** from combustion of HD is small and is equal to or less than 0.007%. The 1,1,2 isomer of trichloroethane was carcinogenic in mice but not rats when administered orally **(NCI** Technical Report Series 1978). For the rat, an LC_{50} of 2000 **ppm/4** h and an LC_{Lo} of 500 **ppm/8** h have been reported [Vershueren 1983, p. *606; Am. Indust. Hyg. Assoc. J.* **30:470 (1969)**]. Human exposure to 2000 **ppm/5** min produces equilibrium disturbances and acts as a depressant on the central nervous system (Arena and Drew 1986, pp. 259-260). Like the **1,1,1** isomer, conversion of HD to **1,1,2-trichloroethane** under various combustion conditions is small--less than 0.001% (Brooks and Parker 1979). The **1,1,1,2** and **1,1,2,2** isomers of tetrachloroethane are similar in that by oral administration they produce liver tumors in mice but *not* in rats and are negative for mutagenicity in the *Salmonella* test **(Zeiger** 1987). Except for **1,2-dichloroethane**, no information on the carcinogenic potential of inhaled chlorinated ethanes was found. **Dose-**dependent symptoms including headache, vertigo, nervousness, numbness, narcosis, and tremors are associated with human exposure to 1,1,1,2-tetrachloroethane (NRC 1977, p. 768). Chronic exposure to 1,1,2,2-tetrachloroethane causes nephritis and severe toxic hepatitis with acute atrophy of the liver (Arena and Drew 1986, p. 260). Maximum conversion of HD to tetrachloroethanes, under the various conditions employed by Brooks and Parker (1979) was less than 0.004%. Hydrogen chloride (HCl) is a strong acid and an irritant to skin and mucous membranes at high concentrations. Inhalation can be fatal to experimental animals and humans. **HCl** was not carcinogenic to rats under conditions where formaldehyde was (Sellakumar et al. 1985). Chromosome aberrations were produced in fruit flies exposed to 100 ppm **HCl** for 24 h **(***Theor. Appl. Genet.* 1969). **HCl** applied one day prior to mating produced unspecified reproductive effects in rats *(Akushcherstvo* i *Ginekologiya* 1977).

As for HD, **a** number of potential products of Lewisite combustion have been identified (Table B.18). Acetylene is not very toxic, with LC_{Lo}s in the parts per hundreds (instead of millions) range. Acetylene was negative for mutagenicity in the S. *typhimurium* test (Hughes et al. 1984). No biological effects data were found for acetylene monochloride. The arsenic-containing compounds arsenic trichloride and arsenic trioxide are produced under both incomplete and complete combustion conditions. Arsenic trichloride is the single major combustion product of Lewisite in essentially a one-to-one yield. Arsenic compounds are

recognized human carcinogens (IARC 1980). At relatively low concentrations, 28.5 mg/m^3, arsenic trioxide produced chromosomal aberrations and reproductive effects in mice (Nagymajtenyi et al. 1985). Chlorine gas is acutely toxic to experimental animals and to humans. Chlorine is irritating to mucous membranes and can corrode the teeth (Arena and Drew 1986, pp. 316-318). Methylchloride is acutely toxic only at rather large concentrations and/or exposure periods (HSDB 1987, p. 883). It is mutagenic in the Ames bacterial test (Andrews et al. 1976) and is reported to cause unspecified paternal reproductive effects in rats (HSDB 1987, p. 883). No values for the acute toxicity of vinyl chloride were obtained. Vinyl chloride is a human carcinogen (IARC 1979d). It is mutagenic in various test systems (IARC 1979d). Workers exposed to vinyl chloride have increased frequencies of chromosomal aberrations; wives of workers who were exposed to levels equivalent to 30 mg/m^3/5 years had nearly twice the incidence of fetal deaths as controls (IARC 1979d). Acetylene dichloride is reportedly carcinogenic in mice and mutagenic in the Ames bacterial test (Reichert et al. 1980, 1983). From the data of Reichert et al. (1978), we calculate an LD_{Lo} of approximately 200 ppm/1 h for acetylene dichloride in the rabbit. In that study, renal and hepatic toxicities were observed. The polymeric product C_6H_5, detected but not identified as to structure by Brooks and Parker (1979), is listed for completeness. For obvious reasons, toxicity data for this product could not be located. No data were obtained for arsenic oxychloride; toxic properties similar to those of $AsCl_3$ and As_2O_3 might be expected. Our only information about chlorovinyl arsenous oxide is that it has vesicant properties comparable to those of Lewisite (see Lewisite hydrolysis in Sect. B.5.1.3).

Clearly the acute toxic effects of the gases (e.g., HCN, NO_2, HF, SO_2, and C12) would be of immediate local concern in event of explosion and/or fire involving chemical agents. Of greater concern are the chronic effects, including carcinogenicity, of the arsenic-containing compounds produced from combustion of Lewisite. The chronic health effects associated with the chlorinated hydrocarbons produced from incomplete combustion of HD and Lewisite are tempered in large measure by the fact that they would be produced in trace or small quantities. Under the conditions of temperature and residence time proposed for incinerator operation, no chlorinated'hydrocarbon releases are expected.

B.6 REENTRY CRITERIA FOR AGENT CONTAMINATION OF FOOD, WATER, AND SURFACES

In the event of an unplanned release of agent during handling or transport, a potential exists for contamination of drinking water, forage crops, grains, garden produce, and livestock (either by surface contact or ingestion of contaminated food or water). Persistent agents, such as VX or the mustards, pose the greatest human health concern for reentry.

B.6.1 DISPOSITION OF LIVESTOCK AND FOOD PRODUCTS

Prior to the current analysis, the disposition of food products and livestock originating from an area of unplanned agent release had not been systematically consideredby most civilian agencies with regulatory responsibilities for monitoring crops, livestock and water [e.g., U. S. Department of Agriculture (USDA), U.S. Environmental Protection Agency (EPA), Food and Drug Administration (FDA)]. Each agency received a letter request from CDC/Center for Environmental Health (CEH) in March 1987, requesting assistance in developing guidelines for safe use of agricultural products and water. These discussions are currently in progress but have generated the following general concepts:

filtered prior to consumer use, and leafy vegetables are washed. Documentation for the MPCs recommended in Table B.21 is provided below.

B.6.2.1 Drinking Water Criteria

Existing literature on water criteria is more fully developed than that for foodstuffs, primarily because of the militarily strategic need to determine safe drinking water concentrations for troops performing missions in the field. At present, the U.S. Department of Defense utilizes the following allowable concentration levels established in 1970 by the North Atlantic Treaty Organization (NATO) (Ward 1970): GA at 0.1 mg/L, GB at 0.02 mg/L, VX at 0.02 mg/L, and mustards at 0.20 mg/L. These values assume individual adult consumption at 5 L/d for a period of seven days or less. It is further assumed that the water consumed

analysis of vesicant exposure in drinking water is still in preparation and will not be completed in time to incorporate into the present assessment.

Existing data on differential toxicity and red blood cell (RBC) ChE activity following human and animal exposure to the nerve agents VX, GB, and GA have been considered in developing MPCs for military personnel. Daniels has assumed consumption of 5 to 15 L/d of field water over a 7-d period. The calculated MPC estimates are based on RBC-ChE depression of 50% and 20% of normal baseline (resulting in an RBC-ChE level of 50% and 80% of baseline, respectively; see Tables B.22 and B.23). A 50% lowering of RBC-ChE is considered "a conservative estimate of the threshold above which performance-degrading effects could occur" that would alter the ability of military personnel to perform routine duties (Daniels, in press). Please note that the threshold for headache and irritability in

- Forage, grains, and garden produce could be quarantined until tested [R. Parker at the FDA National Center for Toxicologic Research (NCTR) states that the NCTR laboratories have developed a method for analyzing the capacity of foods to inhibit cholinesterase. Detection limits are 5 μg/sample (Parker 1987)].

Vesicant agents are less likely than nerve agents to induce livestock fatalities. For example, for inhalation exposure among goats, VX is 200 times more potent than mustard; there is a 400-fold difference between VX and mustard potency for percutaneous exposure among dogs (U.S. Army 1974; Dick 1981). However, vesicants can still induce eye damage, blindness, and severe skin lesions. Meat and milk from such animals could contain agent residues from surface contamination unless the animals were thoroughly decontaminated. It is possible that some livestock would be killed, and severely injured animals would need to be destroyed. Residual effects could include permanent respiratory and eye damage as well as scarring of hides, resulting in economic losses for the producer.

An alternate approach is presented by Mershon and Tennyson (1987), who considers it more reasonable to dispose of agent-contaminated agricultural commodities, unless particularly valuable seed stock are affected. Crops considered salvageable would be those present in peripheral areas or which have otherwise become lightly contaminated. The logic here is that a country with abundant food supplies, such as the United States, has no pressing need to place its civilian population at risk from ingesting contaminated food when necessary foodstuffs can be readily transported to affected sites from uncontaminated areas and abundant commodity food stockpiles. If rigidly enforced and mobilized quickly, this approach would eliminate any potential for human contamination via ingestion and reduce the potential for human surface contact with contaminated crops. However, many agricultural resources that would naturally decontaminate with time (on the order of months for VX; Dewey and Fish undated) would be unnecessarily destroyed. In any case, some criteria to distinguish between "contaminated" and "uncontaminated" would still need to be established.

B.6.2 CRITERIA FOR REENTRY BY THE GENERAL PUBLIC

Specific recommendations for determining safe reentry by the general public to an area previously contaminated with agent are presented in Table 8.21 as maximum permissible concentrations (MPCs). Concentrations equal to or less than these should be used to000 determine an "all clear" designation; greater concentrations indicate that unprotected members of the general public should either be denied entry or provided alternate sources of drinking water, air, or food. Development of these recommendations assumes direct consumption of drinking water from the tap, lack of air filtration, and ingestion of raw, unwashed, green leafy vegetables (e.g., lettuce and spinach). These assumptions are considered protective, because most surface water supplies are chlorinated and

filtered prior to consumer use, and leafy vegetables are washed Documentation for the MPCs recommended in Table B.21 is provided below.

B.6.2.1 Drinking Water Criteria

Existing literature on water criteria is more fully developed than that for foodstuffs, primarily because of the militarily strategic need to determine safe drinking water concentrations for troops performing missions in the field. At present, the U.S. Department of Defense utilizes the following allowable concentration levels established in 1970 by the North Atlantic Treaty Organization (NATO) (Ward 1970): GA at 0.1 mg/L, GB at 0.02 mg/L, VX at 0.02 mg/L, and mustards at 0.20 mg/L. These values assume individual adult consumption at 5 L/d for a period of seven days or less. It is further assumed that the water consumed contains no other toxic materials. The basis for selecting these particular maximal values for nerve agents is not described; however, the mustard guideline was stated to be based on data provided by volunteers who ingested 1 to 3 mg of nitrogen mustard (HN3) in drinking water with "development of moderate symptoms but few, if any, casualties" (Ward 1970). The 1-mg dose produced no symptoms "except a feeling of fullness in the stomach."

If one assumes an adult body mass of 70 kg, a l-day exposure, and complete absorption, the stated drinking water concentrations of nerve agent provide doses ranging from 1.4 to 7.1 μg/kg. The cholinesterase-lowering threshold for VX administered intravenously (i.v.) is 0.225 μg/kg over a 30-s administration time in an adult male (Kimura, McNamara, and Sim 1960). Toxic signs of irrationality and bronchospasms are observed when 2.12 μg/kg VX is administered i.v. over a 5.5-h period (Kimura, McNamara, and Sim 1960). It is not appropriate to compare oral and i.v. exposures of nerve agent; mode of entry was the source of major differences in cholinesterase (ChE) lowering observed among human volunteers receiving i.v. or oral doses of VX (Sidell'and Groff 1974). Intravenous exposure produces a 50% ChE depression from individual baseline at roughly 0.5 the dose (1.1 μg/kg) as oral exposure (2.3 μg/kg). This comparison does indicate, however, that current military field water recommendations would benefit from systematic analysis on the basis of a readily observable biological endpoint. Cholinesterase lowering is such an endpoint for comparing nerve agent exposures.

The U.S. Army Medical Research and Development Command at Ft. Detrick, Md., is apparently rethinking the existing Army/NATO guidelines, as evidenced by their current support of an ongoing evaluation of drinking water standards by Lawrence Livermore National Laboratory. The work is in draft form and currently undergoing internal review but will eventually be published as Evaluation *of* Military *Field-Water Quality, Vol.* 4, Criteria *and* Recommendations for *Standards for Chemical Constituents of* Military Concern by Anspaugh et al. (in press). One of the completed chapters (Chapter 10, "Threat Agents, Organophosphato Section") addresses nerve agents (Daniels, in press). An

Table B.21. Recommended maximum permissible concentrations (MPCs) to the public for various media following an unplanned agent release[a]

Agent[b]	Medium			
	Drinking water (μg/L)	Air (mg/m^3)[d]	Garden produce (μg/kg)[e]	Surfaces
VX	1.5	3×10^{-6}	1.4-1.9	Undeveloped
GB (sarin)	2.8	3×10^{-6}	2.6–3.5	Undeveloped
GA (tabun)	2.8	3×10^{-6}	2.6-3.5	Undeveloped
HD/H	Not yet available	1×10^{-4}	Not yet available	Undeveloped
L	Not yet available	3×10^{-3}	Not yet available	Undeveloped

[a]Unprotected members of the general public should not be exposed to concentrations greater than these. Alternate supplies should be provided and quarantine measures taken in any case where greater concentrations are detected.

[b]Agents GA (Tabun) and L (Lewisite) are stockpiled only at Tooele Army Ammunition Depot (TEAD) and arc thus considered a site-specific concern. Estimation of reentry MPCs for these agents will be included in the site-specific document for TEAD.

[c]Derived from Daniels (in press) by method described in accompanying text. Methods have not yet been developed to detect agent concentrations at these levels in water.

[d]Approved by DHHS as a safe, 72-h weighted average, exposure for the general public.

[e]Estimate assumes raw and unwashed produce is consumed and that the agent is evenly distributed over the edible surface. Green, leafy vegetables possess a large scavenging surface area and are recommended samples for monitoring purposes. Analytical methods have not yet been developed to detect these recommended MPCs at the required degree of confidence.

analysis of vesicant exposure in drinking water is still in preparation and will not be completed in time to incorporate into the present assessment.

Existing data on differential toxicity and red blood cell (BBC) ChE activity following human and animal exposure to the nerve agents VX, GB, and GA have been considered in developing MPCs for military personnel. Daniels has assumed consumption of 5 to 15 L/d of field water over a 7-d period. The calculated MPC estimates are based on RBC-ChE depression of 50% and 20% of normal baseline (resulting in an RBC-ChE level of 50% and 80% of baseline, respectively; see Tables B.22 and B.23). A 50% lowering of RBC-ChE is considered "a conservative estimate of the threshold above which performance-degrading effects could occur" that would alter the ability of military personnel to perform routine duties (Daniels, in press). Please note that the threshold for headache and irritability in adults is attained prior to the point at which 50% ChE depression is observed.

Note that blood cholinesterase lowering, the basis for the present analysis, is known to be rate-related. That is, a nerve agent dose administered in small increments over a period of days or weeks can be tolerated without toxic manifestations (Sect. B.2). The same nerve agent dose, administered rapidly over a period of minutes, would have severe or lethal consequences. Ingestion of potentially contaminated water or food is likely to occur over an extended period. The present analysis assumes prompt physiological response and is thus likely to be protective.

If performance criteria for military personnel charged with accomplishing highly technical tasks or operating complex equipment is a consideration, Daniels (in press) recommends the MPCs for 20% ChE inhibition be applied. Daniels acknowledges that these values are quite protective and based on speculation that RBC-ChE lowering to 80% of individual baseline would result in noticeably impaired performance.

Daniels' MPCs are based on sound consideration of nerve agent biological effects. It is reasonable to modify his estimates for application to civilian populations whose drinking water supplies could become contaminated through unplanned agent releases. It is assumed that most adults consume 2 L water/d [the usual adult intake as estimated by Snyder et al. (1975)]; thus, an estimate of safe agent intake based on 5 L water/d would be protective. It is also known that threshold ChE depression is noted at a dose equal to 20% of the dose observed for 50% ChE depression [threshold ChE lowering-has been obtained at an i.v. dose of 0.225 μg/kg, and 50% ChE lowering has been observed at an i.v. dose of 1 μg/kg (Kimura, McNamara, and Sim 1960)]. If we incorporate an additional adjustment of 0.5 to accommodate anemic individuals in the general population (a maximal estimate of RBC mass reduction for victims of anemia is 50% of normal) (S. S. Leffingwell, Center for Environmental Health, DHHS, Atlanta, Ga., letter to A. P. Watson, Health and Safety Research Division, ORNL, June 25, 1987), the overall adjustment to Daniels' recommended MPCs in Table B.22 for 50% ChE depression would

Table B.22. Maximum permissible concentrations (MPCs) for nerve agents, based on RBC-ChE activity's not being depressed below 50% of normal in 70-kg military personnel[a]

Nerve agent	Consumption rate and corresponding MPC (μg/L)[b]	
	5 L/d	15 L/d
VX	15	5.0
GB (sarin)	28	9.3
GA (tabun)[c]	140	46

[a]Daniels, J. I. (in press). "Threat Agents through 10-30) in *Evaluation* of *Military Field-Water* Qualify, Vol. 4: Criteria *and Recommendations for Standards for Chemical Constituents of Military Concern.* Lawrence Livermore National Laboratory, University of California, Livermore, CA 94550. Draft document for Project Order 82PP2817 supported by the U.S. Army Medical Research and Development Command, Ft. Detrick, MD 21701.

[b]Field-water consumption rate for up to 7 d.

[c]Agent GA is stockpiled only at Tooele Army Ammunition Depot (TEAD) and is included here for the sake of completeness.

Table B.23. Maximum permissible concentrations (MPCs) for nerve agents, based on RBC-ChE activity's not being depressed below 20% of normal (e.g., resulting in 80% of normal baseline) in 70-kg military personnel[a]

Nerve agent	Consumption rate and corresponding MPC (μg/L)[b]	
	5 L/d	15 L/d
VX	6.0	2.0
GB (sarin)	II	3.7
GA (tabun)[c]	56	18

[a]Daniels, J. I. (in press). 'Threat Agents," Chapter 10 (pp. IO- 1 through in *Evaluation of Military Field-Water* Quality, *Vol.* 4: Criteria *and Recommendations for Standards for Chemical Constituents* of *Military Concern.* Lawrence Livermore National Laboratory, University of California, Livermore, CA 94550. Draft document for Project Order 82PP28 17 supported by the U.S. Army Medical Research and Development Command, Ft. Detrick, MD 21701.

[b]Field-water consumption rate for up to 7 d.

'Agent GA is stockpiled only at Tooele Army Ammunition Depot (TEAD) and is included here for the sake of completeness.

equal 0.1 (i.e., 0.20 x 0.50). This safety factor provides a protective estimate of threshold effects. The resulting modified MPCs suggested for consideration as civilian drinking water criteria are provided in the first column of Table B.21. Development of these values assumes no other source of agent exposure (i.e., food and air). An analysis of potential cumulative food and water exposure is presented in Sect. B.6.2.4.

The detection limits of current analytical methods (Hines 1984) would have to be improved by a factor of 10 to monitor the recommended MPCs with any consistency. Although considered theoretically possible, such techniques for *nerve* agents and mustard have not yet been demonstrated. It is recommended that an Army research and development program be initiated by the appropriate command(s) to develop necessary methods for reliably detecting agents at estimated safe MPC levels.

B.6.2.2 Air Criteria

Maximum permissible concentrations for air (Table B.21) are those previously approved as safe for general public exposure by CDC (DHHS). These estimates incorporate safety factors of at least two orders of magnitude and should be measured as an average over a 72-h sample period.

B.6.2.3 Garden Produce Criteria

Although limited, studies of plant uptake from hydroponic soiutions containing radiotagged VX indicate little agent translocation to leaves, stems, or fruit. Study of a variety of species [the ornamental house plants coleus (Coleus *blumei* Benth.), wandering jew (Zebrina *pendula* Schnizl.), arrowleaf (*Maranta bicolor* Kev.) and petunia (*Petunia hybrida* Vilm.), and the garden vegetable tomato (Lycopersicon *esculentum* Mill.)] demonstrated that the maximal transfer (7.8%) of tagged VX from root uptake took place in the fruit and seed of petunia after a 42-d growing period (Worthley 1971). In tomato, the only food crop plant examined, maximal activities were detected after 12-d growth; 1.4% and 2.9% of the tagged VX was observed in roots and leaves respectively. Tomato stem activity was 0.8% at the same harvest date. No tomato flowers or fruit had developed by the 42nd day of growth, when the experiment was terminated (Worthley 1971). These data indicate that any agent ingestion hazard would be more likely to be a result of surface contamination than translocation.

Estimating MPCs for garden produce considers that crops possessing a large surface area and that are usually eaten raw offer the greatest potential for surface contamination. Classic examples are leaf spinach and leaf lettuce, particularly the savoy varieties, which present a "scavenging" surface. The water content of spinach and lettuce exceeds 90% [92.7% for spinach and 94.8% for lettuce (Spector 1956)]; the first of the two recommended MPCs per agent for garden produce in Table B.21 was calculated by assuming that the water portion of these crops should contain no more than the previously documented protective MPC for

drinking water. An alternate approach is to consider that the body burden of agent imposed by consuming fresh garden produce should be no greater than that generated by consumption of water at estimated reentry criteria levels. Thus, one day's consumption (assume 2.0 L) of contaminated water at 1.5 μg VX/L would provide a daily intake of 3.0 μg [in the unlikely event of complete absorption into the bloodstream, this concentration would be equivalent to a dose of 0.04 μg/kg in an adult; this is considered the threshold for headache and irritability. No RBC/ChE depression is noted at this dose of VX (Kimura, McNamara, and Sim 1960).] If we further assume the average daily consumption of food in the United States to be 1.6 kg (Snyder et al. 1975), the concentration of VX in garden produce that could be daily ingested with no toxic response would be

$$\frac{3\ \mu g}{1.6\ kg} = 1.87\ \mu g/kg\ .$$

This analysis assumes that exposed individuals are vegetarians and consume only unwashed garden produce. A similar analysis with agent GB generated the value of 3.5 μg/kg presented in Table B.21. Development of these values assumes no other source of agent (e.g., water and air). An analysis of potential cumulative food and water exposure is presented in Sect. B.6.2.4.

It is not known whether these recommended MPCs can be detected at the required level of confidence; chemical analysis of garden produce for microgram quantities of agent has never been performed to any extent. It is again recommended that an Army research and development program be initiated to develop appropriate analytical methods. Crops contaminated to a greater extent should be quarantined or destroyed.

B.6.2.4 Food + Water Ingestion Criteria

Several transport accident scenarios (e.g., spills) describe unplanned releases to water bodies used for public water supplies. In such cases, only the reentry criteria for drinking water proposed in Table B.21 would be pertinent because contamination of food crops would be minimal during most spills into streams or lakes. Nevertheless, some accident scenarios involving explosive release could include surface contamination of crops *and* public water supplies. In such cases, we recommend that the total daily intake from combined food and water not exceed the body burden obtained by consuming 2 L of water at the MPCs presented in Table B.21.

For VX, this daily total body burden would equal 3 μg (i.e., 1.5 μg/L x 2 L/d = 3 μg/d). For GB, this daily total body burden would equal 5.6 mg (i.e., 2.8 μg/L x 2L/d = 5.6 μg/d). Paired values of estimated safe water and food concentrations represent a continuum of solutions to straight line equations. An example follows.

If the daily intake of VX should not exceed 3.0 μg/d, and adult consumption of water and food are 2 L/d and 1.5 kg/d, respectively (Snyder et al. 1975), then

$$2\ \text{L/d}\ (x) + 1.6\ \text{kg/d}\ (y) = 3\ \mu\text{g/d}\ ,$$

where

$$x = \text{VX water concentration in } \mu\text{g/L}\ ,$$

$$y = \text{VX food concentration in } \mu\text{g/kg}\ .$$

If 5 L water are consumed per day, the equation becomes

$$5\ \text{L/d}\ (x) + 1.6\ \text{kg/d}\ (y) = 3.0\ \mu\text{g/d}\ ,$$

with x and y defined as above. Any combination of food and water concentrations may be evaluated, as long as the sum does not exceed 3.0 μg/d.

Similar analysis for GB provides the following:

$$2\ \text{L/d}\ (x) + 1.6\ \text{kg/d}\ (y) = 5.6\ \text{mg/d}\ ,$$

$$5\ \text{L/d}\ (x) + 1.6\ \text{kg/d}\ (y) = 5.6\ \text{mg/d}\ .$$

An application of this approach can be illustrated. If an individual consumes 2.0 L/d of WC-contaminated tap water at 0.5 μg VX/L, he or she could also consume 1.6 kg food per day at 1.25 μg VX/kg or 2 kg food per day at 1.0 μg VX/kg, etc.

(1) $2\ \text{L/d}\ (0.5\ \mu\text{g/L}) + 1.6\ \text{kg/d}\ (y) = 3\ \mu\text{g/d}$

$$y = \frac{2\ \mu\text{g}}{\text{d}} \times \frac{\text{d}}{1.6\ \text{kg}} = 1.25\ \mu\text{g/kg}\ ,$$

(2) $2\ \text{L/d}\ (0.5\ \mu\text{g/L}) + 2.0\ \text{kg/d}\ (y) = 3\ \mu\text{g/d}$

$$y = \frac{2}{\text{d}} \times \frac{\text{d}}{2\ \text{kg}} = 1.0\ \mu\text{g/kg}\ .$$

For adult individuals consuming average daily intakes [2 L/d water and 1.6 kg/d food (Snyder et al. 1975)] of a contaminated diet, water

should contain no more than 0.85 μg VX/L or 1.56 μg GB/L and food should contain no more than 0.83 μg VX/kg or 1.56 μg GB/kg.

B.6.2.5 Contaminated Surfaces Criteria

Guidelines for contact with contaminated surfaces (e.g., building materials, fabrics, metal, glass, etc.) are a particular concern in the event of an off-site transportation accident. However, such guidelines are relatively undeveloped. Present Army regulations require that any object whose surface is potentially contaminated by lethal agents be decontaminated to a "5X" standard before it can be released from military custody to the public (U.S. Army 1982). The 5X state is achieved by heating the (disassembled) object and holding it at a minimum temperature of 1000°F (538°C) for 15 min. Assembled objects are required to be held at the minimum temperature for longer time periods. While suitable for many metal objects, this treatment would result in destruction of or severe damage to wood, plastics, and fibers. Additional problems might be encountered in gaining possession of objects to be "5X'd" and in determining which articles have actually been contaminated.

Decision criteria for decontaminating military hardware for military use are currently based on analysis of solvent-extracted swab samples or analysis of air drawn over a potentially contaminated item enclosed in a plastic bag to provide a "yes/no" indication of agent presence. Current, best available technology for certifying rooms in demilitarization facilities as free of vapor contamination makes use of replicate air samples after the questionable area has been sealed to prevent air inflows. At a standard operating temperature of 21°C (70°F), three 12-h samples are pulled through existing instrumentation. If the measured agent concentration in air is at or below the (general public) MPC for air presented in Table 8.21 (e.g., 3×10^{-6} mg/m^3 for VX and GB and 1×10^{-4} mg/m^3 for mustards), the area is certified as safe to enter by unprotected operations personnel. Because of the total lack of available data correlating agent concentrations in sample air with the degree of surface contamination, it is not yet possible to certify this procedure as an adequate decision criterion for unlimited public access.

There are no data available to use in correlating the amount of agent on or absorbed into any given surface with the concentration observed in off-gases. Therefore, it is considered prudent to prohibit access to contaminated buildings and their contents by the general public until the area can be determined to be acceptably safe. This time period is currently indefinite, given that the most sensitive form of commercially available off-gas analysis is unable to detect when porous surfaces such as wood, fabric, or plastic are free of agent contamination. Desorption of agent from contaminated porous surfaces could continue over time by means of elevated temperature *or* passive diffusion (Trapp 1985). This is an obvious area for further investigation.

DEFINITION OF TERMS

Acetylcholine (ACh): a choline derivative, found in many parts of the body, that plays an important role in the transmission of an impulse from one nerve fiber to another.

Acetylcholinesterase (AChE): an enzyme that hydrolyzes acetylcholine into acetic acid and choline, thereby preventing excess buildup of the neurotransmitter and regulating the transmission of nerve impulses.

Additive effects: the combined action of two agents, being equal to the sum of the effects of each used alone. For comparison, see Potentiation.

Ames test: the use of certain specific strains of Salmonella *typhimurium*, with and without metabolic activation, to detect gene mutations.

Basal cells: skin cells between epidermis and dermis: the epidermal layer from which the remainder of the epidermis is generated.

Bioassay: determining the activity of an agent by noting its effect on a live animal, an isolated organ preparation, or a cell line (as opposed to assaying activity by noting changes to a chemical preparation).

Bowen's disease: intraepidermal squamous cell carcinoma (Friel 1974, p. 222).

Bronchitis: inflammation of the bronchi (i.e., larger air passages of the lung).

Carcinogenesis: the production of malignant neoplasms.

Carcinoma: a malignant new growth made up of epithelial cells (Friel 1974, p. 261).

CHO forward mutation assay: an assay using as an endpoint mutation at a specific (enzyme) genetic site (locus) in Chinese hamster ovary (CHO) cells.

Conjunctivitis: inflammation of the conjunctiva (membranes that line the eyelids) (Friel 1974, p. 352).

Cornea : the clear part of the outer layer. of the eye.

Cytogenetic analysis: assay utilizing cultured cells or cell lines to assay for chromosomal aberrations following an administration of chemical substances (Tatken and Lewis 1983, p. xxvii).

Cytology: the study of cells.

Delayed neuropsthy: degeneration of certain peripheral nerves at some delayed time (days to weeks) after causative agent exposure.

Dermis: layer of skin below the epidermis.

DNA damage: damage to DNA strands, including strand breaks, crosslinks, and other abnormalities (Tatken and Lewis 1983, p. xxvii).

Dominant lethal mutation: a genetic change in a gamete that kills the zygote produced by that gamete. In mammals, the dominant lethal test measures the reduction of litter size. In insects, it measures the number of unhatched eggs (Tatken and Lewis 1983, p. xxvii).

ECt50: the dosage (airborne concentration of the agent multiplied by the time of exposure) that produces some effect in 50% of exposed, unprotected personnel or animals at some given breathing rate. The unit used by the Army to express ECt50 is milligram-minutes per cubic meter ($mg\text{-}min/m^3$) (U.S. Army 1975).

Electroencephalogram (EEG): the tracings of brain waves recorded by an instrument (electroencephalograph) designed to measure the brain's electrical currents.

Emphysema: a disease characterized by over-accumulation of air in lungs and caused by degeneration of lung tissue. (Friel 1974, p. 510).

Epithelial: pertaining to the cells covering internal and external body surfaces.

Epidermis: outermost layers of skin.

Erythema: redness of the skin.

Fetotoxicity: toxicity to the unborn offspring (e.g., low birth weight, fetal deaths).

Hematopoietic system: tissues that produce or develop blood cells.

Heritable translocation: pertaining to a measure of the transmissibility of induced translocations to subsequent generations. In mammals, the heritable translocation test assays sterility and reduced fertility in the progeny of the treated parent as endpoints. In addition, cytological analysis of the F1 (first generation) progeny of the treated parent is performed to prove the existence of the induced translocation. In *Drosophila,* heritable translocations are detected genetically using easily distinguishable phenotypic markers, and these translocations can be verified with cytogenetic techniques (Tatken and Lewis 1983, p. xxvii).

Incapacitating dosage (ICt_{50}): the incapacitating dosage of a chemical agent is generally expressed as the median incapacitating dosage (i.e., the amount of agent that is sufficient to disable 50% of exposed, unprotected personnel). For inhalation effect, the median incapacitating dosage is expressed as the ICt_{50}. Incapacitating dosages vary in accordance with the protection provided by masks and clothing worn by personnel and by the breathing rate. The unit used to express ICt_{50} is milligram-minutes per cubic meter ($mg\text{-}min/m^3$) (U.S. Army 1975).

Interstitial: situated within the connective tissue that surrounds and binds organs and other tissues.

Intravenous: administration directly into the bloodstream via a vein.

Iritis: inflammation of the iris (Friel 1974, p. 798).

In vitro: refers to experiments performed in glass, using chemical or cellular test systems.

In vivo: refers to experiments performed with live animals as test systems.

Keratitis: inflammation of the cornea (Friel 1974, p.815).

Laryngitis: inflammation of the larynx.

Larynx: the region of the respiratory tract at the top of the trachea and below the root of the tongue (Friel 1974, p. 838).

LCt_{50}: the dosage (airborne concentration of the agent multiplied by the time of exposure) that is lethal to 50% of exposed, unprotected personnel or animals. The unit used to express LCt_{50} is milligram-minutes per cubic meter ($mg\text{-}min/m^3$) (U.S. Army 1975).

LD50: a calculated dose of a substance that is expected to cause the death of 50% of an entire defined experimental animal population. It is determined from the exposure to the substance, by any route other than inhalation, of a significant number of animals from that population (Tatken and Lewis 1983, p. xxxii).

Lymphoma forward mutation: mutation at a specific genetic locus in lymphoma (transformed leukocyte) cells.

Mucocutaneous: referring to junctions of mucous membranes with skin (e.g., mouth).

Mutation: any heritable change in genetic material (Tatken and Lewis 1983, p. xxvii).

Necrosis: death of tissue, usually as individual cells.

Percutaneous: absorption through the skin (not injected).

Pharynx: area between mouth and larynx.

Phenotypic: as related to mutation, a change in the observable appearance of an organism.

Pleural: referring to the membrane around the lungs and lining the rib cage.

Potentiation: the combined action of two agents, being greater than the sum of the effects of each used alone. For comparison, see Additive Effects.

Rhinitis: internal inflammation of the nose.

Sex chromosome loss and nondisjunction: a bioassay that measures the nonseparation of homologous chromosomes at meiosis and mitosis (Tatken and Lewis 1983, p. xxvii).

Sex-linked lethal mutation: an alteration of genetic material in the sex chromosome of the gamete that produces such a severe effect that all zygotes of a single gender fail to live (or hatch).

Sister chromatid exchange: interchange of DNA in cytological preparations of metaphase chromosomes between replication products at apparently homologous loci (Tatken and Lewis 1983, p. xxvii).

Specific locus test: bioassay utilizing a method for detecting and measuring rates of mutation of any or all of several recessive loci (Tatken and Lewis 1983, p. xxvii).

Squamous: scaly or platelike (Friel 1974, p. 1462).

Subcutaneous: administration of a substance under the skin.

Teratogenicity: the production of physical defects in offspring in utero after maternal exposure (Friel 1974, p. 1549).

Tracheitis: inflammation of the trachea (airway from lower larynx to the division into right and left main bronchi).

Unscheduled DNA synthesis: synthesis of DNA during usually nonsynthetlc phases (Tatken and Lewis 1983, p. xxvii).

REFERENCES FOR APPENDIX B

Abernethy, D. J., Frazella, J. H., and Boreiko, C. J. 1982. "Effects of Ethanol, Acetaldehyde, and Acetic Acid in the C3H/10T1/2 Cl 8 Cell Transformation System," *Environ. Mutagen. 4,* 331.

Abou-Donia, M. B. 1981. "Organophosphorus Ester-Induced Delayed Neurotoxicity," *Ann. Rev. Pharznacol. Toxicol.* 21, 511-48.

Adams, G. K., III, Yamamura, H. I., and O'Leary, J. F. 1976. "Recovery of Central Respiratory Function Following Anticholinesterase Intoxication," *Eur. J. Pharmacol. 38,* 101-12.

Adelson, L. 1962. "Fatal Intoxication with Isopropyl Alcohol (Rubbing Alcohol)," *Am. J. Clin. Pathol. 38,* 144-51.

Ainsworth, M. 1954. *Some Factors Affecting* the *Percutaneous Toxicity of GB*, Porton Technical Paper 426 (Secret), as quoted in Fielding 1960.

Amacher, D. E., and Paillet, S. C. 1979. "Point Mutations at the Thymidine Kinase Locus in L5178Y Mouse Lymphoma Cells. I. Application to Genetic Toxicological Testing," Mutation Res. 64, 391-406.

American Conference of Governmental Industrial Hygienists (ACGIH) 1971. "Ethyl Chloride," p. 106 in *Documentation of the Threshold Limit Values for* Substances *in Workroom Air,* 3rd ed., Committee on Threshold Limit Values, Cincinnati.

American Conference of Governmental Industrial Hygienists (ACGIH) 1980. Threshold Limit Values, 4th ed. and Supplement, ACGIH Cincinnati, pp. 91, 189.

Ames, B. N., Magaw, R., and Gold, L. S. 1987. "Ranking Possible Carcinogenic Hazards," *Science 236,* 271-80.

Anderson, D., et al. 1984. "The Reproductive Genotoxicity of Ethylene Glycol Monomethyl Ether (EGME) in Male Rats and Mice After Single Oral Doses," *Environ.* Mutagen. 6, 390.

Andrews, A. Q., Zawistowski, E. S., and Valentine, C. R. 1976. "A Comparison of the Mutagenic Properties of Vinyl Chloride and Methyl Chloride," *Mutat. Res. 40, 273-76.*

Anonymous 1982. "Mutagenesis Testing Results,'; *NTP Technical Bulletin 7, 5-9.*

Anslow, W. P., et al. 1948. "The Intravenous, Subcutaneous and Cutaneous Toxicity of Bis (Beta-Chloroethyl) Sulfide (Mustard Gas) and of Various Derivatives," *J. Pharmacol. and Expt. Therapeutics 93,* l-9.

Anspaugh, L. R., et al. In press. *Evaluation of Military Field-Water* Quality, *Vol. 4.* Criteria *and* Recommendations for *Srandards for Chemical Constituents of Military Concern.* Lawrence Livermore National Laboratory, University of California, Livermore, CA 94550. Draft document for Project Order 82 PP 2817 supported by the U.S. Army, Medical Research and Development Command, Ft. Detrick, Md. 21701.

Antonova, V. I., and Salmina, Z. A. 1978. "Maximum Permissible Concentration of Isopropyl Alcohol in Water Bodies Taking into Account Its Effect on the Gonads and Progeny," *Gigiena* i *Sanitariya* 43(1), 8-11 (Russian with English summary).

Aposhian, H. V., et al. 1984. "DMSA, DMPS, DMPA--As Arsenic Antidotes," *Fund. Appl.* Toxicol. 4, S58-70.

Arena, J. M., and Drew, R. H., eds. 1986. *Poisoning--Toxicology, Symptoms and Treatment,* 5th ed., Charles C. Thomas, Springfield, Ill., pp. 188-192, 192-194, 212-214, 259-260, 290-292, 313-314, and 316-318.

Ariens, A. T., et al. 1969. "Reversible Necrosis at the End-Plate Region in Striated Muscles of the Rat Poisoned with Cholinesterase Inhibitors," *Experentia* 25(1), 57-59.

Arsura, E. L., et al. 1987. "Adverse Cardiovascular Effects of Anticholinesterase Medications," Am. *J. Med. Sci.* 293(1), 18-23.

Auerbach, C. H. 1950. "Differences Between Effects of Chemical and Physical Mutagens," *Pubbl. della Stazione Zoologica di Napoli 22* Suppl., l-21.

Auerbach, C., and Robson, J. M. 1944. "Production of Mutations by Ally1 Isothiocyanate," *Nature* 154, 81.

Auerbach, C., and Robson, J. M. 1946. "Chemical Production of Mutations" (Letter), *Nature 157. 302.*

Auerbach, C., and Robson, J. M. 1947a. "Tests of Chemical Substances for Mutagenic Action," *Proc. Royal Society of Edinburgh* 62B, 284-91.

Auerbach, C., and Robson, J. M. 1947b. "The Production of Mutations by Chemical Substances," *Proc. Royal* Society *of Edinburgh* 62B, 271-83.

Augustinsson, K. B., and Heimburger, G. 1954. "Enzymatic Hydrolysis of Organophosphorus Compounds. I. Occurrence of Enzymes Hydrolyzing Dimethyl-amino-ethoxy-phosphoryl cyanide (Tabun)," *Acta Chem. Scand.* 8, 753-61.

Back, K. C., Thomas, A. A., and MacEwen, J. D. 1972. *Reclassification of Material Listed as Transportation Health Hazards,* TSA-2072-3, Office of Hazardous Materials, Office of the Assistant Secretary for Safety and Consumer Affairs, Dept. of Transportation, PB-214 270/1.

Bajgar, J. 1971. "Inhibition of Acetylcholinesterase in Different Parts of the Brains of Mice by Isopropyl Methylphosphonofluoridate In Vitro and In Vivo," *Arch, Toxikol. 27,* 233-41.

Bajgar, J. 1972. "Inhibition of Acetylcholinesterase in Rat Brain Subcellular Fractions Following O-Isopropyl Methylphosphonofluoridate Intoxication," Toxicol. *Appl.* Pharmacol. 22, 93-96.

Bakhishev, G. N. 1975. "Relation Between the Chemical Structure and Toxicity of Some Halogenated Aliphatic Hydrocarbons," Fiziol. *Akt. Veshchestva* 7, 35-36, Russian.

Ball, C. R., and Roberts, J. J. 1971/1972. "Estimation of Interstrand DNA Cross-linking Resulting from Mustard Gas Alkylation of HeLa Cells," *Chem.-Biol.* Interact. 4, 297-303.

Barr, S. J. 1985. "Chemical Warfare Agents," *Topics* in Emergency Medicine 7(1), 62-70.

Baxley, M. N., et al. 1980. "Effects of Oral Sodium Arsenite on Mouse Development," *Teratology* 21, 27A.

Baxley, M. N., et al. 1981. "Prenatal Toxicity of Orally Administered Sodium Arsenite in Mice," *Bull. Environ. Contam. Toxicol. 26,* 749-56.

Beal, D. D., et al. 1976. "Effects of 5-(3,3-Dimethyl-1-triazeno) imidazole-4-carboxamide and its Metabolics on Novikoff Hepatoma Cells," *Cancer Res. 36,* 2827-31.

Beebe, G. 1960. "Lung Cancer in World War I Veterans: Possible Relation to Mustard-Gas Injury and 1918 Influenza Epidemic," *J. Natl. Cancer* Inst. 25, 1231-52.

Berenblum, I. 1929. "The Modifying Influence of Dichloroethyl Sulphide on the Induction of Tumors in Mice by Tar," *J. Path. and Bact. 32,* 425-34.

Berenblum, I., and Shubik, P. 1949. "An Experimental Study of the Initiating Stage of Carcinogenesis, and a Re-examination of the Somatic Cell Mutation Theory of Cancer," *Brit. J. Cancer 3,* 109-18.

Blanchet, G., et al. 1986. "Modulation of the Number of Muscarinic Receptors in Mouse Neuroblastoma Cells by Soman, " *Biochem. Pharmacol.* 35(22), 4077-81.

Blewett, W. 1986. "Tactical Weapons: Is Mustard Still King?," *NBC Defence and Technology International* 1(3), *64-66.*

Bodansky, 0. 1945, "Contributions of Medical Research in Chemical Warfare to Medicine," *Science* 102, 517-21.

Boskovic, B. 1981. "The Treatment of Soman Poisoning and its Perspectives," *Fund. and Appl. Toxicol. 1,* 203-13.

Boskovic, B., et al. 1981. "Acute Toxicity of Sarin and VX Administered Simultaneously and the Protective Effect of Oximes and Atropine," *Navcno-Teh. Pregl.* 31, 39-45.

Boskovic, B., Kovacevic, V., and Jovanovic, D. 1984. "PAM-2 Cl, HI-6, and HGG-12 in Soman and Tabun Poisoning," *Fund. and Appl. Toxicol.* 4, S106-15.

Brimblecombe, R. W., French, M. C., and Webb, S. N. 1979. "Effects of Certain Muscarinic Antagonists on the Actions of Anticholinesterases on Cat Skeletal Muscle," *Br. J. Pharmac. 65,* 565-71.

Brookes, P., and Lawley, P. D. 1961. "The Reaction of Mono- and Di-Functional Alkylating Agents with Nucleic Acids," Biochem. *J. 80, 496-503.*

Brooks, M. E. and Parker, *G. A.* 1979. *Incineration/Pyrolysis of* Several *Agents and Related* Chemical *Materials Contained in Identification Sets,* ARCSL-TR-79040, Aberdeen, Md.

Brusick, D. 1986. "Genotoxic Effects in Cultured Mammalian Cells Produced by Low pH Treatment Conditions and Increased Ion Concentrations," *Environ. Mutagen. 8,* 879-86.

Buckley, R. D., and Balchum, 0. J. 1965. "Acute and Chronic Exposures to Nitrogen Dioxide," *Arch. Environ.* Health 10, 220-23.

Burchfiel, J. L., Duffy, F. H., and Sim, V. M. 1976. "Persistent Effects of Sarin and Dieldrin upon the Primate Electroencephalogram," *Toxicol.* and *Appl. Pharmacol. 35, 365-79.*

Calesnick, B., Christensen, J. A., and Richter, M. 1967. "Human Toxicity of Various Oximes 2-Pyridine Aldoxime Methyl Chloride, Its Methane Sulfonate Salt, and 1,1'-Trimethylenebis-(4-Formylpyridinium Chloride)," Arch. *Environ. H.* 15, 599-608.

Callen, D. F., et al. 1980. "Cytochrome P-450 Mediated Genetic Activity and Cytotoxicity of Seven Halogenated Aliphatic Hydrocarbons in *Saccharomyces cereviciae," Mutat. Res. 77, 55-63.*

Cameron, G. R., Carleton, H. M., and Short, R. H. D. 1946. "Pathological Changes Induced by Lewisite and Allied Compounds," *J. of Pathol. Bacteriol.* 58, 411-22.

Capizzi, R. L., et al. 1973. "A Host-Mediated Assay for Chemical Mutagens Using the L5178Y/Asn-Murine Leukemia," *Mutat. Res. 21, 6.*

Carter, Jr., W. H., Jones, D. E., and Carchman, R. A. 1985. "Application of Response Surface Methods for Evaluating the Interactions of Soman, Atropine, and Pralidioxime Chloride ," *Fund. and Appl.* Toxicol. 5, S232-41.

Casadei, E., et al. 1984. "Mantakassa: an Epidemic of Spastic Paraparesis Associated with Chronic Cyanide Intoxication in a Cassava Staple Area of Mozambique: 2. Nutritional Factors and Hydrocyanic Acid Content of Cassava Products," *Bull.* WHO 62, 485-92.

Casale, G. P., Cohen, S. D., and Dicapua, R. A. 1983. "The Effects of Organophosphate-Induced Cholinergic Stimulation on the Antibody Response to Sheep Erythrocytes in Inbred Mice," Toxicol. *Appl. Pharmacol.* 68, 198-205.

Case, R., and Lea, A. 1955. "Mustard Gas Poisoning, Chronic Bronchitis and Lung Cancer. An Investigation into the Possibility that Poisoning by Mustard Gas in the 1914-18 War Might Be a Factor in the Production of Neoplasia," *British J. Pres. Coc. Med. 9, 62-72,* as cited in IARC 1975, *Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Man from Aziridines, N-, S-, and O-Mustards and* Selenium, *Vol.* 9, World Health Organization, Geneva, Switzerland.

Catalini, N., et al. 1971. "Comparative Study of the Biological Effects of X-Rays and of Mustard Gas (beta-beta Dichloro diethylsulfide) on the Thymus of Young Rats," *atti Della Academia Nazionale dei Lincei, classi di* Scienzi Fisichi 51, 81-92.

Cetkovic, S., et al. 1984. "Effect of PAM-2 Cl, HI-6, and HGG-12 in Poisoning by Tabun and Its Thiocholine-like Analog in the Rat," Fund. and *Appl. Toxicol. 4,* S116-23.

Chen, S.-M., and Chi, M.-G. 1986. "Direct Effect of VX and Soman on Nicotinic Receptors," *Acta. Pharmacologica Sinica* 7(5), 401-06.

Christen, P. J., and Cohen, E. M. 1969. "Binding of (32)P-Sarin to Esterases and Other Proteins in Plasma from Rat, Man and Guinea-Pig," *Acta* Physiologica *et* Pharmacologica Neerlandica 15, 36-37.

Cipollaro, M., et al. 1986. "Sublethal pH Decrease May Cause Genetic Damage to Eukaryotic Cell: A Study on Sea Urchins and ***Salmonella typhimurium,"*** *Teratog. Carcinog. Mutagen. 6, 275-87.*

Clarkson, T. W., et al. 1981. "Tests of Efficacy of Antidotes for Removal of Methylmercury in Human Poisoning During the Iraq Outbreak," *J. Pharmacol. Exp. Ther.* 218, 74-83, as cited in Aposhian, H. V., et al. 1984. ***Fund. Appl. Toxicol. 4,*** S58-70.

Clement, J. G. 1981. "Toxicology and Pharmacology of Bispyridinium Oximes --Insight into the Mechanism of Action vs Soman Poisoning In Vivo ," ***Fund. and Appl. Toxicol.*** 1, 193-202.

Clement, J. G. 1985. "Hormonal Consequences of Organophosphate Poisoning," ***Fund. Appl. Toxicol. 5, 561-77.***

Clement, J. G., and Copeman, H. T. 1984. "Soman and Sarin Induce a Long-Lasting Naloxone-Reversible Analgesia in Mice," Life Sci. 34(15), 1415-22.

Clive, D., et al. 1979. "Validation and Characterization of the L5178Y/TK+/- Mouse Lymphoma Mutagen Assay System," Mutation Res. 59, 61-108.

Code of Federal Regulations 1986a. Title 40, part 50.11, U.S. Government Printing Office, Washington, D.C.

Code of Federal Regulations 1986b. Title 40, Part 50.4, U.S. Government Printing Office, Washington, D.C.

Cogan, D. G. 1943. "Lewisite Burns of the Eye," *JAMA* 122(7), 435-36.

Correll, L., and Ehrich, M. 1987. "Comparative Sensitivities of Avian Neural Esterases to In Vitro Inhibition by Organophosphorus Compounds," ***Toxicol. Lett.*** 36, 197-204.

Costa, L. G., Schwab, B. W., and Murphy, S. D. 1982. "Tolerance to Anticholinesterase Compounds in Mammals," ***Toxicology 25, 79-97.***

Craig, F. N., Cummings, E. G., and Sim, V. M. 1977. "Environmental Temperature and the Percutaneous Absorption of a Cholinesterase Inhibitor, VX," *J. Invest. Dermatol. 68, 357-61.*

Crespi, C., et al. 1985. "Mutagenicity of 1,2-Dichloroethane and 1,2-Dibromoethane in Two Human Lymphoblastoid Cell Lines," *Mutat. Res.* 142, 133-40.

Crook, J. W., et al. 1983. *The Effects of Subacute Exposures of the* Mouse, Rat, Guinea Pig, *and* Rabbit to *Low-Level VX* Concentrations ARCSL-TR-82038, U.S. Army Armament Research and Development Command, Aberdeen Proving Ground, Md. 21010.

Cullumbine, H., et al. 1954. *Percutaneous Toxicity of G-Compounds,* Porton Technical Paper 399 (Secret), as quoted in Fielding 1960.

D'Mello, G. D., and Duffy, E. A. M. 1985. "The Acute Toxicity of Sarin in Marmosets (*Callithrix jacchus):* A Behavioral Analysis," *Fund. and Appl.* Toxicol. 5, S169-74.

Dacre, J. C. May 1, 1987. U.S. Army Biomedical Research and Development Laboratory, Fredrick, Maryland, letter to A. P. Watson, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Dahl, H., et al. 1985. "Eye Lesions Induced by Mustard Gas," *Acta Ophthalmologica* 63(Suppl. 173), 30-31.

Danielli, J. F., et al. 1947. "BAL-INTRAV: A New Non-toxic Thiol for Intravenous Injection in Arsenical Poisoning," *Biochem. J.* 41, 325-33.

Daniels, J. I. In press. "Threat Agents," pp. 10-l-10-30 in *Evaluation of Military Field-Water Quality, Volume 4. Criteria and* Recommendations *for* Standards *for Chemical* Constituents *of* Military *Concern.* Draft document for Project Order 82 PP 2817 supported by the U.S. Army Medical Research and Development Command, Ft. Detrick, Md.

Darmer, K. I., Haun, C. C., and MacEwen, J. D. 1972. "The Acute Inhalation Toxicology of Chlorine Pentafluoride," Am. Ind. Hyg. *Assoc. J.* 33, 661-68.

Dasheiff, R. M., Einberg, E., and Grenell, R. G. 1977. "Sarin and Adrenergic-Cholinergic Interaction in Rat Brain," *Exp. Neurol. 57, 549-60.*

Davidson, I. W. F., Sumner, D. D., and-parker, J. C. 1982. "Chloroform and Review of Its Metabolism, Teratogenic, Mutagenic, and Carcinogenic Potential," *Drug and Chem.* Toxicol. 5(1), l-87.

Davies, D. R., Holland, P., and Rumens, M. J. 1960. "The Relationship Between the Chemical Structure and Neurotoxicity of Alkyl Organophosphorus Compounds," *Brit. J. Pharmacol.* 15, 271-78.

Deichman, W. B., and Gerarde, H. W. 1969. *Toxicology of Drugs and* Chemicals, Academic Press, New York, pp. 67, 317-18, and 339.

Delga, J., et al. 1961. "Recherches sur le Traitement de l'Intoxication Cyanhydrique par l'Hydroxocobalamine," *Annales Pharm. Franc.* 19, 740-52.

Denk, J. R. *1975. Effect of GB on Mammalian Germ* Cells and Reproductive *Performance,* EB-TR-74087, as cited in Weimer et al. 1979.

de Raat, W. K., Davis, P. B., and Bakker, G. L. 1983. "Induction of Sister-chromatid Exchanges by Alcohol and Alcoholic Beverages after Metabolic Activation by Rat-Liver Homogenate," *Hut. Res.* 124, 85-90.

Dewey, J. M., and Fish, H. J. undated. *An Assessment of the Performance of 25 PR BE/CHEM Shell Charged VX,* Canada Suffield Experimental Station Report 184 (Secret Report, Confidential Title), as quoted in Fielding 1960.

Dick, C. J. 1981. "Soviet Chemical Warfare Capabilities," *Internal. Defense Review 1,* reprinted as pp. 1-9 *in Selected Readings in Nuclear,* Biological, *and Chemical* Operations, 1984, U.S. Army Command and General Staff College, Ft. Leavenworth, Kans.

Dickey, F. H., Cleland, G. H., and Lotz, C. 1949. "The Role of Organic Peroxides in the Induction of Mutations," Natl. *Acad. Sci.* (*Proc.*) 35, 581-86.

Dixon, M., and Needham, D. 1946. "Biochemical Research on Chemical Warfare Agents," Nature 158, 432-38.

Doull, J., Klaassen, C. D., and Amdur, M. 0. 1980. *Casarett and Doull's Toxicology: The Basic Science of Poisons,* Macmillan Publishing Co., New York, pp. 19-21, 471, 622-5, 687.

Drury, J., and Hammons, A. 1979. Investigations *of Selected Environmental Pollutants: 1,?-Dichloroethane*, EPA-560/2-78-006, U.S. Environmental Protection Agency, pp. 96-104.

Duffy, F. H., et al. 1979. "Long-Term Effects of an Organophosphate upon the Human Electroencephalogram," *Toxicol. and Appl. Pharmacol. 47,* 161-76.

Duffy, F. H., and Burchfiel, J. L. 1980. "Symposium Proceedings: Long Term Effects of the Organophosphate Sarin on EEGs in Monkeys and Humans," Neurotoxicology 1, 667-89.

Dulaney, Jr., M. D., Hoskins, B., and Ho, I. K. 1985. "Studies on Low Dose Sub-acute Administration of Soman, Sarin and Tabun in the Rat," *Acta pharmacol. et* **toxicol. 57, 234-41.**

Dunn, P. 1986. "The Chemical War: Iran Revisited--1986," *NBC Defense and* Technology International 1(3), 32-39.

Dzhanashvili, G. D. 1967. "Maximum Permissible Concentration of Dimethylamine in Water Bodies," *Hygiene and Sanitation (Gigiena i Sanitariya) 32, 329-35.*

Erdmann, W. D. 1976. "**Success** and Failure of **Oxime** Therapy in Acute Poisoning by Organophosphorus Compounds," pp. 46-52 in Medical *Protection against Chemical-Warfare Agents,* Almqvist & Wiksell International, Sweden.

Fahmy, 0. G., and Fahmy, M. J. 1971. "Mutability at Specific Euchromatic and Heterochromatic Loci with Alkylating and Nitroso Compounds in *Drosophila melanogaster," Mutat. Res.* 13, 19-34.

Fahmy, 0. G., and Fahmy, M. J. 1972. "Mutagenic Selectivity for RNA-Forming Genes in Relation to the Carcinogenicity of Alkylating Agents and Polycyclic Aromatics," *Cancer Res. 32, 550-57.*

Farquharson, D. A., et al. 1980. "Penetration of VX into Nerve Cells, and Effects on Electrical Function," *Bull Environ. Contam. Toxicol.* 24(5), 719-26.

Fell, H., and Allsopp, M. 1948. "The Effect of Repeated Applications of Minute Quantities of Mustard Gas on the Skin of Mice," *Cancer Res.* 8, 177-81.

Fernando, J. C. R., Hoskins, B., and Ho, I. K. 1984. "A Striatal Serotonergic Involvement in the Behavioral Effects of Anticholinesterase Organophosphates," *Eur. J. Pharmacol.* 98(1), 129-32.

Fernando, J. C. R., et al. 1985. "Variability of Neurotoxicity of and Lack of Tolerance to the Anticholinesterases Soman and Sarin in the Rat," *Res. Commun. Chem. Pathol. Pharmacol.* 48(3), 415-30.

Fernando, J. C. R., Hoskins, B., and Ho, I. K. 1985. "Rapid Induction of Supersensitivity to Muscarinic Antagonists-Induced Motor Excitation by Continuous Stimulation of Cholinergic Receptors," Life Sci., 37, 883-92.

Fernando, J. C. R., Hoskins, B., and Ho, I. K. 1986. "Hypersensitivity to Antimuscarinic Agents Following Brief Exposure to Soman and Sarin," *J. Toxicol. and Environ. Health* 18, 103-09.

Fielding, G. H. 1960. *V Agent Information Summary,* NRL 5421, U.S. Naval Research Laboratory, Washington, D.C.

Flamm, K. 1986. Chemical *Agent and* Munition Disposal. *Summary of the U.S. Army's Experience,* AMCPM-CD-TR-87115 (draft), Office of Program Manager for Chemical Munitions, Aberdeen Proving Ground, Md.

Fleisher, J. H., Harris, L. W., and Berkowitz, P. T. 1969. *Metabolism of P(32)-Isopropyl Hethylphosphonofluoridate* (Sarin) in *Dogs* EATR 4316, Department of the Army, Medical Research Laboratory, Edgewood Arsenal, Md.

Fleisher, J. H., Harris, L. W., and Berkowitz, P. T. 1970. "Dephosphorylation In Vivo of Brain Acetylcholinesterase Inhibited by Isopropyl Methylphosphonofluoridate (Sarin)," *Biochem. Pharmacol.* 19, 421-26.

Florin, I., et al. 1980. 'Screening of Tobacco Smoke Constituents for Mutagenicity Using the Ames Test," *Toxicology* 15, 219-32.

Flynn, C. J., and Wecker, L. 1986. "Elevated Choline Levels in Brain: A Non-Cholinergic Component of Organophosphate Toxicity,' *Biochem. Pharmacol.* 35(18), 3115-21.

Fonnum, F., and Sterri, S. H. 1981. 'Factors Modifying the Toxicity of Organophosphorus Compounds Including Soman and Sarin," Fund. *and Appl. Toxicol.* 1, 143-47.

Food and Drug Research Laboratories 1974. *Teratologic Evaluation of FDA 71-78 (Apple Cider Vinegar* [Acetic Acid]; *Table Strength 5%) in Mice, Rats and Rabbits*, PB-234-869.

Fox, M., and Scott, D. 1980. "The Genetic Toxicology of Nitrogen and Sulphur Mustard,' *Mutation Res. 75,* 131-68.

Frawley, J. P., et al. 1957. 'Marked Potentiation in Mammalian Toxicity from Simultaneous Administration of Two Anticholinesterase Compounds," *J. Pharmacol. Exp. Therapeu.* 121, 96-106.

Freeman, G. et al. 1953. *The Toxicity of Liquid GB Applied to the Skin of Man,* Chemical Corps Medical Labs., MLRR 217 (Secret), as quoted in Fielding 1960.

Freireich, A. W., et al. 1967. "Hemodialysis for Isopropanol Poisoning," *N. Engl. 3. Med. 277, 699-700.*

Friedberg, K., Mengel, K., and Schlick, E. 1983. "The Action of Azimexone on the Cells of the Hemopoietic System in Mice, Especially After Damage with X-Rays," *Radfat. Environ. Biophys. 22,* 117-31.

Friedham, E., et al. 1978. "Treatment of Lead Poisoning by 2,3-Dimercaptosuccinic Acid," *Lancet 2,* 1234-36, as cited in Aposhian, H. V., et al. *1984. Fund. Appl. Toxicol. 4,* S58-70.

Friedlander, B. R., Hearne, T., and Hall, S. 1978. "Epidemiologic Investigation of Employees Chronically Exposed to Methylene Chloride," *J. Occup. Med. 20, 657-66.*

Friel, J. P., ed. *1974. Dorland's Illustrated Medical Dictionary,* 25th ed., W. B. Saunders, Philadelphia, pp. 222, 261, 352, 510, 798, 815, 838, 1462, and 1549.

Gall, D. 1981. "The Use of Therapeutic Mixtures in the Treatment of Cholinesterase Inhibition," *Fund. and Appl. Toxicol.* 1, 214-16.

Gammage, R. B. 1983. "Polycyclic Aromatic Hydrocarbons in Work Atmospheres," pp. 653-707 in *Handbook of Polycylic-Aromatic Hydrocarbons,* ed. A. Bjorseth, Marcel Dekker, Inc., New York.

Geeraets, W., Abedi, S., and Blanke, R. 1977. "Acute Cornea1 Injury by Mustard Gas," Southern Med. *J.* 70, 348-50.

General Electric Co. 1984. *Material Safety Data Sheet, No. 8,* Genium Publishing Corp., Schenectady, N.Y.

Gerber, G. J., and O'Shaughnessy, D. 1986. "Comparison of the Behavioral Effects of Neurotoxic and Systemically Toxic Agents: How Discriminatory are Behavioral Tests of Neurotoxicity?," *Neurobehav. Teratol.* Toxicol. 8, 703-10.

Gerdes, R. A. 1971. "The Influence of Atmospheric Hydrogen Fluoride on the Frequency of Sex-Linked Recessive Lethals and Sterility in *Drosophila melanogaster," Fluoride 4, 25-29.*

Gershon, S., and Shaw, F. H. 1961. "Psychiatric Sequelae of Chronic Exposure to Organophosphorus Insecticides," *Lancet* 1, 1371-74.

Ghiringhelli, L., and Di Fabio, A. 1957. "Patologia da acido acetico: osservazioni negli animali da esperimento e nell'uomo," *Med. Lavoro* 48, 559-65 (Italian with English summary).

Gill, G. R., and La Ham, Q. N. 1972. "Histochemical and Radiographic Investigations of Malathion-Induced Malformations in Embryonic Chick Limbs," *Canad. J. Zool. SO,* 349-51.

Glow, P. H., Richardson, A., and Rose, S. 1966. "Effects of Acute and Chronic Inhibition of Cholinesterase Upon Body Weight, Food Intake, and Water Intake in the Rat," *J. Comp. Physiol. Psychol.* **61**(2), 295-99.

Glukharev, Yu. U. A., et al. 1980. "Data for Use in Setting Hygienic Standards for Divinyl Sulfide in Reservoir Water," Gig. *Sanit. 5, 69-70.*

Gordon, J. J., et al. 1983. "The Delayed Neuropathic Effects of Nerve Agents and Some Other Organophosphorus Compounds," *Arch. Toxicol.* **52**(2), 71-82.

Gosselin, R. E., et al. 1976. Clinical Toxicology *of* Commercial *Products,* 4th ed., Section III, The Williams 6 Wilkins Co., Baltimore, pp. 169-73.

Gosselin, R. E., et al. 1984. Clinical *Toxicology of Commercial Products* 5th ed., Williams and Wilkins, Baltimore, pp. I-2, 11-236, 11-387, 11-382, III-47 to III-SO, III-50 to III-52, III-174 to III-175, and III-336 to 111-43.

Goudou, D. and Rieger, F. 1983. "Recovery of Acetylcholinesterase and of its Multiple Molecular Forms in Motor End-Plate-Free and Motor End-Plate-Rich Regions of Mouse Striated Muscle, after Irreversible Inactivation by an Organophosphorus Compound (Methyl-Phosphorothiolate Derivative)," Biol. *Cell 48,* 151-57.

Grant, W. M. 1986. Toxicology *of the Eye* 3rd ed., Charles C. Thomas, Springfield, Ill., pp. 213, 336, 495-97, and 643-46.

Gray, E. L., et al. 1954. "Toxicity of the Oxides of Nitrogen," *Arch. Ind. Hyg. Occup. Med. 10,* 418-22.

Greenberg, J., and La Ham, Q. N. 1969. "Malathion-Induced Teratisms in the Developing Chick," *Canad. J. Zool. 47, 539-42.*

Griffiths, A. J. F. 1981. "Neurospora and Environmentally Induced Aneuploidy," pp. 187-199 in *Short-Term Tests for Chemical Carcinogens,* ed. H. G. Stich and R. H. C. San, Springer-Verlag, New

York.

Grob, D. and Harvey, A. M. 1953. "The Effects and Treatment of Nerve Gas Poisoning," Am. *J. Med.,* 52-63.

Grob, D. and Harvey, J. C. 1958. "Effects in Man of the Anticholinesterase Compound Sarin (Isopropyl Methyl Phosphonofluoridate)," *Johns Hopkins Ued. J. 37, 350-68.*

Gross, C., et al. 1985. "Sulfur Mustard Lowers Nicotinamide Adenine Dinucleotide Concentrations in Human Skin Grafted to Athymic Nude Mice," *Toxicol. Appl. Pharmacol.* 81, 85-90.

Guest, D., Katz, G. V., and Astill, B. D. 1982. "Aliphatic Carboxylic Acids," pp. 4901-88 in Patty's Industrial Hygiene and Toxicology, 3rd ed., rev., Vol. 2C, Toxicology, ed. G. D. Clayton and F. E. Clayton, John Wiley & Sons, New York, p. 4914.

Haber, L. 1986. *The Poisonous Cloud,* Clarendon Press, Oxford, England, pp. 250-57.

Haddad, L. M., and Winchester, J. F. 1983. "The Organophosphate Insecticides," pp. 704-710 in *Clinical Management of Poisoning and Drug Overdose. Section F. Pesticides.* W. B. Saunders Company, Philadelphia.

Hanley, T., et al. 1984. "Comparison of the Teratogenic Potential of Inhaled Ethylene Glycol Monomethyl Ether in Rats, Mice and Rabbits," *Toxicol. Appl. Pharmacol. 75, 409-22.*

Hardin, B. D. 1983. "Reproductive Toxicity of the Glycol Ethers," *Toxicology 27,* 91-102.

Harris, L. W., et al. 1967. Aging *and Dealkylation of Rat-Brain ChE Poisoned with Isopropyl Methyl-Phosphonofluoridate (Sarin, GB),* EATR 4047, Medical Research Laboratory, Edgewood Arsenal, Md.

Harrison, W. P., and Hood, R. D. 1981. "Prenatal Effects Following Exposure of Hamsters to Sodium Arsenite by Oral or Intraperitoneal Routes," *Teratology 23,* 40A.

Hartley, W. R. 1987. "A Summary of Current U.S. Army Toxicity Studies on Agent VX," presented at Workshop on Exposure Limits for Agent VX: Safe Exposure Levels for VX (Surgeon General's Panel), Atlanta, Ga., April 2-3, 1987.

Hauser, W., and Weger, N. 1979. "Therapeutic Effects of the Bis-pyridinium Salts HGG-12, HGG-42, and Atropine, Benactyzine in Organophosphate Poisoning of Dogs," Arch *of* Toxicol. 2, 393-96.

Heilbronn, E., and Tolagen, B. 1965. "Toxogonin in Sarin, Soman and Tabun Poisoning," *Biochem. Pharmacol.* 14, 73-77.

Heldman, E., et al. 1986. "Sugar Conjugates of Pyridinium Aldoximes as Antidotes against Organophosphate Poisoning," *Carbohydr. Res.* 151, *337-47.*

Hellmann, W. 1970. "Klinisch-genetische Untersuchungen an Familien von durch N- and S-Lost Chemisch Gewerblich Geschadigten," Doctoral dissertation, University of Marburg. Taken from Lohs, K. *1975 Delayed Toxic Effects of Chemical Warfare* Agents, A SIPRI Monograph, Almqvist and Wiksell International, Stockholm.

Hemminki, K., and Neimi, M.-L. 1982. "Community Study of Spontaneous Abortions: Relation to Occupation and Air Pollution by Sulfur Dioxide, Hydrogen Sulfide, and Carbon Disulfide ," *Int. Arch. Occup. Environ. Health 51, 55-63.*

Heston, W. E. 1950. "Carcinogenic Action of the Mustards," *J. Natl. Cancer Inst.* 11, 415-23.

Heston, W. E. 1953. "Occurrence of Tumors in Mice Injected Subcutaneously with Sulfur Mustard and Nitrogen Mustard," *J. Natl. Cancer Inst.* 14, 131-40.

Heston, W. E., and Levillain, W. D. 1953. "Pulmonary Tumors in Strain A Mice Exposed to Mustard Gas," *Proc. Soc. Exp. Biol.* 82, 457-60.

Hine, C. H., et al. 1958. "The Toxicology of Epoxy Resins," *A.M.A. Arch. Ind. Health* 17, 129-44. Taken from RTECS (data base) Registry of Toxic Effects of Chemical Substances. Oak Ridge National Laboratory, Oak Ridge, Tenn. 1986. IE1225000.

Hines, M. M. 1984. *Statistical Analysis of* VX *Data from Brine Sampling* at *the* Chemical *Agent* Munitions Disposal System (*CAMDS*) at *Tooele Army Depot, Tooele, Utah,* Subcontractor Report by Computer Sciences Corporation, National Space Technology Laboratories, NSTL, Miss.

Hirshberg, A., and Lerman, Y. 1984. "Clinical Problems in Organophosphate Insecticide Poisoning: The Use of a Computerized Information System," *Fund. and Appl. Toxicol. 4,* S209-14.

Hoffman, D. J., and Campbell, K. I. 1977: "Embryotoxicity of Irradiated and Nonirradiated Catalytic Converter-Treated Automotive Exhaust," J. *Toxicol. and Environ. Health 3,* 705-12.

Holmstedt, B. 1959. "Pharmacology of Organophosphorous Cholinesterase Inhibitors," *Pharmacol. Rev. 11, 603.*

Hood, R. D., and Harrison, W. P. 1982. "Effects of Prenatal Arsenite Exposure in the Hamster,* *Bull. Environ. Contam. Toxicol. 29,* 671-*78.*

Hopff, W. H, Riggio, G., and Waser, P. G. 1984. "Sarin Poisoning in Guinea Pigs Compared to Reactivation of Acetylcholinesterase In Vitro as a Basis for Therapy," *Acta Pharmacol. et Toxicol. 55, 1-5.*

Hoskins, B., Liu, D. D., and Ho, I. K. 1986. "Acute Effects of Soman, Sarin, and Tabun on Microsomal and Cytosolic Components of the Calmodulin System in Rat Striatum," *J. Neurochem.* 46(1), *265-69.*

Hoskins, B., et al. 1986. "Relationship Between the Neurotoxicities of Soman, Sarin and Tabun, and Acetyylcholinesterase Inhibition," *Toxicol. Lett. 30,* 121-29.

Hsie, A. W. 1980. "Quantitative Mutagenesis and Mutagen Screening with Chinese Hamster Ovary Cells," pp. 89-102 in *The* Predictive Value of *Short-Term Screening Tests in Carcinogenicity Evaluation,* ed. G. M. Williams, R. Kroes, H. W. Waaijers and K. W. van de Poll, Elsevier-North Holland Biomedical Press, Amsterdam.

Hughes, T. J., Sparacino, C., and Frazier, S. 1984. *Validation of* Chemical *and* Biological Technique *for* Evaluation *of Vapors in* Ambient Air/Mutagenicity Testing *of* Twelve Vapor-Phase Compounds, ISS EPA-600/l-84-005, U.S. Environmental Protection Agency, Research Triangle Park, NC, PB84-164219, pp. 3, 177-189.

Idriss, M. K., et al. 1986. "Organophosphate and Carbamate Compounds Have Pre- and Postjunctional Effects at the Insect Glutamatergic Synapse," *J.* Pharmacol. *and Exp. Ther.* 239(1), 279-85.

Inada, S., et al. 1978. "Multiple Bowen's Disease Observed in Former Workers of Poison Gas Factory in Japan, with Special Reference to Mustard Gas Exposure," *J. Dermatol. Tokyo 5, 49-60.*

Inns, R. H., and Leadbeater, L. 1983. "The Efficacy of Bispyridinium Derivatives in the Treatment of Organophosphonate Poisoning in the Guinea-pig," *J.* Pharm. *Pharmacol. 35, 427-33.*

International Agency for Research on Cancer (IARC) 1975. "Mustard Gas," pp. 181-192 in *IARC Monographs* on *the Evaluation of the Carcinogenic Risk of Chemicals to Nan: Aziridines, N-, S-,* and *O-Mustards and Selenium, Vol.* 9, World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1979a. "Chloroform," pp. *401-427 in IARC Monographs on the Evaluation of the Carcinogenic* Risk *of Chemicals* to *Humans: Some Halogenated Hydrocarbons, Vol.* 20, World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1979b. "Dichloromethane," pp. 449-465 in *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Halogenated Hydrocarbons, Vol. 20,* World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1979c. "1,1,2-Trichloroethane," pp. 533-43 in *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Halogenated Hydrocarbons, Vol. 20,* World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1979d. "Vinyl Chloride, Polyvinyl Chloride and Vinyl Chloride--Vinyl Acetate Copolymers," pp. *377-438* in *IARC Monographs on the Evaluation of the* Carcinogenic Risk *of Chemicals* to *Humans: Some Monomers, Plastics and* Synthetic Elastomers, *and Acrolein, Vol. 19,* World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1980. "Arsenic and Arsenic Compounds," pp. 39-141 in *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans:* Some Metals *and* **Metal** *lic* Compounds, Vol. 23, World Health Organization, Geneva, Switzerland.

International Agency for Research on Cancer (IARC) 1982. "Chloroform (Group 2B)," pp. *87-88* in *IARC Monographs on the Evaluation of the* Carcinogenic Risk *of Chemicals to Humans: Chemicals, Industrial Processes and Industries Associated* with Cancer *in Humans, Supplement 4, Vols. l-29,* World Health Organization, Lyon, France.

Ishidate, M., et al. 1952. "Experimental Studies on Chemotherapy of Malignant Growth Employing Yoshida Sarcoma Animals. (III) The Effect of N-Oxide Derivatives of Nitrogen Mustards," *GANNA (The Japanese Journal of Cancer Research) 43,* 171-74.

Ishidate, M., Jr., Sofuni, T., and Yoshikawa, K. 1981. "Chromosomal Aberration Tests In Vitro as a Primary Screening Tool for Environmental Mutagens and/or Carcinogens," Gann *Monogr.* Can. *Res.* 27, 95-108.

Ishidate, M., Jr., et al. 1984. "Primary Mutagenicity Screening of Food Additives Currently Used in Japan," *Food Chem. Toxicol. 22, 623-36.*

Isomura, K., et al. 1984. "Induction of Mutations and Chromosome Aberrations in Lung Cells Following In Vivo Exposure of Rats to Nitrogen Oxides," *Mutat. Res.* 136, 119-25.

Jackson, H., and Craig, A. W. 1966. "Antifertility Action and Metabolism of Hexamethylphosphoramide," *Nature 212, 86-87.*

Johns, R. J. 1952. *The Effects of Low Concentrations of GB on the Human Eye* CMLRE-ML-52, Chemical Corps, Medical Laboratories, Army Chemical Center, Md.

Jones, D. E., Carter, Jr., W. H., and Carchman, R. A. 1985. "Assessing Pyridostigmine Efficacy by Response Surface Modeling," *Fund. and Appl.* Toxicol. 5, S242-51.

Jones, T. D., Walsh, P. J., and Zeighami, E. A. 1985. "Permissible Concentrations of Chemicals in Air and Water from RTECS Entries: A 'RASH' Chemical Scoring System," *Toxicol. and Ind. Health* 1(4), 213-34.

Jones, T. D., et al. In press. "Chemical Scoring by a Rapid Screening Hazard (RASH) Method Mimics Decisions of Expert Committees," *Risk Analysis.*

Jongen, W. M. F., et al. 1981. "Mutagenicity Testing of Dichloromethane in Short-Term Mammalian Test Systems," *Mutat. Res.* 81, 203-13.

Jorgenson, T. A., et al. 1985. "Carcinogenicity of Chloroform in Drinking Water to Male Osborne-Mendel Rats and Female B6C3F1 Mice," *Fund. Appl. Toxicol. 5, 760-69.*

Karczmar, A. G. 1984. "Acute and Long Lasting Central Actions of Organophosphorus Agents," *Fund. and Appl. Toxicol. 4,* Sl-17.

Karczmar, A. G. 1985. "Present and Future of the Development of Anti-OP Drugs," *Fund. and Appl. Toxicol. 5,* S270-79.

Kawakami, T. G., Schreider, J. P., and Rosenblatt, L. S. 1984. Mutagenicity *of* the Organophosphate *Agent,* VX, in the Mouse Lymphoma *Assay,* Laboratory for Energy-Related Health Research, University of

California, Davis, Calif.

Kepner, L. A., and Wolthuis, 0. L. 1978. "A Comparison of the Oximes HS-6 and HI-6 in the Therapy of Soman Intoxication in Rodents," *Eur. J. Pharmacol. 40, 377-82.*

Kiffer, D., and Hinard, P. 1986. "Reactivation by Imidazo-Pyridinium Oximes of Acetylcholinesterase Inhibited by Organophosphates: A Study with an Immobolized Enzyme Method," *Biochem. Pharmacol.* 35(15), 2527-33.

Kimball, R. F., and Munro, N. B. 1981. A Critical Review *of the Mutagenic and Other Genotoxic Effects of Direct Coal Liquefaction,* ORNL-5721, Oak Ridge National Laboratory, Oak Ridge, Tenn., pp. 3-5.

Kimura, K. K., McNamara, B. P., and Sim, V. M. 1960. *Intravenous* Administration *of VX* in Man CRDLR 3017, U.S. Army, Chemical Corps Research and Development Command, Chemical Research and Development Laboratories, Army Chemical Center, Md.

Kircher, M., and Brendel, M. 1983. "DNA Aklylation by Mustard Gas in Yeast Strains of Different Repair Capacity," *Chem.-Biol. Interact.* 44, 27-39.

Kirkland, D. J., Smith, K. L., and Van Abbe, N. J. 1981. "Failure of Chloroform to Induce Chromosome Damage or Sister-Chromatid Exchanges in Cultured Human Lymphocytes and Failure to Induce Reversion in *Escherichia coli*," *Food Cosmet. Toxicol. 19, 651-b.*

Kiss, Z., and Fazekas, T. 1979. "Arrhythmias in Organophosphate Poisonings," *Acta. Cardiol. 34, 323-30.*

Klaassen, C. D. 1980, "Heavy Metals and Heavy Metal Antagonists," pp. *1615-1637* in *Goodman and Gilman's The Pharmacological Basis of Therapeutics,* 6th ed., A. G. Gilman, L. S. Goodman, and A. Gilman, eds., Macmillan Co., New York, as cited in Aposhian, H. V., et al. 1984. *Fund. Appl. Toxicol. 4,* S58-70.

Klehr, N. 1984. "Cutaneous Late Manifestations in Former Mustard Gas Workers," *Z. Hautkr.* 59(17), 1161-70.

Klimova, K. K. 1958. "Pharmacology of a New Unithion, " *Farmakol. Toksikol* (Moscow) 21, 53-59, as cited in Aposhian, H. V., et al. *1984. Fund. Appl. Toxicol. 4,* S58-70.

Koelle, G. B. 1981. "Organophosphate Poisoning--An Overview," *Fund. and Appl. Toxicol.* 1, 129-34.

Koike, M., Ito, T., and Yamamoto, M. 1983. "Cytogenetic Effects of Maternal Alcohol Uptake on F1 Mouse Fetuses," *Teratology 28,* 31A.

Koslow, E. 1987. "Decontamination," pp. 28-30 in *Nuclear, Biological and Chemical Defense & Technology International 1987 Yearbook.*

Krackow, E. H. 1956. Toxicology *of V Agents,* Army Chemical Center, Chemical Warfare Labs., CWLR 2065 (Secret Report, Unclassified Title), as quoted in Fielding 1960.

Krause, H., and Grussendorf, E. I. 1978 "Syntopy of Bowen's Disease and Lewisite Scar," Hautarzt **29**(9), 490-3.

Krop, S. and Kunkel, A. M. 1954. "Observations on Pharmacology of the Anticholinesterases Sarin and Tabun," *Proc. Soc. Exp. Biol. Med.* 86, 530-33.

Kusic, R., et al. 1985. "HI-6 in Man: Blood Levels, Urinary Excretion, and Tolerance after Intramuscular Administration of the Oxime to Healthy Volunteers," *Fund. and Appl.* Toxicol. 5, S89-97.

LaBorde, J. B., and Bates, H. K. 1986. *Developmental Toxicity Study of Agent GB-DCSM Types I and II in CD Rats and NZW Rabbits. Final Report,* National Center for Toxicological Research, FDA, Jefferson, Ark.

Land, P. C., et al. 1981. "Morphologic Changes in Mouse Spermatozoa after Exposure to Inhalation Anesthetics during Early Spermatogenesis," *Anesthesiology* **54**(1), 53.

Landauer, M. R. and Romano, J. A. 1984. "Acute Behavioral Toxicity of the Organophosphate Sarin in Rats," *Neurobehav. Toxicol. Teratol.* 6, 239-43.

Landolph, J. R. 1983. "Transformation of C3H/10T1/2 Mouse Fibroblasts by Carcinogenic Metal Salts in the Absence of Base Substitution Mutations," *Proc. Am. Assoc. Cancer Res. 24,* 100.

Larramendy, M. L., Popescu, N. C., and DiPaolo, J. A. 1981. "Induction by Inorganic Metal Salts of Sister Chromatid Exchanges and Chromosome Aberrations in Human and Syrian Hamster Cell Strains," *Environ. Mutagen. 3, 597-606.*

Lazarew, N. W. 1929. "Uber die Marketische Wirkungskraft der Dampfe der Chlorderivaten des Methans, des Athans und des Athylens," *Naunyn-Schmiedeberg's Archiv fur Experimentelle Pathologie und Pharmakologie* 141, 19-24.

Leadbeater, L., Inns, R. H., and Rylands, J. M. 1985. "Treatment of Poisoning by Soman," *Fund. and Appl.* Toxicol. 5, S225-31.

LeBlanc, F. N., Benson, B. E., and Gilg, A. D. 1986. "A Severe Organophosphate Poisoning Requiring the Use of an Atropine Drip," *Clin. Toxicol.* 24(1), 69-76.

Lee, T.-C., Oshimura, M., and Barrett, J. C. 1985. "Comparison of Arsenic-Induced Cell Transformation, Cytotoxicity, Mutation and Cytogenetic Effects in Syrian Hamster Embryo Cells in Culture," Carcinogenesis 6, 1421-26.

Leffingwell, S. S. August 24, 1987. Special Programs Group, Center for Environmental Health and Injury Control, Centers for Disease Control, Atlanta, Georgia, letter to A. P. Watson, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Lehmann, K. B. 1887. "Experimentelle Studien uber den Einfluss technisch und hygienisch wichtiger Gase und Dampfe auf den Organismus. Theil III und IV: Chlor und Brom," *Archiv. fuer Hygiene 7, 231-85.*

Lenchenko, V. G., and Sharipova, N. P. 1974. "Teratogenic and Embryotoxic Effect of Small Concentrations of Inorganic Fluorine Compounds in the Air," pp. 160-64 in *Voprosy Eksperimental No I I Klinicheskoi Terapii I profilaktiki Promyshlennykh Intoksikatsii.*

Lenz, D. E., and Maxwell, D. M. 1981. "Protection Afforded by the H Oximes," pp. 15-34 in *Evaluation* of H-Series *Oximes: Proceedings of a Symposium* USABML-SP-81-001, ed. C. A. Broomfield, D. E. Lenz, B. E. Hackley, Jr., D. M. Maxwell, and F. E. Hahn, U.S.A. Biomedical Laboratory, Aberdeen Proving Ground, Md.

Lenz, K., et al. 1981. "2,3-Dimercapto Succinic Acid in Human Arsenic Poisoning," *Arch. Toxicol.* 47, 241-43, as cited in Aposhian, H. V., et al. 1984. *Fund. Appl. Toxicol. 4,* S58-70.

Leopold, W. R., Miller, J. A., and Miller, E. C. 1982. "Comparison of Some Carcinogenic, Mutagenic, and Biochemical Properties of *S*-Vinylhomocysteine and Ethionine," *Cancer Res. 42, 4364-74.*

Levin, H. S., Rodnitzky, R. L., and Mick, D. L. 1976. "Anxiety Associated with Exposure to Organophosphate Compounds," *Arch. Gen. Psychiatry 33, 225-28.*

Levy, M. 1965. "The Effect of Mustard Gas upon a Property of Nucleated Erythrocytes and Their Ghosts," *Cancer Res. 25, 752-59.*

Liang, Y., et al. 1957. "Studies on Anti-Biharzial Drugs VI. The Antidotal Effects of Sodium Dimercapto Succinate and BAL-Glucoside Against Tartar," Emetic. *Acta Physiol. Sin.* 21, 24-32, as cited in Aposhian, H. V., et al. 1984. *Fund. Appl. Toxicol. 4,* S58-70.

Little, P. J., et al. 1986. "Tissue Disposition of [(3)H]Sarin and. Its Metabolites in Mice," Toxicol. *and Appl. Pharmacol. 83,* 412-19.

Liu, D.-D., et al. 1986. "Acute Effects of Soman, Sarin, and Tabun on Cyclic Nucleotide Metabolism in Rat Striatum," *J. Toxicol. and Environ. Health* 19, 23-32.

Lohs, *K.* 1975. *Delayed Toxic Effects of Chemical Warfare* Agents: *A SIPRI Monograph,* Almqvist 6 Wiksell, International, Stockholm.

Loomis, T. A., and Salafsky, B. 1963. "Antidotal Action of Pyridinium Oximes in Anticholinesterase Poisoning; Comparative Effects of Soman, Sarin, and Neostigmine on Neuromuscular Function," *Toxicol. Appl.* Pharmacol. 5, 685-701.

Loveless, A. 1951. "Qualitative Aspects of the Chemistry and Biology of Radiomimetic (Mutagenic) Substances," Nature 167, 338-42.

Ludomirsky, A., et al. 1982. "Q-T Prolongation and Polymorphous ("Torsade de Pointes") Ventricular Arrhythmias Assocated with Organophosphate Insecticide Poisoning," *Amer. J. Cardiol. 49,* 1654-58.

Lund-Karlsen, R., and Fonnum, F. 1976. "The Effect of Locally Applied Cholinesterase Inhibitors and Oximes on the Acetylcholinesterase Activity in Different Parts of the Guinea-Pig Eye," *Acta. Pharmacol. Toxicol.* 38, 299-307.

Lynch, M. R., Rice, M. A., and Robinson, S. W. 1986. "Dissociation of Locomotor Depression and ChE Activity after DFP, Soman and Sarin," *Pharmac. Biochem.* Behav. 24, 941-47.

Machle, W., et al. 1942. "The Effect of the Inhalation of Hydrogen Chloride," *J. Ind. Hyg. Toxicol. 24, 222-25.*

Malcolm, D., and Paul, E. 1961. "Erosion of the Teeth Due to Sulphuric Acid in the Battery Industry," *Br. J. Ind. Med.* 18, 63-69.

Malhi, P. K., and Grover, I. S. 1987. "Genotoxic Effects of Some Organophosphorus Pecticides. II. In Vivo Chromosomal Aberration Bioassay in Bone Marrow Cells in Rats," *Mutat. Res.* 188, 45-51.

Maltoni, C., Valgimigli, L., and Scarnato, C. 1980. "Long-Term Carcinogenic Bioassays on Ethylene Dichloride Administered by Inhalation to Rats and Mice," pp. 3-33 in *Banbury Report 5 Ethylene Dichloride: A* Potential *Health Risk?,* ed. B. Ames, P. Infante, and R. Reitz, Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.

Manning, K., et al. 1981. "Cancer of the Larynx and Other Occupational Hazards of Mustard Gas Workers," *Clin. Otolaryngol.* 6, 165-70.

Marquis, J. *K.* 1986. *Contemporary Issues in Pesticide* Toxicology *and Pharmacology.* Karger, Basel, Switzerland, pp. 53-71.

Martin, L. J., et al. 1985. "Protective Effect of Diazepam Pretreatment on Soman-Induced Brain Lesion Formation," *Brain Res. 325, 287-89.*

Marzulli, F. N., and Williams, M. R. 1953. *Studies on the Evaporation, Retention, and Penetration of GB Applied to Intact Human and Intact and Abraded Rabbit Skin,* Chemical Corps Medical Labs., MLRR 199 (Secret), as quoted in Fielding 1960.

Marzulli, F. *N.* et al. 1957. *Factors Affecting* Percutaneous *Toxicity to Rabbits of GB, VE, and EA 1677,* Army Chemical *Center,* Chemical Warfare Labs., CWLR 2121 (Secret Report, Unclassifed Title), as quoted in Fielding 1960.

Marzulli, F. N., and Wiles, J. *S.* 1957. *Rate of Transfer of VX Across the Epidermal Barrier with Special Reference to Skin-Surface* Contact Area *and* Contact *Time* Army Chemical Center, Chemical Warfare Labs., CWLR2153 (Confidential Report, Unclassified Title), as quoted in Fielding 1960.

Mason, R. V. 1974. "Smoke and Toxicity Hazards in Aircraft Cabin Furnishings," *Ann. Occup. Hyg. 17, 159-65.*

McLeod, A. A., et al. 1982. "Cardiac Sequelae of Acute Head Injury," *Br. Heart J. 47,* 221-26.

McLeod, Jr., C. G. 1985. "Pathology of Nerve Agents: Perspectives on Medical Management," *Fund. and Appl. Toxicol. 5,* S10-16.

McNamara, B. P., and Leitnaker, F. C. 1971. *Toxicological Basis for Controlling Emission of GB into the Environment,* EASP 100-98, Department of the Army, Medical Research Laboratory, Edgewood Arsenal, Md.

McNamara, B. P., Vocci, F. J., and Leitnaker, F. C. 1973. *Proposed Limits for Human* Exposure *to VX Vapor* in *Nonmilitary Operations.* EASP 1100-l (R-l), Edgewood Arsenal, Aberdeen Proving Ground, Md.

McNamara, B. P., et al, 1975. Toxicological Basis *for Controlling Levels of Mustard in the Environment,* EB-SP-74030.

Mecler, F. J. 1981. *Mammalian Toxicological Evaluation of DIMP and DCPD (Phase III--IMPA),* AD-A107574, U.S. Army Medical Research and Development Command, Fort Detrick, Frederick, Md., pp. 6-20, 31-56.

Medema, J. 1986. "Mustard Gas: *The* Science of H," *NBC Defense and Technology International* 1(4), 66-71.

Meeter, E., and Wolthuis, 0. L. 1968a. "The Effects of Cholinesterase Inhibitors on the Body Temperature of the Rat,,, *Eur. J. Pharmacol.* 4, 18-24.

Meeter, E., and Wolthuis, 0. L. 1968b. "The Spontaneous Recovery of Respiration and Neuromuscular Transmission in the Rat After Anticholinesterase Poisoning,,, *Eur. J. Pharmacol. 2, 377-86.*

Mellerio, J., and Weale, R. A. 1965. "Hazy Vision in Amine Plant Operatives,,, *Br. J. Ind. Med. 23, 153-54.*

Menschick, H. 1971. "Dichloroethanes," pp. *385-86* in *Encyclopedia of Occupational Health and Safety, Vol. 1,* McGraw-Hill, New York.

Mershon, M. M., and Tennyson, A. V. 1987. "Chemical Hazards and Chemical Warfare,,, *JAVMA* 190(6), *734-45.*

Meshul, C. K., et al. 1985. "Comparison of the Ultrastructural Myopathy Induced by Anticholinesterase Agents at the End Plates of Rat Soleus and Extensor Muscles,,, *Exp. Neurol. 89, 96-114.*

Metcalf, D. R., and Holmes, J. H. 1969. "EEG, Psychological, and Neurological Alterations in Humans with Organophosphorus Exposure,,, *Ann. N.Y. Acad.* Sci. 160(1), 357-65.

Mill, T., and Gould, C. W. 1979. "Free-Radical Oxidation of Organic Phosphonic Acid Salts in Water Using Hydrogen Peroxide, Oxygen, and Ultraviolet Light ," *Environ.* Sci. and *Technol. 13, 205-08.*

Miller, R., et al. 1983. "Ethylene Glycol Monomethyl Ether. I. Subchronic Vapor Inhalation Study with Subchronic Vapor Inhalation Study with Rats and Rabbits,,, *Fund. Appl.* Toxicol. 3, 49-54.

Morin, M. L., and McKinley, E. R. 1976. *Evaluation of the Effect of Low-Level GB Exposure on Testicular Atrophy in the Fischer 344 Rat,* EB-TR-75031, Edgewood Arsenal, as cited in Weimer et al. 1979.

Moriya, M., et al. 1983. "Further Mutagenicity Studies on Pesticides in Bacterial Reversion Assay Systems," *Mutat. Res. 116,* 185-216.

Munro, N. B., and Travis, C. C. 1986. "Drinking Water Standards," *ES&T,* 768-79.

Murphy-Corb, M., Kong, H-L., and Murray, M. 1983. "Mutagenic Activity from Nitrosation of Ligoamines," *Environ. Mutagen. 5,* 101-09.

Murray, F. J., et al. 1979. "Embryotoxicity of Inhaled Sulfuric Acid Aerosol in Mice and Rabbits," *J. Environ. Sci. Health, Part C 13,* 251-66.

Murtha, E. F., and Harris, L. W. 1980. "Effects of 2-Pyridine Aldoxime Methochloride on Cerebral Acetylcholinesterase Activity and Respiration in Cats Poisoned with Sarin," Life Sci. 27, 1869-73.

Musselman, N. P., *et* al. 1967. *The Percutaneous Toxicity of VX Vapor to Clipped, Clothed Rabbits* in *Still* Air (*U*) EATR 4121, Department of the Army, Medical Research Laboratory, Edgewood Arsenal, Md.

Nagy, S., et al. 1946. "The Penetration of Vesicant Vapors into Human Skin," *J. General Physiol. 29,* 441-69.

Nagymajtenyi, L., Selypes, A., and Berencsi, G. 1985. "Chromosomal Aberrations and Fetotoxic Effects of Atmospheric Arsenic Exposure in Mice," *J. Appl. Toxicol. 5,* 61-63.

Namba, T., et al. 1971. "Poisoning Due to Organophosphate Insecticides," *Am. J. Med. 50, 475-92.*

National Council on Radiation Protection and Measurements (NCRP) 1987. *Recommendations on Ionizing Radiation* Exposure *Limits,* NCRP Rept. No. 91, NCRP, 7910 Woodmont Ave., Bethesda, Md.

National Research Council 1982. *Possible Long-Term* Health *Effects of Short-Term Exposure to Chemical Agents. Vol. 1. Anticholinesterases and Anticholinergics,* Panel on Anticholinergic Chemicals, Committee on Toxicology, Board of Toxicology and Environmental Health Hazards, Assembly of Life Sciences, National Academy P, Washington, D.C.

National Research Council 1984. *Disposal of Chemical Munitions and Agents.* Committee on Demilitarizing Chemical Munitions and Agents, Board on Army Science and Technology, Commission on Engineering and Technical Systems, National Academy, Washington, D.C.

National Safety Council (NSC) 1985. "Accident Facts 1984," National Safety Council, Chicago, Ill., as cited in NCRP 1987. NCRP Report No. 91.

Nelson, B., et al. 1983. "Behavioral and Neurochemical Alterations in Offspring of Rats after Maternal Inhalation Exposure to the Industrial Solvent 2-Methoxyethanol," *Teratology 27,* 65A.

Nelson, W. 1981. "Cancer Prevention: Environmental, Industrial, and Occupational Factors," *Cancer 47,* 1065-70.

Nishimoto, Y., et al. 1983. "Long Term Observation of Poison Gas Workers with Special Reference to Respiratory Cancers," *J. Uuoeh. 5* (Suppl.), 89-94.

Nishimoto, Y., et al. 1986. "Cancer of the Respiratory Tract Observed in Workers Having Retired from a Poison Gas Factory," *Gan To Kagaku Ryoho* 13(4), 1144-48 (abstract in English, paper in Japanese).

Nishio, A., and Uyeki, E. M. 1981. "Induction of Sister Chromatid Exchanges in Chinese Hamster Ovary Cells by Organophosphate Insecticides and Their Oxygen Analog," *J. Toxic Environ. Health 8, 939-46.*

Nishioka, H. 1975. "Mutagenic Activities of Metal Compounds in Bacteria," *Mutat. Res.* 31, 185-89.

Nixon, G. A., Tyson, C. A., and Wertz, W. C. 1975. "Interspecies Comparisons of Skin Irritancy," Toxicol. *and Appl. Pharmacol.* 31, 481-90.

Norman, C. 1987. "Regulating Pesticides: The 'Delaney Paradox'," Science 236, 1054-55.

Norman, J. 1975. "Lung Cancer Mortality in World War I Veterans with Mustard-Gas Injury: 1919-1965," *J. Natl. Cancer. Inst. 54,* 311-17.

NTIS 1946. *Chemical Warfare Agents and Related Chemical Problems. Parts III-VI,* Office of Scientific Research and Development, PB 158-507. Taken from HSDB (data base) Hazardous Substances Data Base, Oak Ridge National Laboratory, Oak Ridge, Tenn. 1987. HSN 58,411,46.

O'Leary, J. F., Kunkel, A. M., and Jones, A. H. 1961. "Efficacy and Limitations of Oxime--Atropine Treatment of Organophosphorus Anticholinesterase Poisoning," *J. Pharmacol. Exptl. Therap.* 132, 50-56.

O'Neill, J. J. 1981. "Non-cholinesterase Effects of Anticholinesterases," *Fund. and Appl. Toxicol. 1,* 154-60.

Oberst, F.W., et al. 1968. "Retention of Inhaled Sarin Vapor and Its Effect on Red Blood Cell Cholinesterase Activity in Man," *Clin. Pharmacol. Ther. 9,* 421-27.

Oehme, F. W. 1979. "British Antilewisite (BAL); The Classic Heavy Metal Antidote," pp. *945-952* in *Toxicity of Heavy Metals in the Environment,* Part 2, ed. F. W. Oehme, Marcel Dekker, New York.

Ohno, H., Hanaoka, F., and Yamada, M. 1982. "Inducibility of Sister-Chromatid Exchanges by Heavy-Metal Ions," *Mutat. Res.* 104, 141-45.

Oldiges, H. 1976. "Comparative Studies of the Protective Effects of Pyridinium Compounds Against Organophosphate Poisoning," pp. 101-108 in *Medical Protection Against Chemical-Warfare Agents,* ed. J. Stares, Almqvist & Wiksell International, Stockholm.

Oldiges, H., and Schoene, K. 1970. "Pyridinium and Imidazolium Salts as Antidotes for Soman and Paraoxon Poisoning in Mice," *Arch. of* Toxicol. 26, 293-305.

Opdyke, D. L. J. 1981. "Monographs on Fragrance Raw Materials," *Food* Cosmet. Toxicol. 19, 97-116.

Orr, J. W. 1938. "The Changes Antecedent to Tumor Formation During the Treatment of Mouse Skin with Carcinogenic Hydrocarbons," *J. Parhol. Bacteriol. 46,* 495-515.

Overstreet, D. H., et al. 1974. "Involvement of Muscarinic and Nioctinic Receptors in Behavioral Tolerance to DFP," *Pharmacol.* Biochem. *Behav.* 2, 45-54.

Papirmeister, B., et al. 1984. "Pathology Produced by Sulfur Mustard in Human Skin Grafts on Athymic Nude Mice. 1. Gross & Light Microscopic Changes," *J, Toxicol. Cut. and Ocular* Toxicol. 3, 371-91.

Papirmeister, B., et al. 1985. "Molecular Basis for Mustard-Induced Vesication," *Fund. Appl. Toxicol. 5,* S134-49.

Parker, R. M., May 22, 1987. Pathology Associates, Inc. National Center for Toxicologic Research, Jefferson, Ark., letter *to S. S.* Leffingwell, Special Programs Group, Center for Environmental Health and Injury Control, Centers for Disease Control, Atlanta, Ga.

Patty, F. A., ed. 1962. *Industrial Hygiene and Toxicology,* Vol. II, 2nd ed., rev., Interscience Publishers (John Wiley & Sons), New York, pp. 1275, 1282, and 1778.

Peoples, S. A., et al. 1977. "Poison Exposures of Children in California Due to Ingestion of Liquid-Formulation Pesticides Containing Sodium Arsenite," *Clin. Toxicol. 10, 477.*

Petrunkin, V. E. 1956. "Synthesis and Properties of Dimercapto Derivatives of Alkylsulfonic Acids," *Ukr. Khem. Zh. 22, 603-7,* as cited in Aposhian, H. V., et al. 1984. *Fund. Appl. Toxicol. 4,* S58-70.

Plaa, G., and Duncan, W., eds. *1978. Proceedings of the International Congress on Toxicology, Toxicology as a Predictive Science, lst,* Academic Press, New York, p. 450. Taken from RTECS (data base) Registry of Toxic Effects of Chemical Substances. Oak Ridge National Laboratory, Oak Ridge, Tenn. 1986. SZ9090000.

Polak, R. L., and Cohen, E. M. 1969. "The Influence of Triorthocresylphosphate on the Distribution of (32)P in the Body of the Rat after the Injection of (32)P-Sarin," *Biochem. Pharmacol. 18,* 813-20.

Polak, R. L., and Cohen, E. M. 1970. "The Binding of Sarin in the Blood Plasma of the Rat," *Biochem. Pharmacol.* 19, 877-81.

Prentiss, A. M. 1937. *Chemicals in War,* McGraw-Hill, New York, pp. 148-151.

Radike, M. J., Stemmer, K. L., and Bingham, E. 1981. "Effect of Ethanol on Vinyl Chloride Carcinogenesis," *Environ.* Health *Perspect.* 41, 59-62.

Rao, K. S., et al. 1980. "Teratogenicity and Reproduction Studies in Animals Inhaling Ethylene Dichloride," pp. 149-166 in *Banbury Report 5 Ethylene* Dichloride: *A Potential Health* Risk?, ed. B. Ames, P. Infante, and R. Reitz. Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.

Reichert, D., Henschler, D., and Bannasch, P. 1978. "Nephrotoxic and Hepatotoxic Effects of Dichloroacetylene," Fd. Cosmet. Toxicol. 16, 227-35.

Reichert, D., *et* al. 1980. "Carcinogenic Potential of Dichloroacetylene," pp. 269-272 in *Mechanisms of* Toxicity *and Hazard* Evaluation, ed. B. Holmstedt et al., Elsevier/North Holland Biomedical Press, New York.

Reichert, D., et al. 1983. "Mutagenicity of Dichloroacetylene and its Degradation Products, Trichloroacetyl Chloride, Trichloroacryloyl Chloride and Hexachlorobutadiene," Hut. Res. 117, 21-29.

Reid, B. D., and Walker, I. G. 1969. "The Response of Mammalian Cells to Alkylating Agents II. On the Mechanism of the Removal of Sulfur-Mustard-Induced Cross Links," Biochim. *Biophys. Acta*. 179, 178-88.

Remsen, J. F., Schreider, J. P., and Rosenblatt, L. S. 1984. Mutagenicity *of the Organophosphate Agent, VX,* in Salmonella, Laboratory for Energy-Related Health Research, University of California, Davis, Calif.

Rengstorff, R. H. 1985. "Accidental Exposure to Sarin: Vision Effects," *Arch. Toxicol. 56. 201-03.*

Riccio, E., et al. 1983. "A Comparative Mutagenicity Study of Volatile Halogenated Hydrocarbons Using Different Metabolic Activation Systems," *Environ. Mutagen. 5, 472.*

Rickett, D. L., Glenn, J. F., and Beers, E. T. 1986. "Central Respiratory Effects vs. Neuromuscular Actions of Nerve Agents," Neurotoxicology 7(1), 225-36.

Rickett, D. L., Glenn, J. F., and Houston, W. E. 1987. "Medical Defense Against Nerve Agents: New Directions," Milit. *Med.* 152(1), 35-41.

Robineau, P., and Guittin, P. 1987. "Effects of an Organophosphorus Compound on Cardiac Rhythm and Haemodynamics in Anaesthetized and Conscious Beagle Dogs," *Toxicol. Lett. 37, 95-102.*

Robinson, J. P. 1967. "Chemical Warfare," Science *J. 4, 33-40.*

Rodgers, K. E., et al. 1986. "Rapid In Vitro Screening Assay for Immunotoxic Effects of Organophosphorus and Carbamate Insecticides on the Generation of Cytotoxic T-Lymphocyte Responses," Pest. *Biochem. Physiol. 26,* 292-301.

Romano, Jr., J. A., and Landauer, M. R. 1986. "Effects of the Organophosphorus Compound, 0-ethyl-N-dimethyl-phosphoramidocyanidate (Tabun), on Flavor Aversions, Locomotor Activity, and Rotarod Performance in Rats," *Fund. and Appl. Toxicol. 6, 62-68.*

Rommerein, R., and Hackett, P. 1986. "Evaluation of the Teratogenic Potential of Orally Administered Sulfur Mustard in Rats and Rabbits," *Teratology* 33(3), *70C.*

Ross, R. H., et al. 1982. *Problem Definition Study of Agent VX: Demilitarization, Health Effects, and Toxicology,* ORNL/EIS-200, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Rossman, T. G., et al. 1980. "Absence of Arsenite Mutagenicity in *E. coli* and Chinese Hamster Cells," *Environ. Mutagen. 2,* 371-79.

Rousseaux, C. G. 1987. "The Use of Animal Models for Predicting the Potential Chronic Effects of Highly Toxic Chemicals," *J. Hazard. Mater. 14, 283-92.*

Rowntree, D. W., Nevin, S., and Wilson, A. 1950. "The Effects of Diisopropylfluorophosphonate in Schizophrenia and Manic Depressive Psychosis," *J.* Neurol. *Neurosurg. Psychiat.* 13, 47.

Rozimarek, H., et al. 1973. "Mutagenic Activity in Somatic and Germ Cells Following Chronic Inhalation of Sulfur Mustard," *Mu&a&. Res.* 21, 13-14.

Russell, R. W., and Overstreet, D. H. 1987. "Mechanisms Underlying Sensitivity to Organophosphorus Anticholinesterase Compounds," *Progress in* Neurobiology, 28, 97-129.

Rylands, J. M. 1982. "A Swimming Test for Assessing Effects of Drugs Upon Motor Performance in the Guinea-Pig (Cavia *porcellus*)," *Neuropharmac.* 21, 1181-85.

Salvini, M., Binaschi, S., and Riva, M. 1971. "Evaluation of the Psychophysiological Functions in Humans Exposed to the 'Threshold Limit Value' of 1,1,1-Trichloroethane," *Brit. J. Industr. Med. 28, 286-92.*

Sammet, R. 1983. "Kinetik von 14-Sarin und deren Beeinflussung durch Obidoxim--eine Ganztierautoradiographische Untersuchung an der Maus" Dissertation ETH Nr. 7288, Zurich, as quoted in Hbpff et al. 1984.

Saracci, R. 1981. "The IARC Monograph Program on the Evaluation of the Carcinogenic Risk of Chemicals to Humans as a Contribution to the Identification of Occupational Carcinogens," pp. 165-176 in *Banbury Report Vol. P--Quantification of Occupational Cancer,* ed. R. Peto and M. Schneiderman, Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y.

Sasser, L. B. 1985a. *Toxicology Studies on Sulfur Mustard and Lewisite,* Monthly Progress Report No. 8 (June 11, 1985), Pacific Northwest Laboratory, Richland, Wash.

Sasser, L. B. 1985b. *Toxicology Studies on Sulfur* Mustard *and Lewisite,* Monthly Progress Report (Oct. l-31, 1985), Pacific Northwest Laboratory, Richland, Wash.

Savage, J., and Breckon, G. 1981. "Differential Effects of Sulfur Mustard on S-Phase Cells of Primary Fibroblast Cultures from Syrian Hamsters," *Mutat. Res. 84, 375-87.*

Schoene, K. 1981. *Monogr.* Neurol. *Sci.* 7, as cited in Lenz, D. E., and Maxwell, D. M. 1981.

Schoene, K., and Oldiges, H. 1973. "Die Wirkungen von Pyridiniumsalzen gegenuber Tabun- und Sarinvergiftungen *In* Vivo und *In Vitro," Arch. Int. Pharmacodyn. 204,* 110-23.

Schoene, K., Steinhanses, J., and Oldiges, H. 1976. "Protective Activity of Pyridium Salts Against Soman Poisoning In Vivo and In Vitro," *Biochem. Pharmacol. 25,* 1955-58.

Schreider, J. P., et al. 1985. *The Teratologic Effects of VX in Rats,* Laboratory for Energy-Related Health Research, University of California, Davis, Calif.

Selby, L. A., et al. 1971. "Outbreak of Swine Malformations Associated with the Wild Black Cherry, *Prunus serotina," Arch. Environ.* Health 22, 496-501.

Sellakumar, A. R., et al. 1985. 'Carcinogenicity of Formaldehyde and Hydrogen Chloride in Rats," *Toxicol. and Appl. Pharmacol.* 81, 401-06.

Senanayake, N., and Karalliedde, L. 1987. "Neurotoxic Effects of Organophosphorous Insecticides: An Intermediate Syndrome," *N. Engl. J. Med.* 316. 761-63.

Sevaljevic, L., et al. 1981. 'The Effect of Organophosphate Poisoning on Plasma Cyclic AMP in Rats," *Biochem. Pharmacol.* 30(19), 2725-27.

Shalamberidze, 0. P., and Tsereteli, N. T. 1971. "Effect of Low Concentrations of Sulfur and Nitrogen Dioxides on the Estrual Cycle and Reproductive Functions of Experimental Animals,' Hygiene *and Sanitation (Gigiena i* Sanitariya) 36, 178-81.

Shapiro, R. 1977. "Genetic Effects of Bisulfite (Sulfur Dioxide)," *Mutat. Res. 39,* 149-76.

Shifrine, M., Schreider, J. P., and Rosenblatt, L. S. 1983. *The Effect of Agent VX on* Mitotic *Recombination in* Saccharomyces cerevisiae, Laboratory for Energy-Related Health Research, University of California, Davis, Calif.

Shimizu, H., et al. 1985. "The Results of Microbial Mutation Test for Forty-Three Industrial Chemicals," *Jpn. J. Ind. Heal&h 27, 400-19.*

Sidell, F. R. 1974. "Soman and Sarin: Clinical Manifestations and Treatment of Accidental Poisoning by Organophosphates," *Clin. Toxicol.* 7(1), l-17.

Sidell, F. R. 1976. "Clinical Aspects of Intoxication by Cholinesterase Inhibitors," pp. *22-35* in *Medical Protection Against Chemical-Warfare Agents,* ed. J. Stares, Almqvist & Wiksell International, Stockholm.

Sidell, F. R. 1986. General Guidelines *for* Therapy of *Acetylcholinesterase Intoxication* (course handout), Biomedical Laboratory, Medical Research Division, Aberdeen Proving Ground, Md.

Sidell, F. R. 1987. Untitled manuscript available from the U.S. Army, Program Executive Office, Office of the Program Manager for Chemical Demilitarization, PE-PMCD (M. Tishbin), Aberdeen Proving Ground, Md. 21010-5401.

Sidell, F. R., and Groff, W. A. 1974. "The Reactivatibility of Cholinesterase Inhibited by VX and Sarin in Man," *Toxicol. and Appl. Pharmacol. 27, 241-52.*

Sim, V. M. 1962. *Variability of Different Intact Human-Skin Sites to the* Penetration of VX, U.S. Army Chemical Research and Development Laboratories Technical Report CRLDR 3122, as quoted in Craig, Cummings, and Sim 1977.

Sim, V. M. 1971. "Chemicals Used as Weapons in War," Chap. 59, pp. 1232-1248 in Drill's Pharmacology in *Medicine*, ed. J. R. DiPalma, 4th ed.. McGraw-Hill Book Co.

Simeon, V., et al. 1979. "1,3-Bispyridinium-Dimethylether Mono- and Dioximes: Synthesis, Reactivating Potency and Therapeutic Effect in Experimental Poisoning by Organophosphorus Compounds," *Arch. of Toxicol.* 41, 301-06.

Singer, A. W., et al. 1987. "Cardiomyopathy in Soman and Sarin Intoxicated Rats," Toxicol. *Lett. 36, 243-49.*

Singh, A. K., Zeleznikar, R. J., Jr., and Drewes, L. R. 1986. "Protection from Quinidine or Physostigmine Against In Vitro Inhibition by Sarin of Acetylcholinesterase Activity," *Life Sci.* 38(2), 165-72.

Sittig, M. 1981. *Handbook of Toxic and Hazardous* Chemicals, Noyes Publications, Park Ridge, N.J., pp. 20-21.

Sivam, S. P., Hoskins, B., and Ho, I. K. 1984. "An Assessment of Comparative Acute Toxicity of Diisopropylfluorophosphate, Tabun, Sarin, and Soman in Relation to Cholinergic and GABAergic Enzyme Activities in Rats," *Fund. and Appl. Toxicol. 4, 531-38.*

Small, *M.* J. 1984. *Compounds Formed from the* Chemical *Decontamination of HD, GB, and VX and Their Environmental Fate.* Technical Report 8304, U.S. Army Medical Bioengineering Research & Development Laboratory, Fort Detrick, Frederick, Md; AD A149515.

Smyth, H. F., et al. 1954. "Range-Finding Toxicity Data: List V," *A. M. A. Arch. Ind. Hyg. Occup. Med. 10,* 61-68.

Smyth, H. F., Jr., et al. 1962. "Range-Finding Toxicity Data: List VI," *Am. Ind. Hyg. Assoc. J. 23,* 95-107.

Snyder, W. S., et al. 1975. *Report of the Task Group on Reference Man,* International Commission on Radiological Protection Report No. 23, Pergamon Press, Oxford, pp. 348-349, 357-358.

Sollman, T. H. 1957a. "Chlorosulfides, Chloroethylamines, Ethyleneimines (Sulfur and Nitrogen Mustards): Melamine," pp. 193-201 in Sollman, T. H. *A Manual of Pharmacology and its Applications to Therapeutics and Toxicology,* 8th ed., W. B. Saunders Co., Philadelphia.

Sollman, T. H. 1957b. "Lewisite," pp. 192-193 in Sollman, T. H. *A Manual of Pharmacology and its Applications to Therapeutics and Toxicology,* 8th Ed., W. B. Saunders Co., Philadelphia.

Spector, W. S., ed. 1956. *Handbook of* Biological *Data,* Table 156, p. 187. W. B. Saunders Co., Philadelphia.

Spencer, H. C., et al. 1951. "Vapor Toxicity of Ethylene Dichloride Determined by Experiments on Laboratory Animals ," *A. M. A. Arch. Ind. Hyg. Occup. Med. 4, 482-93.*

Stares, J. E. S. 1976. "Medical Protection Against Chemical-Warfare Agents," pp. 157-166 in *Medical Protection Against Chemical-Warfare Agents,* ed. J. Stares, Almqvist & Wiksell International, Stockholm.

Stewart, R. D., *et* al. 1972. "Carboxyhemoglobin Elevation after Exposure to Dichloromethane," *Science* 176, 295-96.

Stewart, W. *C. 1956. The Effects of Varying Concentrations of GA and GB, as Compared with VE and VG on Neuromuscular Conduction in the Isolated Diaphragm of the Rat,* Canada, Suffield Experimental Station, STP 79 (Secret), as quoted in Fielding 1960.

Stoller, A., *et* al. 1965. "Organophosphorus Insecticides and Major Mental Illness: An Epidemiological Investigation," *Lancet* 1, 1387-88.

Streissguth, A. P., et al. 1980. "Teratogenic Effects of Alcohol in Humans and Laboratory Animals," *Science 209,* 353-61.

Tai, C. C., and **Ting**, R. C. 1979. "Induction of Increased Frequencies of Sister Chromatid Exchange in V79 Cells by Butyric Acid," *In Vitro* 15, 172.

Tatken, R. L., and Lewis, Sr., R. J., eds. 1983. *Registry of Toxic Effects of Chemical Substances,* 1981-82, Vol. 3, NIOSH, U.S. Department of Health and Human Services, pp. xxvii and xxxii, PB 85-218071.

Taylor, P. 1980. "Anticholinesterase Agents," pp. 100-119 in Goodman and *Gilman's The Pharmacological Basis of Therapeutics,* Macmillan Publishing Co., New York.

Thorpe, J. F., and Whiteley, M. A., eds. 1939. *Thorpe's Dictionary of Applied Chemistry,* Vol. 3, 4th ed., Longmans, Green and Co., New York, p. 8.

Tokuoka, S., et al. 1986. "Early Cancer and Related Lesions in the Bronchial Epithelium in Former Workers of Mustard Gas Factory," *Acta Pathol. Jpn 36, 533-42.*

Topham, J. C. 1981. "Evaluation of Some Chemicals by the Sperm Morphology Assay," pp. 18-20 in *Evaluation of Short-Term Tests for Carcinogens. Report of the International Collaborative Program,* ed. F. J. de Serres and J. **Ashby**, Elsevier/North-Holland, New York *(Progress in Mutation Research,* Vol. 1).

Torkelson, T. R., Oyen, F., and Rowe, V. K. 1976. "The Toxicity of Chloroform as Determined by Single and Repeated Exposure of Laboratory Animals," Am. *Ind. Hyg. Assoc. J. 37,* 697-705.

Torkelson, T. R., and Rowe, V. K. 1981. "Halogenated Aliphatic Hydrocarbons Containing Chlorine, Bromine, and Iodine," pp. 3433-*3602* in *Patty's Industrial Hygiene and Toxicology,* 3rd ed., rev., vol. 2B, Toxicology, ed. G. D. Clayton and F. E. Clayton, John Wiley & Sons, New York, p. 3463.

Towill, L. E., et al. 1978. Reviews *of the Environmental Effects of Pollutants: 5. Cyanide,* EPA-600/l-78-027, U.S. Environmental Protection Agency, Cincinnati, pp. 7, 127.

Trapp, R. 1985. *The* Detoxification *and Natural Degradation of Chemical Warfare Agents,* Stockholm International Peace Research Institute, Taylor & Francis, London.

Travis, C. C., et al. 1987. "Cancer Risk Management: A Review of 132 Federal Regulatory Decisions," *Envir.* Sci. & *Tech. 21, 415-20.*

Trosko, J. E., et al. 1981. "In Vitro Assay for Tumor Promoters," pp. *420-27* in *Short-Term Tests for Chemical* Carcinogens, ed. H. F. Stich and R. H. C. San, Springer-Verlag, New York.

Tsuda, H., et al. 1981. "Chromosomal Aberrations and Sister-Chromatid Exchanges Induced by Gaseous Nitrogen Dioxide in Cultured Chinese Hamster Cells," *Mutat. Res. 89,* 303-9.

U.S. Department of the Army 1974. *Chemical Agent Data Sheets, Vol. 1* Technical *Report,* EO-SR-74001, (Edgewood Arsenal Special Report) Defense Technical Info. Ctr., Alexandria, Va.

U.S. Department of the Army 1975. Military Chemistry *and Chemical Compounds,* FM 3-9/AFR355-7.

U.S. Department of the Army 1979. *Safety Regulations for Chemical Agents H, HD and HT,* DARCOM-R 385-31, H.Q., U.S. Army Material Development and Readiness Command, Alexandria, Va.

U.S. Department of the Army 1982. Safety Regulations *for Chemical Agents GB and VX,* DARCOM-R-385-102, U.S. Army Materiel Development and Readiness Command, Alexandria, Va.

U.S. Department of the Army and U.S. Department of the Air Force 1975. Military *Chemistry and Chemical Compounds.* Field *Manual.* Army 9, Air Force AFR 355-7, Department of the Army Headquarters, Washington, D.C.

U.S. EPA 1979. "National Interim Primary Drinking Water Regulations; Control of Trihalomethanes in Drinking Water. Final Rule," *Fed Reg.* 44(231), 68696-98, 68703.

U.S. EPA 1985. "National Primary Drinking Water Regulations; Synthetic Organic Chemicals; Inorganic Chemicals and Microorganisms. Proposed Rule," *Fed. Reg.* 50(219), 46957.

U.S. EPA 1986a. *Health Assessment Document for Beryllium. Review Draft,* EPA 600/8-84/026B, U.S. Environmental Protection Agency, Research Triangle Park, N.C., pp. 7-77 to 7-80.

U.S. EPA 1986b. "Hazardous Waste Management System; Identification and Listing of Hazardous Waste; Notification Requirements; Reportable Quantity Adjustments; Proposed Rule," *Fed. Reg.* 51(114), 21666.

Van Abbe, N. J., et al. 1982. "Bacterial Mutagenicity Studies on Chloroform In Vitro," *Food Chem. Toxicol. 20,* 557-61.

van Helden, H. P. M., van Der Wiel, H. J., and Wolthuis, 0. L. 1983. "Therapy of Organophosphate Poisoning: The Marmoset as a Model for Man," *Br. J. Pharmac. 78, 579-89.*

Van Hooidonk, C., et al. 1980. "Rate of Skin Penetration of Organophosphates Measured in Diffusion Cells," pp. 643-46 in *Mechanisms of* Toxicity *and Hazard Evaluation* Elsevier/North-Holland Biomedical Press.

Van Hooidonk, C., et al. *1983. CW Agents and the Skin. Penetration and Decomposition. Proc. Int. Symp. Protection Against Chemical Warfare Agents,* Stockholm, Sweden, June 6-9, 1983, pp. 153-60.

Van Kampen, K. R., et al. 1969. "Organic Phosphate Poisoning of Sheep in Skull Valley, Utah," J.A.V.M.A. 154(6), 623-630.

Van Kampen, K. R., et al. 1970. "Effects of Nerve Gas Poisoning in Sheep in Skull Valley, Utah," *J.A.V.M.A.* 156(1), 1032-35.

Van Meter, W. G., Karczmar, A. G., and Fiscus, R. R. 1978. "CNS Effects of Anticholinesterases in the Presence of Inhibited Cholinesterases," *Arch.* int. Pharmacodyn. 231, 249-60.

Velazquez, A., et al. 1986. "Indication for Weak Mutagenicity of the Organophosphorus Insecticide Dimethoate in *Drosophila Nelanogaster," Mutat. Res. 41, 372-77.*

Velazquez, A., et al. 1987. "Lack of Mutagenicity of the Organophosphorus Insecticide Malathion in *Drosophila melanogaster," Environ. Uutagen. 9, 343-48.*

Vestweber, J. G., and Kruckenberg, S. M. 1972. "The Effect of Selected Organophosphorus Compounds on Plasma and Red Blood Cell Cholinesterase in the Dog," *Vet Med. Small Anim. Clin. 67, 803-6.*

Vogt, R., et al. 1984. "Pathogenesis of Skin Lesions Caused by Sulfur Mustard," *Fund. & Appl. Toxicol. 4,* 571-83.

Vojvodic, V., et al. 1985. "The Protective Effect of Different Drugs in Rats Poisoned by Sulfur and Nitrogen Mustards," *Fund. Appl.* Toxicol. *5,* S160-68.

Volpe, L. S., Biagioni, T. M., and Marquis, J. K. 1985. "In Vitro Modulation of Bovine Caudate Muscarinic Receptor Number by Organophosphates and Carbamates," *Toxicol. and Appl. Pharmacol. 78, 226-34.*

Voroshilin, S. I., Plotko, E. G., and Nikiforova, V. Ya. 1975. "Mutagenic Effect of Hydrogen Fluoride on Animals," *Cytology* and Genetics 9, 40-42.

Vozovaya, M. 1974. "Development of Posterity of Two Generations Obtained from Females Subjected to the Action of Dichloroethane," *Cigiena I. Sanitariya* 7, 25-28, as cited in *Reproductive Hazards of Industrial Chemicals,* ed. S. Barlowk and F. Sullivan, Academic Press, New York, 1982, pp. 310-15.

Vranken, M. A., De Bisschop, H. C., and Willems, J. L. 1982. "In Vitro Inhibition of Neurotoxic Esterase by Organophosphorus Nerve Agents," *Arch. Int. Pharmacodyn. Ther.* 260(2), *316-18.*

Wada, S., et al. 1962a. "Malignant Respiratory Tract Neoplasms Related to Poison Gas Exposure," *Hiroshima J. Med. Sci.* 11(3), 81-91.

Wada, S., et al. 1962b. "Review of Okuno-jima Poison Gas Factory Regarding Occupational Environment," Hiroshima *J. Med.* Sci. 11(3), *75-80.*

Wada, S., et al. 1968. "Mustard Gas as a Cause of Respiratory Neoplasia in Man," *Lancet* 1 (553), 1161-63.

Wan, B., Christian, R. T., and Soukup, S. W. 1982. "Studies *of* Cytogenetic Effects of Sodium Arsenicals on Mammalian Cells In Vitro," Environ. *Mutagen. 4, 493-98.*

Wands, R. C. 1981. "Alkaline Materials," pp. *3045-70* in *Patty's Industrial Hygiene and Toxicology,* 3rd ed., rev., vol. 2B, *Toxicology,* ed. G. D. Clayton and F. E. Clayton, John Wiley 6 Sons, New York, pp. 3048-52.

Ward, D. M. *1970. Maximum Allowable Concentration of Agents in Drinking Water,* SMURA-RILO-TIC, Toxicological Information Center, ILO/RSCH Laboratories.

Watson, A. P., Jones, T. R., and Griffin, G. In review. "Sulfur Mustard as a Carcinogen: Application of Relative Potency Analysis to the Chemical Warfare Agents H, HD, and HT."

Weger, N., and Szinicz, L. 1981. "Therapeutic Effects of New Oximes, Benactyzine and Atropine in Soman Poisoning: Part I. Effects of Various Oximes in Soman, Sarin, and VX Poisoning in Dogs," *Fund. and Appl.* Toxicol. 1, 161-63.

Weidler, D. J. 1974. "Myocardial Damage and Cardiac Arrhythmias After Intracranial Hemorrhage. A Critical Review," *Stroke 5, 759-64.*

Weimer, J. T., et al. 1979. *Proposed Revision of Limits of Human Exposure to GB Vapor in Nonmilitary Operations Based on One-Year Exposures to Laboratory Animals* to *Low Airborne Concentrations,* ARCSL-TR-78056, U.S. Army Armament Research and Development Command, Chemical Systems Laboratory, Aberdeen Proving Ground, Md.

Weiss, A. 1959. "Kampfstoffe als Carcinogene Substanzen," *Conf. Nordwestdeutschen Gesell. Inn. Med.* [*Proc.*] *51st, 1958,* Hansisches Verlagskontor H. Scheffler, Lubeck, as cited in K. Lohs 1975. *Delayed* Toxic *Effects of Chemical Warfare-Agents: A SIPRI Monograph,* Almquist and Wiksell International, Stockholm.

Whong, W. Z., Specinor, N. D., and Edwards, G. D. 1979. "Mutagenicity Detection of In Vivo Nitrosation of Dimethylamine by Nitrite," *Environ. Mutag.* 1, 277-82.

Wiles, J. S., and Alexander, T. B. 1960. *Comparative Toxicity of VX Applied to the Unclipped and Clipped* Skin *of Bare and Clothed Rabbits* (*U*) AD839329, Directorate of Medical Research, U.S. Army Chemical Research & Development Laboratories, Army Chemical Center, Md.

Wilhelm, K., et al. 1979. "Protective Effect of Dexetimide and HI-6 in Poisoning with Highly Toxic Organophosphorus Compounds," *Arhiv za Higijenu Rada* i *Toksikologiju 30,* 147-51.

Willems, J. L., Nicaise, M., and De Bisschop, H. C. 1984. "Delayed Neuropathy by the Organophosphorus Nerve Agents Soman and Tabun," Arch. *Toxicol. 55, 76-77.*

Wilson, B., et al. 1984. *The Action of a Single VX Dosage in Chickens,* Laboratory for Energy-Related Health -Research, University of California, Davis, Calif.

Windholz, M., et al. 1983. *The Merck Index. An Encyclopedia of Chemicals, Drugs*, and *Biologicals*, Merck and Co., Rahway. N. J. pp. 34-35, 229, 230, 234, 385, 540-41, 692, 696, 947, 1059, 1235, 1288-9, 1375, and 1430.

Wolfsie, J. H., and Winter, G. D. 1952. "Statistical Analysis of Normal Human Red Blood Cell and Plasma Cholinesterase Activity Values," *Arch. Ind. Hyg. Occup.* Med. 6, 43-49.

Wolthuis, 0. L., and Meeter, E. 1968. "The Effects of DFP on the Rat Heart," *Ind. Med.* Surg. 37, 535.

Wolthuis, 0. L., and Vanwersch, R. A. P. 1984. "Behavioral Changes in the Rat after Low Doses of Cholinesterase Inhibitors," *Fund. and Appl.* Toxicol. 4, S195-208.

Worthley, E. G. 1971. "Uptake and Distribution of VX in Plants," EATM 100-15, Edgewood Arsenal Technical Memo, Department of the Army, Medical Research Laboratory, Edgewood Arsenal, Md.

Wulf, H., et al. 1985. "Sister Chromatid Exchanges in Fishermen Exposed to Leaking Mustard Gas Shells," *Lancet 1* (8430), 690-91.

Wysocki, J., Kalina, Z., and Owczarzy, I. 1987. "Effect of Organophosphoric Pesticides on the Behaviour of NBT-Dye Reduction and E Rosette Formation Tests in Human Blood," *Int. Arch. Occup. Environ. Health 59,* 63-71.

Yamakido, M., et al. 1985a. "Study of the Genetic Effects of Sulfur Mustard Gas on Former Workers of Ohkunojima Poison Gas Factory and Their Offspring," *Hiroshima J. Med.* Sci. 34, 311-22.

Yamakido, M., et al. 1985b. "The Causes of Death in the Retired Workers of Okuno-Jima Poison Gas Factory," *Jpn. J. Med.* 24, 394.

York, R. G., et al. 1982. "Evaluation of Teratogenicity and Neurotoxicity with Maternal Inhalation Exposure to Methyl Chloroform," *J. Toxicol. Environ. Health 9,* 251-66.

Young, L. 1947. *Observations on the Effects of Mustard Gas on the Rat," *Canad. J. of Res.* Section E, Medical Sciences, 25, 141-51.

Zeiger, E. 1987. "Carcinogenicity of Mutagens: Predictive Capability of the *Salmonella* Mutagenesis Assay for Rodent Carcinogenicity," *Cancer Res.* 47, 1287-96.

Zhao, D.-L., et al. 1983. "Effects of Soman, Sarin, and VX on the Specific Binding of 3H-QNB in Rat Cerebral *Cortex* Homogenates," *Acta Pharmacologica Sinica* 4(4), *225-28.*

Appendix C

DEMILITARIZATION PLANT DESIGN

C.1 INTRODUCTION

The Johnston Atoll Chemical Agent Disposal System (JACADS) technology is used as the basis for disposal of the stockpile in the Chemical Stockpile Disposal Concept Plan (U.S. Army 1986). The JACADS process uses thermal destruction to destroy the chemical agents contained in the munitions. The munition propellants, explosives, fuzes, and associated dunnage are also thermally destroyed, and the metal components of each munition are thermally treated to ensure complete agent destruction. Several types of furnaces are used, depending on the types of munition that is being processed.

All five of the chemical agents (GB, VX, mustard, GA, and Lewisite) are combustible and have relatively high adiabatic flame temperatures. However, the incineration process must be controlled to ensure complete combustion of the agent and to control the combustion products. Combustion products are processed through pollution abatement systems to remove particulates and acidic gases from the furnace exhaust before discharge to the atmosphere. The pollution abatement systems for the incinerators (except for the dunnage incinerator) include quench towers, venturi scrubbers, packed scrubber towers, and mist eliminators. Liquid wastes from the pollution abatement systems are concentrated and dried, and the residues are loaded into containers for disposal in an approved landfill.

The design for the Chemical Stockpile Disposal Program (CSDP) facilities represents the current state of the art in large-scale toxic chemical destruction technology. Toxic materials are confined within specific areas of the plant. This design protects plant workers and the environment from exposure to toxic chemical agents.

Figure C.1 is an artist's concept of the proposed plant based on the JACADS design. The plant site will be enclosed by a double security fence and will have a guard house to ensure controlled access, which would be strictly enforced. The demilitarization facility will include a disposal building that will house the entire disposal process (note the adjacent air-pollution-control equipment); a laboratory; a multipurpose building (which will include personnel facilities, a maintenance area, and emergency medical facilities); an engineering office area; a boiler house; and a standard reinforced concrete igloo to be used to store a portion of the munitions ready for disposal. Revisions to the JACADS design will be made as necessary for site adaptation (e.g., weather enclosures for the pollution control equipment and the liquid waste treatment system).

C.2 PLANT DESCRIPTION

The heart of the facility is the munitions demilitarization building (MDB), which typically contains four incinerators supported by equipment designed to prepare the munitions for deactivation and detoxification. The JACADS design will be modified as appropriate for the inventory at each installation (e.g., Aberdeen Proving Ground would have only two

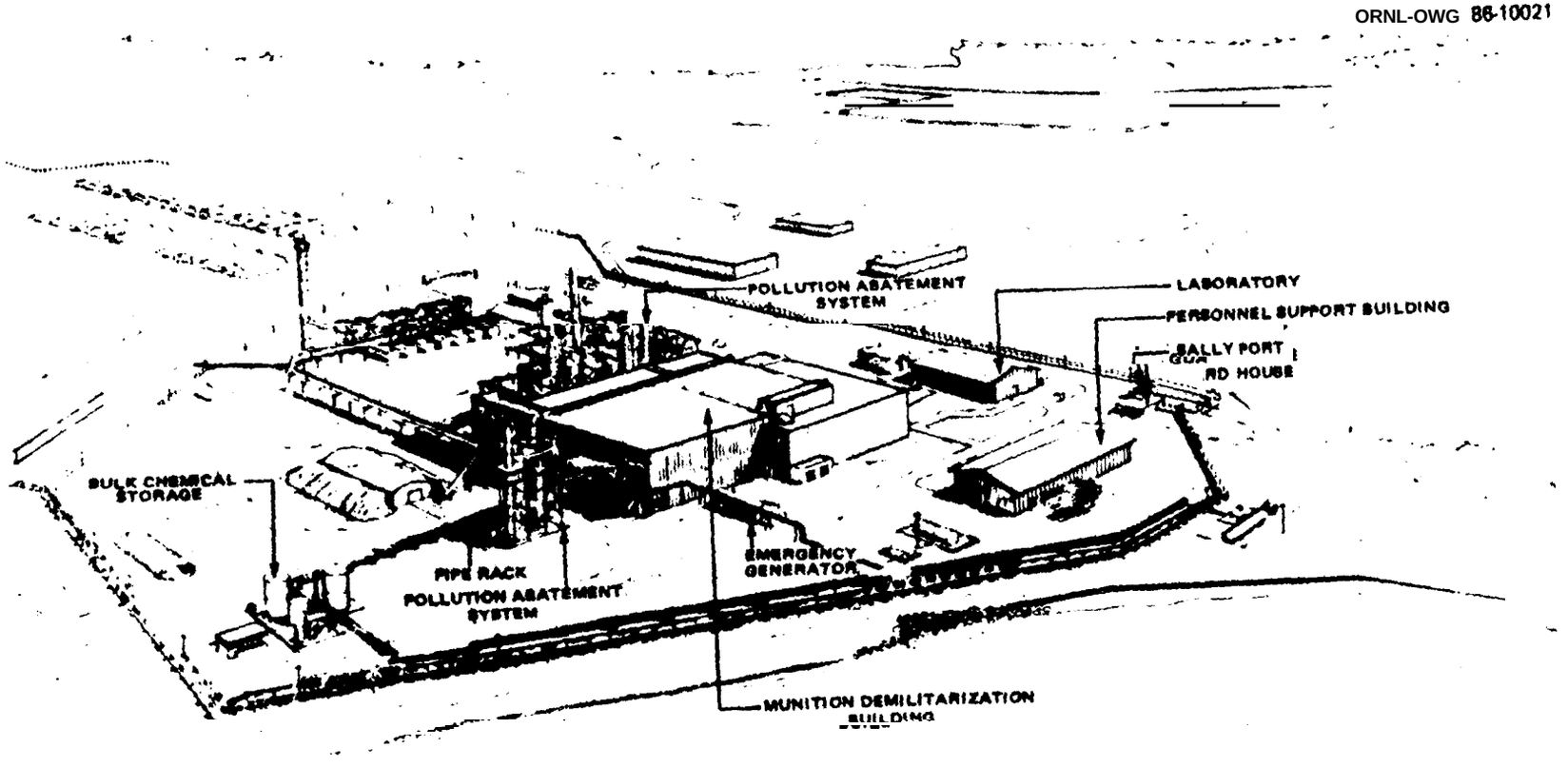


Fig. C.1. Artist's concept of proposed chemical disposal facility (based on JACADS).

incinerators because only ton containers of mustard would be processed in the on-site disposal alternative). Figure C.2 presents the overall flow of the munitions demilitarization process. Axonometric drawings of the first and second floor of the JACADS process building are shown in Figs. C.3 and C.4. The CSDP plants will be similar to JACADS. These figures identify the major processing areas and also identify potential agent contamination zones. The brine reduction area (i.e., the liquid waste treatment system) and the air pollution abatement systems for the incinerators are adjacent to the building. The destruction facility is designed to prevent the escape of agent vapor into the atmosphere. This safeguard is achieved through the design of ventilation and filter *systems,* airlocks, explosive-containment walls, and special partitions.

The destruction facility processes only one chemical agent at a time. Agent detection/monitoring equipment is specifically configured for the agent being destroyed. An important, integral feature of the demilitarization process is that each type of munition has its own individually tailored, computer-controlled program for management of the destruction process. The computer programs are written to oversee all process steps and include an extensive network of interlocks and program safeguards. Additionally, emergency response information is provided by the programs. All programs are extensively checked during a year of preoperational testing.

C.2.1 MUNITIONS TRANSFER FROM STORAGE

The munitions to be demilitarized are listed in Appendix A. When the munitions are delivered to the munitions holding igloo (MHI) the physical and accounting responsibility will be transferred from the storage account to a demilitarization account, and after certification of demilitarization the items will be removed from the Army's inventory of lethal chemical agents and munitions.

Munitions destined for demilitarization are designated by the Department of the Army. Designated munitions are removed from the installation's chemical surety materiel storage area at a rate compatible with the operating schedule of the facility to preclude accumulation of an inventory within the MDB greater than that equivalent to 4 h of operating time.

Chemical munitions are stored in configurations generally suitable for transport during wartime (i.e., boxes, protective tubes, or metal overpacks on pallets). Munitions and bulk containers (except the ME-116 weteye and ME-94 (500-lb) bombs, and the TMU-28/B spray tanks) will be transported from the storage area to the MDB in a specially designed on-site transport container which provides protection in case of an accident. For analysis purposes in the FPEIS, the on-site transport container was assumed to hold one pallet of munitions or one bulk container. The storage area and munitions are monitored for agent. Munitions which have been verified as not leaking will be moved from their storage location in their storage pallets or boxes and placed in

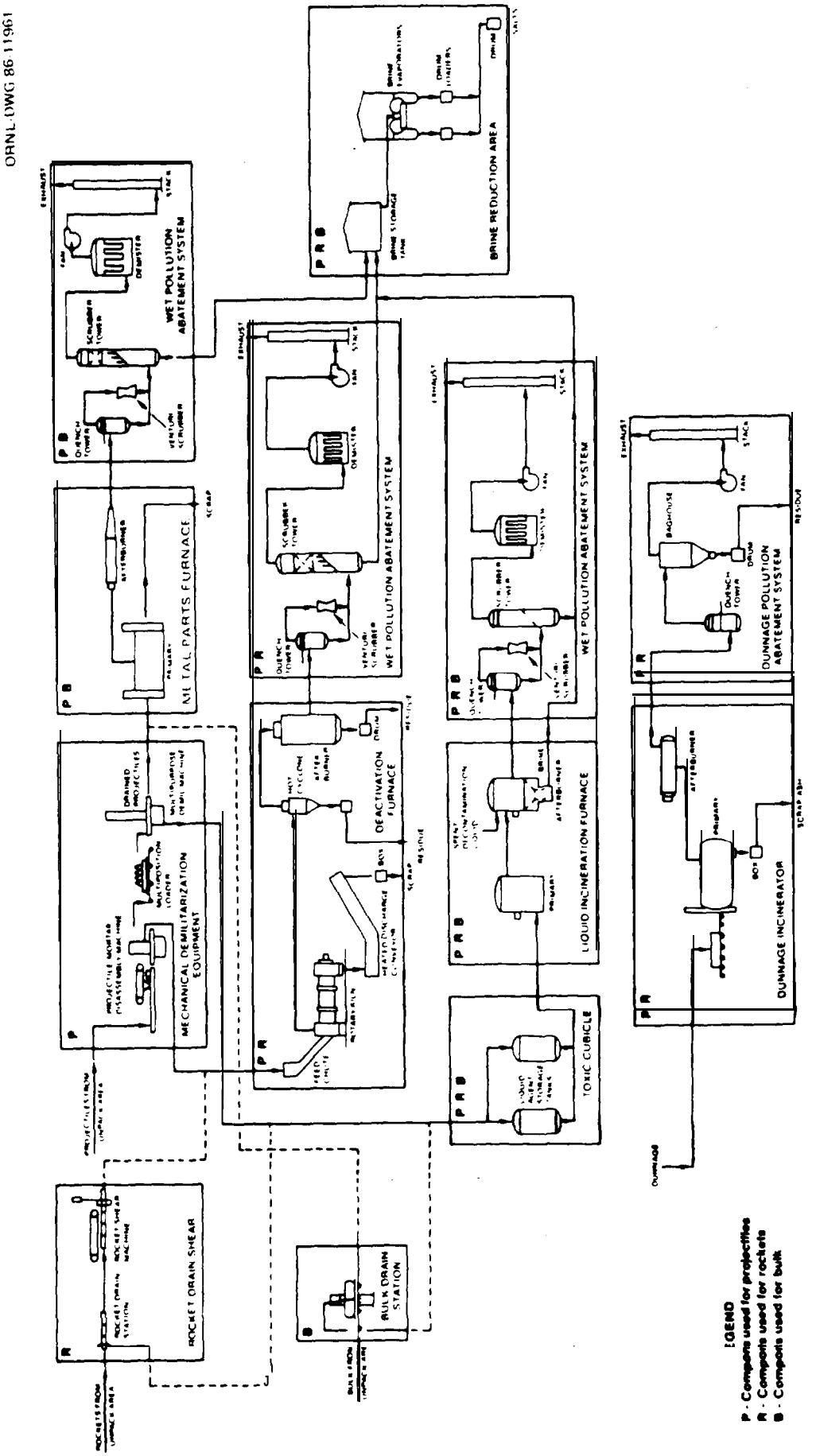


Fig. C.2. Munitions processing flows.

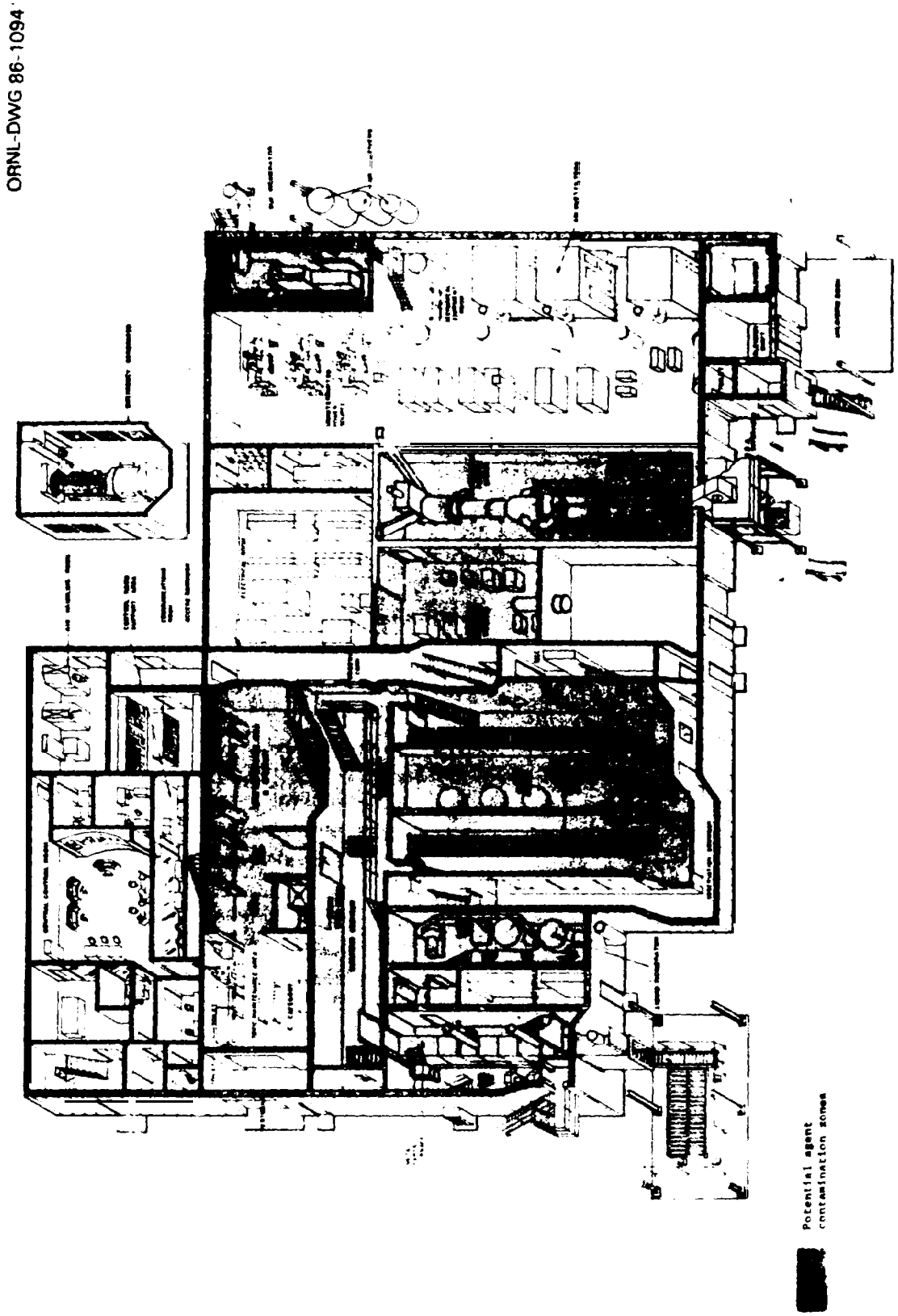


Fig. C.3. JACADS munition demilitarization building, first floor.

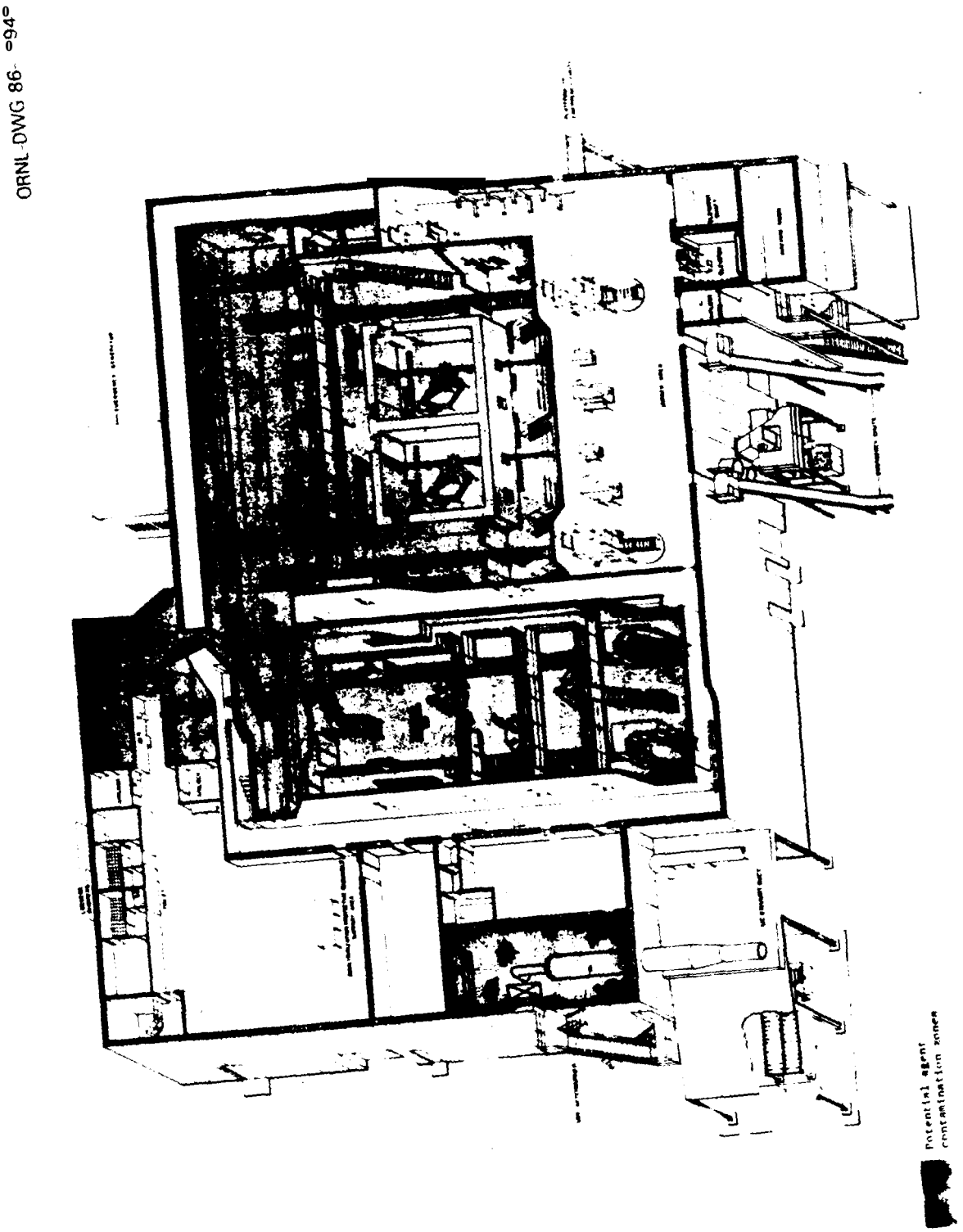


Fig. C.4. JACADS munition demilitarization building, second floor.

the transport container using an electric forklift. Loading will occur on the pad in front of the storage igloo (i.e., the igloo apron). The M55 rockets will be placed first in a specially designed overpack called the single pallet only rocket transporter (SPORT); the SPORT will be loaded into the on-site transport container. (The SPORT is vapor tight under normal handling but does not provide collision or fire protection.) If any leaking munitions are found, they will be decontaminated and packaged in a munition-specific steel overpack which provides agent vapor and liquid containment. The overpacked leakers would be placed in the transport container. The transport containers are loaded on a flatbed truck. The MK-116 weteye and the MK-94 (500-lb) bombs, and the TMU-28/B spray tanks will be transported on-site in the protective steel overpacks in which they are stored (i.e., without the transport container).

At the demilitarization site, the munitions are inventoried and logged into the demilitarization account. The transport containers are removed from the truck by personnel using a forklift and are transferred to the MHI for temporary storage *or* directly to the MDB.

C.2.2 MUNITIONS PROCESSING PRIOR TO INCINERATION

The lethal chemical agents and munitions in the on-site transportation container are removed from the MHI and brought by forklift to the MDB, where they are placed in an elevator (Fig. C.3) and raised to the second floor (Fig. C.4). On the second floor, the transport container is moved from the elevator into the unpack area by a battery-powered forklift.

The transport container is unpacked. Palletized munitions are unbanded and placed onto a loading table feeding a conveyor system (Fig. C.5). An automated loader/airlock then sets the munitions onto the conveyor. Burstered munitions (i.e., munitions containing explosives) are transferred by conveyer into the explosive containment room vestibule (ECV) (Fig. C.4) and bulk items to the munitions processing bay. The vestibule can be used for preparing overpacked, leaking munitions, if necessary. Leaking munitions are delivered to the MDB in overpacks and are manually removed from the overpacks in the ECV only and inserted into the process by operators dressed in protective clothing. Passing through the vestibule, burstered munitions move through blast doors into one of two explosive containment rooms (ECRs) (Fig. C.4). These rooms are reinforced concrete enclosures designed to totally contain the effects of an accidental explosion. Processing of munitions with explosive components is accomplished in the rooms by the rocket punch and drain system and the rocket shear machine; the projectile/mortar disassembly machine and the burster size reduction machine; or the mine punch and drain system and mine machine for burster removal.

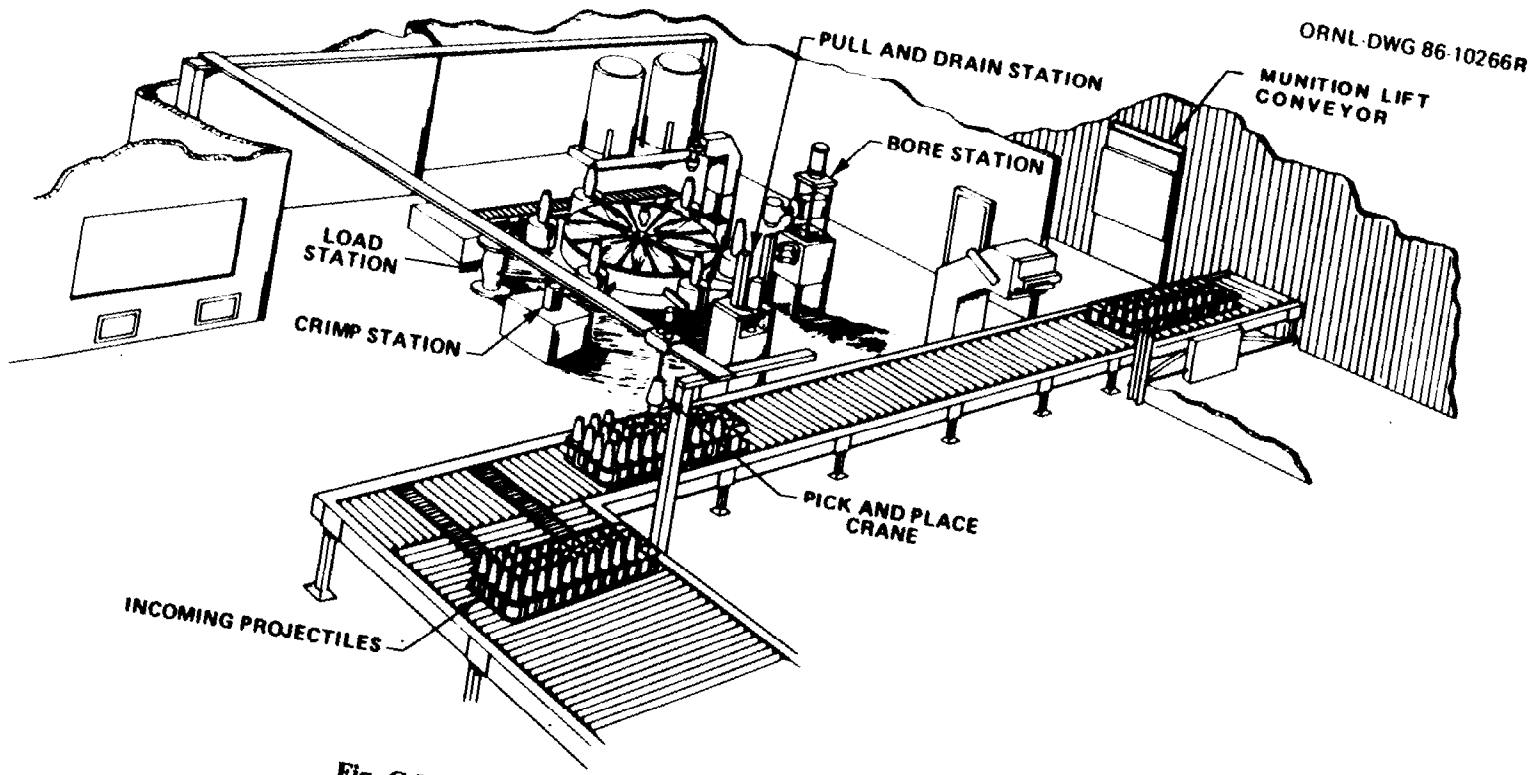


Fig. C.5. Artist's concept of process operations conveyors.

C.2.2.1 Rocket Processing

Once inside the ECR, rockets are punched and drained of the lethal chemical agent and then sheared into five sections using the rocket shear machine. The pieces slide down a chute, through blast gate valves, and into the deactivation furnace system, located on the first floor (see Sect. C.2.3.3). The propellant and explosive components are thermally destroyed, and any residual agent remaining in the rocket cavity is incinerated.

The agent drained from rockets is pumped to a short-term storage tank located in the toxic cubicle and later transferred to the liquid incinerator (see Sect. C-2.3.1) for destruction. Pallets and packing materials are conveyed to the dunnage incinerator (see Sect. C.2.3.4).

C.2.2.2 Projectile Processing

Burstered projectiles are processed by the projectile/mortar disassembly machine located in the ECR. A rotating table incorporated into the machine advances the projectiles through several separate work stations, where the following operations are performed: (1) nose closure/fuse removal, (2) supplemental charge removal, and (3) burster removal. All parts and pieces removed from projectiles are fed into the deactivation furnace system (see Sect. C.2.3.3). However, the bursters from 155-mm and 8-in. projectiles are automatically sheared by machine before being sent to the incinerator.

The projectile bodies are then conveyed out of the ECR to a tilting conveyor that sets them upright for manipulation by a multiposition loader, which places them into unit pallet assemblies after burster removal has been verified. These unit pallet assemblies carry the projectiles throughout the remaining processing steps.

The deburstered projectiles in the unit pallet assemblies are transferred on conveyors to the munitions corridor, where they are placed onto a charge car. The charge car moves along the corridor, loading the unit pallet assemblies either onto the tray buffer tables or onto conveyors leading to the munitions processing bay (Fig. C.4). Three multipurpose demilitarization machines located in the bay have a pick-and-place mechanism that lifts a munition out of the pallet, places it on an operating turntable, and returns a completed munition to the pallet. The operations on the turntable include (1) removal of a portion of the metal from the top of a welded or stuck burster well by a milling head (if required), (2) removal of the burster well, (3) draining of the chemical agent, (4) crimping of the burster well, and (5) replacing the burster well part way into the munition. Finally, the projectile is returned to the indexed point of origin, where the pick-and-place mechanism removes the empty projectile and loads another onto the turntable. The drained agent is pumped to the agent storage tank located in the toxic cubicle and then sent to the liquid incinerator (Sect. C.2.3.1) for destruction.

Drained projectile bodies processed by the machines are sent by conveyor to the lift car (a munitions elevator) and lowered to the first floor. On the first floor, the drained projectile bodies are loaded onto buffer storage conveyors. From the buffer storage areas, the bodies move to another charge car, which loads the metal parts furnace (see Sect. C.2.3.2). All drained projectile bodies are thermally treated in the furnace, which burns any remaining agent and thermally decontaminates the drained projectile bodies and the trays. The fully demilitarized projectile bodies are placed in storage to await disposal or metal recovery. Trays are reused in the facility.

C.2.2.3 Land Mine Processing

Land mines arrive at the facility packed in drums. These drums are fed into a glove box located in the ECV. The mines, fuzes, and actuators are manually removed from the drums in the glove box and are sent to the ECR for further processing. The packing materials and drums are fed to the dunnage incinerator (see Sect. C.2.3.4). In the ECR, mines are punched and drained of agent and the booster explosive charge is punched out. The explosive charge and mine body are then fed by gravity to the deactivation furnace system (see Sect. C.2.3.3.)

C.2.2.4 Processing Bulk Items: Bombs, Ton Containers, Spray Tanks

Because bulk items such as bombs, ton containers, and spray tanks contain no explosive, they are conveyed from the unpack area directly to the bulk drain station. Spray tanks will be removed from their shipping containers in the unpack area and transferred to tray assemblies on the input conveyor. Unpalletized bulk items, such as ton containers, will be placed directly on tray conveyors. The trays will be conveyed to the bulk drain station, which is equipped with a large punch and an agent pump and removal tube and is operated remotely. The punch will produce a hole in the top of the bulk item, and the removal tube will be inserted through the hole to allow removal of the liquid agent. The agent will be transferred by pipeline to the agent storage tank in the toxic cubicle for destruction in the liquid incinerator (see Sect. 2.3.1).

The tray containing the drained bulk item will be transported to the munitions lift car, which descends to the first floor to discharge the tray to the buffer storage conveyor, and into the metal parts furnace. Residual agent will be burned in the metal parts furnace (Sect. C.2.3.2).

C.2.3 INCINERATION

The MDB typically has four incinerators: the liquid incinerator, the metal parts furnace, the deactivation furnace system, and the dunnage incinerator. Ton containers are processed principally in the liquid incinerator and metal parts furnace. The deactivation furnace system, liquid incinerator, and dunnage incinerator are used to process rockets.

The processing of projectiles requires the use of all four incinerators. Descriptions of the incineration processes are provided below.

C.2.3.1 Liquid Incinerator

The liquid incinerator system (shown in Figs. C.6 and C.7) is a two-stage, refractory-lined incinerator. It is designed to incinerate all the chemical agents (GB, VX, mustard, GA, and Lewisite) as well as spent decontamination solutions. The drained agent collected in the toxic tank is pumped at a uniform, continuous rate to the primary chamber with air atomizing nozzles and mixed with combustion air. Natural gas or propane is used to ensure a stable flame pattern within the primary chamber. Combustion is controlled at or above 2500°F by controlled addition of waste, fuel, and air. The combustion flue gases flow into the afterburner. Spent decontamination solution is injected into the afterburner for destruction. A minimum temperature of 2000°F is maintained in the afterburner by a gas burner. The overall gas residence time is a minimum of 2 s. Sufficient excess air is provided in both chambers to ensure complete combustion.

The flue gases from the afterburner are ducted to the pollution abatement system discussed in detail in Sect. C.2.4.1. The exhaust blower pulls exhaust gases through the entire system. A damper controls the flow to maintain a negative pressure in the furnace system. The clean gas is discharged to the atmosphere through the stack. The liquid incinerator, the metal parts furnace, and the deactivation furnace system share a common stack.

Salts generated from incinerating spent decontamination solution collect in the afterburner base. They are dissolved in water, and the resulting brine solution is mixed with spent brine from the scrubbing tower and pumped to the brine reduction area (Sect. C.2.4.3).

C.2.3.2 Metal Parts Furnace

The metal parts furnace is used to thermally decontaminate drained munition bodies and ton containers that do not contain explosive or propellant. It is also used to decontaminate scrap metal resulting from facility maintenance or closure. This furnace can also be used to incinerate charcoal filters, charcoal canisters, high-efficiency particulate air (HEPA) filters, and prefilters from the ventilation systems.

The metal parts furnace (Figs. C.8 and C.9) consists of two major subsystems: the furnace (which includes the inlet airlock, the burnout chamber, and exit airlock) and the afterburner. The metal parts furnace is a horizontal, three-chambers-in-series, roller-hearth unit. The first chamber is an airlock, sealed by vertical doors at both ends. This chamber has a capacity for one tray of drained munition bodies or bulk containers. The chamber holds a tray ready to be sent into the burnout chamber. With this system, the burnout chamber is never open to the room air in the MDB while in operation. The burnout chamber is at negative

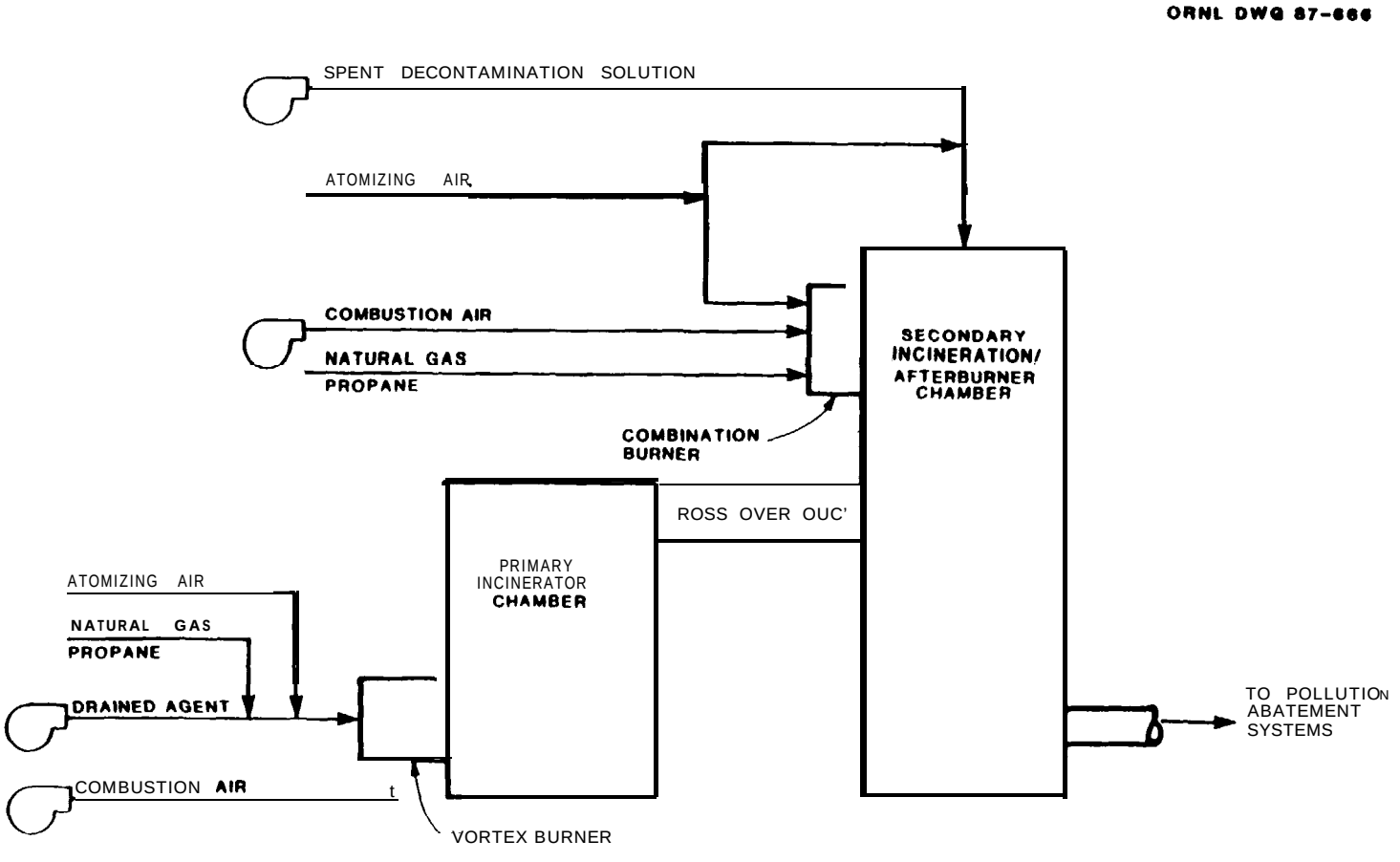


Fig. C.6. Schematic diagram of the liquid incinerator.

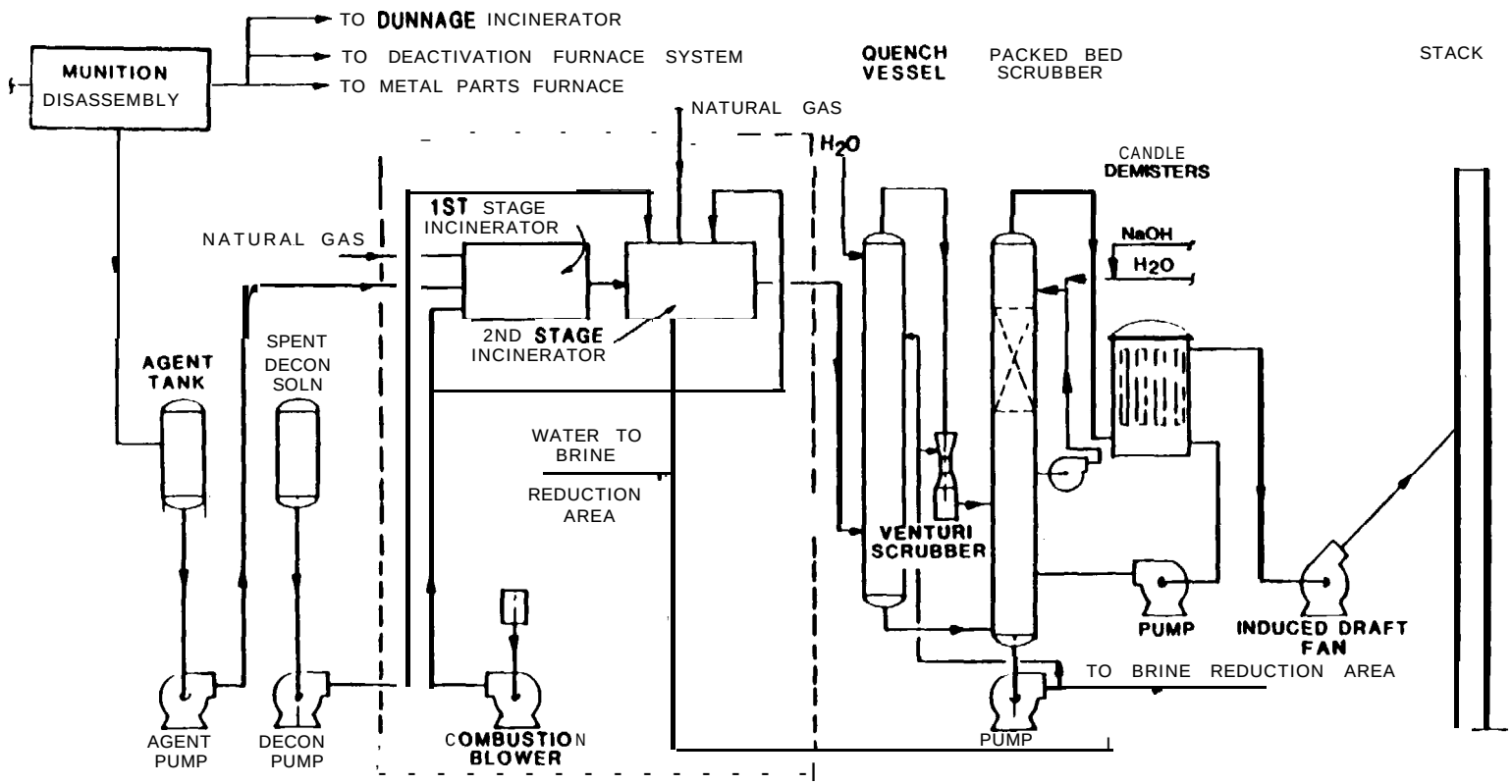


Fig. C.7. Liquid **incinerator process flow diagram.**

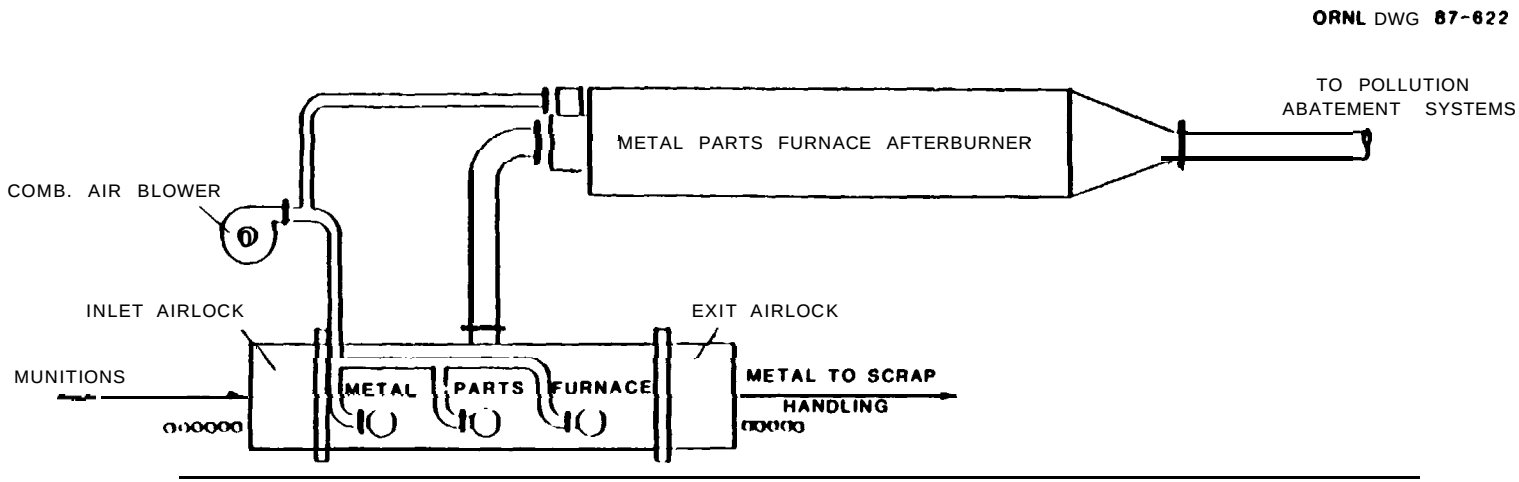


Fig. C.8. Schematic diagram of the metal parts furnace.

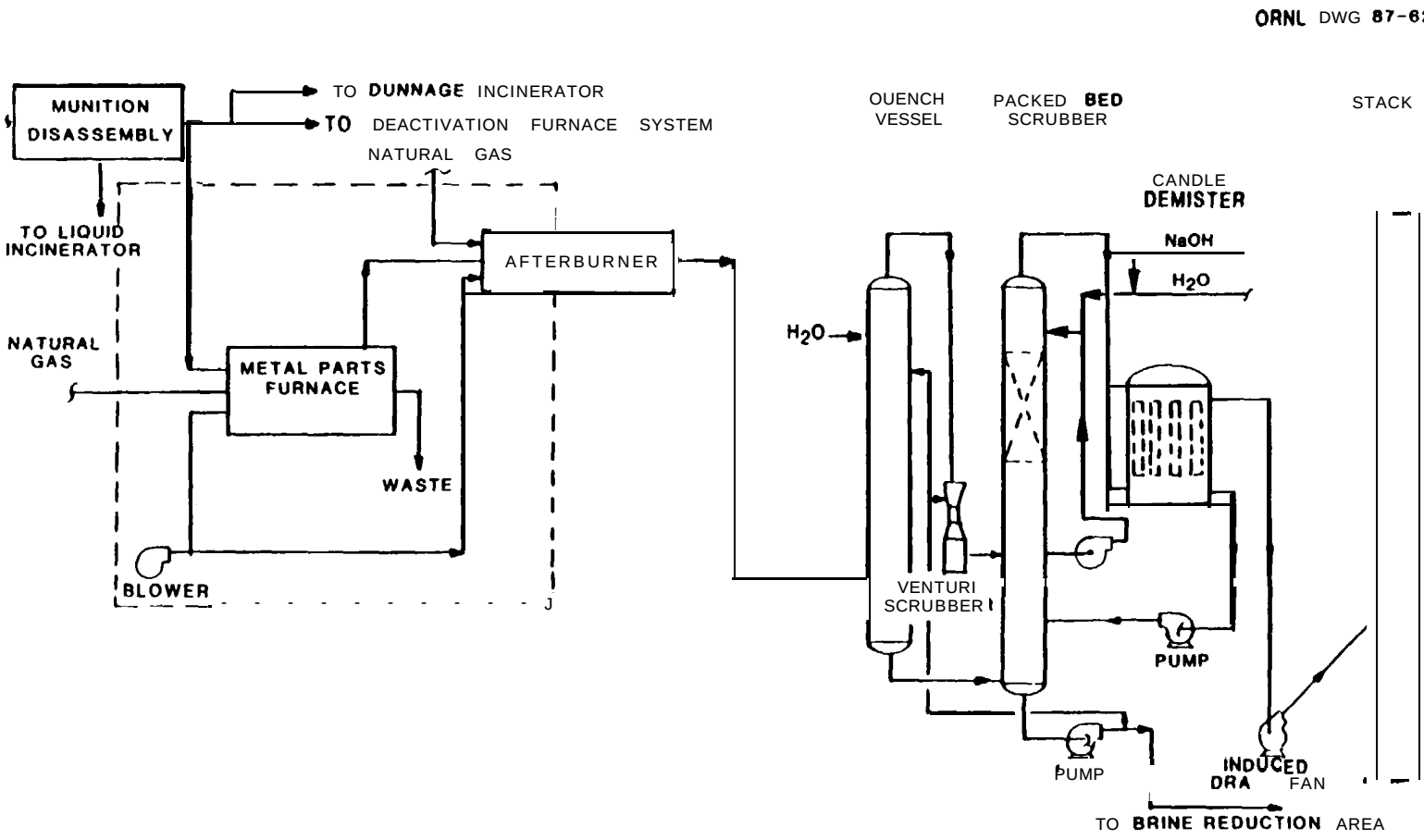


Fig. C.9. Metal parts furnace process flow diagram.

pressure with respect to the room air, providing containment of the flue gases in the furnace system. Any vapors generated in the first chamber (airlock) are vented to the afterburner.

The second chamber (burnout chamber) has a capacity for three trays of drained munition bodies or bulk containers. The metals and residual solids are heated in this chamber to a minimum temperature of 1000°F for a minimum time of 15 min [5X level of decontamination]. This method has been proven to destroy residual agent contamination on metal surfaces, rendering the metal suitable for sale as scrap metal. The burnout chamber is heated throughout its length by natural gas or propane burners. Excess air is maintained in the chamber to ensure combustion. Flue gases from this chamber are vented to the afterburner. Decontaminated munition bodies and other solid residuals are left in the trays and discharged from the burnout chamber to the third chamber, the exit airlock. After the door to the burnout chamber is closed, the exit air lock door is opened and the tray is sent to the cooling rack area. After the decontaminated munition bodies have sufficiently cooled, they are sent to the scrap metal bins.

The purpose of the afterburner is to ensure the complete incineration and detoxification of gases from the inlet airlock, exit airlock, and burnout chamber. The afterburner is a horizontal, refractory-lined, cylindrical unit that operates at a temperature of 2000°F, with a minimum retention time of 0.5 s. The afterburner is fired by natural gas. Gases leaving the afterburner enter the metal parts furnace pollution abatement system.

C.2.3.3 Deactivation Furnace System

The deactivation furnace system consists of four separate sections: (1) the rotary kiln, (2) a heated discharge conveyor, (3) the cyclone, and (4) the afterburner (Figs. C.10 and C.11). It is designed to process drained rockets and mines. In addition, it also processes fuzes, explosives, and propellants from other munitions. At any given time, the deactivation furnace system will process components from only a single munition type and a single agent (e.g., M55 rockets containing GB or VX are processed separately). This material is 5X decontaminated. The furnace is also used to destroy spent decontamination-solution and rinse water generated from the decontamination of the feed chutes.

The furnace system consists of two slide chutes, each with two blast gate valves, a charge-end subassembly, a rotary kiln with shroud, a kiln drive mechanism, a discharge-end subassembly, and a heated discharge conveyor. Feed to the kiln is accomplished via the two blast gate valves, which isolate the kiln from the ECR.

Pieces enter the deactivation furnace system through the blast gate valves and slide down a chute into the rotary kiln, then move through the kiln as thermal processing occurs. The kiln is fabricated from flanged fabricated-alloy steel sections and operates with a gas temperature of 1000 to 1800°F. The drive mechanism rotates the kiln at a

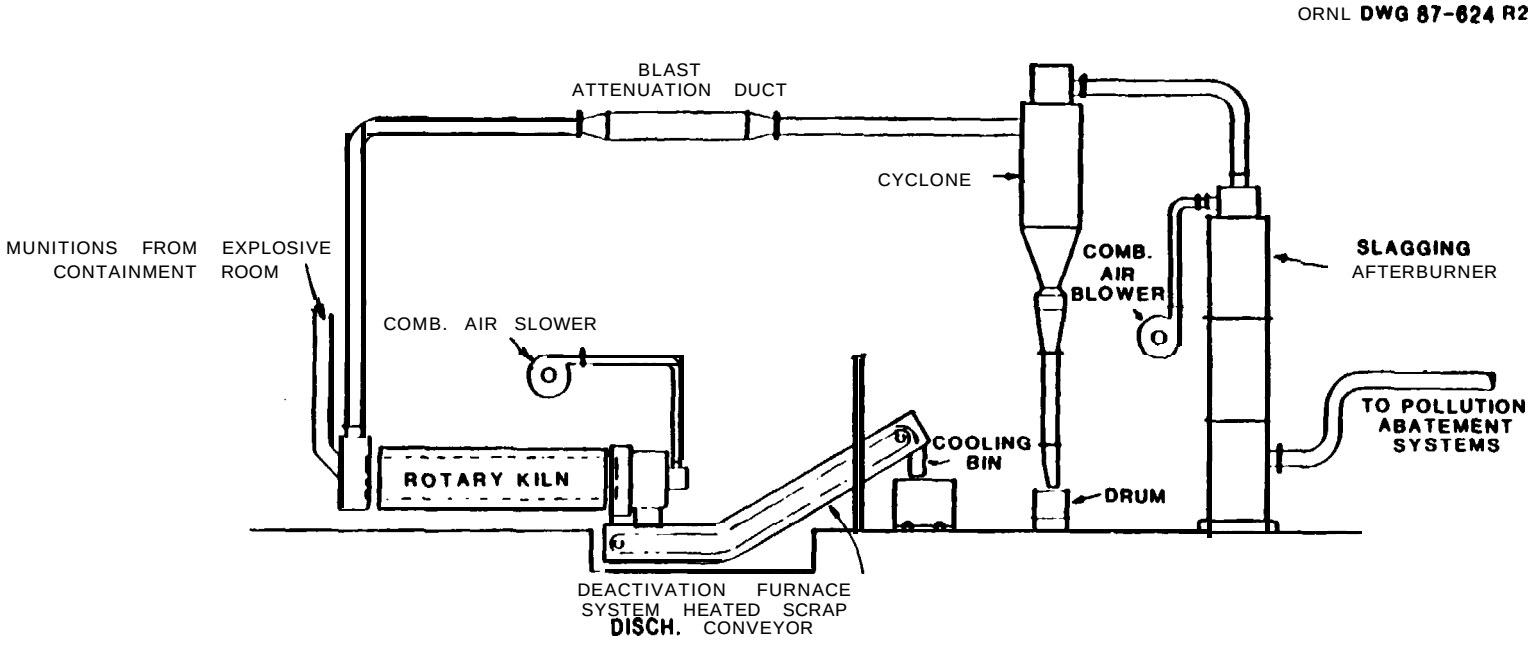


Fig. C.10. Schematic diagram of the deactivation furnace system.

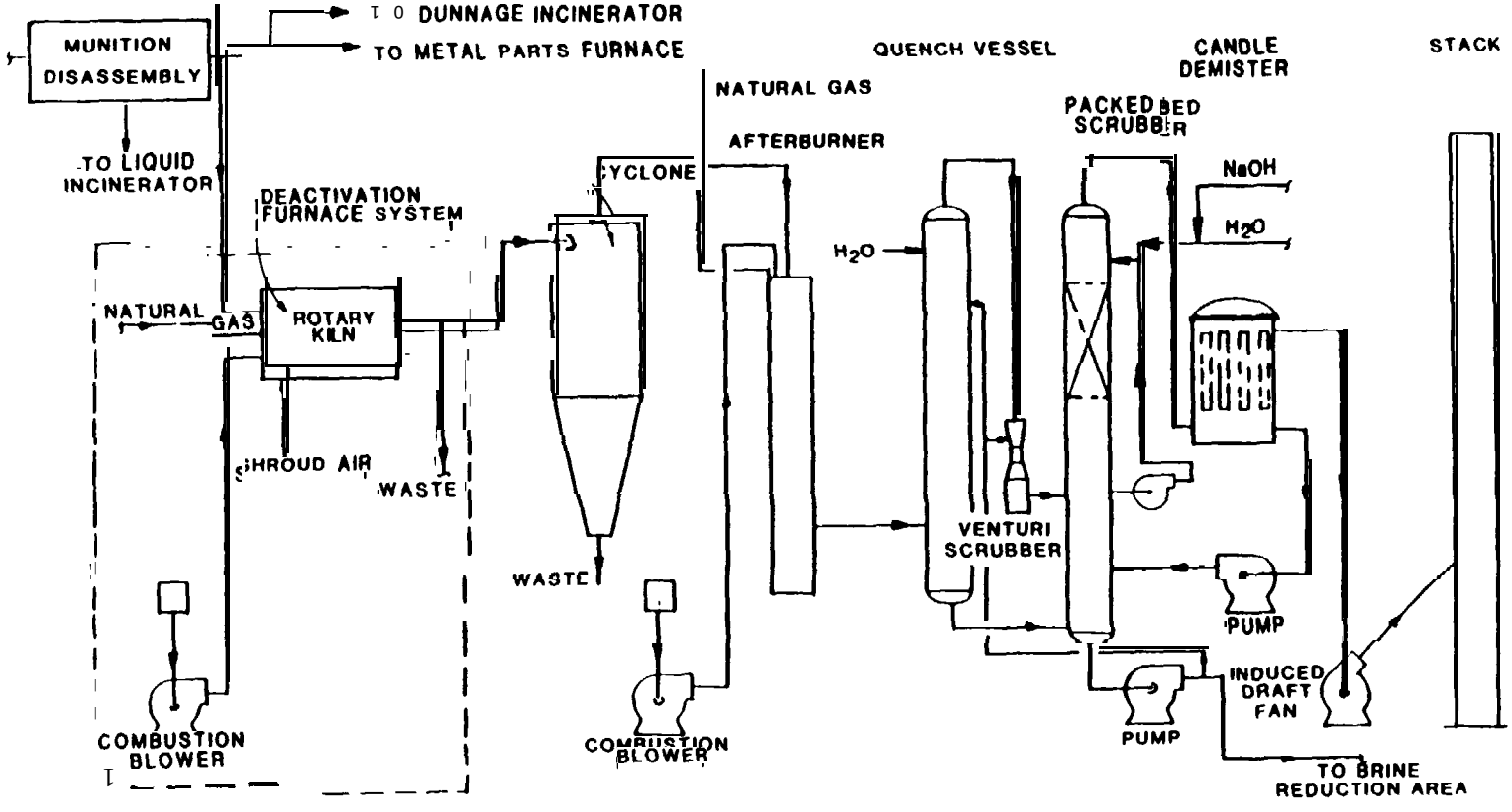


Fig. C.11. Deactivation furnace system process flow diagram.

preselected constant speed, which is adjustable in a narrow range for complete deactivation of explosive components. Metal pieces and ash exit the kiln at the discharge-end subassembly. At this point, they transfer to the heated discharge conveyor, which conveys the material while providing sufficient holding time at an elevated temperature of 1000°F to ensure decontamination (5X). The discharge conveyor is a steel enclosure containing a continuous herringbone-weave conveyor belt. The heated conveyor discharges the ash and scrap metal through a chute with a blast gate valve to a portable residue bin. When full, the bin is transferred to the residue handling area by forklift. The gates are interlocked so that both can never be open at the same time.

Flue gas from the kiln exits through a blast-attenuating duct and then enters the cyclone that separates large particulate matter from the gas stream. The cyclone is a cylindrical vessel with a cone-shaped bottom. Large particles removed from the flue gas drop into a container below the cyclone. These wastes will be characterized and disposed of in an appropriate manner.

Flue gas from the cyclone then passes into the slagging afterburner, a refractory-lined fume incinerator. The afterburner operates at 2200°F, providing a minimum of 2-s retention time. The afterburner ensures complete combustion of agent vapors, hydrocarbons, and organic vapors from incineration of fiberglass. (Fiberglass is present only during rocket processing.) The afterburner is the final thermal process step before the flue gas enters the deactivation furnace pollution abatement system.

C.2.3.4 Dunnage Incinerator

The dunnage incinerator system (shown in Figs. C.12 and C.13) is designed to incinerate a combination of wooden pallets, laboratory solid wastes, protective clothing, charcoal canisters (from masks), charcoal and HEPA filters from ventilation systems, rubber boots, metal packaging material, combustible dunnage, and any other miscellaneous wastes that may be contaminated with agent. It consists of a primary combustion chamber and an afterburner.

The primary combustion chamber is a horizontal refractory-lined chamber operating at approximately 1600°F. Feed enters the system through an airlock that leads to the primary combustion chamber. The gaseous products of combustion travel from the primary combustion chamber to the afterburner, a refractory-lined unit operating at approximately 2000°F with a retention time of 2 s. The afterburner incinerates all remaining combustible material from the primary combustion chamber. Both chambers are independently fired with natural gas or propane and air. Excess air is provided to incinerate combustibles within the afterburner. Gases leaving the afterburner enter the dunnage incinerator pollution abatement system (see Sect. C.2.4.2).

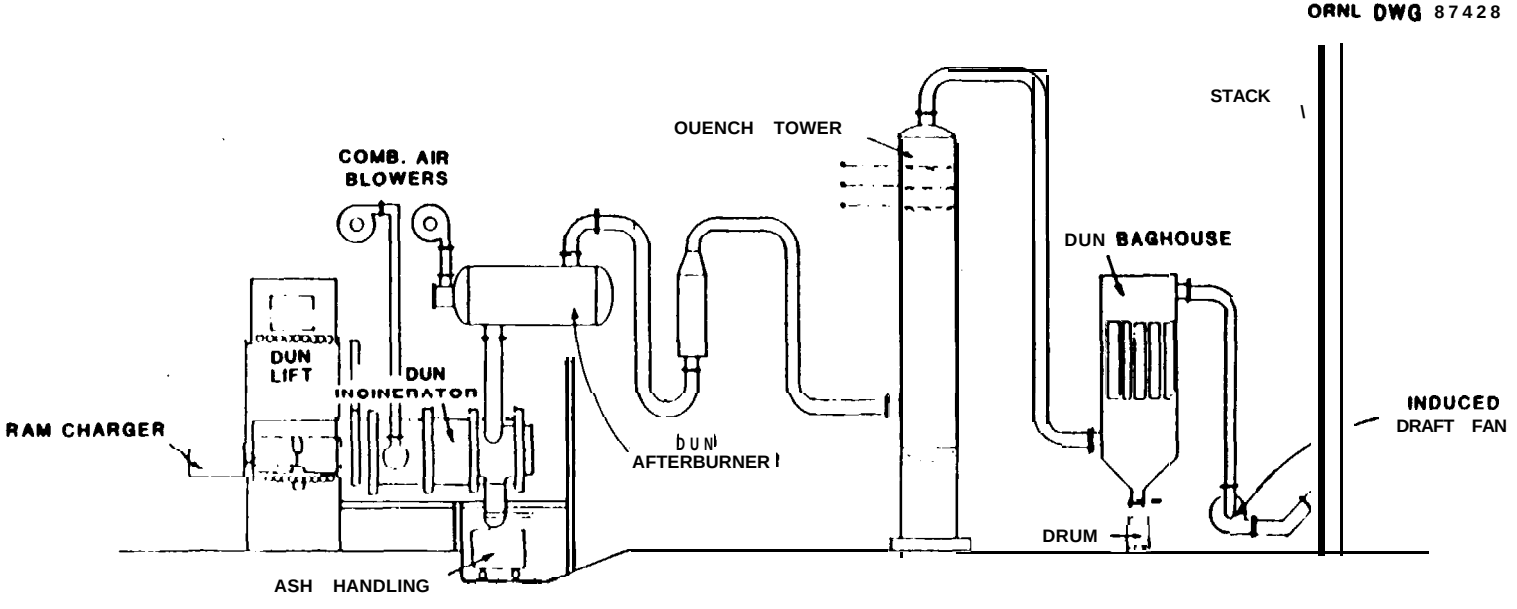


Fig. C.12. Schematic diagram for the dunnage incinerator and the pollution abatement system.

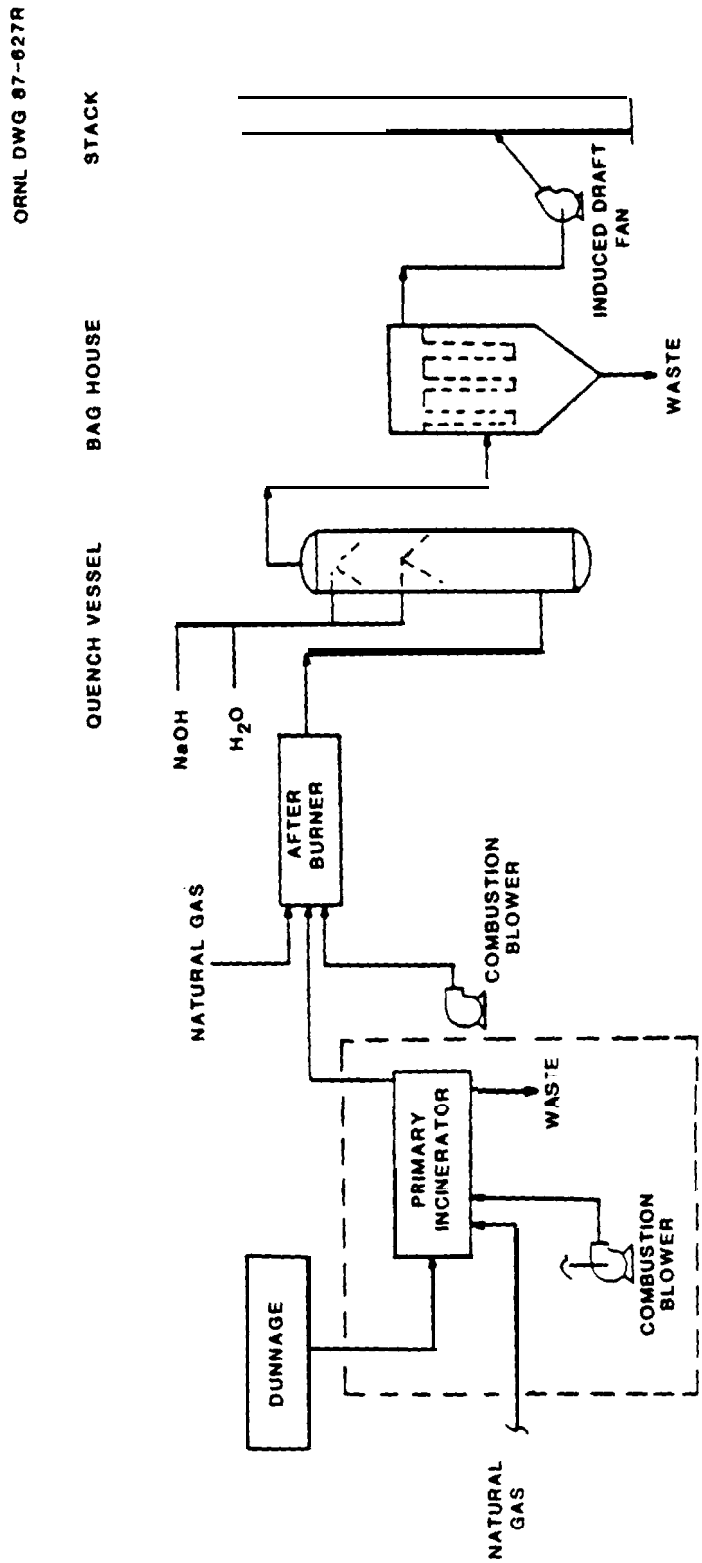


Fig. C.13. Dunnage incinerator process flow diagram.

C.2.4 POLLUTION ABATEMENT SYSTEMS FOR INCINERATORS

The pollution abatement systems control emission of acidic gases and particulates in the flue gas from the incinerators. There is an independent pollution. abatement system for each incinerator. The pollution abatement systems for the liquid incinerator, metal parts furnace, and deactivation furnace system each have a quench tower, a venturi scrubber, a packed bed scrubber tower, demister vessel, and associated pumps and blowers. Spent brine (liquid waste) from the scrubbers is treated in the brine reduction area (Sect. C.2.4.3). The system for the dunnage incinerator is different from the others and is described separately.

C.2.4.1 Liquid Incinerator, Hetal Parts Furnace, and Deactivation Furnace System Pollution Abatement Systems

The pollution abatement systems, as shown in Figs. C.7, C.9, and c.11, consist of a quench tower, a venturi scrubber, a packed bed scrubber tower, demister vessel, associated pumps and blowers, and a stack. The pollution abatement system configuration is identical (except for equipment size) for the liquid incinerator, the metal parts furnace, and the deactivation furnace. Therefore, only the pollution abatement system for the liquid incinerator is described. Although each system has the same basic equipment, the flows of flue gas and scrubber brine vary to match the varying duties of each incinerator. Also, the flow and pressure control mechanism is different for the induced draft fan for each incinerator. The treated effluents from the three incinerator pollution abatement systems are released to the atmosphere through a common stack.

The pollution abatement systems for these incinerators are designed to provide removal efficiencies which meet or exceed 99% for HCl and 99.8% for particulates (including metal oxides and P_2O_5).

The flue gas enters the quench tower, where it contacts a brine spray pumped by the quench brine pump from the scrubber tower sump. Water from the brine is evaporated into the gas, cooling the gas to its dew point. Brine is sprayed into the tower at a rate in excess of the maximum expected evaporation load. Water required to maintain the level in the scrubber tower sump is added to the quench.

The exhaust gas from the quench tower effluent enters the venturi scrubber, where it contacts an alkali brine. The venturi scrubber has a variable throat controlled to maintain a pressure drop of 40-in. water column across the venturi scrubber. Brine flow is controlled to ensure efficient collection of particulates. Acidic gases react with alkali in the brine to form salts. Caustic (NaOH) is added to the brine as required to maintain a pH of approximately 8 in the scrubber sump.

The two-phase effluent (gas and liquid) from the venturi scrubber enters the scrubber tower below the reservoir tray. The liquid falls to the sump while the gas rises through the chimneys of the reservoir tray. The rising gas contacts alkali scrubbing solution in the packed bed. Acidic gases are absorbed by the solution and react with the alkali to

form salts. The scrubbing solution falling from the packed bed collects on the reservoir tray from where it is pumped back to the top of the packed bed.

Recirculation of solution is controlled to ensure adequate contact of liquid and gas. Caustic is added to the solution to maintain a pH above 8, and water is added to adjust the density and level of the solution. Excess brine overflows the chimneys and falls into the scrubber tower sump. Gas rising from the packed bed passes through a mist eliminator. The mist eliminator coalesces liquid droplets and drains to the scrubber tower sump.

One of the combustion products from the incineration of the nerve agents is P_2O_5. The P_2O_5 in the off-gas reacts with moisture to form a fine mist of H_3PO_4, which is removed by the demister. Liquid accumulates in the vessel bottom and is pumped to the scrubber tower sump.

The exhaust blower pulls exhaust gases through the entire system. A damper controls the flow to maintain a negative pressure in the furnace system. The cleaned gas is discharged to the atmosphere from the stack.

Acid gases react with the caustic in the brine to form salts. Fresh caustic is continuously added to maintain a basic (alkaline) solution. Spent brine is continuously removed from the system. The spent brine is pumped to one of two large brine holding tanks (see Sect. C.2.4.3). The systems serving the liquid incinerator, deactivation furnace system, and metal parts furnace share a pair of tanks. When one tank is filled, flow of the waste stream is automatically routed to the other tank and a "full" signal on the filled tank is activated.

C.2.4.2 Pollution Abatement System for the Dunnage Incinerator

The pollution abatement system for the dunnage incinerator consists of a quench tower, baghouse, blower, and a stack, as shown in Figs. C.12 and C.13.

The afterburner flue gas from the incinerator is quenched with water or NaOH solution in a quench tower from an inlet temperature of about 2000°F to approximately 350°F. Cooling is required to protect the fabric filters in the baghouse from excessive heat. The gas flows from the quench tower to the baghouse where submicron particulates are removed. The particulate ash is collected in drums below the baghouse. Gases pass from the baghouse through an induced draft fan that provides the driving force for the combustion gas through the entire system, and then up an exhaust stack and into the atmosphere. The dunnage incinerator has its own stack.

Wastes are processed batch wise by type (e.g., wooden pallets, agent-contaminated filters, etc.). Caustic is used in the quench tower when incinerating wastes which generate acidic gases (e.g., HCl from certain plastics; SO2 from mustard-contaminated filters).

The combined quench and baghouse systems are designed to remove 95% of the chlorides and 99% of the particulates. The 95% HCl removal efficiency will ensure compliance with the 4-lb/h HCl emission

limitation.

C.2.4.3 Brine Reduction Area (Brine Drying)

The spent brines (liquid wastes) generated by the pollution abatement systems for the metal parts furnace, the liquid incinerator, and the deactivation furnace system and the waste brines from the secondary chamber of the liquid incinerator are dried to a salt in the brine reduction area (Fig. C.14). This system consists of an evaporator and two steam-heated rotary double-drum dryers. The brines from the various incinerator pollution abatement systems are collected in one of the two brine feed tanks, while the other tank is used to speed the drying operations. The waste is analyzed to verify that it is "agent free" before being dried. Flow into the drum dryers is regulated by level controllers. Salt generated in the final drying of the brine is packed in containers. The waste will be characterized and disposed of in an appropriate manner.

C.2.5 BUILDING VENTILATION

Areas of the MDB are categorized into air-ventilation zones. The categories are based on the anticipated type and degree of agent contamination as defined in Table C.l. The zones are established to confine agent to controlled areas within the facility. Ventilation zones are delineated by physical barriers to confine hazardous materials and by zone pressure differentials to direct airflow from zones of lower potential for contamination to zones of higher potential. The hazard categories for different areas of the MDB are shown on Figs. C.15 and C.16. All Category A, B, and C areas are provided with an appropriate ventilation system to (1) collect, treat, and monitor ventilation air from the work area that may contain lethal chemical agent vapors before being exhausted to the ambient air; (2) provide mixing of air that is essential for monitoring work areas with agent detection devices; and (3) provide a negative pressure within the work areas to contain agent vapors.

Air is collected from the more contaminated areas and passed through a filter system before being exhausted to the atmosphere. The filter system uses a series of filter units, with each unit containing a filter train and a motor/blower. The filter train consists of prefilters; HEPA filters; activated charcoal filters; a second bank of charcoal filters; and, finally, a second bank of HEPA filters. Each filter bank is provided with gauges to indicate pressure drop across the filters. Sampling ports are provided between the banks of charcoal filters and in the exhaust stack. Category E areas (e.g., the control room) are positive pressure with charcoal-filtered supply air. Category D areas provide standard industrial ventilation, but neither supply nor exhaust air is filtered.

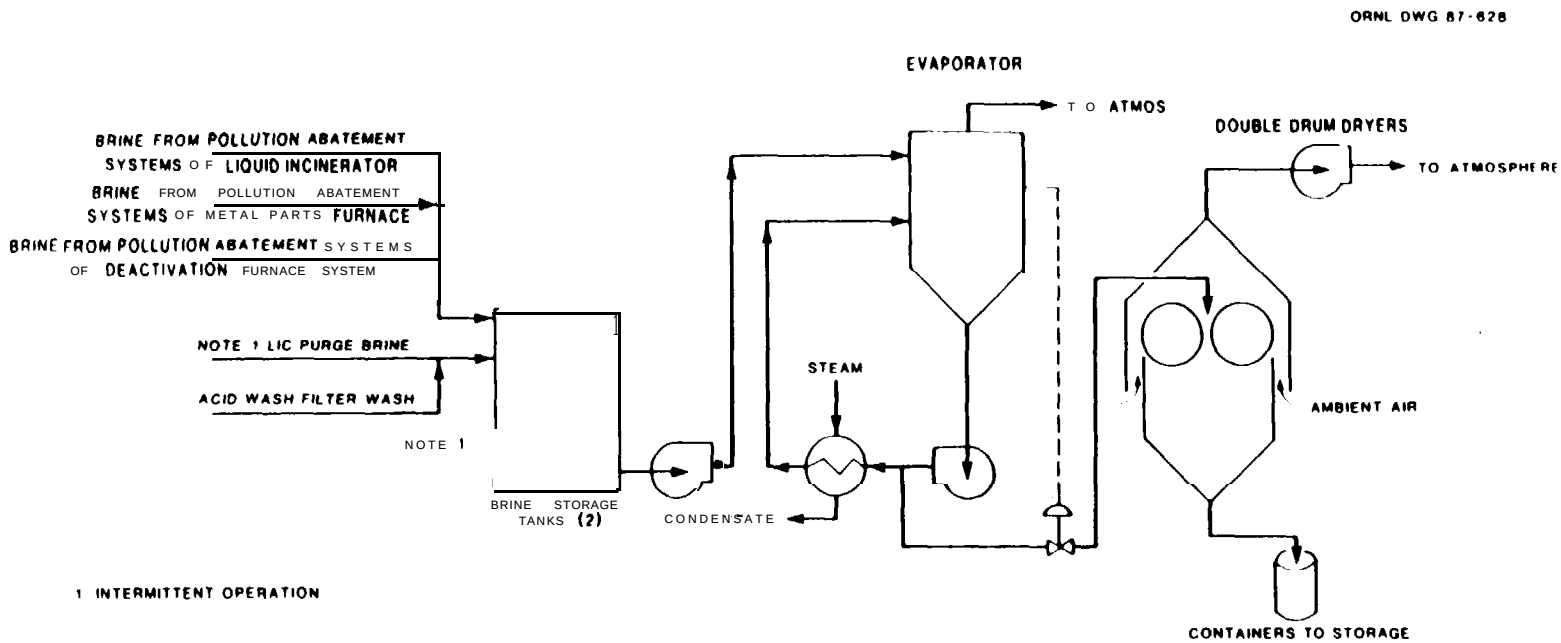


Fig. C.14. Brine reduction area flow diagram.

Table C.l. CSDP plant ventilation/agent categories

Ventilation category	Agent contamination	Treatment
A	Probable liquid and vapor	Used as combustion air (incinerated) or HEPA/charcoal filtered
B	Possible vapor	Cascaded to higher category or HEPA/ charcoal filtered
C	Not expected, but possible	Cascaded to higher category or HEPA/ charcoal filtered
D	No agent	None
E	No agent	HEPA/charcoal filter on inlet

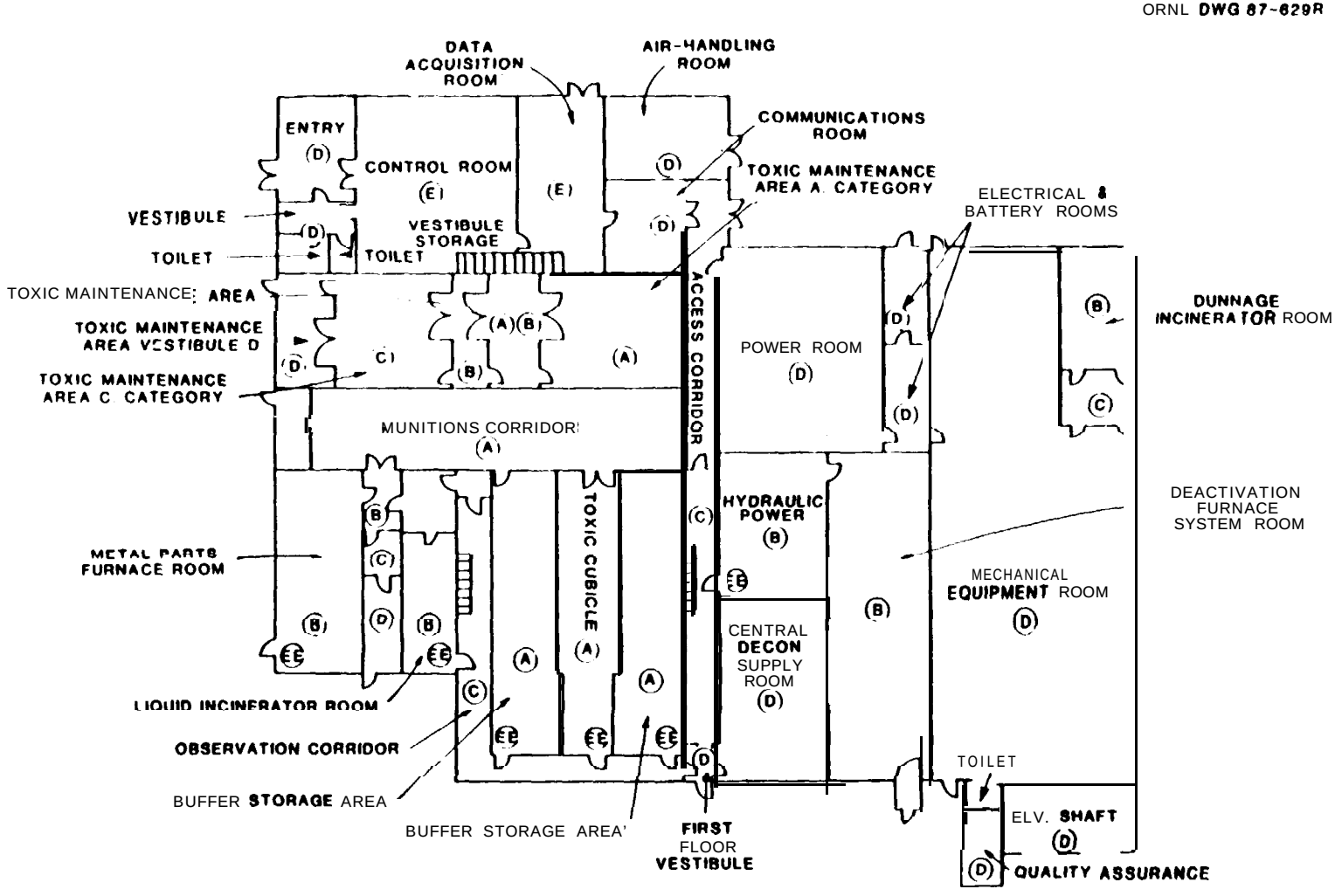


Fig. C.15. First floor munitions demilitarization building hazard categories. (Codes defined in Table C.1.)

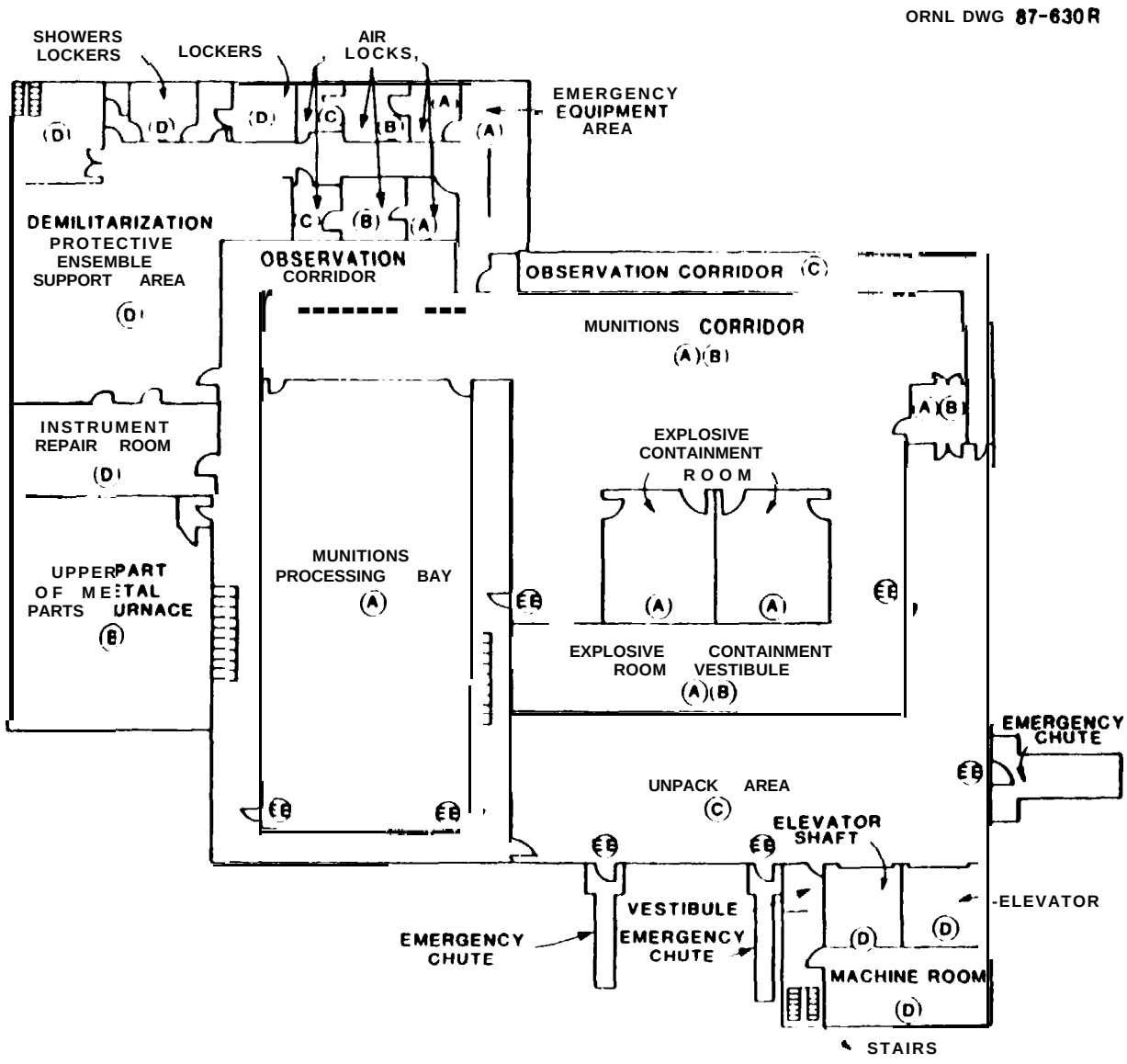


Fig. C.16. Second floor munitions demilitarization building hazard categories. (Codes defined in Table C.1.)

Ventilation flow requirements vary with each process area. The filter units are a common type for all areas. The varying capacity requirements are met by using multiple filter units in parallel for areas requiring ventilation at rates higher than one unit can provide. The basic filter unit is a skid-mounted design with welded housing, access doors, interior lighting, and observation and sample ports. This basic unit is designed to handle a nominal 15,000 scfm at a 5-in. water column pressure drop across each element. The filter elements are changed when the pressure drops across any element exceeds 6-in. water column. Redundant charcoal filter trains are provided. The filter locations and duties for a typical CSDP plant are summarized in Table c.2.

C.2.6 PROCESS SUPPORT AND UTILITIES

C.2.6.1 Cooling Tover Circulation System

The closed-loop cooling tower circulation system provides the primary cooling water for the secondary (i.e., process) cooling water system, for compressor cooling, and for building cooling systems. It consists of a forced air circulation cooling tower, water circulation pumps, piping, and control system. Cooling tower blowdown wastewater will either be used for makeup water for the pollution abatement systems or be pumped to the brine reduction system for processing with pollution abatement system brines.

C.2.6.2 Laboratory

The laboratory facility is provided on-site to conduct routine analyses of samples from the processes and monitoring stations at the facility. Equipment will include extraction/concentration apparatus for sample preparation, gas chromatography, high-performance liquid chromatography, and atomic absorption spectrometry equipment to carry out the waste analysis plan. The laboratory may be used to conduct routine chemical analyses to determine compliance with the Resource Conservation and Recovery Act (RCRA), the Clean Air Act, and the Toxic Substances Control Act.

The analyses include (1) determination of agent concentration in samples collected using bubblers or the depot area agent monitoring system (DAAMS) and (2) maintenance and testing of the automatic continuous agent monitoring system (ACAMS) monitors located in instrument stations within the site complex. Because the site will be processing only one agent at any given time, the analytical procedures will be tuned to detect one specific agent at any time.

Table C.2. Filter locations rod duties for typical CSDP plant

Location	Design capacity (scfm)	No. of units installed	
		Operating	Spare
MDB[a] control room	12,000	1	1
MDB process area	75,000	5	2
Laboratory	8,000	1	1

[a] MDB = munitions demilitarization building.

Table D.1. Summary of Army's experience in industrial-scale chemical agent/munitions diil

Operation	Description	Date	Agent	Site[a]	Process[b]	Quantity[c]
		Agent neutralization experience				
Project Eagle (Phs 11)	M34 cluster bombs	Oct. '73–Nov. '76	GB	R	N/I	4. I 29.6
Expanded Project Eagle (Phs II)	Underground storage tanks	Sept. '74–Nov. '74	GB	R	N	378.0
Expanded Project Eagle (Phs II)	Ton containers	May '75 -Nov. '75	GB	R	N/I	3,604.5
Expanded Project Eagle (Phs II)	Honest John warhead (M139)	April '76–Nov. '76	GB	R	N/I	76.5
M55 rocket disposal		Sept. '79–April '81	GB	C	N/I	128.0
155-mm projectile disposal		July '81 - July '82	GB	C	N	*d*
105-mm projectile disposal		March '82–July '82	GB	C	N	60.5[d]
Total neutralization						8,377.1
		Agent incineration experience				
Project Eagle (Phs I)	Ton containers	July '72–March '74	H	R	I	4,428.0
Project Eagle (Phs I)	Ton containers	July '72–March '74	HD	R	I	1,714.0
CAIS disposal	Chemical agent identification sets	May '8 1 -Dec. '82	*e*	R	I	36.7
Agent injection incineration tests		March '81–Jan. '84	GB	C	I	II.2
Agent injection incineration tests	Ton containers	April '84–July '84	v x	C	J	7.9
In situ agent incineration		Oct. '82 - Nov. '83	GB	C	I	17.6
M55 rocket incineration		Feb. '85–July '86	GB	C	I	2.3
Liquid incineration test		Aug. '85–Aug. '86	GB	C	I	37.9
Total incineration						6.2556
Grand total disposal						14.632.7

[a]R = Rocky Mountain Arsenal; C = Chemical Agent Munitions Disposal System.

[b]N = agent neutralization only; I = incineration of agent and explosive (and/or metal parts thermal decontamination); N/I = agent neutralization and explosive incineration (and/or metal parts thermal decontamination).

'Quantity is expressed in thousands of pounds (quantity x IO'). Original source in English units. For metric conversion I lb = .454 kg.

[d]Total of 155-mm and 105-mm projectile neutralization in July 1981-July 1982 and March 1982 to July 1982 periods, respectively.

'Agents include: phosgenc. chloropicrin, mustard, Lewisite, cyanogen chloride. nitrogen mustard, and GB.

Source: U.S. Army 1987a. *Chemical Agent and Munition Disposal: Summary of the* U.S. *Army's Experience,* Report SAPEO-CDE-IS-87005, Office of the Program Manager for Chemical Munitions (Demilitarization and Binary), Aberdeen Proving Ground, Md.

C.2.6.3 Electrical Power System

The electrical power supply and distribution network for the process system are provided by the local utility and by the installation.

C.2.6.3.1 **Emergency pover** system

Because a power loss could either create a safety hazard or cause major damage to equipment, a diesel-driven emergency electrical generation system is provided to supply essential power to the MDB and associated facilities for a safe and orderly shutdown. This includes power for emergency lighting, the instrumentation and control system, the building ventilation system, and key process equipment such as **pumps** and blowers. Plant security lighting is supplied from a separate power source. There are redundant emergency power systems.

Specific computer programs will be prepared to react automatically to electrical power supply malfunctions according to the emergency power contingency plans adopted for each site.

C.2.6.3.2 Uninterruptible pover supply

The uninterruptible power supply will provide power to electrical loads that cannot be interrupted for any length of time, such as instrument control cabinets, computer, closed-circuit television, demilitarization protective ensemble radio system, flashing warning lights, fire alarms, exit and emergency lights, and agent monitoring systems. The uninterruptible power supply system for the MDB utilizes offset battery racks in separate rooms to facilitate access for maintenance and to accommodate cable connections to each battery. Positive room air circulation is utilized to avoid hazardous concentrations of hydrogen gas.

C.2.7 PROCESS CONTROL

Instrumentation is provided to (1) monitor process conditions, (2) provide data for ensuring compliance with regulatory requirements, (3) ensure appropriate process response and control, and (4) ensure operational flexibility, safety interlocking, and shutdown features. A centralized process control system is used with a centralized control console, including closed-circuit television monitors for observing operations at various locations and locally mounted programmable logic controllers. Processing and sequencing operations are controlled automatically through the programmable logic controllers. Interlocks are monitored and continuous checking is undertaken to determine any lack of completion of a programmed step. All abnormal conditions, operator entries into the system, and starting and stopping of equipment are logged with the time of occurrence by the data acquisition and recording system. The control system provides continuous automatic control of the incineration process. System interaction by the operator is limited to initiation of process systems or reaction to abnormal conditions. In

ACRONYMS AND INITIALISMS FOR APPENDIX C

ACAMS	automatic continuous agent monitoring system
CSDP	Chemical Stockpile Disposal Program
DAAMS	depot area agent monitoring system
ECR	explosives containment room
ECV	explosives containment room vestibule
HEPA	high-efficiency particulate air (filter)
JACADS	Johnston Atoll Chemical Agent Disposal System
MDB	munitions demilitarization building
MHI	munitions holding igloo
RCRA	Resource Conservation and Recovery Act
SPORT	single pallet only rocket transporter

Appendix D

BACKGROUND OF THE CHEMICAL STOCKPILE DISPOSAL PROGRAM

D.l INTRODUCTION

The purpose of this appendix is to describe the Army's experience in the disposal of chemical agent/munitions and to explain how this experience led to the choice of incineration for the disposal of the chemical stockpile. This appendix summarizes the information contained in U.S. Army (1987a).

This appendix discusses (1) the historical background of various disposal processes, (2) the scientific reviews that led to the use of chemical neutralization, (3) the problems encountered with neutralization that led to adoption of incineration, (4) the experience with incineration, (5) the incineration and pollution abatement systems to be used in the proposed chemical stockpile disposal program, and (6) the test program being conducted by the Army to verify performance and environmental compliance.

D.2 HISTORICAL BACKGROUND

Between World War I and 1969, chemical agent/munitions were disposed of by open pit burning, atmospheric dilution, burial, and ocean dumping. Such methods of chemical disposal were also commonly practiced by industry in this period before environmental, health, and safety issues became critically important to the public. In response to public concerns arising in May 1969, the National Academy of Sciences (NAS) performed a scientific review of chemical agent/munitions disposal methods. In June 1969, the NAS recommended abandoning ocean dumping as a method of disposal. Two alternatives to ocean dumping were suggested based on the Army's experience at that time: chemical neutralization of the nerve agent GB and incineration of the blister agents H and HD.

In 1972, a Senior Advisory Panel report confirmed the original NAS-recommended dual-method approach to disposal and added that the Army should continue to test incineration for disposal of GB and VX. Incineration of hazardous wastes was a relatively new technology at that time. However, by 1970, the Army had limited experience in incinerating mustard at Rocky Mountain Arsenal and limited field experience in neutralizing GB. The Army's industrial-scale experience with these two disposal methods is summarized in Table D.l and is discussed in greater detail in Sects. D.3 and D.4.

D.3 CHEMICAL NEUTRALIZATION TECHNOLOGY

D.3.1 BACKGROUND

In response to public concerns about the environmental implications of ocean dumping of chemical agent/munitions, the Army considered chemical neutralization as a method for industrial-scale disposal of chemical agent GB. Neutralization was attempted before incineration because of the Army's familiarity with neutralization in field disposal and decontamination operations. The term "neutralization" is used in the generic sense to mean a chemical reaction that counteracts the toxic effect of the chemical agent, yielding an innocuous product. In some cases, the reaction is literally a true chemical neutralization in which the chemical agent, which acts as an acid, undergoes reaction with a base to form a salt.

Table D.I. Summary of Army's experience in industrial-scale chemical agent/munitions disposal

Operation	Description	Date	Agent	Site[a]	Process[b]	Quantity[c]
		Agent neutralization experience				
Project Eagle (Phs II)	M34 cluster bombs	Oct. '73–Nov. 76	GB	R	N/I	4,129.6
Expanded Project Eagle (Phs II)	Underground storage tanks	Sept. 74 -Nov. 74	GB	R	N	378.0
Expanded Project Eagle (Phs II)	Ton containers	May '75–Nov. '75	GB	R	N/I	3,604.5
Expanded Project Eagle (Phs II)	Honest John warhead (M 139)	April '76–Nov. 76	GB	R	N/I	76.5
M55 rocket disposal		Sept. '79–April 81	GB	C	N/I	128.0
155-mm projectile disposal		July 8 1--July 82	GB	C	N	d
105-mm projectile disposal		March '82–July 82	GB	C	N	60.5[d]
Total neutralization						8,377.1
		Agent incineration experience				
Project Eagle (Phs I)	Ton containers	July '72–March 74	H	R	I	4,428.0
Project Eagle (Phs I)	Ton containers	July '72–March 74	HD	R	I	1,714.0
CAIS disposal	Chemical agent identification sets	May 8 1-Dec. 82	e	R	I	36.7
Agent injection incineration tests		March '81–Jan. 84	GB	C	I	11.2
Agent injection incineration tests	Ton containers	April '84–July 84	v x	C	I	7.9
In situ agent incineration		Oct. '82–Nov. 83	GB	C	I	17.6
M55 rocket incineration		Feb. '85–July 86	GB	C	I	2.3
Liquid incineration test		Aug. '85–Aug. 86	GB	C	I	37.9
Total incineration						6,255.6
Grand total disposal						14,632.7

[a]R = Rocky Mountain Arsenal; C = Chemical Agent Munitions Disposal System.

[b]N = agent neutralization only; I = incineration of agent and explosive (and/or metal parts thermal decontamination); N/I = agent neutralization and explosive incineration (and/or metal parts thermal decontamination).

[c]Quantity is expressed in thousands of pounds (quantity x 10^3). Original source in English units. For metric conversion 1 lb = .454 kg.

[d]Total of 155-mm and 105-mm projectile neutralization in July 1981–July 1982 and March 1982 to July 1982 periods, respectively.

[e]Agents include: phosgene, chloropicrin, mustard, Lewisite, cyanogen chloride, nitrogen mustard, and GB.

Source: U.S. Army 1987a. ***Chemical Agent and Munition Disposal: Summary of the U.S. Army's Experience,*** Report SAPEO-CDE-IS-87005, Office of the Program Manager for Chemical Munitions (Demilitarization and Binary), Aberdeen Proving Ground, Md.

Mustard agent can be neutralized by hydrolysis or by reacting with an excess of monoethanolamine. However, the homogeneous liquid organic waste by-product creates disposal problems. Because this organic waste can be disposed of by incineration, it was thought best to incinerate mustard directly, thus eliminating the need for neutralization. Therefore, no industrial-scale neutralization was carried out for mustard agents.

Chemical agent VX can be neutralized by acid chlorinolysis. However, neutralization of VX was never demonstrated on the pilot-plant scale.

The only chemical agent using which the Army has had industrial-scale neutralization experience is GB. GB was neutralized using caustic sodium hydroxide in an exothermic process (i.e., a process in which heat is generated by the chemical reactions). This neutralization reaction is sensitive to concentration, pH, and temperature. It is also reversible, albeit marginally, and reformation of GB is possible under suitable conditions. Approximately 3.7 million kg (8.4 million lb) of GB have been chemically neutralized on an industrial scale by the Rocky Mountain Arsenal (RMA) in Denver, Colorado, and by the Chemical Agent Munitions Disposal System (CAMDS) at Tooele, Utah.

D.3.2 ROCKY MOUNTAIN ARSENAL

The neutralization operations at RMA occurred under two different projects: PROJECT EAGLE--Phase II, and EXPANDED PROJECT EAGLE--Phase II. (PROJECT EAGLE--Phase I consisted of the incineration of ton containers of mustard between July 1972 and March 1974 and is discussed in Sect. D.4.2.) PROJECT EAGLE--Phase II consisted of the demilitarization of 21,114 M34 cluster bombs between October 1973 and November 1976. EXPANDED PROJECT EAGLE--Phase II consisted of three suboperations: demilitarization of (1) five underground storage tanks; (2) 2422 ton containers; and (3) 106 Honest John Ml90 warheads (each containing 358 Ml39 bomblets), 1222 fused Ml39 bomblets, and 39,632 unfused Ml39 bomblets. EXPANDED PROJECT EAGLE--Phase II took place between September 1974 and November 1976. The purpose of PROJECT EAGLE--Phase II and EXPANDED PROJECT EAGLE--Phase II was to dispose of the excess stocks of chemical munitions stored at RMA since the early 1940s.

The process of demilitarizing the M34 cluster bombs in PROJECT EAGLE-'-Phase II consisted of declustering of the M125 bomblets (76 per bomb), deactivating the fuze in each bomblet, punching the bomblet, draining the liquid GB, weighing the drained bomblet, chemically decontaminating the empty bomblet, shearing the burster wells, incinerating the explosive, thermally decontaminating metal parts, and storing the GB for neutralization.

No processing was required for disposal of the GB held in underground storage tanks other than that of in-line filtering to remove solids. The GB was directly pumped from these tanks into neutralization reactors.

The ton containers were demilitarized by draining the liquid GB by vacuum transfer, by thermally decontaminating the containers, and by storing the GB for neutralization.

The Honest John Ml90 warheads and Ml39 bomblets were demilitarized by stripping the explosive (if present), removing the bomblets (if not already removed), punching the bomblets, draining the liquid GB,

incinerating the explosive assemblies, thermally decontaminating metal parts, and storing the GB for neutralization.

During all demilitarization operations and suboperations at RMA, the same process was used for neutralizing GB. The GB to be neutralized was pumped to a holding tank. From there it was pumped to a venturi-like mixing tee, where it was mixed'with a caustic solution consisting of 18% sodium hydroxide. The mixture of GB and caustic solution flowed from the mixing tee into a reactor, which had been previously filled with some caustic solution. The brine resulting from the reaction mixture was continuously agitated and recirculated. Heat generated from the neutralization process was removed by a heat exchanger. When the neutralization was determined to be complete by sampling and testing, the brine was transferred to a spray dryer to evaporate the water and reduce the brine to salt. The water vapor was scrubbed before being discharged to the atmosphere, and the salt was packed in drums for disposal. Wastewater from the scrubbing process and from periodic washdown of the reactors was transferred to an industrial sump or lagoon.

The neutralization process at RMA was successful, for the most part, in its mission but not without presenting some environmental problems. These environmental problems must be considered in a historical context. For instance, although the wastewater contributed to contamination of the underground water, it was then common industrial practice to dispose of wastewater by allowing it to evaporate from a lagoon.

The solid waste included decontaminated metal parts, furnace ash, and the spray-dried salt. The decontaminated metal parts were sold as scrap. Furnace ash was not disposed of until 1986, subject to the Resource Conservation and Recovery Act (RCRA) that, because of the presence of heavy metals, required placement of the ash in a hazardous-waste landfill. The 10 million kg (21.5 million lb) of spray-dried salt generated by the process became subject to RCRA. The salt was placed into an approved hazardous-waste landfill in 1986. Of greatest concern was the reformation of minuscule amounts of GB in the spray-dryer gas stream. Solution of the problem required the performing of tedious studies that resulted in a method that combines adjusting the pH and brine flow rate, reducing operating temperature, and/or changing the fuel.

D.3.3 CHEMICAL AGENT MUNITIONS DISPOSAL SYSTEM

Several demilitarization operations involving neutralization of GB occurred at CAMDS, which is located at Tooele Army Depot (TEAD), near Salt Lake City, Utah. Two major operations are discussed in this appendix-- the M55 rocket disposal and 155-mm/105-mm projectile disposal.

Between September 1979 and April 1981, 13,951 M55 rockets were demilitarized at CAMDS. The process consisted of draining the liquid GB, cutting the rocket into pieces, incinerating the explosive/propellant, thermally decontaminating the metal parts, and storing the GB for neutralization.

Between July 1981 and July 1982, 12,673 nonburstered 155-mm and 105-mm projectiles were demilitarized. The process consisted of extracting the nose closure, pulling the burster well, draining the liquid GB, thermally decontaminating the empty projectiles, and storing the GB for neutralization.

The GB was neutralized at CAMDS using the Agent Destruction System (ADS). The ADS was modeled after the existing facilities at Rocky Mountain Arsenal (RMA) with certain modified equipment configurations. In the ADS design, the caustic solution and GB are blended in the reactor rather than in a mixing tee, as done at RMA. Also, the heat of the reaction is removed by a reactor recirculation system and cooling jacket in the ADS rather than a heat exchanger downstream of the mixing tee. These modifications were done to eliminate foaming and line-plugging problems experienced at RMA and to improve process operations in general. Also, these changes were necessary to accommodate caustic neutralization of the VX acid brine in the second step of the VX neutralization process.

The other significant difference involved the changeover from the use of spray dryers at RMA to drum dryers at CAMDS for evaporating the water and reducing the brines from neutralization to salts. The main reason for the changeover was to avoid the conditions present in spray drying that were found to be conducive to GB reformation; these conditions were the high operating temperature and exposure to acidic combustion gases. Furthermore, the drum dryers involved much less air volume because air was used not for heat transfer but only as an effluent to carry away water vapor; thus, a smaller volume of air had to be dealt with in the event of GB emission. The operation of the drum dryers was also more cost-effective than the spray dryers.

Significant problems were encountered with the GB neutralization process at CAMDS. The neutralization process did not follow the expected course and presented two major problems: (1) minute quantities of GB were found in the brines and (2) the process took significantly longer than expected. A significant portion of the problems CAMDS was experiencing can be explained by the change in the standard for certifying that the brine was agent-free. At RMA, the standard that was imposed was the mere presence of 5% excess caustic in the brine. However, after RMA faced problems with GB emission in the spray dryer, the U.S. Department of Health and Human Services adopted a more stringent standard requiring testing of the brine. This standard was converted into the certification target level of no more than 2 ng of GB per mL of brine. Thus, faced with a much stricter standard, CAMDS had more difficulty. The Army spent over $7 million in attempts to determine the cause of the presence of minute quantities of GB in the brine after neutralization. Four predominant theories were proposed but not confirmed: reversal of the reaction under equilibrium conditions, occlusion or encapsulation with solids, introduction as an artifact under the analytical procedures used to detect GB, and false positives resulting from the complex sample matrix. In efforts to achieve the certification target level, the process took much longer than the expected four hours. Some neutralization reactions took as long as 40 days while most lasted 10 to 20 days. Excess caustic was added to accelerate the neutralization reaction and this resulted in significantly high salt production. Other problems had to do with residual water left in the storage tank before filling with GB. Apparently, the water reacted with the GB in a hydrolysis that resulted in a very low-pH solution that consumed more caustic than expected and formed a hard to pump sledge that occasionally led to pipe-clogging. On other occasions, although GB was added to the reactor in a controlled

manner to reduce heat build-up, the use of a cooling system resulted in too-low temperatures that caused precipitation to clog pipes from the reactor vessels.

The problems encountered with industrial-scale neutralization of GB led the Army to abandon neutralization for disposal of chemical munitions regardless of the type of chemical agent present. As previously mentioned, the NAS 1969 report had recommended incinerating mustard. VX, which was next to be neutralized, was not neutralized on an industrial scale because of the problems experienced with GB neutralization. The rationale for abandoning neutralization was based on a number of factors: (1) the sheer complexity of the process (as compared to incineration, which was the emerging industrial technology for disposal of organic substances) and the sensitivity of the process-to numerous parameters that would slow the reaction or even promote hydrolysis reversal, reforming GB, (2) the quantity and nature of the waste that was produced, and (3) the high capital costs (for the complex of equipment required) and operating costs. On March 9, 1982, at a Configuration Policy Board meeting, the Army officially decided to abandon neutralization and adopt incineration for disposal of chemical agent/munitions.

D.4 INCINERATION TECHNOLOGY

D.4.1 BACKGROUND

The National Research Council restudied the question of the most effective, economical, and safe means for chemical agent/munitions disposal at the request of the Army in 1982. After reviewing the advances in technology since the 1969 NAS report, the National Research Council (1984) concluded that thermal destruction was the best means for disposal.

Incineration is the thermal decomposition of organic compounds into simpler inocuous inorganic compounds, mainly water and carbon dioxide. In addition to these two compounds, acid gases such as hydrogen fluoride (from GB), phosphorus pentoxide (from GA, GB, and VX), nitrogen dioxide (from GA and VX), and hydrogen chloride (from mustard) arise from combustion of the chemical agents considered here. These acid gases can be removed by scrubbing. Incineration is a safe, environmentally sound method of destroying toxic organic compounds. Incineration is used commercially for disposal of polychlorinated biphenyls (PCBs), pesticides, herbicides, and hazardous organic waste products.

As an alternative to chemical neutralization, incineration was seriously considered for a number of reasons.

- It was already in use for disposal of mustard agent.
- It is fairly simple and straightforward.
- The products do not allow reformation of the reactants (such as GB).
- It does not produce as much waste as neutralization, and the waste is generally inorganic.

- The capital and operating costs are much less than those of neutralization.

- It was already required to augment chemical neutralization in the demilitarization of chemical munitions.

Because neutralization applies only to the agent, incineration had to be used to deactivate explosive/propellant components and to thermally decontaminate munition cavities and metal parts. Because incineration would also be required with chemical neutralization, there was little reason to not use it for the complete destruction and eliminate neutralization altogether.

The Army's experience in incineration of chemical agents includes demilitarization of over 2.8 million kg (6.2 million lb) of agent (over 60,000 munitions and containers) at RMA and CAMDS together.

D.4.2 ROCKY MOUNTAIN ARSENAL

Two major demilitarization operations using incineration have been accomplished at RMA. The first, designated as PROJECT EAGLE--Phase I, occurred between August 1972 and February 1974 and disposed of 2.78 million kg (6.14 million lb) of mustard agent (H and HD) in ton containers. The second operation involved disposal of over 21,000 chemical agent identification sets (CAIS), which contained about 16,000 kg (36,700 lb) of various chemical agents.

PROJECT EAGLE--Phase I was intended to dispose of excess stocks of mustard agent stored at RMA since the 1940s. They were scheduled for ocean dumping until the NAS recommended incineration. PROJECT EAGLE--Phase I was carried out in three stages: incineration (1) at 1 gpm and (2) at 2 gpm and (3) incineration of residue remaining in the ton containers. The demilitarization process consisted of the following steps: preheating the ton container, draining of the mustard agent, incineration of the agent, thermal decontamination of the container, and furnace emission control.

The primary incinerator for disposal of the drained mustard agent was a modified hydrazine furnace. It did not have a separately fired afterburner but used the long residence time between the combustion chamber and the pollution abatement system (PAS) to ensure complete destruction. Two ton-container furnaces containing 8 and 14 natural gas burners, respectively, were used to incinerate the residue left in the ton containers (i.e., to thermally decontaminate it). The PAS for the furnaces consisted of two caustic quench and scrubber systems connected to a single electrostatic precipitator and stack. An electrostatic precipitator was added to ensure that particulate emission and stack opacity limits were met because rust (iron oxide) from inside the tan containers posed potential emissions problems. All wastewater and scrubber brines generated by the PAS were dried into waste salts using a spray-dryer.

The stack and work area were monitored for mustard during incineration. The perimeter was also monitored for nitrogen dioxide, sulfur dioxide, ozone, suspended particulate, and HCl with sequential samples on a 6-h-cycle monitor. Although particulate emission standards were occasionally exceeded, PROJECT EAGLE--Phase I had no significant impact on ambient air quality.

A total of 6.4 million kg (14 million lb) of spray-dried salt was produced during PROJECT EAGLE--Phase I. Although the test results did not show the majority of salt samples to be hazardous waste, under RCRA guidelines, excessive arsenic and lead in some samples and sample variability forced a decision to place all of the salt in a RCRA-approved hazardous-waste landfill. The decontamined ton containers were sold as metal scrap. The ash and electrostatic precipitator residue were disposed of by land dilution.

The second major incineration operation at RMA was the disposal of chemical agent identification sets (CAIS). CAIS were used for identification of various chemical agents during training but were declared obsolete in 1971. There were 18 different configurations; each set contained from one to five different agents. Although normally only one chemical agent at a time is processed during disposal, the agents could not be easily separated and, hence, were incinerated simultaneously. This circumstance made the CAIS disposal operation unique.

The CAIS were first incinerated in the RMA deactivation furnace followed incineration in the RMA decontamination furnace. The deactivation furnace was modified by the addition of an afterburner to accommodate glass ampules and bottles. The decontamination furnace was modified by the installation of a new high-temperature refractory and pedestals to support the CAIS shipping containers and reconfiguration of the burners to ensure uniform heating. An electrostatic precipitator was added to the PAS to remove arsenic oxide and other particulates in the furnace exhaust. The PAS, in addition, had a quench and dual-packed column scrubbers to remove acid gases. All wastewater, quench, and PAS scrubber brines were dried into salt with a spray dryer.

The work areas and stack emissions were monitored for the presence of all nine chemical agents. The spray-dried salts were drummed and disposed of in 1985 in a hazardous-waste landfill. The electrostatic precipitator residue, found to contain excessive arsenic, lead and zinc, was drummed and also placed into a hazardous-waste landfill. The furnace residue, in spite of its acceptability for disposal in a sanitary landfill, was, likewise, placed in a hazardous-waste landfill. The lead from the gaskets in the CAIS shipping containers was sold as scrap metal, and the CAIS shipping containers, themselves, were retained after thermal decontamination for use as overpacks for leaking chemical munitions.

D.4.3 CHEMICAL AGENT MUNITIONS DISPOSAL SYSTEM

The primary purpose of the CAMDS at Tooele Army Depot is to test and evaluate equipment and processes to be used in chemical agent/munitions disposal plants. CAMDS is authorized to dispose of some chemical agents/munitions, pursuant to acquiring specific data. CAMDS experience with incineration of chemical agent consists of disposing 34,000 kg (75,000 lb) of GB and 3600 kg (8000 lb) of VX (i.e., 38,000 munitions). Three furnaces have been used at CAMDS: a deactivation furnace system, a metal parts furnace (MPF), and a liquid incinerator.

The purpose of the deactivation furnace system is to incinerate residual chemical agent and deactivate explosives/propellant. The deactivation furnace system experience consists of the incineration of 18,300 M55 rockets. It consists of a feed chute with double tipping

blast valves, rotary kiln furnace protected and isolated by a reinforced concrete enclosure (to contain a potential detonation), heated discharge conveyor, scrap conveyor, cyclone separator, slagging afterburner, and PAS. Segmented rocket pieces, munition bodies and cavities with or without residual agent, and all other charge for the deactivation furnace system are fed from the explosive containment cubicle.

The purpose of the PAS for the deactivation furnace system is to remove acid gases and particulate matter from the furnace exhaust before atmospheric release. The PAS consists of a quench tower, variable-throat venturi scrubber, packed-bed wet scrubber tower, demister, induced-draft fan and exhaust stack. The deactivation furnace system exhaust is monitored for oxygen, carbon monoxide, carbon dioxide, sulfur dioxide, nitrogen dioxide, and the presence of chemical agents. A system of eight perimeter monitoring stations is used to check ambient air quality.

To date, five incineration tests have been completed on the deactivation furnace system: (1) GB challenge test, (2) undrained M55 rocket incineration test, (3) PCB incineration test, (4) drained M55 rocket incineration test, and (5) ocean dumping permit application analysis of deactivation furnace system scrubber brines.

The GB challenge test was conducted in April 1977 to determine the GB destruction and removal efficiency of the deactivation furnace system PAS with the kiln and afterburner on and off. The results were 99.9% GB removed efficiently (at a maximum GB challenge of 0.36 mg/m^3) without the afterburners and a 99.9998% GB destruction and removal efficiency (at a GB challenge rate of 82 mg/min), with the afterburner.

The undrained M55 rocket incineration tests were conducted in May 1981 and July 1982 to determine the ability of the deactivation furnace system to incinerate undrained M55 rockets containing GB. Results demonstrated that the deactivation furnace system has a GB destruction and removal efficiency of 99.999999% based on a feed rate of 2.25 L GB per min, which is equivalent to the agent from one undrained rocket every two 'minutes. The deactivation furnace system demonstrated it can process simulated M55 rockets at a feed rate of one-third undrained M55 rocket per minute, at a GB destruction and removal efficiency of 99.999994%.

The PCB incineration test was conducted in March 1986 to evaluate the ability of the deactivation furnace system to meet the Toxic Substances Control Act (TSCA) solid PCB incineration criteria when burning M55 rockets with shipping and firing tubes. The outcome was that the approved Environmental Protection Agency (EPA) analytical procedures were not sensitive enough to demonstrate the 99.9999% destruction and removal efficiency required by TSCA. In comparing test results to commercial PCB incinerators, the deactivation furnace system emissions are an order of magnitude less. A Department of Health and Human Services review of the test results concluded that there is no threat to human health from the level of emissions coming from the deactivation furnace system.

The drained M55 rocket incineration test was run in May 1986 to establish baseline performance data on the deactivation furnace system. Test results were as follows:

- Drained M55 rockets can be incinerated at up to 18 rockets per hour with a GB destruction and removal efficiency of 99.9999%.

- The combustion efficiency of the slagging afterburner was greater than 99.9%.
- The particulate emission rate was less than the RCRA standard of 180 mg/m^3.
- The PAS brines did not exhibit RCRA extraction procedure toxicity.
- Ash samples from the deactivation furnace system exceeded the RCRA toxicity limit for cadmium content and cyanide reactivity.
- No products of incomplete combustion (PIG), in terms of RCRA-specified compounds, were detected.

The ocean dumping permit application analysis of the deactivation furnace system scrubber brine was conducted in June 1986 to determine the composition and concentration of pollutants. These tests indicated that the deactivation furnace system brines are non-RCRA-hazardous, although post-burn increases in S04, P04, F, and Cl were indicated. While some semi-volatile organic priority pollutants were present, no volatile organic priority pollutants were found.

The primary purpose of the CAMDS metal-parts furnace is to thermally decontaminate munition bodies, ton containers, projectiles, and other metal parts having residual chemical agent (in the absence of explosive/propellant). In addition, the MPF can be used to incinerate bulk agent. The MPF experience comprises the incineration of 16,032 kg (35,271 lb) of GB, 3575 kg (7866 lb) of VX, and 19,631 projectiles. The MPF system consists of a charge car, a roller hearth furnace containing three chambers--punch, volatilization, and burnout; a primary fume burner; an auxiliary fume burner; and a PAS. Moreover, the MPF system includes scrap handling and cooling equipment. Items are fed from the multipurpose demilitarization machine or bulk drain station to the MPF by the power-driven charge car. The pollution abatement system for the MPF is identical to the deactivation furnace system PAS, except for size/capacity differences because of differences in the volumetric flow rate of the MPF exhaust gas.

Six types of incineration tests have been conducted with the MPF: (1) evaluation of the PAS, (2) GB agent injection incineration, (3) thermal decontamination of drained 105-mm projectiles, (4) in situ incineration, (5) development of design data for the Johnston Atoll Chemical Agent Disposal System (JACADS) MPF and liquid incinerator, and (6) VX agent incineration tests.

The metal-parts furnace PAS was evaluated in August 1978 in terms of its ability to remove agent vapors from the furnace exhaust in the event of a furnace upset, Test results showed that the GB destruction and removal efficiency of the PAS under cold conditions was at least 99.99% up to GB challenge rates of 3.17 mg/m^3; under normal operating and upset conditions, with both partial and total failure, the GB destruction and removal efficiency was greater than 99.999%.

Agent injection incineration tests were conducted in July 1981 to determine the limiting factor for agent incineration in the MPF and in March 1982 to determine if incineration was -a viable alternative to neutralization for GB destruction. Test results showed that the limiting

factor was the stack opacity standard. However, the stack opacity limit could be met at a higher GB flow rate if the liquid flow rate to the quench tower was increased. The GB destruction and removal efficiency was greater than 99.99999%, when the GB flow rate was equal to 446 pounds per minute. Thus, MPF incineration of GB was a viable alternative.

The MPF was tested for its capability to thermally decontaminate 105-mm projectiles (drained of GB) in May 1982, but the results were inconclusive.

Undrained 155-mm GB-filled projectiles were processed through the MPF between October and December 1982 to test in situ incineration of munitions (as opposed to injection incineration in which munitions are first drained, of the agent and empty munition incinerated separately). The test demonstrated that in situ incineration was feasible, but further testing was necessary to improve it.

Testing with the MPF was carried out between 1980 and 1984 to develop design data for the JACADS metal-parts furnace and liquid incinerator. Instability of furnace drafts with the above in situ incineration led to its rejection in favor of agent injection incineration. The test developed the result that the maximum GB incineration flow rate is 4 kg/min (550 lb/h) at a destruction and removal efficiency of 99.999999%. A complete chemical characterization of the brine from the metal-parts furnace PAS was obtained.

VX injection incineration testing was conducted in June 1984 on the MPF. Results demonstrated that the MPF can incinerate VX at flow rates of up to 3 kg/min (400 lb/h) at a destruction and removal efficiency of 99.9999998%. Particulate emissions from the incineration of VX were well below the RCRA limit, and chloride levels from spent decontamination were below the detection limit.

The purpose of the CAMDS liquid incinerator is to burn (1) chemical agent drained from munitions or bulk containers and (2) organic matter in waste liquids such as spent decontamination solution. The liquid incinerator was operated for over a year and incinerated 17,200 kg (37,930 lb) of GB. The liquid incinerator has a primary burner and afterburner, and it is designed to take advantage of the high heat of combustion of the chemical agent. The CAMDS liquid incinerator has one-third the capacity of the liquid incinerator to be used at JACADS and the proposed chemical stockpile disposal plants. The CAMDS liquid incinerator also differs in that it does not have a separate PAS. Instead it is hooked up to the CAMDS metal-parts furnace PAS; the result being that the liquid incinerator and MPF cannot be operated simultaneously.

Between December 1985 and February 1986, a series of tests was conducted to obtain (1) emissions data on the liquid incinerator and the metal-parts furnace PAS (with the liquid incinerator operating) and (2) effluent data on the liquid incinerator sump brines, PAS scrubber brines, and salts formed from drying the brines. Both the particulate and hydrogen fluoride emissions results were considered inconclusive. No significant emissions or volatile or semivolatile products of incomplete combustion were identified. The liquid incinerator sump brines, PAS scrubber brines, and salts formed from drying the brines were all found to be non-RCRA-hazardous.

Tests were conducted in June 1986 to obtain the composition of and concentration of pollutants in liquid incinerator (LIC) scrubber brines. The LIC scrubber brines did not exhibit any metals exceeding the RCRA limit. Large post-burn increases in phosphate and fluoride concentrations were shown (due to GB incineration). No organic priority pollutants, either volatile or semi-volatile, were detected in significant amounts.

0.5 BASELINE INCINERATION PROCESS

Incineration is the proposed method of disposal for the Chemical Stockpile Disposal Program (CSDP). The incinerators will be based on JACADS design (as shown in Fig. C.2 in Appendix C), differing only in-fuel used and capacity because of inventory variations from site to site. Four incinerators are discussed in this section: (1) deactivation furnace system, (2) liquid incinerator, (3) metal-parts furnace, and (4) toxic dunnage incinerator. Each incinerator will have its own pollution abatement system, and the pollution abatement system will also be discussed.

D.5.1 Incinerators

The CSDP deactivation furnace system will consist of four separate sections: (1) rotary retort, (2) blast attenuation duct, (3) cyclones, and (4) afterburner. In addition to its originaily designed purpose of destroying residual agent and deactivating explosives/propellants, the deactivation furnace system can be used to thermally decontaminate spent filters. At any given time, the deactivation furnace system will process components from only one munition type and one agent.

The rotary retort of the deactivation furnace system consists of two feed chutes, each with two blast gates in series, a charge end subassembly, a furnace retort drive mechanism, a discharge end subassembly, and a heated discharge conveyor. The feed chutes connect with the explosive containment room. The blast load attenuation duct conducts the flue gases from the retort to the cyclone, which separates solid particulates from the gas stream. The afterburner combusts any organic vapor not combusted in the off gases leaving the cyclone. The gases from the afterburner flow to the quench tower in the deactivation furnace system pollution abatement system.

The CSDP liquid incinerator is a two-stage refractory lined incinerator designed to combust drained liquid chemical agent and organic matter in waste liquor, such as spent decontamination solution. The liquid incinerator consists of a primary combustion chamber and afterburner. Drained chemical agent stored in the toxic tank is pumped through duplex strainers to the primary combustion chamber, where it is air-atomized and burned. The afterburner ensures complete combustion of any persistent chemical agent present in the gases leaving the primary combustion chamber. Spent decontamination solution is atomized to the stream of gases entering the afterburner from the primary combustion chamber and is incinerated in the afterburner. The resultant flue gas from the afterburner flows to the liquid incinerator PAS.

The CSDP metal-parts furnace is designed to thermally decontaminate drained, defused, and nonburstered projectiles; bulk items such as ton containers and bombs; equipment; and contaminated combustible dunnage.

The MPF consists of (1) a horizontal, three-chamber, roller hearth unit and (2) an afterburner. The hearth unit's first chamber is an, inlet airlock that receives items for feed. The hearth's second chamber is a refractory-lined burnout chamber. split into three individually controlled temperature zones. The hearth's third chamber is an egress airlock. Gases from all three chambers are vented to the afterburner, a horizontal refractory-lined cylindrical vessel, to ensure complete combustion. Exhaust from the afterburner flows to the metal-parts furnace PAS. Handling of molten aluminum in special feed cars is accommodated for processing spray tanks and M116 weteye bombs.

The CSDP toxic dunnage incinerator is designed to incinerate agent-contaminated (as well as uncontaminated) dunnage. Dunnage includes scrap wood, pallets, shipping boxes, laboratory solid wastes, work garments, and miscellaneous waste. Metal mine drums having small amounts of combustible packing will also be disposed of in the toxic dunnage incinerator. The toxic dunnage incinerator consists of two separate chambers: the primary combustion chamber and an afterburner. Solid wastes are charged into the primary chamber via an airlock, elevator, and ram feeder. The flue gases from the primary combustion chamber are ducted to an afterburner to ensure complete combustion. Exhaust from the afterburner is vented to the PAS.

D.5.2 POLLUTION ABATEMENT SYSTEMS

Each CSDP incinerator will have its own pollution abatement system. The PAS configurations for the deactivation furnace system, liquid incinerator, and MPF are identical but differ in equipment size. The PAS consists of the quench tower, venturi scrubber, packed-bed scrubber tower, demister vessel, induced-draft fan, and a common stack. The quench tower cools by means of caustic in countercurrent flow, the hot gases entering from the afterburner until the adiabatic saturation temperature is reached. The cooled gases from the quench tower enter the venturi scrubbers, which are variable-plug throat scrubbers with a normal operating pressure drop across the throat. Scrub solution is sprayed into the gas stream to remove particulates. The scrubber tower separates the two-phase flow from the venturi scrubbers. The separated gas phase enters the demister vessel to capture acid mist and particulate. The induced-draft fan provides the motive force for the exhaust gases throughout the incinerator and PAS. The common stack provides atmospheric discharge of the scrubbed flue gases from the PAS for all three incinerators. Sampling ports are furnished in the stack for monitoring.

The toxic dunnage incinerator PAS differs from the PAS for the other three incinerators. It consists of a quench tower, baghouse, induced-draft fan, and a stack. All of the toxic dunnage incinerator PAS components function identically to those discussed previously, except for the baghouse. The baghouse is used to remove particulates from the gas stream by forcing the stream through a fabric. Dislodged solids are collected in a hopper beneath the bags and packed into drums.

D.6 INCINERATION AND POLLUTION ABATEMENT SYSTEMS TEST PROGRAM

A comprehensive test program is under way at CAMDS to verify the performance of the munition disassembly machines, incineration, and PAS

to be used in JACADS and the proposed CSDP. The test program is divided into agent test windows because only one chemical agent is processed during any one period, although more than one type of munition may be involved. For example, the liquid incinerator can incinerate VX from ton containers while the deactivation furnace system is incinerating drained VX M55 rockets. Three types of data will be obtained during incineration system testing and evaluation: (1) performance, including heat and material balances, temperature profiles, furnace stability, etc.; (2) environmental compliance, which includes analysis of exhaust gases and residues from the incinerators; and (3) environmental concerns, including information on the exhaust gases and solid residues, which is not required by current environmental regulations, but may be required to support permitting actions or may be covered by proposed or future regulations. A comprehensive report on performance and operational characteristics will be prepared at the end of each agent test window. Test reports involving environmental compliance and environmental concerns data will be obtained by conducting special test burns with the aid of a contractor (because of the special sampling and analysis procedures).

Incineration system tests were run in May 1986 for GB agent for products of incomplete combustion (PICs). No PICs, in terms of RCRA-specified compounds, were detected in the exhaust gases and PAS brines. Table D.2 presents the results of the PIC analysis completed for the composite furnace residue sample; however, these PIC results cannot be considered definitive. Similar tests for VX are scheduled at CAMDS from January to June 1988, and HD tests are scheduled for August to December 1988.

Environmental compliance data will be obtained with respect to standards established by the Clean Air Act (as administered by all eight states where chemical agent munitions stockpiles are located), including new-source review for nonattainment areas and prevention of significant deterioration. RCRA compliance data will be obtained with respect to incineration emission standards. Incineration emission standards cover the chemical agent, POHC, HCl, particulates, SO_2, and opacity. RCRA compliance data will be obtained for solid waste residue to determine if it is a characteristic waste; this applies only in states where chemical agents are classified as characteristic waste. In states where the agents are "listed," this data could be used for delisting petitions. Environmental compliance data will also be -obtained for the Marine Protection, Research, and Sanctuaries Act.

Environmental data will be obtained on: (1) products of incomplete combustion, (2) heavy-metal emissions from the incinerators, and (3) toxicity characteristic leaching procedures as applied to solid waste residues.

Each test burn in the test program will consist of at least one baseline or background trial plus three trials in which the chemical agents/munitions are processed. With the exception of chemical agent sampling and analysis and the brine and nitroglycerine analytical procedures, all of which are Army procedures, EPA-approved sampling and analysis procedures will be used.

To be permitted under RCRA, an incinerator and its pollution abatement system must complete a special trial burn supervised by representatives from EPA and the state involved. The purpose of the

Table D.2. Product of incomplete combustion analysis of DFS residue from May 1986 rocket incineration test

Parameter	Trial 1[a] (μg/kg)	Trial 2[a] (μg/kg)	Trial 3" (μg/kg)
Unknown	23	<10	<10
1,4-dioxane	92	Cl0	230
2,2-dimethylhexane	<10	1400[b]	<10
Chloromethane	<38	160	120
Dichloromethane	<11	210	2ooo
Chloroform	<6.2	<6.2	11
4-methy/-2-pentanone	<6.2	<6.2	11
Carbon tetrachloride	<11	<11	15
Benzene	190	350	46

[a]Values reported with a "less than" sign indicate that the results are below detection limits of the analytical method used.

[b]Suspect result due to laboratory contamination. Verified by blank analysis.

Source: US. Army 1987a. ***Chemical Agent and Munition Disposal: Summary of the U.S. Army's Experience,*** Report SAPEO-CDE-IS-87005, Office of the Program Manager for Chemical Munitions (Demilitarization and Binary), Aberdeen Proving Ground, Md.

trial burn is to ensure compliance with the standards for POHC, HCl, and particulate emissions described in Tabie D.3. The operating permit issued to the incinerator facility will be based on the results of the trial burn. A trial burn will take approximately two weeks to complete.

Under RCRA, an incinerator must demonstrate a 99.99% destruction (incinerator) and removal (pollution abatement system) efficiency (DRE) for each POHC designated in the feed. POHCs for the trial burn- are usually selected by EPA or the state from the list of RCRA compounds (40 CFR 261, Appendix VIII) based on the concentration or degree of difficulty of incineration of the hazardous organic constitutents in the waste feed.

The state and federal hazardous waste regulators have indicated that each chemical agent and nitroglycerin (an energetic compound contained in the M55 rocket motor) should be selected as POHCs for the CSDP disposal plant trial burns. In addition, the regulators have requested that surrogates be used to demonstrate incinerator performance prior to the chemical agent/munition trial burns. The POHCs for the blister and nerve agent surrogates are carbon tetrachloride and 1,1,1-trichloroethane. Beyond being more difficult to incinerate, the surrogates will simulate the agents' particulate matter and HCl emission characteristics.

In addition to demonstrating agent DRE during the CAMDS test burns, special test burns will be conducted with the chemical agent surrogate selected for the CSDP trial burns. This will allow the Army to become familiar with surrogates and to evaluate the performance of the furnaces and incinerators from this perspective before the CDSP disposal plants are constructed.

0.7 EXPERIENCE WITH ACCIDENTAL RELEASES AT CAMDS

An incident at the liquid incinerator facility of the CAMDS at Tooele Army Depot (TEAD) raises a series of concerns about the susceptibility of the incineration process to failures resulting in the release of chemical agent. The incident began when the real-time monitor on the liquid incinerator building exhaust filter stack and an automatic continuous air-monitoring system in the liquid incinerator motor control room activated at approximately 7:50 a.m., January 28, 1987.

Although no chemical agent was being processed at the time, GB was being stored in the agent tank located in the liquid incinerator primary combustion chamber (PCC). The PCC room is maintained at a negative pressure with respect to its surrounding environment, and incinerator exhaust is filtered before it is released. Liquid. chemical agent (GB) apparently leaked from the primary containment piping network located inside the PCC of the liquid incinerator facility. *The Technical Investigation Board Report, Chemical Incident at the Chemical Agent Munition Disposal System (CAMDS)', 28 January 1987* (U.S. Army 1987c), indicates that the exhaust from the liquid incinerator PCC was switched from the primary filter unit to the secondary (backup) filter unit. Changeout of the charcoal on the primary filter was initiated. No personnel were exposed to agent. Agent was not detected outside of the liquid incinerator or metal-parts furnace buildings. The Army's *Report of CAMDS Follow-up Assessment Panel, 24 July 1987* (U.S. Army 1987b) reports that, the source of contamination appeared to be an accumulation of residual agent from prior agent transfer. A secondary containment

Table D.3. CSDP incinerator emission standards

Emission	Standard	Regulation/requirement[a]
Agent[b]	0.003 mg HD/m^3	DOD 6055.9-STD
	0.0001 mg L/m^3	DOD 6055.9-STD
	0.0003 mg GB/m^3	DOD 6055.9-STD
	0.0003 mg GA/m^3	*c*
	0.00003 mg VX/m^3	DOD 6055.9-STD
POHC[d]	99.99% DRE	*40* CFR 264.343(a)(1)
HCl[d]	The larger of 4 lb/h or 99% removal efficiency	*40* CFR 264.343(b)
Particulates[d,e]	0.08 gr/dscf at 7% O_2	*40* CFR 264.343(c)
	0.03 gr/dscf at 12% CO_2	COMAR 10.18.08
SO_2	500 ppm (volume)	CAMDS FEIS

[a]State regulation citations obtained from U.S. Army 1987a.

[b]One-hour average.

[c]No current standard, assumes same standard as GB.

[d]States have adopted RCRA standards for POHC, HCl, and particulate emissions from hazardous waste incinerators.

[e]RCRA particulate standard is more stringent than state air quality particulate standards except in Maryland.

Source: U.S. Army 1987a. ***Chemical Agent and Munition Disposal. Summary of the U.S. Army's Experience,*** Report SAPEO-CDE-IS-87005, Office of the Program Manager for Chemical Munitions (Demilitarization and Binary), Aberdeen Proving Ground, Md.

breach occurred when GB vapor migrated from the controlled environment of the PCC room to an unprotected work area of the liquid incinerator building corridors. The pathway of agent migration appeared to be through a louver located in the wall adjacent to the liquid incinerator agent storage tank. In addition, agent GB vapor remaining in the liquid incinerator PCC was not totally filtered from the system exhaust stream, and minute quantities were later released into the atmosphere. The Department of Health and Human Services determined that the event posed no health hazard to the general public.

A series of conclusions and recommendations are derived as a result of the incident. These programmatic-level conclusions and recommendations are derived from the CAMDS facility but apply to all future disposal sites as well. Six basic conclusions and resulting Army actions are presented below:

1. No independent chemical disposal program inspection team exists to verify chemical disposal program compliance with regulatory, statutory, and safety guidelines. As a consequence, the Army will establish an independent chemical disposal program inspection team (e.g., a sanctioned ad hoc team, contractor, or Inspector General) chartered to periodically evaluate compliance with appropriate regulatory, statutory, and safety guidelines at current and future chemical disposal sites.

2. Quality assurance for plant operations within the chemical disposal program is inadequate. As a result, the PEO-PM will establish an overall quality assurance program for chemical disposal operations.

3. Baseline operating procedures and changes thereto are not effectively established, evaluated, and documented. The PEO-PM will establish standard baseline assessment procedures and ensure that changes to the baselines of all disposal sites are adequately evaluated.

4. The initial Army report (1987c) concluded that technology transfer throughout the chemical disposal community was inadequate. A subsequent Army revision (1987b) failed to concur with this finding. Nevertheless, emphasis on ensuring that a viable technical information exchange program for Army chemical disposal to include lessons learned will be continued.

5. Shortfalls in technical expertise exist within the chemical disposal program that have not been resolved through exploitation of outside assistance. The PEO-PM will determine shortfalls in technical expertise and supplement with outside assistance (e.g., ventilation, filtration, and chemical process engineering). The PMCM will review, with appropriate authorities, pay scales and grade structures for attracting required technical personnel.

6. The initial review board (Army 1987c) found that no standard exists for conducting preoperational surveys within the chemical disposal program. Subsequent Army review (1987b) failed to concur with this finding. The PMCM will continue to ensure that preoperational

surveys will be conducted to meet the overriding standards of safety.

REFERENCES FOR APPENDIX D

National Research Council 1984. Disposal *of Chemical Munitions and Agents,* National Academy Press, Washington, D.C.

U.S. Army 1987a. *Chemical Agent and Munition Disposal: Summary of the U. s. Army's Experience,* Report SAPEO-CDE-IS-87005, Office of the Program Manager for Chemical Munitions (Demilitarization and Binary), Aberdeen Proving Ground, Md.

U.S. Army 1987b. *Report of CAMDS Follow-Up Assessment Panel, 24 July 1987.*

U.S. Army 1987c. *Technical Investigation Board Report, Chemical Incident, at the Chemical Agent Munition Disposal System (CAMDS), 30 March 1987.*

Appendix E

LEAKING MUNITIONS AND THE DRILL AND TRANSFER SYSTEM

E.l OVERVIEW

Because of the age of the chemical stockpile, some munitions have begun to leak. The MS5 rockets have exhibited the largest incidence of leakage in the stockpile, while other munitions in the stockpile, such as the bombs, projectiles, spray tanks, mines, and bulk storage tanks, have relatively few leakers.

Special surveillance procedures to detect and containerize so-called "leakers" have been developed. It costs approximately $4,000 to containerize a leaker (Fig. E.l). It is decontaminated, encapsulated, sealed, and put back into segregated storage so that it is not a risk to anyone. Under the current procedures, storage areas are checked at least weekly for agent leakage. Igloos that contain munitions which have a history of leakage are checked daily with bubblers.

Leakage is apparent through visual evidence such as encrustation, exudate, localized paint discoloration, blistering, or peeling on the surface of the munition or storage container; or liquids on the surface of the munition, storage container, pallets/dunnage used for storage, and the storage igloo floor. Test evidence may be obtained by atmospheric sampling within the igloo and/or within storage containers using chemical agent detectors designed or modified especially to detect the presence of chemical agent vapor.

Starting in 1979, the U.S. Army Toxic and Hazardous Materials Agency (USATHAMA) instituted a Drill and Transfer System (DATS) to dispose of leaking munitions that were declared unserviceable, unrepairable, or obsolete, or that were recovered from firing ranges and disposal grounds.

DATS is a transportable facility, mounted on a series of trailers, designed to drain chemical agents from leaking munitions at the installations where they are currently stored. As the name implies, the agent is accessed by drilling through the munition wall. The agent is not destroyed; rather, it is transferred to a suitable container for safe storage. After being drained, the munition casings are decontaminated chemically and then fractured by detonation in a fragment-containing chamber.

The spent decontamination solutions are stored for subsequent drying; scrap metal and inert munitions bodies are stored for thermal decontamination. DATS has operated at Dugway Proving Ground (DPG), Pine Bluff Arsenal (PBA), Anniston Army Depot (ANAD), Lexington-Blue Grass Army Depot (LBAD), Umatilla Depot Activity (UMDA), and Pueblo Depot Activity (PUDA). To date, it has successfully processed about 900 munitions.

The design capacity of DATS is small; a production rate of approximately three to six rounds per day has been experienced. DATS will be placed on layaway status at DPG in FY 1988, because CSDP will handle the leaking munitions more efficiently.

ORNL DWG 85-13659

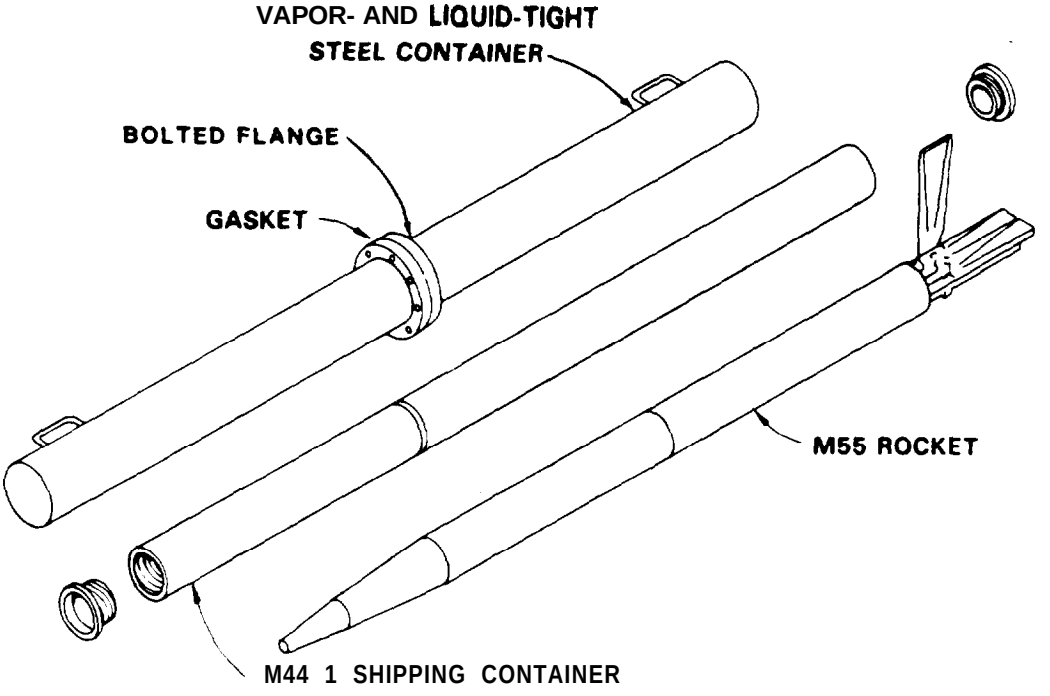


Fig. E.1. Overpack for leaking M55 rockets.

Appendix F

COMPLIANCE WITH THE ENDANGERED SPECIES ACT

Compliance with the Endangered Species Act of 1973 (ESA), as amended, requires each federal agency to ensure that any action it authorizes, funds, or carries out does not jeopardize the continued existence of endangered or threatened species or result in the destruction or adverse modification of their critical habitat.

The first stage in the compliance process is informal consultation consisting of a request to the U.S. Fish and Wildlife Service (FWS) and/or the National Marine Fisheries Service (NMFS) for information on whether any endangered or threatened species or critical habitat is present in the area of the site.

This appendix contains copies of the letters sent to FWS regional offices requesting information on federally listed threatened and endangered species for each of the disposal sites and FWS responses to the letters. Long species lists are not included but are available upon request. For some sites, responses from the states are included in order to document additional information for federally listed threatened and endangered species.

Because federally listed species may be affected by this action, formal consultation may have to be initiated before the disposal project is started. The Army must request, in writing, formal consultation from the U.S. Department of the Interior, FWS office with responsibility for the project area (e.g., the FWS office in Jackson, Mississippi, for the Arkansas and Alabama transportation areas). Requests for formal consultation must include (1) a description of the action to be considered; (2) a description of the specific area that may be affected by the action; (3) a description of any listed species that may be affected by the action; (4) a description of the manner in which the action may affect any listed species and an assessment of any cumulative effects; (5) reports including any environmental impact statement, environmental assessment, or biological assessments; and (6) any other relevant information available on the action or the affected listed species. Attention is also directed to Sect. 7(d) of the Endangered Species Act, as amended, which underscores the requirement that the federal agency and/or the permit or license applicant shall not make any irreversible or irretrievable commitment of resources during the consultation period that, in effect, would deny the formulation or implementation of reasonable and prudent alternatives regarding their actions on any listed endangered or threatened species (D. B. Jordon, U.S. FWS, Jackson, Miss., personal communication to L. L. Sigal, Oak Ridge National Laboratory, Oak Ridge, Tenn., April 30, 1984).

The results of this informal consultation with the regional offices of the FWS, NMFS, and the states were used to prepare the appropriate sections of Sects. 3 and 4 and Appendix 0.

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS INC

POST OFFICE BOX X
OAK RIDGE TENNESSEE 37831

May 21, 1986

Mr. Howard N. Larsen, Regional Director
U.S. Flsh and Wildlife Service
Boston Regional Office
One Gateway Center, Suite 700
Newton Corner, Mdssdchusttts 02158

Dear Mr. Larsen:

In order to prepare the terrestrial and aquatic ecology sections of the Department Of Army programmatic environmental impact statement for the dlsposdl of chemical agents and munitions, I am requesting information about terrestrial dnd aquatic species Of plants dnd animals listed or proposed to be listed as endangered, threatened. or of special concern which may be present in d corridor surrounding d proposed route for the transport of chemical agents and munitions. Enclosed art state maps and lists of counties of Interest. Please number the species and indicate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict impacts for d vdrlety Of potential accident scenarios.

Please also indicate, by county, for the drtd within the corridor:

(1) critical habltat for threatened dnd endangered species,

(2) major wildlife use artds for game dnd nongdme species,

(3) fish management areas within the corridor,

(4) important wetlands, and

(5) state-protected groundwater recharge artds.

We realize that thls is d lot to ask and we want you to know that we appreciate your help. If there are questions, please call me at (615) 574-7266 (FTS 624-7266) or Virginia Tolbtrt (615) 574-7288 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Envlronnntal Compliance and
Impact Analysts Group
Environmental Analysts Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

UNITED STATES GOVERNMENT

memorandum

U.S. FISH AND WILDLIFE SERVICE

ONE GATEWAY CENTER
SUITE 700
NEWTON CORNER, MASSACHUSETTS 02158

TO: Director, Fish & Wildlife Service
Washington, DC (Attn: DEC)

FROM: ACTING Regional Director
Region 5

DATE: AUG 1 , 1986

SUBJECT: Review of Draft Environmental Impact Statement for Chemical Stockpile Disposal Program (ER 86/852)

The May 21, 1986 letter which transmitted the-subject document directly from Oak Ridge National Laboratory requested an array of information regarding fish, wildlife, wetlands and groundwater, much of which we donothavc.

We have attached maps of affected counties in Maryland, Delaware, District of Columbia, Virginia, Pennsylvania and West Virginia, displaying the location of Federally listed threatened and endangered species and candidate species. We did not receive responses from two States so the data from Delaware are incomplete and the data from New Jersey are sbsent. Additional data from Delaware as well as the data from New Jersey will be forwarded directly to Oak Ridge in a separate submittal.

Army should be advised that candidate species are not legally protected under the Endangered Species Act. However, we do encourage them to afford special protection to such species when possible.

For Army's further information, we have enclosed copies of the Endangered Species Act Biological Assessment Guidelines and a list of National Wetlands Inventory map distribution centers.

Information regarding the location of fish and wildlife management areas and State protected groundwater recharge areas should be available from the respective State agencies.

Attachments

R-5-38

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS, INC.

POST OFFICE BOX X
OAK RIDGE, TENNESSEE 37831

July 9, 1987

tlr. Paul Nickerson
U.S. Fish and Wildlife Service
One Gateway Center, Suite 700
Newton Corner, Massachusetts 02 158

Dear Mr Nickerson:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munitions, we are requesting information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present in a corridor surrounding the proposed route for transport of chemical agents and munitions. Enclosed are state maps and lists of counties of interest. Pleese number the species and indicate their distribution by putting their numbers in the appropriate counties on the maps. This will allow us to predict impacts for a variety of potential accident scenarios. Please also indicate by county if there is critical habitat for threatened and endangered species or important wetlands that could be impacted.

Because of an accelerated time schedule for preparation of the FEIS, we would very much appreciate hearing from you by the first of August. Thank you for your assistance. If there are questions, please call me et (6 15) 574-7288 (FTS 624-7288) or Lorene Sigal (6 15) 574-7266 (FTS 624-7266).

Sincerely,

Virginia R. Tolbert

Virginia R. Tolbert, PhD.
Environmental Compliance and
Impact Analysis Group
Environmental Analyses Section
Environmental Sciences Division

United States Department of the Interior

FISH AND WILDLIFE SERVICE
ONE GATEWAY CENTER SUITE 700
NEWTON CORNER MASSACHUSETTS 02158

JUL 16 1987

Dr. Virginia R. Tolbcrt
Environmental Compliance 6
Impact Analysis Group
Oak Ridge National Laboratory
Post Office Box X
Oak Ridge, TN 37831

Dear Dr. Tolbert:

I have enclosed lists of Endangered and Threatened Species in the four states (MD, PA, VA, WV) that you inquired about in your July 15, 1987 letter. I have also forwarded a copy of your letter and the maps to our offices in Annapolis MD and State College, PA. If you need additional information about endangered and threatened species in Maryland, Delavare and Virginia contact Andy Mose. or Judy Jacobs in Annapolis, Maryland at (301) 269-6324. Information about Pennsylvania and Uest Virginia can be obtained from Sharon Morgan, State College, PA at (814) 234-4090

Sincerely,

Chief, Endangered Species

Enclosures

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS INC

POST OFFICE BOX X
OAK RIDGE TENNESSEE 37831

May 21, 1986

Mr. James W. Pullman, Jr., Rcglonal Director
U.S. Fish and Wildlife Service
Atlanta Rcglonal Office
Richard B. Russell Building
75 Sprlng Street, S.W., Room 1200
Atlanta, Georgia 30303

Dear Mr. Pullman:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munitions, I am requesting information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present In a corridor surrounding a proposed route for the transport of chemical agents and munitions. Enclosed are state maps and lists of counties of Interest. Please number the species and indicate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict impacts for a variety of potential accident scenarios.

Please also indicate, by county, for the area ulthln the corridor:

(1) critical habltat for threatened and endangered species,

(2) major wildlife use areas for game and nongame species,

(3) fish management areas ulthln the corridor,

(4) important wetlands, and

(5) state-protected grounduater recharge areas.

We realize that this is a lot to ask and we want you to know that we appreciate your help. If there are questions, please call me at (615) 574-7266 (FTS 624-7266) or Vlrginia Tolbert (615) 574-7288 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Environmental Compliance and
Impact Analyses Group
Environmental Analyses Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

United States Department of the Interior
FISH AND WILDLIFE SERVICE
ENDANGERED SPECIES FIELD STATION
100 OTIS STREET. ROOM 224
ASHEVILLE, NORTH CAROLINA 28801

July 7, 1986

Dr. Lorene L. Sigal
Environmental Analyses Section
Oak Ridge National Laboratory
P.O. Box X
Oak Ridge, Tennessee 37831

RE: 4-Z-86-463

Dear Dr. Sigal:

Your May 21, 1986, letter regarding proposed transport routes for chemicals and munitions in seven states was received June 9, 1986. We have reviewed the project for counties in Kentucky and Tennessee with regard to endangered and threatened species. The transport routes for the other states will be reviewed by our field offices in Jackson, Mississippi (Mississippi, Alabama, Arkansas, and Louisiana), and Jacksonville, Florida (Georgia).

The enclosed pages describe the federally listed endangered (E) and/or threatened (T) and/or species proposed for listing as endangered (PE) or threatened (PT) which may occur in the area of influence of this action.

The legal responsibilities of a Federal agency under Section 7 of the Endangered Species Act of 1973 (as amended) were detailed in material sent to you previously. If you would like another copy of this material, or if you have questions, please contact us at 704/259-0321 (FTS 672-0321).

Your concern for endangered species is appreciated, and we look forward to working with you on endangered species matters in the future.

Sincerely yours,

Richard G. Biggins
Acting Field Supervisor

Enclosure

United States Department of the Interior
FISH AND WILDLIFE SERVICE
ENDANGERED SPECIES FIELD STATION
2747 ART MUSEUM DRIVE
JACKSONVILLE, FLORID.1 32207

June 19, 1986

Ms. Lorene L. Sigal
Environment Analysis Section
Environment Sciences Division
Oak Ridge National Laboratory
P.O. Box x
Oak Ridge, TN 37831

Dear Ms. Sigal:

We have reviewed the proposed routes for chemical agents and munitions transport as requested by your letter of May 21, 1986.

The following endangered (E) or threatened (T) species may occur along the proposed transport routes in Georgia. These classifications apply to the Federal listing only, the State of Florida may have other State listed species. Georgia ?

Mammals

1. Gray bat (Myotis grisescens) - (E)
2. Indiana bat (Myotis sodalis) - (E)

Birds

3. Red-cockaded woodpecker (Picoides borealis) - (E)
4. Bald eagle (Haliaeetus leucocephalus) - (E)

The distribution for each species has been identified by Its corresponding number on the enclosed map. This is not to imply a particular species is found in this county, but only that the county is within historic range. There are no critical habitats or species proposed for listing found within this area.

If you have further questions please contact Mr. Don Palmer in this office.

Sincerely yours,

David J. Wesley
Field Supervisor

Enclosure

United States Department of the Interior
FISH AND WILDLIFE SERVICE
JACKSON MALL OFFICE CENTER
300 WOODROW WILSON AVENUE, SUITE 331
JACKSON, MISSISSIPPI 39213

June 26, 1986

IN REPLY REFER TO:
Log No. 4-3-86-522

Ms. Lorene L. Sigal
Oak Ridge National Laboratory
Post Office Box X
Oak Ridge, TN 37831

Dear Ms. Sigal:

This responds to your letter of May 21, 1986. requesting information on listed species which may be present in a corridor surrounding a proposed route for the transportation of chemical agents and munitions. We have also included information on proposed dnd candidate species. This information is provided on a county basis with (E) for endangered, (T) for threatened, (P) for proposed, dnd (C) for candidate species.

Alabama

Autauga	-	Alabama canebrake pitcher plant (C)
Bibb	-	red-cockaded woodpecker (E)
Blount	-	flattened musk turtle (P)
Bullock	-	eastern indigo snake (T)
Butler	-	red hills salamander (T)
Calhoun	-	red-cockaded woodpecker (E)
Chambers	-	little amphianthus plant (C)
Cherokee	-	green pitcher plant (E), Mohr's barbara's buttons plant (C), Krdl water plantain plant (C), Alabama leather flower plant (C)
Chilton	-	Alabama Cdnebrdke pitcher plant (C)
Choctaw	-	gopher tortoise (P)
Clarke	-	Alabama shovel-nosed sturgeon (C)
Clay		red-cockaded woodpecker (E)
Colbert	-	gray bat (E), pink mucket pearly mussel (E), rough pigtoe pearly mussel (E), orange-footed pimpleback mussel (E)
Conecuh	-	gray bat (E), red hills salamander (T), eastern indigo snake (T)
Cullman	-	flattened musk turtle (P)

Dallas	-	Alabama shovel-nosed sturgeon (C)
DeKalb	-	green pitcher plant (E), gray'bat (E), gopher tortoise (P), Krdl wdter plantain plant (C)
Elmote		Alabama Cdnebrdke pitcher plant (C)
Etowah		green pitcher plant (E), flattened musk turtle (P)
Fayette		flattened musk turtle (P)
Greene	-	Marshall's mussel (P), Judge Tdit's mussel (P), penitent shell mussel (P), stirrup shell mussel (P), relict trillium plant (C)
Jackson	-	gray bat (E), Indiana bat (E), Alabama lamp pearly mussel (E), fine-rayed pigtoe pearly mussel (E), shiny pigtoe pearly mussel (E), pink mucket pearly mussel (E), pale lilliput pearly mussel (E), snail darter (T)
Jefferson	-	watercress darter (E), flattened musk turtle (P)
Lauderdale		gray bat (E), pink mucket pearly mussel (E), white warty-back pearly mussel (E), rough pigtoe pearly mussel (E), ordnge-footed pimpleback mussel (E), slackwater darter (T), Alabamacavefish (E), golf stick mussel (C)
Laurence	-	red-cockaded woodpecker (E), pink mucket pearly mussel (E), flattened musk turtle (P)
Lee	-	relict trillium plant (C)
Limestone	-	gray bat (E), pink mucket pearly mussel (E), slackwater darter (T)
Macon	-	red-cuckaded woodpecker (E)
Madison	-	gray bat (E), fine-rayed pigtoe pearly mussel (E), pink mucket pearly mussel (E), shiny pigtoe pearly mussel (E), snail darter (T), Alabama cave shrimp (C)
Marshall	-	gray bat (El, green pitcher plant (E), red-cockaded woodpecker (E), fine-rayed pigtoe pearly mussel (E), shiny pigtoe pearly mussel (E), pink mucket pearly mussel (E), flattened musk turtle (P)
Monroe	-	red hills salamander (T), Alabama shovel-nosed sturgeon (C)
Morgan	-	gray bat (E), pink mucket pearly mussel (E)
Perry	-	Alabama shovel-nosed sturgeon (C)
Pickens	-	Curtus' mussel (P), Marshall's mussel (P), Stirrup shell mussel (P), penitent shell mussel (P)
Randolph	-	little amphianthus plant (C)
St. Cldir	-	Alabama leather flower plant (P), Alabama live-bearing snail (C)
Sumter	-	Marshall's mussel (P), Judge Tdit's mussel (P), penitent shell mussel (P), stirrup shell mussel (P)
Talladega	-	red-cockaded woodpecker (E), Alabama live-bearing snail (C)
Tuscaloosa	-	red-cockaded woodpecker (E), flattened musk turtle (P)
Walker	-	flattened musk turtle (P), Mohr's barbara's buttons plant (C)

Washington	-	gopher tortoise (C)
Winston	-	red-cockaded woodpecker (E), flattened musk turtle (P)

Arkansas

Ashley	-	red-cockaded woodpecker (E)
Baxter	-	gray bat (E), bald eagle (E)
Benton	-	bald eagle (E), gray bat (E), Ozark cavefish (T)
Boone	-	gray bat (E)
Bradley	-	red-cockaded woodpecker (E), Geocarpon minimum plant (P)
Calhoun	-	red-cockaded woodpecker (E)
Carroll	-	bald eagle (E), gray bat (E)
Clark	-	red-cockaded woodpecker (E), pink mucket pearly mussel (E), Wheeler's mussel (C)
Clay	-	pink mucket pearly mussel (E), pondberry plant (P)
Cleburne	-	Strecker's mussel (C)
Cleveland	-	Geocarpon minimum plant (P)
Cross	-	flat pocketbook pearly mussel (E)
Desha	-	bald eagle (E)
Drew	-	red-cockaded woodpecker (E), Geocarpon minimum plant (P)
Franklin	-	Geocarpon minimum plant (P)
Fulton	-	pink mucket pearly mussel (E)
Garland	-	Ouachita madtom (C)
Grant	-	red-cockaded woodpecker (E)
Hot Spring	-	Wheeler's mussel (C)
Independence	-	gray bat (E)
Izard	-	gray bat (E), Indiana bat (E)
Jackson	-	pondberry plant (P)
Laurence	-	pink mucket pearly mussel (E), pondberry plant (P)
Madison	-	gray bat (E)
Marion	-	gray bat (E), Ozark big-eared bat (E), bald easle (E)
Mississippi	-	bald eagle (E)
Monroe	-	pondberry plant (P)
Newton	-	gray bat (E), Indiana bat (E)
Perry	-	red-cockaded woodpecker (E)
Poinsett	-	fat pocketbook pearly mussel (E)
Randolph	-	pink mucket pearly mussel (E)
St. Francis	-	fat pocket pearly mussel (E)
Saline	-	red-cockaded woodpecker (E), Ouachita madtom (C)
Searcy	-	gray bat (E)
Sharp	-	gray bat (E)
Stone	-	gray bat (El, Indiana bat (E), cave crayfish (C)
Union	-	red-cockaded woodpecker (E)
Van Buren	-	gray bat (E), Strecker's mussel (C)
Washington	-	Ozark big-eared bat (E), gray bat (E)

Louisiana

Union - red-cockaded woodpecker (E), bald eagle (E)

Mississippi

Itawamba - Curtus' mussel (P), penitent shell mussel (P)
Lowndes - Marshall's mussel (P), Judge Tait's mussel (P), penitent shell mussel (P), stirrup shell mussel (P)
Monroe - Curtus' mussel (P), penitent shell mussel (P)
Neshoba - ringed sawback turtle (P)
Noxubee - red-cockaded woodpecker (E)
Oktibbeha - red-cockaded woodpecker (E)
Sunflower - pondberry plant (P)
Wayne - red-cockaded woodpecker (E), gopher tortoise (C)
Winston - red-cockaded woodpecker (E)

If you need more specific locations for any of these species, please let us know.

We appreciate your participation in the efforts to enhance the existence of endangered species.

Sincerely yours,

R. M. Dawson

for Dennis 8. Jordan
Field Supervisor
Endangered Species Field Office

cc:
ES, FYS, Vicksburg, MS
ES, FYS, Daphne, AL
Game and Fish Commission, Little Rock, AR
Department of Wildlife & Fisheries, New Orleans, LA
Department of Wildlife Conservation, Jackson, MS
Division of Game and Fish, Montgomery, Al

ARKANSAS NATURAL HERR-AGE COMMISSION
THE HERITAGE CENTER, SUITE 200
225 EAST MARKHAM
LITTLE ROCK, ARKANSAS 72201
Phone: (501) 371-1706

Harold K. Grimmett
Director

Bill Clinton
Governor

Date: August 8, 1986
Subject: Disposal of chemical agents
ANHC Job #CF-78
Dated June 17 and July 10, 1986
Received June 23 and July 17, 1986

Dr. Lorene L. Sigal
Environmental Compliance and
Impact Analyses Group
Environmental Analyses Section
Environmental Sciences Division
Oak Ridge National Laboratory
Post Office Box X
Oak Ridge, Tennessee 37831

Dear Dr. Sigal:

Arkansas Natural Heritage Commission staff members have reviewed our files for designated natural areas and known occurrences of all the plant and animal species included in the categories that you specified in your letter of July 13, 1986. The information about the county distribution of designated natural areas was sent to you a few days ago. A state highway map showing some of the information that you required was sent earlier. All species occurrences fitting your criteria are presented in the accompanying computer printout. The occurrences are aggregated by county and then listed alphabetically by county under the headings of two major criteria. You will find much overlap in the data included under the two headings.

Please keep in mind that the area covered by this review may contain important natural features of which we are unaware. Likewise, some of the features reported to have occurred historically within the area may exist no longer. The quantity and quality of data collected by the Natural Heritage Inventory Program are dependent on the research and observations of many individuals and organizations. In most cases the information is not the result of comprehensive or site-specific field surveys. Many natural areas in Arkansas never have been surveyed thoroughly. for these reasons, the Natural Heritage Commission cannot provide a definitive statement on the presence, absence, or condition of biological elements in any part of Arkansas. Natural Heritage Inventory reports summarize the existing information known to the Program at the time of the request. They should never be regarded as final statements on the elements or areas under consideration, nor should they be substituted for on-site surveys required for environmental assessments, Because our files are updated constantly as additional data are received, you may want to check with us again at a later date.

The importance of the various features on which we keep records varies over a wide range. Some are habitats of animals on the federal list of endangered and threatened species. Some records represent the -best known examples of certain types of natural communities. Others locate habitats of plant and animal species that, though very rare in Arkansas, are common elsewhere. Please be advised that we can discuss with you in depth any record about which you need to know more for your project planning.

If the information presented here is used in any publication, please cite the Arkansas Natural Heritage Commission as the source.

Thank you for consulting us. It has been a pleasure to work with you on this study.

Sincerely,

Bill Shepherd

William M. Shepherd
(for Harold K. Grimmett, Executive Director)

Enclosed:
1) printout
2) information about critical habitat
3) legislation
4) invoice
5) fee schedule

Under separate cover at an earlier date: maps

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS INC

POST OFFICE BOX X
OAK RIDGE TENNESSEE 37831

Ray 21, 1986

Hr. Harvey K. Nelson, Regional Director
U.S. Fish and Wildlife Service
Twin Cities Regional Office
Federal Building, Fort Snelling
Twin Cities, Minnesota 55111

Dear Hr. Nelson:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munltlons, I am requesting Information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present in a corridor surroundlng a proposed route for the transport of chemical agents and munltlons. Enclosed are state maps and lists of counties of Interest. Please number the species and lndlcate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict Impacts for a variety of potential accident scenarios.

Please also indicate, by county, for the area wlthln the corridor:

(1) critical habitat for threatened and endangered species,

(2) major wildlife use areas for game and nongame species,

(3) fish management areas within the corridor,

(4) Important wetlands, and

(5) state-protected groundwater recharge areas.

We realize that this is a lot to ask and we want you to know that we appreciate your help. If there are questions, please call me at (615) 574-7266 (FTS 624-7266) or Virginia Tolbert (615) 574-7288 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Environmental Compliance and
Impact Analyses Group
Environmental Analyses Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

United States Department of the Interior

FISH AND WILDLIFE SERVICE

IN REPLY REFER TO:

ROCK ISLAND FIELD OFFICE (ES)
1830 Second Avenue, Second Floor
Rock Island, Illinois 61201

COM: (309) 793-5800
FTS: 386-5800

June 17, 1986

Hr. Lortnt L. Sigal, Ph.D.
Environmental Sciences Division
Oak Ridge National Laboratory
Post Office Box x
Oak Rldgt, Tennessee 37831

Attention : Virginia Talbot

Dear Dr. Slgal:

The following is our reply to your Hay 21, 1986 letter to our Regional Director which requested information nttdtd to prepare an environmental impact statement for the Department of the Army's plan to dispose of chemical agents and munitions. The information we art forwarding is for the states of Iowa and Illinois, which are the responsibility of this office.

Ye htvt prtptrtd atparatt packages for each state which includes the list of federally threatened and tndangtrad species, a map of the state with corridors marked, and a separate map marked per your Instructions. You indicated in our June 12, 1986 ttltcon that separate mailings were being sent to the individual states, therefore, we limited the additional information supplied to areas of ftdtral concern, i.e. fish and wildlife refuges, waterfowl resting awes.

We are pleased to cooperate in your data gathering efforts. If you have questions on the information enclosed, or desire additions? information, please contact me or Wayne Fischer of this office.

Sincerely,

Richard C. Nelson
Field Supervisor

Enclosures

cc: AH-TES

United States Department of the Interior

FISH AND WILDLIFE SERVICE

IN REPLY REFER TO:

BLOOMINGTON FIELD OFFICE (a)
718 North Walnut Street
Bloomington, Indiana 47401

July 11, 1986

Dr. Lorene L. Sigal
Environmental Sciences Division
Oak Ridge National Laboratory
Post Office Box X
Oak Ridge, Tennessee 37831

Dear Dr. Sigal:

This letter is in response to your May 21, 1986 request for information on endangered species, wildlife use areas, and wetlands for use in your preparation of a programmatic environmental impact statement (EIS) for the disposal of chemical agents and munition. We will be providing you information for the State of Indiana as our Regional Office has assigned individual field offices the responsibility for responding to requests such as yours. As you mentioned in your letter, the information sought is very extensive, therefore, we have limited our response for wildlife use areas and wetlands to major areas only. Every county in Indiana has sane areas that are important to sane form of fish or wildlife. If you need more specific information than we are providing, please contact us directly.

Your map and additional information indicate that, except for a few counties in extreme northeastern Indiana, virtually the entire state will be potentially affect&. In a telephone conversation between Don Steffeck of this office and Virginia Tolbert of your staff on July 8, 1986, it was agreed that tne information requested would be sent on state maps that include counties.

Federally endangered species that occur within Indiana are list& in Table 1. The occurrence of these species by county is presented in Figure 1. The Indiana bat (Myotis sodalis) is found statewide. This species winters in caves in southern Indiana and of these areas are Cesignated as critical habitat. During the spring and summer Indiana bats disperse for breeding am may be found statewide. The bald eagle (Haliaeetus leucocephalus) occurs in various counties during the winter months. There is an ongoing program in progress at Lake Monroe, Monroe County, where young eagles are being hacked (reared) to encourage breeding of this species in the state. Tnere are currently 4 mussels on the endangered species list for the State of Indiana. However, an additional 3 species may occur within the state but have not been collected in recent years. These additional species include the orange-footed pimpleback mussel (Plethobasis cooperianus), the rough pigtoe (Pleurobema plenum), and the white cats paw pearly mussel (Epioblasma obfiquata erobliqua). In addition, there are a number of plant and animal species currently under review for inclusion on the Indiana endangered species list.

Major wildlife and fish management areas are shown in Figure 2. This list includes mostly State areas, but does show Indiana's only National Wildlife Refuge. he Patoka River bottomlands in Pike county are currently under review

fcr inclusion into the National Wildlife Refuge system. Indiana Dunes National Lakeshore in Lake and Porter Counties is part of the National Park System, and is managed by the National Park Service. In addition, there are streams in Porter, LaPorte, and St. Joseph counties that are tributaries to Lake Michigan an4 are managed as migratory salmonid waters.

Your request for important wetlands is especially difficult for us to respond to. The great majority of Indiana's original wetlands have been drained for various reasons. Hence, existing wetlands have a relatively increased value because of their scarcity. We have indicated the major rivers, reservoirs, and lakes in Figure 3. However, smaller wetland areas of importance are too numberous to list seperately. The U. S. Fish and Wildlife Service and the Indiana Department of Natural Resources (IDNR) are currently involved in a joint project to inventory the states existing wetlands. The northern one-third of Indiana is canpleted, with the rest of Indiana due to be done within the next 3 years. If ycu have specific questions regarding wetlands at a certain location please contact this office. The completion of the wetland inventory maps will provide much more detailed information than we can efficiently do in this correspondence. Indiana has a number of waterways am associate? wetlands, hence the movement of chemical agents and munitions will occur in areas that contain important wetlands virtually throughout the state.

To our knowledge the State has not designated specific protected groundwater recharge areas. The State is currently reviewing and updating its groundwater protection policies, therefore, we recommend you get additional information from the IDNR or Indiana Department of Environmental Management.

We hone the enclosed information is helpful to you and look forward to reviewing the EIS and subsequent documents. If ycu have any questions regarding information contained this letter please contact me or Don Steffeck of my staff at 812-334-4261.

Sincerely yours,

David C. Hudak
Supervisor

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS, INC

POST OFFICE BOX X
OAK RIDGE, TENNESSEE 37831

May 21, 1986

Mr. Michael J. Spear, Regional Director
U.S. Fish and Wildlife Service
Albuquerque Regional Office
500 Gold Avenue, S.W., Room 9018
Albuquerque, New Mexico 87103

Dear Mr. Spear:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munitions, I am requesting information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present in a corridor surrounding a proposed route for the transport of chemical agents and munitions. Enclosed are state maps and lists of counties of Interest. Please number the species and indicate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict impacts for a variety of potential accident scenarios.

Please also Indicate. by county, for the area within the corridor:

(1) critical habitat for threatened and endangered species,

(2) major wildlife use areas for game and nongame species,

(3) fish management areas wlthin the corridor,

(4) important wetlands, and

(5) state-protected groundwater recharge areas.

We realize that this Is a lot to ask and we want you to know that we appreciate your help. If there are questions, please call me at (615) 574-7266 (FTS 624-7266) or Virginia Tolbert (615) 574-7288 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Environmental Compliance and
Impact Analyses Group
Envlronmental Analyses Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

UNITED STATES
DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
Ecological Services
222 S. Houston, Suite A
Tulsa, Oklahoma 74127

June 18, 1986

(2-14-86-f-60

Dr. Lorene L. Sigal
Environmental Sciences Division
Oak Ridge National L&oratory
Post Office Box X
Oak Ridge, Tennessee 37831

Dear Or. Sigal:

his is in reply to your letter dated May 21, 1986, requesting information regarding the corridor surrounding • proposed route for the transport of chemical agents and munitions. Although the proposed route for transportation of these materials does not extend into Oklahoma, 13 northeastern Oklahoma counties lie within a 100 kilometer distance and could potentially be effected by trenaportetion of chemical stockpiles. These 13 counties are Adair, Cherokee, Craig, Delaware, Kay, Mayes, Nowata, Osage, Ottawa, Rogers, Tulsa, Wagoner, snd Washington. In response to your request we compiled information concerning species federally listed or proposed to be listed as threatened or endangered, major fish and wildlife use areas, and other valuable habitats that occur in *the* 13 *county* area. We are unable to provide *information* regarding stattprotected groundwater recharge areas since they are not under our jurisdiction. Ye suggest you contact the Oklahoma State Water Resources Board for groundwater recharge locality information.

Within the project area that you described there are six federally listed species, five endangered and one threatened. Table 1 lists the six species and the counties in which they are known to occur. The endangered bald eagle (Haliaeetus leucocephalus) winters in Oklahoma along the state's major rivers and reservoirs and occasionally in rangeland areas. In early winter bald eagles mainly feed on fish but as the season progresses the diet switches to waterfowl. They prefer tree perches, over other perch types, that are located near feeding areas. Species authority for the bald eagle is Dr. James Lish, Zoology Department, Oklahoma State University, Stillwater, Oklahoma 74078. 405/624-5555.

The endangered gray bat (Myotis grisescens) is a year-round resident that hibernates in deep pit-type caves and typically forms maternity colonies in large caves containing streams. Gray bats forage on insects over rivers and reservoirs and adjacent riparian areas that occur within 2-S miles of their summer caves. Species authorities for the gray bat are Dr. Everett Crigsby, Division of Natural Science and Mathematics, Northeastern State University, Tahlequah, Oklahoma 74464, 918/456-5511 ext. 321, and William Puckette, Colcord, Oklahoma 74338, 918/326-4578.

Table 1. Federally listed threatened OK endangered species prerent in the counties which comprise the corridor of the proposed transportation route.

County	Species					
	Bald eagle	Gray bet	Indiana bet	Interior least tern	Ozark big-oared bet	Ozark cavefish
Adair		X	X		X	
Cherokee	X	X			X	
Delaware	X	X	X		X	X
Kay	X					
Mayes	X					X
Nowata	X					
Osage	X			X		
Ottawa	X	X				X
Rogers	X			X		
Tulsa	X			X		
Wagoner	X			X		
Washington	X					

Endangered Indians bat (Myotis sodalis) are rare summer migrants in Oklahoma. They roost in hollow trees, under bsrk, or under bridgss. The bats forage on insects near foliage of riparian and floodplain trees. Dr. Everett Crigsby and William Puckette are species authorities for this bat.

Interior least terns (Sterna ● ntillarum athalassos) nest along major rivers, barren shores of large reservoirs, and on the barren flats of the Salt Plains National Wildlife Refuge in Oklahoma. The presence of bare or nearly bare ● lluvisl islands or sandbars, the existence of favorable water levels during nesting season and the availability of a food source such as small minnows influence the distribution of this species. Laura Hill of this office is ● species authority.

Ozark big-eared bats (Plecotus tovnsendii ingensl are yesr-round residents in Oklahoma. The bats winter in caves with SO-90 percent relative humidity and temperatures of 4-9 °C and summer in warmer and more humid caves. Ozark big-eared bats forage in forested areas near lakes or rivers. Forested corridors are important travel routes for the bat. The Fish and Wildlife Service recently has obtained fee title or easement to four areas surrounding Ozark big-eared bat caves in Adair and Delaware Counties. Management goals are to minimize disturbance in the caves and to protect nearby foraging habitat. Species authorities are Dr. Everett Crigsby and William Puckette.

The threatened Ozark cavefish (Amblyopsis rosae) is a true troglobitic cavefish that is endemic to northeast Oklahoma, northwest Arkansas, and southwest Missouri. The species mainly preys on small crustaceans but also will eat larval salamanders. Species authority for the cavefish is Dr. Arthur Brown, Zoology Department, University of Arkansas, Fayetteville, Arkansas 72701, 501/575-3251.

A number of important fish and wildlife areas occur in the 13 county area of influence. Table 2 lists the public lands managed by the Oklahoma Department of Wildlife Conservation (ODWC) and Table 3 lists reservoirs and high qulaity streams that lie in the area potentisllly affected by the transport route. In addition to these existing areas there is a proposed Tallgrass Prairie Preserve north of Pawhuska, Osage County that would include about 50,000 acres of tallgrass prairie.

Bottomland hardwood forest and other wetlands are well distributed throughout the 13 county ares. The Frsh and Wildlife Service and the ODWC have recently completed a report on the bottomland hardwoods of eastern Oklahoma 1/. They Identified three forested areas which occur in northeast Oklahoma as having exceptional ecological and management potential. These tracts

---

1/ Brabander, J.J., R.E. Masters, and R.M. Short. Bottomland hardwoods of eastern Oklahoma, a special study of their status, trends, and values. U.S. Fish and Wildlife Service, Tulsa, Oklahoma and Oklahoma Dept. of Wildlife Conservation, Oklahoma City, Oklahoma. 148 pp.

Table 2. Wildlife management drcds (WMA) operated by the Oklahoma Department of Wildlife Conservation.

WMA	County	Size (Acres)	Important species
Chou teau	*Wagoner*	*2,197*	*Waterfowl, deer,* squirrel, furbearers, dove, rabbit
Cookson Hills	Cherokee, Adair	13,640	Deer, squirrel, elk, turkey
Copan	Wdahinqton	7,500	Waterfowl, deer, rabbit, prairie chicken, squirrel, furbearers
Fort Gibson	Wagoner, Cherokee	21,790	Waterfowl, dove, deer, quail, turkey, rabbit, squirrel
Huldh	Osage	14,112	Waterfowl, deer, quail, rabbit, dove, squirrel, furbearers, turkey
Oologah	Nowata	12,941	Waterfowl, rabbit, turkey, deer, squirrel, dove, furbearers
Osage	Osage	320	Prairie chicken, dove, quail
Spavinaw Hills	Delaware	13,740	Deer, turkey, rabbit, squirrel, furbedrcrs
Tenkiller	Cherokee	1,950	Quail, deer, squirrel, rabbit, furbearers

Table 3. Reservoirs and high quality streams in the project area.

Lakes	county	Streams	County
Birch	Osage	Baron Fork	Adair/Cherokee
Blues tem	Osage	Caney Creek	Washington/Rogers
Copan	Washington	Flint Creek	Adair
Eucha	Delaware	Fourteenmi le Creek	Cherokee
Fort Gibson	Wagoner/Cherokee/Mayes	Illinois River	Adair/Cherokee
Grand	Mayes/Delaware/Ottawa	Neosho/Grand River	Ottawa
Hulah	Osage	Snake Creek	Mayes/Cherokee
Kaw	Osage	Spavinaw Creek	Delaware
Keys tone	Osage/Tulsa	Spring Creek	Hayes/Cherokee
Lake Hudson	Mayes		
Oologah	Rogers/Nowata		
Skiatook	Osage		
Spa vi naw	Mayes/Delaware		
Tenki ller Ferry	Cherokee		

are: 1) 2,990 acres on the Grand River vest of Commerce, Ottawa County; 2) 2,122 acres on the Verdigris River east of Verdigris, Rogers County! and 3) 666 acres on Caney Creek south of the confluence with Hogshooter Creek, Washington County. Two additional smaller tracts of bottomland forest, one on the ⊜⋊♦♦●ℳ Caney Creek ● amt of Dewey, Washington County and another on the Verdigris River above Oologah Lake, Nowata County, occur in the area potentially affected by the route. Emergent, open-water end other wetland types are ● rrcciated with all these forerted tracts.

If there are my further questions please call this office et FTS:745-7458.

Sincerely yours,

Sidney H. Wilkirson
Field Supervisor

cc:
Regional Director, FWS, Albuquerque, New Mexico (AHR)
Director, ODWC, Oklahoma City, Oklahoma

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS INC

POST OFFICE BOX X
OAK RIDGE TENNESSEE 37831

May 21, 1986

Mr. Galen L. Buterbaugh, Regional Director
U.S. Fish and Wildlife Service
Denver Regional Office
134 Unlon Boulevard
P.O. Box 25486
Denver Federal Center
Denver, Colorado 80225

Dear Mr. Dutetbaugh:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munitions, I am requesting information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present In a corridor surroundlng a proposed route for the transport of chemical agents and munitions. Enclosed are state maps and lists of counties of interest. Please number the species and indicate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict impacts for a variety of potential accident scenarios.

Please also Indicate. by county, for the area within the corridor:

(1) critical habitat for threatened and endangered species,

(2) major wildlife use areas for game and nongame species,

(3) fish management areas within the corridor,

(4) Important wetlands, and

(5) state-protected groundwater recharge areas.

We realize that this Is a lot to ask and we want you to know that we appreciate your help. If there art questions, please call me at (615) 574-7266 (FTS 624-7266) or Virginia Tolbert (615) 574-7268 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Environmental Compliance and
Impact Analyses Group
Environmental Analysts Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

United States Department of the Interior
FISH AND WILDLIFE SERVICE

MAILING ADDRESS
Post Office Box 25486
Denver Federal Center
Denver, Colorado 80225

STREET LOCATION:
134 Union Blvd
Lakewood, Colorado 80228

IN REPLY REFER TO:
HR
MAIL STOP 60120

JUN 10 1986

Dr. Lorene L. Sigal
Environmental Sciences Division
Oak Ridge National Laboratory
P.O. Box X
Oak Ridge, Tennessee 37831

Dear Or. Sigal:

We have directed our Ecologlcal Services field offices to respond directly to you with information concerning questions 2 through 4 in your letter of May 21, 1986, to Regional Director Buterbaugh. Collectively, the responses from these field offices should provide useful information on each of the eight States (Colorado, Kansas, Montana, North Dakota, Nebraska, South Dakota, Utah, and Wyoming) in our Region. The State water resource agencies would be a better source of information for question 5 than would the Fish and Wildlife Service.

The Regional Endangered Species Office will be responding to you separately regarding question 1 and the occurrence of threatened and endangered species along your designated route.

We are pleased to be of assistance and if you have questions concerning any of our responses you may contact the field offices concerned or Joe Ellis (3031236-8180) of my staff.

Sincerely,

William E. Martin
Assistant Regional Director,
Habitat Resources

cc: Endangered Species
FWS, Regional Office
ES Field Offices, FWS
Salt Lake City, UT
Billings, MT
Grand Island, NE
ES Field Suboffices, FWS
Manhattan, KS
Cheyenne, WY
ES Field Substation, FWS
Grand Junction, CO

United States Department of the **Interior**
FISH AND WILDLIFE SERVICE

MAILING ADDRESS
Post Office Box 25486
Denver Federal Center
Denver, Colorado 80225

STREET LOCATION
134 Union Blvd.
Lakewood, Colorado 80228

IN REPLY REFER TO
FA/SE/Army-
Informal
Mail Stop 60153

JUL 8 1986

Dr. Lorcnc L. **Sigal**
Environmental **Sciences Division**
Oak Ridge National Laboratory
Post Office Box X
Oak Ridge, Tennessee 37831

Dear Dr. Sigal:

This responds to your May 21, **1986,** letter requesting various information, **including** that on endangered and thrtrttntd species for preparation of the Department of Army **programmatic** environmental Impact statement for the disposal of chemical agents and **munitions.** This **letter** and **the** attachments pertain to proposed and listed threatened and endangered species and their critical habitat. The information you requested on **the** other items will be handled by separate responses.

Region 6 has the following crltical **habitat** for threatened and **endangered** species that may occur within the corridor:

Colorado

Clay-loving wild-buckwheat (Erlogonum pelinophilum)
Delta County.

Kansas

Whooping crane (Grus **americana)**
Quivira National Wildlife Refuge - Stafford, Reno, and Rice Counties
Cheyenne Bottoms Yaterfowl Management Area - Barton County

Nebraska

Whooping crane **(Grus americana)**
Dawson **County**
Buffalo County
Hall County
Phelps County
Kearny County
Adams County

Utah

Listed Species
June sucker (Chasmistes liorus)
Utah County

Proposed Species
Heliotrope milk-vetch (Astragalus limnocharis var. montii)
Sanpete County

Toad-flax cress (Glaucocarpum suffrutacens)
Uintah County

Wyoming
No designated critical habitat for endangered or threatened species in Wyoming.

Attached are maps with the distribution of species for the States of Kansas and Nebraska. Endangered species that occur or may occur within the Yyoming portion include:

1. Black-footed *ferret* (Mustela nigripes)
2. Bald eagle (Haliaeetus leucocephalus)
3. Peregrine falcon (Falco peregrinus)
4. Yhooping crane (GNS americana)
5. Wyoming toad (Bufo hemiophrys baxteri)

Species 1-3 *may occur in* all *Wyoming counties indicated* on the *attached* Wyoming *map*. We have put the appropriate numbers in the counties where the whooping crane (4) and the Uyaning toad (5) nay occur. The whooping crane can be expected to occur in Lincoln, Uinta, Sublette, Hot Springs, and Sweetwater Counties. The Wyoming toad occurs or may occur in Albany County.

Tnere are too many species listed in Colorado and Utah to place numbers in the counties. Ye have therefore numbered the species for each county in Colorado and prepared a chart which includes each county in the proposed corridor in Utah.

If you have any questions, please contact us.

Sincerely yours,

/s/ John L. Spinks Jr.
Acting Regional Director

Attachments

OAK RIDGE NATIONAL LABORATORY
OPERATED BY MARTIN MARIETTA ENERGY SYSTEMS, INC

POST OFFICE BOX X
OAK RIDGE, TENNESSEE 37831

May 21, 1986

Mr. Richard J. Myshak, Regional Director
U.S. Fish and Wildlife Service
Portland Regional Office
500 N.E. Multnomah Street, Suite 1692
Portland, Oregon 97232

Dear Mr. Myshak:

In order to prepare the terrestrial and aquatic ecology sections of the Department of Army programmatic environmental impact statement for the disposal of chemical agents and munitions, I am requesting information about terrestrial and aquatic species of plants and animals listed or proposed to be listed as endangered, threatened, or of special concern which may be present In a corridor surrounding a proposed route for the transport of chemical agents and munitions. Enclosed are state maps and lists of counties of interest. Please number the species and indicate their distribution by putting their numbers In the appropriate counties on the maps. This will allow us to predict impacts for a variety of potential accident scenarios.

Please also indicate, by county, for the area within the corridor

(1) critical habltat for threatened and endangered species,

(2) major wildlife use areas for game and nongame species,

(3) flsh management areas within the corridor,

(4) important wetlands, and

(5) state-protected groundwater recharge areas.

We realize that this is a lot to ask and we want you to know that we appreciate your help. If there are questions, please call me at (615) 574-7266 (FTS 624-7266) or Virginia Tolbert (615) 574-7288 (FTS 624-7288).

Sincerely,

Lorene L. Sigal

Lorene L. Sigal, Ph.D.
Envlronmental Compliance and
Impact Analyses Group
Envlronmental Analyses Section
ENVIRONMENTAL SCIENCES DIVISION

LLS:lkm

Enclosures

United States Department of the Interior

FISH AND **WILDLIFE** SERVICE

Portland Field Office
727 N. E. 24th Avenue
Portland, Oregon 97232

Re: 1-7-86-SP-151

July 9, 1986

Oak Ridge National Laboratory
P. O. Box X
Oak Ridge, Tennessee 37831

Dear Sir:

As requested by your letter, dated May 21, 1986 and received by us on June 10, 1986, we have attached a list of endangered and threatened species that may be present within the area of the proposed chemical transport through Nevada, California, Oregon, Washington, and Idaho. The list fulfills the requirement of the Fish and Wildlife Service under Section 7(c) of the Endangered Species Act of 1973, as amended. Your requirements under the Act are outlined in Attachment B.

Should your biological assessment determine that a listed species is likely to be affected (adversely or beneficially) by the project, the Department of the Army should request formal Section 7 consultation through this office. Even if your biological assessment shows a "no effect" situation, we would appreciate receiving a copy for our Information.

We have also included a list of candidate species presently under review by this Service for consideration as endangered or threatened. It should be noted that candidate species have no protection under the Endangered Species Act. Should you determine your project may affect candidate species, you are not required to perform a biological assessment or to consult with the Fish and Wildlife Service. Candidates are included simply as advance notice to Federal agencies of species which may be proposed and listed in the future. If early evaluation of your project indicates that it is likely to adversely impact a candidate species, the Oepartment of the Army may wish to request technical assistance from this office.

Your interest in endangered species is appreciated. If you have any additional questions regarding your responsibilities under the Act, please contact Diana Hwang at our office, phone (503) 231-6179 or FTS 429-6179. All correspondence should include the above referenced case number

Sincerely yours,

Attachments

Russell 0. Peterson
Field Supervisor

cc:
RO (AFA/SE)
PFO (ES
BFO (SE)
ODFW (Nongame)
ONHP
SESO
GBC-Nevada
OFO

J. Leonard Ledbetter
COMMISSIONER
Leon Kirkland
DIVISION DIRECTOR

Department of Natural Resources
Game and Fish Division
Non-Game Endangered Species Program
Route 2 Box 119
Social Circle Georgia 30279
(404) 557.2532

August 6, 1986

Dr. Lotene L. Sigal
Oak Ridge National Laboratory
P.O. Box X
Oak Ridge, TEnnessee 37831

Dear Dr. Sigal:

Enclosed is the map you sent with endangered species use indicated. i have also enclosed a copy of Georgia's Protected Wildlife for your reference. The additional information you requested can best be obtained by contacting:

Chuck Rabolli
Endangered Plant Program
Department of Natural Resources
Suite 1362-East Tower
205 Butler Street S.E.
Atlanta, CA 30334

Gike Gennings
Dept. of Natural Resources
Fisheries Section
Suite 1350-East Tower
205 Butler Street, S.E.
Atlanta, GA 30334

Harold F. Reheis
Environmental Protection Division
Suite 1152-East Tower
205 Butler St., S.E.
Atlanta, GA 30334

Please feel free to contact us for further information.

Sincerely,

Ron R. Odom
Senior Wildlife Biologist

RRO/rd

Enclosures

Appendix G

TRANSPORTATION CONCEPTS

G.1 INTRODUCTION

In planning for the disposal of the chemical munitions stockpile, the Army is considering moving the munitions to one national disposal center (NDC) or two regional disposal centers (RDCs). Operation of RDCs or an NDC requires transport of large segments of the lethal chemical munitions stockpile on a scale unprecedented since the munitions were originally deployed to eight continental United States (CONUS) sites and overseas (OCONUS). In the deployment phase, the munitions were moved within CONUS by truck and rail over periods of many years. In the proposed disposal program, the entire stockpile must be moved in a period of three years or less.

All but two of the disposal alternatives (Continued Storage and On-site Disposal) now under study by the Army for its Chemical Stockpile Disposal Program require off-site transport of chemical agents and munitions. All alternatives require transport of the munitions within the boundaries of the eight installations. The principal purpose of this Appendix is to address the nature of both kinds of transport.

G.2 PREVIOUS LETHAL CHEMICAL MATERIAL TRANSPORT

Most of the lethal chemical munitions currently in the national stockpile were transported from manufacturing locations or other storage locations to their current storage site. Most of these movements were by truck, ship, or rail, Some munitions were moved by aircraft. Movement of items currently in the inventory began as early as 1942 and continued into the late 1960s. In addition, there were movements made for overseas deployment and return during and following Word War II, movements involving captured German and Japanese munitions, movements involving test or experimental munitions, and movements to ports for sea disposal. These early moves were usually conducted without incident, but during the period of the 1940s and 1950s, when requirements were less stringent, some chemical leaks and spills occurred. On several occasions in the late 1940s, some military and civilian personnel were injured in moves involving captured German mustard munitions.

Public Law 91-121 was passed in 1969 and amended by Public Law 91-441 in 1970. These laws limit the open-air testing, disposal, and movement of chemical weapons. They also mandate the stringent review of plans for any proposed movement. Some moves that occurred during the period since 1969 are described below. A detailed compilation for shipments dating from 1946 can be found in the Chemical *Weapons Movement History* Compilation (U.S. Army 1987b).

1. Operation CHASE X. Obsolete M55 rockets were encased in solid concrete coffins and shipped by rail from Lexington-Blue Grass Army Depot and Anniston Army Depot to the Military Ocean Terminal at Sunny Point, North Carolina. This move was conducted in August 1970 without incident. The munitions were disposed of at sea in deep water without incident.
2. Operation *Red* Hat. The entire contents of Chibana Army Depot in Okinawa was loaded onto six ships and moved to Johnston Island in the Pacific Ocean. Mustard munitions were moved in January 1971; the remaining items followed in August and September 1971. During the

loading of one vessel, a pallet of 15 M55 rockets was accidentally dropped from a crane approximately 40 ft into the hold of the vessel. Although subsequent examination showed that some of the rockets were severely damaged, no significant leak or spill occurred and there was no harm to operators or the general public.

3. Operation FMA. A small number of mustard-filled ton containers? HD projectiles, and GB projectiles were moved about 20 road miles by truck convoy, removing all chemical munitions remaining at Fort McClellan, Alabama to Anniston Army Depot, Alabama. This operation was conducted in December 1976 without incident.
4. Operation TNS. Mustard-filled mortar cartridges were transported by rail approximately 25 miles form the North Area to the South Area of Tooele Army Depot. This operation was conducted in August 1977. Prior to Army inspection and prior to arrival in the North Area, one of two engines scheduled to pull the munitlons train was involved in a collision when the brakes failed, allowing it to roll into another train. This engine was replaced, and the actual movement of munitions proceeded without incident.
5. Operation DTS. A variety of GB, VX, and phosgene munitions was moved by truck convoy from Dugway Proving Ground, Utah, to the South Area of Tooele Army Depot, Utah (approximately 50 miles). This operation was conducted in August 1977 without incident.
6. Operation SETCON 1. This operation Involved the movement of about 10% of the inventory of Chemical Agent Identification Sets containing actual agents formerly used In chemical warfare training from several locations to Rocky Mountain Arsenal for incineration. The move was conducted by air and truck convoy in January 1978 without incident.
7. Operation SETCON II. This operation involved the movement of the remaining Chemical Agent Identification Sets to Rocky Mountain Arsenal for disposal. The move was conducted in May and June 1980 using air and truck convoys. During the movement operation, a helicopter transferring sets from Crane Army Ammunition Activity, Indiana, to Fort Campbell Army Airfield experienced engine failure and crashed. No agent was spilled. There were no other incidents.
8. Operation RMT. The stock of GB-filled Weteye bombs and three l-ton containers of GB at Rocky Mountain Arsenal, Colorado, were moved to Dugway Proving Ground, Utah, by military aircraft and, subsequently, to the South Area of Tooele Army Depot by truck convoy. This operation was conducted In July 1981 without incident.

G.3 REGULATIONS GOVERNING TRANSPORTATION

Transportation of chemical agents and munitions is governed by regulations of the U.S. Department of Defense (DOD) and other federal agencies. Federal agencies with oversight and/or enforcement roles in the transportation program include the U.S. Department of Transportation (DOT), the U.S. Department of Health and Human Services (DHHS), and the U.S. Environmental Protection Agency (EPA). In addition, states affected by transportation may have regulatory jurisdiction over certain aspects of the transportation program. These states to be affected, and any relevant regulations, would be determined If and after a decision to transport is made and routes are selected.

G.3.1 DEPARTMENT OF TRANSPORTATION

The Hazardous Materials Transportation Act of 1974 (Public Law 93-633) seeks to "protect the nation adequately against the risks to life and property inherent in the transportation of hazardous materials in commerce." Under this act, the DOT formulates regulations to ensure safety in transit. These regulations cover packaging, marking, loading, and handling of materials in transit and the precautions necessary to determine whether materials to be shipped are in proper condition for transport.

Regulations of the DOT (49 CFR Pts. 146, 171-178, 297, and 379) govern truck and rail transport of hazardous materials. The DOD Explosives Safety Board assigns proper shipping names prior to shipment and also assigns the DOD Hazard Class/Division, DOT marking and label, and DOD compatibility. The hazard class is a numerical designator of explosiveness (1) or toxicity (6) of the material. The hazard class or division is a numerical designator assigned to denote the character and the predominance of the associated hazards or property damages. Within class 1 (explosives), four divisions indicate the type of hazard expected.

Hazard Class/Division	Hazards
1.1	Mass detonating
1.2	Nonmass detonating fragment producing
1.3	Mass fire
1.4	Moderate fire--no blast

For hazard class 6, only one division is utilized for ammunition (division 1), which denotes poisonous (toxic) substances:

Once the proper shipping name is obtained, the hazardous materials table in 49 CFR Part 172.101 may be used to determine applicable packaging requirements and transportation mode limitations.

According to 49 CFR 173.59, the requirements for transporting explosive chemical munitions are the same as for Class A explosives as described in Part 173.56. These items can be shipped by and for the DOD and in accordance with established practices and procedures specified by the DOD. Nonexplosive chemical munitions may be shipped only by, for, or to the DOD. The packaging, marking, and labeling is as required by DOD regulations.

G.3.2 DEPARTMENT OF DEFENSE

Public Law 91-121 requires the following actions be completed prior to the transportation of chemical munitions between military installations:

1. The Secretary of Defense has determined that the proposed transportation is necessary and in the interest of national security.

2. The Secretary of Defense has brought the particulars of the proposed move (disposal or transportation) to the attention of the Secretary of DHHS.

3. The Secretary of Defense has implemented any precautionary measures recommended by the Secretary of DHHS.

4. The Secretary of Defense has provided notification of the move to the Congress of. the United States and the governors of the states affected by the move.

In addition, the Defense Appropriations Act of 1986 (Public Law 99-145) requires coordination with the DHHS, EPA, and DOD Explosives Safety Board.

Army Regulation 50-6 implements the Chemical Surety Program, and includes safety guidelines, chemical surety duty positions, transportation policies, and DOD physical security requirements for chemical surety material. Section 3.0 of this regulation delineates requirements for personnel selection and tracking for chemical surety duty positions. Section 4.0 states policies for transport of chemical surety material including: "safety procedures for movement within the continental United States will provide for a level of protection equal to or greater than that required by the DOT." Other policies, administrative and operational procedures, and requirements may be found in Sect. 4.0. Section 5.0 addresses the Army's Chemical Accident and Incident Response and Assistance.

System Safety Program Requirements for the DOD are contained in MIL-STD-882B:

> This standard provides uniform requirements for developing and implementing a system safety program of sufficient comprehensiveness to identify the hazards of a system and to impose design requirements and management controls to prevent mishaps by eliminating hazards or reducing the associated risk to a level acceptable to the managing activity.

Implementation of selected portions of this standard could be used to comprehensively address the safety requirements in the transport of the chemical stockpile program. For example, hazard analyses, human factors considerations, and system safety program plan guidelines should be integrated into the plan, if transportation alternatives are implemented.

C.3.3 DEPARTMENT OF HEALTH AND HUMAN SERVICES

Pursuant to Public Law 91-121 as amended by PL 91-441, the Secretary of DHHS may direct the Suregon General of the Public Health Service and other qualified people to review the particulars of any proposed chemical agent transportation, testing, or disposal and to recommend necessary precautionary measures. Although the Secretary typically establishes such committees, this has not always been done. The committee has approved the basic concept plan for demilitarization but does not have power to approve specific procedures and equipment. Historically, the Army has complied with DHHS recommendations.

C.3.4 COUNCIL ON ENVIRONMENTAL QUALITY

In November 1980, the Army made effective a new policy and guidance for considering environmental effects in the Army decision-making process (32 CFT, Pts. 650-651). This policy is responsive to a Council

on Environmental Quality Regulation to implement the National Environmental Policy Act. Therefore, an environmental impact statement is required for proposed actions that involve the production, storage, transportation, use and disposal of hazardous or toxic materials that have potential to cause significant environmental effects.

G.3.5 ENVIRONMENTAL PROTECTION AGENCY (EPA)

The Army has declared that the M55 rockets are obsolete. In discussions with EPA, the rockets were classified as hazardous wastes subject to management regulations promulgated under the Resource Conservation and Recovery Act (RCRA). Thus, transportation of M55 rockets will be conducted in compliance with all applicable RCRA regulations, whether the program is administered on a state or federal level.

G.4 EXPERT PANEL ON TRANSPORTATION METHODS AND PROCEDURES

The Transportation *of* Chemical *Agents and Munitions*: *A Concept Plan (U.S.* Army 1987c), as summarized herein, is a conceptual plan for on-site and off-site movement of munitions. The transportation concept plan conforms *to* the guidance provided to the Army by a panel of nongovernment experts, assembled to advise the Army on transportation methods and procedures.

To assist in an objective determination of appropriate transportation methods, the Army, through the MITRE Corporation, assembled a panel of experts from outside the Federal government who are knowledgeable about transportation methods and operations, transportation of hazardous material, and transportation safety. The charge to the panel was to develop system requirements and criteria for the safe transportation of chemical munitions, both within the boundaries of the installations at which the munitions are stored, and in off-site transportation by railroad, aircraft, and waterway. The panel was not asked to make recommendations regarding which transport mode is preferable. The members of the panel and their areas of expertise are listed in Table G.l.

G.5 GUIDANCE OF THE TRANSPORTATION PANEL

The panel developed the generic criteria that the major elements of the overall transportation system must satisfy to ensure safety. Three distinct transportation elements were identified and addressed: packaging; cargo handling operations at the sites; and transportation mode operational procedures (MITRE 1987b). Also identified and addressed were program management functions crucial to the safety of the transport operations.

G.5.1 BASIC CONSIDERATIONS

In developing the criteria for the transportation system, the panel was motivated by a number of basic considerations.

First, and foremost, is recognition that the selection of a disposal option, which requires transportation, would initiate a program with a highly specialized and singular mission. Because of the toxicity of the agents and the potential scale of the operations, the mission goes

Table G.1. Members of the expert panel oa transportation concepts for chemical munitions

Panelist and title	Area of expertise
Michael S. Bronzini Professor and Head Department of Civil Engineering Pennsylvania State University	Freight transportation with emphasis on marine transportation
Charles E. Dettmann Vice President, Transportation Union Pacific Railroad	Rail transportation of hazardous materials
Charles 0. Miller President System Safety, Inc.	System safety with emphasis in aviation and human factors
Bruce J. Williams Manager, Land Transportation Dow Chemical Company	Shipping of hazardous materials

beyond the routine transportation of the most hazardous of materials in commerce. Therefore, the mission requires special attention and treatment. A particularly strong safety emphasis in the overall program management is necessary.

Second, for munitions having both chemical agent and explosives, the toxic hazard and the explosive hazard should be physically separated prior to shipment, whenever feasible.

Third, redundancy in systems to protect against release of agent into the atmosphere during normal transport is necessary.

Fourth, the munitions should be packed into a transportation container at the origin site as early in the transport process as possible. *Once* in a container, subsequent direct handling of the munitions would be eliminated and handling mishaps minimized.

Fifth, packaging is the most crucial component in the entire transportation system. It provides containment of the hazard; it affects the extent and the manner of the handling of the munitions; and it has a significant influence on the choice of equipment used in transport. Packaging should be considered in a system context, taking into account the munition type, the bracing and **dunnage**, overpacking, human factors, and the vehicles.

Finally, recognition should be given to the obvious--as is true with almost any human activity, it is not possible to guarantee that chemical munition transportation can take place with zero risk.

G.5.2 PACKAGING CRITERIA FOR OFF-SITE TRANSPORTATION

The panel believes packaging to be the crucial component in the entire transportation system. Packaging, here, is meant in the system sense: the outermost transport container; **dunnage** inside; inner containers, if any; and possibly the munition casings themselves, The most important role for the packaging system is the containment of the agent, both during normal transportation and during an accident. In an accident, the package provides the impact and thermal protection necessary to prevent agent release.

Packaging also plays a significant role in the way the cargo is handled throughout the transport process. Containerization early in the transportation sequence minimizes handling of the munitions themselves and reduces the potential for handling accidents. If the container is standardized, it enables intermodality, such as transferring a container from a truck to a train at the loading area or removing a container from the scene of a derailment by truck.

Finally, a packaging system with demonstrable accident survivability enhances public confidence in the safety of the transport operations. The panel recommends that the transportation package be evaluated against either a series of accident scenarios developed in Sect. 2.2 of the *Transportation of Chemical Agents and Munitions: A Concept Plan* (1987c) or a series of tests developed by the Nuclear Regulatory Commission (10 CFR 71.73). The scenarios portray severe transportation accidents that have occurred in hazardous materials transportation experience. Prevention of agent release under these scenarios would provide a high degree of assurance that the packaging system would survive virtually all credible accidents. The tests developed for nuclear materials define a hypothetical accident. A series of drop, puncture, and fire and water immersion tests are used as surrogates for

severe transportation accidents. The packaging system concept should be designed to prevent agent release under either the type of accident described in the panel's scenarios (see above) or the Nuclear Regulatory Commission's test requirements, whichever is more stringent.

Motivated by the foregoing considerations, the panel arrived at the following four packaging system criteria:

1. The system should provide redundant protection against agent release during normal transport.
2. The system should prevent agent release into the environment in the event of an accident. For munitions with any explosives and/or propellants, the risk from release of agent due to detonation is expected to be small compared with other accident scenarios.
3. The transportation container should be compatible with standard commercial handling and carrier equipment. This eliminates the need for specialized handling procedures and equipment and enables the use of current equipment technology.
4. The transportation containers should be equipped with automated agent and temperature monitors and alarms. This enables the timely detection of possible leaks within the container.

G.5.3 TRANSPORT MODES

The panel made recommendations on transport operations by the rail, air, and marine modes. They pertain to operational elements such as preparation for transport, equipment, routing, and inspections. For pre-shipment preparation, the munitions should be placed in the transport container as early in the transport sequence as possible. For rail transport, equipment should be of current technology, routes should be chosen principally on the basis of track quality, and speeds be 10 miles per hour less than the designated limit, but never more than 50 miles per hour. For air transport, the panel recommended the airfield should be at least 200 ft wide and permit a straight-in approach of at least 3 miles.

G.6 PACKAGING SYSTEM CONCEPT

Packaging is the crucial component in the entire transportation system. It provides protection against agent release, affects the manner of handling the agents and munitions, and Influences the choice of equipment used in transport.

Chemical munitions of various types, sizes, and quantities may be transported by rail or air from storage sites to destruction facilities. The fundamental transportation criterion recommended by the independent panel of experts to be met under all modes of transport is that there should be no release of chemical agent into the environment during normal conditions of transport or as the result of an accident. The panel's principles developed to achieve the primary objective are summarized below.

. Redundant protection against leakage of chemical agents to the environment shall be inherent in the basic equipment design of handling and shipping containers.

- Containers must have automatic monitoring for leakage of chemical agent and/or test points for sampling air within the container.

- Shipping containers must be compatible with existing cargo handing and transport systems.

- Handling of the munitions shall be minimized,

Accident conditions that the munitions shipping containers must be able to withstand include dynamic impact, static load, fire, and immersion in water.

The transportation criteria noted above, along with the anticipated accident conditions, are the baseline for development of the conceptual design of the chemical munitions transport system. The resultant conceptual design used a combination of several materials and geometric configurations to maximize protection against mishaps in the operating environment and to ensure the safety of the public and transport personnel.

The basic off-site container concept consists of a double-walled cylindrical inner container surrounded by thermal insulation and supported on shock-isolating springs within an outer rectangular support frame and shell. The outer frame conforms to International Organization for Standardization (ISO) requirements with outside dimensions of 8 ft 6 in. high, 8 ft wide, and 20 ft long. Munitions *on* their original pallets are placed on special pallets for efficient packing into the inner cylindrical shipping container. They are restrained from shifting within the container by a tiedown system and expendable inflatable dunnage. Figure G.1 is a schematic drawing of the packaging system concept. Beyond the panel's recommendations for off-site transportation packages or containers, the Army has committed to the use of a package for shipments associated with the on-site disposal alternative.

The Army commissioned a transportation package survey by the MITRE Corporation. The resulting Analysis *of* Existing Hazardous Materials Containers *for Transporting Chemical* Nunitions (MITRE 1987a) identifies as many of the implemented packaging strategies for safe shipment of the most hazardous cargos as possible, emphasizing those that may be adapted with the fewest alterations to chemical munition shipment. The types of requirements envisioned for munitions package systems already exist as performance requirements in regulations covering hazardous chemical containers and radioactive material containers. Requirements for impact, static load, puncture, deceleration, fire, and water immersion have all been applied to existing containers. Furthermore, experiments on existing containers demonstrate that certain specific features of several package types meet or come near to meeting the proposed chemical munitions package requirements.

Some promising features are specific to individual packages and some are incorporated virtually universally *to* meet common performance requirements. The major applicable design features are listed below.

1. Cylindrical geometry. Cylindrical geometry is common to nearly all containers designed to meet extreme internal and/or external pressure criteria. Spherical geometry is used for a few designs; rectangular geometry is used as the primary containment barrier only where extreme pressures are not encountered.

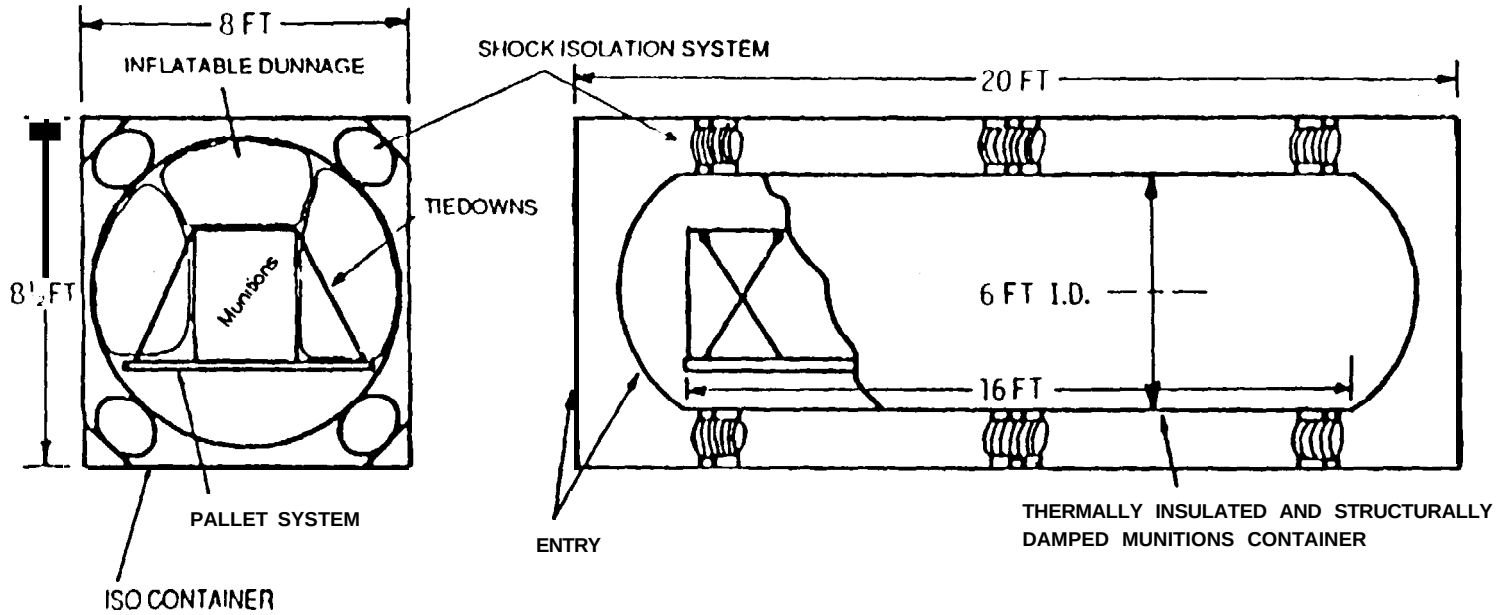


Fig. G.1. Schematic diagram of the off-site transportation container for the packaging and shipping of chemical munitions/agents.

2. Double containment. Multiple barriers to the release of contents is common to containers for the most hazardous material. Double containment is a regulatory requirement for plutonium packages (over 20 Ci) and is used in practice for most high-radiation material packages.
3. Thermal insulation and thermal **mass.** Designs for thermal protection vary. Thin-walled bulk chemical containers such as U.S. rail car tanks and IS0 intermodal tanks have additional thermal insulation surrounding the primary container. In contrast, nuclear spent-fuel casks rely on thick, massive walls to provide high thermal mass as a means of insulation. This thermal protection strategy arises naturally from the concurrent radiation protection requirement, which calls for massive shielding.
4. Overpacks. Many package designs include various types of overpacks (i.e., outer packages placed around the primary container). Overpacks typically provide primary protection against different hazards than does the protection provided by the primary container. For example, TRUPACT (Transuranic Package Transporter), an insulated rectangular box using composite material construction, provides structural strength, and impact, puncture, and thermal resistance for inner containers for certain nuclear waste. The containers loaded into TRUPACT provide the principal radiation protection and the primary barrier to release of contents.
5. Structural frame. Large bulk chemical tanks, such as the ISO intermodal tanks, incorporate structural support frames as an integral part of the packaging system. Like overpacks, frames serve different safety functions than the primary containers.

Structural frames provide the main protection against deceleration, impact, and crushing events (but not puncture) for thin-walled bulk chemical containers.

In conclusion, although it appears that no one existing container or packaging system is entirely appropriate to the present task, a number of existing packages designed to transport either toxic chemical substances or highly radioactive materials have specific promising features from existing packages and current safety concepts.

The redundancy criteria will be met by designing into the packaging at least two layers of agent containment. The packaging system will be designed to prevent agent release to the environment in case of an accident. The Army's package will conform to either the transportation panel's recommendations or the Nuclear Regulatory Commission's regulations (10 CFR 71.73), whichever is more stringent.

The packaging will be designed with standard exterior dimensions (probably, 8½ x 8 x 20 ft) and equipped with standardized corners for handling by standard hoisting machinery consistent with the panel's recommendations.

The objectives of the panel's criteria for temperature and **agent** monitoring are met for rail shipments through the pre-placement of low-level monitors at planned stops required by the Power Brake Law (49 CFR 232). Each container will be monitored by sampling air from the outermost airspace of the container. During air movement of munitions in protective packages, the air in the cargo bay of the aircraft will be sampled continuously using a low-level monitor. *The Monitoring Concept Plan* provides additional detail regarding monitoring (Army 1987c).

Temperature monitoring is intended to -detect unusual elevation of temperature within a container carrying munitions that are "energetic" (e.g., rockets having propellants). The transport packaging system will be designed with an automatic temperature-monitoring capability.

The purpose for automatic agent monitoring is the timely detection of agent presence within the walls of the transport container. The automatic monitors currently available can detect only relatively high levels of agent presence. More importantly, there is a question about their reliability with respect to giving false positive or false negative signals. This is a serious consideration for rail transport because of the large quantity of containers involved.

The containerized concept for transportation of the chemical. munitions stockpile minimized handling. Furthermore handling requirements en route under normal conditions are eliminated.

G.7 PROGRAMMATIC CONSIDERATIONS COMMON TO RAIL AND AIR TRANSPORTATION

The Army will carry out several planning and coordination activities before a rail or air transportation program is begun (see Appendix S). These activities include (1) the preparation of a transportation operation plan, safety plan, vulnerability analysis, medical support plan, and emergency response plans; (2) determination of personnel required to be in the Army Chemical Personnel Reliability Program and the implementation of the requirements of this program; (3) development and implementation of personnel training programs; (4) establishment of command and control procedures and a central office; and (5) selection of transportation routes.

G.7.1 ON-SITE TRANSPORTATION AND ASSOCIATED OPERATIONS

Regardless of which disposal alternative is selected, there will be movement of the chemical munitions within the boundaries of the current storage locations. The operational procedures associated with on-site movement include

- activities necessary to prepare and move the munitions and ton containers from the site where they are currently stored to the loading area where off-site transport will begin
- procedures for handling munitions or ton containers once they reach the destination site where destruction will occur, and
- activities involved in preparing and moving munitions and ton containers from the current storage location to a nearby destruction facility on-site.

The activities to be carried out will conform to established Army regulations and standing procedures on safety and security.

Preparations for movement at an origin site begin with monitoring and inspection of stockpile items to be moved. Leaking munitions are identified and contained. For off-site transport, the munition and agents are then packed into the transport container and taken to a holding area where containers accumulate and await off-site transport. For on-site disposal, the munitions are trucked to the on-site disposal facility. At a destination site, a reverse sequence begins with the

unloading of the containers from the transport vehicle and ends with the container's arrival at a storage facility to await disposal.

G.7.2 TRANSPORTATION OPERATING PLAN

An operating plan based on the proposed transportation concept will be prepared for use by program personnel before a transportation program may begin. The operating plan will include detailed procedures on operations, security, and safety. Some of the elements of this plan will be

- material to be transported,
- schedules,
- location of scheduled stops, if any,
- delineation of authority and responsibilities,
- training requirements for personnel,
- security procedures,
- safety and emergency response procedures **and** responsibilities,
- special operating requirements, and
- munition monitoring procedures.

G.7.3 SAFETY SUBMISSIONS AND SITE PLANS

Safety submissions and site plans will be prepared for new facilities located within the boundaries of the Army installations as required by Army Material Command Regulations 385-100 and 385-31. These documents specify distances, methods, and procedures for safe operation of facilities. Some elements of these plans are

- hazard-zone calculations,
- munition container handling requirements,
- inspection and testing requirements,
- emergency equipment and protective clothing requirements,
- contamination control requirements,
- monitoring requirements, and
- special operating procedures.

Site-specific plans will include hazards analyses, which are specific to the site terrain and prevailing weather. The safety submission will be prepared by the individual installations and coordinated subsequently with U.S. Depot System Command; U.S. Army

Armament, Munitions, and Chemical Command; U.S. Army Test and Evaluation Command; Army Material Command Field Safety Activity; and the Department of Defense Explosives Safety Board. The DHHS will also review the plans. These reviews are conducted to ensure that no unsafe operations are implemented, and that all possible measures are taken to avoid accidents or injury to operating personnel or to the surrounding community. All approvals must be obtained before construction of facilities can begin.

G.7.4 PREOPERATIONAL SURVEY

Prior to beginning transportation operations, a preoperational survey will be conducted. The preoperational survey will involve simulating operations beginning at the storage site where activities begin. Inert materials will be used in place of munitions. The purpose of the survey is to identify and eliminate potential safety problems and to ensure effective operations. The preoperational survey will be conducted by an independent team that will observe all aspects of the operations.

The Army will establish procedures for cooperating with local communities. Involvement with local emergency response personnel will be necessary during the simulated transportation activities, Lessons learned from the preoperational survey and simulation of the operational activities will help identify problems and help develop program changes to eliminate these problems. These procedures will be integrated into the emergency response plan.

G.7.5 VULNERABILITY ANALYSIS

A sabotage vulnerability assessment has been carried out to determine the vulnerability of chemical munitions to terrorist activities during transport (Science Applications 'International Corporation 1987). This assessment takes into consideration the elements of transport operations and the variety of threat potentials. It assesses the vulnerability of the operational elements and identifies measures to be taken to reduce vulnerability due to sabotage.

G.7.6 MEDICAL SUPPORT PLAN

A medical support plan for transportation program personnel will be developed for approval by the Army Health Services Command and for review and approval by DHHS. This plan will describe the medical procedures, personnel, facilities, and equipment to be used in supporting the transport of chemical munitions. Medical response to a chemical accident during loading, transport, and unloading will be developed as part of the emergency response plans (see Sect. G.7.6).

G.7.7 EMERGENCY RESPONSE PLANS

A generic Emergency Response Concept Plan has been prepared by the Army (Jacobs Engineerings Group Inc., and Schneider EC Planning and Management Services 1987). If a disposal alternative requiring transportation is selected, specific emergency response plans will be prepared for shipping and receiving installations and for the transportation routes. These plans will be specific for each installation, mode of transport, and transportation route. State and

local authorities will participate, as necessary, in the development of these plans. The nature of these plans will be determined by the characteristics of credible accidents identified in a risk analysis for the munition inventory to be shipped from each location.

G.7.8 PUBLIC AFFAIRS PLANS

Before a transportation program is implemented, the Public Affairs Office of the PEO-PM Cml Demil will prepare public affairs plans for the originating and destination installations. A generic public affairs plan will be prepared for communities along the transportation routes that could be affected.

G.7.9 CHEMICAL PERSONNEL RELIABILITY PROGRAM

The Army Chemical Personnel Reliability Program is used to identify chemical surety duty positions and to manage the personnel assigned to the positions. Features of the program (Army Regulation 50-6) include

- identification and designation of chemical surety positions;
- selection, screening and evaluation of Chemical Personnel Reliability Program candidates on the basis of
  - favorable current personnel security investigation,
  - screening of local personnel records, and
  - evaluation of medical history and physical condition;
- certification of acceptability for the Chemical Personnel Reliability Program by the certifying official;
- personal interview and briefing conducted by the certifying official;
- continuing evaluation by the certifying official and coworkers;
- disqualification of personnel who no longer meet applicable personnel reliability standards; and
- administrative termination of Chemical Personnel Reliability Program personnel no longer assigned to chemical duties.

Personnel with responsibility for chemical munitions and agents must comply with the requirements of the Chemical Personnel Reliability Program. At a minimum, these will be personnel having access to or controlling chemical surety material. Personnel who must be in the Chemical Personnel Reliability Program will be determined and a pool of qualified personnel will be established before a transportation program begins.

G.7.10 PERSONNEL TRAINING PROGRAMS

Personnel must be well trained and certified in accordance with Army Material Command Regulation 350-9 for the tasks involved in the

transportation program to ensure the safety of the operations and the security of the material. Personnel to be trained may include loading and unloading crews; operating crews; convoy command, safety and security personnel; other convoy support personnel such as medical teams; and en route civilians that might be involved in emergency response activities and transportation security. Additional information specific to each transportation mode is given in the appropriate section.

G.7.11 COMMAND AND CONTROL

A Central Command and Control Office will also be established to provide a centralized point of contact for the munition movement operation. All communications to the munition convoy will originate from this office upon approval by the officer in charge of the Command and Control Unit. The duties of the central Command and Control Office will be

- to function as the primary communication link during movement of munitions,
- to receive communication checks and status reports on movements of chemical munitions,
- to relay reports and information to participating and supporting organizations, and
- to provide current information on status of normal and emergency operations to supporting and participating organizations.

G.7.12 ROUTE SELECTION

For rail and air transportation, hypothetical routes are identified in Sect. 2.3 of the FPEIS. Should a disposal alternative requiring transportation be selected, the Army will seek the advice of and coordinate with the appropriate federal agencies, affected commercial carriers, and the states to be traversed in selecting specific routes.

G.8 RAILROAD TRANSPORTATION

The transport of chemical munitions by railroad to a national site or to regional destruction sites will be accomplished by loading sealed munition containers on a unit train dedicated to munitions carriage. For the national destruction site alternative, munitions will be shipped to Tooele Army Depot, Utah, from the seven other storage locations. For the regional destruction alternative, munitions will be shipped (1) to Tooele Army Depot from both Umatilla Depot Activity, Oregon, and Pueblo Depot Activity, Colorado, and (2) to Anniston Army Depot, Alabama, from Lexington-Blue Grass Army Depot, Kentucky; Pine Bluff Arsenal, Arkansas; Newport Army Ammunition Plant, Indiana; and Aberdeen Proving Ground, Maryland.

Munition containers will probably be carried on a type of rail car currently used in rail commerce that is built specifically for carrying stacked shipping containers of standard dimensions. The advantages of using this type of rail car instead of an 89-ft flatcar are superior

ride quality and capacity to carry more munition containers on a train of any given length.

The containers may be carried stacked in two layers or in a single layer. Carrying two layers per rail car (double stacking) is desirable over carrying a single layer because of the doubling of capacity of each munition train, which reduces the number of trainloads needed. Double stacking, however, may reduce routing flexibility because of the height above rail of the stacked containers. Other factors such as security, emergency response, and cost may also affect the decision on whether to double stack. This decision will be part of the development of a rail operating plan.

Shipments of munitions will consist of a munition train preceded by an escort train. Munition trains may carry several types of munitions, but they will contain only one type of chemical agent. The munition train will carry the munition containers, support equipment, the convoy commander, security forces as required by Army Regulation SO-6 and Army Regulation 50-6-1, and other support personnel. The escort train will carry medical supplies and personnel, additional security forces, and other support personnel.

Munition trains will be a maximum 8000 ft long, a length chosen to be compatible with many mainline sidings. Trains may be shorter than 8000 ft for security, emergency response, or other operational reasons. The determination of train length and operating procedures will be part of the development of a rail operating plan if a decision to implement rail transport is made.

The number of trainloads required to transport the stockpile of chemical munitions will depend on train length, the type of rail car used, and whether containers are double stacked. Using trains of 8000 ft and double-stacked containers will result in the fewest trainloads. For these conditions, about 70 to 75 trainloads will be required for the national destruction center alternative. For the regional destruction center alternative, about 50 to 55 trainloads will be required.

G.9 AIRCRAFT TRANSPORTATION

For air transport, the panel recommended the airfield should be at least 200 ft wide and permit a straight-in approach of at least three miles. Crew assignments should be given with due consideration to the normal work/rest cycle, and emphasis should be given to critical component maintenance problem areas. The panel also made recommendations on a number of program management functions addressing public involvement, packaging system testing, and a staff safety function with a central overview of the various safety elements within the transportation program.

Transport of chemical munitions by aircraft will be accomplished by loading the sealed munitions containers (identical to rail transport containers) onto U.S. Air Force C-141 or C-5 transport planes. The munitions containers will require no handling or opening during a flight. However, emergency landing sites will be designated along the route for handling containers if needed in an emergency. Special operating procedures will be developed for the airlift mission.

Two locations are currently under study for air shipment of munitions. These are Lexington-Blue Grass Army Depot, Kentucky, with about 2% of the stockpile by agent weight, and Aberdeen Proving Ground,

Maryland, with 5% of the stockpile by agent weight. The destination will be Tooele Army Depot, South Area, Utah; The two origin sites are being considered for munition removal in response to comments received on the Draft Environmental Impact Statement on the chemical Stockpile Disposal Program. The Army is considering Tooele Army Depot as the only receiving installation for all destruction alternatives because of the low population density in the vicinity of Tooele Army Depot and-the difficulty of constructing an airfield at Anniston Army Depot.

Discussions of the air alternatives are generic in nature at this time. If either of the two site-specific air alternatives is chosen, operational details will then be developed. Details will be provided at the time such an option is chosen in comprehensive site-specific environmental documentation. Included in this documentation will be a detailed design of an air transport container.

G.10 SAFETY ASSURANCE FUNCTIONS WITHIN THE TRANSPORTATION PROGRAM

The safety program management function will be directed by the Safety and Surety Division of the PEO-PM Cml Demil. It is a centralized staff function to ensure that safety in the diverse operational aspects of the transportation program are adequately addressed in the overall program plan and that safety responsibilities for the operational units are identified and properly assigned. A system safety program will be established applying the principles of MIL-STD-882B to the transportation program.

In addition to this safety function, which is aimed at activities internal to the Army, a safety promotion and outreach program will be established. This program will assist those states located along the transport routes in enhancing their emergency preparedness capabilities to respond to chemical munition transportation accidents should they occur. It will provide the states with an understanding of the nature of the hazards and what that implies in terms of preparedness components such as training, equipment, and medical treatment. The program will also provide for channels of communication between the Army and the states to resolve issues that may arise as a result of munitions transport. This program will be developed by the Public Affairs Office in the Office of the PEO-PM Cml Demil and will be coordinated with the affected states.

A program carrying out the transportation of the chemical munitions will be necessarily complex. It will require an organization with elements executing many of the functions addressed in this report--functions such as packaging design and testing, monitoring, loading and unloading, etc. Some of these functions will be organized and implemented in the early stages of the transportation program; others will be carried out only when the program becomes fully operational.

The transportation program will be managed by the Office of the Program Manager for Chemical Munitions, located at Aberdeen Proving Ground, Edgewood Area, Maryland. A detailed program plan covering all aspects of the transportation program will be established early in the program and updated as needed. Overall program direction, responsibilities of the Office of the Program Manger for Chemical Munitions, supporting elements and federal agencies, and integration activities will be defined in the program plan. Specific areas of responsibility will be identified and elements of work assigned. Program

management activities will include management of a safety program and implementation of a public outreach program.

The safety program management function will be directed by the Safety and Surety Division of the Office of the Program Manager for Chemical Munitions; a public outreach program will be developed by the Public Affairs Office and coordinated with the affected states.

A major element of program management is management of the safety program. The safety program includes many elements, such as

- system safety engineering and management,
- site plans/safety submissions,
- preoperational surveys,
- test and evaluation,
- system safety working group,
- accident/incident reporting and investigation,
- emergency preparedness and respons.

G.11 SECURITY PROCEDURES DUPING RAILROAD TRANSPORT OF CHEMICAL AGENTS AND MUNITIONS

The convoy commander will be responsible for the custody, safety, and security of the chemical munition stocks while en route. A total of 26 technical escort personnel, divided between the weapons and pilot trains, will be required. These individuals would be on'duty on 12-h shifts and would be responsible for their supplies, including decontamination equipment throughout the trip.

The two-man rule will apply during all safety and security operations. The two-man rule requires the presence of two authorized personnel, each capable of detecting incorrect or unauthorized procedures with respect to the task to be performed and familiar with applicable safety and security requirements. Two authorized personnel shall be considered to be present when they are in a physical position from which they can positively detect incorrect or unauthorized procedures with respect to the task and/or operation being performed. *When* application of the two-man rule is required, it shall be constantly enforced by the personnel who make up the team, both while they are accomplishing the task or operation assigned and until they leave the area which permits access. The security force shall ensure that no lone individual is permitted in a limited and/or exclusion area unless he is further controlled by technical escort personnel.

Sufficient guards will accompany the trains en route such that there are two guards per munition car. At any stop, guards will dismount, using the two-man rule and maintain a walking guard on each side of the rail car until time for the train to depart. During this period, the guards will inspect the exterior of the rail cars. If an unsafe condition is found, this information will be reported immediately to the convoy Technical Escort Unit Commander. Caution must be exercised during short nonscheduled stops. Direction to dismount will be provided by the

train technical escort commander or his designee.

The following security control procedures will be adhered to:

- The cargo will be under surveillance of the assigned security escort guard personnel at all times while in transit.
- Security surveillance over moving carriers will be maintained by security guards posted in strategically located guard rail cars to observe and preclude unauthorized access or damage to the material being protected.
- Security surveillance over fixed or stationary carriers awaiting further movement or awaiting release to an authorized consignee will be maintained by close-in dismounted security guards. When close-in security surveillance might be better maintained from guard escort vehicles, or similar locations, to avoid inviting public attention to the protected material, the use of dismounted or walking security guards will not be required.
- Written guard orders will be provided for each guard post and will be carried by the guard at all times while on duty, except as provided in the following procedures. Guard orders will be worded carefully, clearly, and as concisely as possible to facilitate thorough understanding by the guards.
- In cases where displayed or carried guard orders would constitute a security risk in themselves, they will not be carried.
- The train TE commander will ascertain immediately before each mission begins that each guard thoroughly understands the guard, orders for his assigned post.

REFERENCES FOR APPENDIX G

Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987. *Emergency Response* Concept Plan *for the* Chemical *Stockpile* Disposal *Program.*

MITRE 1987a. Analysis *of Existing Hazardous* Material *Containers for Transporting* Chemical *Munitions*, Report UP-8700263, The MITRE Corporation, McLean, Va.

MITRE 1987b. *Conceptual Design of a Chemical Munitions Transport Packaging System,* Report UP-8700347, The MITRE Corporation, McLean, Va.

Science Applications International Corporation 1987. *Analysis of the Vulnerability of Chemical Stockpile Disposal Program Alternatives* to *Sabotage or Terrorism* (classified).

U.S. Army, 1987a. Chemical Stockpile Disposal *Program:* Monitoring Concept *Study,* U.S. Army Program Executive Officer--Program Manager for Chemical Demilitarization, Aberdeen Proving Ground, Md.

U.S. Army 1987b. *Chemical Stockpile Disposal Program:* Chemical *Weapons* Movement History *Compilation,* Program Executive Officer--Program Manager for Chemical Demilitarization, Aberdeen Proving Ground, Md.

U.S. Army, 1987c. *Transportation of Chemical Agents and Munitions: A Concept Plan,* Report SAPEO-CDE-IS-87003, Program Executive Officer--Program Manager for Chemical Demilitariztion, Aberdeen Proving Ground, Md.

Appendix H

SEISMICITY

H.1 INTRODUCTION

This appendix provides a summary of seismic risk at eight chemical munitions storage sites (Figs. H.1-H.8) in the conterminous United States. These figures visually illustrate the wide variability of seismic risk among the sites. The north-south-trending Wasatch Mountains immediately east of TEAD (Fig. H.7) are seismically active, whereas at other sites [LBAD (Fig. H.3), ANAD (Fig. H.2), and PUDA (Fig. H.6)] seismicity is rather low and scattered. At two sites [PBA (Fig. H.5) and UMDA (Fig. H.8)] active seismic zones are confined to distant sources in one quadrant (the New Madrid region of southeast Missouri and the volcanically active Cascade Mountain Range, respectively). At NAAP (Fig. H.4) a moderately active seismic zone extends from the southwest quadrant up the Wabash Valley almost to Newport, Indiana. The APG site (Fig. H.l) lies within a moderately active southwest-to-northeast-trending seismic zone, but the storage site is in the Chesapeake Bay region, which appears to be less active. All the previous figures are computer-generated maps from current data files of the National Oceanographic and Atmospheric Administration's (NOAA) National Geophysical Data Center of Boulder, Colorado.

H.2 PROBABILISTIC RISK ANALYSIS

Estimated mean value peak horizontal accelerations (Am) in rock for a 475-year return period (475YRP) are used to estimate the annual probabilities of exceedance as a function of Am at each storage site (Sect. 4). These estimates are based on Applied Technology Council (1978) data. In this analysis the 475YRP Am is estimated'to the nearest 0.05 of the earth's gravitational acceleration (g). At TEAD and NAAP the 475YRP Ams are 0.20 and 0.10 g, respectively. At each of the other sites the 475YRP Am is 0.05 g. Actual Ams are probably not known well enough to justify greater precision. The reader is reminded.that these are mean value predictions and that they apply only where sites are on firm ground (shallow deposits of stiff cohesive and dense granular soils, as well as rock). The Applied Technology Council (1978) suggests multiplication factors of 1.2 and 1.5 for intermediate and soft ground, respectively.

H.3 INFLUENCE OF NATURAL FACTORS ON SEISMIC RISK

Although peak horizontal acceleration is a very important consideration in seismic risk analysis, there are a number of other natural and cultural factors that require consideration as well. Natural factors include foundation conditions and an earthquake's magnitude and location with respect to the storage site, both of which profoundly influence the Am and the duration of shaking (defined variously as the length of time that horizontal acceleration remains above 0.05 to 0.10 g). The influence of natural factors is considered in Sect. 3, and the effects on various facilities are considered in Sect. 4.

ORNL-DWG 86- 11996

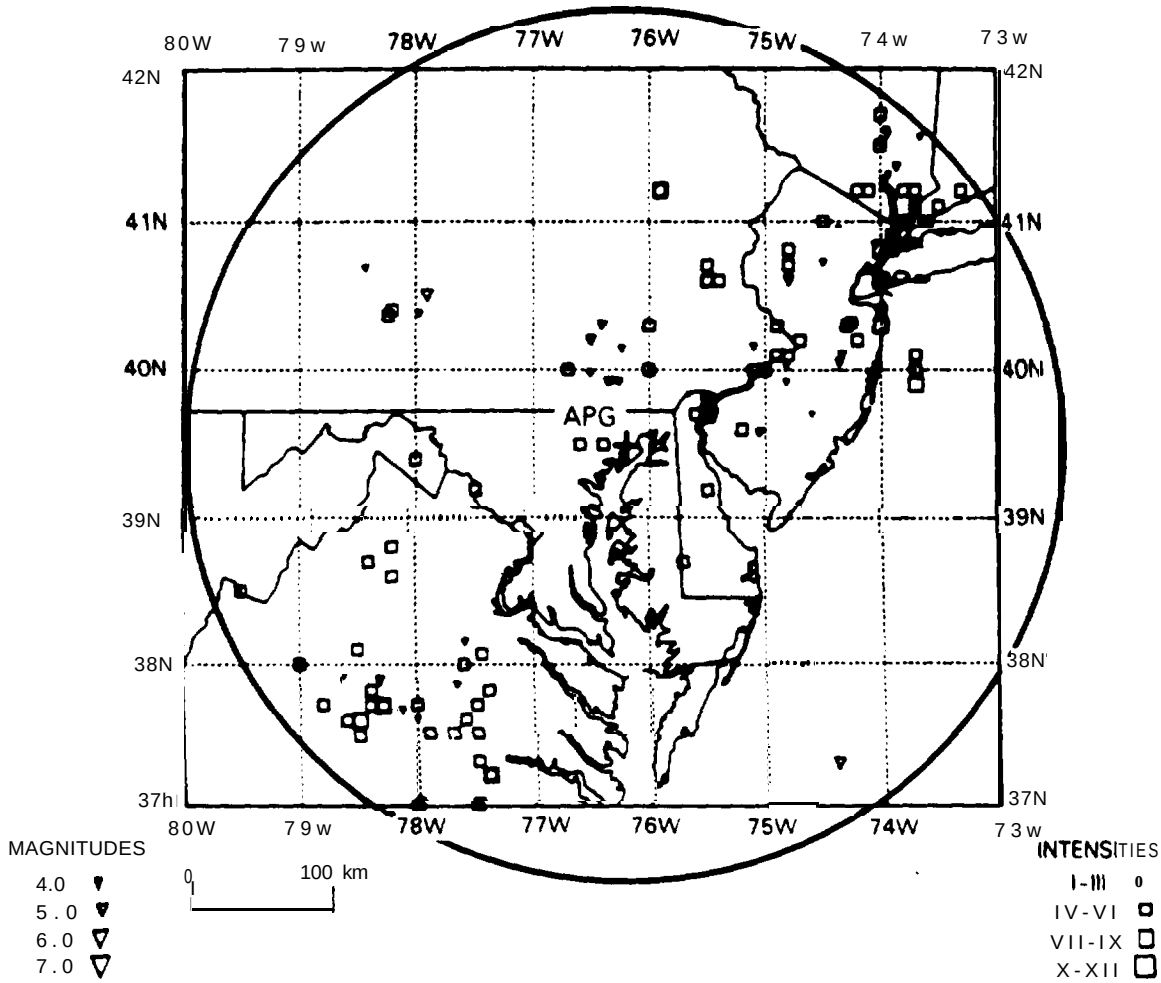


Fig. H.1. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of APC.

ORNL-DWG 86-11995

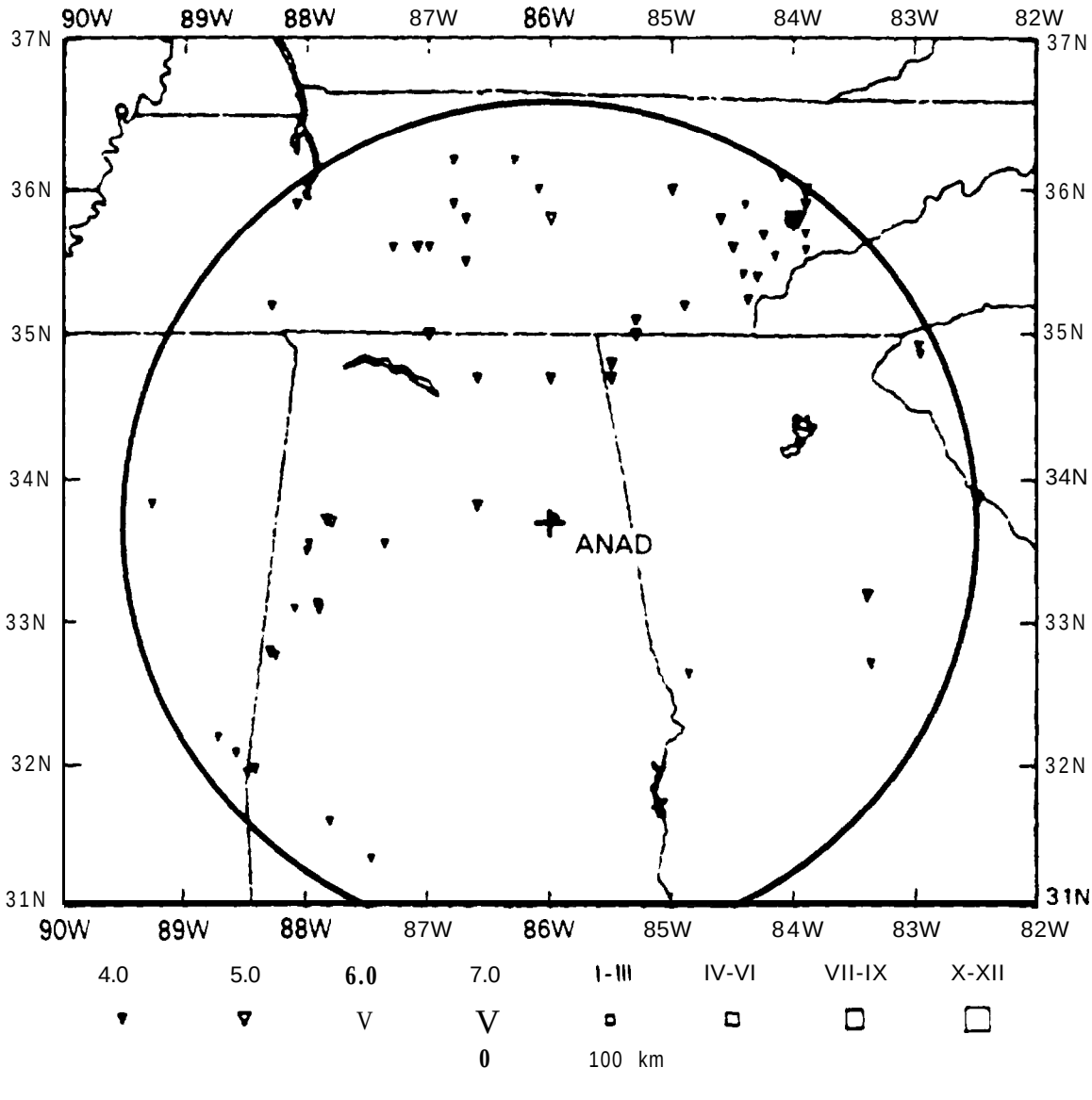


Fig. H.2. Historically and instrumentally recorded eartbquakes 01 magnitude equal to or greater than 4 within 320 km of ANAD.

ORNL-DWG 86- 11997

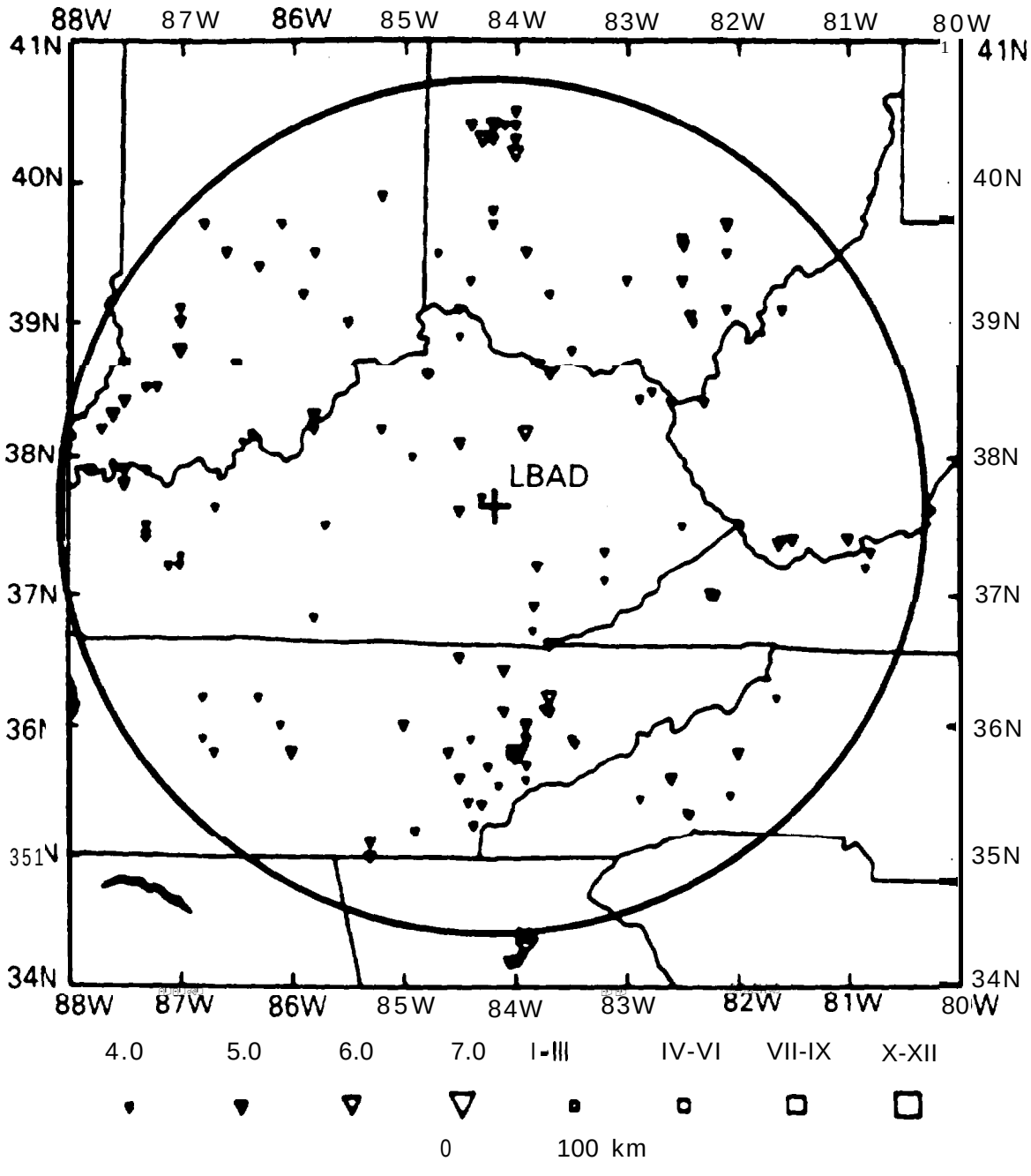


Fig. H.3. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of LBAD.

ORNL-DWG 86-I 1994

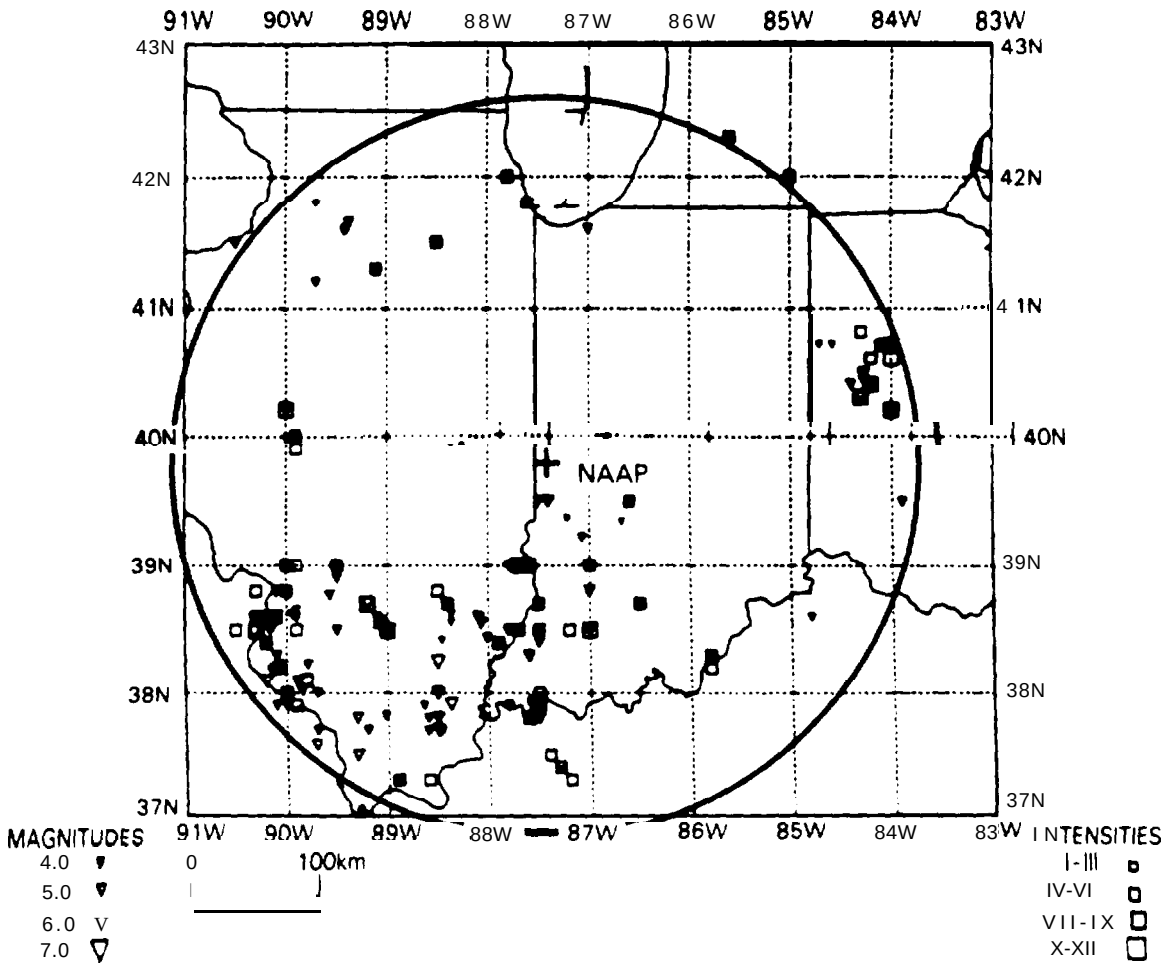


Fig. H.4. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of NAAP.

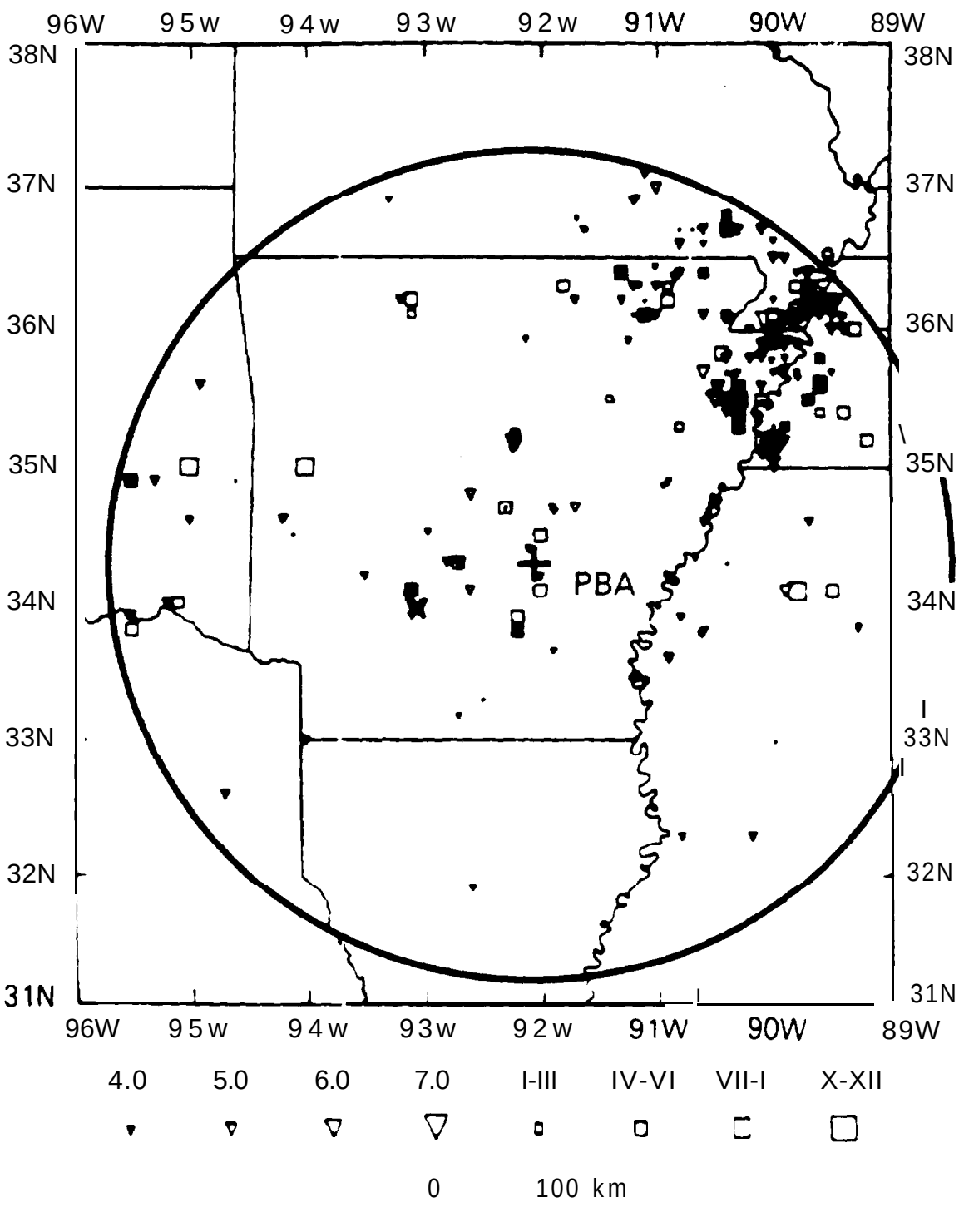


Fig. H.5. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of PBA.

ORNL-DWG 86- 11992

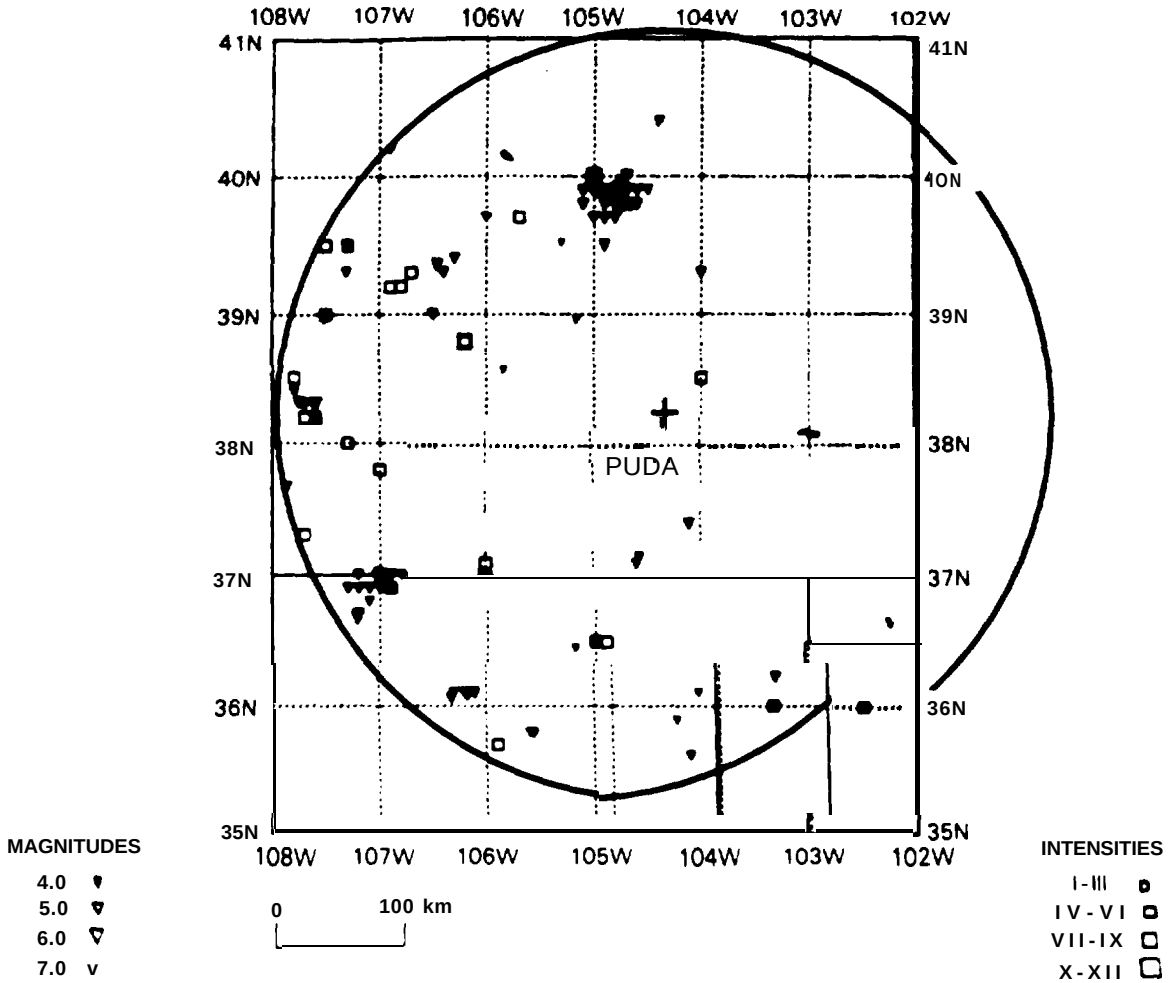


Fig. H.6. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of PUDA.

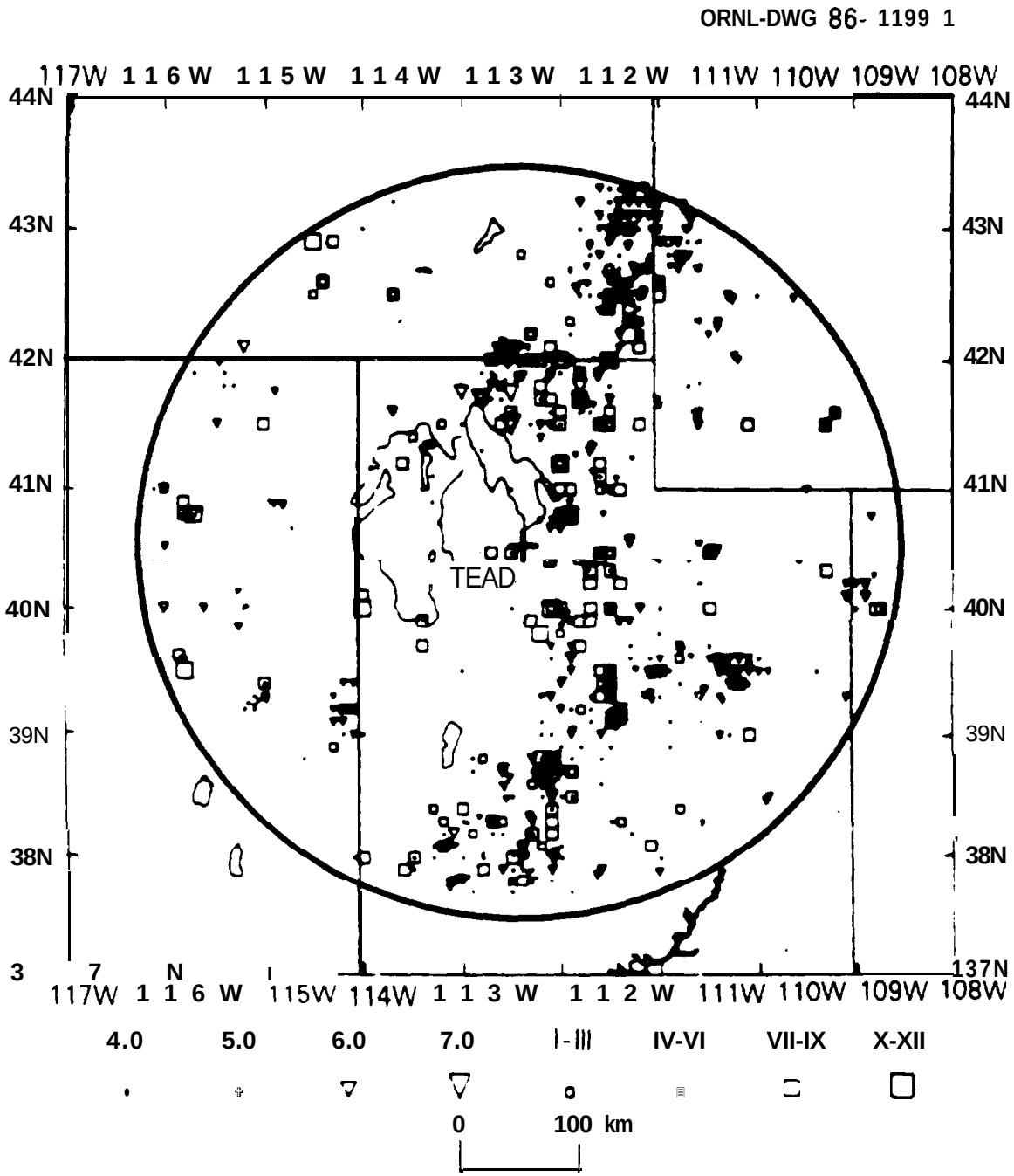


Fig. H.7. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of TEAD.

ORNL-DWG 86- 11990

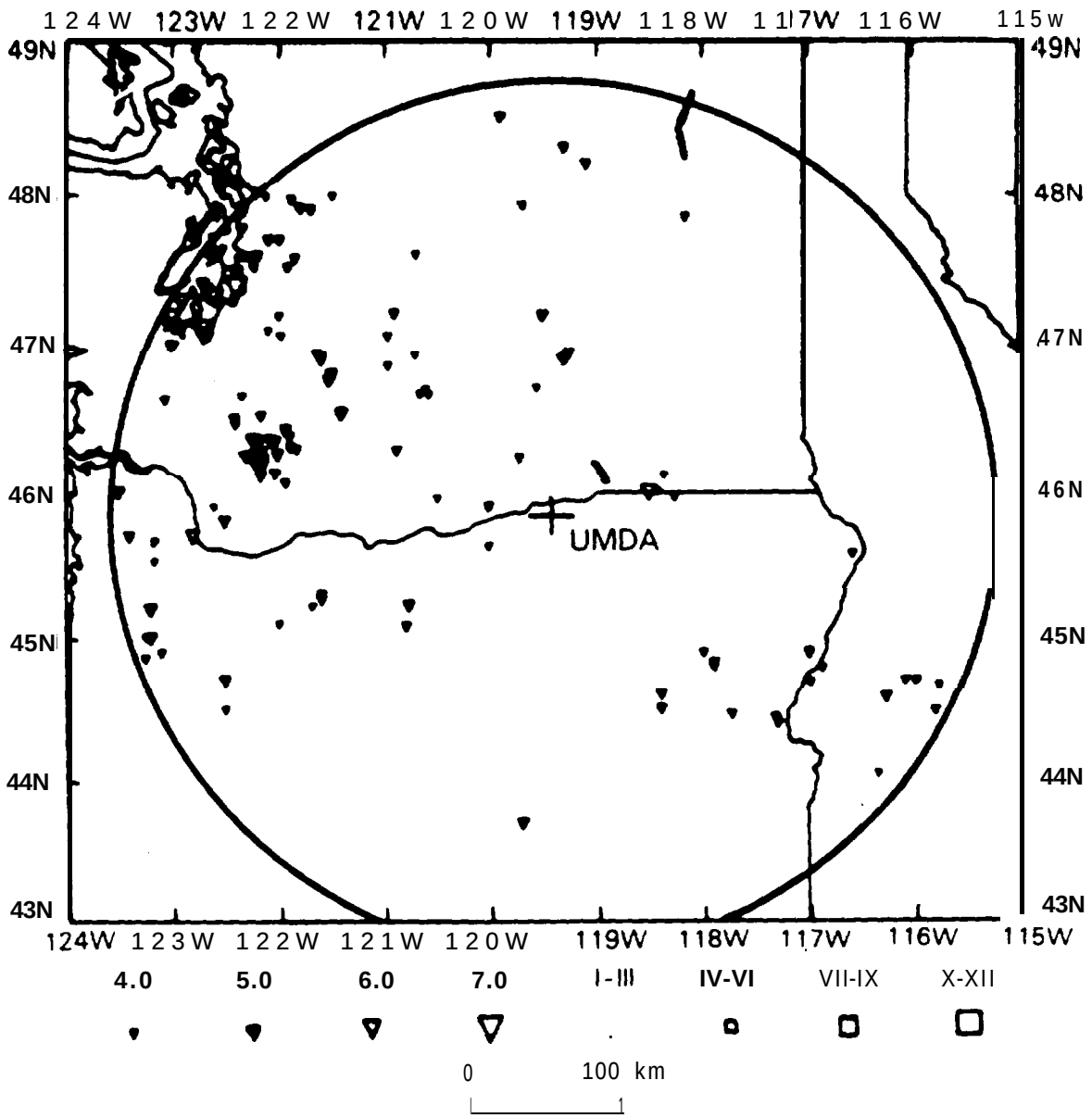


Fig. H.8. Historically and instrumentally recorded earthquakes of magnitude equal to or greater than 4 within 320 km of UMDA.

H.3.1 FOUNDATION CONDITIONS

Ground motions vary significantly as a function of foundation conditions. These conditions include whether a structure is placed on consolidated rock or unconsolidated sediments, the degree of consolidation, the thickness and type of unconsolidated sediments, and the position of the water table with respect to unconsolidated sediments. Foundation liquefaction may occur at an Am as low as 0.05 g. Liquefaction commonly occurs in unconsolidated sands and silts where the water table is shallow. By contrast, the threshold of failure for ordinary structures on rock varies between Am = 0.15 to 0.20 g. Highly variable and site-specific foundation conditions are not amenable to probabilistic seismic risk analysis. However, generalized foundation conditions are sufficiently well known at various storage sites to qualitatively assess their responses to earthquakes. The effects of foundation conditions on facilities during an earthquake are addressed in Sect. 3.

H.3.2 EARTHQUAKE LOCATION AND INTENSITY

Ground motions also vary significantly as functions of an earthquake's location and epicentral intensity. Ground motions are large but uncertain for modified Mercalli intensities (MMI) IX or greater. Major earthquakes (MMI XI or XII) produce more uncertain ground motions even at great distances from their epicenters. Ground responses also differ for sites located in the near- and far-fields of an earthquake. Krinitzsky and Marcuson (1983) define the boundary between the near- and far-fields as 15 and 25 km from the epicenter of earthquakes of MMI VII and VIII, respectively.

H.3.3 RESONANCE

When the natural frequency of vibration of a foundation matches the frequency of a seismic wave impressed upon it, a condition of resonance exists. Near-resonance or resonance magnify ground motions from several-fold to more than tenfold, increasing the peak horizontal acceleration and prolonging the duration of shaking with respect to nonresonant conditions. The frequency of vibration of seismic waves varies as a function of epicentral distance. High-frequency seismic energy is more rapidly absorbed than low-frequency seismic energy by the earth through which it passes, so that seismic waves are predominately low frequency at sites located in an earthquake's far-field. The natural frequency of vibration for rock foundations is generally above that of seismic waves (increasingly true in the far-field), and in such cases resonance rarely occurs. By contrast, unconsolidated sediments have a low natural frequency of vibration, and resonance may occur during an earthquake if conditions are right. Resonance may promote liquefaction beneath any type of building, but the most spectacular damage occurs in high-rise structures as they are whipped back and forth by horizontal

ground motions over an extended duration of shaking.

H.3.4 CASE HISTORIES

Case histories provide vivid evidence of the effects of foundation conditions on ground motions during an earthquake. The Sharpsburg, Kentucky, earthquake is an example illustrating higher MMIs on unconsolidated sediments in contrast to adjacent areas where rock is close to the surface. The Acapulco earthquake illustrates liquefaction and resonance of ancient lake bed sediments at a distant site (Mexico City).

H.3.4.1 Sharpsburg, Kentucky

The Sharpsburg earthquake of July 27, 1980, was felt over a wide region of the eastern United States (Fig. H.9). At Sharpsburg (the nearest municipality to the epicenter), the earthquake was felt as MMI VI, but at Maysville, Kentucky (60 km to the north), the local MMI reached as high as VII. At Richmond, Kentucky (60 km to the south), the MMI was IV (NOAA 1982). Intensities were higher in northern Kentucky and southwestern Ohio along the floodplain of the Ohio River and adjacent glaciated regions but lower in central and southern Kentucky, where bedrock lies close to the surface. Newspaper accounts generally referred to this event as the Maysville earthquake because the perceived intensity was higher than that at Sharpsburg.

H.3.4.2 Acapulco, Mexico

A severe earthquake occurred on September 19, 1985, in the eastern Pacific Ocean about 50 km from the port city of Acapulco, Mexico. Damage in the epicentral area was restricted to a few tourist resorts and industries along the coast, and damage further inland was restricted to particularly weak buildings. Those parts of Mexico City (more than 350 km from the epicenter) built on solid rock were left undamaged or only slightly damaged. In contrast, areas of Mexico City built on soft, water-saturated sediments of an ancient lake bed suffered catastrophic damage [over 10,000 fatalities, 770 buildings completely destroyed, another 1,665 severely damaged, and 5,000 slightly damaged, at a cost of about $4 billion in United States dollars]. The extensive damage was attributed to resonance coupling between earthquake waves and the widely distributed lake-bed sediments and high-rise buildings (Munich Reinsurance Company 1986). News media referred to this catastrophic event as the Mexico City earthquake despite the closer proximity of Acapulco to the epicenter.

The potential for widespread damage must be considered in terms of its effect on emergency planning. Hospitals were among the structures extensively damaged or destroyed at Mexico City. Although MMI IX was

ORNL DWG 87-16813

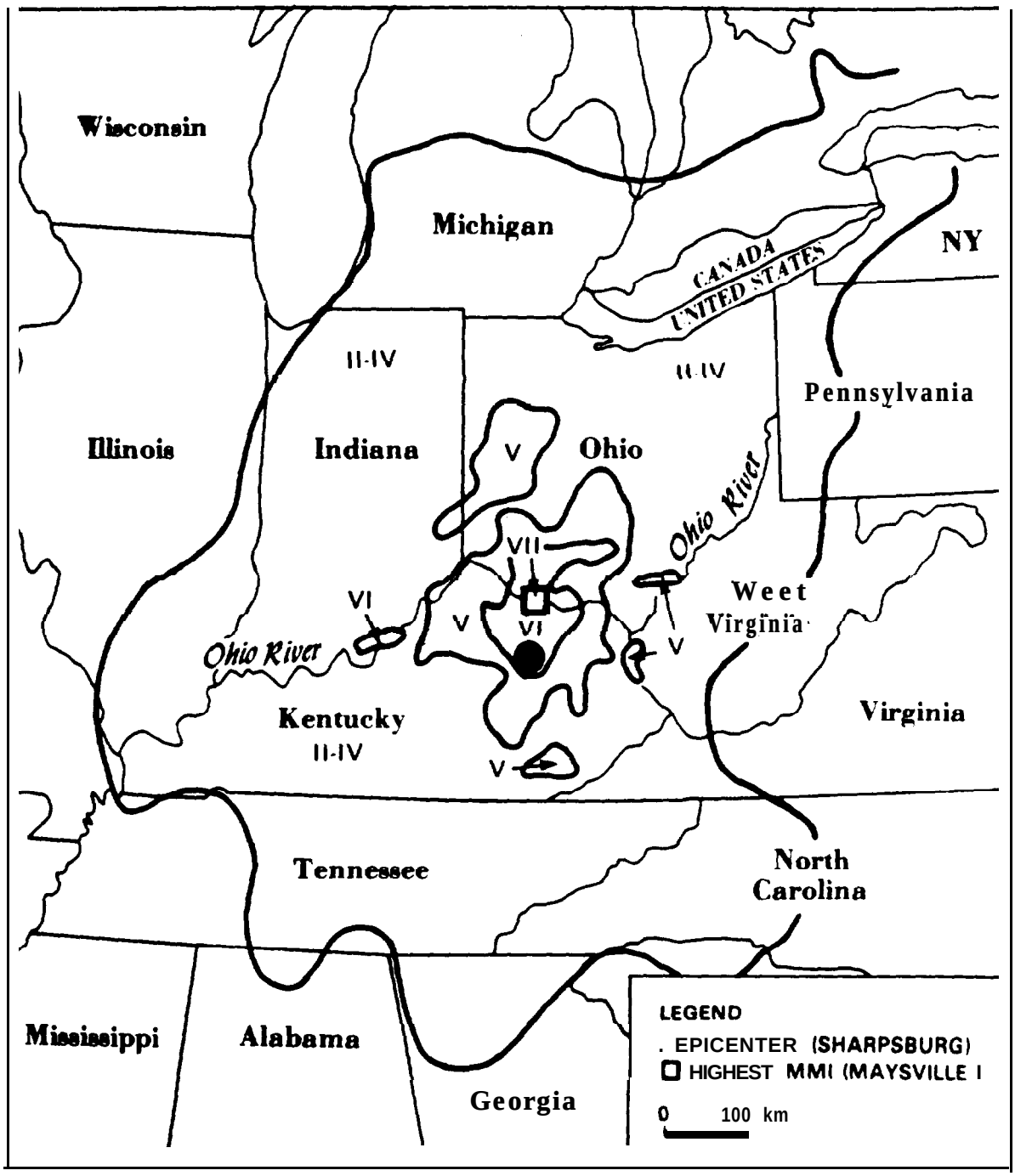


Fig. H.9. Isoseismal map for the northern Kentucky earthquake of July 27, 1980 [modified after NOAA (1982)].

assigned to the earthquake at both Acapulco and Mexico City, only scattered areas were damaged at Acapulco. Medical facilities at Acapulco remained essentially intact.

H.4 METHODOLOGY FOR COMPARING STORAGE SITES

Tables H.l and H.2 illustrate the uncertainties and variations in ground motions with respect to site conditions and earthquake sources. Estimated ground motions for a far-field "comparison" earthquake are listed for given site conditions. These data are generalized, and they are not intended for use in structural design.

A far-field, 500-year earthquake was arbitrarily selected as the basis for comparison of ground motions among storage sites. Too much uncertainty is associated with earthquakes of longer recurrence intervals, and near-field, 500-year earthquakes are generally too low in intensity to yield significant differences in ground motions among storage sites. A 500-year earthquake may occur anywhere within a 10,000-km^2 area surrounding each site. The MMI will be reduced by one unit between the earthquake's epicenter and the site (assuming the site is located in the same seismic source zone as the earthquake, foundation conditions are similar between the epicenter and the site, and the site is in the far-field of the earthquake). The MMI of the far-field, 500-year earthquake is estimated from data provided by Algermissen et al. (1982), supplemented by data from Hadley and Devine (1974) and open-file data (Figs. H.1-H.8) from the National Oceanographic and Atmospheric Administration, National Geophysical Data Center (NOAA). The most conservative data source is used where discrepancies are encountered among data sources.

On-site MMIs for the far-field, 500-year earthquake are used to estimate ground motions at each storage site. Krinitzsky and Marcuson (1983) established empirical relationships between MMI and ground motions for both firm and soft foundation material. Their data are used to estimate mean value and one standard deviation above the mean ground acceleration (Am), velocity (Vm), and duration of shaking (Dm) for both firm ground and soft sediments. Region-wide foundation conditions from various open-file sources and ground motions from Krinitzsky's data provide the basis for seismic risk analysis in Sect. 3.

Some sites are located in comparatively inactive seismic source zones (SSZs) as defined by Algermissen et al. (1982). In these cases 500-year earthquakes from more distant SSZs cause greater ground motions at the site than local 500-year earthquakes. For example, a far-field, 500-year earthquake located in the same SSZ as PBA is a MMI VI earthquake, but it would be attenuated to MMI V at the storage site. In comparison, a 500-year earthquake in the New Madrid SSZ (180 km from PBA) has a MMI of IX, but it would be attentuated to MMI VI or VII at PBA. In such cases, the most damaging earthquakes are most likely to occur outside the SSZ in which the site is located. Anderson's attenuation curves for eastern and western United States (Chandra 1979)

Table H.1. Foundation conditions, estimated modified Mercalli intensities, and perk horizontal accelerations in rock for a far-field, 500-year earthquake at eight chemical weapons storage rites in the conterminous United States

site	Foundation conditions'	500-year earthquake[b]			475-year return period Am (% g)	
		MMI Epicenter/ on-site	Minimum distance (km)	Active fault?	c	d
ANAD	Firm	VII/VI	15	No	5	9
APG	Soft	VII/VI	15	No	5	5
LBAD	Firm	VII/VI	15	No	5	6
NAAP	Firm	VII+/VI+	15	No?'	10	8
PBA	Soft	IX/VI +	180[f]	Yes	5	6
PUDA	Soft	VI/V	15	No	5	5
TEAD	Soft	IX/VIII	35'	Yes	20	17
UMDA	Firm	XI/VI	250[h]	Yes	5	6

"Based on open-file data.

[b]Based on data provided by Algennisscn et al. (1982), supplemented by open-file data from NOAA's National Geophysical Data Center (Figs. H.I-H.8) and (for eastern sites) Hadley and Devine (1974).

'Based on contoured data from Applied Technology Council (1978), p. 316.

"Nearest posted value, Algermissen et al. (1982).

'Nearby Wabash Valley graben, activity on faults speculative.

'Distant New Madrid seismic zone, active fault identified.

'Nearby Wasatch Mountains, active faults identified.

"Distant volcanically active Cascade Mountains.

Table H.2. Estimated ground motions (for foundation conditions, MMI, and epicentral distances as given in Table H.l) for 8 far-field, 500-year earthquake at eigbt chemical weapons storage sites in the conterminous United States[a]

Site	A (% g)		V (cm/s)		D (s)	
	Am[b]	Am + 1 std[c]	Vm[b]	Vm + 1 std	Dm[b]	Dm + 1 std
ANAD	7	10	5	7	1	3
APG	5	8	7	11	1	3
LBAD	7	10	5	7	1	3
NAAP	8	12	8	13	3	5
PBA	8	12	10	16	3	5
PUDA	3	5	3	6	1	2
TEAD	20	30	21	32	14	20
UMDA	5	10	5	11	2	5

[a]Mean value and one standard deviation above the mean ground motions, based on data provided by Krinitzsky and Marcuson (1983); A-acceleration, V-velocity, and D-duration of shaking.

[b]Am, Vm, Dm = mean values.

[c]std = standard deviation.

were used to estimate on-site MMIs from distant SSZ SOO-year earthquakes.

Other sites are located in mildly or moderately *active* SSZs. In these cases, earthquakes occurring in the same SSZ as the storage *site* cause greater ground motions than earthquakes from *more* distant SSZs at the 500-year recurrence interval.

REFERENCES FOR APPENDIX H

Algermissen, S. T., et al. *1982. Probabilistic Estimates of Maximum Acceleration and* Velocity *in Rock in* the *Contiguous United States,* U.S. Geological Survey, Open-File Report 82-1033, Denver, Colo.

Applied Technology Council 1978. *Tentative Provisions for the Development of Seismic Regulations for Buildings,* U. S. Department of Commerce, National Bureau of Standards, Special Publication 510, Washington, D.C.

Chandra, U. 1979. "Attenuation of Intensities in the United States," *Bull. Seismol. Soc.* Am., **60(3),** 2003-2024.

Hadley, J. B., and Devine, J. F. 1974. "Seismotectonic Map of the Eastern United States," MF-620, U. S. Geological Survey, Denver, Colo.

Krinitzsky, E. L., and Marcuson, W. F. 1983. "Principles for Selecting Earthquake Motions in Engineering Design," *Bull. Assoc. Eng. Geol.,* **20(3)**, 253-265.

Munich Reinsurance Company 1986. *Earthquake Mexico* '8.5, Munich, Federal Republic of Germany.

NOAA *1982. United* States *Earthquakes, 1980,* jointly published by the USGS and NOAA, Golden, Colo.

NOAA n.d. National *Geophysical Data File,* National Geophysical Data Center, Boulder, Colo.

Appendix I

COORDINATION WITH THE PRESIDENT'S COUNCIL ON ENVIRONMENTAL QUALITY

EXECUTIVE OFFICE OF THE PRESIDENT
COUNCIL ON ENVIRONMENTAL QUALITY
722 JACKSON PLACE N W
WASHINGTON D C 20006

June 2, 1986

Ms. Amoretta M. Hoeber
Deputy Under Secretary of the Army
The Pentagon, Washington, D.C. 20310

Dear Ms. Hoeber:

I am writing in reference to the Department of the Army's compliance with the National Environmental Policy Act, as it pertains to disposal of the U.S. stockpile of lethal chemical agents and munitions, required by Congress to be completed by September 30, 1994. As you know, I attended the briefing on March 28, 1986, concerning the Chemical Munitions Demilitarization Program and was pleased to note the Army's commitment to the spirit of the National Environmental Policy Act (NEPA). Since that time, the CEQ General Counsel has met with Army representatives who are involved in the NEPA compliance for this proposal, and attended the public scoping meeting at Aberdeen Proving Ground, Maryland, in connection with this proposal.

My understanding at this time is that the Army is preparing d programmatic environmental impact statement (PETS) which examines the Chemical Stockpile Disposal Program. Alternatives to be examined in this PEIS include incineration of each portion of the stockpile on-site where it is currently located, transporting the stockpiles to two regional centers, or transporting the stockpiles to d national Site for disposal. I understand that the Army views incineration as the only sound disposal method currently available. Other technology alternatives eliminated from further consideration, largely as the result of a 1984 National Research Council study, will also be identified and briefly discussed. The PEIS will serve as the environmental documentation necessary to aid the decisionmaker in his or her decision regarding the selection of a disposal alternative, which will then be documented in a Record of Decision.

After completion of the PEIS and the decision as to which disposal alternative will be implemented, further NEPA documents will be prepared to document the implementation of thdt decision - for example, further analysis of specific transportation routes or operations at site-specific disposal faciities. This further NEPA documentation (which would be in the form of either environmental impact statements or environmental assessments, as appropriate) would be tiered to the PEIS, thus providing for d comprehensive analysis of both the program dnd the specific implementation of the program.

Ms. Hoeber
Page Two

This plan for NEPA compliance in relation to the Chemical Munitions Demilitarization Program is consistent with the CEQ regulations implementing the procedural provisions of NEPA. As used in those regulations, "tiering" refers to the coverage of general matters in broader environmental impact statements (such as national programs • here, the Chemical Stockpile Demilitarization Program) with subsequent narrower environmental analyses (such as regional or site-specific environmental impact statements or environmental assessments), incorporating by reference the general discussions and concentrating solely on the issues specific to the statement subsequently prepared. (40 C.F.R 1508.28) Agencies are encouraged to tier their environmental impact statements to eliminate repetitive discussions of the same issues and to focus on the actual issues ripe for decision at each level of environmental review. (40 C.F.R. 1502.20) The Council believes that tiering can be a useful method of reducing paperwork and duplication when used carefully for appropriate types of plans, programs and policies which will later be translated into site-specific projects. See, Guidance Regarding NEPA Regulations, 48 FR 34263, 34268 (1983).

The Army's plan for preparing a PEIS, with further NEPA documentation tiered to the PEIS, is appropriate and consistent uith the letter and spirit of the NEPA regulations. The scoping meetings which have been held, both with the public and with federal and state agencies, should be valuable in framing the issues to be addressed in the PEIS.

Let me reiterate my support for your careful consideration of how best to comply with NEPA in preparing for this difficult task. Please let me know if the Council can be of further assistance to you in implementing this compliance.

Yours truly,

A. Alan Hill
Chairman

Appendix J

RISK ANALYSIS SUPPORTING THE FINAL ENVIRONMENTAL IMPACT STATEMENT FOR THE CHEMICAL STOCKPILE DISPOSAL PROGRAM

APPENDIX J

CONTENTS

	Page
J.l INTRODUCTION	J-l
J.1.1 PURPOSE OF THE RISK ANALYSIS	J-l
J.1.2 RISK ELEMENTS OF THE CSDP	J-2
J.1.2.1 The Disposal Alternatives	J-3
J.1.2.2 Disposal Activities	J-3
J.1.2.3 Agents and Munition Types	J-7
J.1.2.4 Accident Types	J-7
J.1.3 PRIOR STUDIES	J-8
J.1.4 DATA SOURCES FOR THIS ANALYSIS	J-8
5.2 METHODOLOGY OF THE RISK ANALYSIS	J-11
J.2.1 INTRODUCTION TO RISK ASSESSMENT CONCEPTS	J-11
J.2.2 APPLICATION OF RISK CONCEPTS TO THE CSDP	J-12
J.2.3 PRINCIPAL MEASURES OF RISK	J-13
J.2.4 METHODS FOR PORTRAYING RISK	J-16
5.3 PRESENTATION AND DISCUSSION OF RESULTS	J-19
J.3.1 OVERVIEW OF RISK DATA	J-19
J.3.1.1 Disposal Alternatives and Site-Stockpiles Biological Considered	J-20
J.3.1.2 Treatment of Mitigation	J-20
J.3.1.3 Treatment of Uncertainty	J-22
J.3.1.4 Description of Data	J-23
J.3.2 PROGRAMMATIC RISK OF ALTERNATIVES	J-23
J.3.2.1 General Comparison	J-23
J.3.2.2 Major Sources of Risk in Each Alternative	J-31
J.3.3 LOCATION-SPECIFIC RISK	J-49
J.3.3.1 Distribution of Programmatic Risk by Location	J-49
J.3.3.2 Factors Contributing to Community/Societal Risk, by Location	J-93
J.3.3.3 Time and Person-Years at Risk	J-109
J.3.4 CAVEATS AND LIMITATIONS	J-114
J.3.4.1 Frequency and Consequence Screening	J-114
J.3.4.2 Potential Fatality Estimates and Site Boundaries ,	J-114
5.4 ACCIDENT SCENARIO DESCRIPTIONS	J-117
J.4.1 ACCIDENT SCENARIO IDENTIFICATION	J-117
J.4.2 SUMMARY DESCRIPTIONS OF ACCIDENT SCENARIOS	J-119
REFERENCES	J-129

J.1.0 INTRODUCTION

J.1.1 Purpose of The Risk Analysis

This appendix presents a summary of the results of a comprehensive risk analysis performed in support of the Chemical Stockpile Disposal Program (Fraize et al., 1987a). The major purpose of this risk analysis is to provide the Army with a consistent and quantitative comparison of the risks associated with each of the disposal alternatives. The relative risk to public safety of the alternatives has been evaluated on the basis of risk to the public (individuals outside the boundaries of the military installation) at proposed disposal sites and along potential transportation corridors.

This document reports the following risk analysis results:

- programmatic and site-specific [illegible] to affected populations;
- major contributors to risk; and
- differences among alternatives.

The risk analysis is intended to meet information needs of several audiences. The principal audiences are:

- Army decision-makers who must select a disposal alternative;
- local governments and community groups who need to understand and evaluate the potential impacts on their local populations;
- individuals who are concerned about their personal risk, given their locations with respect to storage sites, disposal sites, or transportation routes; and
- Army program managers responsible for implementing a disposal alternative, who must be aware of activities that have the potential for high risk to the public, and who must ensure that the CSDP is implemented safely.

Identifying the major sources of risk requires that the risk analysis be performed on an accident-specific basis. Data presented in Fraize et al., 1987a supports analysis at that level of detail. This appendix presents more aggregated information on risk to society, using a set of complementary measures of risk.

Since risk analysis deals with potential future occurrences, uncertainty in the results is unavoidable. In addition, uncertainty in the

risk analysis arises from gaps in data and in our understanding of the accident phenomena, which require that many assumptions be made in the analysis. Estimates of uncertainty in the probability of accident occurrence have been developed, and are displayed with the risk estimates.

Despite uncertainties in the results, risk analysis remains the best available means for systematically identifying major sources of risk, quantifying safety concerns, and comparing the relative risk of the different alternatives. Subjective factors related to developing a sound safety philosophy (e.g., administrative controls) and to managing risks that are difficult to quantify (e.g., sabotage, procedural errors) are important also, and need to be considered along with the insights offered by quantitative risk analysis.

The data used in this risk analysis are of two broad types: historical data -- that is, data derived from records of a large number of actual events which are related to specific types of accidents, or events leading to them; and hypothesized data -- data derived from largely subjective modeling of assumed accident sequences with the aid of fault and event trees describing the process. (The use of fault and event trees is a standard procedure to investigate sequences of occurrences in a complex system.)

J.1.2 Risk Elements of The CSDP

To understand the ways in which the Chemical Stockpile Disposal Program (CSDP) might present risk to the public, one needs first to identify the major features of the CSDP, including:

- the disposal alternatives, including the "no-action" alternative (continued storage);
- the disposal activities (e.g., handling, transportation, plant operations) that make up the alternatives;
- the chemical agents themselves and the munition configurations in which they are stored; and
- the various accident initiators (e.g., human error, equipment failure, natural event) and accident types that could lead to agent release.

Each of these features is discussed below.

J.1.2.1 The Disposal Alternatives

For purposes of this risk assessment, the disposal alternatives are defined by where, *not* how, the destruction of the chemical stockpile takes place. The disposal technology assumed here for all alternatives is the "baseline" technology which consists primarily of mechanical disassembly of the munitions, draining of the chemical agent, destruction of the agent in liquid incinerators, incineration of "energetics" (propellants, bursters, etc.) in deactivation furnaces or kilns, and destruction of residual agent in metal parts and dunnage furnaces. The disposal alternatives are, therefore, distinguished by the logistics of munition movement and the location of the disposal activities. These alternatives can be summarized as follows:

- on-site disposal: all chemical agents are destroyed at the sites where they are now stored;
- regional disposal: munitions stored in the eastern region of the country are shipped by rail to Anniston Army Depot, Alabama, while those in the west are shipped to Tooele Army Depot, Utah;
- national disposal: all munitions in the Continental U.S. are shipped by rail to Tooele Army Depot for destruction; and
- partial relocation: on-site disposal at all sites except for relocation of the stockpile to Tooele Army Depot by C141 aircraft from two sites: Aberdeen Proving Ground, Maryland, and Lexington-Blue Grass Army Depot, Kentucky.

The risk implications of the disposal alternatives are apparent in the potential for the redistribution of overall risk. Movement of the stockpile from one site, in what could be a densely populated region, to a second site, in what could be a sparsely populated region, might reduce the risk to the population around the first site, at the expense of added risk to people along the transportation corridor and around the second *site.* The magnitude of these risk differences is one of the questions answered by the risk analysis.

J.1.2.2 Disposal Activities

Each of these disposal alternatives comprises many activities. These range from the relatively simple activities associated with continuing to store the munitions, to the more complex activities associated with handling, shipping, or disassembly/destruction of the stockpile elements. Since these activities involve some contact with the chemical stockpile, they all could pose some risk to the public.

Figure J.l illustrates the major activities associated with each disposal alternative. Many of these activities are common to some or all of the disposal alternatives.

The "no-action" alternative, continued storage, involves the risks associated with storage in fixed sites (igloos, warehouses, or open fields). The major risk elements are relatively rare, external or natural catastrophic events, such as tornadoes and aircraft crashes; maintenance and surveillance activities for the stored stockpile also contribute to risk. Storage-related accidents are typically very low in their probability of occurrence, but very high in potential consequence, because of the large inventory of agent likely to be affected by any one event. In the continued storage alternative, all potential agent-releasing events (including leaks and maintenance-related handling accidents) are assumed to pose risk over an indefinite period -- taken for this analysis to be 25 years. No account has been taken in this analysis of the presumed risk of eventual disposal after a long period of continued storage.

The on-site disposal alternative involves risk posed by the following activities:

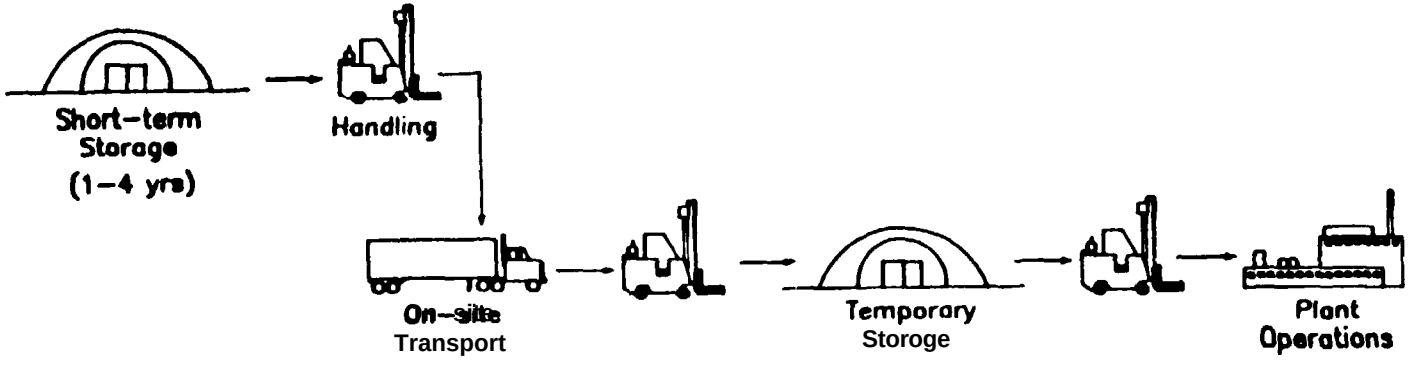
- handling activities, required to move the stockpile elements from their storage areas to on-site transportation containers, and from the transportation containers to the on-site disposal facility, and from one operation to another within the facility;
- on-site transport activities, moving the stockpile by truck from storage area to plant over on-site roads; and
- plant operations activities, including all steps required to disassemble, drain, and incinerate the chemical agents and munitions.

The national. regional. and partial relocation disposal alternatives introduce several different classes of activity posing some risk:

- additional handling activities, involving stockpile movement from storage to the packing/holding/loading areas (essentially the same risk as movement of the stockpile from storage to on-site disposal plant) for subsequent off-site transport, plus handling at the transportation container unloading/holding/unpacking areas and handling at the destination site (essentially a reversal of the activities at the sending site); and
- off-site (inter-site) transport activities, involving long distance transport by one of two modes, depending on which disposal alternative is being considered. (See S.C. Chu et al., 1987.)

Long-term
storage
(25+ Yrs)

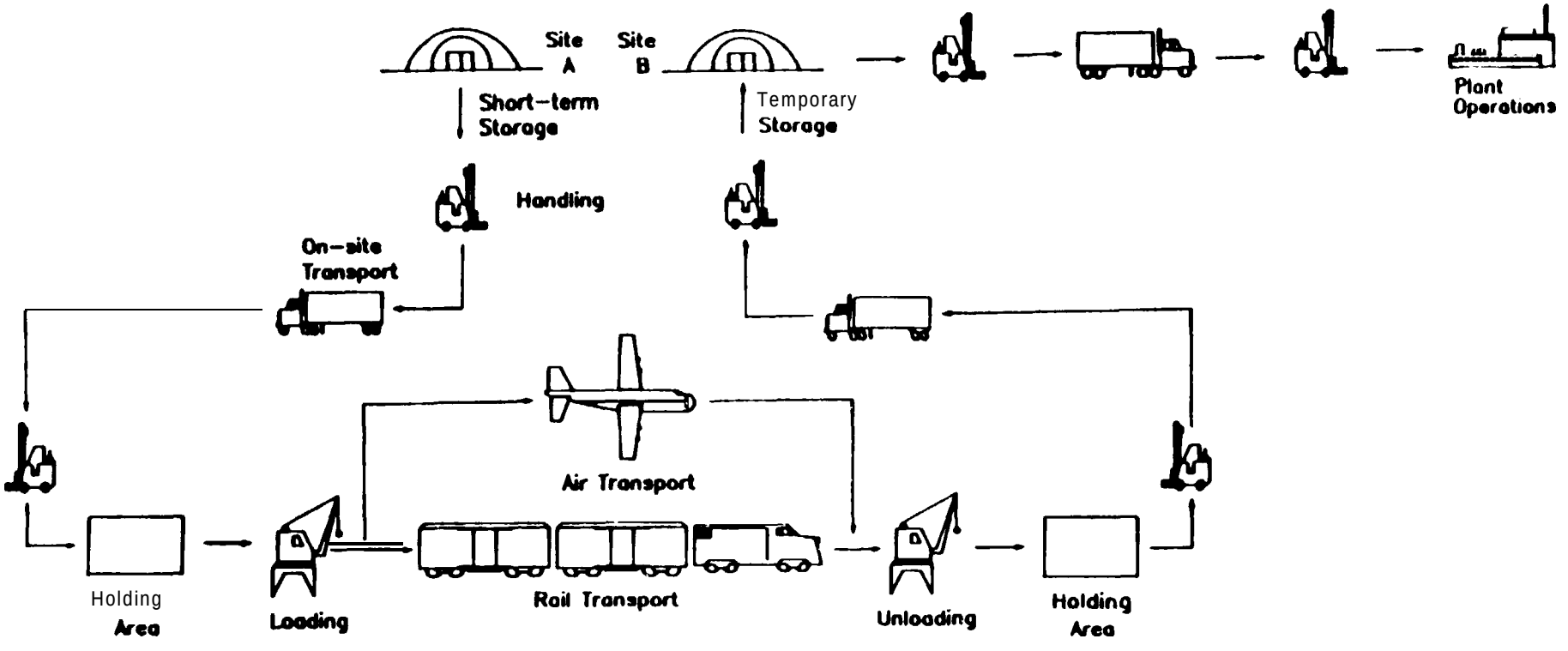
Continued Storage Alternative



On-site Disposal

FIG. J.I

DISPOSAL ACTIVITIES INVOLVING SOME RISK



Disposal Alternatives Involving Transportation

FIG. J.I (Concluded)

DISPOSAL ACTIVITIES INVOLVING SOME RISK

For all the disposal alternatives, the risk of continued storage remains until the local stockpiles have been destroyed. However, this risk contribution is not included as an element of risk for the alternatives involving demilitarization of the stockpiles, because it is common to each of them, and would not help to differentiate among them.

J.1.2.3 Agents and Munition Types

Each of the disposal alternatives involves the full range of chemical agent and munition types in the chemical stockpile. The characteristics of each are accounted for in the risk analysis. Risk associated with each of the agent types is different, since their physical and toxicological properties differ. Physical properties of greatest importance in estimating risk as a function of agent type include: vapor pressure (determines the rapidity with which spilled agent might evaporate); freezing point; and molecular weight. These and other physical properties, as well as toxicological characteristics, are encoded into the Army's D2PC computer model for chemical hazard prediction (C.G. Whitacre et al., 1987), which provides estimates of the downwind distance the chemical hazard might extend in a particular accident. Use of the model in this risk analysis is described in Fraize et al., 1987[a]

The munition types included in the stockpile are described in Appendix A of this FPEIS. Major munition characteristics accounted for in the risk analysis include: munition size and agent inventory; susceptibility to failure by puncture, crush, fire or impact; packing density; and presence of energetic materials (bursters, fuzes, and propellants).

J.1.2.4 Accident Twes

Potential chemical accidents are defined in specific accident scenarios, which are sequences of possible events leading to a release of agent. Accident scenarios for which lethal exposures would not be experienced beyond 0.5 km from the storage and disposal sites under worst-case meteorological conditions have been excluded. Accident scenarios have been identified for major classes of accident causes, including natural phenomena (e.g., wind, flood, lightning), other external events (e.g., aircraft crash), equipment failures (e.g., pipe rupture, control system breakdown), and human error. The scenarios are presented in Section 5.4 of this appendix.

The threat of sabotage is being addressed elsewhere and is omitted from the accident scenario data base considered here.

J.1.3 Prior Studies

This risk analysis is founded on a number of prior hazard and risk analyses. Quantitative hazard analyses were performed on the proposed disposal of M55 rockets utilizing a technique known as hazard and operability analysis (HAZOP) (Arthur D. Little, 1985a, 1985b, 1985c, 1985d). Qualitative analyses of the Johnston Atoll Chemical Agent Disposal System (JACADS), using a failure mode and effects analysis (FMEA) method were carried out by the R.M. Parsons Company (1983, 1985).

Deductive system logic models, such as fault trees, were used to assess the probability of agent release in off-site transportation accidents (Rhyne, 1985a, 1985b). Rhyne's study incorporated the transportation accident data base prepared by Sandia National Laboratories (Clark et al., 1976). An analysis of disposal of M55 rockets by Science Applications International Corporation (1985) focused on the storage, handling and on-site transportation of chemical munitions, using both the event tree and fault tree methodologies.

For the draft programmatic environmental impact statement (DPEIS) for the CSDP (U.S. Army Program Manager for Chemical Demilitarization, 1986), the MITRE Corporation outlined an approach for using the risk data prepared in support of the M55 rocket disposal program as the basis of an accident scenario data base applicable to the entire stockpile (Fraize et al., 1987b). MITRE then identified gaps in the accident scenario data base (subsequently addressed by GA Technologies), and proceeded to develop a framework for analyzing the risk associated with this resulting accident scenario data base and identifying representative worst case accidents for the CSDP/EIS. This framework and the preliminary accident scenario data base, as updated and completed by GA Technologies, was used to prepare the risk analysis supporting the DPELS (U.S. Army Program Manager for Chemical Demilitarization, 1986).

J.1.4 Data Sources for This Analysis

With the studies listed above as the starting point, GA Technologies (GA Technologies, 1987a, 1987b, 1987c), with technical assistance from H&R Technical Associates, JBF Associates, and Battelle-Columbus Laboratories, conducted a comprehensive assessment of accident probabilities for all munition types. Event and fault tree analyses, together with information on mechanical and thermal failure threshold conditions for each munition type, were used to estimate the probability of agent release in each of nearly 3000 potential accidents, and the amount of agent that could be released.

Downwind dispersion of lethal plumes was determined by a method incorporated in the D2PC plume dispersion model developed by the Army

(Whitacre *et al.*, 1987). Demographic data and potential fatality estimates for generic accidents (defined by lethal plume length and meteorological conditions) for all sites and transportation corridors were provided by Oak Ridge National Laboratory. An overview of the approach used for assimilating the probability and consequence information is depicted in Figure 5.2. The ovals on the left edge of Figure 5.2 represent the four major data inputs to the risk analysis:

- probability data (from GA Technologies)
- agent release data (from GA Technologies)
- meteorological data (from ORNL)
- fatality data (from ORNL)

These four major data sets are then integrated in ways that represent the disposal alternatives defined by the Army to yield measures of risk. More detail on the risk data integration process is presented in Fraize *et al.*, 1987[a]

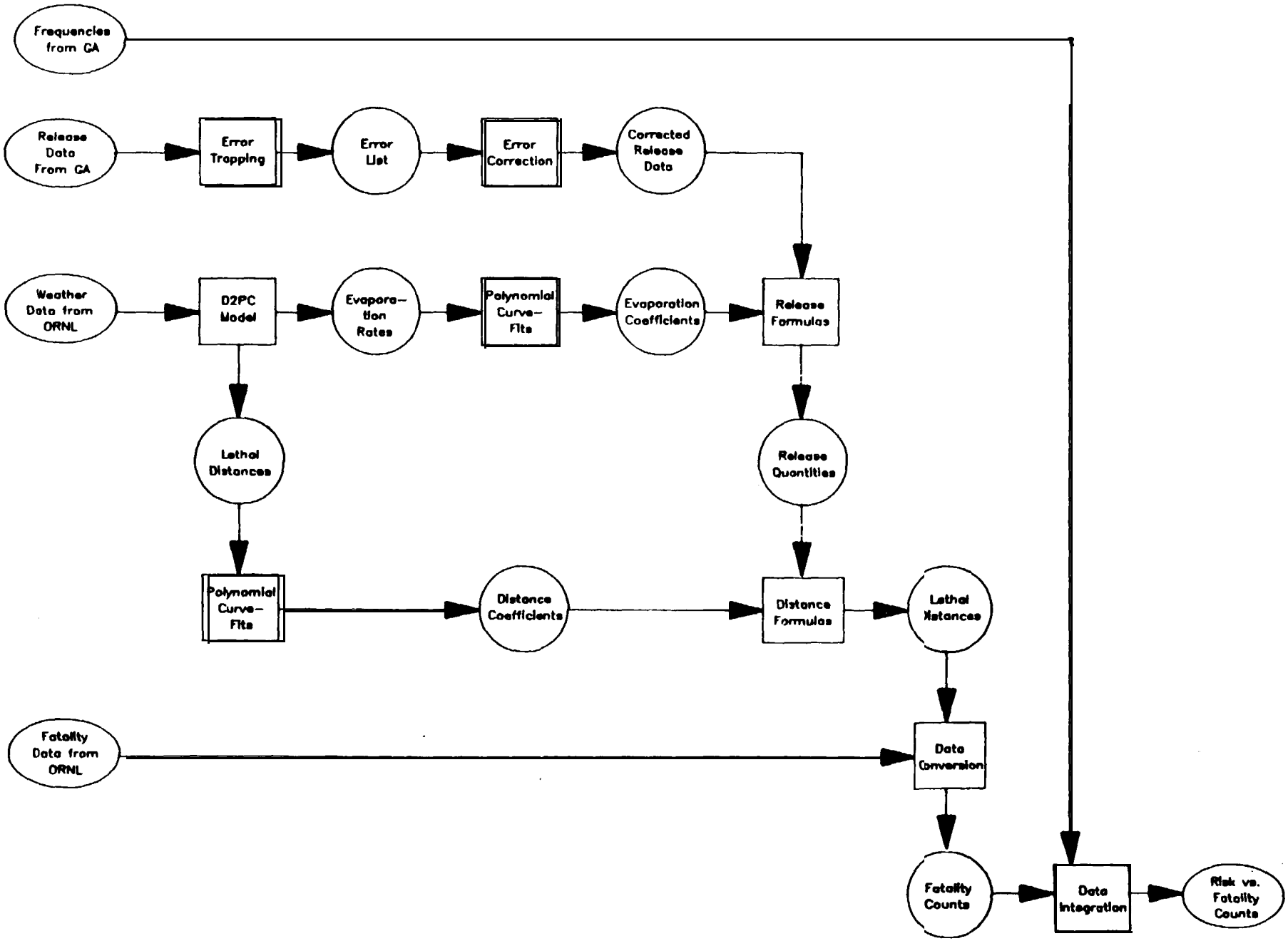


FIG. J.2

SCHEMATIC DIAGRAM OF RISK DATA PROCESSING

J.2.0 METHODOLOGY OF THE RISK ANALYSIS

The purpose of this section is to present basic principles involved in estimating risk to the public, and to show how these principles have been applied to the Chemical Stockpile Disposal Program (CSDP).

J.2.1 Introduction to Risk Assessment Concepts

Risk is a measure of the potential for exposure to unwanted events or consequences (e.g., injuries or fatalities). Any danger to the public associated with the proposed Chemical Stockpile Disposal Program may be described in *terms* of risk. For purposes of this study, risk is considered to be that due only to accidental release of, and potential public exposure to, chemical agent. *Only* accidents that could result in a release of agent sufficient to expose the public to potentially lethal doses are included. For purposes of this study, the term "public" excludes persons within the boundaries of the military installations.

The risk associated with any activity (e.g., living near a geologic fault, driving a car, riding a roller-coaster, or living under an airplane flight path) may be described as the product of two quantities: the probability of the unwanted event occurring and the conseauence to an individual or the public, if the event does occur.

The probability of a potential accident is a quantitative statement of the "odds" of that accident occurring, given many repetitions of the activity or condition that can lead to the accident. For instance, analysis of the accident and all of the separate events leading up to it might show that the odds of the accident occurring at some time during the CSDP might be 1 in 200,000; we can express the probability of that event occurring in just that way -- 1 in 200,000 -- or in the following equivalent ways: 0.000005; l/200,000; or, in scientific notation, 5×10^{-6}. For this analysis, the probability of an accident is expressed as the likelihood (or "odds") of its occurring once during the stockpile disposal program. The only exception is for long-term storage accidents where probability has been expressed as the likelihood of occurrence during a 25-year period (the assumed duration of the "no-action" alternative).

The **consequence** of a potential accident can be expressed in several ways, depending on the intended use of the results. For the purposes of the CSDP risk analysis, there are two principal measures of the consequence of any given accident:

- **size** of the lethal plume produced by the accident. Size of the lethal plume is defined as the distance to the downwind location where the "exposure" (the product of agent concentration and time) is equal to the estimated minimum lethal value. This distance is also referred to as the "no-deaths" hazard distance. Plume size,

or downwind hazard distance, is dependent on the agent type (physical characteristics), agent quantity released and the meteorological conditions governing the atmospheric dispersion of the agent.

- potential fatalities per event. This measure is the most direct indicator of potential accident consequences to the population. Estimation of potential fatalities requires knowledge of the source term (quantity and mode of agent release), the atmospheric dispersion mechanism (specified by local meteorological conditions), the population distribution (by distance and direction), and the estimated human response to chemical agent exposure.

The present risk analysis is limited to airborne release of agent. Other modes for dispersion of released agent, such as through ground water or surface water, are beyond the scope of this analysis. Only acute and lethal toxicity are considered in the analysis; chronic and sub-lethal effects are not evaluated. (See Appendix B of this FPEIS for details.)

J.2.2 Application of Risk Concerts to the CSDP

The factors defining risk to people in the vicinity of a stockpile or transportation corridor include the following:

- the probability that an accidental release will occur;
- the probability (along transportation corridors) that a transport vehicle will be in the vicinity of the individual when the accident occurs;
- the distribution of people downwind of the release;
- the probability of being within the plume width; and
- the probability that individuals within each lethality zone of the plume will die.

Risk to the public was calculated for each accident by overlaying the lethal plume (under "most-likely" weather conditions) associated with the accident on a map of the residential population about the site *or* adjacent to a transportation corridor and estimating the number of potential fatalities within the plume. Next, expected fatalities from each accident were computed as the product of potential fatalities and the probability of the accident occurring. The total population risk was then determined by summing expected fatalities for all applicable accidents.

This concept is illustrated by Figure 5.3. The concentric arcs in the figure represent hazard distance zones from the potential accident site. For example, the distance zones used in this analysis are the following:

>0.0 - 0.1 km	> 2 - 5 km
>0.1 - 0.2 km	> 5 - 10 km
>0.2 - 0.5 km	> 10 - 20 km
>0.5 - 1.0 km	> 20 - 50 km
>1 - 2 km	> 50 - 100 km

An accident having a "no-deaths" plume length of 12 km is assumed *to* result in a fatality count for the zone which is 10 - 20 km from the accident site. If an accident causes a plume that reaches into the 10 - 20 km population zone, then all those in the inner population rings, closer to the agent source, are at even more risk since the dosages become higher as one approaches the accident site. Similarly, within a given distance zone, individuals will be affected not only by those scenarios for which the plume just reaches their zone, but also those accidents of greater magnitude for which the plume reaches into the outer zones. *While* plume lengths exceeding 100 km may be estimated in the D2PC model for the worst of the potential accidents, a correction has been made to exclude fatalities that would occur farther than 100 km from the potential accident location. (Such travel distances are unrealistic; see Appendix K of this FPEIS for an explanation.)

J.2.3 Principal Measures of Risk

To compare the public risks of the disposal alternatives, the following measures, each of which provides a different perspective on program risk, are used:

- probability of one or more fatalities, a public risk indicator equal to the chance that there will be at least one fatality at a given site or for the nation as a whole during the CSDP. This measure is calculated by summing the probabilities of all accidents that could cause one or more fatalities. Included in this sum are all accidents for which the potential fatality estimate, based on assuming uniform population densities, is less than unity. (This means that that accident is expected to cause a fatality for only a fraction of the times it occurs; for the remaining fraction of occurrences, that event would not cause a fatality. For such accidents, the probability of occurrence is reduced so that only the fraction of events expected to cause a fatality are counted);

- maximum number of fatalities, equal to the maximum consequence of all accidents at a site or for the nation, This risk measure is

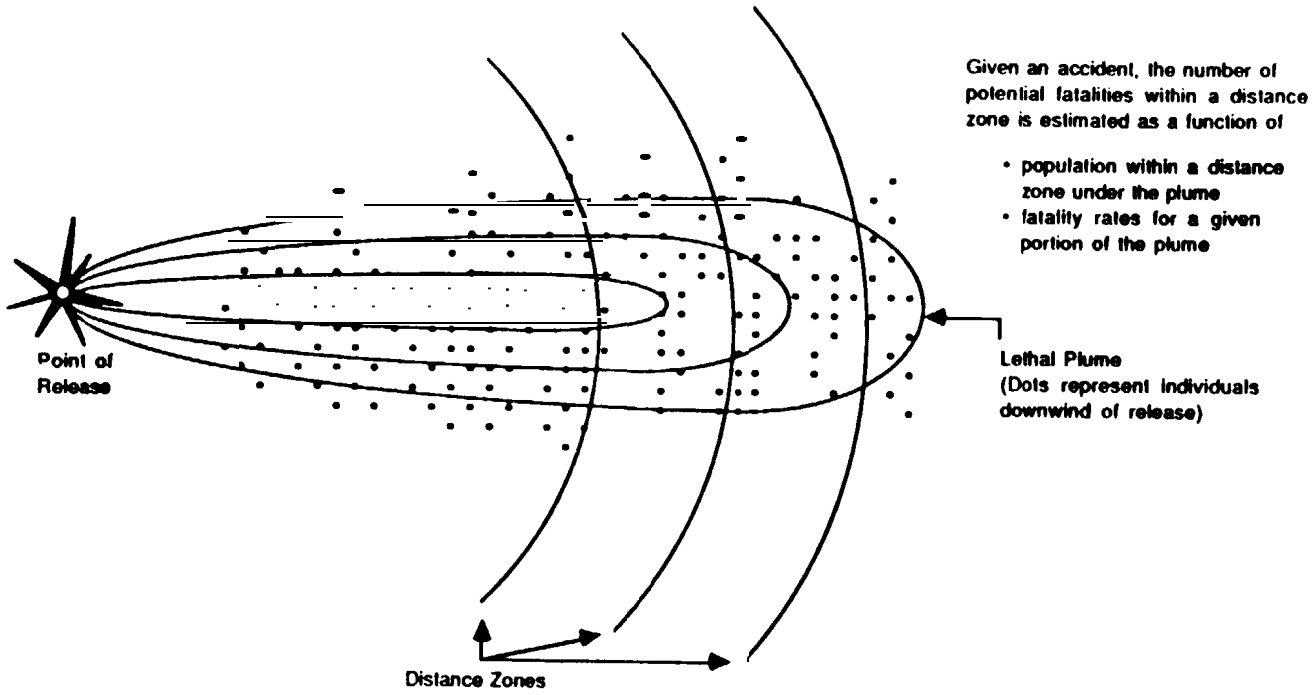


FIG. J.3

ILLUSTRATION OF POPULATION RISK

based on worst-case weather conditions, actual population densities (1980 census data, as analyzed by Oak Ridge National Laboratories), and worst possible wind direction (i.e., plume striking the highest number of people without any allowance for preventive/emergency response measures);

- expected fatalities, equal to the sum of the risk contribution of all accidents at a site or for the nation, where risk for each accident is the potential fatality count (if the accident were to occur) multiplied by the probability of the accident occurring. Note that expected fatalities is proportional to the probability of a fatality-causing event occurring, and will nearly always be a small number -- well less than unity. For example, an accident with a potential fatality estimate of 12 and a probability of 10^{-6} (odds of 1 in a million of occurring during the CSDP) would have an expected fatality value of 12×10^{-6}. At the programmatic level, the expected fatalities value is the sum of the expected fatality contribution of several hundreds of potential events and might lie somewhere in the range of 10^{-3};

- person-years-at-risk, equal to the population living within all zones (defined in Section 2.2.2) that could experience potentially lethal agent exposure multiplied by the time period over which that worst-case event could take place (typically, the duration of disposal operations at fixed sites or the time during which transport vehicles might be within lethal plume reach of population groups along the corridors). This measure does not account for the fact that individuals within the affected population groups who are farther from the potential accident site are at lower risk of suffering ill effects of exposure; all affected individuals are counted if they have any risk at all. (This measure is discussed in more detail in Section J.3.3.3.2.); and

- maximum total time at risk, representing the maximum length of time an individual could be at risk at a fixed location near a site or along a transportation corridor. For those living within a radius equal to or less than the maximum lethal hazard distance, the time at risk is the total time for stockpile disposal at that site regardless of where the individual is located. For those individuals along the transportation corridors, the time depends on the distance from the rail line or air corridor. For those individuals the maximum time is assumed to occur if the individual is located at a 0.1 km distance from the rail track or centerline of the air corridor. These persons are exposed to a hazard only when a train or aircraft is in the vicinity (defined as the maximum lethal hazard distance in either direction) of them. This time is summed for each agent-bearing train *or* aircraft that would pass by in each

alternative. Since maximum lethal hazard distance is used in this determination, the worst case meteorological conditions apply.

J.2.4 Methods for Portraying Risk

Estimates of risk to the population can be displayed in a variety of ways. Those used in this report are illustrated by Figure 5.4:

Item A. Risk curves, which portray, for the full set of applicable accident scenarios:

- the probability of exceeding a given number of potential fatalities per event (vertical axis), against
- the potential fatalities per event (horizontal axis);
- the upper and lower bound estimates, as well as the mean (average) value, reflecting the uncertainty in the probability component of the risk curve -- the uncertainty range defining the 90% confidence limits; and
- the maximum potential fatalities, assuming worst-case meteorology, distributed population, and worst possible wind direction, shown as a dashed vertical line.

Risk curves depict the difference between alternatives dominated by high-probability/low-consequence accidents and those dominated by low-probability/high-consequence accidents.

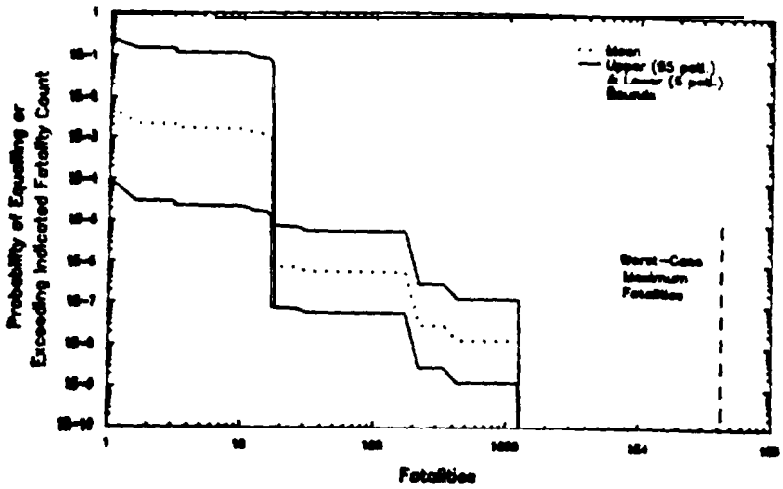
Item B. Risk pictograms, which display:

- a pictorial indicator (the darkness of the shading) of the relative magnitude of each of the measures of risk chosen for this analysis;
- a key to the numerical range represented by each of the shading values; and
- an array of data allowing comparison of risk at all sites for a given disposal alternative or, alternatively, comparison among alternatives for a given site (both approaches are used in this report).

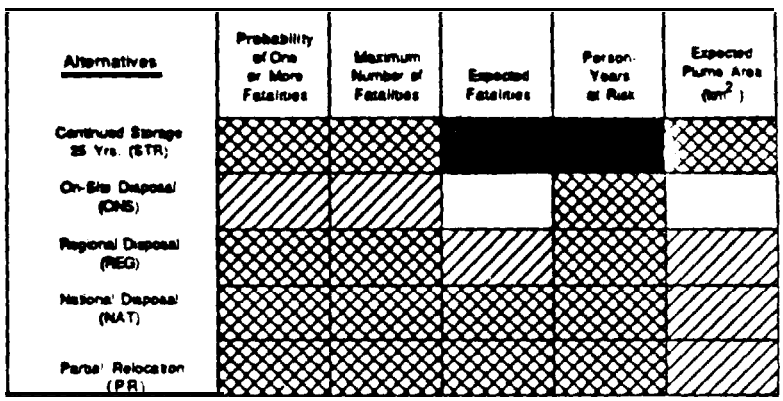
Risk pictograms provide a visual impression of the relative magnitude of public risk for all combinations of alternatives and locations.

Item C. Expected fatalities plots showing mean estimated values of expected fatalities, with uncertainty bands. The expected fatalities

A. RISK CURVE



B. RISK PICTOGRAM



C. EXPECTED FATALITIES PLOT

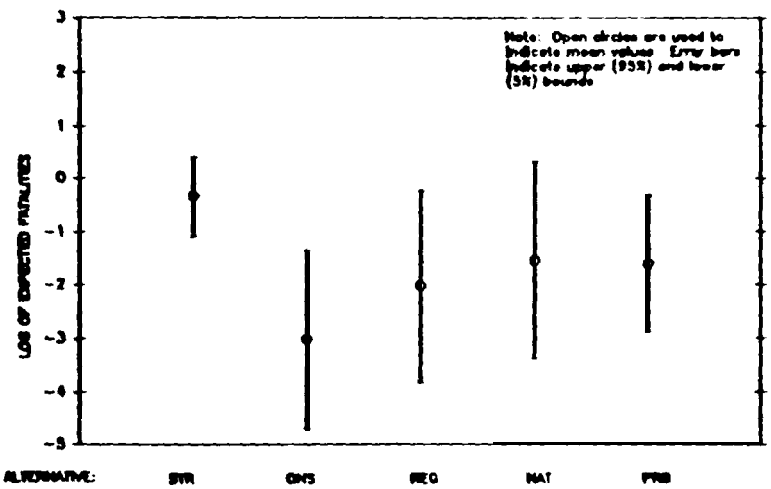


FIG. J.4

PORTRAYAL OF PUBLIC RISK (3 EXAMPLES)

value is defined as the sum of the risk (probability times potential fatalities) for all applicable accidents. While this measure of risk is convenient and consistent, permitting the summing and disaggregation of the contributions to CSDP risk, it provides the least information of any of the risk measures. For example, it does not clearly show the relative contributions of low consequence/high probability accidents and high consequence/low probability accidents, which is often of great interest to the public. As illustrated in Figure 3.4, expected fatality data are presented in this report with error bars indicating the estimated uncertainty in the calculated value. The extremes of the error bars represent the 90% confidence limits -- that is, there is only a 10% probability that the actual expected fatalities value would fall outside the indicated range.

J.3.0 PRESENTATION AND DISCUSSION OF RESULTS

The detailed results of the risk analysis, describing the consequences, probability, and all relevant risk parameters for each identified accident for all locations and disposal alternatives, are presented in the MITRE report (Fraize, et al., 1987a). In this section, we will present a summary of that risk data and will discuss the significance of the results from both programmatic (all locations combined, for each disposal alternative) and location-specific considerations.

Unless stated otherwise, the term risk will refer to expected fatalities, while plume length will mean the 'no-deaths' hazard distance under most-likely meteorological conditions.

J.3.1 Overview of Risk Data

The risk data contained in the MITRE report (Fraize et al., 1987a) include, for each identified potential accident, all the information necessary to determine the major risk parameters identified in section 2.2.4: probability of one or more fatalities; maximum number of fatalities; expected fatalities; and, person-years-at-risk (derived from estimated time-at-risk). Expected plume area is also presented as a measure of ecological risk, Each of these parameters is relevant only to a set of potential accidents which could take place at a specified location (fixed site or along a transportation corridor) for a given site-specific stockpile (identified by its originating site) and a given disposal alternative. Thus, throughout this section, we will refer to the following three descriptors of the accident scenario set of interest:

- Disposal Alternative (see below)
- Site-Stockpile (see below)
- Location/Locale of Risk as defined by:
  - OS = Originating Site
  - DS = Destination Site
  - TC = Transportation Corridor

For programmatic risk portrayal, all three locales are combined. For site- or location-specific risk portrayal, the risk at only one locale is shown.

J.3.1.1 Disposal Alternatives and Site-Stockpiles Considered

The eight site-stockpiles considered in this analysis are identified in the main body of this document. The codes used throughout this analysis to signify particular sites are tabulated below:

G = APG = Aberdeen Proving Ground, MD
L = LBAD = Lexington-Blue Grass Army Depot, KY
B = PBA = Pine Bluff Arsenal, AR
N = NAAP = Newport Army Ammunition Plant, IN
P = PUDA = Pueblo Depot Activity, CO
U = UMDA = Umatilla Depot Activity, OR
A = ANAD = Anniston Army Depot, AL
T = TEAD = Tooele Army Depot, UT

The five alternatives analyzed in the Final Programmatic Environmental Impact Statement (FPEIS), their one-line descriptions, and the codes used to represent them in the analysis and in the presentation of the results are given below:

S = STR = Continued Storage (for 25 years)
0 = ONS = On-Site Disposal
R = REG = Regional Disposal (via rail)
N = NAT = National Disposal (via rail)
B = PRB = Partial Relocation -- On-Site Disposal, except APG & LBAD Stockpiles to TEAD via Air (Cl41 aircraft) [Note: This alternative is also referenced by the code PR in some sections of this appendix and in the body of the FPEIS.]

J.3.1.2 Treatment of Mitieation

The accident scenario data base was analyzed for the unmitigated case plus two levels of mitigation, the details for which are described in a separate Army report (PEO-PM Cml Demil, 1987a) and elsewhere in the FPEIS. The three levels are:

1. Unmitieated. The accident scenarios as defined and characterized by GA Technologies (1987a,b,c) were used.

2. Mitigated, Revision 1. The unmitigated accident scenario data base was modified by the following measures which are expected to significantly reduce the effects of high risk scenarios identified by analysis of the unmitigated data base; expected benefits for each mitigation measure are also indicated:

   - Reduce time required to clean up spill to under 15 minutes by applying foam or other material to a spill occurring during handling or on-site transportation.

     Expected Benefit: Spill duration reduced by 75 percent (handling accidents) or 87 percent (on-site transportation accidents).

- Use battery-powered lifting devices for handling.

  Expected Benefit: 99 percent reduction in probability.

- Install blunt bumpers on lift truck tines.

  Expected Benefit: 65 percent reduction in probability.

- Use improved mobile device to control vehicle fire during on-site transportation.

  Expected Benefit: less than 1 percent reduction in overall probability. Significant benefit in certain accidents included in this category.

- Install seismic-actuated gas cut-off valves and category 3 breakers in the munition demilitarization building (MDB).

  Expected Benefit: Probabilities for earthquake-caused scenarios, plant operations accidents PO 26 and PO 29 (see section 5.4 for descriptions of accident scenarios), are reduced by 80 to 88 percent, depending on the site. Probability of plant operations accident PO 33, also an earthquake scenario, is reduced by 90 percent.

- Install a metal shield over the conveyor at the explosive containment vestibule (ECV).

  Expected Benefit: 99 percent reduction in probability for the munition detonation scenarios, plant operations accidents PO 46 and PO 47.

- Implement changes in the unpack area (UPA) to prevent mines and rockets from being inadvertently conveyed to the dunnage furnace (DUN). All measures necessary to reduce by a factor of 100 the probability of a munition reaching the DUN will be implemented. The measures under consideration are the following:

  - interlock mine and explosive counters at the mine glovebox with the dunnage conveyor using redundant sensors and positive shut-off.

  - independently interlock a drum weighing device at the mine glovebox to the dunnage conveyor.

  - interlock a metal detector with the DUN airlock to preclude transporting a rocket to the DUN.

- inspect the dunnage for munitions.

- replace manual handling of rockets by using a mechanical hoist to lift rockets from the pallet.

  Expected Benefit: 99 percent reduction in probability for the dunnage furnace accident scenario, PO___ 52.

- Transport mustard ton containers in frozen state.

  Expected Benefit: 94 percent reduction in amount evaporated for rail transport accidents involving a spill of mustard agent; 85 percent reduction for the corresponding air transport accidents. [A detailed description of the benefits of low temperature transport of mustard agent may be found in a separate Army report (PEO-PM Cml Demil, 1987[b])]

- Deenergize warehouse electrical system using seismically-actuated circuit breakers or disconnected electrical leads.

  Expected Benefit: 98 percent reduction in probability of a warehouse fire caused by an earthquake.

3. Mitinated. Revision 2. The accident scenario data base, as mitigated by the measures described above (mitigation, Revision 1), was further mitigated by the following additional measure:

   - Restrict air space at all of the *sites* and eliminate military helicopter flights.

     Expected Benefit: 100 percent reduction in probability of air crash accidents of military helicopters, and 92 percent reduction in all other crashes.

J.3.1.3 Treatment of Uncertainty

Uncertainties in risk estimation arise due to many causes, including the inadequacy of data, inaccuracies in modeling, and the incomplete identification and understanding of accident phenomena. The basis for estimating uncertainties when summing probabilities or probability-weighted data with known individual uncertainties is described in Fraize et al., 1987[a].

The analysis of accident scenarios carried out by GA Technologies provides an *error* factor for each accident probability "point estimate". This error factor was used to characterize the uncertainty inherent in each estimate. The contribution *to* risk uncertainty of consequence estimation

(for example, in estimating potential public fatalities as a result of an agent release) is represented separately (though incompletely) by considering most likely and worst case meteorological conditions. However, since worst case conditions occur relatively rarely and have greater consequences, they may have little effect on a risk curve.

In this report, uncertainty is portrayed on the risk curves and on the expected fatality plots where upper and lower uncertainty bounds (at the 95 percent and 5 percent levels) are indicated.

J.3.1.4 Description of Data

Risk data are summarized in several forms. In section J.3.2, risk data for the programmatic level (no location-specific information) are presented for the unmitigated case plus two levels of mitigation in three forms:

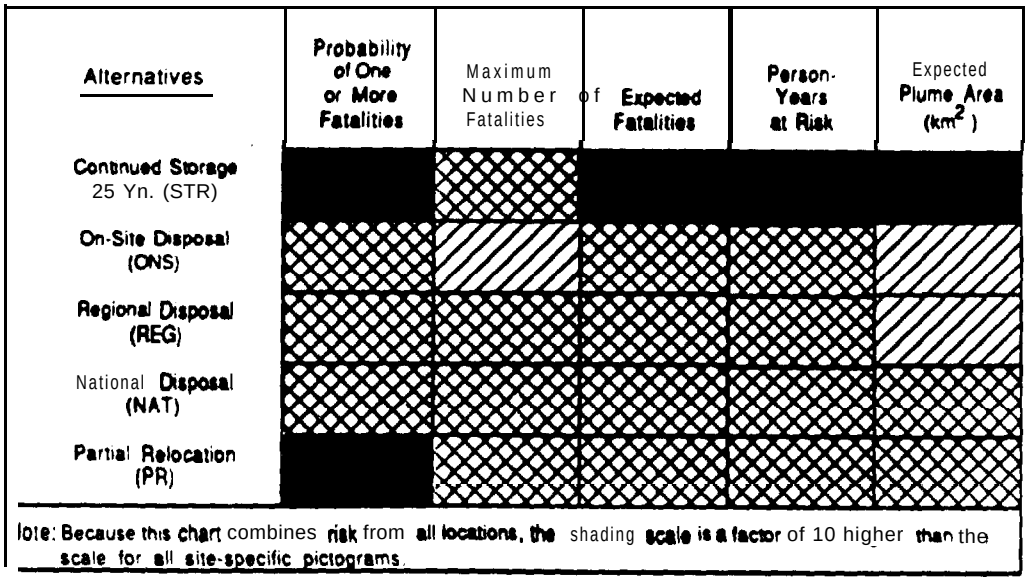
- semi-quantitative, graphical/pictorial comparisons of major risk parameters in 'pictograms'.
- graphical comparisons of expected fatality estimates, with upper and lower uncertainty bounds; and
- cumulative risk curves, with upper and lower uncertainty bounds.

In section J.3.3, location-specific risk data are presented, but in 'pictogram' form; location-specific risk curves are not presented because they could reveal classified information.

J.3.2 Programmatic Risk of Alternatives

J.3.2.1 General Comparison

Figures J.5 through 5.7 display in pictogram format (using matrix elements shaded according to four numerical equivalence ranges) the four major risk measures plus a fifth measure -- the expected value of plume area (requested by ORNL for the purpose of evaluating ecological risk). The shadings are chosen so that higher risk is connoted by darker shading. The numerical ranges were chosen so that the full range of values for all alternatives could be displayed and readily differentiated. The shading assigned to any entry in the pictograms is strictly defined by the mean value of the risk measure relative to the numerical boundaries of the ranges. Note that the numerical equivalence scale chosen for this programmatic chart (involving the larger values associated with the summation of risk at individual locations) is higher (by one order of



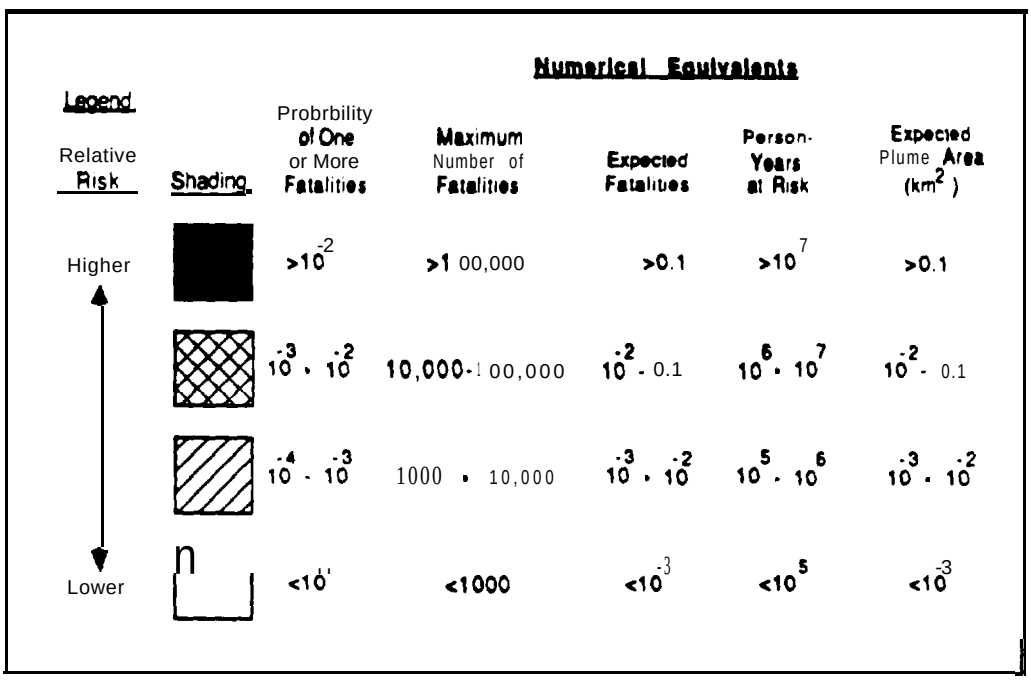
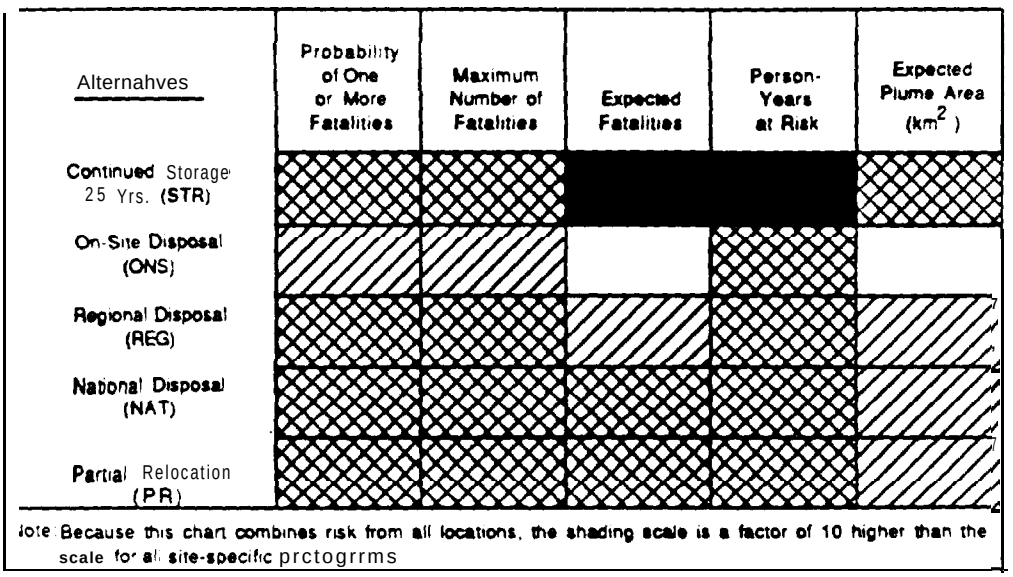


FIG J.5

RISK COMPARISON FOR PROGRAMMATIC ALTERNATIVES ALL LOCATIONS COMBINED



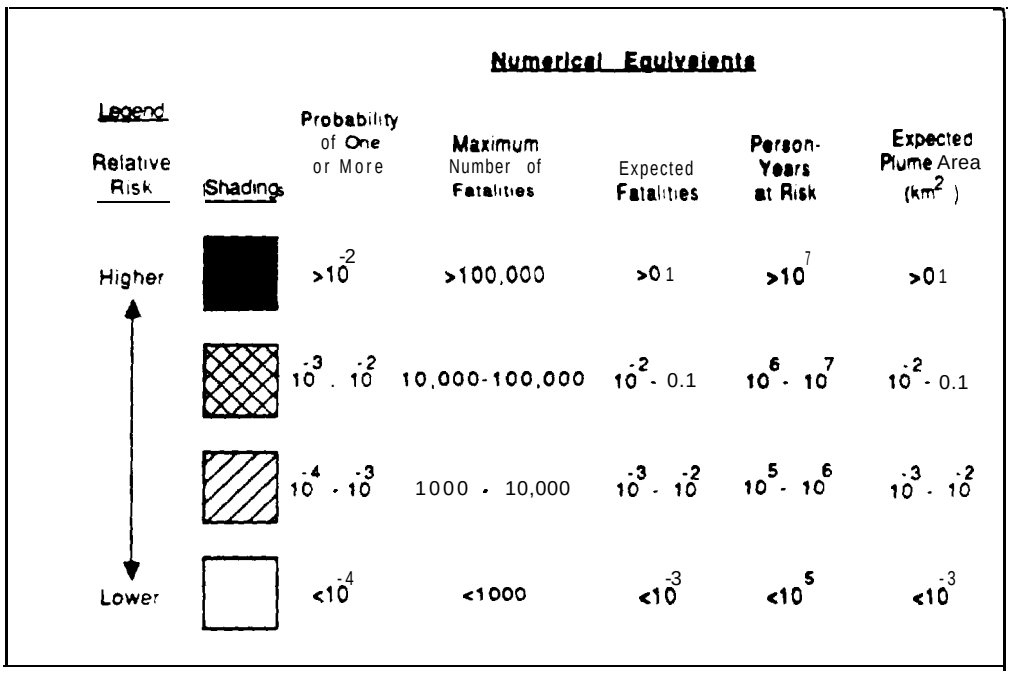
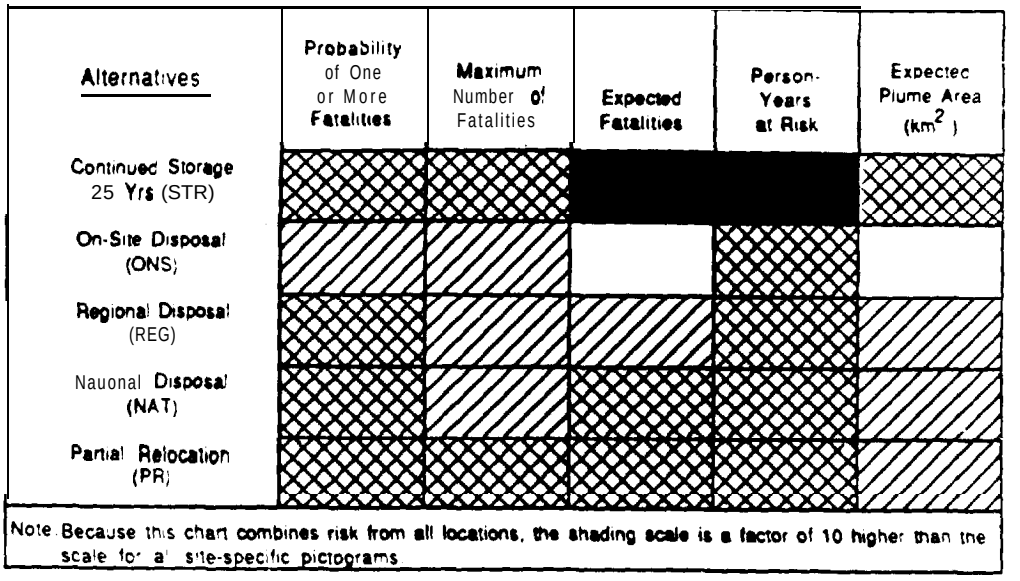


FIG. J.6

RISK WITH MITIGATION (REV. 1):
COMPARISON FOR PROGRAMMATIC ALTERNATIVES
ALL LOCATIONS COMBINED



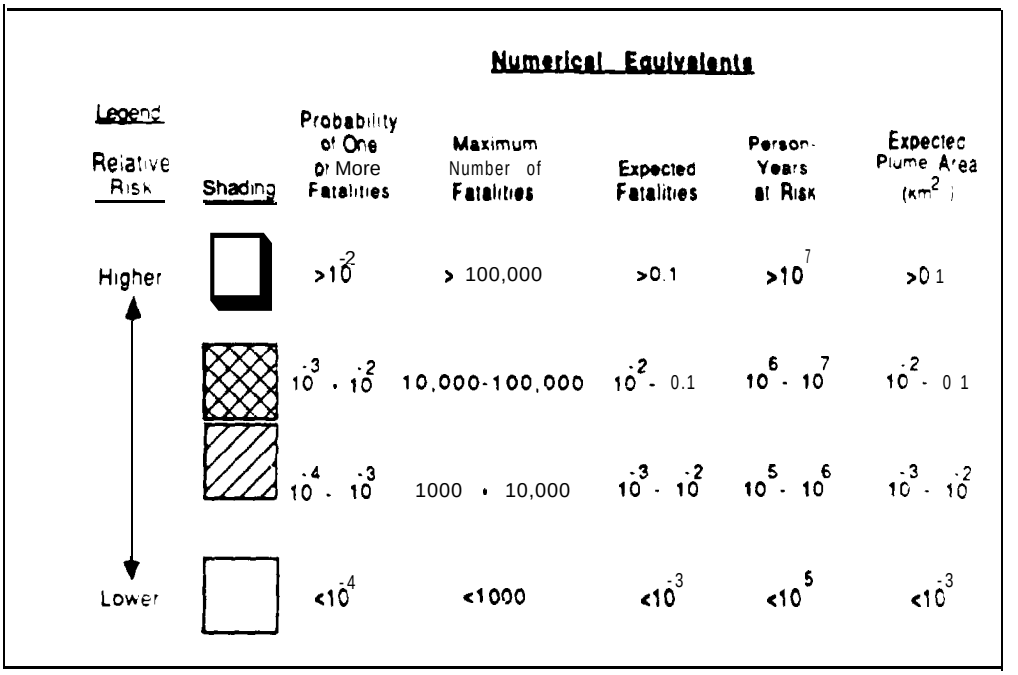


FIG. 5.7

RISK WITH MITIGATION (REV. 2):
COMPARISON FOR PROGRAMMATIC ALTERNATIVES
ALL LOCATIONS COMBINED

magnitude or distance category) than the numerical equivalence scale for the location-specific 'pictograms' displayed in Section 3.3

Discussion of comparative risks, as presented in this section, are based on reference to the actual data from the risk analysis; the quantitative comparisons can not be derived from the 'pictograms'. To support the programmatic (i.e., not location-specific) risk comparisons in this subsection, actual values for the risk measures for the five alternatives are presented in Table J.l; data are provided for the unmitigated case and for the two levels of mitigation.

Considering the case of unmitigated accident scenarios first, the continued storage alternative has the greatest probability of causing one or more fatalities of the five alternatives. The remaining four alternatives have approximately a factor of 10 lower probability of causing one or more fatalities.

The maximum number of fatalities of the five alternatives ranges between approximately 5,000 and 90,000, with continued storage having the greatest number and on-site disposal having the least. Continued storage has 16 times more maximum fatalities than on-site disposal; the national or regional alternatives have 7 times more than the on-site disposal alternative and the partial relocation alternative has 4 times more than the on-site disposal alternative.

The continued storage alternative has the greatest expected fatalities in the unmitigated case, while the on-site disposal and regional disposal alternatives have the least. The value of expected fatalities for the partial relocation and national disposal alternatives is approximately three times that for the on-site disposal alternative, while the value for the continued storage alternative (25 years) is approximately 1,900 times greater than for the on-site disposal alternative. This significant difference is not displayed on the pictogram, since the darkest shading category, in which continued storage falls, is unbounded on the higher end.

The difference between the expected fatalities for the continued storage alternative and the other four alternatives is more precisely shown in a plot of expected fatality estimates. Figure J.8 portrays the expected fatality estimates, with upper and lower bounds, for each of the five unmitigated alternatives. The data show the dominance of expected fatalities associated with 25 years of continuing storage over expected fatalities associated with any of the four disposal alternatives. Continued storage shows a mean expected fatality value of approximately 20, indicating that the analysis predicts a public fatality rate averaging roughly one per year. The fact that no deaths to the public have occurred after decades of storage does not mean the analysis is greatly in error or unduly conservative, rather, it is because the continued storage accidents

TABLE J.I
QUANTITATIVE COMPARISON OF RISK MEASURES FOR PROGRAMMATIC ALTERNATIVES
-ALL LOCATIONS **COMBINED-**

A: Unmitigated Risk

Alternatives	Probability of One or More Fatalities	Maximum Number of Fatalities	Expected Fatalities	Person-Years at Risk	Expected Plume Area (km^2)
Continued Storage 25 Yrs. (STR)	6.4×10^{-2}	8.9×10^{4}	19a	1.4×10^{8}	2.1
On-Site Disposal (ONS)	7.3×10^{-3}	5.4×10^{3}	1.0×10^{-2}	2.3×10^{6}	7.2×10^{-1}
Regional Disposal (REG)	$4. \times 10^{-3}$	4.2×10^{4}	1.2×10^{-2}	5.5×10^{6}	9.3×10^{-1}
National Disposal (NAT)	51×10^{-3}	4.2×10^{4}	3.1×10^{-2}	5.4×10^{6}	1.2×10^{-2}
Partial Relocation (PR)	1.1×10^{-2}	2.3×10^{4}	3.4×10^{-2}	3.1×10^{6}	1.1×10^{-2}

B Risk with Mitigation (Rev. 1)

Alternatives	Probability of One or More Fatalities	Maximum Number of Fatalities	Expected Fatalities	Person-Years at Risk	Expected Plume Area (km^2)
Continued Storage 25 Yrs. (STR)	2.4×10^{-3}	8.9×10^{4}	4.5×10^{-1}	1.4×10^{8}	4.4×10^{-2}
On-Site Disposal (ONS)	3.2×10^{-4}	5.4×10^{3}	9.4×10^{-4}	2.3×10^{6}	4.6×10^{-4}
Regional Disposal (REG)	1.8×10^{-3}	4.2×10^{4}	8.5×10^{-3}	5.5×10^{6}	2.0×10^{-3}
National Disposal (NAT)	3.4×10^{-3}	4.2×10^{4}	$a.0 \times 10^{-2}$	5.4×10^{6}	3.8×10^{-3}
Partial Relocation (PR)	3.7×10^{-3}	2.3×10^{4}	2.5×10^{-2}	3.1×10^{6}	6.6×10^{-3}

C Risk with Mitigation (Rev. 2--with Air-Space Restrictions)

Alternatives	Probability of One or More Fatalities	Maximum Number of Fatalities	Expected Fatalities	Person-Years at Risk	Expected Plume Area (km^2)
Continued Storage 25 Yrs. (STR)	1.3×10^{-3}	8.9×10^{4}	3.8×10^{-1}	8.9×10^{7}	3.7×10^{-1}
On-Site Disposal (ONS)	3.2×10^{-4}	5.4×10^{3}	9.4×10^{-4}	2.3×10^{6}	4.6×10^{-4}
Regional Disposal (REG)	1.8×10^{-3}	5.0×10^{3}	9.3×10^{-3}	1.9×10^{6}	2.0×10^{-3}
National Disposal (NAT)	3.4×10^{-3}	8.3×10^{3}	2.9×10^{-2}	1.8×10^{6}	3.8×10^{-3}
Partial Relocation (PR)	3.7×10^{-3}	2.3×10^{4}	2.5×10^{-2}	2.7×10^{6}	6.6×10^{-3}

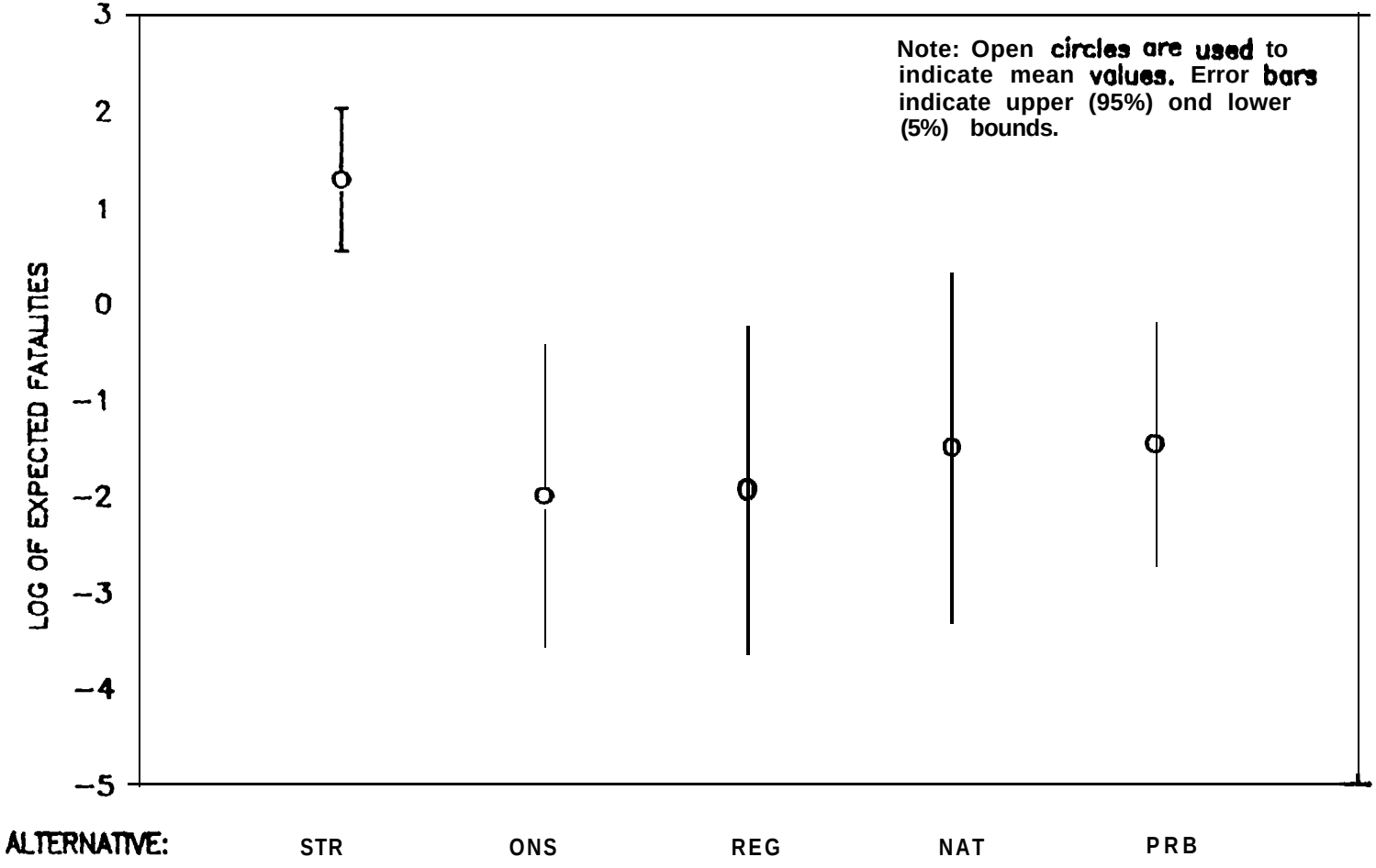


FIG. J.8

SOCIETAL RISK (EXPECTED FATALITIES)
FOR PROGRAMMATIC **ALTERNATIVES**
UNMITIGATED

are predicted to be infrequent (occurring far less frequently than 1 per year) but severe (multiple fatalitiesj.

Person-years-at-risk are approximately equal for the on-site disposal and partial relocation alternatives. The national and regional disposal alternatives have approximately five times more person-years-at-risk than the on-site disposal alternative; and the continued storage alternative has approximately 60 times more person-years-at-risk than the on-site disposal alternative.

Expected plume area is greatest for the continued storage alternative, and least for the on-site disposal alternative.

For the mitigated accident scenarios, Figures J .6 and J. 7 show that on-site disposal has the least probability of causing one or more fatalities. Compared to on-site disposal, the regional disposal alternative has approximately a 5 times greater probability of causing one or more fatalities, continued storage has approximately seven times greater probability of causing one or more fatalities, national disposal has approximately 10 times greater probability of causing one or more fatalities, and the partial relocation alternative has approximately 11 times greater probability of causing one or more fatalities.

There was no change in the number of maximum fatalities possible or the person-years-at-risk for the mitigated accident scenarios. Also, addition of airspace restriction to the mitigation measures did not reduce risk to any significant degree from a programmatic standpoint.

For the mitigated (revision 1) accident scenarios, the continued storage alternative has the greatest expected fatalities, and on-site disposal has the least expected fatalities. Compared to on-site disposal, the value for expected fatalities is approximately 10 times greater for regional disposal, 26 times greater for partial relocation, 30 times greater for national disposal, and nearly 500 times greater for the continued storage alternative.

The greatest reduction in the probability of one or more fatalities for the mitigated accident scenarios, is in the on-site disposal and continued storage alternatives, which both shoved greater than 90 percent reduction. The regional disposal and partial relocation alternatives showed approximately a 60 percent reduction in expected fatalities; and the national disposal alternative shoved approximately a 40 percent reduction in the probability of one or more fatalities.

The greatest reductions in the value of expected fatalities for the mitigated accident scenarios also occurred in the continued storage and on-site disposal alternatives, which both show greater than 90 percent reduction from the unmitigated accident scenarios. The partial relocation

alternative shows approximately a 25 percent reduction in the expected fatality value; the regional disposal alternative shows approximately 20 percent reduction in the expected fatality value; and the national disposal alternative shows approximately a 5 percent reduction in the expected fatality value.

As in the unmitigated case, the continued storage alternative has significantly more expected fatalities than all other alternatives. In addition, however, with addition of the accident mitigation measures, on-site disposal is rendered significantly less risky than the other disposal alternatives. (See Figures J.9 and J.10.)

J.3.2.2. Major Sources of Risk in Each Alternative

Figures J.11 through J.15 display the cumulative risk for the five disposal alternatives, for the unmitigated accident scenarios. Each curve shows the probability that the number of fatalities indicated by the horizontal scale is estimated to occur during the course of the entire disposal program. The intercept of the risk curve with the vertical axis, at a potential fatality of value of 1, is the probability of one or more fatalities -- one of the chosen major measures of programmatic risk. The area under each risk curve (as it would appear if plotted using linear scales -- equal increments for equal changes in the value of the plotted parameters -- instead of the logarithmic scales -- equai increments for each lo-fold multiple of the value of the plotted parameters -- necessitated by the very wide range of the risk data) is numerically equal to another of the principal risk measures -- the expected fatalities of the alternative. Finally, the horizontal intercepts (at probability = 10^{-10}) indicate the maximum fatalities that potentially could occur, although at very low probability, during the execution of the disposal alternative. The intercept for the lower bound curve indicates maximum fatalities for most-likely meteorology with wind directed at the average population density; the dashed vertical line at the right of each curve indicates the maximum fatalities for worst-case meteorology with wind directed toward the maximum potentially affected population. The latter value for maximum fatalities (i.e., worst-case conditions) is the measure represented in the 'pictograms'.

The risk curves show mean values as the dotted curve, as well as upper and lower uncertainty bounds (95 percent and 5 percent levels), The uncertainty bounds have been estimated through the use of the range factor data supplied by GA Technologies as a part of the accident scenario data base (GA Technologies, 1987[a], 1987[b], 1987[c]). The method for estimating uncertainty for a programmatic alternative is presented in Fraize et al., 1987[a]. Note that the uncertainty estimates are based on uncertainty in the estimated probability that an accident will take place, not on uncertainty in the consequence of an accident; nor do the uncertainty estimates include

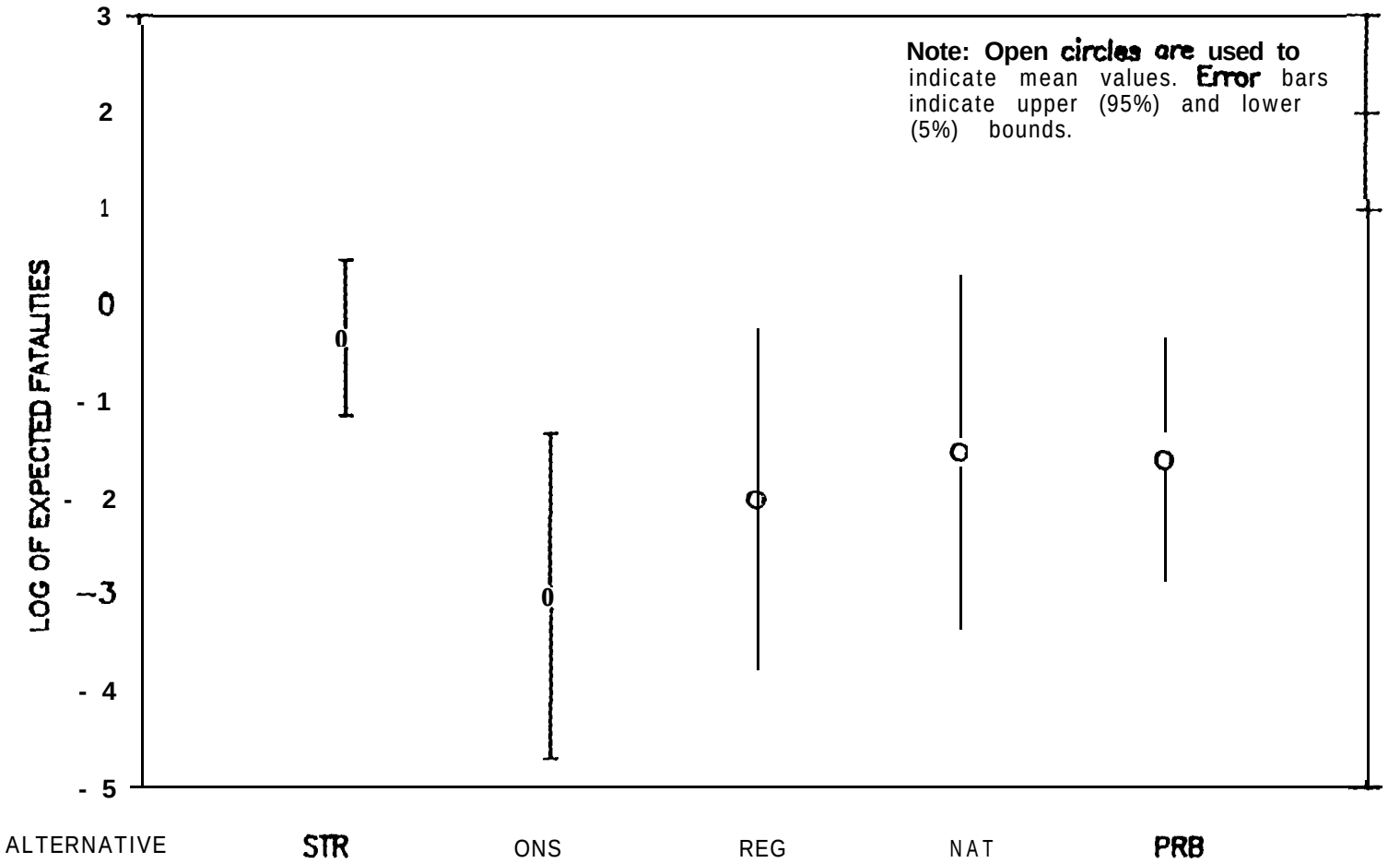


FIG. J.9

**SOCIETAL RISK (EXPECTED FATALITIES)
FOR PROGRAMMATIC ALTERNATIVES
MITIGATED -- NO AIR SPACE RESTRICTION**

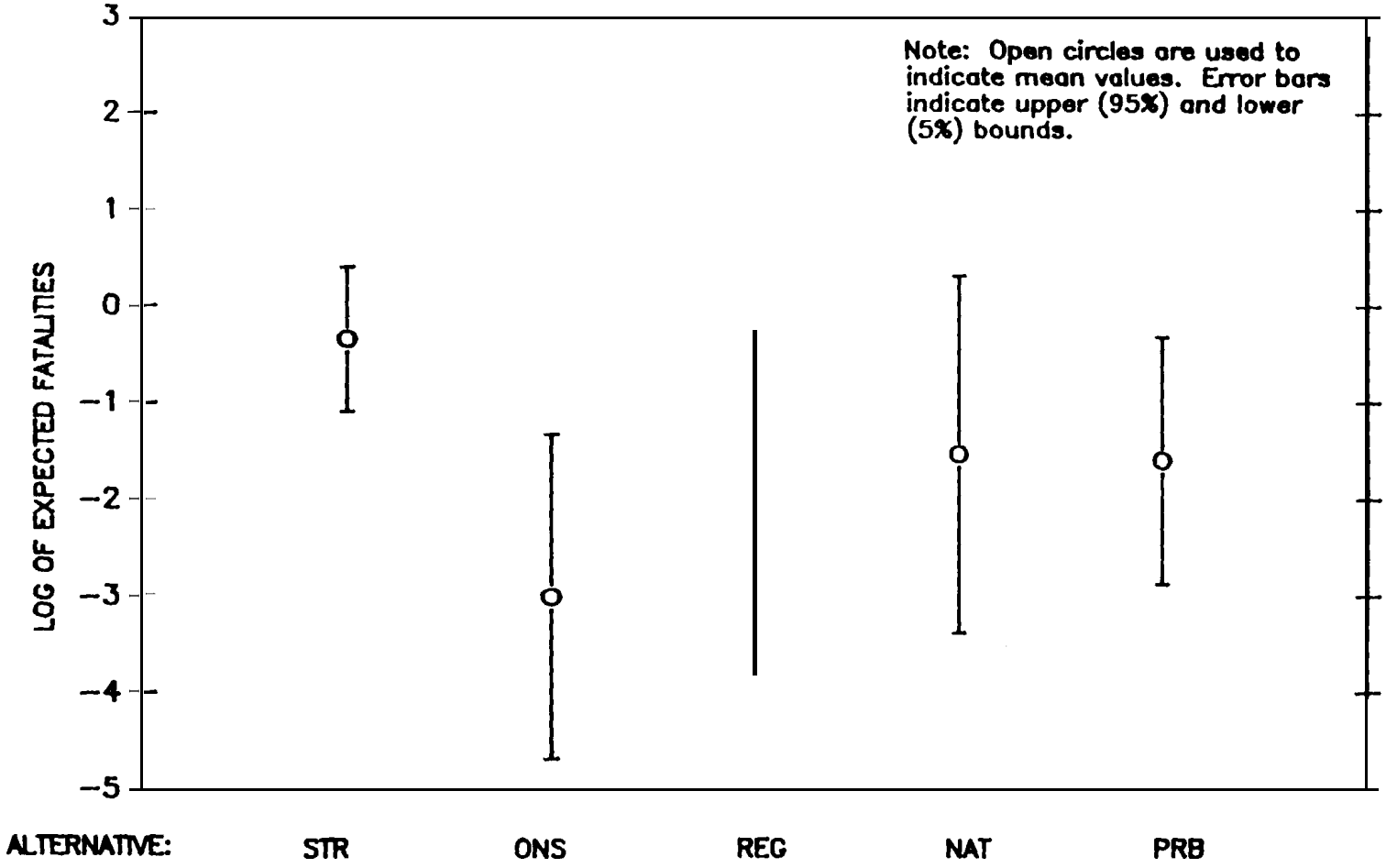


FIG. J.10

SOCIETAL RISK (EXPECTED FATALITIES)
FOR PROGRAMMATIC ALTERNATIVES
MITIGATED – WITH AIR SPACE RESTRICTION

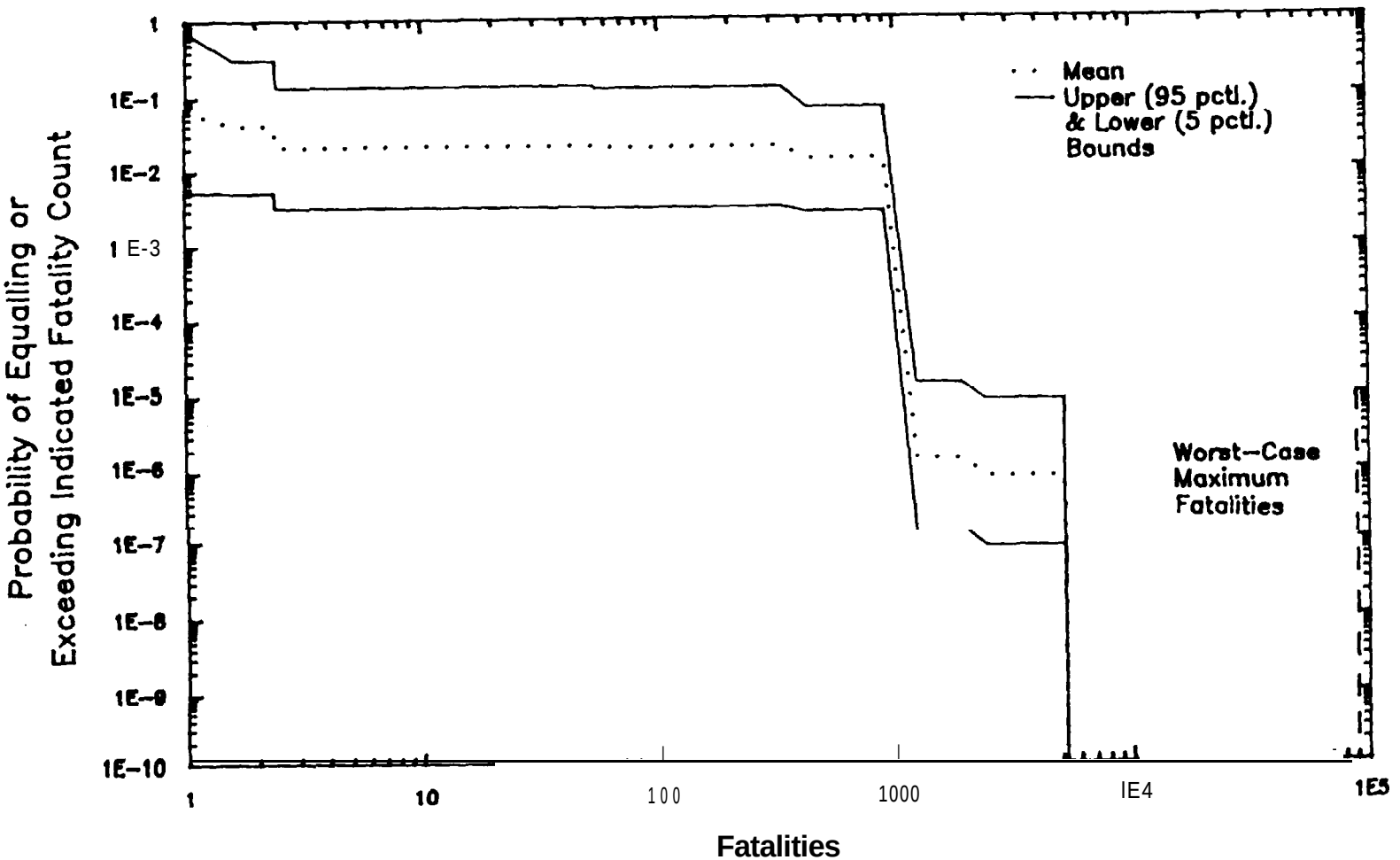


FIG. J.11

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: STR
CONTINUED STORAGE - 25 YEARS

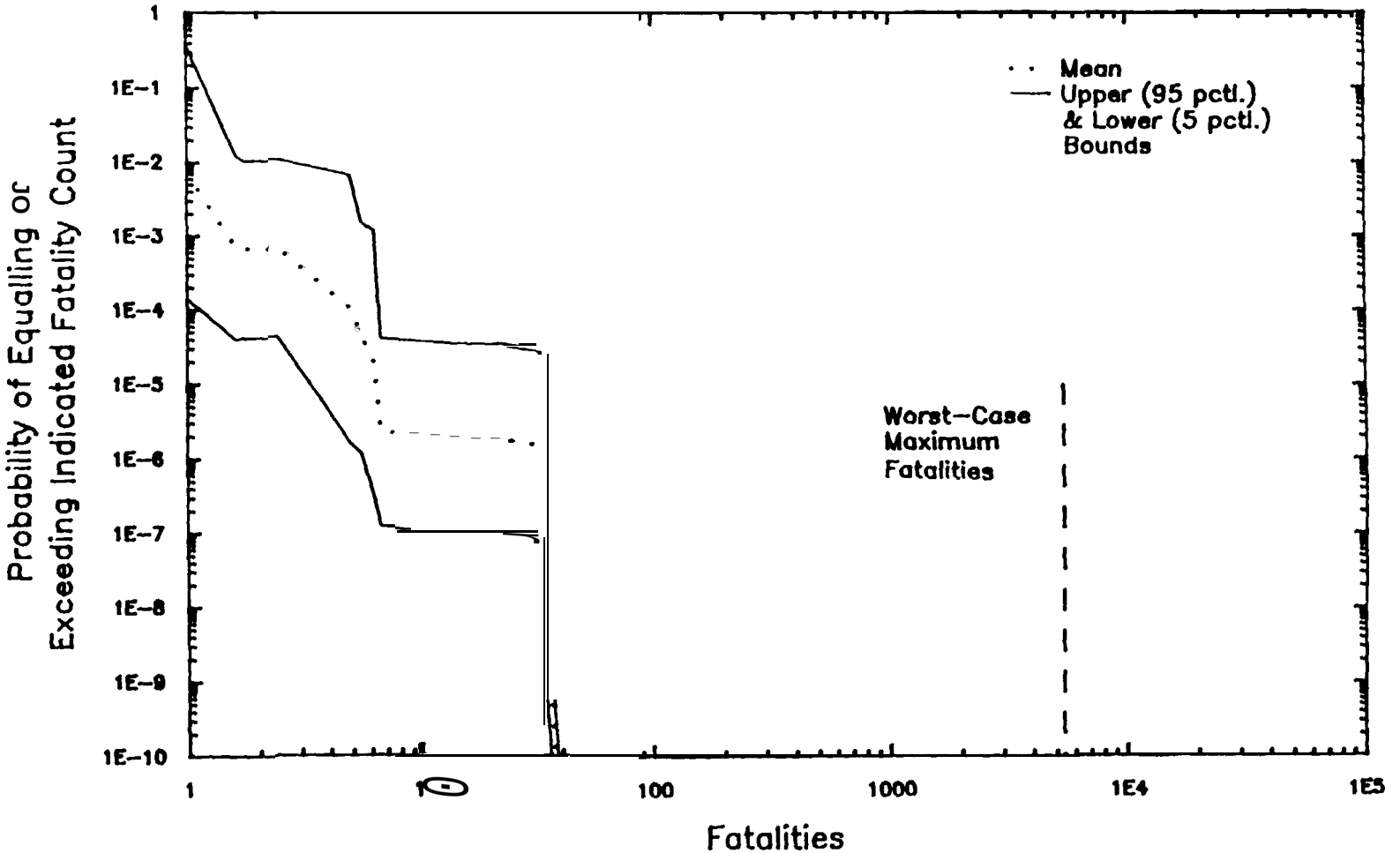


FIG. J.12

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: ONS ON-SITE DISPOSAL

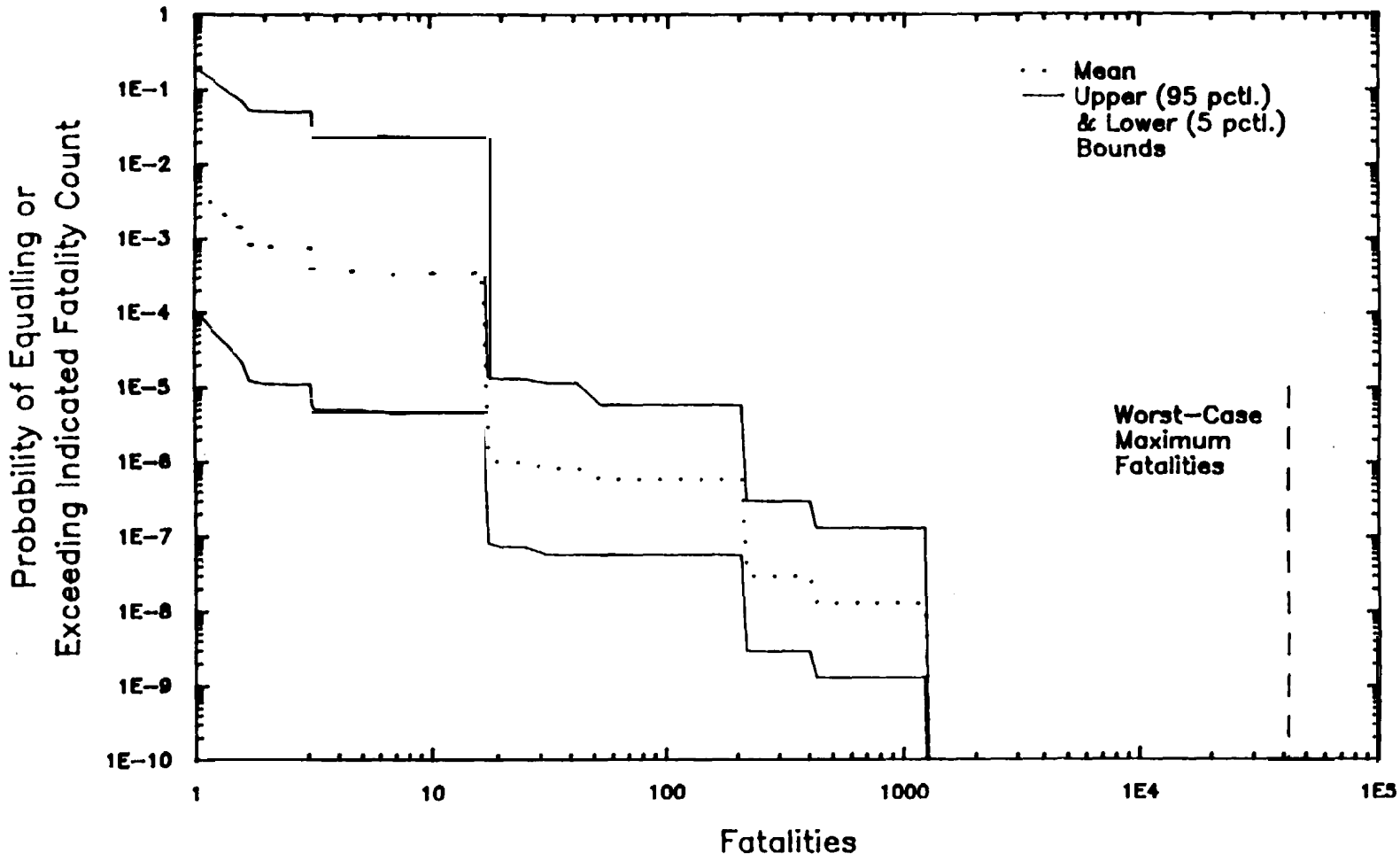


FIG. J.13

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: REG REGIONAL DISPOSAL (RAIL)

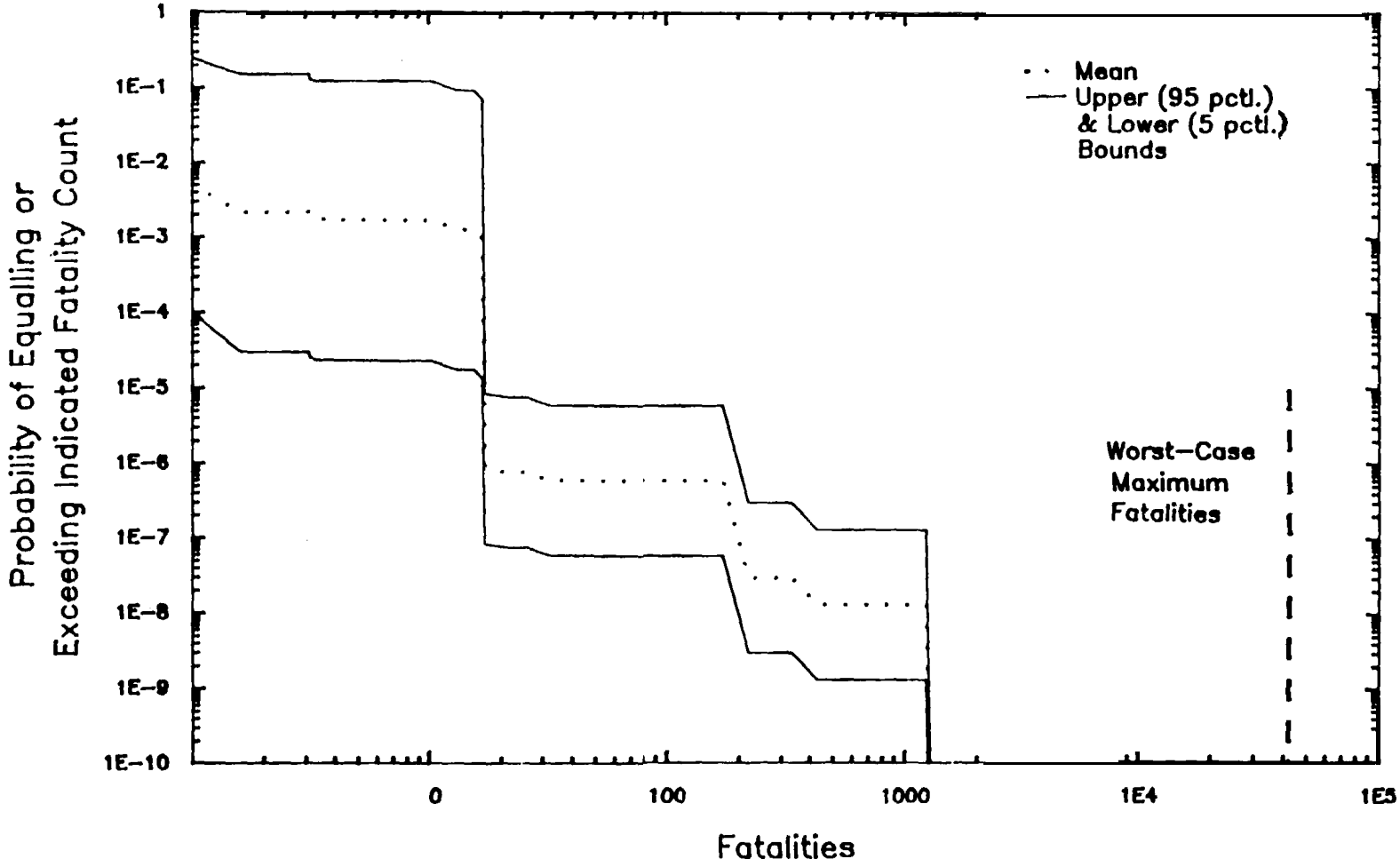


FIG. J.14

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: NAT NATIONAL DISPOSAL (RAIL)

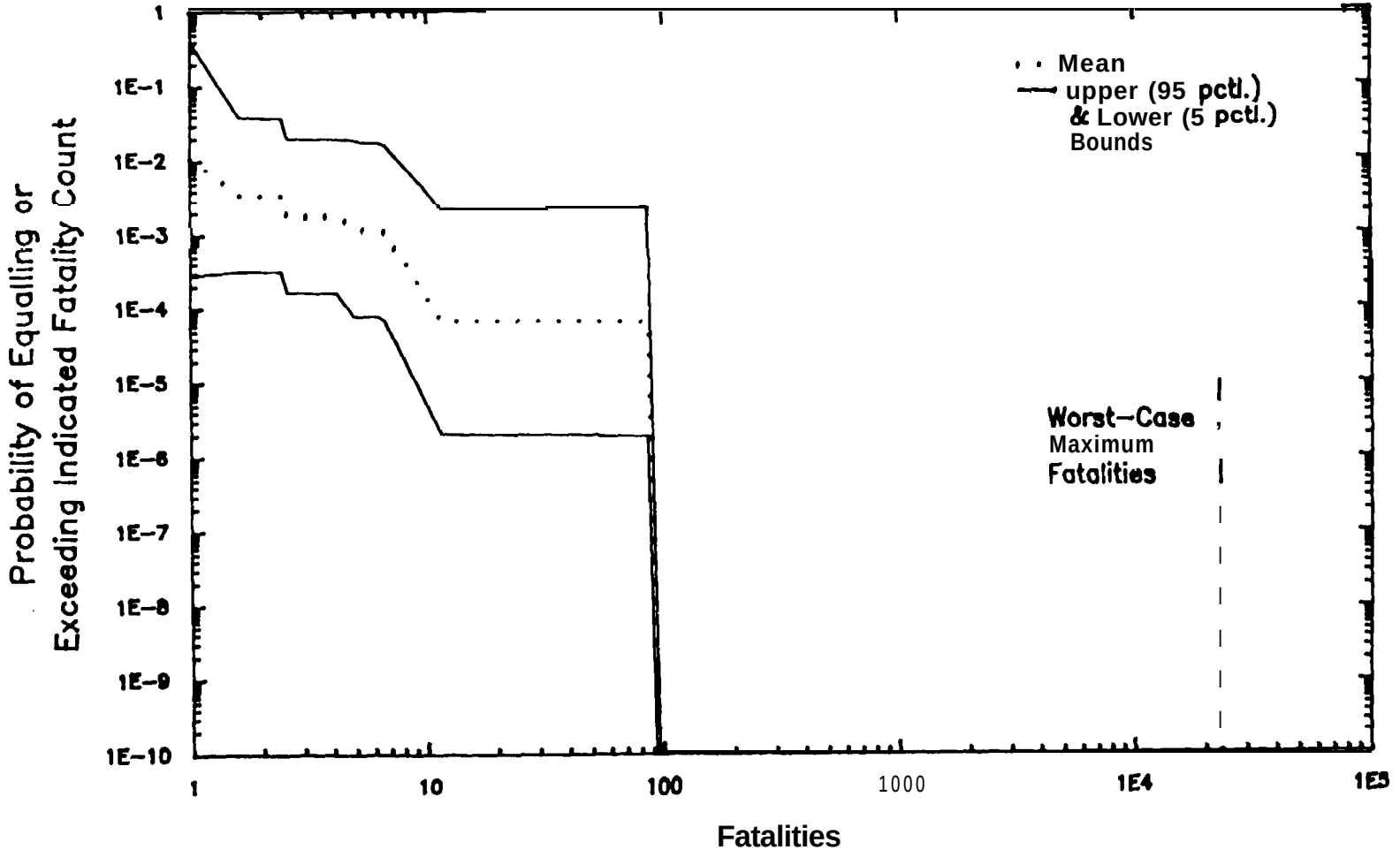


FIG. J.15

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: PRB
PARTIAL RELOCATION: APG & LBAD TO TEAD BY AIR (C141)

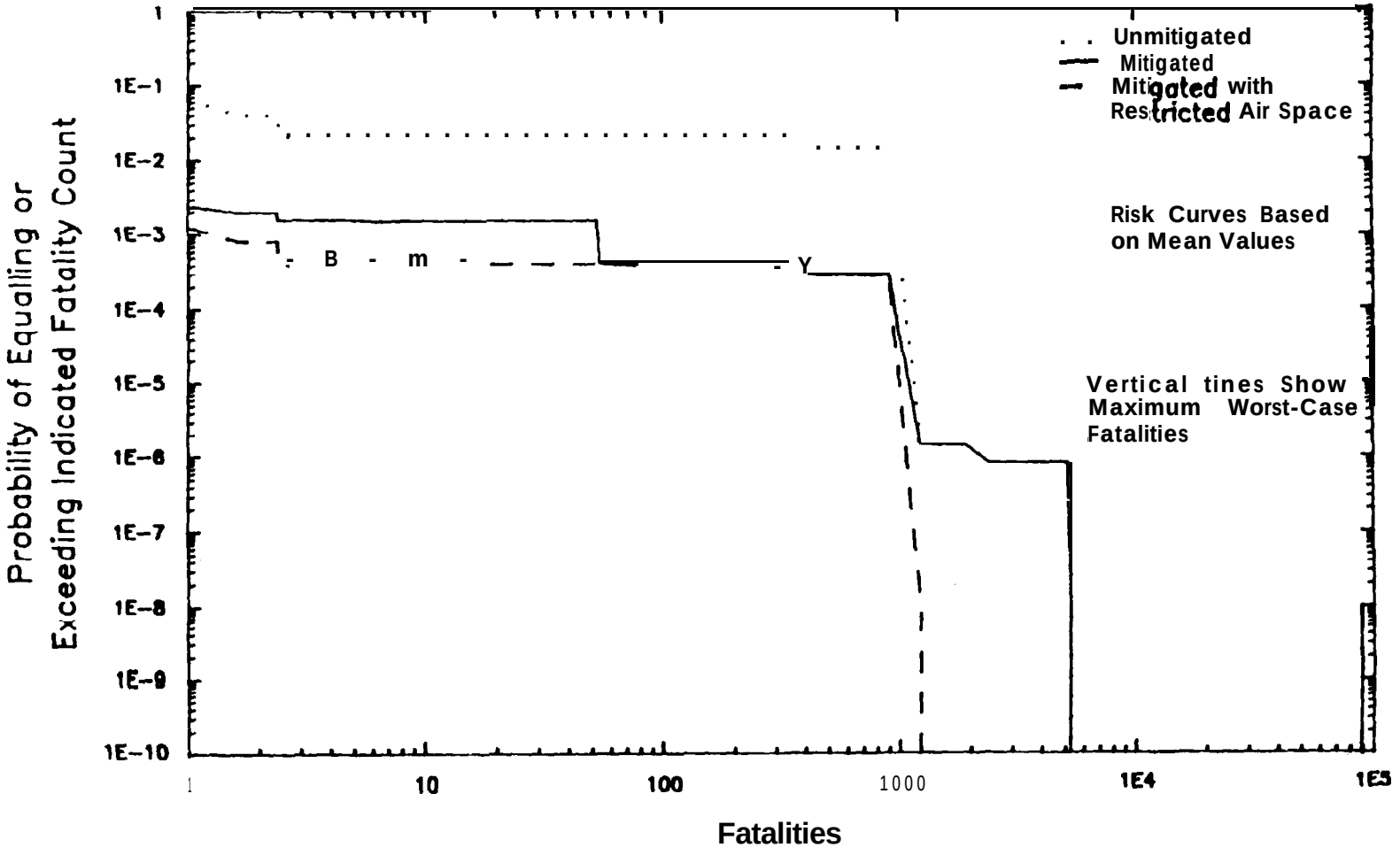
the uncertainty in population density, atmospheric conditions, and dose response. Uncertainty in wind direction is implied by assuming a uniform wind rose (equal probability for any direction) in conjunction with the. assumptions of most-likely meteorological conditions and average population densities used for all probabilistic risk computations.

The effects of mitigation for each disposal alternative are displayed by the cumulative risk curves in Figures 5.16 through J.20. These risk curves do not show the uncertainty bands, which are well represented by Figures J.11 through 5.15 but, instead, show three mean value curves -- one for each level of mitigation.

J-3.2.2.1 Continued Storaee. The programmatic risk due to the continued storage alternative for the unmitigated case is portrayed in Figure J.11. The risk is made up of both internally- and externally-initiated potential accidents. Storage of bulk containers accounts for 99 percent of the risk. Externally-initiated events, and, in particular, relatively mild earthquakes that result in fires affecting warehouse storage of bulk containers of mustard or VX at three sites (NAAP, UMDA, and TEAD), account for nearly all of the risk as measured by expected fatalities (the area under the cumulative risk curve), with earthquakes at the NAAP warehouse dominating. The probability of these earthquake events occurring in any given year is in the range of 10^{-4} to 10^{-5}, and the amount of agent potentially released is in the range of 10,000 to 100,000 pounds or more -- an amount which, because it can released as a vapor cloud fed by burning agent, can cause downwind plume lengths ('no-deaths' hazard distances, most-likely weather) of 20 to 50 km. Such large plumes could result in large numbers of fatalities, even in remote areas.

The next most significant contributor to risk of continued storage is the result of another external event -- a small aircraft crash into the open storage yard, containing ton containers of mustard, at APG. Like the warehouse accidents, this scenario involves the fire-induced release of mustard agent. The frequency expected for this accident is 10^{-5} per year and the quantity of agent released is in the range of 5000 lbs, which can result in a plume length of approximately 3 km.

The analysis has identified a number of highly probable agent-releasing handling accidents associated with movement of the stockpile for maintenance and surveillance. These accidents, although estimated to occur more frequently than earthquakes or aircraft accidents (frequency of 10^{-2} per year) release small quantities of agent since the contents of only one munition or pallet is involved, and the accident can be cleaned up more quickly. Because the release quantities are smaller, the downwind hazard distances are less. Therefore, handling accidents during storage do not contribute significantly to the population risks associated with the continued storage alternative.



J-40

FIG. J.16

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: STR CONTINUED STORAGE -- 25 YEARS -- SHOWING EFFECTS OF MITIGATION

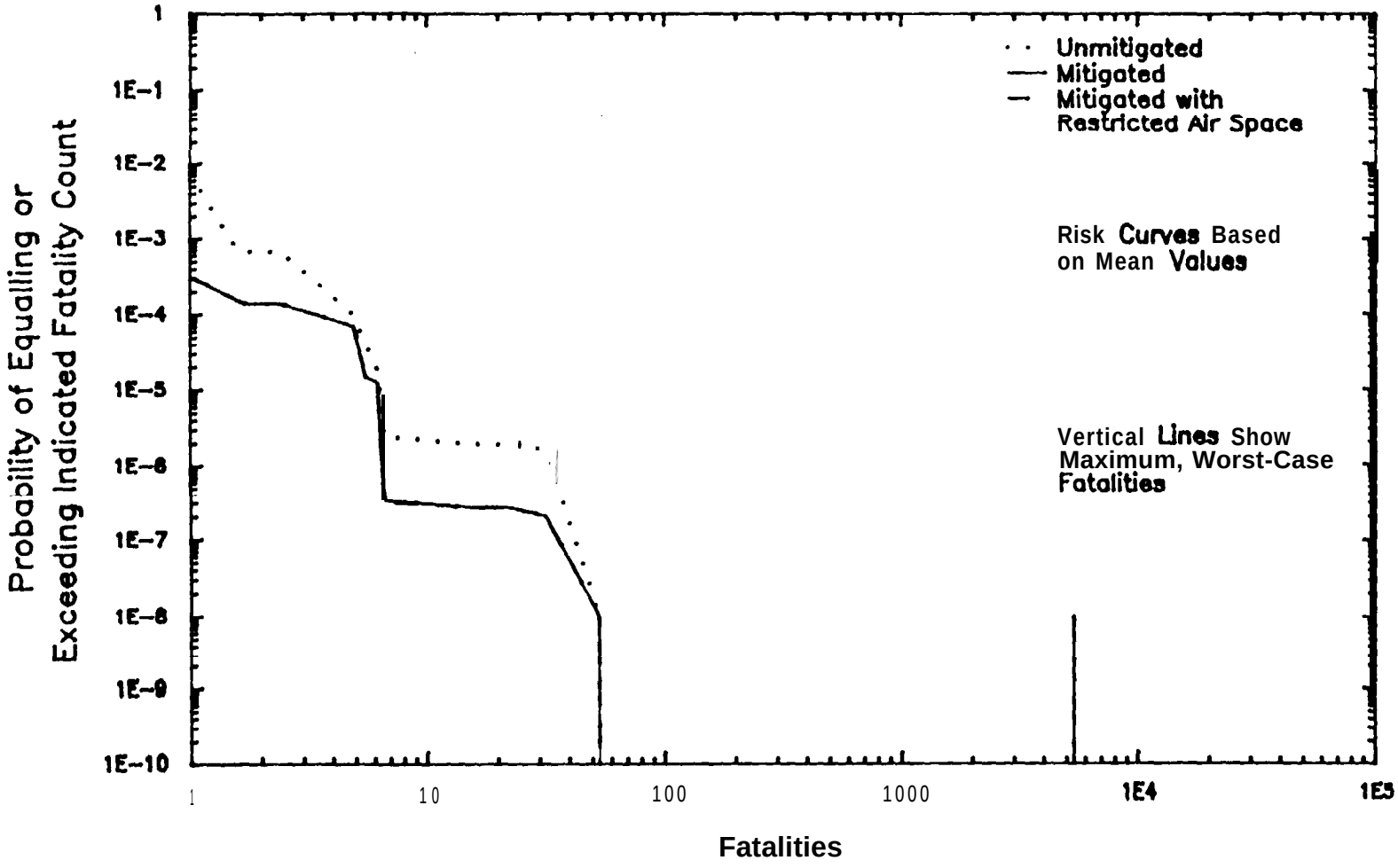


FIG. J.17

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: ONS ON-SITE DISPOSAL – SHOWING EFFECTS OF MITIGATION

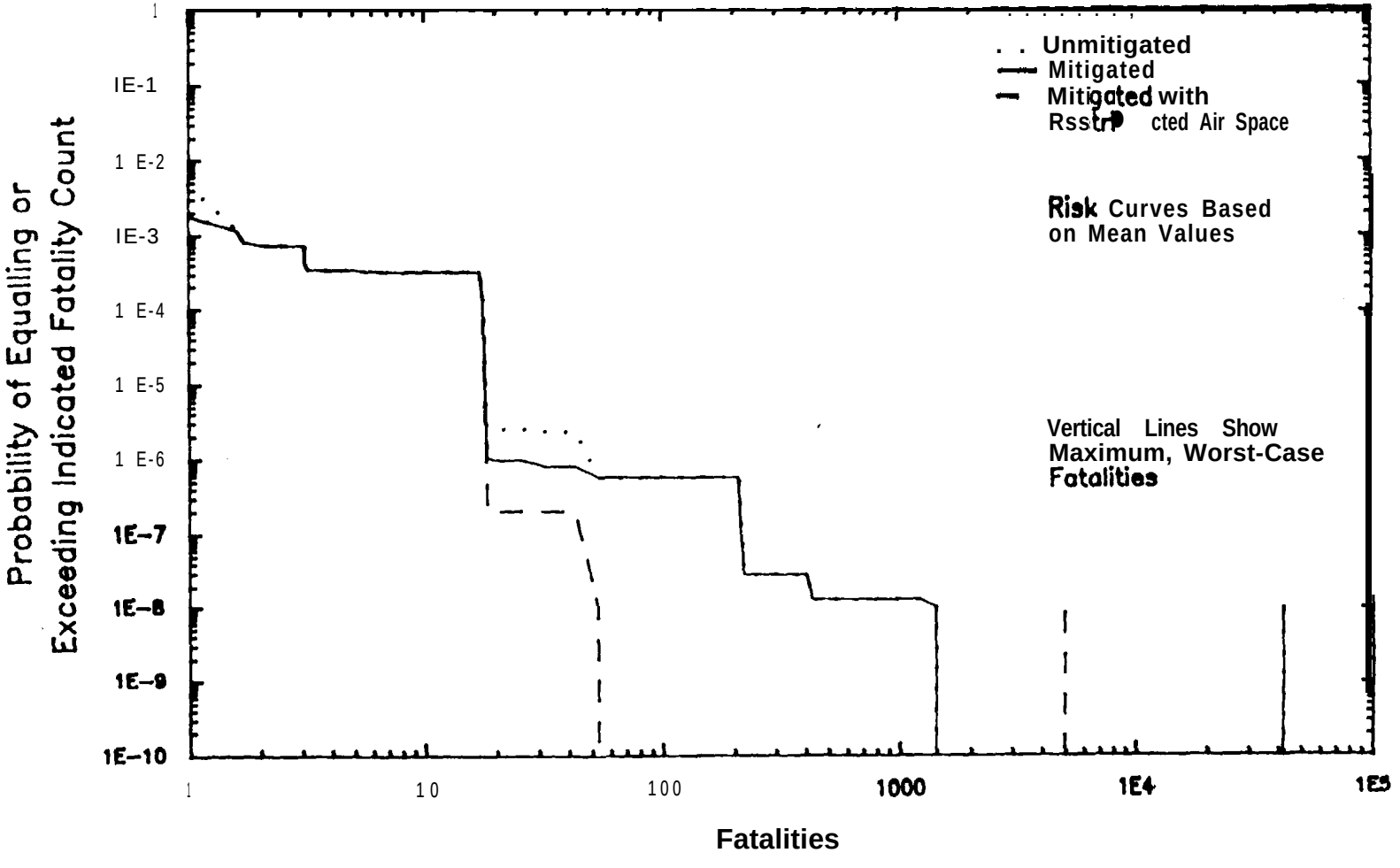


FIG. 3.18

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: REG REGIONAL DISPOSAL (RAIL) -- SHOWING EFFECTS OF MITIGATION

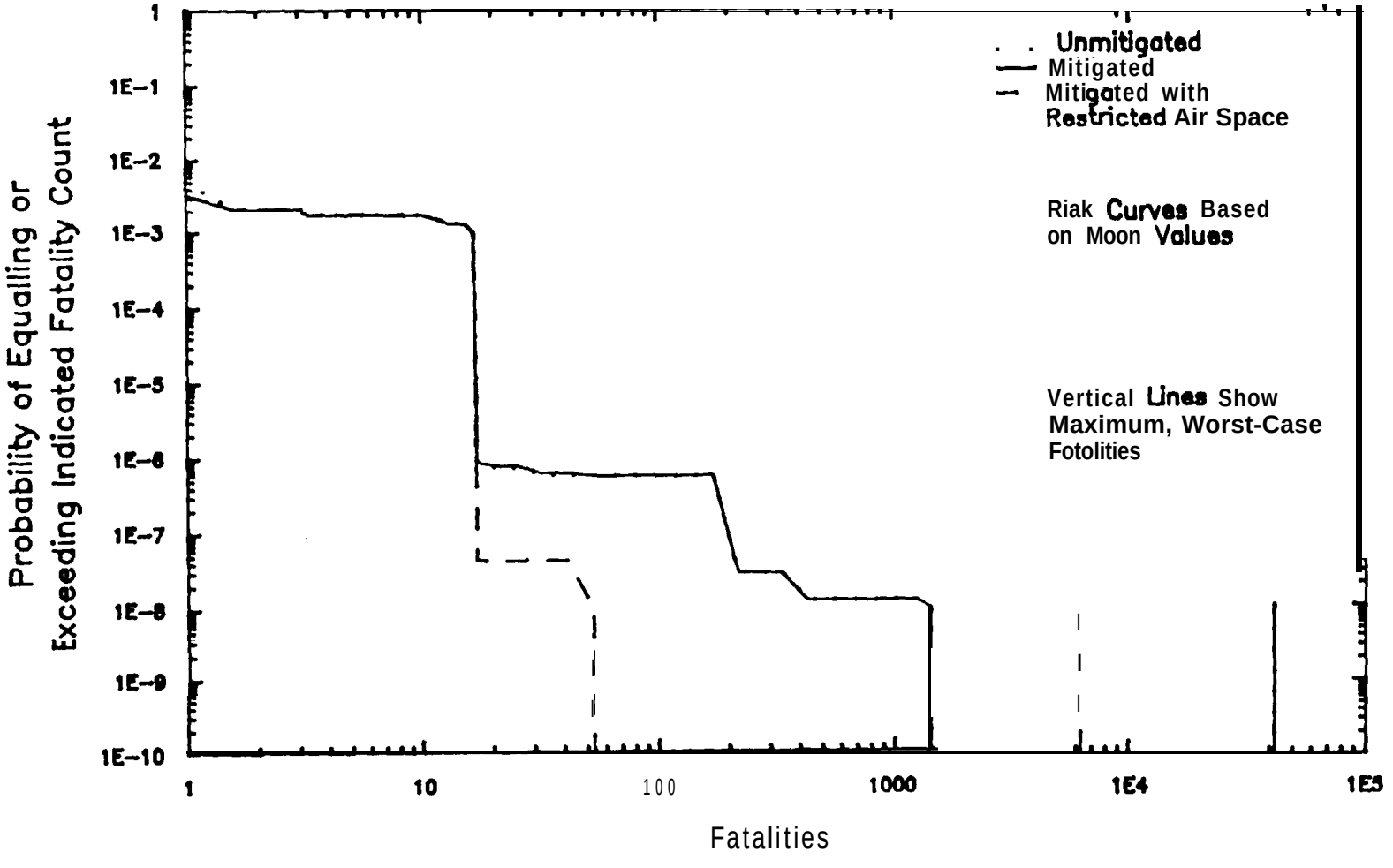


FIG. J.19

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: NAT NATIONAL DISPOSAL (RAIL) -- SHOWING EFFECTS OF MITIGATION

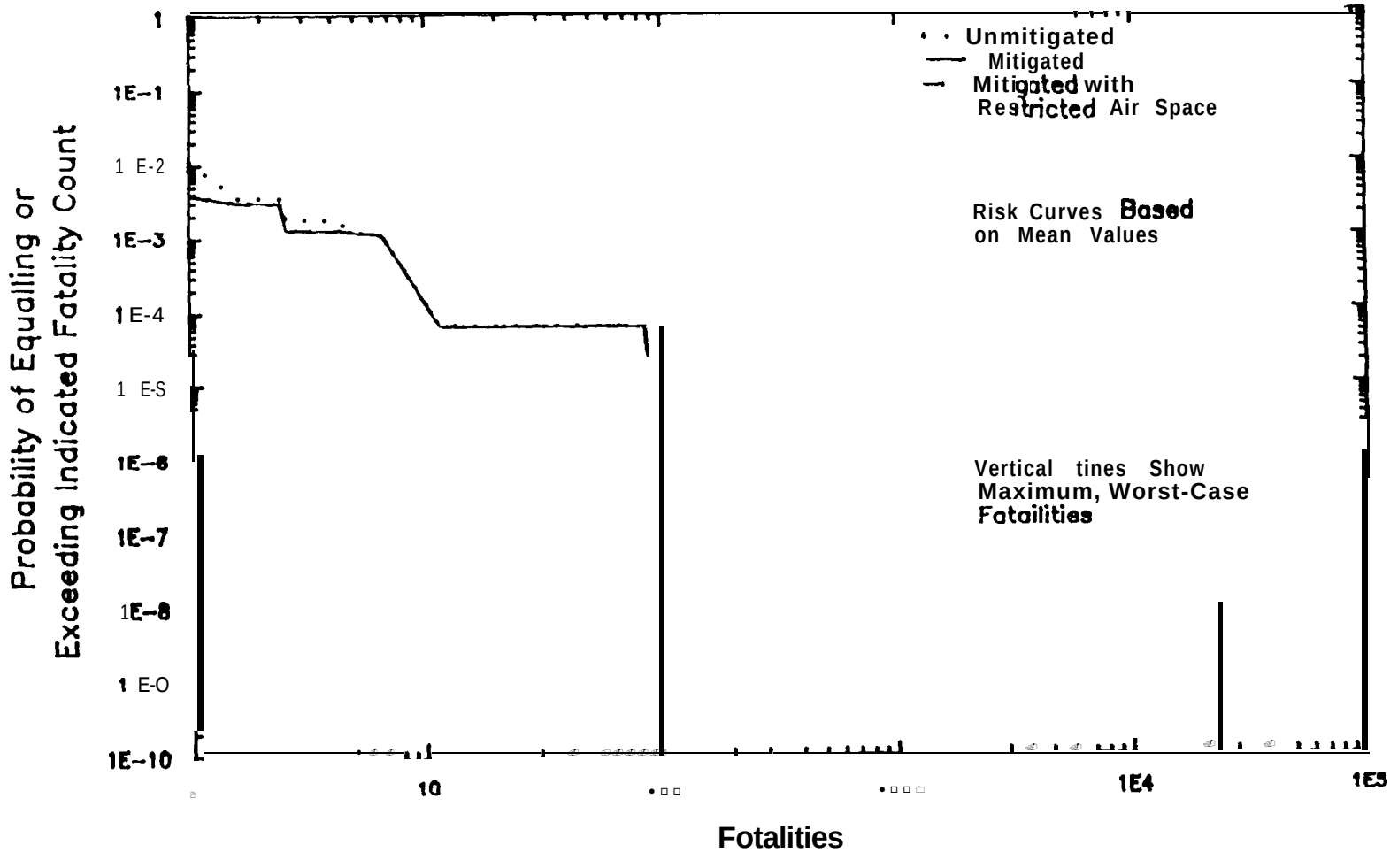


FIG. 5.20

SOCIETAL RISK FOR PROGRAMMATIC ALTERNATIVE: PRB PARTIAL RELOCATION: APG & LBAD TO TEAD BY AIR (C141) SHOWING EFFECTS OF MITIGATION

The risk curve shows that accidents with consequences greater than 5000 fatalities could occur. These accidents involve aircraft crashing into the warehouse at NAAP resulting in fire-induced release of agent VX. However, the probability of these potential accidents is less than 10^{-7}, and the resulting contribution to expected fatalities is relatively low.

The effects of mitigation are portrayed in Figure 5.16. For the case of Mitigation, Revision 1, the risk analysis shows that the probability of one or more fatalities is reduced by a factor of approximately 26, while expected fatalities decreases by a factor of approximately 42. There is no change to the maximum number of fatalities or the person-years-at-risk for this alternative. Storage activities account for approximately 99 percent of the expected fatalities, while handling associated with movement of the stockpile for maintenance and surveillance during continued storage accounts for the remainder. Bulk containers account for approximately 99 percent of the expected fatalities. With the introduction of Mitigation, Revision 2, the probability of one or more fatalities is reduced by approximately a factor of 2, and the expected fatalities are approximately equal.

J.3.2.2.2 On-Site Disposal. The programmatic risk of on-site disposal for the unmitieated case is displayed in Figure J.12. Several activity categories contribute to on-site disposal risk; 93 percent being caused by chemical disposal plant operations; 2 percent caused by handling in the storage area and at the disposal facility; and, 4 percent being caused by on-site transportation. The major contributors to on-site disposal risk are earthquakes damaging the disposal plant and human-error-induced accidents involving inadvertent feed of a burstered munition to the dunnage incinerator. These accidents are among the most frequent of all those identified for this alternative; they have a probability of occurring during the stockpile program of approximately 10^{-3}. The agent release for the earthquake scenario is large because the munition demilitarization building (MDB) is assumed to be severely damaged and bulk agent quantities and/or multiple munitions are involved; the estimated potential release, via fire, is sufficient to generate a lethal plume approximately 3 km long. The dunnage furnace scenarios involve lesser release quantities, since only single munitions are involved. Aircraft crashes into the disposal plant do not contribute significantly to risk because of the relatively small size of the target and of the local inventory available for release, and because of the relatively short time the plant is in operation (<3 years at most sites).

On-site transport of munitions also contributes significantly to on-site risk because large quantities of agent can be involved in vehicle accidents, and because the probability of occurrence, although only 10^{-10} accidents per vehicle-mile, is relatively high because there are many vehicle-miles involved in the CSDP.

Handling accidents which contribute most significantly to on-site risk are the dropping of an on-site container or a pallet of munitions.

The on-site disposal alternative has the lowest maximum consequence accident (most-likely meteorological conditions) of any alternative. The maximum potential fatality event could cause an estimated 54 deaths (under these most-likely conditions) as a result of either an earthquake, leading to a fire in the disposal plant, or a serious on-site transport vehicle accident; all these maximum consequence accidents involve the fire-borne or detonation-caused release of agent VX. (For statistical reasons, explained in Fraize et al. 1987a, the X-axis intercept on Figure 5.12 and other programmatic risk curves appears to occur at a lower value.)

In addition to the dunnage incinerator accidents discussed above, other accidents having probabilities in the range of 10^{-4} to 10^{-3} per stockpile also include handling operations both at the storage yard and at the plant; these handling accidents, with the exception of accidents during handling of ton-containers containing GB, do not result in consequences beyond the boundaries of the military reservation.

The effects of mitigation are portrayed in Figure 5.17. For the case of Mitigation, Revision 1, the probability of one or more fatalities is reduced by a factor of approximately 22, while expected fatalities decreases by a factor of approximately 10. On-site transportation activities account for approximately 44 percent of the expected fatalities, while plant operations account for approximately 48 percent. Fifty percent of the expected fatalities can be attributed to rockets, and approximately 42 percent to bulk containers. Expected fatalities caused by plant operations activities were reduced by a factor of approximately 20 while expected fatalities caused by handling activities were reduced by approximately a factor of 4. There is no additional reduction in risk with the introduction of Mitigation, Revision 2.

J.3.2.2.3 Regional Disposal (Rail). Figure 3.13 illustrates the programmatic risk for the unmitigated regional disposal alternative. Over 60 percent of the total risk (expected fatalities) is due to potential off-site transport accidents; 25 percent of the risk is due to plant operations and less than 10 percent results from on-site transport; short-term storage and handling together contribute less than 5 percent to total risk. Of the risk contributed by off-site transportation, 80 percent is due to the transport of rockets, followed in significance by an 11 percent contribution due to transport of mines. Of total regional disposal risk, over 60 percent is due to rockets.

Among individual accident scenarios, those contributing most to risk are due to off-site rail transport of rockets. Of nearly equal risk are dunnage incinerator accidents involving rockets and mines.

In contrast to the on-site disposal risk curve (Figure J.12), the regional alternative includes potential accidents with much higher consequences (most likely conditions): greater than 1400 maximum fatalities vs. 54 for on-site. The highest consequence event involves short-term storage of the transportation containers of rockets in the holding area. However, the probability of this high consequence accident is low: less than 10^{-7}.

The highest probability accidents for this alternative are those due to inadvertent feeding of burstered munitions into the dunnage incinerator and handling accidents involving single munitions or a pallet of munitions. These high probability accidents are not of sufficient consequence, under most-likely conditions, to cause fatalities beyond the boundaries of the military reservation with the exception of dunnage furnace accidents involving mines and rockets.

The effects of mitigation are portrayed in Figure 5.18. For the case of Mitieation. Revision 1, the probability of one or more fatalities is reduced by a factor of 2, while the expected fatalities does not significantly decrease. Plant operations expected fatalities were decreased by a factor of 47. Off-site transportation activities account for approximately 77 percent of the expected fatalities, on-site transportation accounts for approximately 18 percent of the expected fatalities, and the remainder is distributed among the handling, storage, and plant operations activities. Rockets account for approximately 66 percent of the expected fatalities, while projectiles and mines account for approximately 14 and 11 percent of the expected fatalities respectively. With the introduction of Mitigation, Revision 2, the maximum fatalities are reduced by a factor of approximately 7. There is no reduction in the probability of one or more fatalities or in the expected fatalities.

J.3.2.2.4 National Disposal (Rail). The programmatic risk due to the national disposal alternative without mitigation is portrayed by Figure 5.14. The risk curve appears to be very similar to that for regional disposal, as one might expect since the mix of activities is the same, with the major differences due to where the accidents might take place. Relative to regional disposal, the national alternative involves the transportation of the ANAD stockpile to TEAD and the shift of all plant operations to TEAD.

Of the total risk, approximately 90 percent is caused by off-site transportation and less than 5 percent is caused by chemical disposal plant operations. Of the off-site transportation risk, over 95 percent is caused by transportation of energetic munitions, approximately 55 percent of that being caused by transportation of rockets, and 25 percent by transportation of projectiles.

As with regional disposal, the major contributors to risk among individual accident scenarios are the off-site rail transportation accidents. However, for this alternative, the highest risk accidents include those due to projectiles and mines, representing the risk due to transport of the ANAD stockpile to TEAD.

The highest consequence scenario, involving potential fatalities of over 1400, is the same as for the regional alternative: short-term storage of rockets in the holding area.

Highest probability accidents (probability greater than 10^{-4}) for national disposal are due to plant operations (inadvertent feed of burstered munitions to dunnage incinerator), handling, and off-site rail transportation. Of these, the handling accidents do not lead to plume lengths which exceed the boundary of the military reservation with the exception of those involving ton containers of GB, and therefore do not contribute significantly to risk.

The effects of mitigation are portrayed in Figure J.19. For the case of Mitigation, Revision 1, there is no significant reduction in the probability of one or more fatalities or the expected fatalities. Approximately 97 percent of the expected fatalities are caused by off-site transportation activities. Rockets account for approximately 56 percent of the expected fatalities, while projectiles and mines account for approximately 28 and 7 percent respectively. With the introduction of Mitigation, Revision 2, maximum fatalities are reduced by a factor of approximately 7. There is no significant reduction in the probability of one or more fatalities or in expected fatalities.

J.3.2.2.5 Partial Relocation: APG 6 LBAD to TEAD by Air(C141). Programmatic risk for the unmitigated partial relocation alternative, on-site disposal at all sites except for transport of the APG and LBAD stockpiles to TEAD via C141 aircraft, is shown in Figure J.15. Of total risk, 71 percent is due to off-site transportation and 27 percent results from plant operations. Accidents involving rockets contribute 77 percent of total risk. In-flight air accidents (along the transportation corridor) account for 46 percent of total risk for this alternative.

The highest consequence accidents, under most-likely conditions, for this alternative (112 potential fatalities) are due to aircraft take-off accidents involving rockets and projectiles containing GB.

The probability of one or more fatalities for this alternative is approximately 10^{-2}.

The effects of mitigation are portrayed in Figure 5.20. For the case of Mitination. Revision 1 the probability of one or more fatalities is reduced by approximately a factor of 3, while expected fatalities, the

area under the curve (on rectilinear scales), does not significantly decrease. Plant operations activities show the greatest expected fatalities reductions, reduced by approximately a factor of 21. Expected fatalities caused by mines were reduced approximately a factor of 21, while expected fatalities caused by bulk containers were reduced by approximately a factor of 4. There is no additional reduction in risk with the introduction of Mitigation, Revision 2.

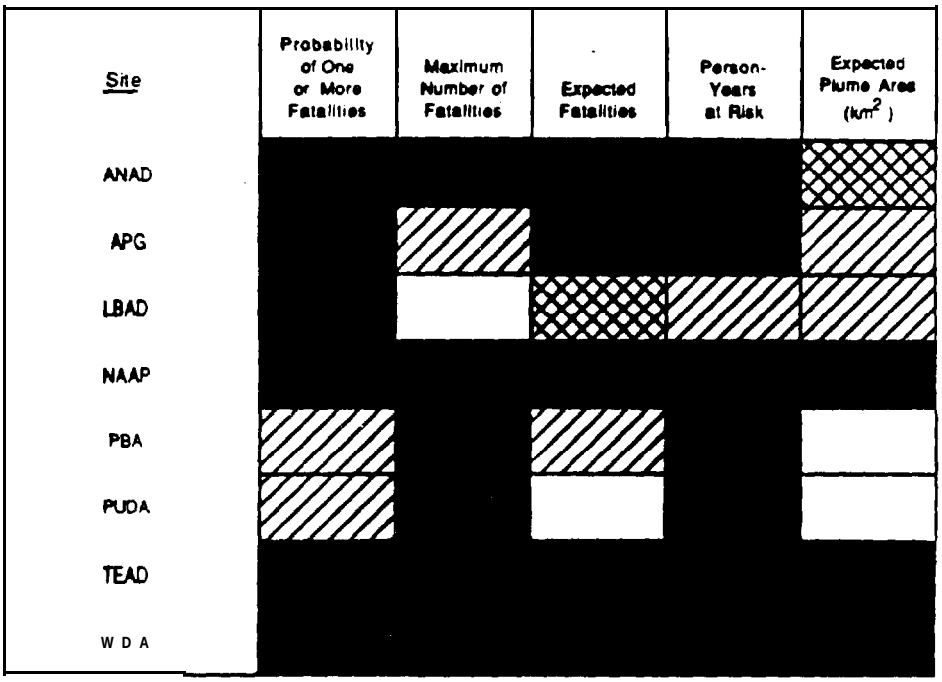
J.3.3 Location-Specific Risk

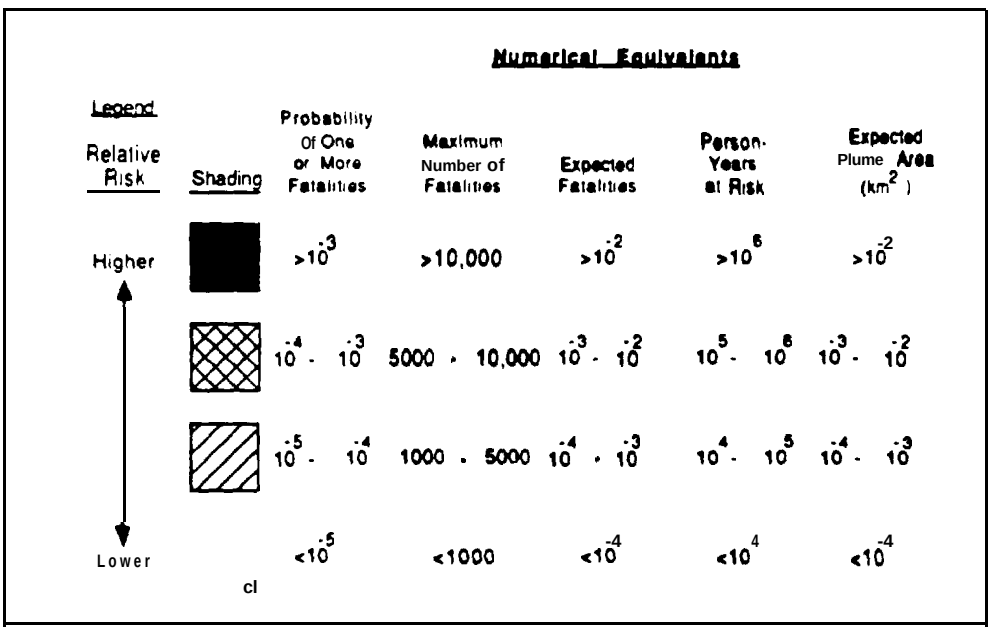
In this section, the distribution of risk according to location (storage/disposal -- i.e., "fixed" -- sites and transportation corridors) is presented by means of the 'pictogram' display of the major risk parameters as shown in Figures 3.21 through 5.62. The first 15 pictograms, Figures 5.21 through 3.35, compare the risk measures for each of the five disposal alternatives at each of the eight sites for the unmitigated case plus two levels of mitigation. Figures 5.36 through 5.38 present the risk to the transportation corridor populations (which is not affected by the proposed mitigation measures) for the three alternatives involving off-site transport of the stockpile. Figures 5.39 through 5.62 present the 'pictogram' displays of risk measures for each of the eight sites and for the unmitigated case plus two levels of mitigation. Although the relevant values (shadings) in Figures 5.21 through 5.35 are identical to those for Figures 5.39 through 5.62, the information is presented on a site-by-site basis for the latter figures to facilitate comparison of risks at a site for the different alternatives. The reader should note that all figures displaying site risk do not incorporate risks along a transportation corridors. (The corresponding 'pictogram' for all locations combined was presented as Figures 5.5 through 5.7, depending on mitigation level.)

In addition, risk to an individual -- a meaningful concept only when a specific location is considered -- is discussed, in section J.3.3.3, in terms of the individual's 'time-at-risk' and the population-based companion measure, person-years-at-risk.

J.3.3.1 Distribution of Pro-tic Risk by Location

In addition to the differences in overall programmatic risk among the disposal alternatives, as presented in section J.3.2, there are major differences in how that risk (as measured by expected fatalities) is distributed among the affected population groups. The pictograms supporting this discussion are those presented in Figures 5.21 through 5.38. In this regard, we note the following:

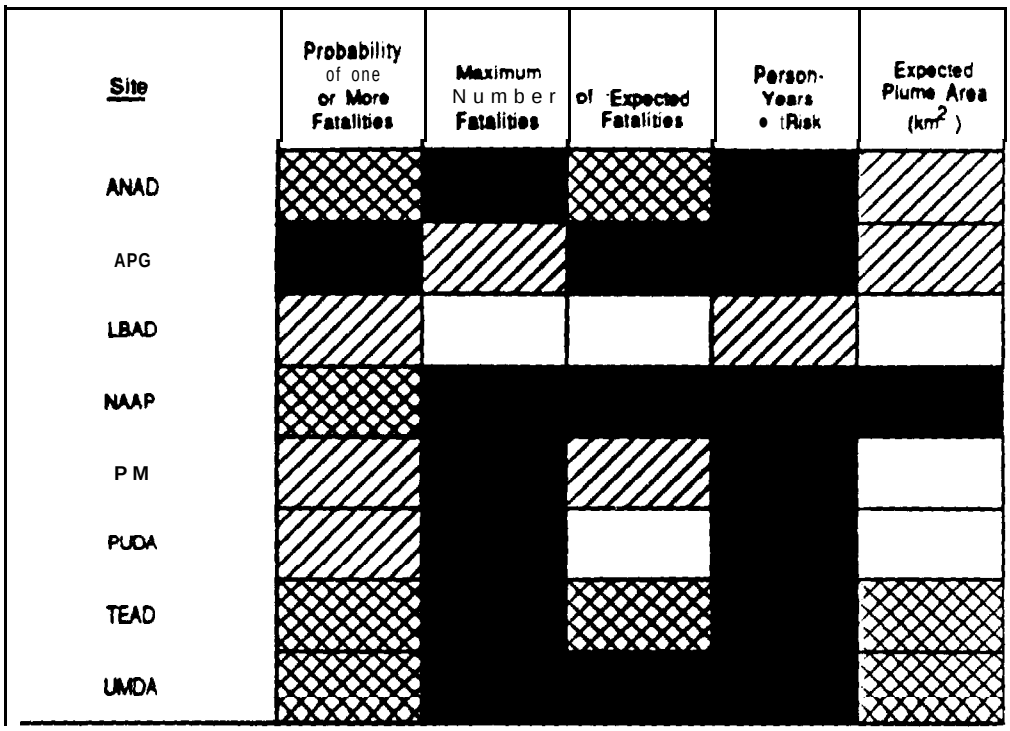




cl

FIG. J.21

SITE-SPECIFIC COMPARISON OF RISK FOR CONTINUED STORAGE (STR) 25 YEARS



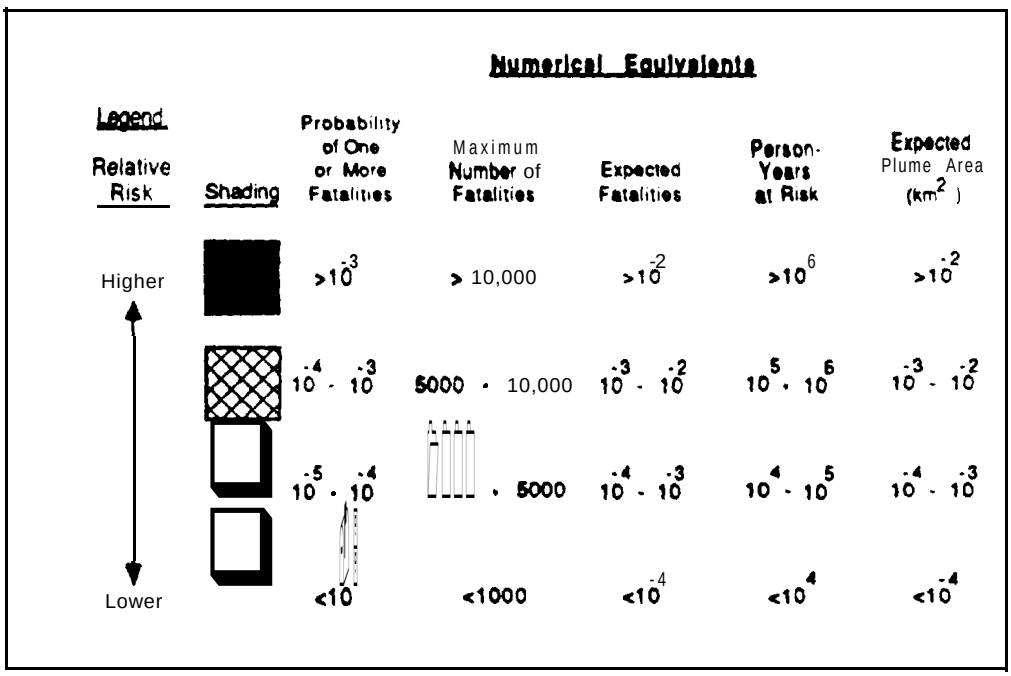
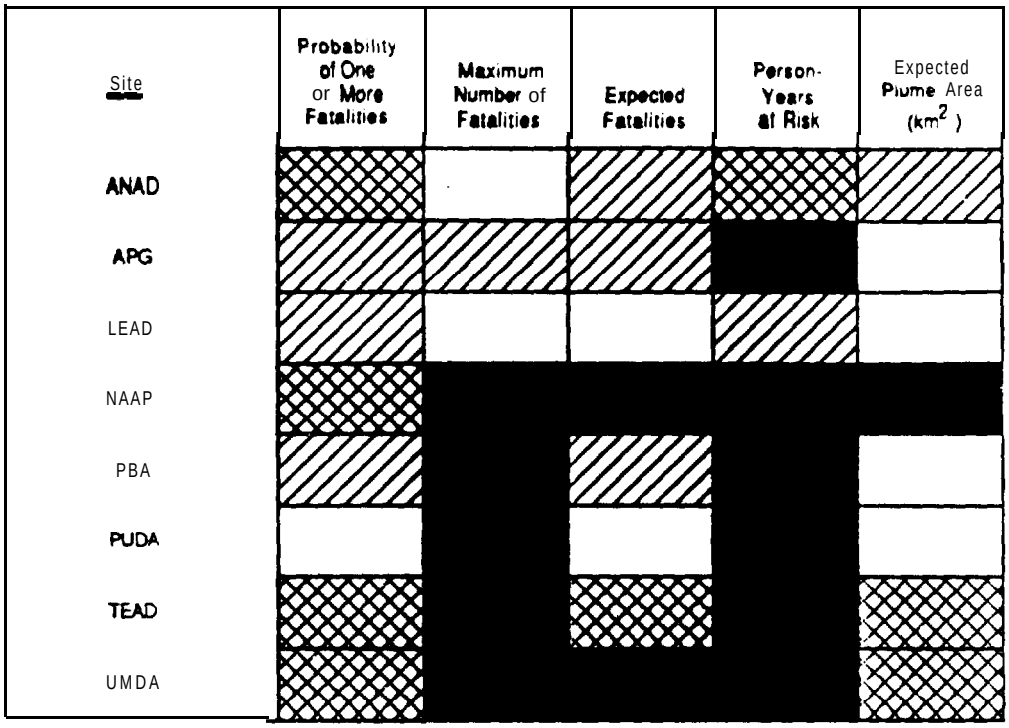


FIG. J.22

**RISK WITH MITIGATION (REV. 1):
SITE-SPECIFIC COMPARISON FOR
CONTINUED STORAGE (STR)
25 YEARS**



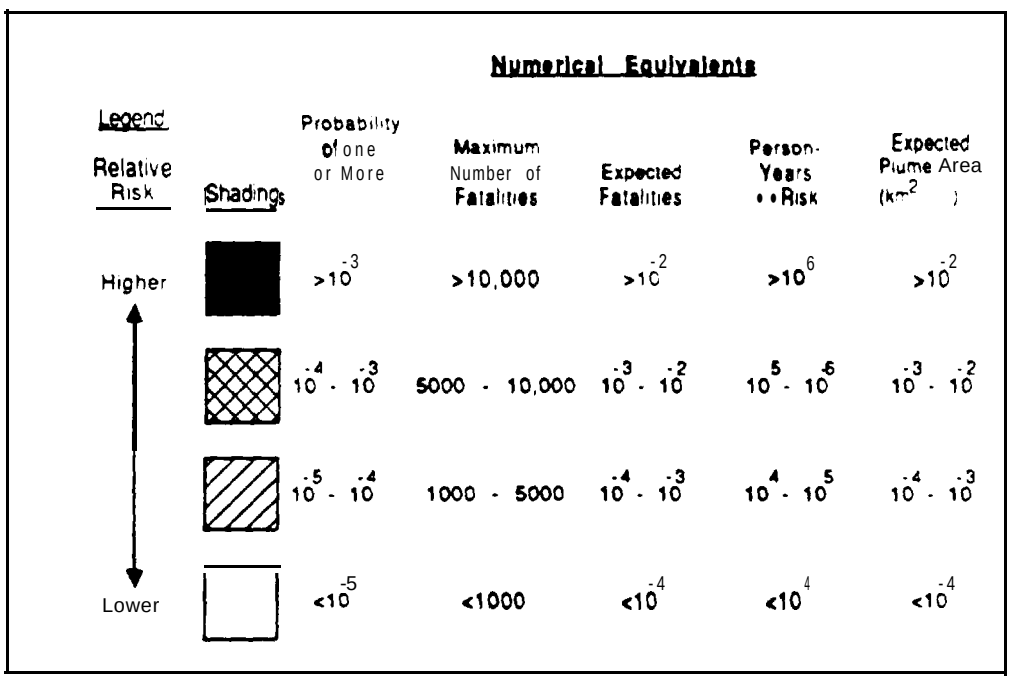
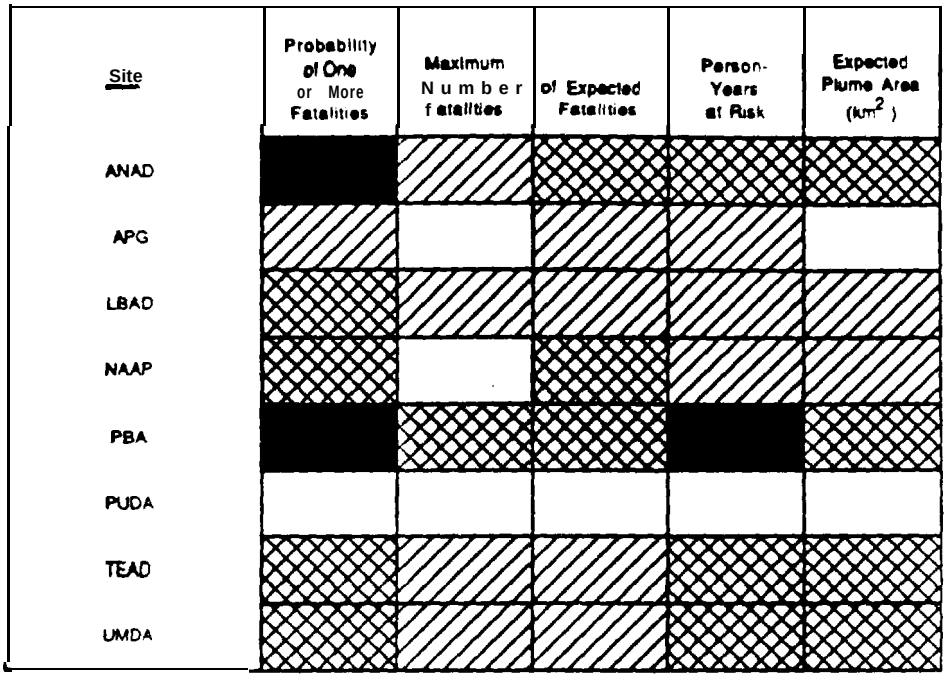


FIG. J.23

RISK WITH MITIGATION (REV. 2):
SITE-SPECIFIC COMPARISON FOR
CONTINUED STORAGE (STR)
26 YEARS



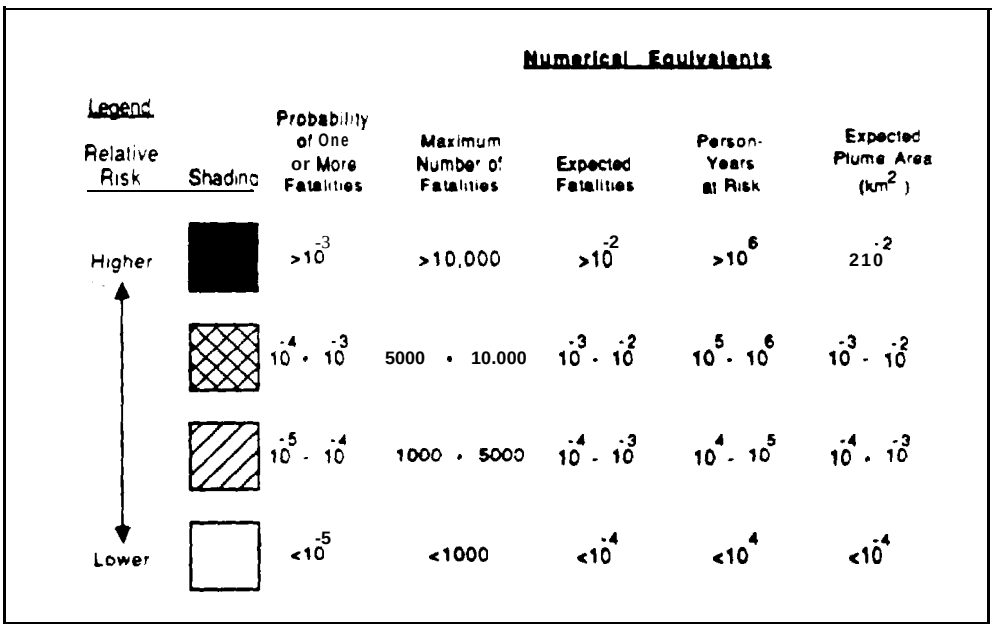
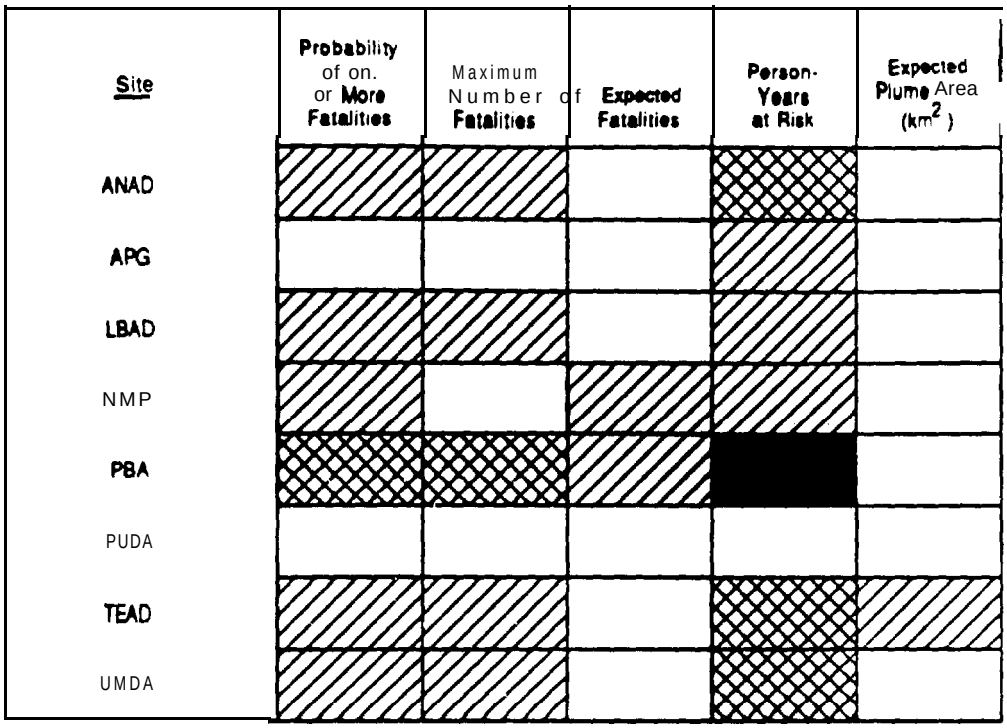


FIG. J.24

SITE-SPECIFIC COMPARISON OF RISK FOR ON-SITE DISPOSAL (ONS)



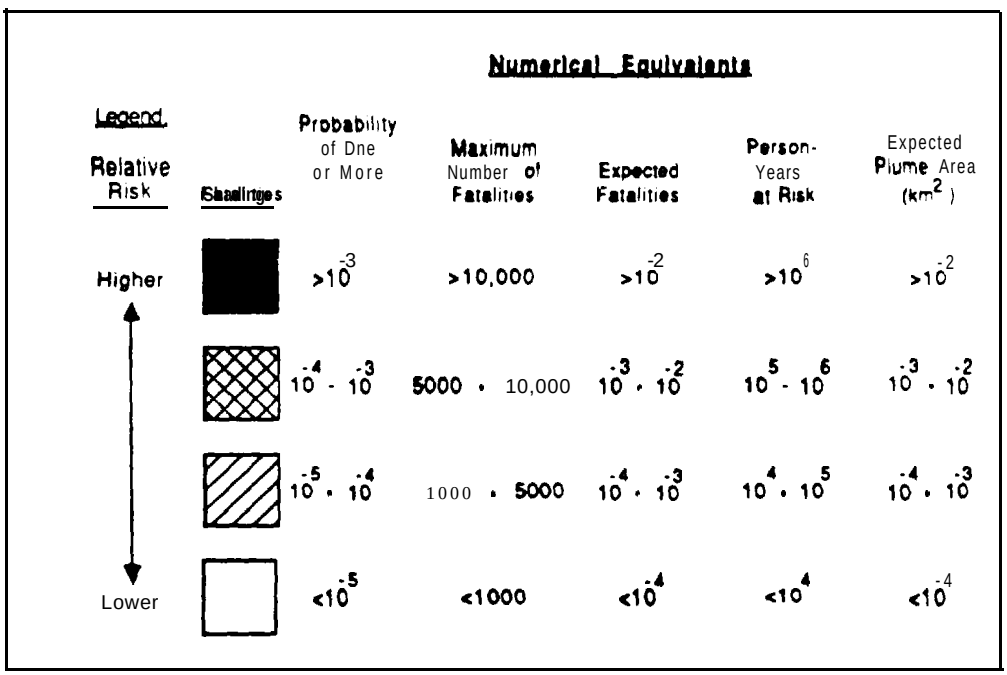
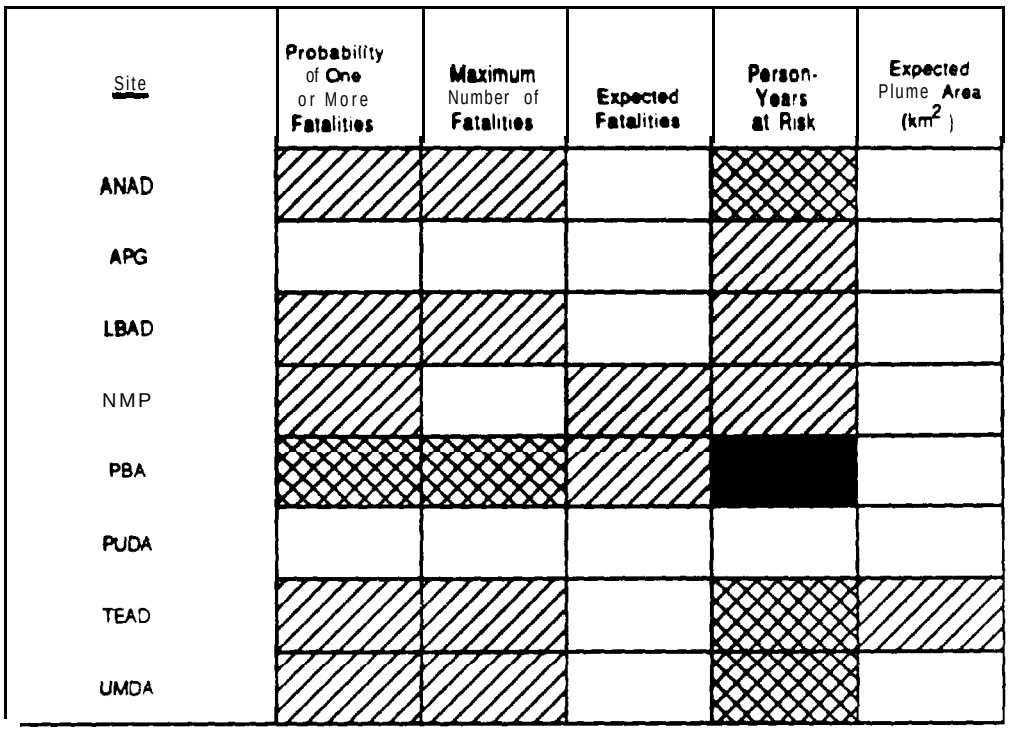


FIG. 5.25

RISK WITH MITIGATION (REV. 1): SITE-SPECIFIC COMPARISON FOR ON-SITE DISPOSAL (ONS)



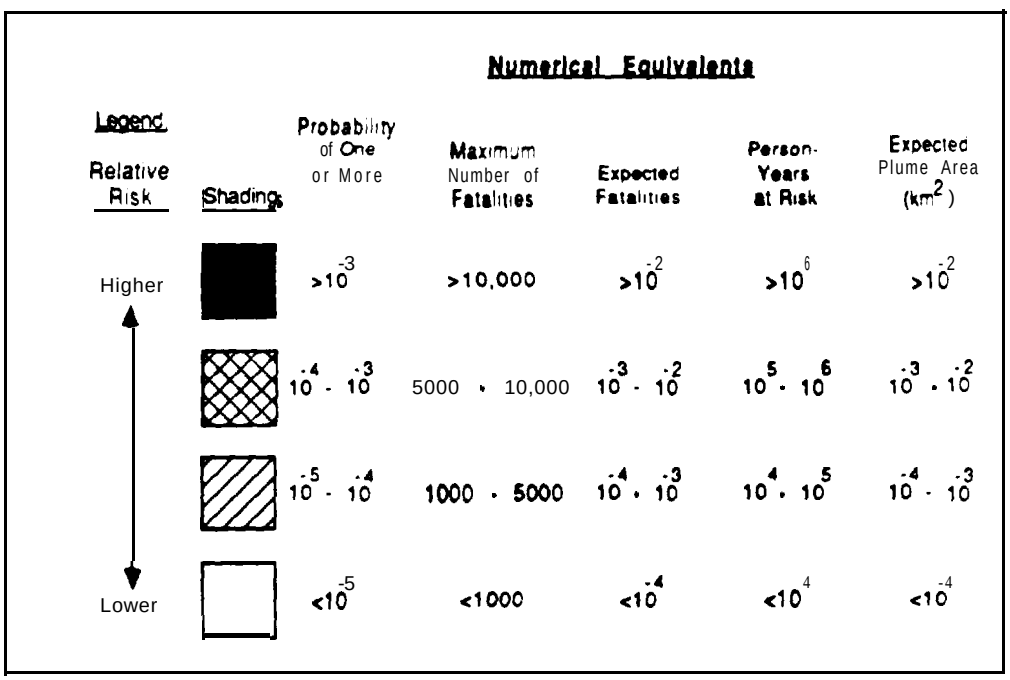
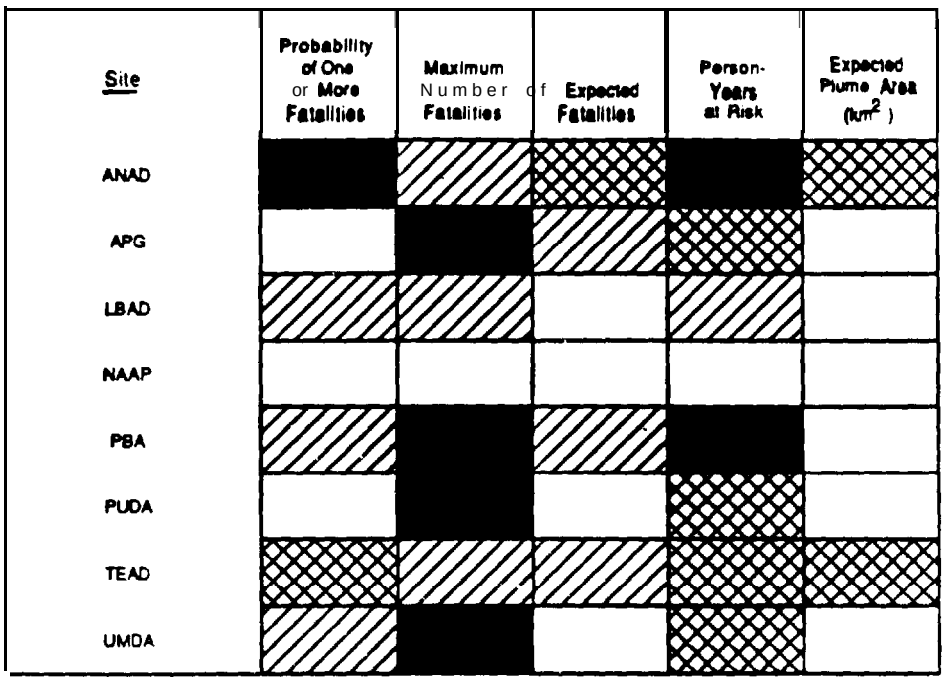


FIG. J.26

**RISK WITH MITIGATION (REV. 2):
SITE-SPECIFIC COMPARISON FOR
ON-SITE DISPOSAL (ONS)**



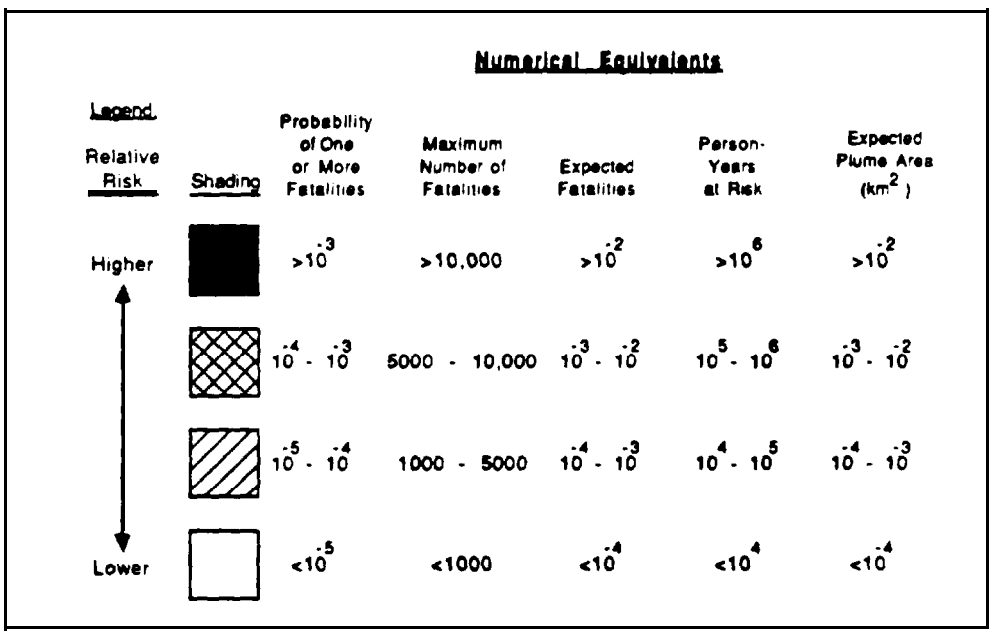
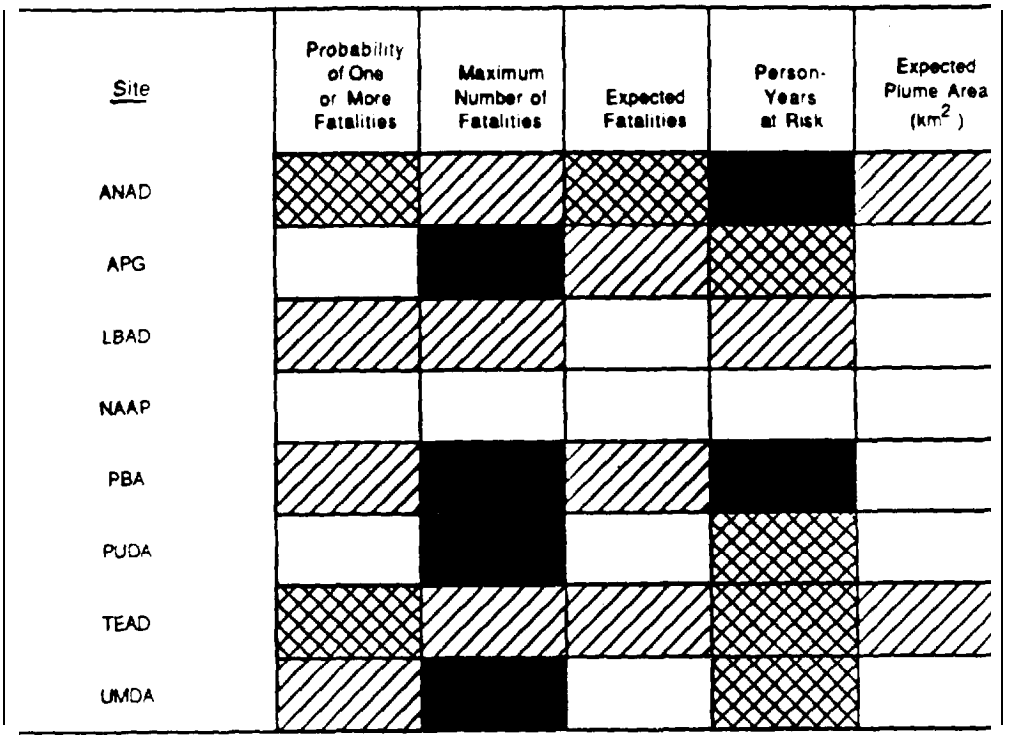


FIG J.27

SITE-SPECIFIC COMPARISON OF RISK FOR
REGIONAL DISPOSAL (REG)

(Risk along transportation corridors not included)

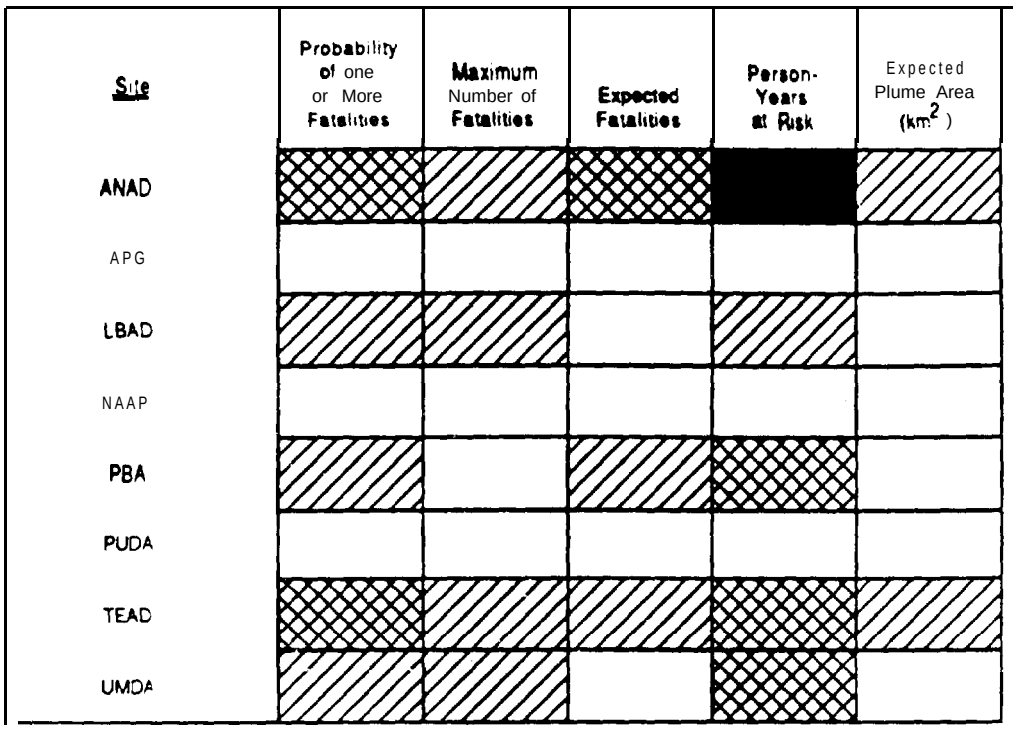


Numerical Equivalents

Legend: Relative Risk	Shading	Probability of One or More Fatalities	Maximum Number of Fatalities	Expected Fatalities	Person-Years at Risk	Expected Plume Area (km^2)
Higher		$>10^{-3}$	>10,000	$>10^{-2}$	$>10^{6}$	$>10^{-2}$
		$10^{-4} - 10^{-3}$	5000 - 10,000	$10^{-3} - 10^{-2}$	$10^{5} - 10^{6}$	$10^{-3} - 10^{-2}$
		$10^{-5} - 10^{-4}$	1000 - 5000	$10^{-4} - 10^{-3}$	$10^{4} - 10^{5}$	$10^{-4} - 10^{-3}$
Lower		$<10^{-5}$	<1000	$<10^{-4}$	$<10^{4}$	$<10^{-4}$

FIG. J.28

RISK WITH MITIGATION (REV. 1):
SITE-SPECIFIC COMPARISON FOR
REGIONAL DISPOSAL (REG)
(Risk along transportation corridors not included)



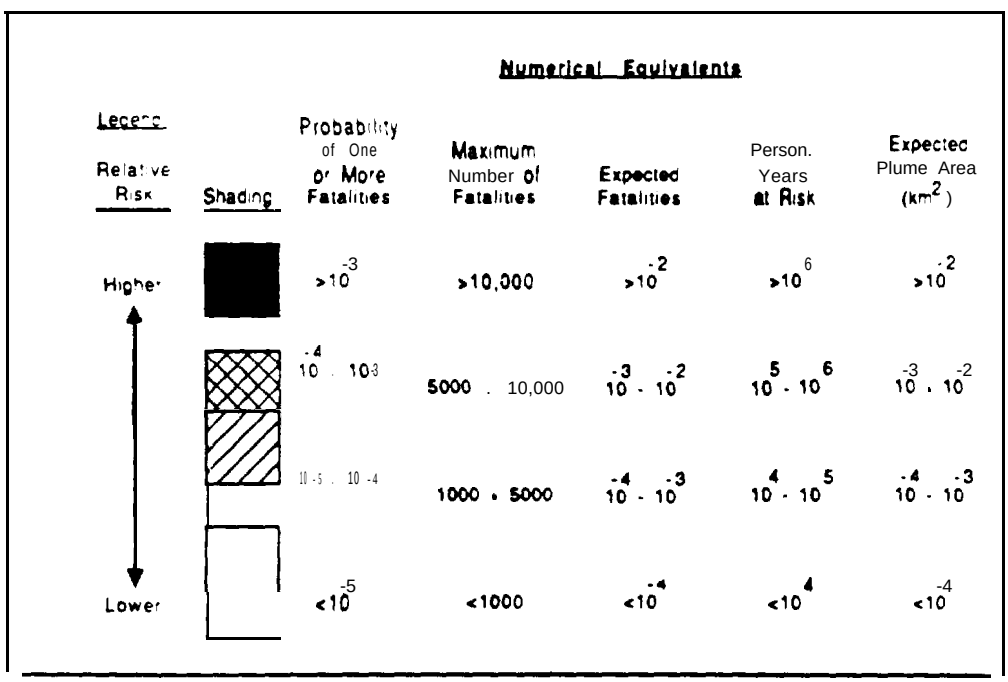
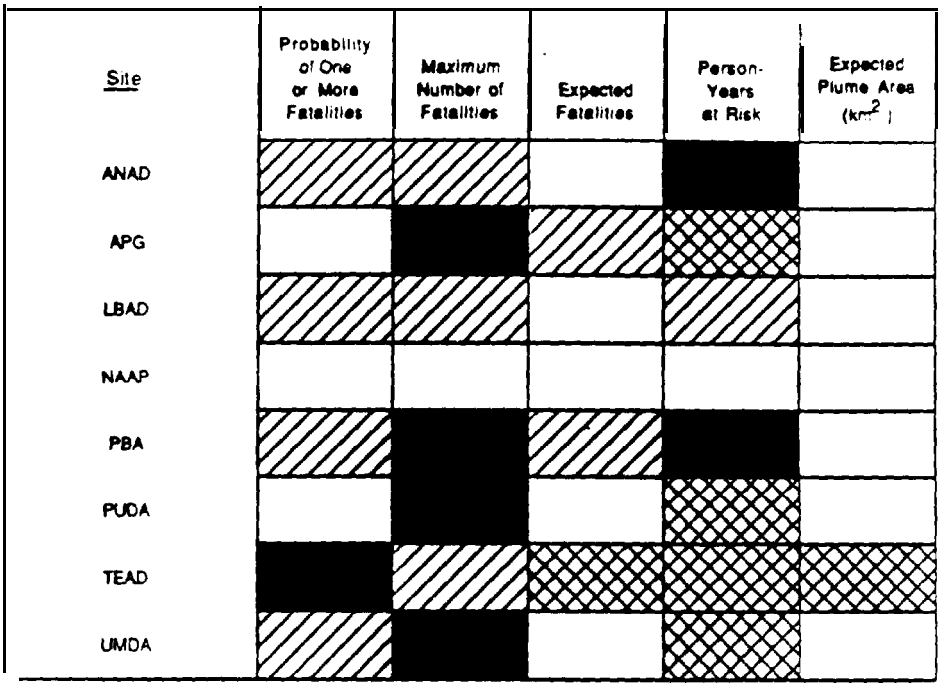


FIG. **J.29**

RISK WITH MITIGATION (REV. 2):
SITE-SPECIFIC COMPARISON FOR
REGIONAL DISPOSAL (REG)

(Risk along transportation corridors not included)



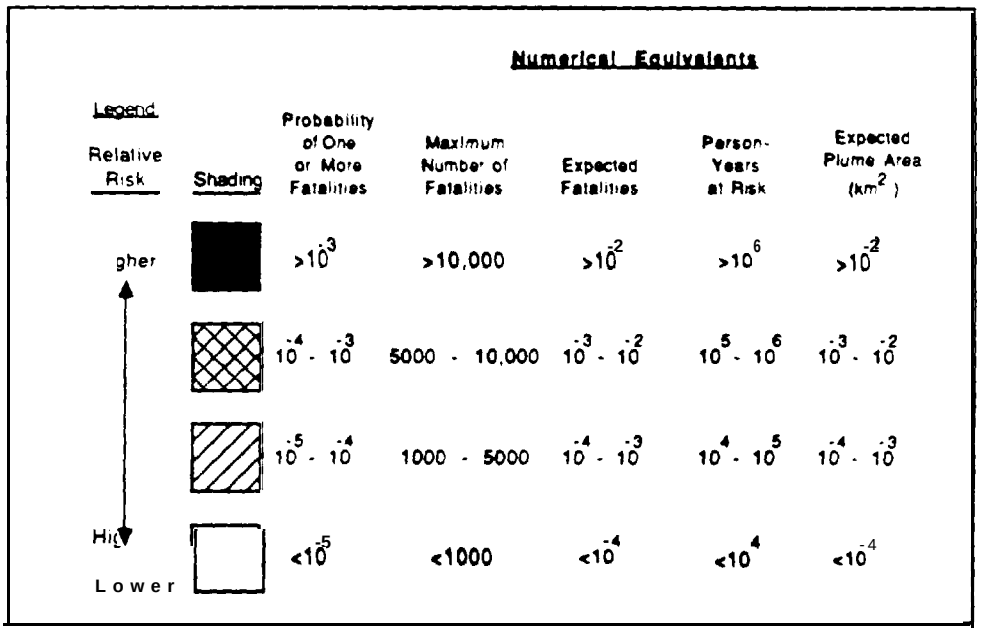
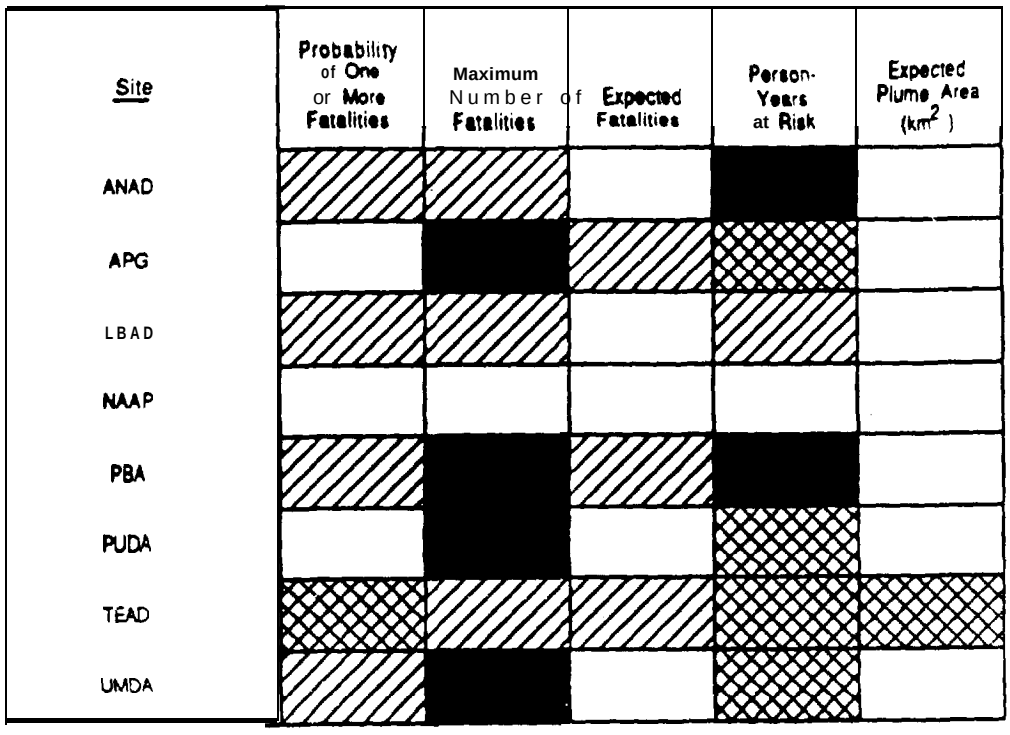


FIG. J.30

SITE-SPECIFIC COMPARISON OF RISK FOR NATIONAL DISPOSAL (NAT)

(Risk along transportation corridors not included)



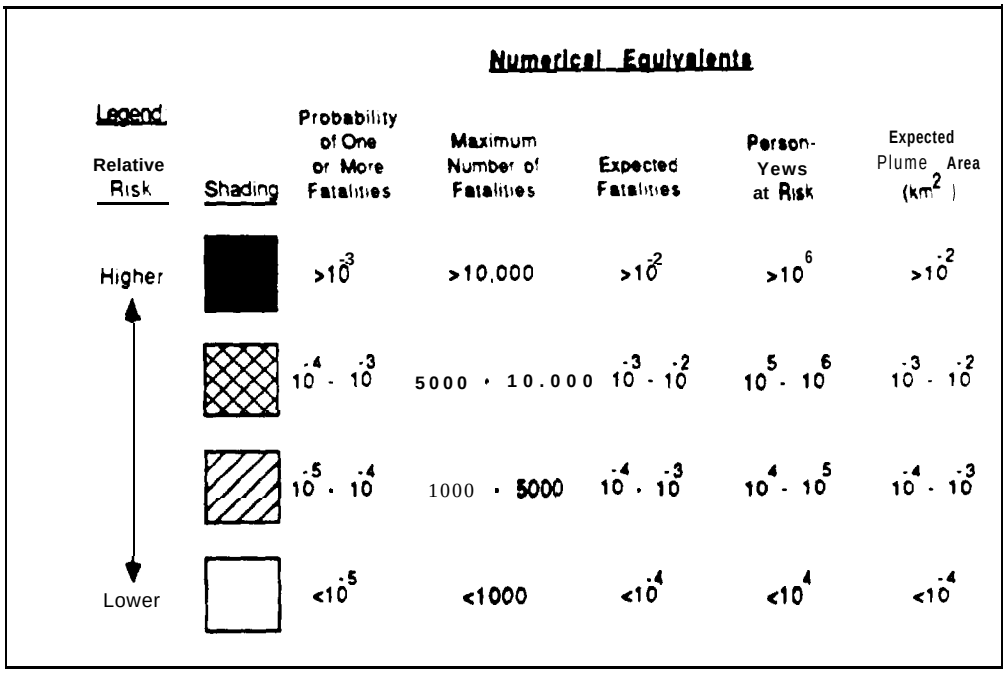
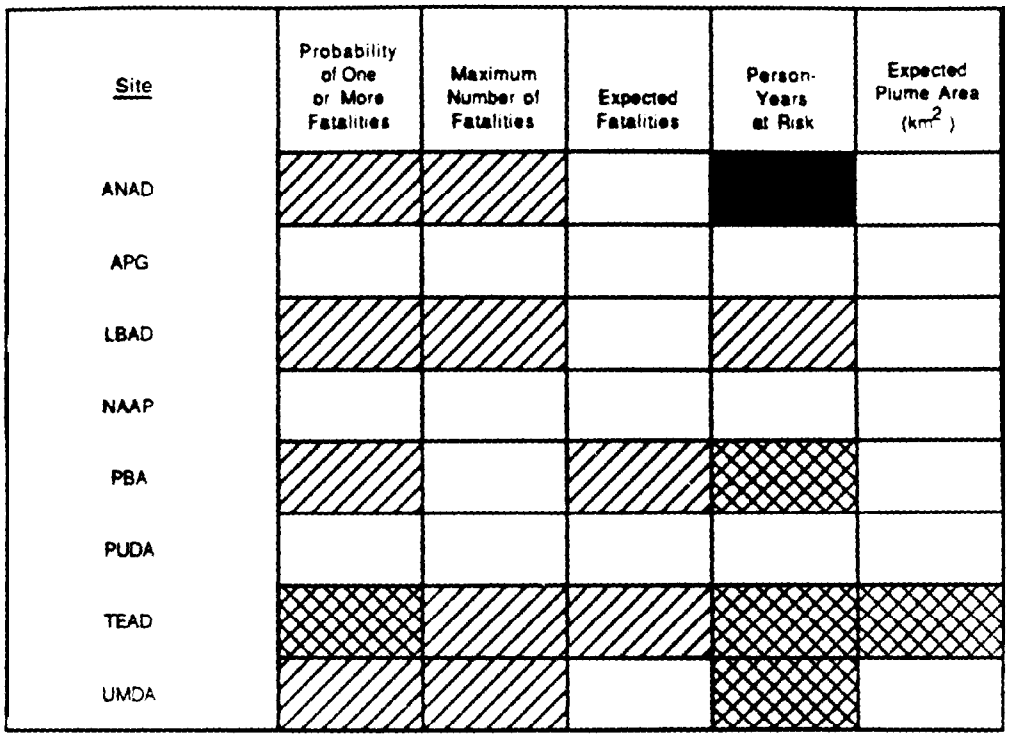


FIG. J.31

RISK WITH MITIGATION (REV. 1):
SITE-SPECIFIC COMPARISON FOR
NATIONAL DISPOSAL (NAT)

(Risk along transportation corridors not included)



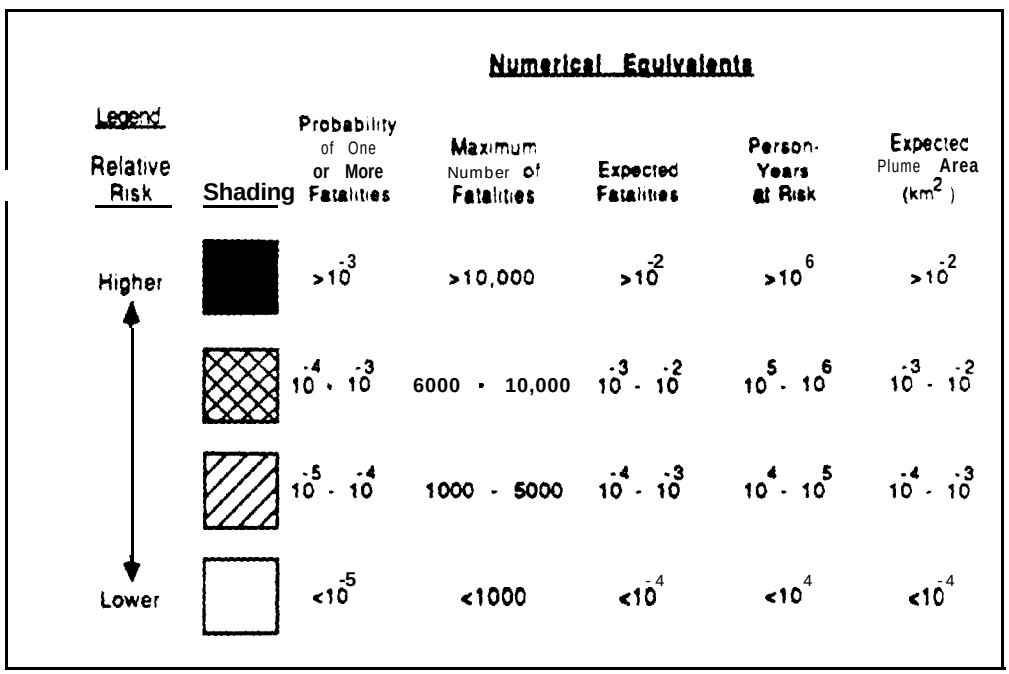
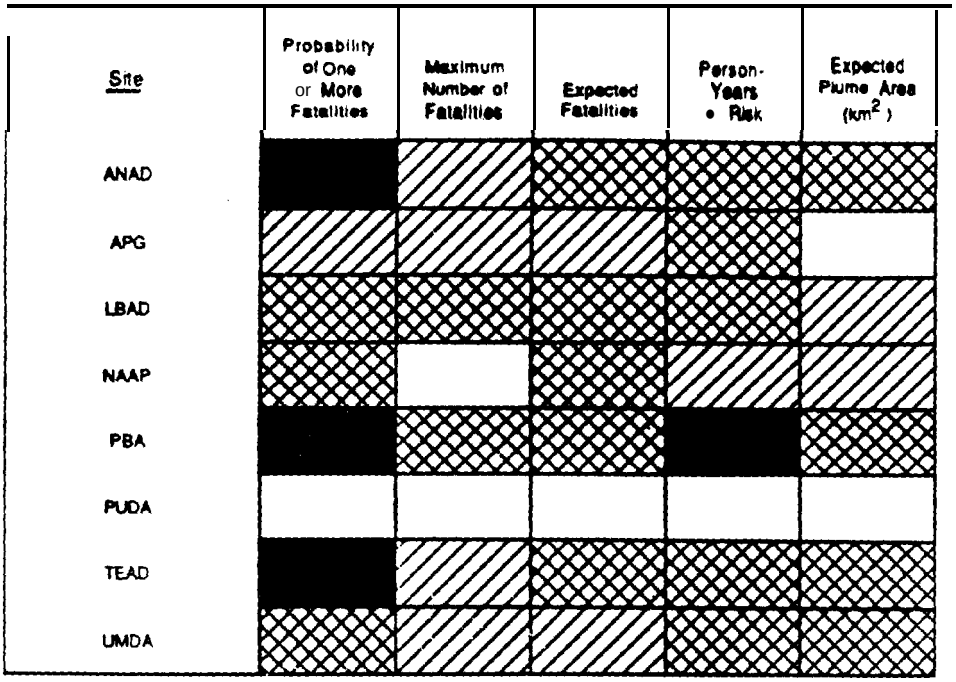


FIG. 5.32

RISK WITH MITIGATION (REV. 2): SITE-SPECIFIC COMPARISON FOR NATIONAL DISPOSAL (NAT)

(Risk along transportation corridors not included)



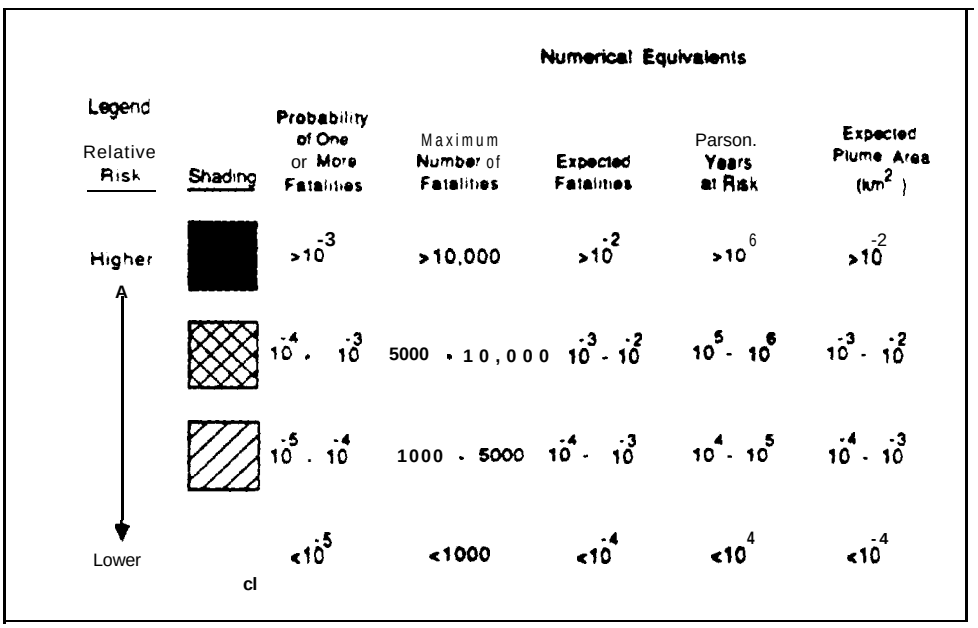
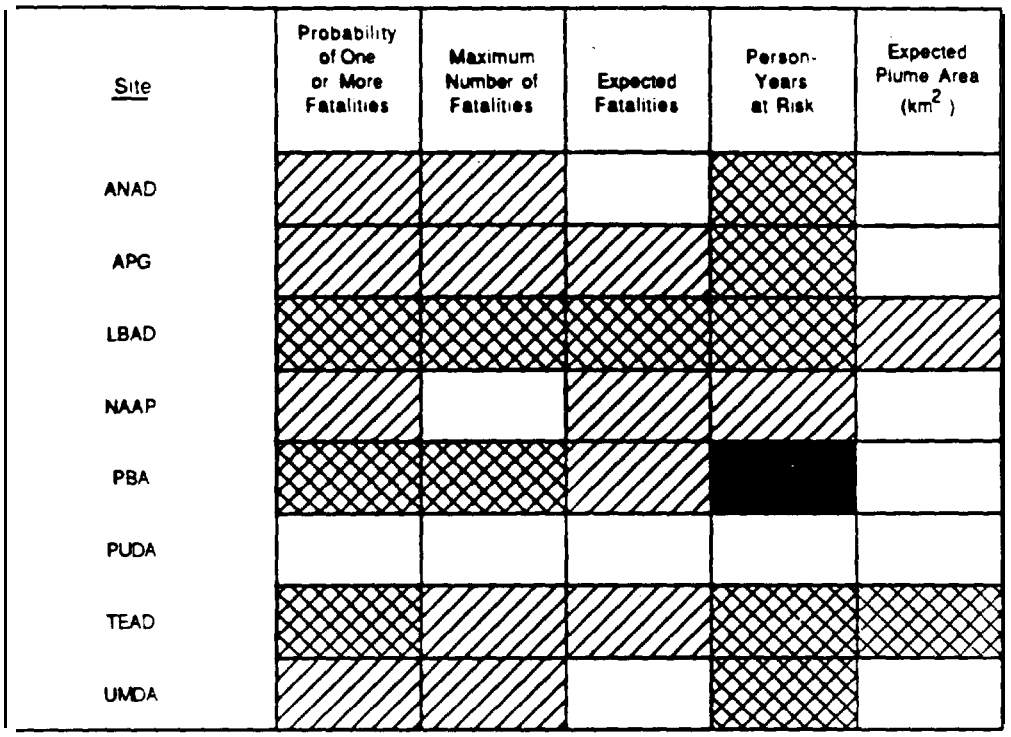


FIG. J.33

SITE-SPECIFIC COMPARISON OF RISK FOR PARTIAL RELOCATION (PR)

(Risk **along trrnsponetion corridors not included)**



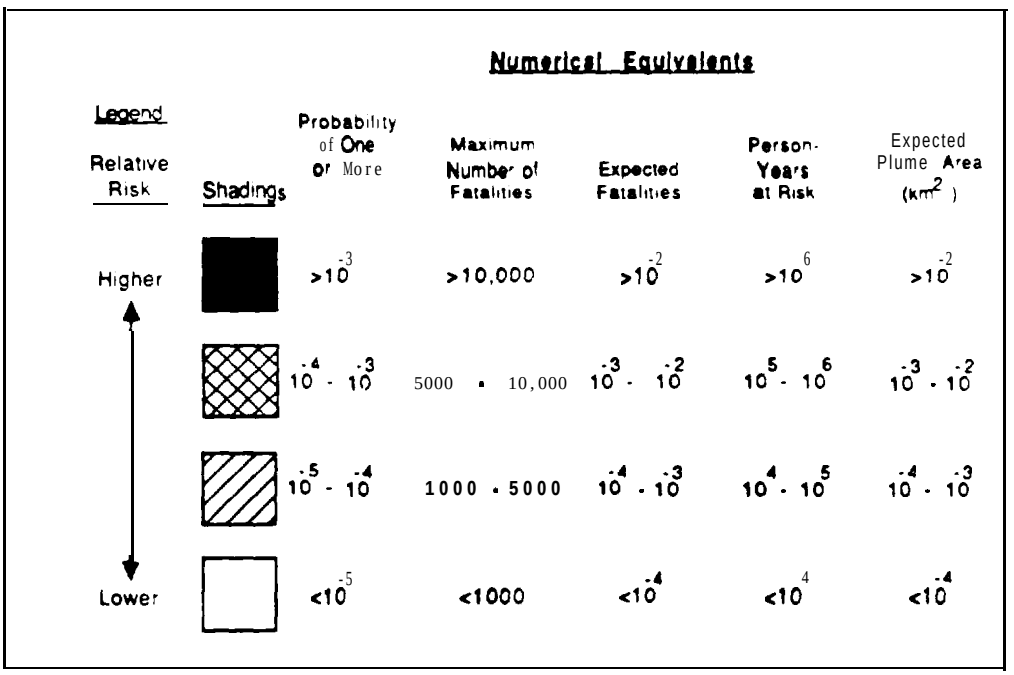
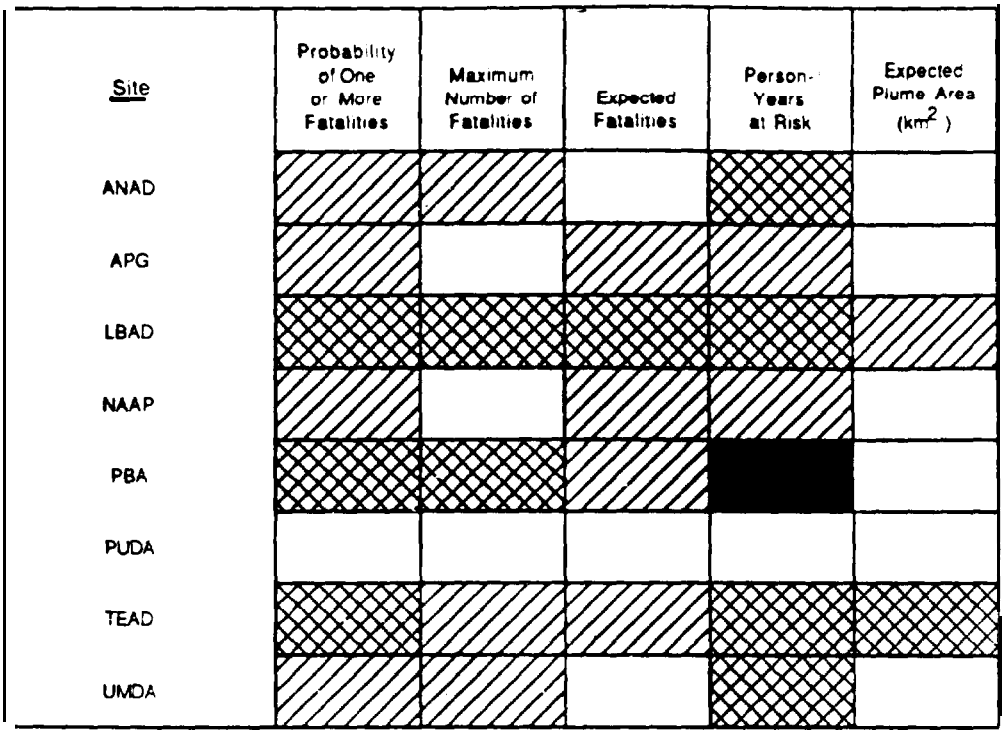


FIG. 5.34

RISK WITH MITIGATION (REV. 1): SITE-SPECIFIC COMPARISON FOR PARTIAL RELOCATION (PR)

(Risk along transportation corridors not included)



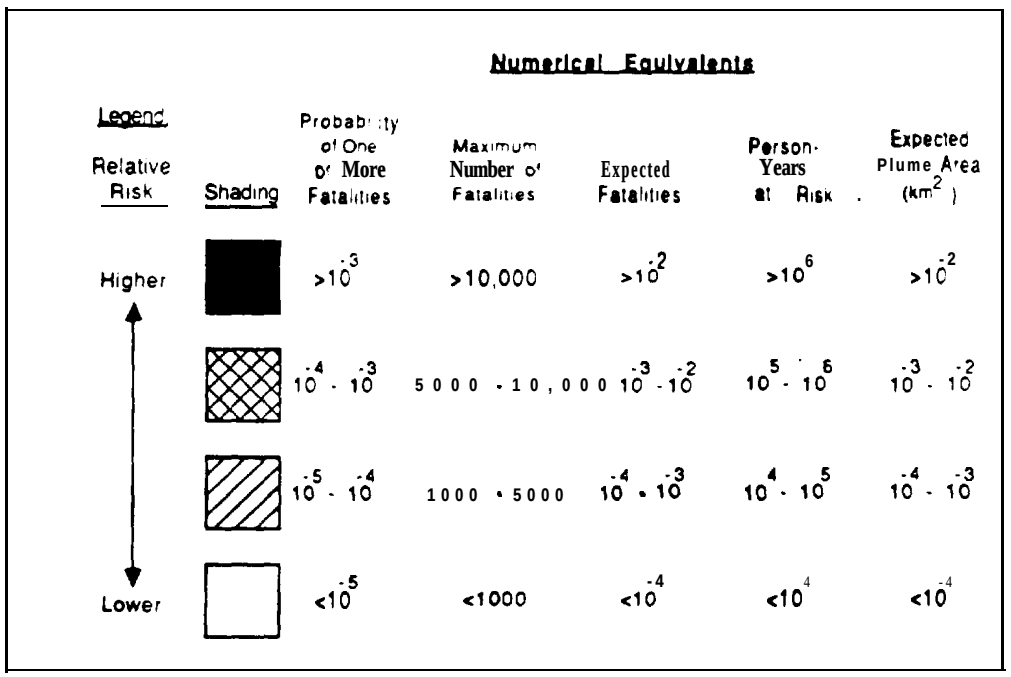
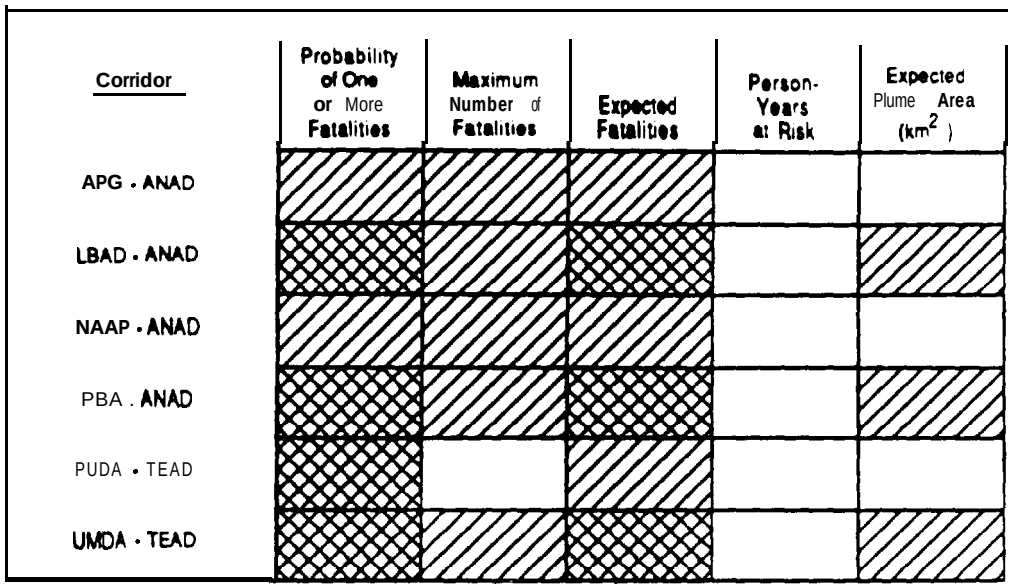


FIG. J.35

RISK WITH MITIGATION (REV. 2):
SITE-SPECIFIC COMPARISON FOR
PARTIAL RELOCATION (PR)

(Risk along transportation corridors **not** included)



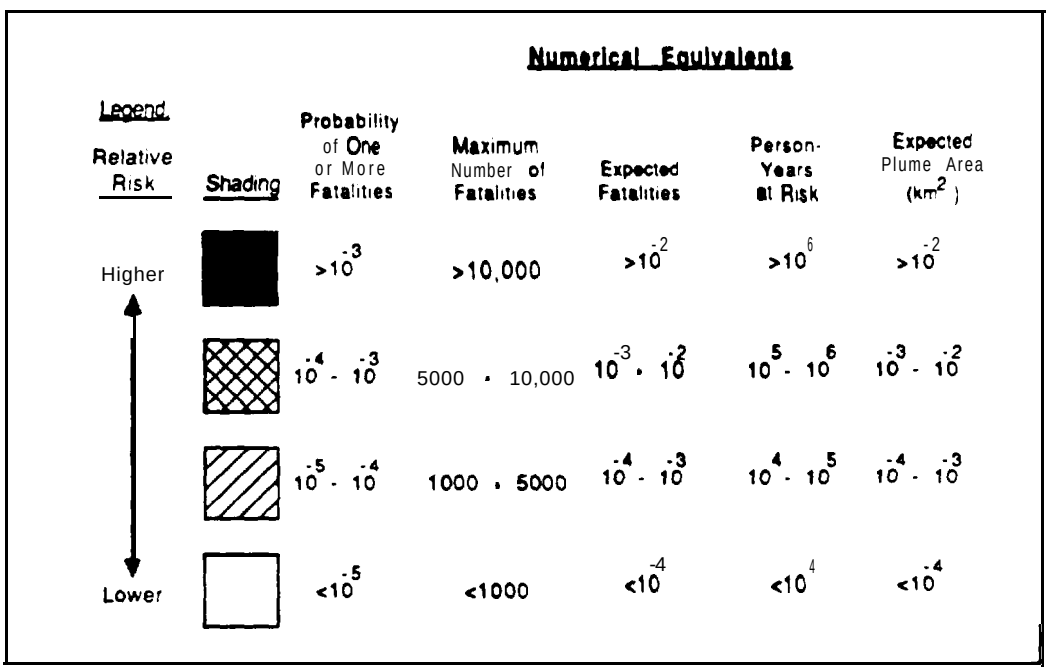
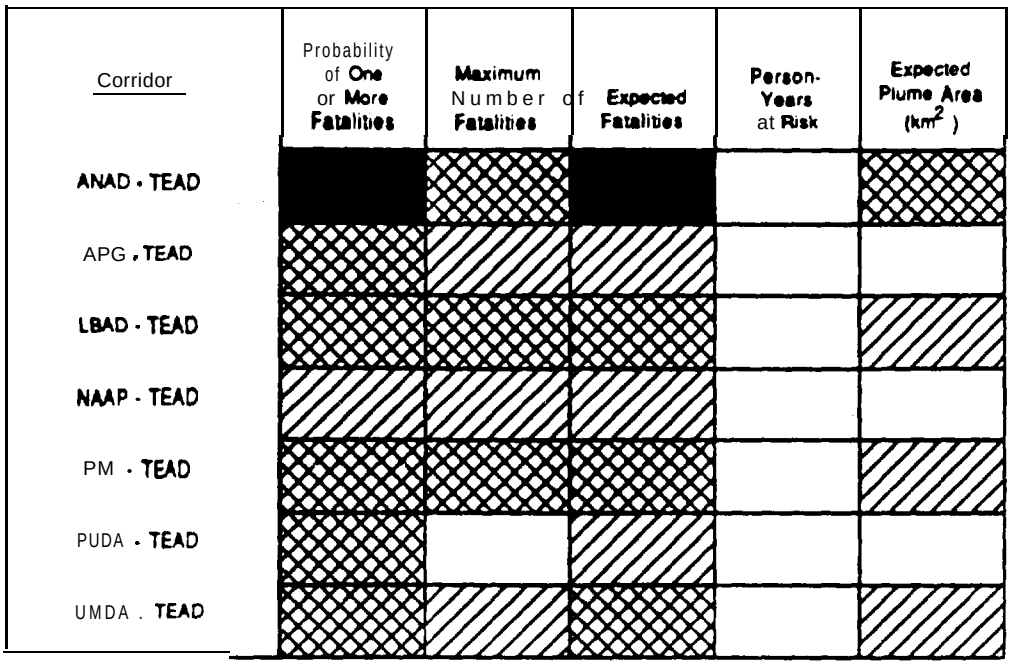
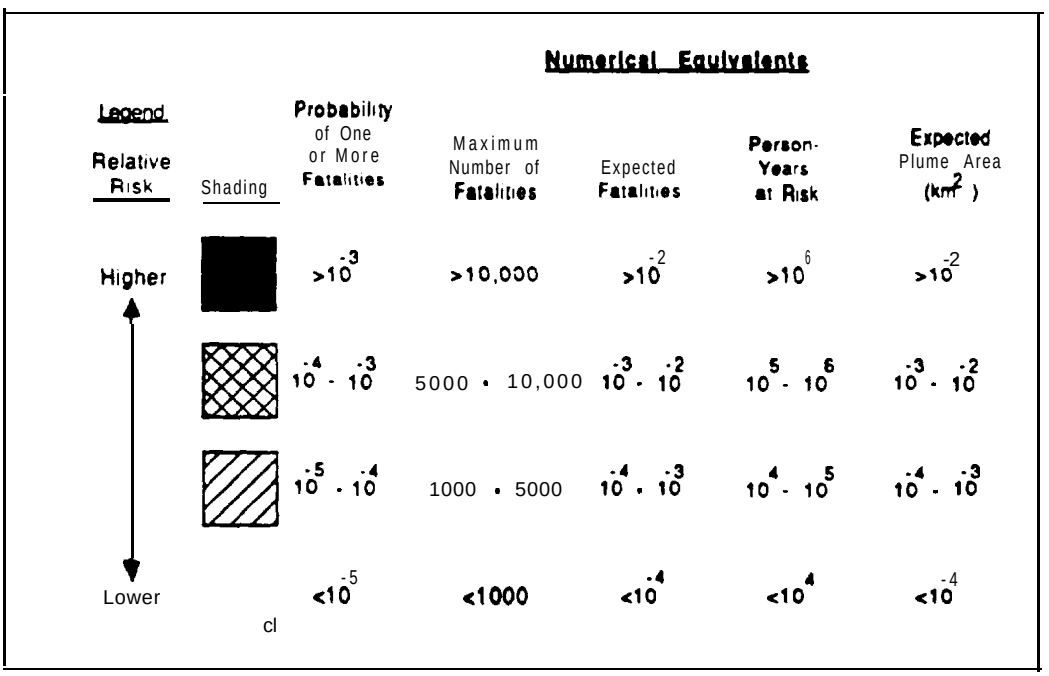


FIG. 5.36

RISK ALONG RAIL TRANSPORTATION CORRIDORS FOR REGIONAL DISPOSAL • ALL MITIGATION LEVELS •

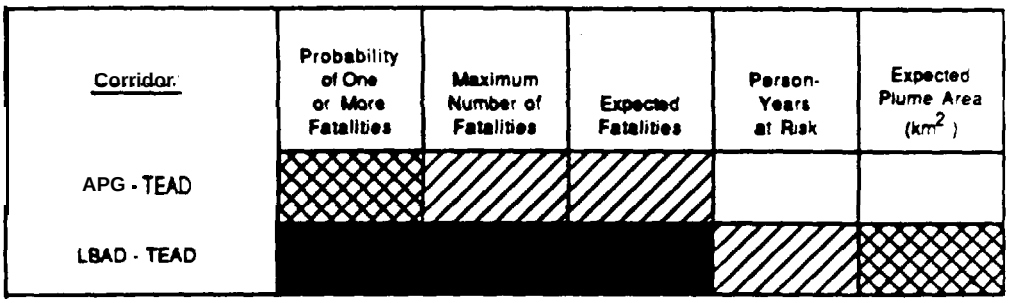




cl

FIG. 3.37

RISK ALONG RAIL TRANSPORTATION CORRIDORS FOR NATIONAL DISPOSAL - ALL MITIGATION LEVELS -



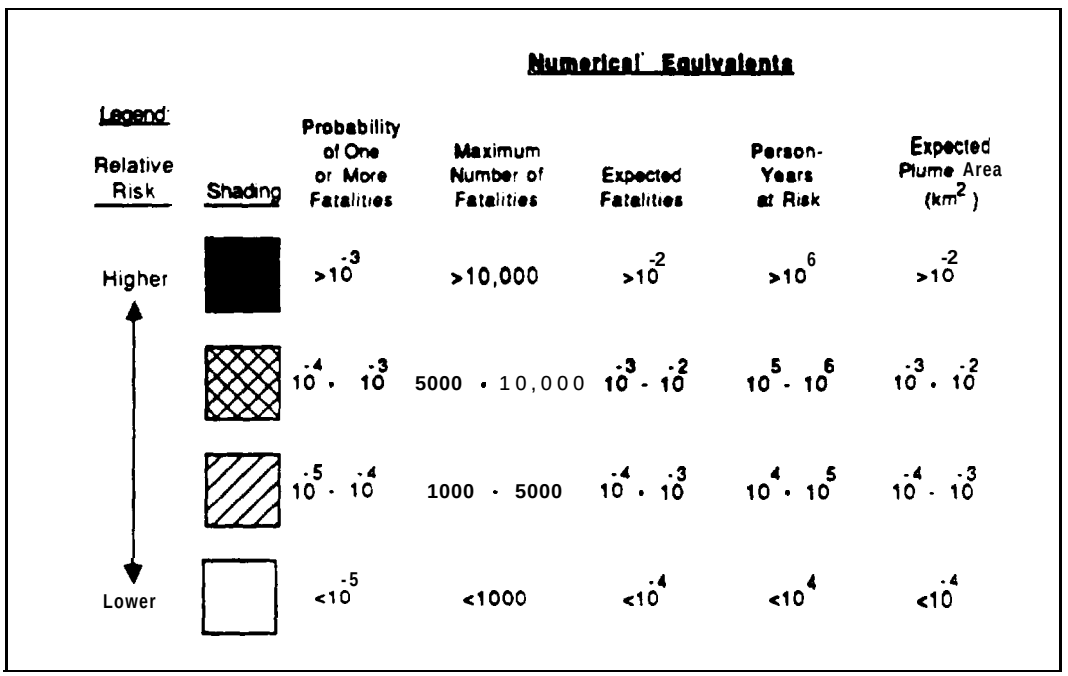
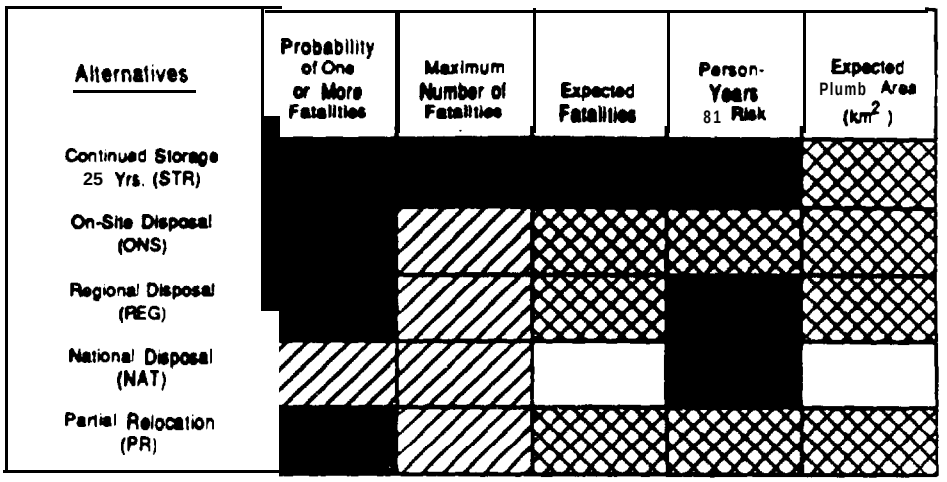


FIG. J.38

RISK ALONG AIR TRANSPORTATION CORRIDOR FOR PARTIAL RELOCATION DISPOSAL • ALL MITIGATION LEVELS •



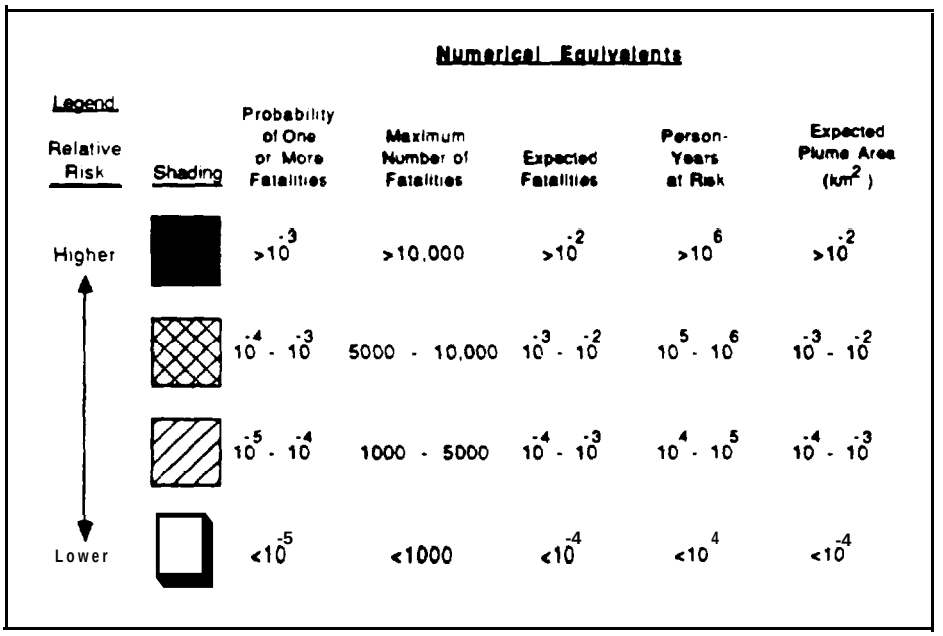
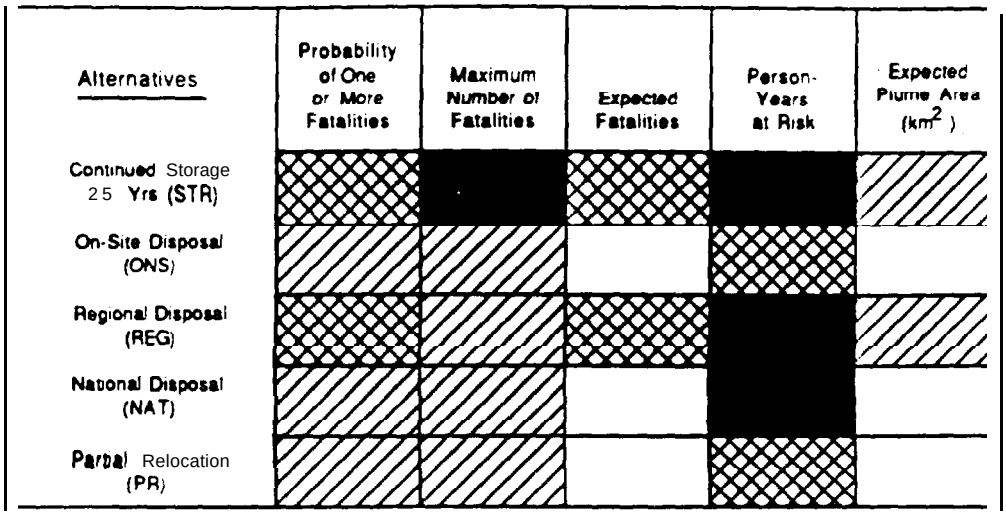


FIG. J.30
RISK IN THE VICINITY OF
ANNISTON ARMY DEPOT **(ANAD)**
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transponation corridors not included)



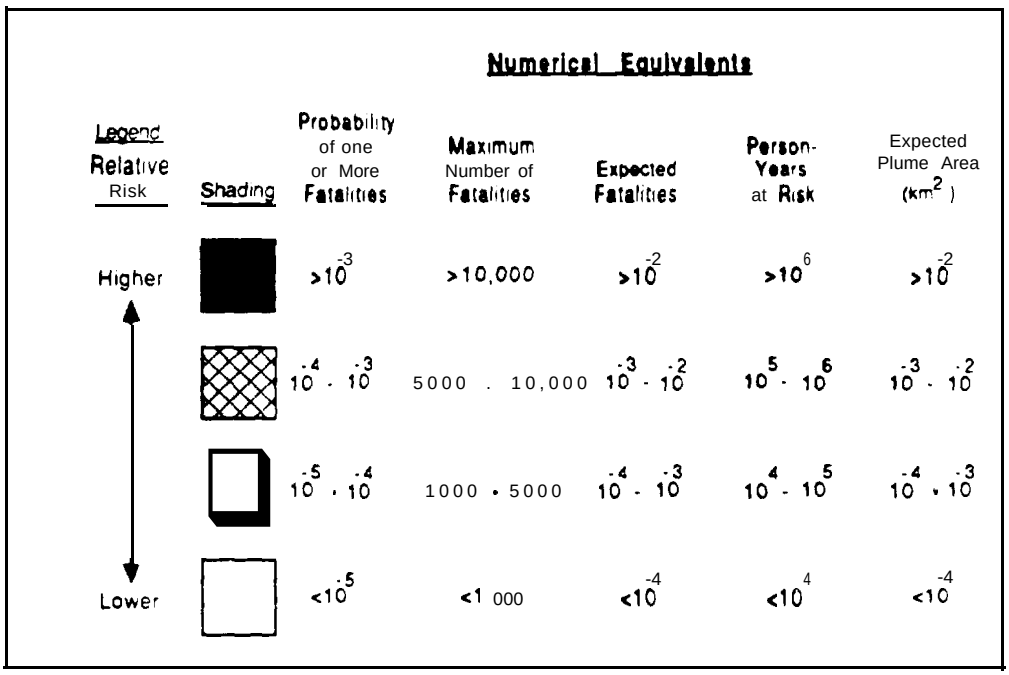
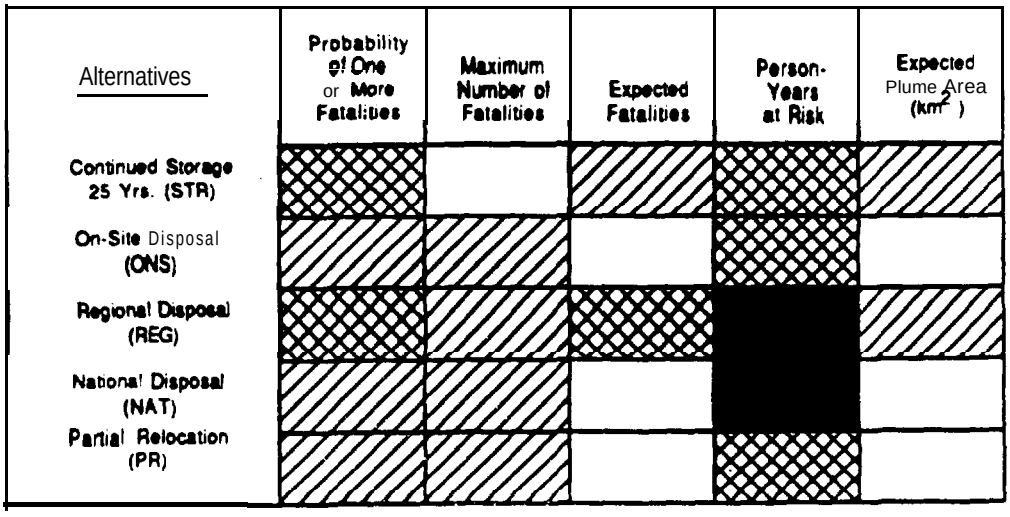


FIG. **J.40**

RISK, WITH MITIGATION (REV. **1),**
IN THE VICINITY OF
ANNISTON ARMY DEPOT **(ANAD)**
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



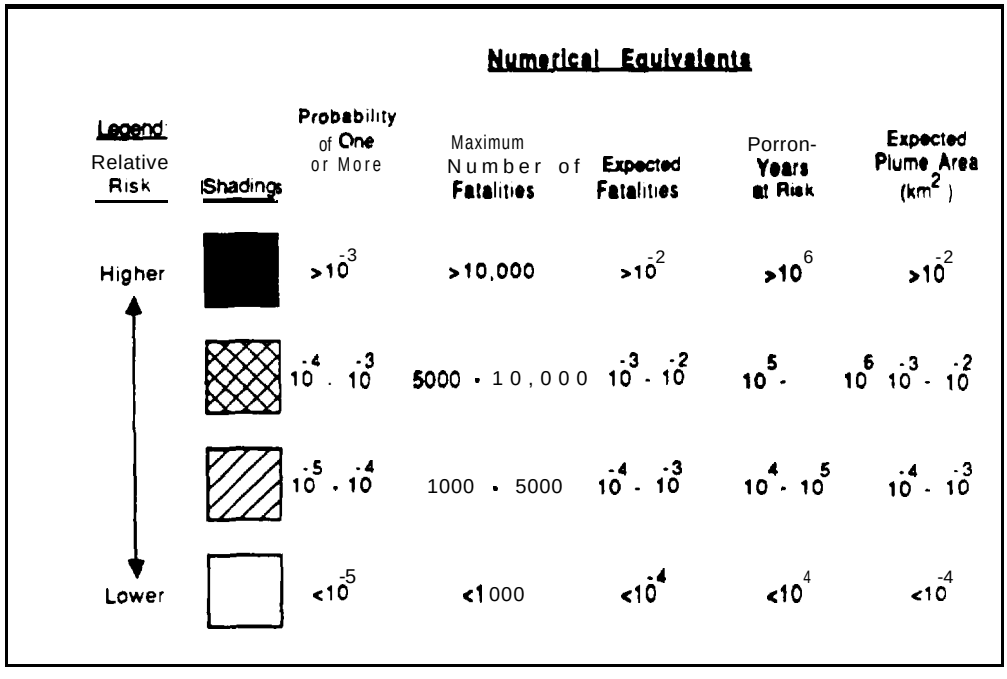
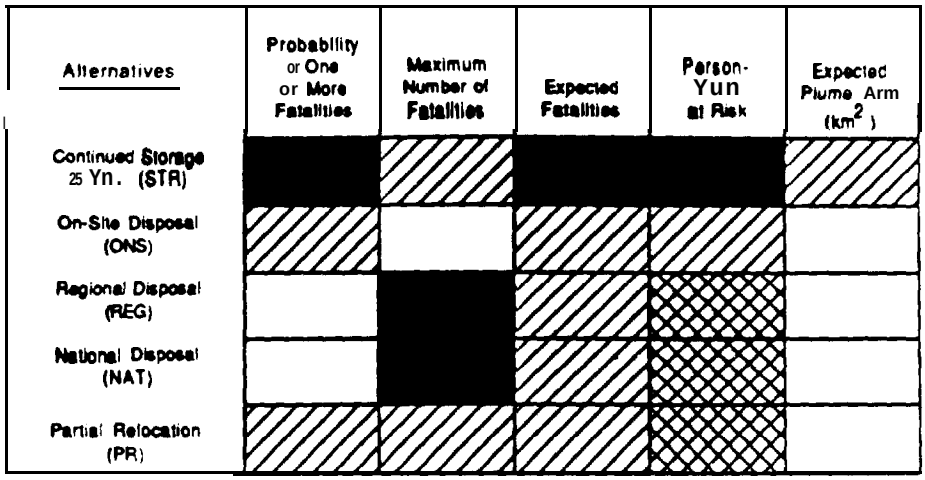


FIG. 5.41

RISK, WITH MITIGATION (REV. 2),
IN THE VICINITY OF
ANNISTON ARMY DEPOT (ANAD)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



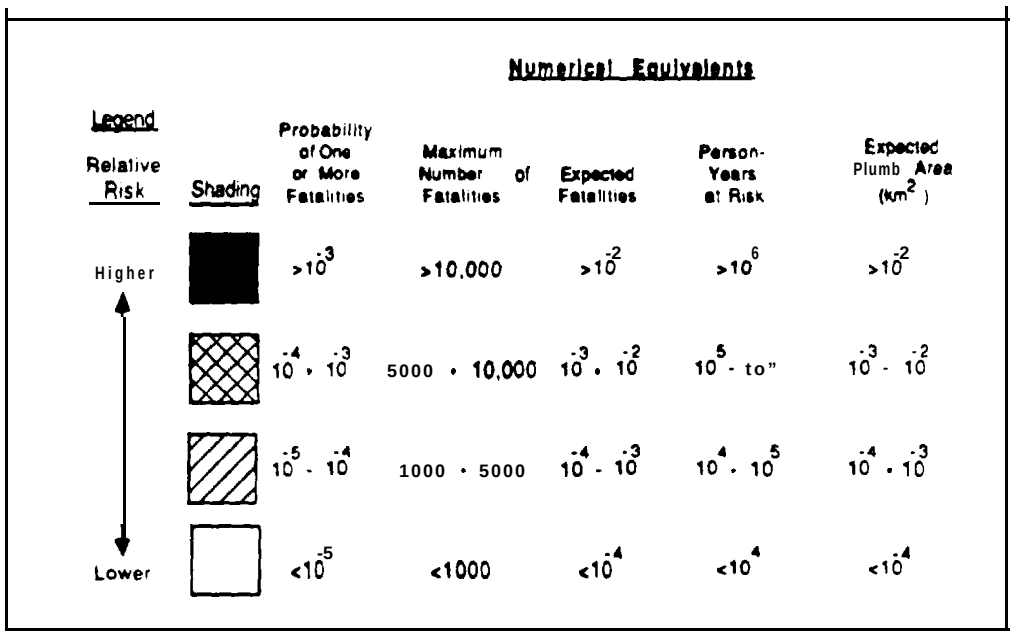
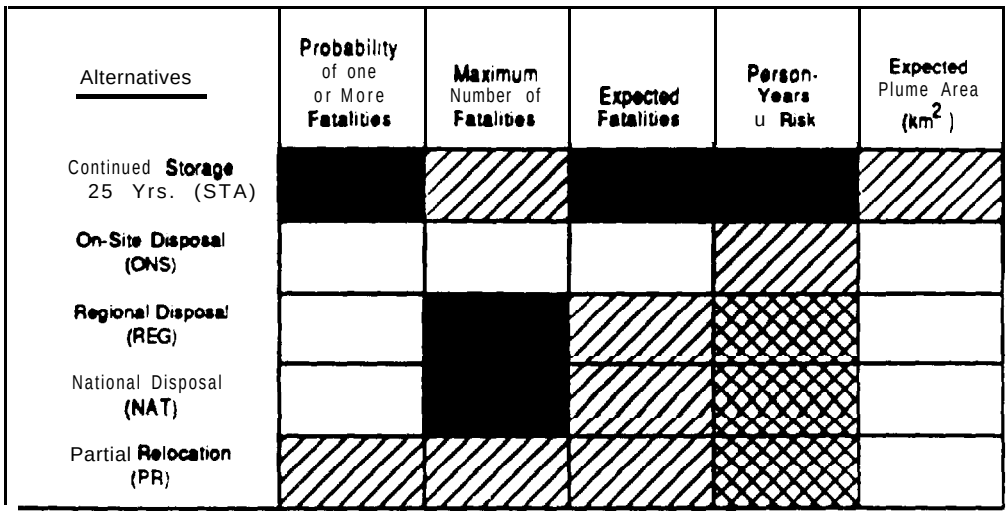


FIG. J.42

RISK IN THE VICINITY OF ABERDEEN PROVING GROUND (APG) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors or not included)



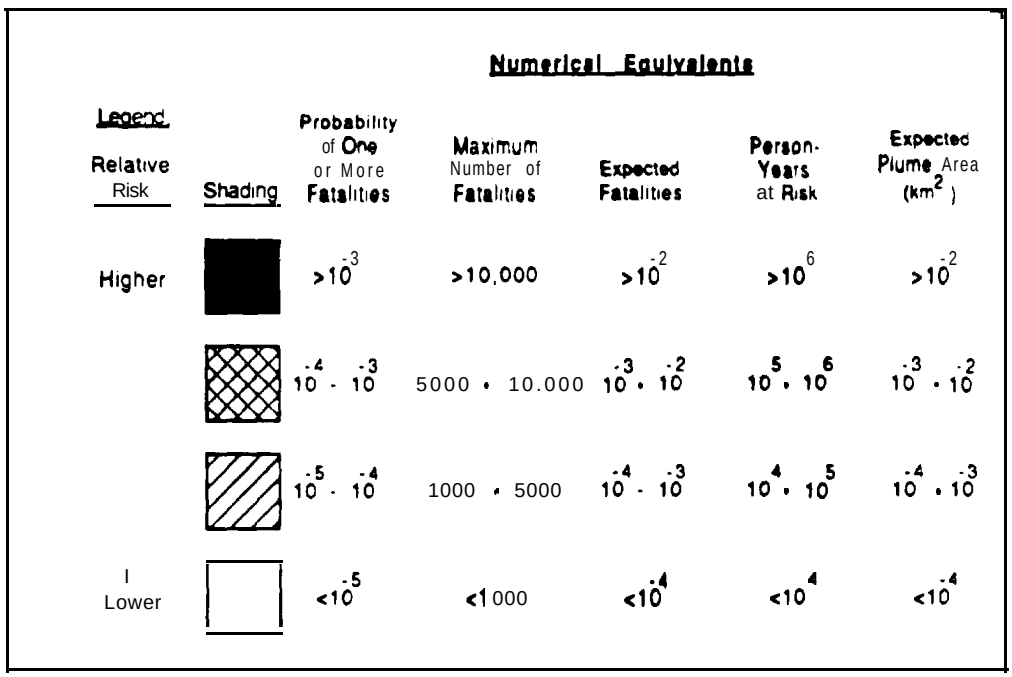
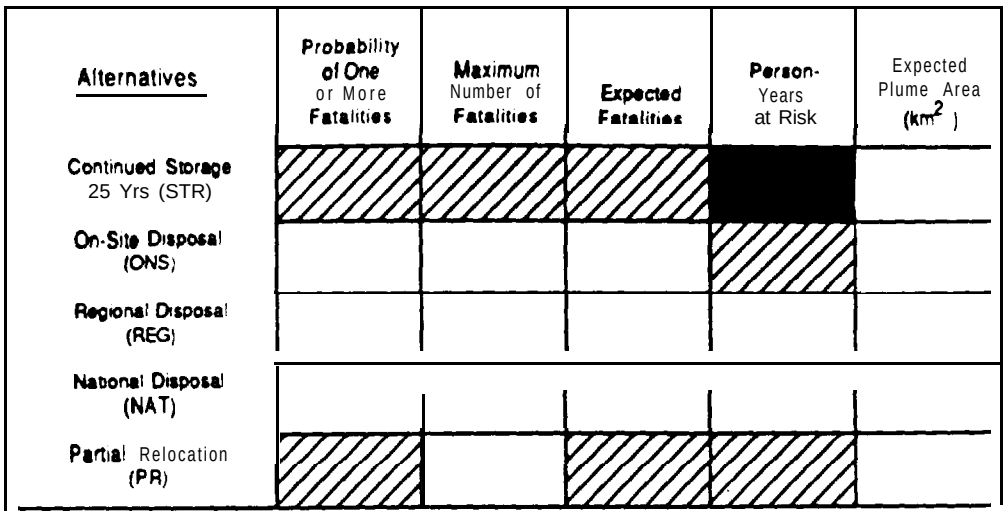


FIG. 5.43

RISK, WITH MITIGATION (REV. 1), IN THE VICINITY OF ABERDEEN PROVING GROUND (APG) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



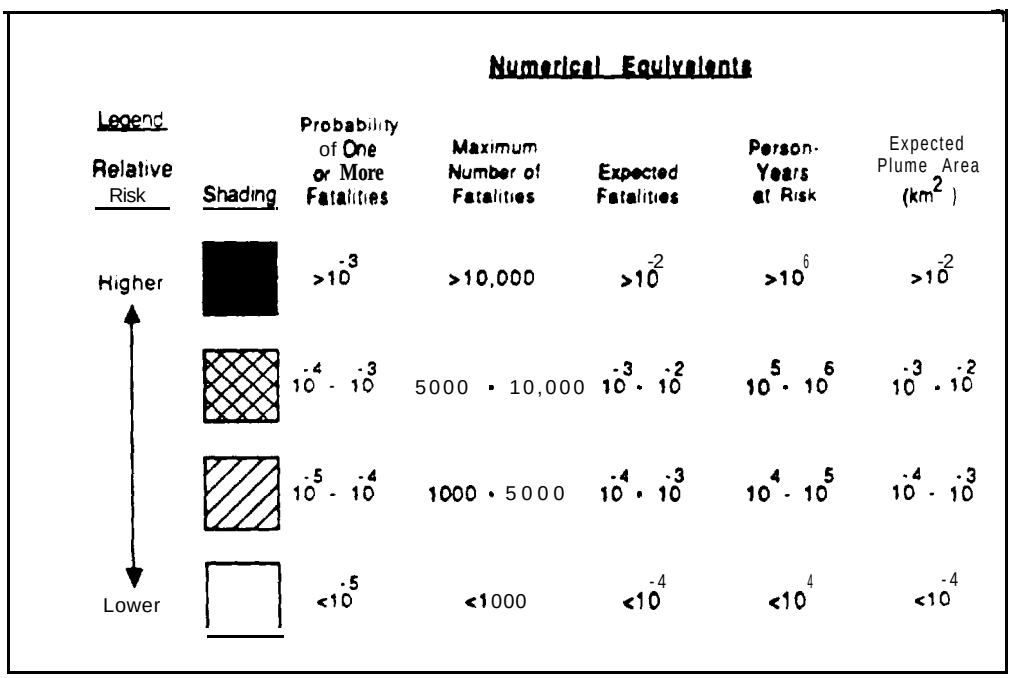
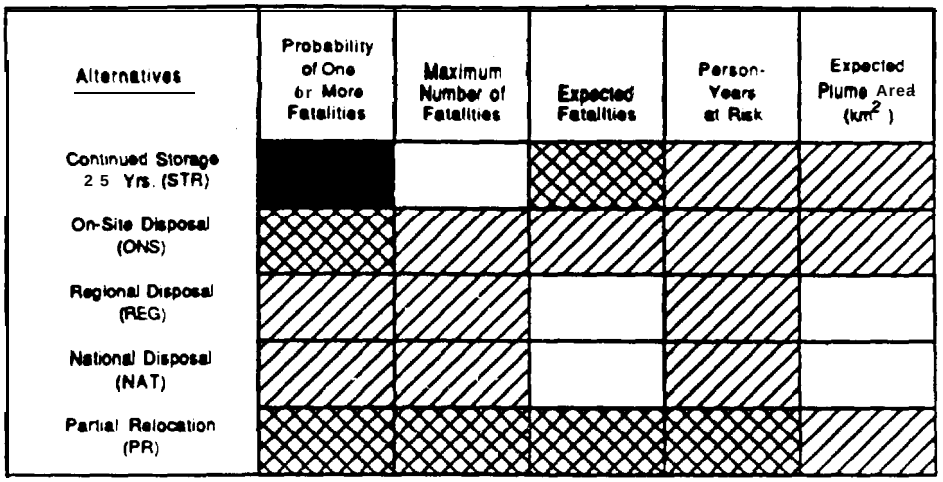


FIG. 5.44

RISK, WITH MITIGATION (REV. 2), IN THE VICINITY OF ABERDEEN PROVING GROUND (APG) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



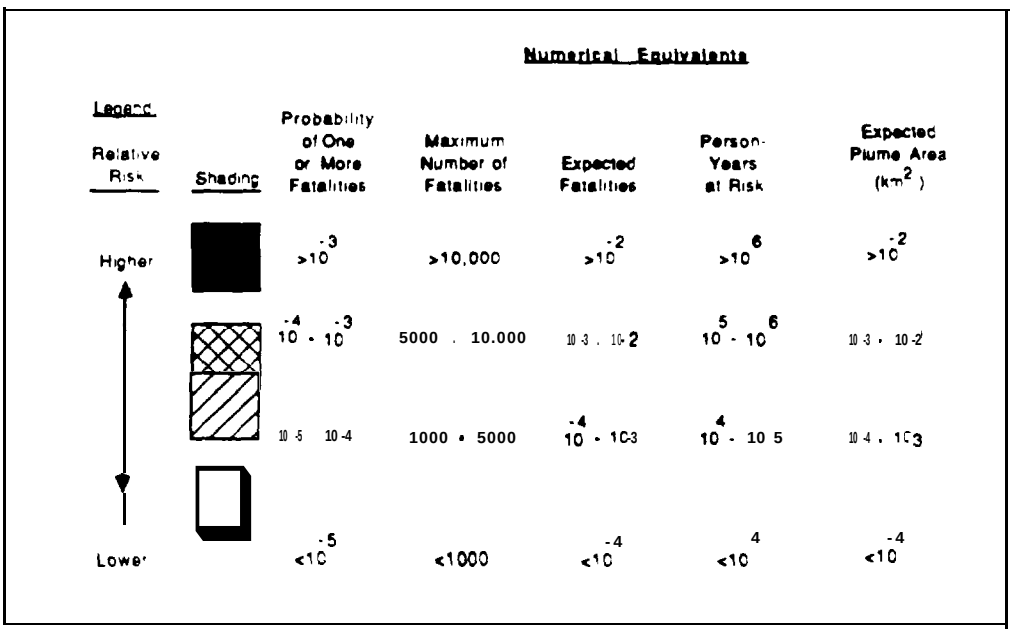
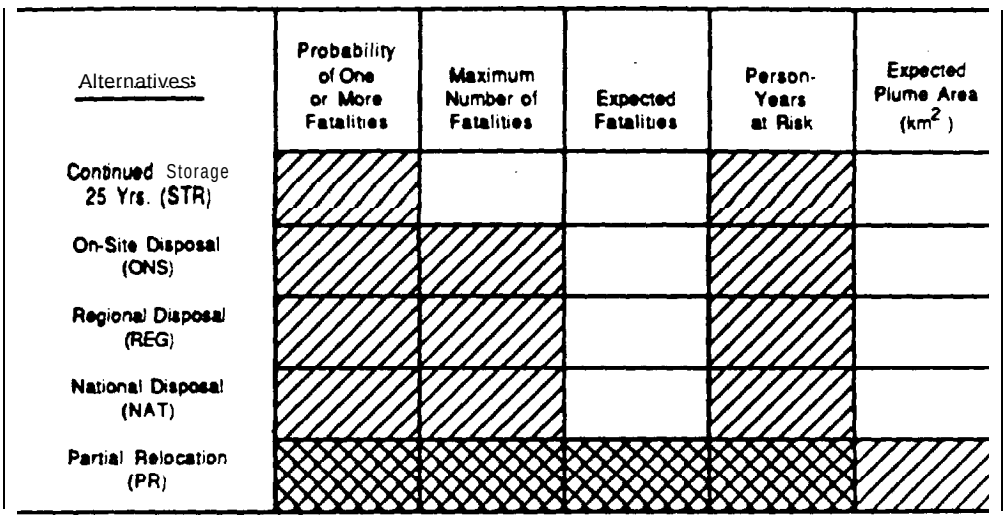


FIG. J.46

RISK IN THE VICINITY OF
LEXINGTON-BLUE GRASS ARMY DEPOT (LBAD)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



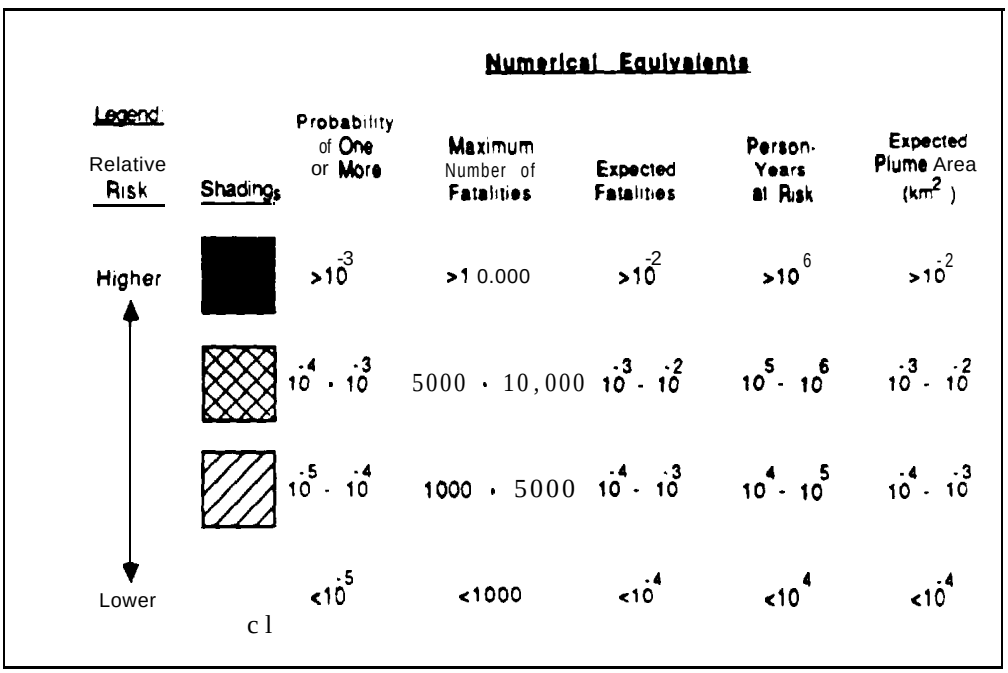
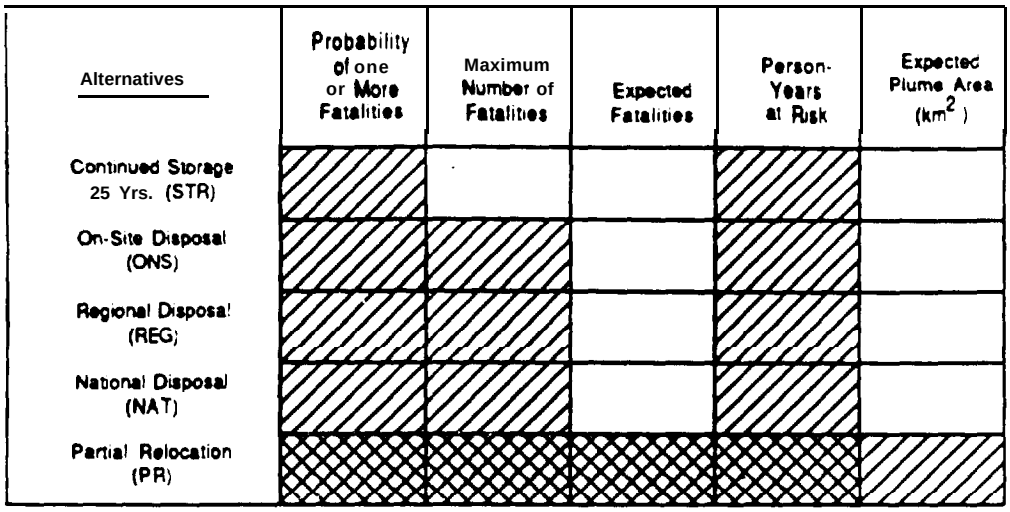


FIG. 5.46

RISK, WITH MITIGATION (REV. 1), IN THE VICINITY OF LEXINGTON-BLUE GRASS ARMY -DEPOT (LBAD) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



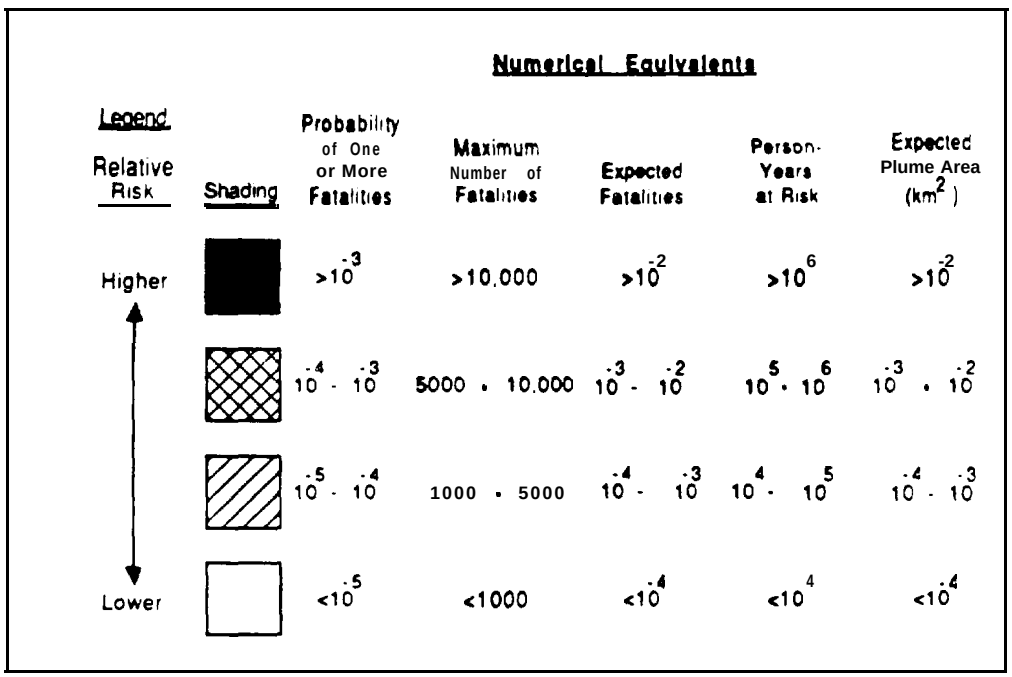
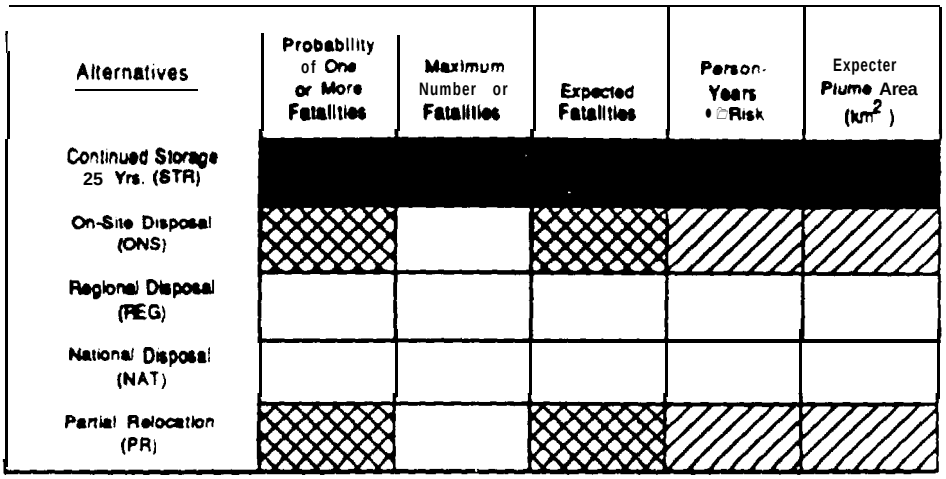


FIG. 5.47

RISK, WITH MITIGATION (REV. **2),**
IN THE **VICINITY** OF
LEXINGTON-BLUE GRASS ARMY **DEPOT** (LBAD)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along **transportation** corridors not included)



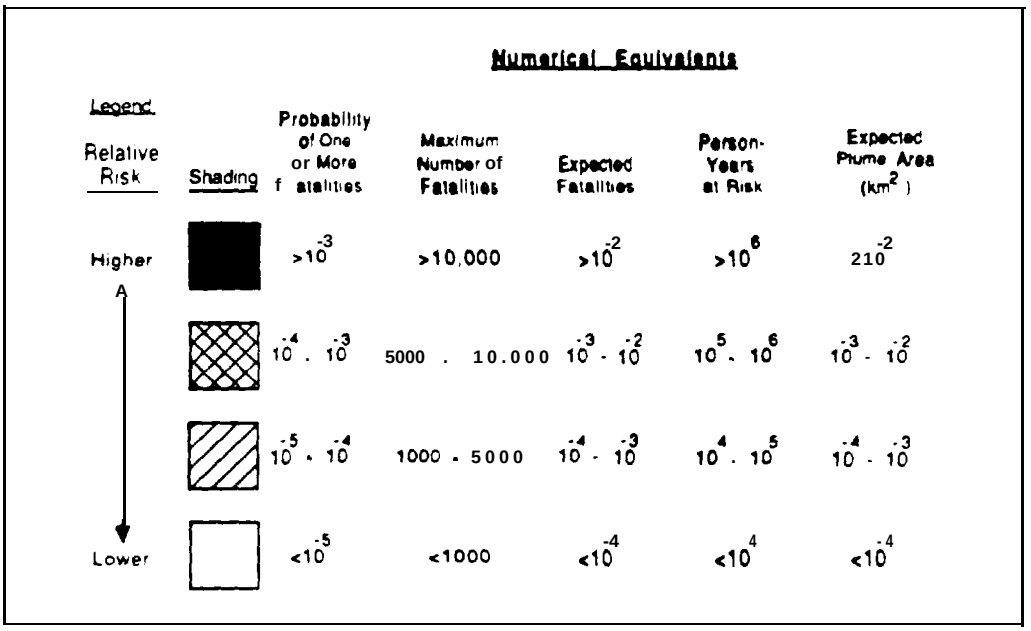
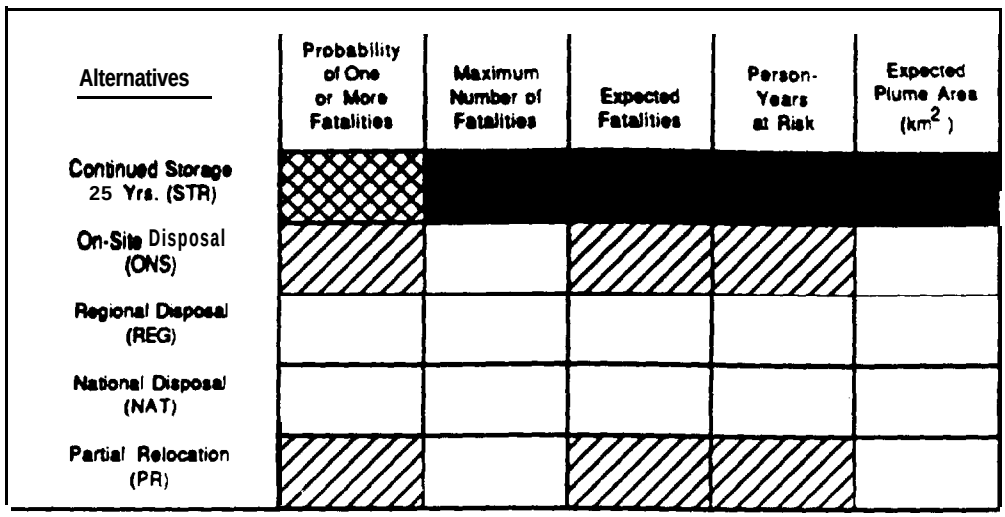


FIG. J.48

RISK IN THE VICINITY OF NEWPORT ARMY AMMUNITION PLANT (NAAP) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridor not included!



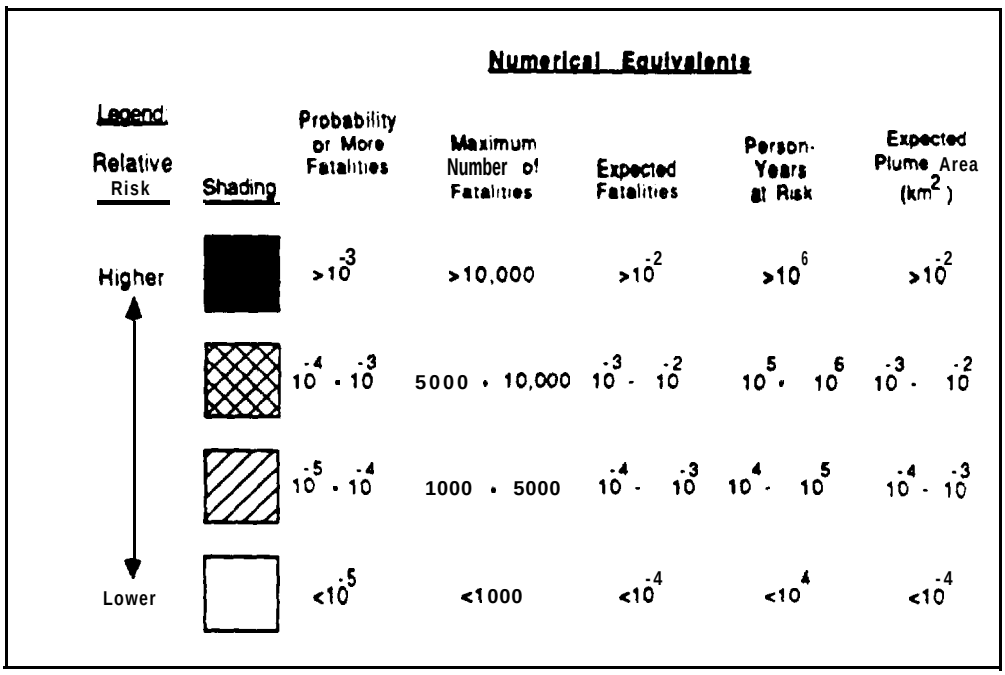
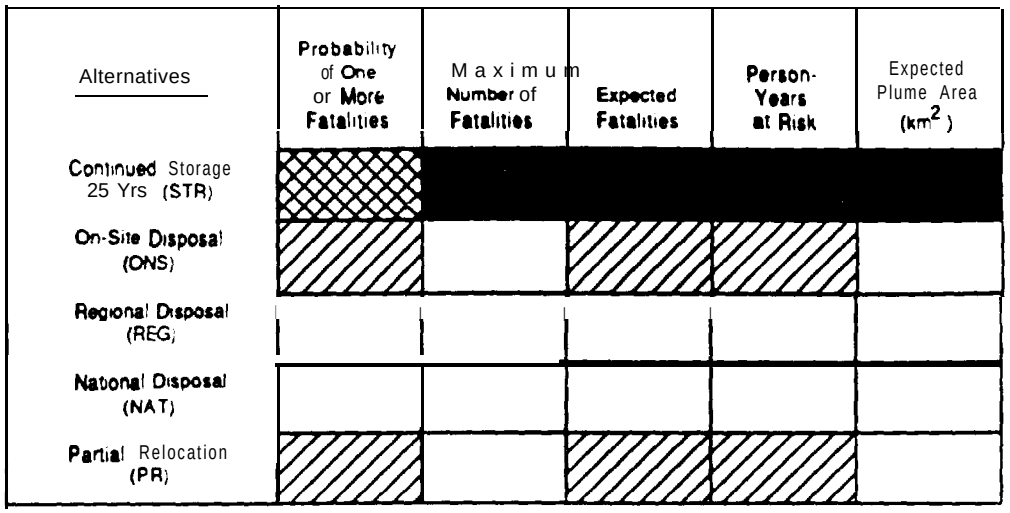


FIG. J.49

RISK, WITH MITIGATION (REV. 1), IN THE VICINITY OF NEWPORT ARMY AMMUNITION PLANT (NAAP) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



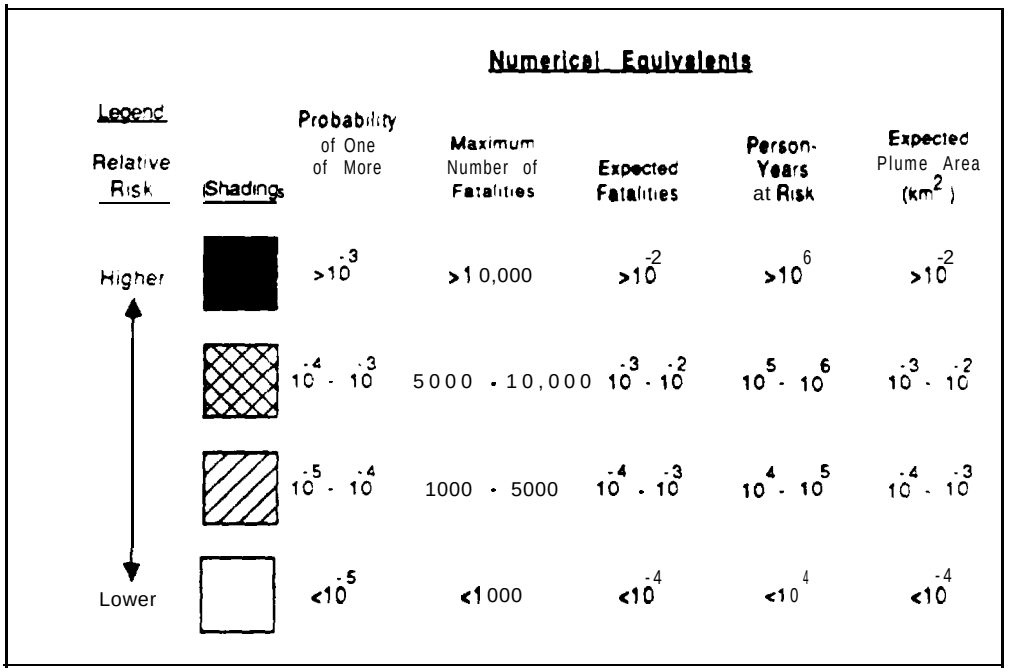
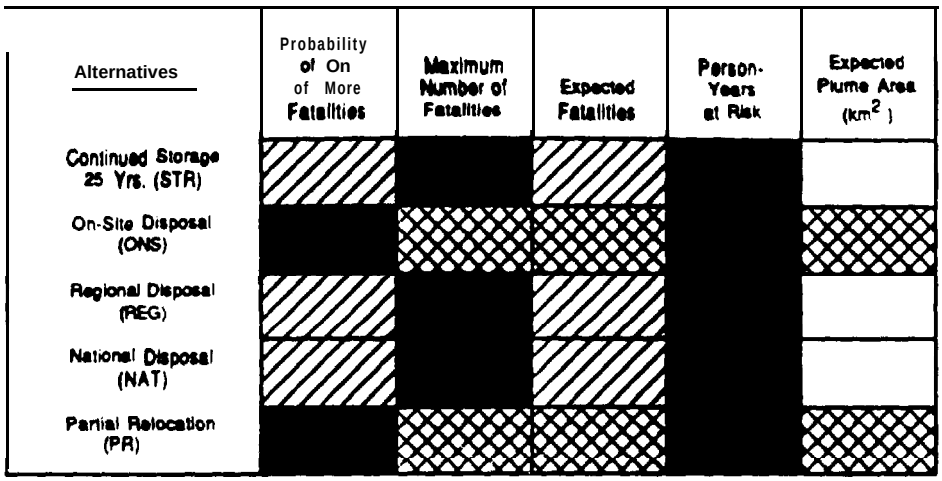


FIG. J.50

RISK, WITH MITIGATION (REV. 2),
IN THE VICINITY OF
NEWPORT ARMY AMMUNITION PLANT (NAAP)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



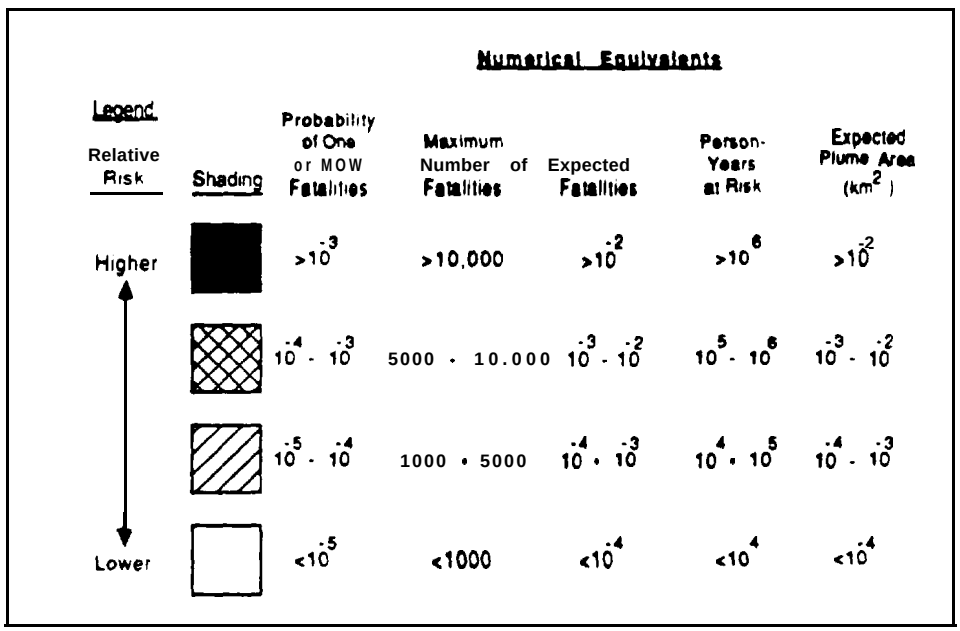
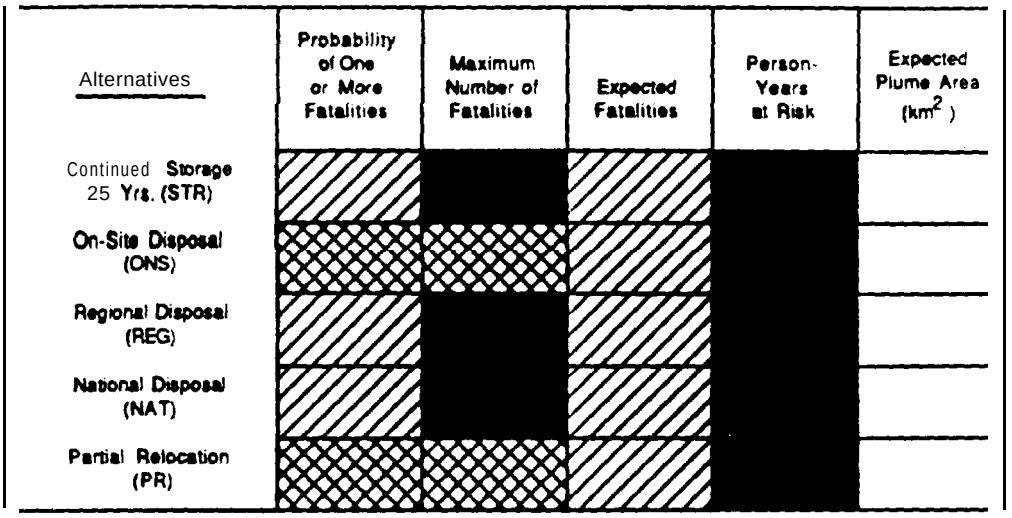


FIG. J.51

RISK IN THE VICINITY OF
PINE BLUFF ARSENAL (PBA)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors or not included)



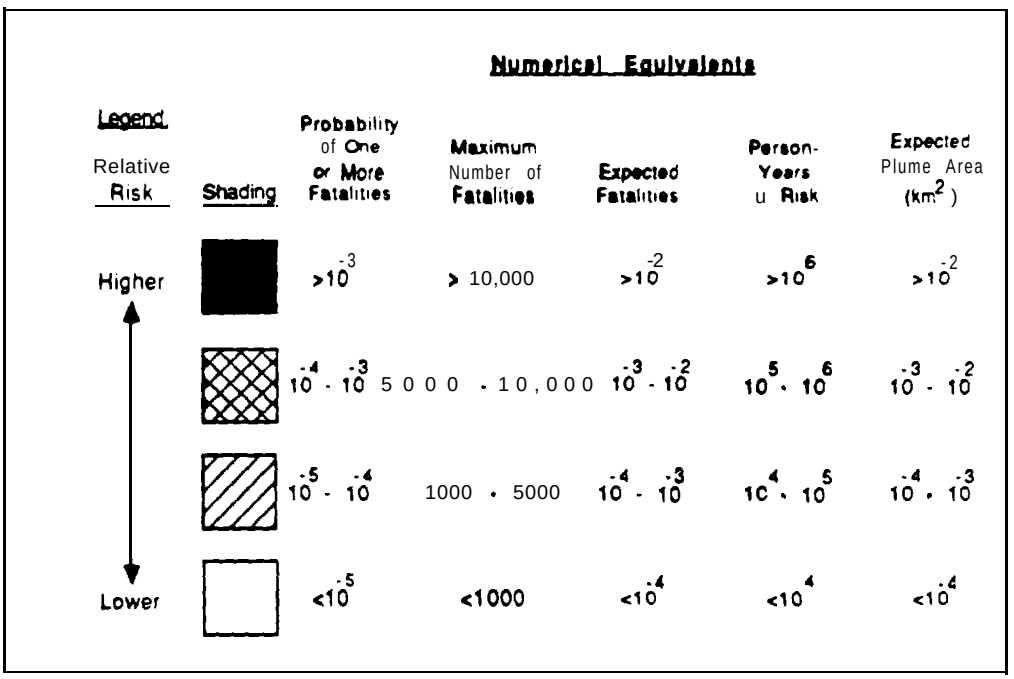
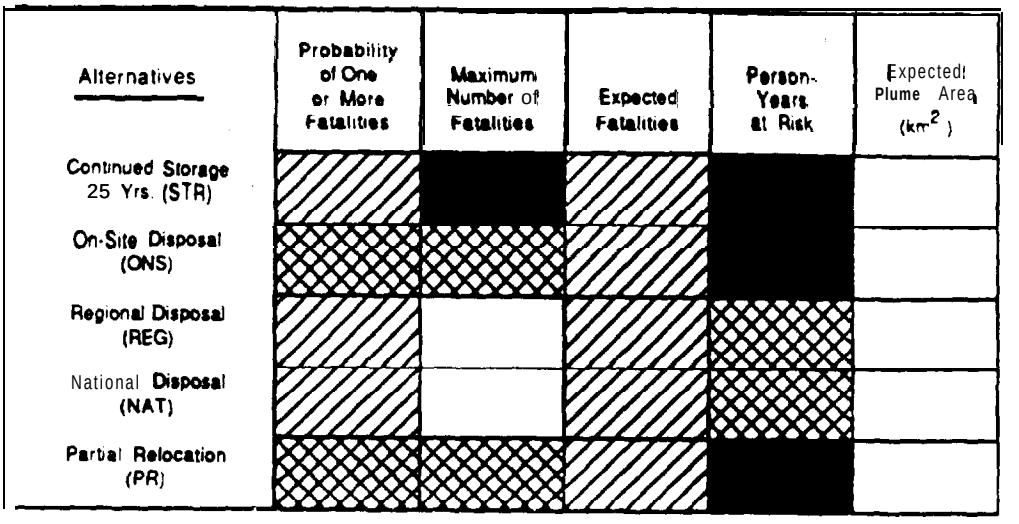


FIG. 5.52

RISK, **WITH MITIGATION (REV. 1), IN THE VICINITY OF PINE BLUFF ARSENAL (PBA) FOR PROGRAMMATIC ALTERNATIVES**

(Risk along transportation corridors not included)



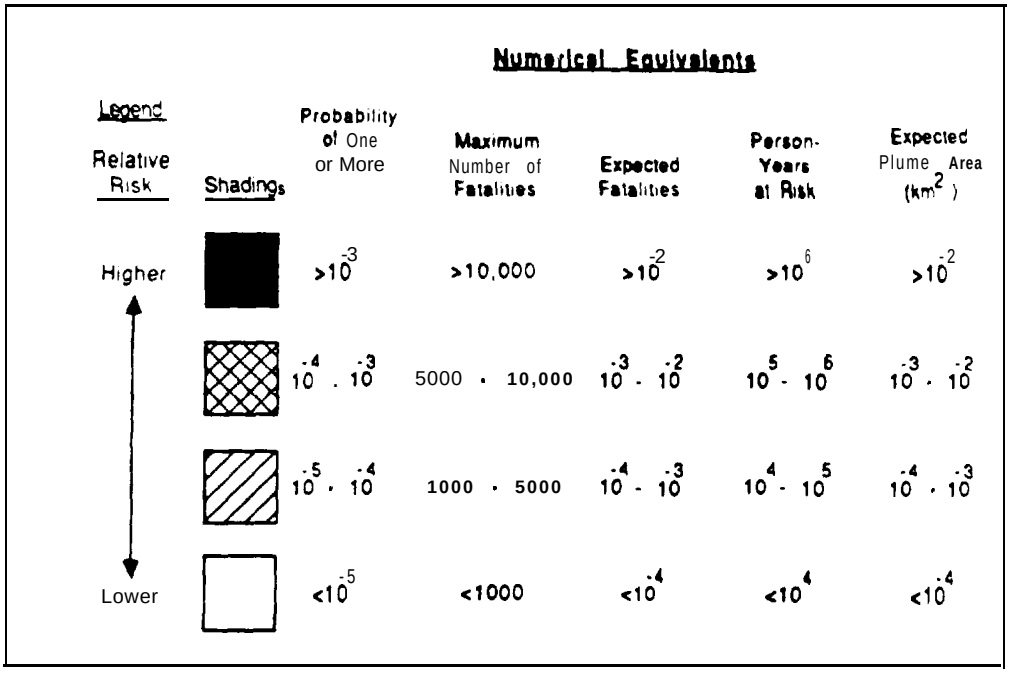
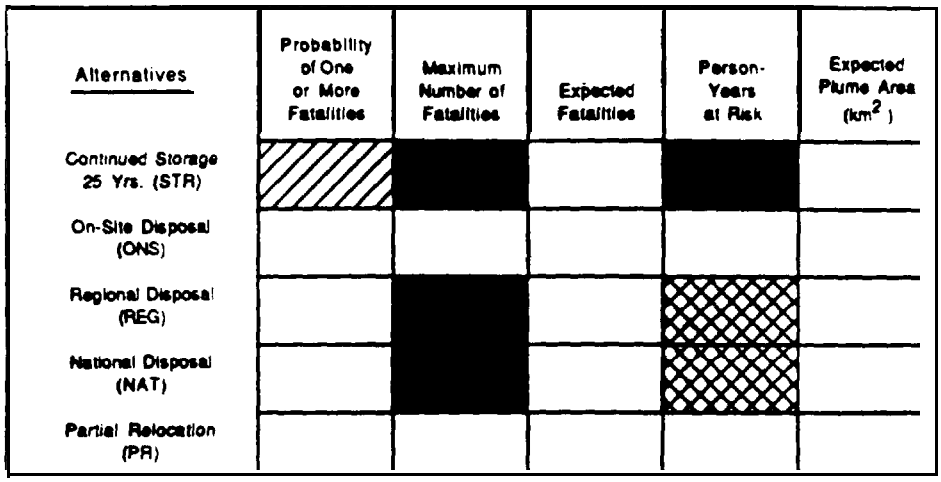


FIG. 5.53

RISK, WITH MITIGATION (REV. 2),
IN THE VICINITY OF
PINE BLUFF ARSENAL (PBA)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



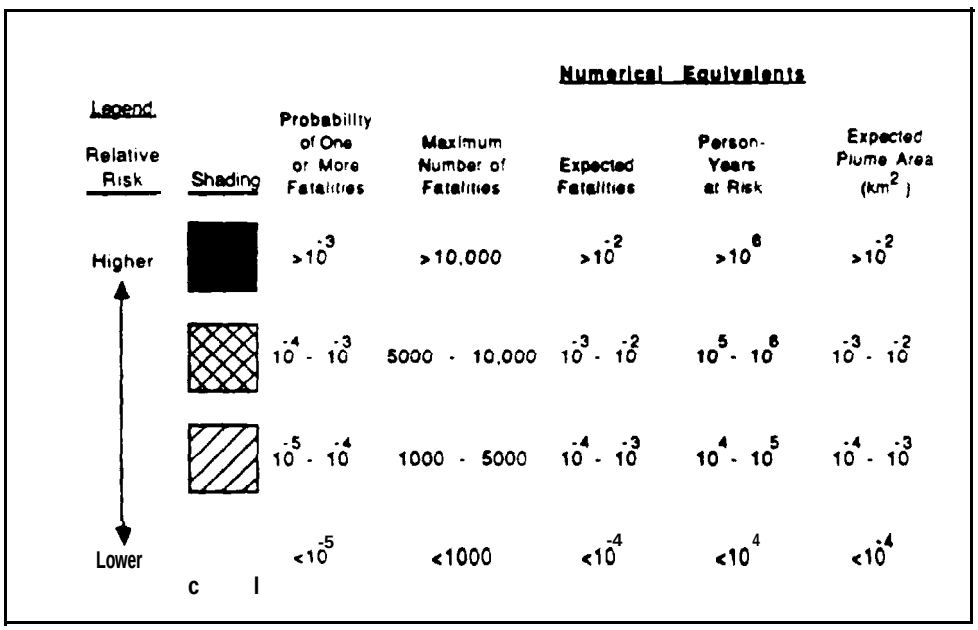
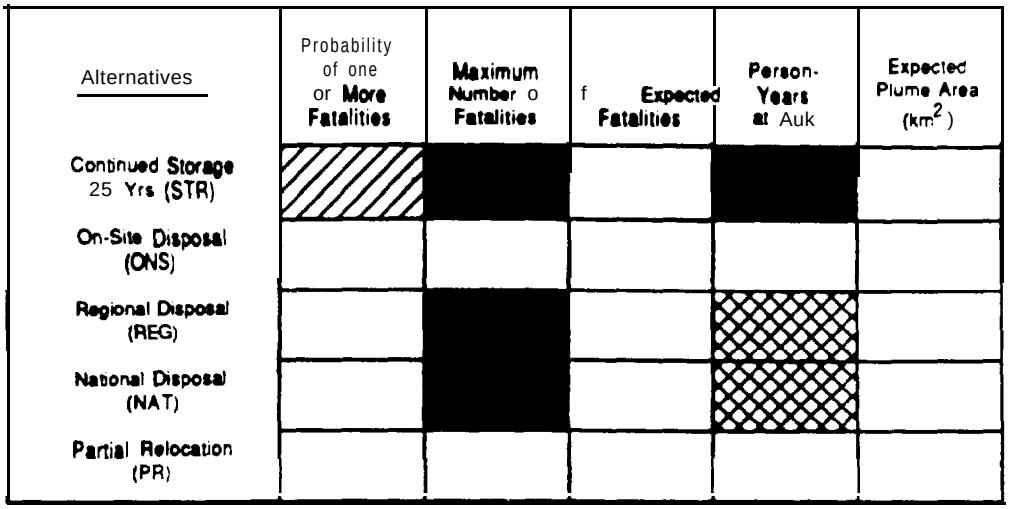


FIG. J.54

RISK IN THE VICINITY OF
PUEBLO DEPOT ACTIVITY (PUDA)
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



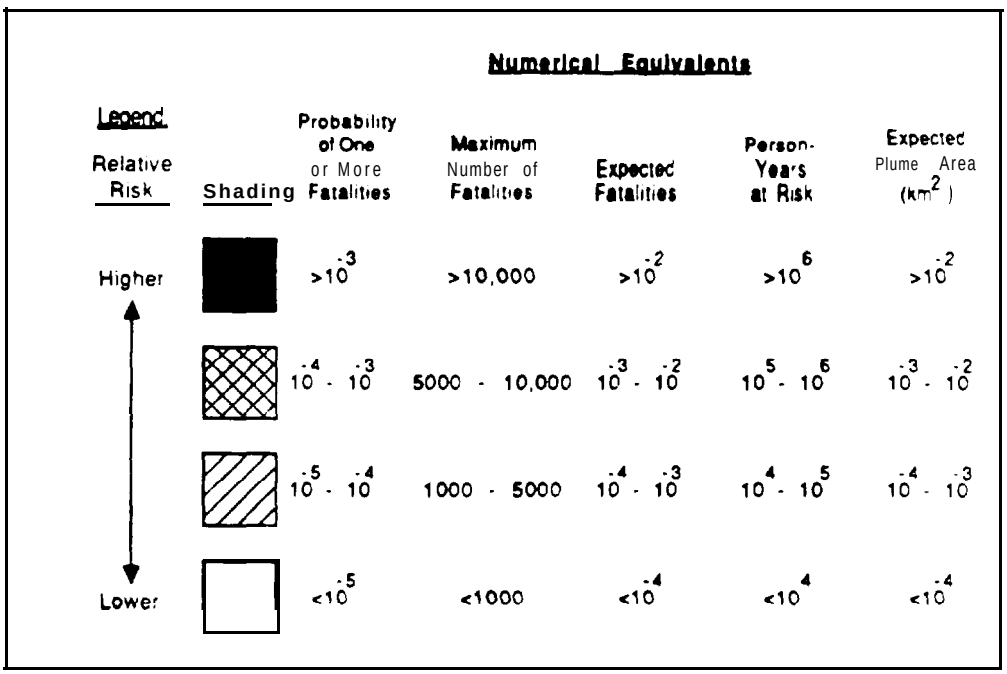


FIG. J.55

**RISK, WITH MITIGATION (REV. 1),
IN THE VICINITY OF
PUEBLO DEPOT ACTIVITY (PUDA)
FOR PROGRAMMATIC ALTERNATIVES**

(Risk along transportation corridors not included)

Alternatives	Probability of one or More Fatalities	Maximum Number of Fatalities	Expected Fatalities	Person-Years at	Expected Plume Area (km^2)
Continued Storage 25 Yrs. (STR)					
On-Site Disposal (ONS)					
Regional Disposal (REG)					
National Disposal (NAT)					
Partial Relocation (PR)					

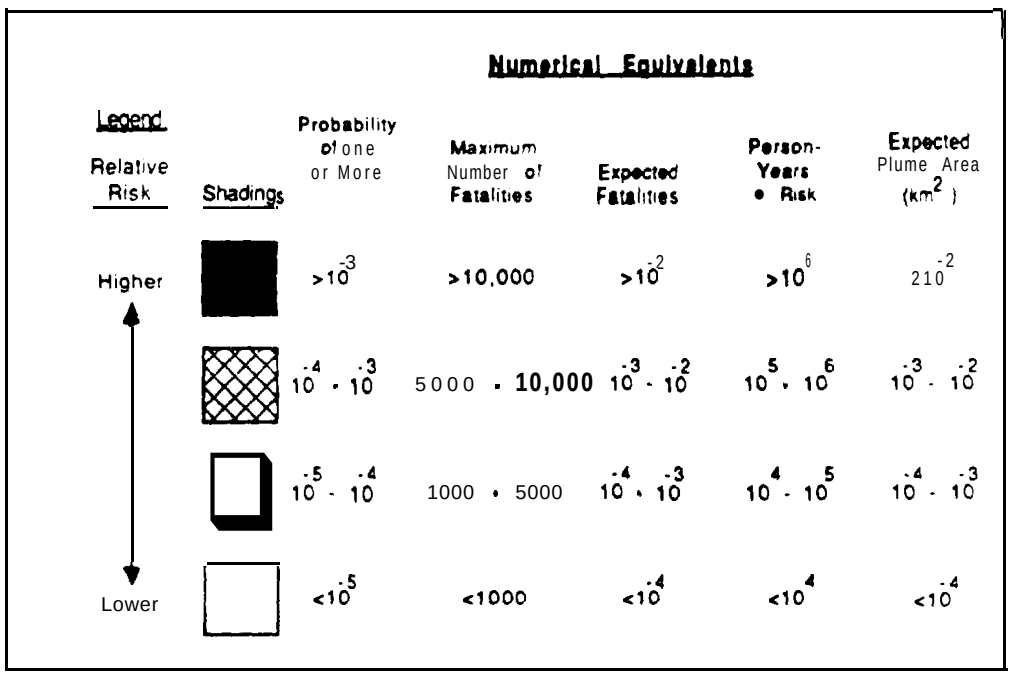
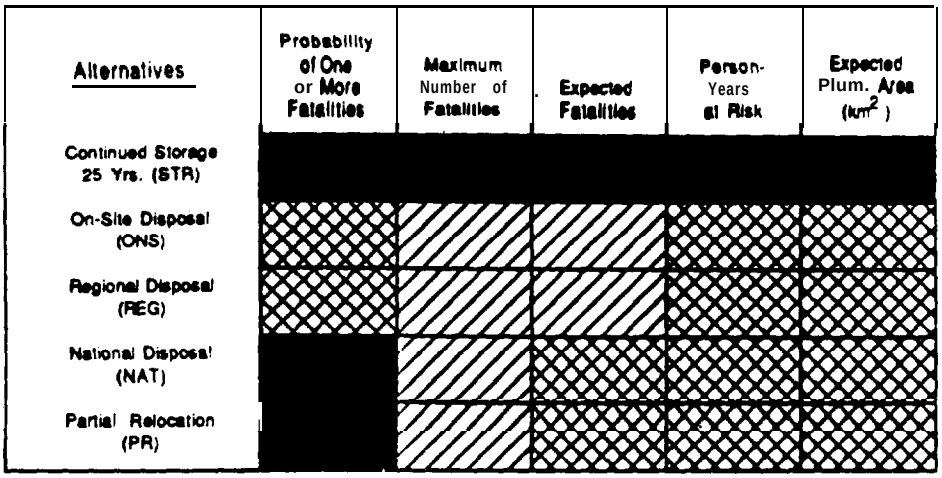


FIG. 5.56

RISK, WITH MITIGATION (REV. 2), IN THE VICINITY OF PUEBLO DEPOT ACTIVITY (PUDA) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



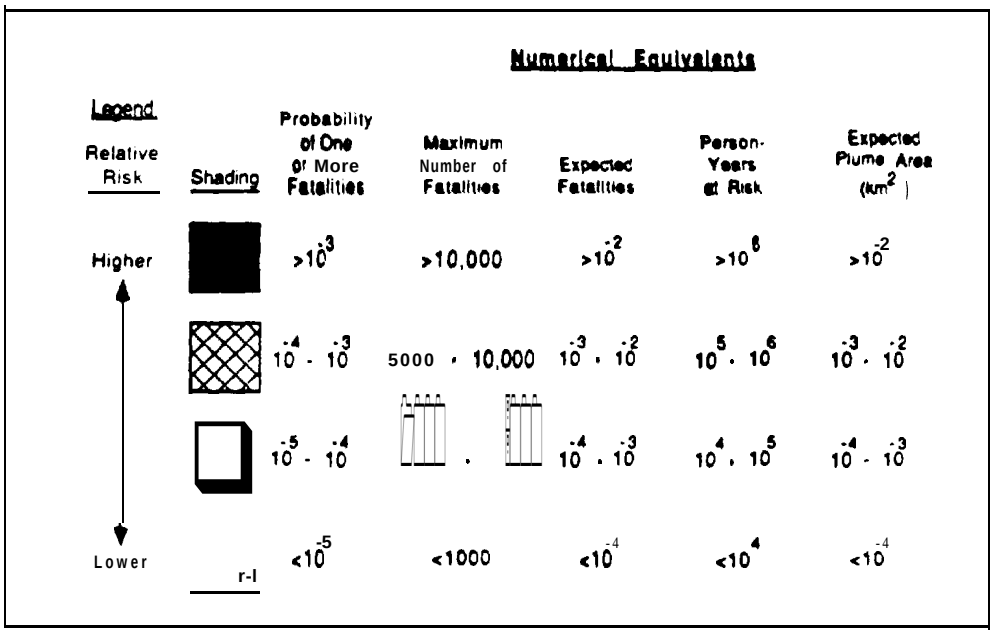
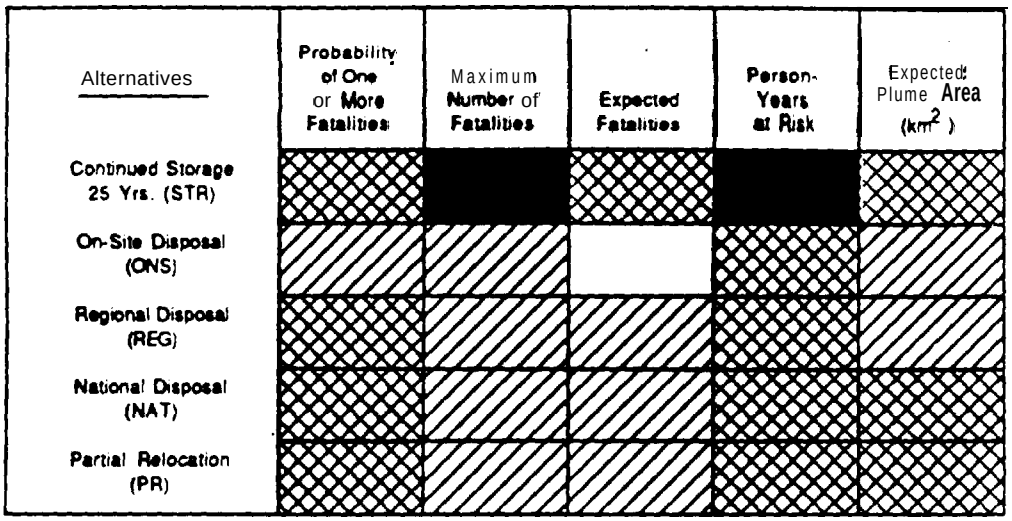


FIG. J.67

RISK IN THE VICINITY OF
TOOELE **ARMY DEPOT (TEAD)**
FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



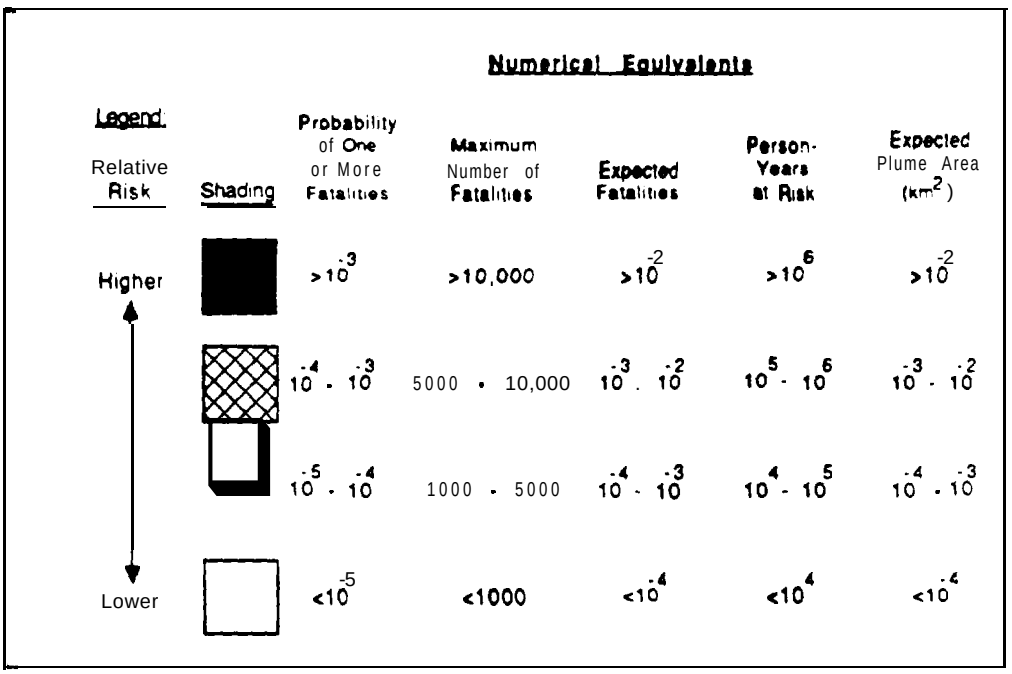
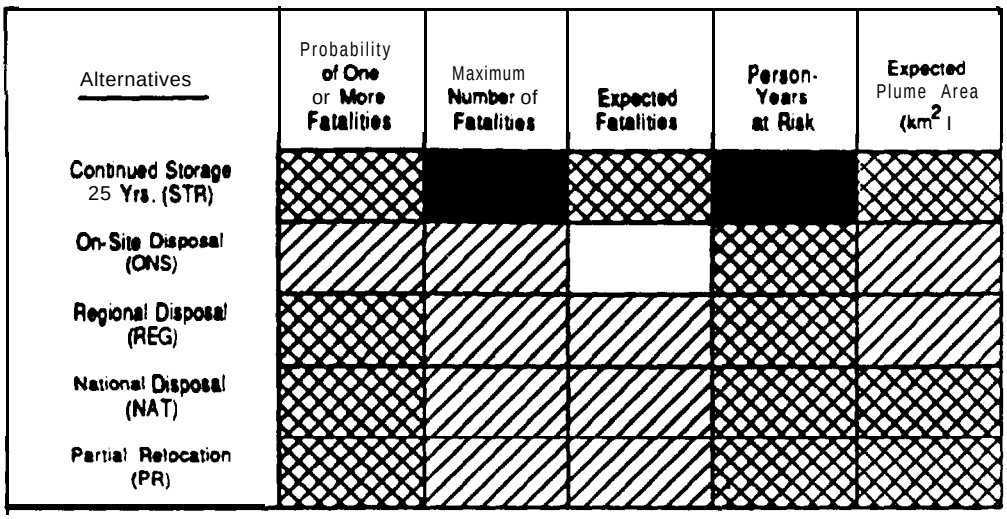


FIG. J.58

RISK, WITH MITIGATION (REV.1), IN THE VICINITY OF TOOELE ARMY DEPOT (TEAD) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



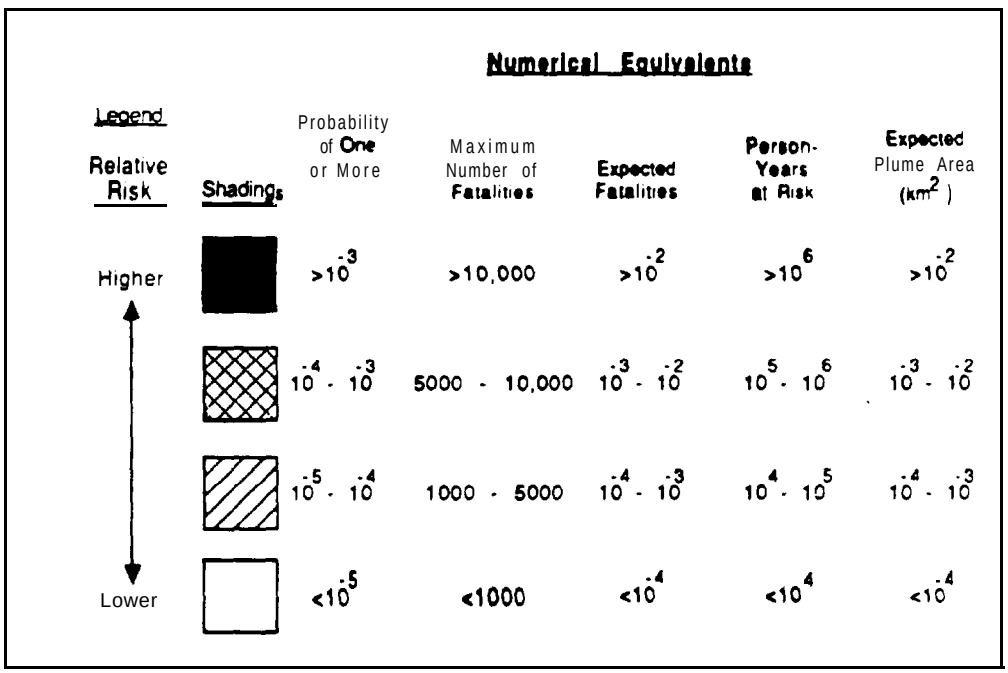
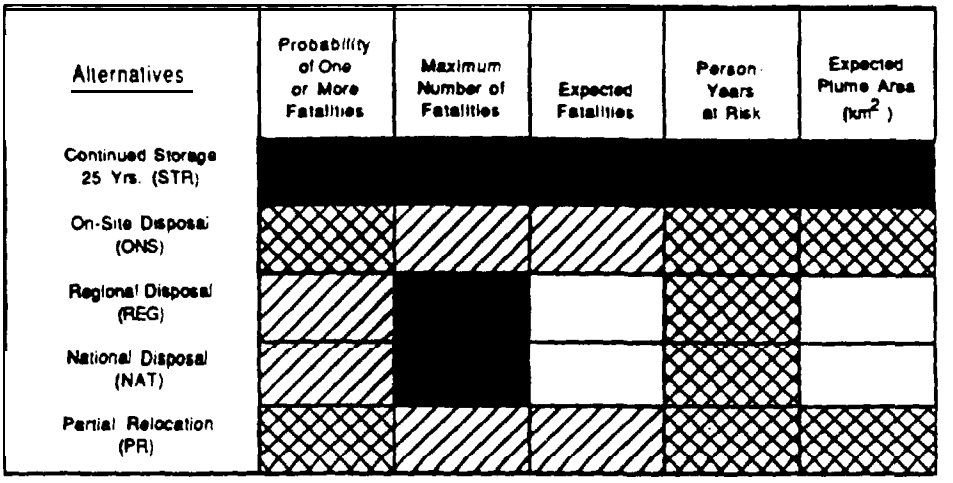


FIG. J.59

**RISK, WITH MITIGATION (REV.2),
IN THE VICINITY OF
TOOELE ARMY DEPOT (TEAD)
FOR PROGRAMMATIC ALTERNATIVES**

(Risk along transportation corridors not included)



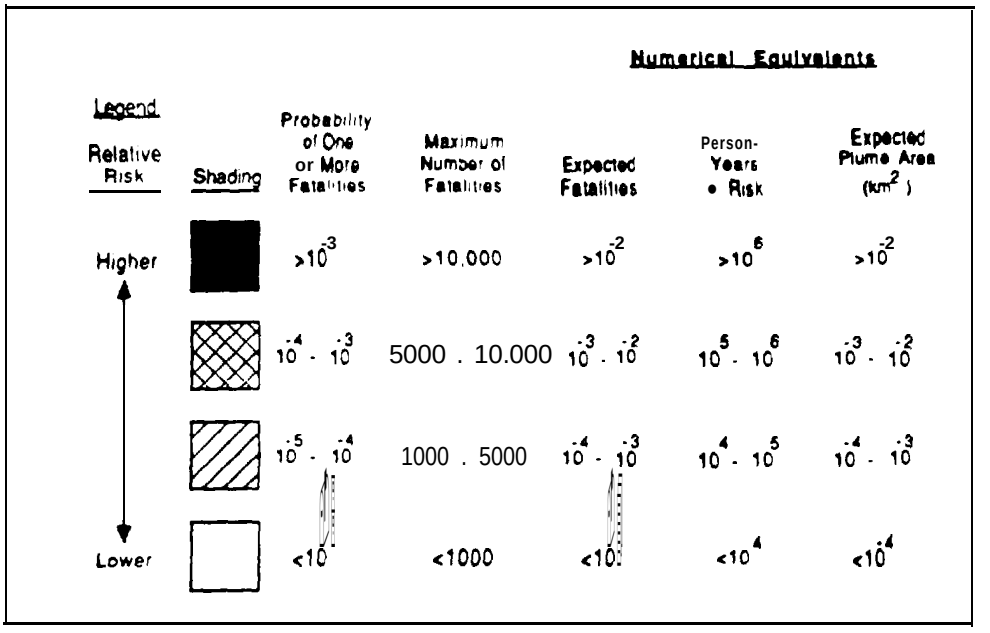
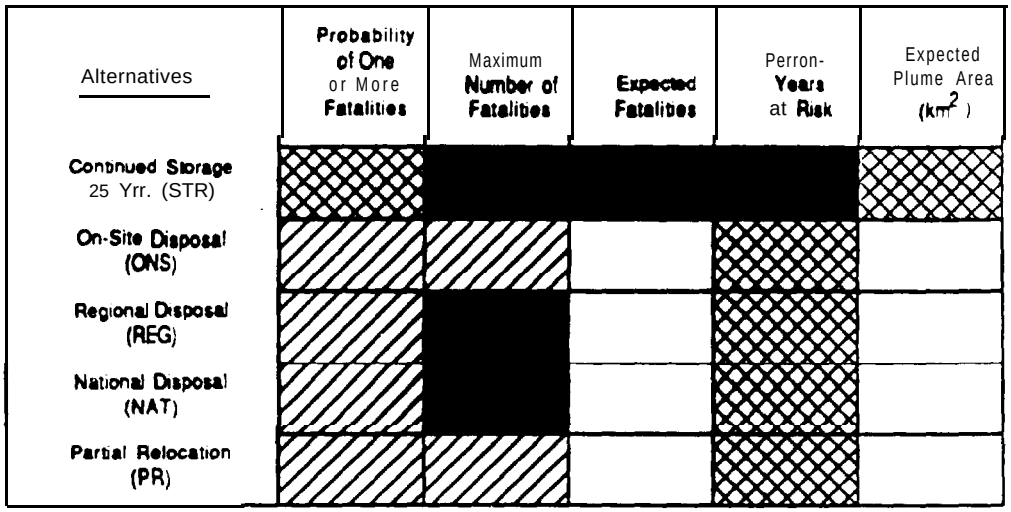


FIG. **J.60**

RISK IN THE VICINITY OF
UMATILLA DEPOT ACTIVITY **(UMDA)**
FOR PROGRAMMATIC ALTERNATIVES

(Risk along **transportation** corridors not included)



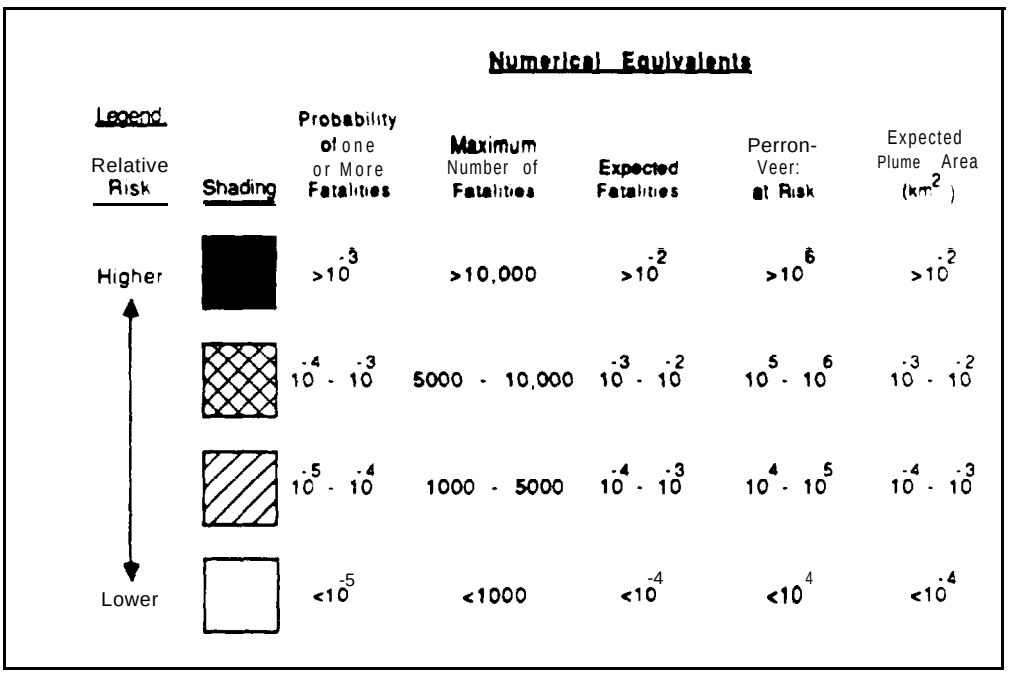
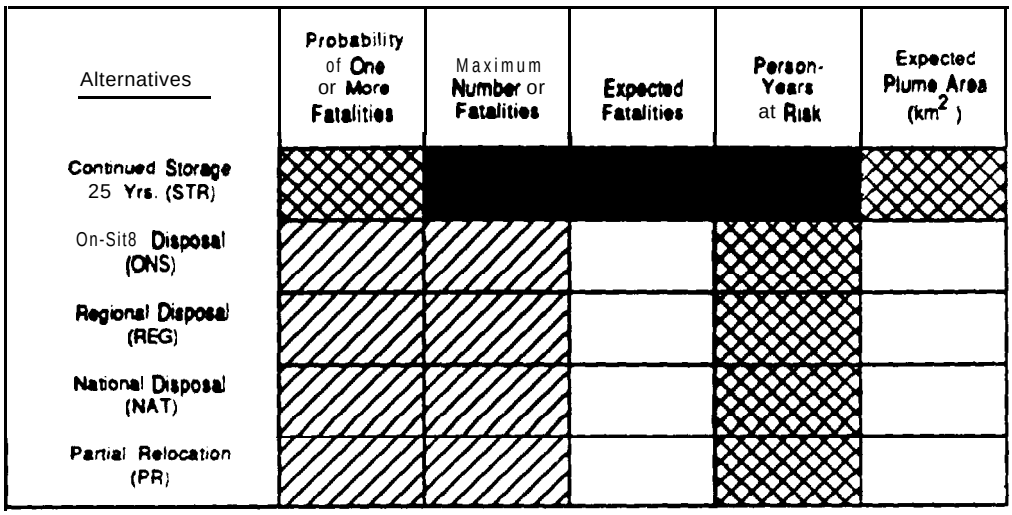


FIG. J.81

RISK, WITH MITIGATION (REV. 1), IN THE VICINITY OF UMATILLA DEPOT ACTIVITY (UMDA) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)



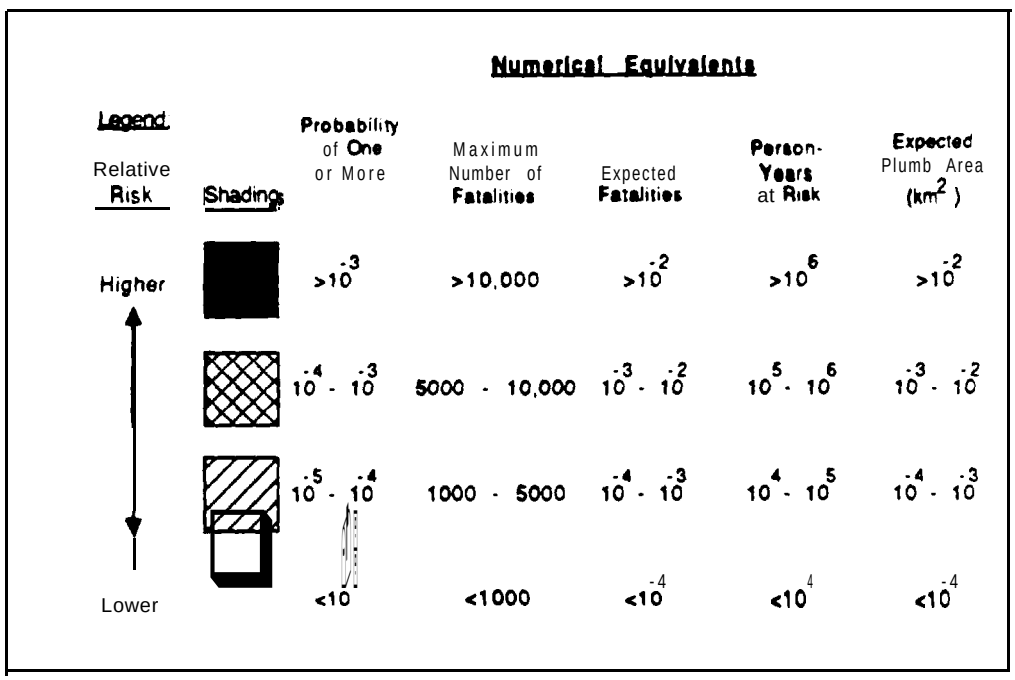


FIG. J.62

RISK, WITH MITIGATION (REV. 2), IN THE VICINITY OF UMATILLA DEPOT ACTIVITY (UMDA) FOR PROGRAMMATIC ALTERNATIVES

(Risk along transportation corridors not included)

- For continued storaee, the risk is borne primarily by two sites: NAAP with 85 percent of the total, and UMDA with 14 percent; when mitigation is introduced, the total program risk burden becomes somewhat more evenly shared, with NAAP dropping to 75 percent of the total (reduced) program risk and APG and UMDA carrying equal portions of the remainder of the risk; the risk of continued storaee is relatively insignificant at the remaining five sites. When air-space restrictions are introduced (mitigation, revision 2), the major beneficiary is the APG area, with its contribution to total programmatic risk dropping from over 10 percent (mitigation,- revision 1) to well less than 1 percent.

- Risk is somewhat more evenly shared for the unmitigated on-site disposal alternative, but even here, large disparities exist: 48 percent of the total risk would be experienced at PBA, with 2 percent or less of the totai borne at each of four sites -- APG, LBAD, PUDA and UMDA. When mitigation is introduced, overall risk drops by more than a factor of 10, and 75 percent of the reduced programmatic risk is borne in approximately equal shares by PBA and NAAP; LBAD, ANAD, and TEAD each experience between 5 percent and 10 percent of the total, with the remaining sites bearing less than 3 percent each of the total (reduced) programmatic risk, Air-space restriction has no effect on this alternative.

- For the regional alternative, 63 percent of the total programmatic risk is borne by the population groups along the transportation corridors, and only 3 percent is felt by the populations near the originating sites; the remaining 34 percent is borne by the destination site populations (ANAD and TEAD), with ANAD's share three times TEAD's. Risk along the transportation corridors is due principally to shipment of the PBA and LBAD stockpiles. When mitigation is introduced, the risk borne by the corridor populations rises to *over* 75 percent of the slightly lower (by 20 percent) total risk; the disposal sites (ANAD and TEAD) now experience less than 20 percent of the total -- most of that risk carried by the ANAD area; transportation corridor risk remains essentially unchanged in an absolute sense. Air-space restrictions would reduce the originating site contribution to total risk, with the major benefit *seen* at APG (where risk is reduced to less than 10^{-6}.

- 92 percent of the risk for the national alternative is distributed along the transportation corridors, leaving less than 2 percent of the risk to be borne by the originating sites. The remaining 6 percent of the total is felt by the TEAD population group. Of the transportation corridor risk, over 50 percent is the result of transporting the ANAD stockpile; the LBAD and PBA stockpiles also contribute nearly 20 percent each. With mitigation (revision 1), disposal site (TEAD) total risk drops from 6 percent to 1 percent

- For pn-site disposal (same as partial relocation at ANAD), mitigation reduces the probability of one or more fatalities and expected fatalities by about 95 percent. Since the highest consequence accident (detonation during on-site transport) is not mitigated significantly, there is no reduction in the maximum number of fatalities or person-years-at-risk measures. With mitigation, the greatest risk contributor is the accident involving detonation during on-site transport, representing 40 percent of the total; plant operations accidents account for nearly 40 percent of the total as well.

- For reeional disposal, mitigation reduces probability of one or more fatalities by about 80 percent and expected fatalities by 50 percent; the other risk measures remain unchanged. Most of the risk (approximately 95 percent) is attributable to an on-site accident involving a detonation. Mines are responsible for about 60 percent of the risk; rockets contribute 10 percent.

- Mitigation has no significant effect on any of the risk measures for the national alternative.

With the addition of air-space restriction (see Figure 3.41) to the mitigation measures discussed above, only the continued storage is affected. Relative to mitigation without air-space restriction (revision 1), expected fatalities would be reduced by nearly 90 percent and maximum number of fatalities and person-years-at-risk would be more than 99 percent lower. However, the probability of one or more fatalities is not reduced significantly by the restriction of air-space -- the reason being that this measure is dominated by other, higher probability events which are not mitigated by air-space restriction.

5.3.3.2.2 Aberdeen Proving Ground (APG). The risk from the various disposal alternatives to the population about APG is illustrated by the 'pictograms' in Figures 5.42 through J.44, covering the unmitigated case plus two levels of mitigation. For the unmitigated case (Figure J.42), it appears that, except for the probability of one or more fatalities, all risk measures indicate that the on-site alternative poses the least risk, On the basis of expected fatalities, the risk due to the disposal alternatives is roughly the same (within 25 percent) for all. Risk associated with the continued storage alternative is greater than for any of the disposal alternatives by a factor of 10 • 100.

The major contributors to risk for each unmitigated alternative at APG are summarized below:

- 100 percent of the continued storage risk is due to external events (aircraft crashes) during storage; there are no handling events of risk significance, a result of the fact that APG's stockpile consists only of mustard agent in bulk containers, and the handling accidents lead only to spills *or* spills with fire which do not create plumes that move beyond the boundary of the military reservation.

- For the on-site alternative, 100 percent of the risk results from plant operations.

- The risk due to regional and national alternatives (the same for those living near APG) is due entirely to accidents during short-term storage related to off-site transportation via rail.

- For the partial relocation -- air mode alternative, more than 95 percent of the risk arises from off-site transportation-related activities -- mainly crashes of aircraft carrying bulk containers of mustard, during take-off. The remainder of the risk results from short-term storage activities.

For the mitigated accident scenario set (all measures except air-space restriction -- see Figure J.43), the following risk reductions are realized, by disposal alternative:

- For continued storage, mitigation does not significantly change any of the measures of risk.

- For on-site disposal, mitigation reduces the probability of one or more fatalities and expected fatalities by almost an order of magnitude. Since the highest-consequence accident (an earthquake) is not sufficiently mitigated to remove it from the set of credible accidents, there is no reduction in the maximum number of fatalities or in person-years-at-risk. With mitigation, the plant operations accidents remain the only contributors to risk.

- For regional and national disposal, mitigation reduces risk by about 5 percent. Accidents during short-term storage remain the only contributors to risk.

- For partial relocation, mitigation leads to a reduction in risk of less than 20 percent. Although the consequences of a crash during take-off of an aircraft loaded with mustard bulk containers are somewhat mitigated by transporting the mustard in a frozen state, this accident remains the major contributor (>95 percent) to the risk at APG.

If air-space restriction2 (mitigation, revision 2 -- see Figure 5.44) are introduced, significant reductions in risk at APG would result for continued storage and the national and regional alternatives. Mitigation of aircraft crashes benefits the partial relocation alternative somewhat but has no effect on the risk of the on-site disposal alternative.

- For continued storage, both expected fatalities and the probability of one or more fatalities are two orders of magnitude lower with the reduction in frequency of aircraft crash accidents. Since the consequences of this accident are not mitigated, the maximum number of fatalities and person-years-at-risk are affected.

- For the on-site disposal alternative, restricting air-space has no effect on the risk because all accidents involving aircraft damage to the MDB have probabilities below 10^{-8} and are screened out of the analysis. Although there is a relatively high potential for small aircraft crashes at APG, such crashes are not considered to have enough impact to damage the MDB. Large aircraft crashes, which could damage the MDB, are expected to be relatively infrequent at APG.

- For the partial relocation alternative, since over 95 percent of the risk is due to crashes of aircraft carrying bulk containers of mustard, reducing the frequency of other aircraft crash accidents does not significantly reduce the risk of this alternative. However, consequences of a crash into temporary storage are greater than for a crash of the aircraft containing mustard. Therefore, eliminating the former accident reduces the maximum number of fatalities and the person-years-at-risk. With restricted air-space, the probability of this accident would drop below 10^{-8}, thus eliminating it from the data base. For this reason, the maximum number of fatalities is reduced from 2300 to about 300 and the person-years-at-risk from 4 X 105 to 2.5 X 10^4.

J.3.3.2.3 Lexington Blue-Grass Army Depot (LBAD). Figures 5.45 through 5.47 contain the 'pictogram' representation of risk at LBAD. The 'pictogram' for the unmitigated case (see Figure 5.45) indicates that the regional and national alternatives (identical in terms of originating site activities and risk at LBAD) pose the least risk to the population surrounding LBAD. On the basis of expected fatalities, the risk due to the national/regional alternatives is less than that due to on-site disposal by a factor of 3 or 4, while the risk due to the partial relocation (air mode) alternatives dominate by one-to-two orders of magnitude.

The contributions to risk for each unmitigated disposal alternative are summarized below:

- ☞□□ continued storaee at LBAD, essentially all of the risk arises from handling accidents associated with the maintenance of projectiles. The highest risk accidents are due to the movement of munitions for maintenance purposes.

- The risk due to **on-site** disposal results primarily (61 percent) from plant operations, with the remainder of the risk coming from on-site transportation **accidents.** Among munition types, rockets contribute 96 percent of the risk.

- For the **regional/national** alternatives, 100 percent of the risk results from rockets. Among activity types, 93 percent of the risk is due to on-site transportation, with handling contributing the remainder.

- For the partial relocation -- r mode alternative, 99 percent of the risk is due to off-site transportation-related accidents -- aircraft crashes on take-off. 90 percent to 95 percent of the total risk results from the transport of rockets.

Mitigation. Revision 1 would significantly reduce risk for continued storage and on-site disposal (see Figure 5.46). It would have no effect on the other alternatives. The major contributors to risk reduction are summarized below:

. For continued **storage**, mitigation would reduce the probability of one or more fatalities and expected fatalities by roughly 98 percent, Since the consequences of the most severe accidents (handling accidents involving fires or detonations) are not mitigated, there is no reduction in the maximum number of fatalities or person-years-at-risk. Essentially all of the risk remains attributable to handling accidents -- about 70 percent of them involving projectiles.

- For pn-site disposal, mitigation would reduce the probability of one or **more** fatalities by nearly 90 percent and expected fatalities by approximately 60 percent. **As** with continued storage, since the consequences of the most severe accidents (plant damage by earthquakes and detonations during on-site transport) are not mitigated, there is no reduction in the maximum number of fatalities or the person-years-at-risk. With mitigation, over 90 percent of the resulting risk is due to accidents involving detonation during **on**-site transportation. Rockets are responsible for essentially all (-99 percent) of the risk.

The introduction of **air-space** restrictions would have no risk reduction benefit for LBAD (see Figure J.47), mainly because the LBAD stockpile is stored in igloos,

J.3.3.2.4 Newport Army Ammunition Plant (NAAP) The risk at NAAP is illustrated by the 'pictograms' in Figures 5.48 through J.50. The comparison of risk among the unmitigated alternatives (Figure 3.48) becomes very obvious for this site: Continued storage poses the highest risk of all measures. Regional and national disposal represent the least (in fact, very low) risk at NAAP, while the risk due to on-site disposal falls between these extremes. At NAAP, the partial relocation disposal alternative is identical to the on-site disposal alternative.

The major contributions to risk for each unmitigated alternative are discussed below:

- The entire risk associated with continued storage at NAAP is due to external events damaging the stored agent (all of which is agent VX in ton containers in a warehouse.) In particular, the potential accident posing the highest risk, by far, is an earthquake-induced failure of the storage warehouse with a resulting fire. Handling during storage poses only negligible risk.

- For on-site disposal, essentially the entire (all but a fraction of a percent) risk is posed by plant operations, for which the major contributing accident, as with continued storage, is an earthquake-induced failure of the demilitarization building and a simultaneous failure of the fire suppression system.

- The very small risk (expected fatalities less than 10^{-4}) of regional and national disposal to the NAAP population is due entirely to a handling accident leading to a short duration fire.

With the introduction of mitigation (see Figure J.49), substantial reductions in risk would result for all alternatives. Since the risk for the regional/national alternatives is small to begin with, these alternatives show no substantial benefits of mitigation. The major contributions to risk reduction with mitigation are summarized below

- For continued storage, mitigation would lead to 98 percent reductions in both probability of one or more fatalities and expected fatalities. The reduction in expactad fatalities is not reflected in the 'pictogram' (Figure 5.49) because the value of expected fatalities remains greater than the 10^{-2} lower boundary of the highest risk category for that measure. Although the probability of the most severe accident (an earthquake-caused fire) is reduced, the accident is not eliminated. Therefore, there is no reduction in the maximum number of fatalities or the perron-years-at-risk measures. The entire risk contribution remains due to external events.

- For the on-site and partial relocation alternatives, mitigation reduces the probability of one or more fatalities by nearly 90 percent. Since the consequences of the most severe accidents (those caused by aircraft crashes and earthquakes) are not mitigated, there is no reduction in the maximum number of fatalities or the person-years-at-risk measures. The major contribution (~98 percent) to risk, with mitigation, remains the earthquake which damages the plant, leading to a fire.

The introduction of air-space restrictions provides only minor benefit (<2 percent risk reduction) to continued storage and none to the disposal alternatives at NAAP (see Figure J.50).

J.3.3.2.5 Pine Bluff Arsenal (PBA). The risk to the PBA population is portrayed by the 'pictograms' in Figures 5.51 through 5.53. The 'pictogram' for the unmitigated case (Figure 5.51) illustrates a more complex situation than is portrayed for NAAP. On-site disposal appears to pose the highest risk, both in terms of the number of risk measures which are in the higher risk categories, and on the basis of expected fatalities. (As for all sites but APG, LBAD, and TEAD, the programmatic partial relocation alternative calls for on-site disposal at PBA). Continued storage poses the least risk, with the regional/national transportation alternatives responsible for an intermediate level of risk.

Contributions to risk for each unmitieated disposal alternative at PBA are discussed briefly below:

- Over 90 percent of the continued storaee risk at PBA results from external events (aircraft crashes or meteorite strikes) causing fire-borne release of mustard agent from the ton containers in open storage. The remainder of the risk is due almost entirely to handling accidents (the dropping of a pallet leading to detonation) affecting stored rockets,

- For on-site disposal, nearly 95 percent of the risk results from plant operations; the remainder is due to on-site transportation. over 90 percent of the plant operations risk is caused by inadvertent feed of rockets and mines to the dunnage incinerator. Rockets and mines, together, are responsible for essentially all of the risk at PBA; bulk containers contribute a negligible fraction (well less than 1 percent).

- For the regional and national disposal alternatives, nearly 60 percent of the risk to the population near PBA results from on-site transportation accidents involving rockets; the remainder of the risk is roughly split between handling and short-term storage, again involving rockets. In fact, all but 2 percent of the risk

for all activities is due to rockets. Accidents involving a release of agent GB dominate the risk.

With the introduction of mitigation (revision 1), the risk of on-site disposal is significantly reduced; mitigation has no effect on the other alternatives at PBA. For the pn-site (and partial relocation) alternatives, mitigation reduces the probability of one **or more fatalities** by 98 percent and **expected fatalities by over 90 percent.** Since the consequences of the most severe accidents (a detonation during on-site transportation and earthquake damage to the MDB) are not mitigated, there is no reduction in the maximum **number** of fatalities or person-years-at-risk. With mitigation, the major contributor (-85 percent) to risk is the accident resulting in a detonation during on-site transport. Over 95 percent of the risk is due to rockets.

If air-space restrictions (revision 2) were introduced, some reduction in risk would result for continued storage and the regional/national disposal alternatives. The risk reductions are summarized below:

- For continued storage, the **probability of one or more fatalities** is reduced by over 40 percent and expected fatalities by -80 percent. Since the aircrash accidents are not mitigated sufficiently to eliminate them from consideration, **the maximum number** of fatalities and **person-years-at-risk** measures are not affected.

- For the regional/national disposal alternatives, there is no significant reduction in the **probability of one or more fatalities** and only a small (<20 percent) **reduction** in **expected fatalities.** There is, however, a significant reduction in the maximum number of **fatalities** -- from 40,000 for no air-space restriction to 900 with the restriction. The reduction in **person-years-at-risk** is, similarly, greater than one order of magnitude. This is because the probability of an aircrash accident, which is the most severe accident for these alternatives, has been reduced below 10^{-8} and, thus, screened out of the data **base.**

J.3.3.2.6 Pueblo Depot Activity (PUDA) Figures 5.54 through 5.56 display, in 'pictogram' form, the comparative risk for the applicable disposal alternatives at PUDA. For the ynmitinated case (Figure J.54), the continued storage alternative appears to **pose the** highest risk, but only on the basis of the **probability of** one **or more fatalities,** relative to the regional and national disposal alternatives. On-site disposal results in the least risk to the population near PUDA if all risk measures are considered. On the basis of **expected fatalities alone,** all disposal alternatives pose low risk (less than 10^{-4}) at PUDA with on-site disposal posing the least.

The factors contributing to unmitigated risk (Figure 5.54) at PUDA are summarized below:

- The risk at PUDA during continued storage arises entirely from potential aircraft crashes in to the storage facility, leading to detonation and/or fire. Projectiles account for nearly 80 percent of the risk. Although the risk, as measured by expected fatalities is very low, that risk is made up of highly improbable but very severe potential accidents for which the 'no-deaths' plume length, worst-case weather, could exceed 50 km and lead to over 15,000 potential fatalities. The fact that probability of one or more fatalities is relatively high is the result of a single highly probable handling accident of negligible consequence.

- For on-site disposal, plant operations account for nearly 95 percent of the risk, with the major event being earthquake-initiated fires in the munition demil building. The most severe accident involves a 'no-deaths' plume length of approximately 1 km (most-likely weather). The remainder of the risk is due to an on-site vehicle accident leading to detonation and fire.

- The risk at PUDA due to regional and national disposal is entirely the result of short-term storage associated with off-site rail transportation. Most (90 percent) of the risk is due to projectiles. The scenarios contributing the most to risk are those involving aircraft crashes into the transportation containers in the holding area. As with continued storage, the risk as measured by expected fatalities is low but it is comprised of a few high-consequence, low-probability events. The most severe accident leads to a worst-case weather, 'no-deaths' plume length greater than 50 km with the potential to cause over 15,000 fatalities.

M' 'gation, with no air-space restriction (Figure J.55), results in some risk reduction for the on-site disposal alternative. None of the other alternatives is affected by mitigation. For the on-site (and partial relocation) alternatives, mitigation reduces the probability of one or more fatalities by about 80 percent. Since the consequences of the most severe accidents (externally-initiated plant operations accidents and on-site transport accidents) are not mitigated, there is no reduction in the maximum number of fatalities or person-years-at-risk measures. Most of the risk is attributable to projectiles.

With air-space restrictions (revision 2) (Figure J.56), significant reductions in the risk for continued storage and the regional/national alternatives would result. Nevertheless, the factors contributing most to the added risk reduction are summarized below:

- For continued storage, the probability of one or more fatalities would be reduced by over 90 percent and expected fatalities, which are very low to start, would be reduced to well below 10^{-6}. Since the possibility of the most severe (aircrash) accident is not eliminated (probability reduced to below 10^{-8}) by mitigation, its consequences remain, and the related measures, maximum number of fatalities and person-years-at-risk, are not affected.

- For the regional/national disposal alternatives, the probability of one or more fatalities is reduced by almost two orders of magnitude while expected fatalities are reduced to below the 10^{-6} level. The maximum number of fatalities is reduced from 16,000, for the case of mitigation, revision 1, to 3 with air-space restriction. Person-years-at-risk is similarly reduced by three orders of magnitude (factor of 1000). The reason for the great reduction in these latter two measures is the fact that the probabilities of the most severe accidents (aircraft crashes into the holding areas) have been reduced to below 10^{-8} and, thus, screened out of the accident set defining the alternative. The reason the most severe accidents are eliminated for these alternatives and not for the continued storage alternative is related to difference in target areas in the two cases.

J.3.3.2.7 Tooele Army Depot (TEAD). Figures 5.57 through J.59 contain the 'pictogram' comparisons of risk measures for TEAD for the the unmitigated case plus two levels of mitigation. For the unmitigated case (Figure J.57), continued storage is seen to be the most risky on the basis of all risk measures. The lowest risk alternatives appear to be on-site and regional disposal

The major contributions to risk for the unmitigated alternatives (Figure 5.57) at TEAD are summarized for each disposal alternative below:

- Over 90 percent of the risk due to continued storage at TEAD is the result of earthquake-initiated damage and/or fire affecting bulk containers of agent VX in warehouse storage. Essentially all of the remainder of the risk is due to handling accidents involving burstered munitions. The events contributing the most to expected fatalities are also those having the most severe consequences (maximum number of fatalities) as well as a relatively high probability of occurring during the CSDP (10^{-4} to 10^{-3}).

- The risk of on-site disposal at TEAD results from plant operations (57 percent of expected fatalities) and handling (42 percent). Nearly half of the risk involves releases from bulk containers or while processing bulk containers, while mines contribute a third of the risk and rockets approximately 10 percent. Over 60 percent of

the risk involves agent GB. The scenarios making the major contributions to risk are handling accidents involving ton containers of GB, inadvertent passing of rockets and mines into the dunnage incinerator, and earthquakes damaging or causing fire in the demil building.

- For regional disposal, 59 percent of the risk comes from plant operations, 27 percent results from handling activities, and 14 percent is due to on-site transportation. Rockets, mines, and bulk containers each contribute about 25 percent to the risk at TEAD. As with on-site disposal, over 60 percent of the risk involves agent GB. Over 40 percent of the regional disposal risk at TEAD is due to inadvertent feeding of rockets and mines into the dunnage incinerator.

- For national disposal, where the entire U.S. stockpile is disposed of at TEAD, the contribution to total risk due to plant operations rises to 70 percent, with approximately 20 percent of the risk resulting from handling activities and 10 percent caused by on-site transport. Mines and rockets each contribute one-third to the total risk with nearly 20 percent resulting from bulk agent disposal. Slightly more than half of the risk arises from potential accidents involving agent GB. The major scenarios contributing to risk are essentially the same ones responsible for risk for the regional disposal alternative at TEAD, although the relative importance of some of the scenarios is slightly different due to the different mix of munitions in the inventory to be disposed. Nearly 60 percent of the total risk is due to inadvertent feeding of rockets and mines to the dunnage incinerator; handling of ton containers of GB are another major risk contributor -- responsible for 10 • 15 percent of the total. Vehicular accidents during On-site transportation of burstered munitions is next in risk significance.

- For the partial relocation disposal alternative involving air shipment into TEAD, the risk picture changes significantly. Approximately 1/3 of the risk is due to off-site transportation -- aircraft crashes during landing; most of the remainder of the risk results from plant operations. Rockets are responsible for 50 percent to 75 percent of the total risk; accidents involving bulk containers are next in importance to risk. Close to 80 percent of risk involves agent GB. The highest risk event is a handling accident involving ton containers of GB.

Mitigation (revision 1), without air-space restriction (Figure J.58), would result in substantial risk reduction for continued storage and all disposal alternatives except for partial relocation for which the risk

benefit is less. The major contributions to this risk reduction are summarized below:

- For continued storage, mitigation would reduce the probability of one or more fatalities and expected fatalities by approximately 98 percent. Since the consequences of the most severe accident (an earthquake-caused storage fire) would not be mitigated, there is no reduction in the maximum number of fatalities or person-years-at-risk measures. Earthquake-initiated accidents remain the major contributors to risk, accounting for about 90 percent of the total.

- For on-site disposal, mitigation reduces the probability of one or more fatalities by about 98 percent. The number of fatalities and person-years-at-risk measures are not affected since the consequences of the most severe accidents (earthquake-initiated damage to the MDB and a detonation during on-site transport) are not mitigated. Handling accidents account for nearly 75 percent of total risk for this alternative. The remainder of the mitigated risk is due to plant operations and on-site transportation. Handling accidents involving GB are still major contributors to risk -- accounting for nearly 60 percent of the total.

- For regional and national disposal alternatives, mitigation results in a reduction in probability of one or more fatalities of 80 percent for regional and nearly 90 percent for national. The reduction for regional disposal is not displayed on the 'pictogram' because both values, unmitigated and mitigated, fall within the risk category range: 10^{-4} to 10^{-3}. As with many other sites and alternatives, the person-years-at-risk and maximum number of fatalities are not affected by mitigation because the consequences for the most severe accidents (in this case, those involving a detonation during on-site transport) are not mitigated. The accident involving a detonation during on-site transport contributes 45 percent to the overall mitigated risk. The remainder of the risk is due to handling (43 percent) and plant operations (12 percent). Projectiles account for about 50 percent of the risk.

- For the partial relocation disposal alternative, both probability of one or more fatalities and expected fatalities are reduced by over 50 percent. Although this is a small change, well under an order of magnitude, it is reflected in the 'pictogram' because both values happen to cross over the boundaries defining their shading (risk measure) categories. The consequences of the most severe accident (crash of an aircraft carrying burstered munitions or a detonation during on-site transport) are not mitigated, so there is no reduction in maximum number of fatalities or person-years-at-risk for this alternative. Mitigation has the effect of increasing

the relative contribution of the aircraft crashes to risk -- to over 80 percent of the total. Rockets account for 80 percent of the mitigated risk.

The introduction of air-space restrictions will have no significant impact on risk at TEAD for any alternative because of the relatively low aircraft traffic density over TEAD.

5.3.3.2.8 Umatilla Depot Activity (UMDA). The 'pictograms' comparing risk measures among the applicable disposal alternatives at UMDA for the unmitigated case plus two mitigation levels are presented in Figures J.60 through 5.62. The only obvious conclusion to be drawn from Figure 5.60, for the unmitigated alternatives, is that the risk due to continued storage exceeds that of any of the disposal alternatives, both in terms of the number of risk measures (all) for which risk is in the maximum category and in terms of expected fatalities. In fact, the risk of continued storage for 25 years, as measured by expected fatalities, dominates disposal risk by several orders of magnitude.

The major contributions to unrnitinated risk (Figure 5.60) for each disposal alternative are summarized below:

- The risk associated with continued storaee is due almost entirely (>99 percent) to earthquake-induced damage with fire in warehouses storing mustard ton containers. This scenario represents a set of potential accidents with probabilities in the range of 10^{-6} to 10^{-4} per year and 'no-deaths' plume lengths of 20 - 30 km, for most-likely weather, and 200 - 300 km, for worst-case weather; potential fatalities exceed 400 for average conditions and approach 50,000 for extreme conditions (worst-case weather and wind direction □▪□ peak population density).

- For on-site disposal, plant operations accidents contribute over 90 percent of the total risk; on-site transportation accidents are responsible for most of the remainder. Two-thirds of all risk is due to rockets, with the remainder resulting from the disposal of mines. The inadvertent feeding of rockets and mines to the dunnage incinerator accounts for over 80 percent of the total risk for this alternative; on-site vehicle accidents and earthquake-initiated fire in the demil building during the processing of rockets and mines account for nearly all the remainder of the identified risk, Agent GB is involved in most of the risk; mustard-related accidents are negligible for plant operations (whereas mustard dominates for storage-related accidents because a much larger source of agent is available for release).

- The risk due to national and regional disposal, as measured by expected fatalities, is quite low (less than 10^{-4}) and arises mostly from on-site transportation accidents; short-term storage and handling accidents also make a significant and equivalent contribution to risk. Again, approximately 90 percent of the risk is GB-related, with rockets making the dominant contribution; bombs are responsible for most of the remainder of the identified risk. The scenarios contributing most to risk are a severe on-site vehicle transporter accident, an aircraft crash into the holding area during short-term storage, and a handling accident leading the drop and detonation of a palletized rocket.

When mitigation (revision 1) is introduced (Figure J.61), the risk of continued storage and on-site disposal are significantly reduced. The regional and national alternatives are not significantly affected. The details of the risk reductions are summarized below:

- For continued storaee, both the probability of one or more fatalities and the expected fatalities measures are reduced by about 98 percent. The reduction in expected fatalities is not reflected in the 'pictogram' (Figure J.61) because even the mitigated value for expected fatalities remains above the break point -- 10^{-2}. Again, the consequences of the most severe accidents (earthquake-induced warehouse fires and an aircraft crash into a warehouse) are not involved in the mitigation process, and the result is no change in the maximum number of fatalities and person-years-at-risk measures. With mitigation, the risk remains almost entirely due to earthquake-induced warehouse fires.

- For the on-site and partial relocation alternatives, mitigation leads to a factor of 10 reduction in probability of one or more fatalities and expected fatalities. The most severe accident (a large aircraft crash onto the MDB) has not been mitigated, leading to no reduction in maximum number of fatalities and person-years-at-risk. Approximately 70 percent of the mitigated risk results from an accident involving a detonation during on-site tranport; the remainder is due to plant operations (17 percent) and handling (12 percent). Agent GB is involved in accidents accounting for 80 percent of the mitigated risk.

If air-space restrictions were to be introduced (Figure J.62), there would be a slight benefit to the rail-mode colocation alternatives. No significant benefit would accrue to the other alternatives. The rail-mode risk benefits *consist* of a reduction in expected fatalities by 28 percent and a lo-fold reduction in maximum number of fatalities, from 24,000, for *no* air-space restriction, to 2500 with the restriction; person-years-at-risk would be reduced by nearly 56 percent. These reductions flow from the fact that the probability of the most *severe* accident (a large aircraft

crashing into the holding area) is reduced to below 10^{-8} and is thereby screened out of the active data base for this alternative.

J.3.3.2.9 Transportation Corridors - Regional (Rail) Alternative. Figure 5.36 summarizes, in 'pictogram' form the risk measures along the regional (rail) transportation corridors. The rail corridors from LBAD, PBA, and UMDA pose the highest risk in terms of expected fatalities. For these three corridors, rockets are responsible for well over half of the risk. For the other corridors (originating from APG, NAAP, and PUDA), the risk is lower primarily because rockets are not a part of the transported stockpile. The risk-dominating accident scenarios are severe train accidents with fire of long enough duration to cause failure of the overpack and the munitions (either by burster detonation or by thermal rupture of bulk containers). The applicable scenario (depends on whether the transported inventory is burstered or not) accounts for at least 95 percent of the risk (>99 percent for three sites) in all corridors.

The most severe potential accidents are those in the LBAD, PBA, and UMDA corridors. They yield worst-case 'no-deaths' plume lengths in the range of 15 to 20 km and could cause, under extreme conditions, 1000 to 2000 potential fatalities.

J.3.3.2.10 Transportation Corridors - National (Rail) Alternative. The risk picture is much the same for national disposal as it is for regional, as seen in Figure 5.37. The major difference is due to the fact that the ANAD stockpile is now transported to TEAD and, because of the size and composition of the ANAD stockpile, the risk in the ANAD - TEAD corridor dominates. In addition, selected risk parameters for APG, LBAD, and PBA are higher for national than for regional, primarily because of the greater travel distance for these stockpiles. For the ANAD - TEAD corridor, virtually all of the risk is associated with the movement of energetic munitions; the largest contribution to risk results from the transporting of projectiles, with the risk due to rockets not far behind.

The scenarios contributing the most to risk are the same severe rail accidents, with long-duration fires, that control risk for the regional corridors. The highest-consequence accident for the ANAD - TEAD corridor is expected to cause a worst-case 'no-deaths' plume length of 19 km with the potential for over 6000 fatalities.

J.3.3.2.11 Transportation Corridors - Partial Relocation Alternative. The risk along the transportation corridors that might be employed during the partial relocation disposal alternative is depicted in Figure 5.38. The route emanating from LBAD represents significantly higher risk than those originating from APG.

The major contributors to risk along the two corridors are discussed briefly below:

- For the LBAD • TEAD Corridog, via air mode, the risk (when measured, as before, by expected fatalities) is largely (90 percent) the result of transporting rockets; the rest is due to projectiles. Accidents involving GB constitute over 80 percent of the total corridor risk. Better than 75 percent of the risk results from a severe potential crash on an aircraft carrying GB-filled rockets; the accident would be of sufficient severity to rupture both the shipping containers and the munitions, with a possible fire. These high risk accident scenarios also represent the most severe consequence as well: a worst-case 'no-deaths' plume length of 31 km with a maximum potential fatalities of 23,000.

- The risk along the APG • TEAD Corridor, via air mode, is comprised of three scenarios, all involving severe aircrashes with breach of the shipping container and the agent containers. The worst-case plume length is considerable smaller than for the LBAD stockpile -- only 2.3 km with a corresponding maximum potential fatalities of 3500.

For all the transportation corridors, the effects of the mitigation measures are negligible -- certainly not enough to alter the 'pictogram' portrayal, a fact noted on Figures 5.36 through 5.38. Mitigation, revision 1 (no air-space restriction) yields, at best, a small reduction (less than 20 percent) in both expected fatalities and probability of one or more fatalities for the APG • TEAD air corridor. Air-space restrictions, as expected, would offer no additional benefit to any of the transportation corridors.

J.3.3.3 Time and Person-Years at Risk

Two time-related risk measures are addressed in this section:

- The total time-at-risk during the CSDP when an individual could be exposed if an accident were to occur;

- The total person-years-at-risk during the CSDP -- a measure equal to the time-at-risk times the number of people experiencing any risk (i.e., being within a zone that could encompass potentially lethal exposures, as defined by the 'no-deaths' plume length under worst-case meteorological conditions).

J.3.3.3.1 Time-at-Risk. Time-at-risk is readily addressed at the storage/disposal sites since the appropriate time measure is simply the duration of disposal activities at a given site. These times vary from less than 1 year to over 4 years, depending on the site. The actual disposal duration time at a given site cannot be stated because of the possibility of revealing classified data regarding stockpile size. By this measure, all individuals within a distance equal to the maximum possible (worst-case weather) 'no-deaths' plume length from a specific site should be considered 'at risk' for the same duration of time; outside this site-specific maximum distance, time-at-risk would be zero.

An approximate upper limit can be set on time-at-risk for both fixed site and transportation corridors:

- For fixed sites, the maximum time-at-risk is in the range of 4 years (35,000 hrs);

- For the regional (rail) corridors, the maximum number of trains is approximately 50, and the maximum hazard distance is in the 20 km range; an individual living within 10 km of the track carrying all 50 (or so) trains would experience a total time-at-risk of:

  MAX. TIME-AT-RISK (REG) = (0.7 hr/train) * (50 trains)
  = 35 hr

- For the national (rail) corridors, the maximum hazard distance is also approximately 20 km. The maximum number of trains for this alternative is approximately 75, leading to:

  MAX. TIME-AT-RISK (NAT) = 53 hr

- For the partial relocation (air-mode) corridor, the maximum hazard distance is approximately 31 km, leading to a time-at-risk of approximately 0.07 hr/aircraft flight. The actual number of flights required to move the APG and LBAD stockpiles is classified; but, it can be said to be in the range of 900 - 1200 air-lifts for the APG stockpile and in the range of 1200 - 1500 air-lifts for the LBAD stockpile, yielding a total number of airlifts in the range of 2100 - 2700 for the combined air-lifted stockpile. Using 2500 air-lifts (which could consist of several flights each, but would not thereby add to an individual's time-at-risk) as as a rough indicator of air traffic intensity, we find that:

  MAX. TIME-AT-RISK (AIR MODE) =
  (0.07 hr/flight) * (2500 flights) = 175 hr

Thus, the time-at-risk for individuals along the transportation corridors is in the range of 100 hr. For individuals around a disposal

site, time-at-risk is measured in the tens-of-thousands of hours -- a hundred-fold greater time than for those along the corridors.

J.3.3.3.2 Person-Years-at-Risk. The societal counterpart to the individual's time-at-risk measure discussed above is the number of person-years-at-risk. Although the time-at-risk for an individual along a transportation corridor is low (by a factor of 100 to 1000) compared to that for individuals about storage/disposal sites, the number of individuals experiencing that time-at-risk is greater (by a factor of roughly 10) along the corridors. Hence, risk, as measured by person-years-at-risk tends to be more evenly shared between fixed site and corridor population groups, although the former group's share is still the greater, by far.

The population data used to estimate potential fatalities among all population groups potentially affected by the CSDP can also be used to estimate person-years-at-risk for each exposed population group and for each disposal alternative. The results of that analysis are displayed in Tables 5.2 and 3.3.

With the aid of Tables 5.2 (for the cases of no mitigation or mitigation without air-space restrictions) and 5.3 (mitigation with air-space restrictions), one can compare the person-years-at-risk for all locations (fixed sites as well as transportation corridors) for all the CSDP alternatives, including 25 years of continued storage; the dominance of risk posed by continued storage is clear. Among the disposal alternatives, on-site disposal poses the least risk while national disposal represents the highest value for person-years-at-risk. Examining person-years-at-risk along the transportation corridors for the various transportation alternatives, one can see that the rail mode alternatives are significantly less risky than the air modes, according to this measure, with regional (rail) responsible for the least person-years-at-risk. The principal reason for the four-fold difference between regional and national is the transportation of the Anniston stockpile for the national alternative.

The distribution of person-years-at-risk among the eight storage/disposal sites can be compared for the various CSDP alternatives. For continued storage, ANAD displays the highest value of person-years-at-risk, with APG and LBAD having the least risk by this measure. Person-years-at-risk resulting from on-site disposal are greatest at PBA, ANAD, and UMDA, in decreasing order, and least at PUDA, APG, NAAP, and LBAD, in increasing order. The distribution of person-years-at-risk about the fixed sites is, as expected, identical for the regional and national alternatives -- the only exception being ANAD for which the difference between alternatives is approximately 5 percent. The partial relocation alternative (air mode) affects only three sites, but the major impact is felt only at APG and LBAD.

TABLE 3.2
PERSON-YEARS-AT-RISK -- HO AIR-SPACE RESTRICTION

- Fixed Sites -

SITE\ALT:	S I R	ONS	REG	NAT	PRB
ANAD	37759175	650156	1147150	1094013	650156
APG	CD75700	22910	975776	975776	308056
LBAD	59525	50579	82466	82466	409223
NAAP	28331075	36242	4509	4509	36242
PBA	20216050	1049450	1617264	1617264	1049458
PUDA	12486100	97	740166	749166	97
TEAD	24203400	113962	132979	132979	132979
UMDA	7794550	340930	779455	779455	340930
TOTAL	135625575	2266353	5488785	5435647	3019140

- Transportation Corridors -
(Originating at Specified Sites)

SITE\ALT:	REG	NAT	PRB
ANAD	0	6642	0
APG	263	307	504
LBAD	1884	2923	61522
NMP	180	282	0
PBA	1177	1277	0
PUDA	65	65	0
UMDA	279	279	0
TOTAL	3849	13777	62026

- All Locations Combined -

SITE\ALT:	STR	ONS	REG	NAT	PRB
ANAD	37759175	650156	1147150	1102654	650156
APG	4975700	22010	976039	976063	398560
LBAD	59525	50579	84350	85389	470745
NAAP	28331075	38242	4689	4791	30242
PBA	20216050	1049458	1618461	1618561	1049458
PUDA	12486100	97	749231	749231	97
TEAD	24203400	113982	132979	132979	132979
UMDA	7704550	340930	779455	779455	340930
TOTAL	135825575	2266353	5492354	5449144	3061166

TABLE 5.3
PERSON-YEARS-AT-RISK -- WITH AIR-SPACE RESTRICTION

- Fixed Sites -

SITE\ALT:	STR	ONS	REG	NAT	PRB
ANAD	462525	650156	1147150	1094013	650156
APG	4975700	22910	6260	6260	25038
LBAD	59525	50579	82466	82466	409223
NAAP	26331075	38242	4509	4509	38242
PBA	20216050	1049458	150488	150488	1049456
PUDA	12486100	97	626	626	97
TEAD	24203400	113962	132979	132979	132979
UMDA	7794550	340930	340930	340930	340930
TOTAL	98526925	2266353	1865407	1812269	2646122

- Transportation Corridors -
(Originating at Specified Sites)

SITE\ALT:	REG	NAT	PRB
ANAD	0	8642	0
APG	263	307	504
LBAD	1884	2923	61522
NAAP	180	262	0
PEA	1177	1277	0
PUDA	65	65	0
UMDA	279	279	0
TOTAL	3649	13777	62026

- All Locations Combined -

SITE\ALT:	STR	ONS	REG	NAT	PRB
ANAD	462525	650156	1147150	1102654	650156
APG	4975700	22910	6523	6567	25542
LBAD	59525	50579	84350	85389	470745
NAAP	26331075	38242	4689	4791	36242
PBA	20216050	1049458	151665	151765	1049458
PUDA	12486100	97	690	690	97
TEAD	24203400	113962	132979	132979	132979
UMDA	7794550	340930	340930	340930	340930
TOTAL	96528925	2266353	1868976	1825766	2700146

Looking at the transportation corridors for the rail- and air-mode alternatives, one can readily see the relatively high risk, as measured by person-years-at-risk, of moving the ANAD stockpile; the next risky stockpile move is that of the LBAD stockpile. However, the highest number of person-years-at-risk along any corridor results from the air movement of the LBAD stockpile -- the reason being the composition of the LBAD stockpile and the the fact that it, unlike the APG stockpile for which air-mode corridor risk is negligible, is comprised of energetic munitions.

Such considerations as type of agent and munitions, numbers of items, distribution of population around sites, and the numbers of items brought together at any one time is a given program alternative lead to sometimes startling contrasts in persons-years-at-risk, as shown in Table 5.2 and 5.3. For example, Table 5.3 indicates only 97 person-years at risk for the on site alternative, while the regional and national alternatives, which involve short-term storage prior to shipping, are assigned almost 750,000 person-years at risk. Until the mustard-containing items at PUDA are brought together for loading on a train, there is little opportunity for an accident that could cause public fatalities (recall that risks of long-term storage have been excluded here). With preparation for shipment, the opportunity for a much larger accident is created.

J.3.4 Caveats and Limitations

J.3.4.1 Freauencv and Consequence Screening

As noted in the companion report (Fraize, et al., 1987[a], the accident scenario data base for this risk analysis was screened so that only those accidents with a potential (under worst-case meteorology) for causing fatalities beyond the installation boundaries (assumed to be 0.5 km for all sites) and having a probability of occurring during the course of the CSDP (or during a one-year period, in the case of continued storage activities) of at least 10^{-8} are included.

J.3.4.2 Potential Fatalitv Estimates and Site Boundaries

The fatality estimates used in the risk analysis were computed by Oak Ridge National Laboratories (ORNL) using population data from census tracts. It is not possible from such data to determine the precise location of populations. Since the risk analysis is concerned with the potential health effects to the general population located outside the military reservations, personnel within the boundaries of the reservations should not be considered. In an effort to exclude these personnel from consideration, ORNL set all fatality rates to zero for distances of up to 0.5 km from the disposal or storage site where accidents may occur. Thus,

no fatalities are computed for low-consequence accidents for which the zero-fatalities distance does not exceed 0.5 km, if the accidents occur within a military reservation.

Using 0.5 km as a cut-off distance for fatality computations is a conservative approach; i.e., the number of fatalities may be overstated. This is because the actual distances from disposal and storage sites to military reservation boundaries range from 0.9 to 3.5 km. Thus, if the actual boundaries had been used to compute the number of fatalities, the value of "expected fatalities" would have been substantially lower in a number of cases. The most significant reduction (about an order of magnitude) in "expected fatalities would be for the on-site disposal alternative at Anniston, Aberdeen, Pine Bluff, Tooele, and Umatilla. The total of expected fatalities at all sites for the on-site alternative would be approximately 75 percent lower.

J.4.0 ACCIDENT SCENARIO DESCRIPTIONS

To demonstrate the range of accident scenarios identified and analyzed during the course of the risk analysis, this section presents both the code used for identifying **and analyzing the many hundreds of scenarios and the** brief textual discussion of **the actual scenario8 themselves.**

J.4.1 Accident Scenario Identification

Accident scenarios are described by the following:

- A unique identification code which defines:
  - operational activity, with the major categories:
    - on-site handling
    - on-site transportation
    - handling at facility
    - plant operations
    - off-site transportation
    - interim storage (associated with off-site transport)
    - long-term storage
  - munition type
  - agent type
  - release mode
    - outdoor spill *or* leak (leading to evaporation)
    - detonation (without fire)
    - fire alone
    - combinations of the above
    - emissions resulting from a complex series of events, including indoor spills or releases
- A brief textual description of each scenario
- Agent release and probability **data**

The accident scenario identification code is described and defined in Table J.4.

TABLE J.4
ACCIDENT SCENARIO **IDENTIFICATION** CODE

Scenario ID **is** of the form: **XXYZWQnnn**
where: **XX** = Activity Code **W** = **Release Mode** Code
Y = Munition Code **Q** = Special **Code**
Z = Agent Code **nnn** = Scenario Number (See Table 5.5)

ACTIVITY CODE (XX) DEFINITION

BF/BL/BT: *Air* transportation (C141) -- **in-**Flight/Landing/Takeoff
HB: Handling associated with air mode
HF: Handling at the disposal facility
HO: On-site handling away from the disposal facility
HR: Handling associated with rail mode
HS: Handling during long-term storage *
PO: Plant operations
RN: Rail transportation, National
RR: Rail transportation, Regional
SL: Long-term storage
SR/SB: Temporary storage associated with transportation by rail or air, respectively
VO/VR/VB: On-site transportation associated with on-site, rail, and air disposal alternatives

MUNITION CODE (Y) DEFINITION

B: Bombs
C: Cartridges **(105mm)**
D: **Mortar** Shells **(4.2in)***
K: Bulk ("ton") containers
M Hines
P: Projectiles **(155mm)**
Q: Projectiles **(8in)***
R: Rockets
S: Spray tanks
V: Wet-eye bombs*
A: All munitions

AGENT CODE (Z) DEFINITION

G: Agent GB **("Sarin")**
H: Agents H, HT, HD ("Mustard")
V: Agent VX
A: All agents

RELEASE MODE CODE (W) DEFINITION

A: Detonation
C: Complex mode **(incl.** indoor releases affected by building systems) or a combination of **simple** nodes
F: Fire (incomplete combustion)
S: Spill (leading to partial evaporation

SPECIAL CODE (Q) DEFINITION

W: Warehouse Storage
0: **Open** Storage
6/8/9: **60/80/89** ft. Igloo

J.4.2 Summary Descriptions of Accident Scenarios

The accident scenario data provided by GA Technologies includes a textual description of each of the accident scenarios. The scenarios are identified by the activity code (first 2 characters of the scenario ID code -- see Table 5.4) and the scenario number. For convenience, MITRE summarized the scenario descriptions provided by GA Technologies into a data base (dBASEIII) format. The results are presented in Table 5.5. To facilitate finding a particular scenario description, the list is ordered alphabetically according to the ID. In general, the scenario descriptions are independent of munition and agent type. Thus, most scenarios could be the basis for describing as many as fourteen separate accidents, each one representing the probability and release characteristics of one of the applicable munition-agent combinations.

TABLE J.5

CSDP/FEIS RISK ANALYSIS
Accident Scenario Descriptions

RECORD NUMBER	ID		SCENARIO
* BF			
399	BF	001	A severe gromd collision involving an aircraft with mnitions occurs and impact forces fail the agent package and mnitions.
400	BF	002	A severe gromd collision involving an aircraft uith munitions occurs and impact forces fail the agent package and munitions. A subsequent fire occurs with a duration less than 2 h.
401	BF	003	A fire occurs aboard an aircraft with mmitions and causes rupture of the compartment due to thermal expansion of the agent.
402	BF	004	A severe ground collision involving an aircraft with munitions occurs and impact forces fail the agent package and mmitions. A subsequent fire occurs with a duration greater than 2 h.
403	BF	005	A moderate gromd collision involving an aircraft with mmitions occurs causing a breach of the package. A subsequent fire occurs causing a breach (by detonation or thermal expansion) of the agent compartment ad agent is released.
** BL			
404	BL	001	A severe gromd collision involving an aircraft with mmitions occurs and impact forces fail the agent package and mnitions.
405	BL	002	A severe ground collision involving an aircraft with munitions occurs and impact forces fail the agent package and mmitions. A subsequent fire occurs with a duration less than 2 h.
406	BL	003	A fire occurs aboard an aircraft with mmitions and causes rupture of the compartment due to thermal expansion of the agent.
407	BL	004	A severe gromd collision involving an aircraft uith munitions occurs and impact forces fail the agent package and munitions. A subsequent fire occurs uith a duration greater than 2 h.
408	BL	005	A moderate ground collision involving an aircraft with munitions occurs causing a breach of the package. A subsequent fire occurs causing a breach (by detonation or thermal expansion) of the agent compartment and agent is released.
BT			
[illegible]	BT	001	A severe ground collision involving an aircraft with munitions occurs and impact force6 fail the agent package and munitions.
410	BT	002	A severe gromd collision involving an aircraft uith mmitions occurs and impact forces fail the agent package and munitions. A subsequent fire occurs uith a duration less than 2 h.
411	BT	003	A fire occurs aboard an aircraft with munitions and causes rupture of the compartment due to thermal expansion of the agent.
412	BT	004	A severe ground collision involving an aircraft with munitions occur6 and impact forces fail the agent package and mnitions. A s&sequent fire occurs with a duration greater than 2 h.
413	BT	005	A moderate gromd collision involving an aircraft with mmitions occurs causing a breach of the package. A subsequent fire occurs causing a breach (by detonation or thermal expansion) of the agent compartment and agent is released.
HB			
[illegible]	HB	001	Drop of bare pallet or single item at storage area.
415	MB	002	Forklift collision with short duration fire at storage area involving bare munitions.
416	HB	003	Forklift tine accident at storage area involving bare mmitions.
417	HB	004	Forklift collision accident et storage area involving bare mnitions.
418	HB	005	Drop of onsite container.
419	HB	006	Forklift collision accident with short duration fire during handling of onsite container.
420	HB	007	Forklift collision without fire during handling of onsite container.
421	HB	008	Drop of offsite container.
422	HB	009	Collision accident with short duration fire during handling of offsite container.
423	HB	010	Collision accident uithout fire during handling of offsite container.

TABLE J.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
• Accident Scenario Descriptions •

RECORD NUMBER	ID		SCENARIO
424	MB	011	Drop of Dare palletized munition leads to detonation.
425	MB	012	Forklift collision occident at storage area leads to detonation.
426	MB	017	Drop of pallet containing a leaking mmition during leaker isolation operations.
427	MB	018	Drop of single leaking in leakers processing facility.
428	MB	019	Forklift tine puncture during leaker isolation.
429	MB	021	Collision accident uithout fire during handling of leaking munition.
430	MB	022	Drop of mmition in onsite container leads to detonation.
431	MB	023	Drop of munition in offsite container leads to detonation.
432	MB	024	Collision accident during munition handling in onsite container leads to detonation.
433	MB	025	Collision accident during munition handling in offsite container leads to detonation.
434	MB	026	Collision occident in onsite container with prolonged fire lead to thermal detonation.
435	MB	027	Collision accident in offsite container with prolonged fire lead to thermal detonation.
436	MB	029	Drop of pallet containing leaker leads to detonation.
437	MB	030	Drop of single leaking munitions leads to detonation.
438	MB	031	Collision accident involving a leaking munitions leads to detonation.
439	MB	032	Failure to detect ıı leak in the offsite container.
• ⊠ MF			
223	MF	001	Munition pellet or container dropped during movement fra MHI to MDB.
12	MF	002	Rare single mmition dropped during handling inside the MDB.
224	MF	003	Forklift collision accident with short duration fire during handling from MHI to MDB.
225	MF	004	Forklift tine accident handling from the MHI to MDB.
226	MF	005	Collision occident with prolonged fire during handling from MHI to MDB leads to detonation or hydraulic rupture.
227	MF	007	Collision accident without fire.
228	MF	008	Munition dropped inside the MDB.
229	MF	009	Forklift tine accident inside the MDB.
13	MF	010	Collision without fire inside the MDB.
230	MF	011	Drop of mmition pallet from the MHI to MDB leads to detonation.
231	MF	012	Drop of Dare single munition inside the MDB leads to detonation.
232	MF	013	Drop of palletized munition (in container) inside the MDB leads to detonation.
233	MF	014	Collision accident from the MHI to the MOE leads to detonation.
• ⊠ PP			
1	MO	001	Drop of bare pallet or single item at storage area.
2	MO	002	Forklift collision with short &ration fire at storage area involving Dare munitions.
3	MO	003	Forklift tine accident involving Dare munitions at storage area.
4	MO	004	Forklift collision accident without fire at storage area involving Dare munitions.
5	MO	005	Drop of onsite container.
6	MO	006	Forklift collision with short duration fire during handling of onsite container.
7	MO	007	Forklift collision without fire during handling of onsite container.
8	MO	011	Drop of Dare palletized nunition leads to detonation.
9	MO	012	Forklift collision accident at storage area leads to detonation of burstered munition.
10	MO	024	Collision accident during munition handling in onsite container leads to detonation due to impact.
11	MO	026	Collision accident in onsite container with prolonged fire leads to thermal detonation.
490	MO	027	Collision accident in offsite container with prolonged fire leads to thermal detonation.
491	MO	028	Drop of single munitions at maintenance facility leads to detonation.
492	MO	029	Drop of pallet containing leaker leads to detonation.

TABLE J.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
• Accident Scenario Descriptions •

RECORD NUMBER	ID		SCENARIO
493	HO	030	Drop of single leaking mmition leads to detmstion.
494	HO	031	Collision accident involving a Leaker leads to detonation due to impact.
495	HO	032	Failure to detect a leak in the offsite container.
• * HR			
440	NR	DO1	Drop of bare pallet or single item at storage area.
441	HR	002	Forklift collision with short &ration fire at storage area involving bore munitions.
442	HR	003	Forklift tine accident at storage area involving Dare munitions.
443	HR	004	Forklift collision accident at storsge area involving bare munitions.
444	NR	005	Drop of onsite container.
445	HR	006	Forklift collision accident with short duration fire during handling of onsite container.
446	NR	007	Forklift collision without fire during handling of onsite container.
447	HR	008	Drop of offsite container.
448	NR	009	Collision accident with short duration fire during handling of offsite container.
449	HR	010	Collision accident without fire during handling of offsite container.
450	HR	011	Drop of bore palletized mmition leads to detonation.
451	NR	012	Forklift collision accident at storage area leads to detonation.
452	HR	017	Drop of pallet containing a leaking munition during leaker isolation operations.
453	HR	018	Drop of single leaking in leakers processing facility.
454	HR	019	Forklift tine puncture during leaker isolation.
455	HR	021	Collision accident without fire during handling of leaking munition.
456	HR	022	Drop of munition in onsite container leads to detonation.
457	HR	023	Drop of munition in offsite container leads to detonation.
458	RR	024	Collision accident during munition handling in onsite container leads to detonation.
459	HR	025	Collision occident during munition handling in offsite container leads to detonation.
460	HR	026	Collision accident in onsite container with prolonged fire lead to thermal detonation.
461	HR	027	Collision accident in offsite container with prolonged fire lead to thermal detonation.
462	HR	029	Drop of pallet containing leaker leads to detonstion.
463	HR	030	Drop of single leaking munitions leads to detonation.
464	HR	031	Collision accident involving a Leaking munitions leads to detonation.
465	HR	032	Failure to detect a leak in the offsite container.
• ⊠ HS			
478	HS	001	Drop of pallet or container in storage area or maintenance facility; munition punctured.
479	HS	002	Forklift collision with short duration fire.
480	HS	003	Forklift tine puncture.
481	HS	004	Forklift collision without fire.
482	HS	005	Drop of munition leads to detonation.
483	HS	006	Collision accident leads to detonation.
C M	HS	007	Collision accident with prolonged fire.
485	HS	008	Munition pallet dropped during pallet inspection.
486	HS	009	Forklift tine puncture during pallet inspection.
487	HS	010	Forklift collision during pallet inspection.
488	HS	011	Munition pallet dropped during pallet inspection; detonation occurs.
489	HS	012	Forklift collision; detonation occurs.

TABLE J.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
. Accident Scenario Descriptions .

RECORD NUMBER	ID		SCENARIO
* PO			
342	PO	001	Tornado-generated missile puncture/crush munitions in the MHI.
343	PO	002	Tornado-generated missile detonate munitions in the MHI.
344	PO	003	Tornado-generated missile puncture/crush munitions in the UPA.
345	PO	004	Tornado-generated missile detcmate munitions in the UPA.
346	PO	005	Tornado-generated missile damages the agent piping system between the BDS and TOX at TEAD (bulk-only facility).
347	PO	006	meteorite strikes the MHI.
348	PO	007	Meteorite strikes the UPA.
350	PO	008	Meteorite strikes the agent piping system between the BDS and TOX at TEAD (bulk-only facility).
351	PO	009	Direct large aircraft crash onto the MHI, no fire.
280	PO	010	Direct large aircraft crash onto the MHI; fire not contained in 0.5 hours
281	PO	011	Direct large aircraft crash onto the MHI; fire contained in 0.5 hours
282	PO	012	Direct large aircraft crash damages the MDB; no fire
283	PO	013	Direct large aircraft crash damages the MDB; fire not contained in 0.5 hours
284	PO	014	Direct large aircraft crash damages the MDB; fire contained in 0.5 hours
285	PO	015	Indirect large aircraft crash damages the MHI; no fire
286	PO	016	Indirect large aircraft crash damages the MHI; fire not contained in 0.5 hours
287	PO	017	Indirect large aircraft crash damages the MHI; fire contained in 0.5 hours
288	PO	018	Indirect large aircraft crash damages the MDB; no fire
289	PO	019	Indirect large aircraft crash damages the MDB; fire not contained in 0.5 hours
290	PO	020	Indirect large aircraft crash damages the MDB; fire contained in 0.5 hours
352	PO	021	Direct crash of a large or smell aircraft damages the outdoor agent piping system at TEAD, no fire.
353	PO	022	Direct crash of a large or small aircraft damages the outdoor agent piping system at TEAD, fire occurs and not contained.
354	PO	023	Earthquake causes the munitions in the MHI to fall and be punctured.
355	PO	024	Earthqwke causes munitions in the MHI to fall and detonate.
356	PO	025	Earthquake damages the MDB structure, nunitions fall and are punctured, fire suppressed.
357	PO	026	Earthquake damages the MDB structure, munitions fall and are punctured, earthquake also initiates fire, fire suppression system fails.
359	PO	028	Earthquake damages the MDB structure, nunitions fall and are punctured, TOX damaged, fire occurs, fire suppression system fails.
291	PO	029	Earthquake damages the MDB; munitions are intact; fire occurs; fire suppression system fails
360	PO	030	Earthquake damages the MDB, munitions are intact, TOX damaged, no fire occurs.
362	PO	031	Earthquake damages the MDB, munitions *are* intact, TOX damaged, fire occurs, fire'not suppressed.
363	PO	032	Earthquake causes mnitions to fall and detonate, MDB breached by detonation, the TOX is intact, no fire.
364	PO	033	Earthquake causes munitions to fall but no detonation occurs, the MDB is intact, the TOX is intact, earthquake also initiates fire, fire suppression system fails.
365	PO	034	Earthquake causes nunitions to fall but no detonation occurs, the MDB is intact, the TOX is damaged, fire occurs, fire suppression system fails.
234	PO	041	Failure to stop agent feed to the LIC, overloads the ventilation system.
235	PO	042	MPF explosion due to failure to stop fuel flow after a shutdown.
236	PO	043	MPF explosion due to hydraulic rupture of an unpunched bulk item. MPF room and ventilation integrity maintained.
237	PO	044	MPF explosion due to hydraulic rupture of an unpunched bulk item. MPF room or ventilation integrity Lost.
238	PO	045	Ton container is spilled in the ECV, MDB structure fails due to subsequent agent fire.
239	PO	046	Munition detonation in the ECV, no fire.
240	PO	047	Munition detonation in the ECV, fire results but does not propagate.
241	PO	048	Munition detonation in the ECV, fire results and propagates.

TABLE J.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
• Accident Scenario Descriptions .

RECORD NUMBER	ID		SCENARIO
242	PO	049	Munition detonation in ECR causes structural and ventilation system failure.
243	PO	050	Munition detonation in ECR causes structural failure, a fire, and ventilation failure.
244	PO	051	Ton container spill in the MPB results in fire and structural failure.
249	PO	052	A burstered munition is fed to the DUN.
266	PO	052	A burstered munition is fed to the DUN.
349	PO	A07	Meteorite strikes the TOX.
358	PO	A28	Earthquake damages the MDB structure, munitions fall and are punctured, TOX damaged, fire occurs, fire suppressed.
361	PO	A31	Earthquake damages the MDB, munitions are intact, TOX damaged, fire occurs, fire suppressed.
• ⊠ o:			
496	RN	001	A train accident involving a munitions railcar occurs and crush forces fail the agent containment.
497	RN	002	A train accident involving a munitions reilcar occurs and impact forces fail the agent containment.
120	RN	003	A train accident involving a munitions railcar occurs and puncture forces fail the agent containment.
121	RN	004	A train accident with fire occurs. Either the package insulation is torn away due to mechnical forces and the fire is able to heat the munitions inside the package, or the fire Last long enough to cause burstered munitions in the package to detonate. Undue force created by the accident may also cause burster detonation.
122	RN	005	A train accident with fire occurs. Either the package insulation is torn sway due to mechanical forces and the fire is able to heat the aunitions inside the package, or the fire lasts Long enough to cause thermal ruture of the munitions inside the package.
123	RN	006	An aircraft crashes on a munitions railcar. No fire occurs, but impact forces lead to detonations end/or failure of agent containment.
124	RN	007	An aircraft crashes on a munitions railcar. Fire occurs, but impact forces lead to detonations and/or failure of agent containment.
498	RN	008	Combined with scenario RN007.
499	RN	009	A severe ● arthguake occurs involving a munitions railcar and crush forces fail the agent containment.
500	RN	010	A severe ● arthguake occurs involving a nunitions reilcar and impact forces fail the agent containment.
125	RN	011	A severe ● srthquske occurs involving a munitions railcar and puncture forces fail the agent containment.
126	RN	012	A severe earthquake occurs involving a munitions railcar and s&sequent fire detonates burstered.
127	RN	013	A severe earthquake occurs involving a munitions railcar and subsequent fire fails nonburstered munitions.
128	RN	014	A tornado-generate missile leads to failure of the agent containment, or a tornado occurs, causing overturn or derailment of a munitions railcar.
129	RN	015	A earthquake or a tornado occurs, generating undue mechanical forces which cause detonation of burstered munitions.
• * RR			
501	RR	001	A train accident involving a nunitions railcar occurs and crush forces fail the agent containment.
502	RR	002	A train accident involving a munitions railcar occurs and impact forces fail the agent containment.
267	RR	003	A train accident involving a munitions railcar occurs and puncture forces fail the agent containment.
268	RR	004	A train accident with fire occurs. Either the package insulation is torn away due to mechnical forces and the fire is able to heat the nunitions inside the package, or the fire last long enough to cause burstered munitions in the package to detonate. Undue force created by the accident may also cause burster detonation.
269	RR	005	A train accident with fire occurs. Either the package insulation is torn away due to mechanical forces and the fire is able to heat the munitions inside the package, or the fire lasts long enough to cause thermal ruture of the munitions inside the package.
270	RR	006	An aircraft crashes on a munitions railcar. No fire occurs, but impact forces lead to detonations and/or failure of agent containment.
271	RR	007	An aircraft crashes on a munitions railcar. Fire occurs, but impact forces lead to detonations and/or failure of agent containment.

TABLE J.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
Accident Scenario Descriptions

RECORD NUMBER	ID		SCENARIO
SOS	RR	008	Combined with scenario RN007.
504	RR	009	A severe earthquake occurs involving a munitions railcar and crush forces fail the agent containment.
505	RR	010	A severe earthquake occurs involving a munitions railcar and impact forces fail the agent containment.
272	RR	011	A severe earthquake occurs involving a munitions railcar and puncture forces fail the agent containment.
273	RR		A severe earthquake occurs involving a munitions railcar and subsequent fire detonates burstered.
274	RR	013	A severe earthquake occurs involving a munitions railcar and subsequent fire fails nonburstered munitions.
275	RR	014	A tornado-generate missile (ed s to failure of the agent containment, or a tornado occurs, causing overturn or derailment of a munitions railcar.
276	RR	015	A earthquake or a tornado occurs, generating undue mechanical forces which cause detonation of burstered munitions.
** SB			
375	SB	001	area a aircraft direct crash onto transportation containers in holding area; no fire.
376	SB	002	Large aircraft direct crash onto transportation containers in holding area, fire not contained.
377	SB	003	Large aircraft direct crash onto transportation containers in holding area; fire contained.
378	SB	004	Small aircraft direct crash onto transportation containers in holding area; no fire.
379	SB	005	Small aircraft direct crash onto transportation containers in holding area; fire not contained.
380	SB	006	Small aircraft direct crash onto transportation containers in holding area; fire contained.
381	SB	007	Tornado-generated missiles strike munitions in transportation containers in holding area; no detonation.
382	SB	008	Tornado-generated missiles strike munitions in holding area; detonation occurs.
383	SB	009	Meteorite strikes munitions in transportation containers in holding area; fire occurs; detonation (if burstered).
** SL			
262	SL	001	Munitions develops a leak during the in between inspection period.
293	SL	002	Munition punctured by forklift tine during leaker-handling activities.
762	SL	003	Spontaneous ignition of rocket during storage (not analyzed (o r lack of quantitative data).
295	SL	004	Large aircraft direct crash onto storage area; (if a aircraft not contained in 30 min. (Note: Assume detonation occurs if burstered munitions hit, fire involving burstered munitions not contained at all.
962	SL	005	Large aircraft indirect crash onto storage area; fire not contained in 30 min. (See note in SL4.)
297	SL	006	Tornado-generated missiles strike the storage magazine, warehouse, or open storage area; munitions breached (no detonation).
298	SL	007	Severe earthquake breaches the munitions in storage igloos, no detonations.
299	SL	008	Meteorite strikes the storage area; fire occurs; munitions breached (if burstered, detonation also occurs).
300	SL	009	Munition dropped during leaker isolation operation, munition punctured.
301	SL	010	Storage igloo or warehouse fire from internal sources.
302	SL	011	Munitions are dropped due to pallet degradation.
303	SL	012	Liquid petroleum gas (LPG) infiltrates igloo/building.
304	SL	013	flammable liquids stored in nearby facilities explode, fire propagates to munition warehouse (applies to NAAP).
SOS	SL	014	Tornado-induced building collapse leads to breaching/detonation of munitions.
306	SL	015	Small aircraft direct crash onto warehouse or open storage yard, fire occurs, not contained in 30 min.
307	SL	016	Large aircraft direct crash, no fire, detonation (if burstered).
308	SL	017	Large aircraft direct crash, fire contained within 30 min (applies to nonburstered munitions only).
309	SL	018	Small aircraft direct crash into warehouse or open storage yard, no fire.
310	SL	019	Small aircraft direct crash into warehouse or open storage yard, fire contained in 30 min.
311	SL	020	Large aircraft indirect crash onto storage area, no fire.
312	SL	021	Large aircraft indirect crash into storage area, fire contained in 30 min.
313	SL		Severe earthquake leads to munition detonation.
314	SL	023	Tornado-generated missiles strike the storage igloo and leads to munition detonation.

TABLE 3.5 (Cont.)

CSDP/FEIS RISK ANALYSIS
• Accident Scenario Descriptions •

RECORD NUMBER		ID	SCENARIO
315	SL	024	Lightning strikes tan containers stored outdoors.
316	SL	025	Munition dropped during Leaker isolation, munition detonates.
317	SL	A26	Earthquake occurs, NAAP warehouse is intact, no ton containers damaged, fire occurs.
322	SL	A27	Earthquake occurs, TEAD uarehwses intact, munitions intact, fire occurs at one warehouse.
327	SL	A28	Earthquake occurs, UMDA warehouses intact, nunitions intact, fire occurs at one warehouse.
318	SL	826	Earthquake occurs, NAAP warehwse is intact, ton container damaged, no fire.
323	SL	827	Earthquake occurs, TEAD warehouses intact, munitions intact, fire occurs at two warehouses.
328	SL	828	Earthquake occurs, UMDA uarehwses intact, munitions intact, fire occurs at two warehouses.
319	SL	C26	Earthquake occurs, NAAP warehouse is intact, ten container damaged, fire occurs.
324	SC	C27	Earthquake occurs, one TEAD warehouse is damaged, nunitions intact, fire occurs at one warehouse.
329	SL	C28	Earthquake occurs, UMDA warehouses intact, aunitions in one warehwse damaged, no fire occurs.
320	SL	D26	Earthquake occurs, NAAP warehouse is damaged, tan containers damaged, fire occurs.
325	SL	D27	Earthquake occurs, one TEAD warehouse is damaged, nunitions intact, fire occurs at two warehouse.
330	SL	D28	Earthquake occurs, UMDA warehouses intact, aunitions in one warehouse damaged, fire occurs at uarehwse with damaged munitions.
321	SL	E26	Earthquake occurs, NAAP warehouse is damaged, ton containers damaged, no fire occurs.
326	SL	E27	Earthquake occurs, two TEAD warehouses damaged, munitions intact, fire occurs at One warehouse.
331	SL	E28	Earthquake occurs, UMDA warehouses intact, munitions in one warehouse damaged, fire occurs at warehouse with undamaged munitions.
513	SL	F27	Earthquake occurs, 2 TEAD uarehwse damaged; aunitions intact; fire occurs at 2 warehouses.
332	SL	F28	Earthquake occurs, UMDA warehwses intact, munitions in one warehwse damaged, fire occurs at tuo warehouses.
333	SL	G28	Earthquake occurs, UMDA warehwses intact, munitions in two uarehwses damaged, no fire occurs.
334	SL	H28	Earthquake occurs, UMDA warehouses intact, munitions in two warehwses damaged, fire occurs at warehouse with damaged munitions.
335	SL	K28	Earthquake occurs, one UMDA warehwse damaged, munitions in one warehouse damaged, fire occurs at warehouse with damaged munitions.
336	SL	L28	Earthquake occurs, one UMDA warehwse damaged, munitions in one warehouse damaged, fire occurs at two warehouses.
337	SL	M28	Earthquake occurs, one UMDA warehouse damaged, munitions in two warehouses damaged, no fire occurs.
338	SL	N27	Earthquake occurs, one UMDA warehouse damaged, munitions in two warehouses damaged, fire occurs in warehouse with damaged munitions.
339	SL	028	Earthquake occurs, one UMDA warehouse damaged, munitions in two warehouses damaged, fire occurs at two warehouses.
340	SL	P28	Earthquake occurs, two UMDA warehwses damaged, aunitions in two uarehwses damaged, no fire occurs.
341	SL	028	Earthquake occurs, two UMDA warehouses damaged, nunitions in two uarehwses damaged, fire occurs at both warehouses.
• * SR			
14	SR	001	Large aircraft direct crash onto transportation containers in holding area; no fire.
15	SR	002	Large aircraft direct crash onto transportation containers in holding area; fire not contained.
16	SR	003	Large aircraft direct crash onto transportation containers in holding area; fire contained.
17	SR	004	Small aircraft direct crash onto transportation containers in holding area; no fire.
18	SR	005	Small aircraft direct crash onto transportation containers in holding area; fire net contained.
19	SR	006	Small aircraft direct crash onto transportation containers in holding area; fire contained.
20	SR	007	Tornado-generated missiles strike munitions in transportation containers in holding area; no detonation.
21	SR	008	Tornado-generated missiles strike munitions in transportation containers in holding area; detonation occurs.
279	SR	008	Tornado-grnrrated missile penetrates containers; detonation occur.
22	SR	009	Meteorite strikes munitions in transportation containers in holding area; fire occurs; detonation (if burstered).

TABLE 5.5 (Conc.)

CSDP/FEIS RISK ANALYSIS
. Accident Scenario Descriptions .

RECORD NUMBER	ID		SCENARIO
* VB			
[illegible]	VB	001	A snmitions vehicle collision/overturn occurs and crush forces fail the agent containment.
467	VB	002	A munitions vehicle collision/overturn occurs and impact forces fail the agent containment.
[illegible]	VB	003	A munitions vehicle collision/overturn occurs and puncture forces fait the agent containment.
469	VB	004	Detonation of burstered munitions occurs by either 1) fire-only accident, 2) mechanical force end fire, 3) truck collision/overturn impact . induced rocket propellant ignition, or 4) truck collision/overturn • induced undue force detonation.
470	VB	006	An aircraft crashes on a sunitions vehicle. No fire occurs; impact forces fail the agent containment.
471	VB	007	An aircraft crashes on a nnitions vehicle. Fire occurs but impact forces fail the agent contsinnent.
472	VB	009	A severe earthquake occurs, causing a nnitions vehicle accident and crush forces fail the agent containment.
473	VB	010	A severe earthquake occurs, causing a munitions vehicle accident and impact forces forces fail the agent containment.
474	VB	011	A severe earthquake occurs, causing a munitions vehicle accident and puncture forces forces fail the agent containment.
475	VB	012	A severe earthquake occurs, causing a munitions vehicle accident and fire detonates burstered munitions.
476	VB	014	A tornado occurs, generating under mechanical forces which cause detonation of burstered munitions.
477	VB	015	A earthquake or tornado occurs, generating under mechanical forces which cause detonation of burstered snmitions.
** VO			
130	VO	001	A munitions vehicle collision/overturn occurs and crush forces fail the agent containment.
131	VO	003	A munitions vehicle collision/overturn occurs and puncture forces fail the agent containment.
132	VO	004	A munitions vehicle accident with fire occurs, causing detonation of burstered munitions. Ignition of the propellant by a probe could also detonate the burster of a cartridge snd the burster of a rocket could be detonated by impact-induced ignition of the rocket propellant.
133	VO	005	A nmitions vehicle accident with fire occurs, causing non-burstered munitions to fail.
134	vo	006	An eircrsft crashes on a munitions vehicle. No fire occurs; impact forces fail the agent containment.
135	vo	007	An aircraft crashes on a munitions vehicle. Fire occurs; but impact forces fail the agent containment.
136	vo	009	A severe earthquake occurs, causing a munitions vehicle accident and crush forces fail the agent containment.
252	vo	010	A severe earthquake occurs, causing a sunitions vehicle accident and impact forces fail the agent containment.
137	vo	011	A severe earthquake occurs, causing a munitions vehicle accident and puncture forces fail the agent containment.
138	vo	012	A severe earthquake occurs, causing a munitions vehicle accident and fire fails detonates burstered munitions.
139	VO	013	A severe earthquake occurs, causing a sunitions vehicle accident and fire fails nonburstered munitions.
140	VO	014	A tornado occurs, generating a missile or causing a truck overturn and mechanical forces fail agent containment.
141	VO	015	A truck collision/overturn occurs generating undue mechanical forces which cause detonation of burstered munitions.

release would be reduced. The worst-case scenario is a credible condition that results in near-maximum doses, This scenario occurs during a stable atmosphere with a wind speed of 1 m/s.

Three modes of release have been used in the analysis: semicontinuous, instantaneous, and evaporative. A semicontinuous release, applicable for all three agents, involves a steady plume- of vapor (e.g., during a fire or from a vent in a storage igloo) over a specified time period (such as 60 min). An instantaneous release is a single burst of agent, such as in an explosion. The instantaneous release was used only for GB because D2PC does not sufficiently incorporate the evaporation of aerosol following a VX or HD explosion and provides better estimates using the semicontinuous release mode. An evaporative release would follow a spill of agent onto a surface. An evaporative release is considered only for GB and HD because the evaporation rate of VX is so small that an accidental spill would not result in an off-post impact via atmospheric dispersion.

The analysis of accidental releases has included comparisons of the quantity of agent that would be necessary to result in 0%, 1%, and 50% fatality rates for different agents, release modes, meteorological conditions, and downwind distances. Tables K.1-K.3 illustrate representative results for GB, VX, and HD, respectively. Not surprisingly, the tables indicate that the amount of agent required during the release is greater for greater distances, and the amount required also increases as the fatality rate increases from 0% to 1% to 50%. The largest downwind distance listed in the tables is 100 km. Because D2PC does not account for changes in wind velocity or atmospheric stability that would occur over long time periods and large distances, the results are overly conservative at large distances, and fatalities are unlikely beyond 100 km.

For a semicontinuous release, VX is the agent for which the smallest quantity results in a given fatality rate; HD requires the largest amount. For an evaporative release, a much smaller quantity of GB than HD is needed because the evaporation rate of GB is so much greater. The amount of agent estimated in the evaporative release is the quantity of liquid that is spilled. For the other types of release, the amount is the total quantity that is airborne over the release period.

K.3 LIMITATIONS OF THE D2PC MODEL

The D2PC model does not account for topography or changes in wind velocity or atmospheric stability with time or location. The code makes a number of adjustments to compensate for these limitations, but the basic shortcomings of the model remain and must be considered in the analysis of the results of the code. Although the shortcomings affect predicted concentrations near the source, the errors are not large, and the results are acceptable. At large distances, errors caused by the model dominate the results. With conservative but realistic estimates of wind speed and stability, the downwind distance to the no-effects

REFERENCES

References Cited

Arthur D. Little, Inc. (1985a), Analysis of Risk Associated of M55 Rocket Demilitarization Plants, M55-OD-3. Prepared for the U.S. Army Toxic and Hazardous Materials Agency.

Arthur D. Little, Inc. (1985b), Analysis of Risks Associated with Operation of the JACADS M55 Rocket Demilitarization Plant, M55-OD-9. Prepared for the U.S. Army Toxic and Hazardous Materials Agency.

Arthur D. Little, Inc. (1985c), Analysis of the Converted M55 Rocket Demilitarization Plant at Pine Bluff Arsenal, M55-OD-10. Prepared for the U.S. Army Toxic and Hazardous Materials Agency.

Arthur D. Little, Inc. (1985d), Analysis of Risks Associated with Operation of the CAMDS M55 Rocket Demilitarization Plant, M55-OD-11. Prepared for the U.S. Army Toxic and Hazardous Materials Agency.

Chu, S.C. et al. (1987), Transportation of Chemical Aeents and Munitions: A Concept Plan, WP-87W00205, McLean, VA: The MITRE Corporation. Prepared for the U.S. Army Office of the Program Executive Officer -- Program Manager for Chemical Demilitarization. SAPEO-CDE-IS-87003.

Clarke, R.K., et al. (1976), Severities of Transportation Accidents, SLA-74-0001, Sandia National Laboratories.

Fraize, W.E. et al. (1987a), Risk Analysis Supporting the Chemical Stockpile Disposal Pronram (CSDP), MTR-87W00203, Vol. 1: Analysis (Unclassified), Vol. 2: Appendices A-C (Unclassified), Vol. 3: Consequence Data (Unclassified), Vol. 4: Risk Analysis Summary Tables (SECRET), Vols. 5 - 8: Detailed Risk Data (SECRET), McLean, VA: The MITRE Corporation, prepared for the U.S. Army, Office of the Program Executive Officer -- Program Manager for Chemical Demilitarization. SAPEO-CDE-IS-87014.

Fraize, W.E. et al. (1987b), Preliminary Risk Analysis Supporting the Chemical Stockpile Disposal Program (CSDP), MTR-86W00004, McLean, VA: The MITRE Corporation, prepared for the U.S. Army Toxic and Hazardous Materials Agency.

GA Technologies Inc. (1987a), Risk Analysis of the Onsite Disposal of Chemical Munitions, GA-C18562, (Aug. 1987). Prepared for the U.S. Army, Office of the Program Executive Officer -- Program Manager for Chemical Demilitarization. SAPEO-CDE-IS-87010.

GA Technologies Inc. (1987b), sk Analvsis of the Disoosal of Chemical Munitions at National or Reeional Sites GA-C18563, (Aug. 1987), prepared for the U.S. Army, Office of the Program Executive Officer -- Program Manager for Chemical Demilitarization. SAPEO-CDE-IS-87008.

GA Technologies Inc. (1987c), Risk Analvsis of the Continued Storaee of Chemical Munitions, GA-C18564, (Aug. 1987), prepared for the U.S. Army, Office of the Program Executive Officer -- Program Manager for Chemical Demilitarization. SAPEO-CDE-IS-87009.

Rasmussen, N. et al. (1975), Reactor Safety Study, WASH-1400, prepared for the Nuclear Regulatory Commission.

Rhyne, W.R., et al. (1985a), Probabilistic Analvsis of Chemical Aeent Release During Transport of M55 Rockets, M55-CD-4, H&R Technical Associates, Inc., Report No. 255-l. Prepared for the U.S. Army Toxic and Hazardous Materials Agency.

Rhyne, W.R., et al. (1985b), Probabilistic Analysis of Chemical Agent Release Durine Transport of Obsolete Munitions With and Without Additional Protective Packaging, 244-3/244-4, H&R Technical Associates, Inc.

Science Applications International Corporation (1985), Probabilities of Selected Hazards in Disposition of M55 Rockets, M55-CS-2. Prepared for U.S. Army Toxic and Hazardous Materials Agency.

The R.M. Parsons Company, (1983), JACADS • Preliminary Hazards Analysis of Final Concept Desien, Task G-5C Interim Report. Prepared for the U.S Army Engineers Division.

The R.M. Parsons Company (1985), Fault Tree Analvsis. Johnston Atoll Chemical Aeent Disposal System, Task G-SC. Prepared for the U.S. Army Engineers Division.

U.S. Army Toxic and Hazardous Materials Agency (1986), Chemical Stockpile Disposal Concept Plan (Final Draft), Aberdeen Proving Ground, MD.

U.S. Army Program Manager for Chemical Demilitarization (1986), Chemical Stockpile Program • Draft Environmental Impact Statement, Aberdeen Proving Ground, MD.

U.S. Army Program Executive Office • Program Manager For Chemical Demilitarization (1987a), Mitigation of Public Safety Risks of the Chemical Stockpile DisDosal Program, SAPEO-CDE-IS-87014, Aberdeen Proving Ground, MD.

U.S. Army Program Executive Office • Program Hanager For Chemical Demilitarization (1987b), Transportation of Chemical Munitions at Reduced Temperature, SAPEO-CDE-IS-87002, Aberdeen Proving Ground, MD.

Whitacre, C.G. et al. (1987), Personal Computer Program for Chemical Hazard Prediction, CRDEC-TR-87021, Aberdeen Proving Ground, MD: Chemical Research, Development & Engineering Center.

Appendix K

ATMOSPHERIC DISPERSION

K.1 INTRODUCTION

The dispersion of chemical agent from an accidental release is of interest in determining the impacts of such a release and the relative risk involved in various disposal alternatives. The analysis of these risks is of utmost importance in the final programmatic environmental impact statement for the disposal of the chemical stockpile. Therefore, the techniques used to estimate the effects of agent releases must be defensible, conservative, and physically consistent. Predicting the areas affected by accidental releases of nerve agents has been accomplished with a Gaussian-plume dispersion model (D2PC) developed by the U.S. Army's Chemical Research and Development Center (Whitacre 1986) and a box model developed by Oak Ridge National Laboratory.

K.2 D2PC ATMOSPHERIC DISPERSON MODEL

The D2PC computer program has been used to predict the areas affected by time-weighted concentrations likely to produce lethalities, the primary concern of the Army. The D2PC computer program (or code) is an air dispersion model that assumes a Gaussian distribution of agent in the vertical and cross-wind directions as the agent disperses downwind. This assumption has been standardly used in atmospheric dispersion modeling. The D2PC code incorporates detailed information on the type of accident, such as type of agent, type of release (e.g., explosion, fire, or spill), and duration of release. A vapor depletion technique is also included in D2PC to estimate the removal *of* agent vapor from the atmosphere by deposition on surfaces.

The D2PC code predicts dosage of agent expected at locations downwind of the release. Dosage is defined as the multiplicative product of agent concentration and the duration of exposure. D2PC has been used to estimate areas receiving dosages that would, result in human fatalities. The dosages that have been used in this study are those resulting in fatality rates of 0%, 1%, and 50%. The dosage corresponding with the 0% rate, also known as the "no-deaths" dose, is the largest dosage that would result in no fatalities to healthy adults.

Atmospheric dispersion varies considerably according to meteorological conditions during an accidental release. Consequently, the impact of a release also varies tremendously depending on meteorological conditions. This study evaluated releases occurring under two different conditions: conservative most-likely and worst-case. The conservative most-likely scenario is a frequently occurring meteorological condition that results in relatively large doses compared with other frequently occurring conditions. Specifically, this scenario occurs during neutral atmospheric stability (halfway between stable and unstable) with a wind speed of 3 m/s. Stability is the change of temperature with height in the atmosphere. The more the atmosphere cools with increasing height, the less stable the atmosphere is, which allows greater mixing and enhances dispersion so that doses resulting from a

release would be reduced. The worst-case scenario is a credible condition that results in near-maximum doses. This scenario occurs during a stable atmosphere with a wind speed of 1 m/s.

Three modes of release have been used in the analysis: semicontinuous, instantaneous, and evaporative. A semicontinuous release, applicable for all three agents, involves a steady plume- of vapor (e.g., during a fire or from a vent in a storage igloo) over a specified time period (such as 60 min). An instantaneous release is a single burst of agent, such as in an explosion. The instantaneous release was used only for GB because D2PC does not sufficiently incorporate the evaporation of aerosol following a VX or HD explosion and provides better estimates using the semicontinuous release mode. An evaporative release would follow a spill of agent onto a surface. An evaporative release is considered only for GB and HD because the evaporation rate of VX is so small that an accidental spill would not result in an off-post impact via atmospheric dispersion.

The analysis of accidental releases has included comparisons of the quantity of agent that would be necessary to result in 0%, 1%, and 50% fatality rates for different agents, release modes, meteorological conditions, and downwind distances. Tables K.1-K.3 illustrate representative results for GB, VX, and HD, respectively. Not surprisingly, the tables indicate that the amount of agent required during the release is greater for greater distances, and the amount required also increases as the fatality rate increases from 0% to 1% to 50%. The largest downwind distance listed in the tables is 100 km. Because D2PC does not account for changes in wind velocity or atmospheric stability that would occur over long time periods and large distances, the results are overly conservative at large distances, and fatalities are unlikely beyond 100 km.

For a semicontinuous release, VX is the agent for which the smallest quantity results in a given fatality rate; HD requires the largest amount. For an evaporative release, a much smaller quantity of GB than HD is needed because the evaporation rate of GB is so much greater. The amount of agent estimated in the evaporative release is the quantity of liquid that is spilled. For the other types of release, the amount is the total quantity that is airborne over the release period.

K.3 LIMITATIONS OF THE D2PC MODEL

The D2PC model does not account for topography or changes in wind velocity or atmospheric stability with time or location. The code makes a number of adjustments to compensate for these limitations, but the basic shortcomings of the model remain and must be considered in the analysis of the results of the code. Although the shortcomings affect predicted concentrations near the source, the errors are not large, and the results are acceptable. At large distances, errors caused by the model dominate the results. With conservative but realistic estimates of wind speed and stability, the downwind distance to the no-effects

Table K.I. Approximate quantity of CB release equivalent to 50%, 1%, and 0% fatality rates as a function of tbe release mode, distance from tbe source, and meteorological conditions

Downwind distance (km)	Fatality rate					
	50%		1%		0%	
	(kg)	(lb)	(kg)	(lb)	(kg)	(lb)
GB, instantaneous release						
	Conservative *most likely meteorological* conditions					
0.5	18	40	3	6	2	4
1	70	150	11	25	7	15
2	320	700	45	100	27	60
5	1,800	4,000	320	700	180	400
10	9,000	20,000	1,600	3,500	900	2,000
20	36.400	80,000	7,000	15,000	3,900	8,500
50	180,000	400,000	45,000	100,000	27,000	60,000
loo	NA"	N A	N A	N A	450.000	1,000,000
	Worst-case meteorological conditions					
0.5	2	5	0.4	0.8	0.2	0.5
1	7	15	1.1	2.5	0.7	1.5
2	36	80	7	15	3	7
5	180	400	23	50	14	30
10	700	1,500	90	200	70	150
20	2,300	5,000	320	700	180	400
50	13.600	30,000	1,800	4,000	900	2,000
100	40,000	90,000	7,000	15,000	4,000	9,000
CB, semicontinuous release (60 min)						
	Coruewative most likely meteorological conditions					
0.5	90	200	14	30	7	15
1	360	800	45	100	27	60
2	1,100	2,500	180	400	90	200
5	7,000	15,000	1,100	2,500	700	1,500
10	36,400	80.000	7,000	15,000	3,000	6,500
20	136,000	300,000	27,000	60,000	13,600	30,000
50	N A	N A	270.000	600,000	90,000	200,000
100	N A	N A	N A	N A	450,000	1,000,000
	Worst-case meteorological conditions					
0.5	9	20	1.5	3	1	2
1	32	70	4.5	10	3	6
2	90	200	14	30	7	15
5	400	900	70	150	40	90
10	1,800	4,000	230	500	140	300
20	7.000	15,000	700	1,500	450	1,000
50	27,000	60,000	4,500	10.000	2,500	5.500
100	70,000	150,000	13,600	30,000	9,000	20,000

Table K.1 (continued)

Downwind distance (km)	Fatality rate					
	50%		1%		0%	
	(kg)	(lb)	(kg)	(lb)	(kg)	(lb)
CB, evaporation from a spill (60 min)						
Conservative most likely meteorological conditions						
0.5	450	1,000	55	120	27	60
1	2,200	4,900	210	460	130	280
2	8,000	17,500	1,100	2.500	450	1,000
5	57.000	125,000	7,600	16,700	4,500	10,000
10	360,000	800,000	52,300	115,000	23,000	50,000
20	N A	N A	270,000	600,000	136,000	300,000
50	N A	N A	N A	N A	N A	N A
100	N A	N A	N A	N A	N A	N A
Worst-case meteorological conditions						
0.5	45	100	4	9	2.7	6
1	200	450	23	50	14	30
2	650	1,400	70	160	36	80
5	2,300	5,000	400	830	230	500
10	13,600	30,000	1,700	3,750	700	1,500
20	55,000	120,000	5,500	12,000	3,600	8,000
50	300,000	650.000	50,000	108,000	27,000	60,000
100	N A	N A	136,000	300.000	90,000	200,000

'Quantity is greater than 450,000 kg (1,000,000 lb), but a specific estimate is not available.

Table K.2. Approximate quantity of VX release equivalent to 50%, 1%, and 0% fatality rates as a function of the release mode, distance from the source, and meteorological conditions

Downwind distance (km)	Fatality rate					
	50%		1%		0%	
	(kg)	(lb)	(kg)	(lb)	(kg)	(lb)
VX, semicontinuous release (60 min)						
	Conservative *most likely* ***meteorological*** conditions					
0.5	32	70	4.5	10	3.2	7
1	136	300	18	40	11	25
2	450	1,000	70	150	45	100
5	3,200	7,000	450	1,000	270	600
10	13,600	30,000	1,800	4,000	900	2,000
20	45,000	100,000	9,000	20,000	4,500	10,000
50	320,000	700,000	70,000	150,000	30,000	65,000
100	NA[a]	NA	450,000	1,000,000	136,000	300,000
	Worst-case *meteorological conditions*					
0.5	4	9	0.7	1.5	0.4	0.8
1	11	25	1.8	4	1.4	3
2	39	85	7	15	3.6	8
5	180	400	30	65	16	35
10	700	1.500	90	200	70	150
20	2,300	5,000	320	700	180	400
50	11,000	25,000	1,600	3,500	900	2,000
100	36,000	80,000	7,000	15,000	3,200	7,000

[a] Quantity is greater than 450,000 kg (1,000,000 lb), but a specific estimate is not available.

Table K.3. Approximate quantity of HD release equivalent to 50%, 1%, and 0% fatality rites as a function of the release mode, distance from the source, and meteorological conditions

Downwind distance (km)	Fatality rate					
	50%		1%		0%	
	(kg)	(lb)	(kg)	(lb)	(kg)	(lb)
HD, **semicontinuous** release (60 **min**)						
	Conservative most likely *meteorological* conditions					
0.5	900	2,000	90	200	70	150
1	3,200	7,000	320	700	230	500
2	11,000	25,000	1,100	2,500	900	2,000
5	70,000	150,000	7,000	15,000	4,500	10,000
10	270,000	600,000	36,000	80,000	20,500	45,000
20	NA[a]	NA	136,000	300,000	90.000	200,000
50	NA	NA	NA	NA	NA	NA
100	NA	NA	NA	NA	NA	NA
	Worst -case meteorological conditions					
0.5	110	250	11	25	7	15
1	320	700	27	60	18	40
2	900	2,000	90	200	70	150
5	3,600	8,000	360	800	230	500
10	13,600	30,000	1,600	3,500	900	2,000
20	36.400	80,000	4,500	10,000	2,700	6,000
50	160,000	350,000	18,000	40,000	13,600	30,000
100	450,000	1,000,000	70,000	150,000	40,000	90.000
HD, evaporation from a spill (60 min)						
	Conservative most likely meteorological conditions					
0.5	180,000	400,000	18,000	40,000	13,600	30,000
1	NA	NA	80,000	170,000	55,000	120,000
2	NA	NA	290,000	630,000	230,000	500,000
5	NA	NA	NA	NA	NA	NA
10	NA	NA	NA	NA	NA	NA
20	NA	NA	NA	NA	NA	NA
50	NA	NA	NA	NA	NA	NA
100	NA	NA	NA	NA	NA	NA
	Worst-case meteorological conditions					
0.5	13,600	30,000	1,400	3.200	900	2,000
1	64,000	140,000	5,500	12,000	4,500	10,000
2	180,000	400,000	18,000	40,000	13,600	30,000
5	NA	NA	71,700	1160,000	45,000	100,000
10	NA	NA	NA	NA	270,000	600,000
20	NA	NA	NA	NA	NA	NA
50	NA	NA	NA	NA	NA	NA
100	NA	NA	NA	NA	NA	NA

[a] Quantity is greater than 450,000 kg (1,000,000 lb), but a specific estimate is not available.

concentration (determined by the Army Surgeon General as the concentration the general public can be exposed to for 72 h with no harm; see Department of Defense 1984) predicted by the D2PC code stretches for hundreds to thousands of kilometers. These estimates are meaningless because they are overly conservative. An attempt to improve the accuracy of the estimates by incorporating plume depletion was made (M. Myirski, U.S. Army Chemical Research Development Center, personal communication to F. C. Kornegay, Oak Ridge National Laboratory, Oak Ridge, Tenn., August 1985), but the areas calculated to be affected by the no-effect concentrations remained unrealistically large. These results are obviously incorrect, because travel time of the plume to 2500 km would be on the order of days, and wind conditions would not remain steady for such a period or distance. For this reason, the D2PC results for the no-effect level are invalid and should not be used in an accident evaluation.

The areas affected by a large release would be large. However, assuming that the plume runs straight from the point of release with no meander and that it spreads only at the rate predicted by the Pasquill-Gifford-Turner curves used in the D2PC code for neutral or stable atmospheres is not plausible. The Pasquill-Gifford curves, used to estimate plume spread, are not valid beyond a few kilometers. Both Pasquill and Gifford have admonished users of the Gaussian-plume technique of the inappropriateness of extrapolating their findings beyond a few kilometers (Pasquill and Smith 1983). The technique is ***not*** to be used to simulate transport of material to distances beyond a few kilometers or to regions with different meteorological conditions than those at the point of release.

A number of techniques are available to overcome this shortcoming and to provide estimates of concentrations at greater downwind distances. Some dispersion codes are used to simulate longer-range transport of material, such as acid rain or nuclear fallout. Codes 'like the ASTRAP code from Argonne National Laboratory (Sheih 1977; Shannon 1981) or the ARAC codes (Dickerson et al. 1974) from Lawrence Livermore National Laboratory are used to estimate concentrations at greater distances. However, the use of such codes is not appropriate for this study. Here, the D2PC code is used in a diagnostic mode, to determine the consequences of an accident under conservative most-likely or worst-case meteorological conditions. To simulate these circumstances with the ARAC code or another longer-range code would require far more meteorological data than are available for any installation and would require the code to be run with numerous meteorological and source events to predict the worst-case consequences. Because of the inaccuracies in the wind field data and the uncertainties in dispersion during a diurnal cycle, the results would not be defensible.

K.4 MODIFIED BOX MODEL

A modified box model was developed by Oak Ridge National Laboratory to estimate the downwind distance to the no-effects concentration from an accidental release. The model was used to calculate all no-effects distances. In past investigations of large-scale problems (e.g., photochemical oxidants or pollution-control strategies), a box-model approach has been used with good results (Hanna, Briggs, and Hosker 1982). This approach has the merits of being simple to implement, is based on historical studies, and is a proven technique. For this study, the parameter to be calculated is the volume of the box that contains the released material remaining after vapor depletion is incorporated at the no-effects concentration (assuming uniform mixing). This approach is applicable, even though it is different from previous studies in which the volume was known and the concentrations were calculated.

The model assumes that the agent contained in the no-deaths downwind area, as calculated by the D2PC code, is further dispersed downwind. The volume of atmosphere required to contain that amount of agent is calculated, assuming uniform lateral and vertical distribution of material. The size of the initial volume of agent is from the D2PC code (from the point of release to the no-deaths distance). This volume moves downwind, with the leading edge traveling at the transport wind speed plus a downwind dispersion factor of 0.33 of the transport velocity, while the trailing edge moves at the transport wind speed minus a lagging dispersion factor, assumed to be 0.66 of the transport velocity. The cloud expands within a 30' arc and is uniformly mixed in the vertical up to an assumed 750-m mixing height.

The assumed configuration is shown in Fig. K.1. At each 15-min time step, the volume of the moving box is calculated and the amount of agent remaining in the cloud is determined by selecting the values from a D2PC code run with vapor depletion over long time periods. The resulting concentration (amount of agent divided by volume) is compared with the no-effects concentration values. Transport continues until the volume contained in the box is greater than that required to dilute the remaining agent to below the no-effects level, at which point the downwind no-effects distance for that release is determined.

This approach represents an attempt to reasonably bound the problem. Because no reliable technique exists to estimate transport and diffusion over these distances, this approach is taken to determine reasonable limits of impacts to ecological systems.

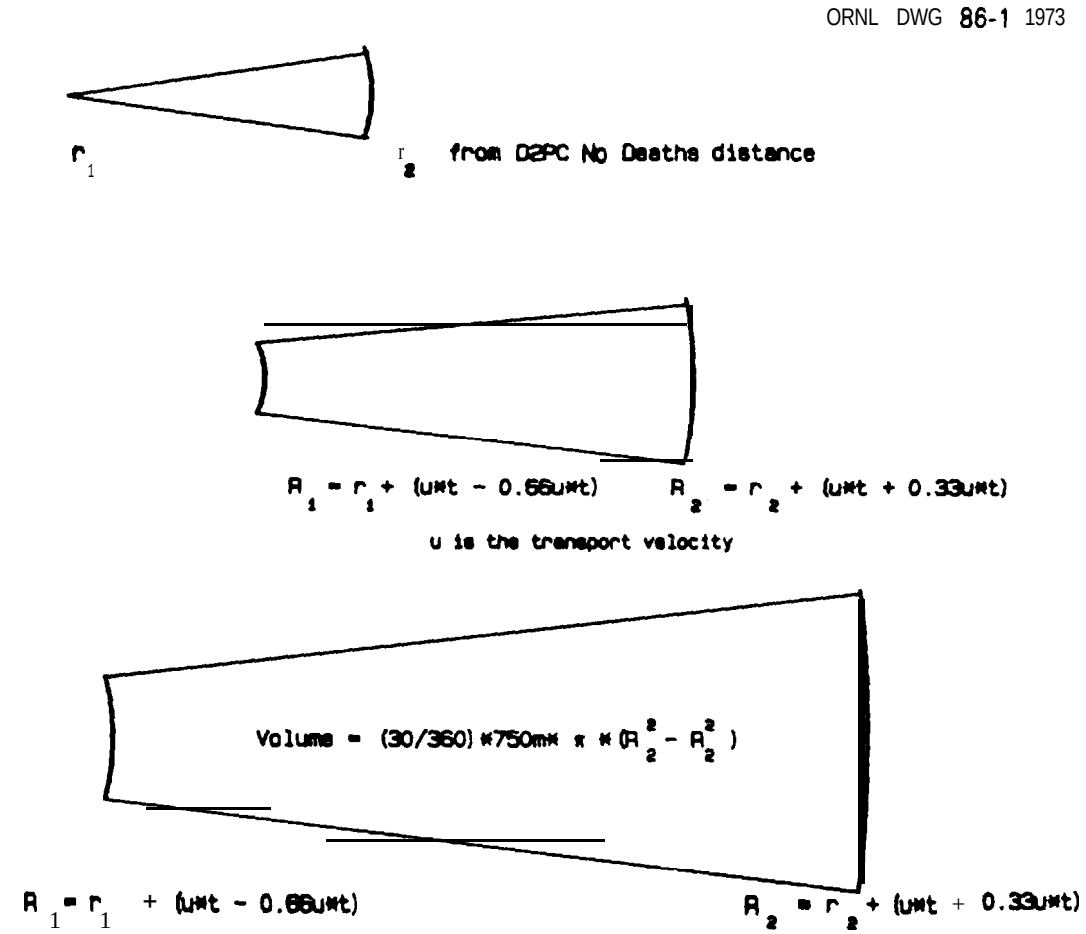


Fig. K.l. Modified box model that estimates the downwind distance to the no-effects concentration from an accidental release.

REFERENCES FOR APPENDIX K

Dickerson, M. H., et al. 1974. A Concept *for an* Atmospheric *Release* Advisory *Capability,* unnumbered reports, Lawrence Livermore National Laboratory, Liver-more, Calif.

Hanna, S. R., Briggs, G. A., and Hosker, R. P., Jr. 1982. *Handbook on Atmospheric Diffusion,* DOE/TIC-11223, U.S. Department of Energy, Oak Ridge, Tenn.

Lewellen, W. S., Sykes, R. I., and Oliver, D. 1982. *The Evaluation of MATHEW/ADPIC as a Real-Time Dispersion Model,* NUREG/CR-2199, U.S. Nuclear Regulatory Commission, Washington, D.C.

Pasquill, F., and Smith, F. B. 1983. *Atmospheric Diffusion,* 3rd edition, Ellis Horwood, Chichester, England.

Science Applications International Corporation 1985. *Probabilities of Selected Hazards in Disposition of M55 Rockets,* La Jolla, Calif.

Shannon, J. D. 1981. "A Model of Regional Long-Term Average Sulfur Atmospheric Pollution, Surface Removal, and Net Horizontal Flux," *Atmospheric Environment 15, 689-701.*

Sheih, C. M. 1977. "Application of a Statistical Trajectory Model to the Simulation of Sulfur Pollution over Northeastern United States," Atmospheric Environment 11, 173-78.

U.S. Department of Defense 1984. *Ammunition and Explosive Safety* Standards, DOD 6055.9-STD, Washington, D.C.

Whitacre, *G. C.,* et al. *1986. Personal Computer Program for Chemical Hazard Prediction (D2PC),* U.S. Army Chemical Research and Development Center, Aberdeen Proving Ground, Md.

Appendix L

GENERIC EMERGENCY RESPONSE CONCEPT PLAN

L.l INTRODUCTION

A comprehensive emergency response program must be implemented at each of the stockpile storage locations for any of the disposal alternatives selected, including continued stockpile storage. The stockpile presents risks to the public that necessitate an emergency response program for each site until the chemical agent is removed. For the national, regional, and limited collocation disposal alternatives, a fixed-site *emergency* response program is necessary at each shipping and receiving point until the stockpiles are completely removed from a site or destroyed. Disposal alternatives involving transportation of chemical agents pose additional emergency response requirements for potential accidents occurring in transit. They involve unique considerations beyond the fixed-site program requirements for the eight stockpile locations. Emergency response concepts for each of the alternatives involving transportation are considered independently, and additional requirements unique to each alternative are presented in the Emergency Response Concept Plan (ERCP) (Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987). The fixed-site emergency response concepts warrant primary attention, in that they apply to all of the disposal alternatives considered and to the present and continued storage of chemical agent at the eight storage depots (see Fig. L.l).

L.2 ALTERNATIVE DISPOSAL OPTIONS

Assessments of the existing emergency response programs at the eight stockpile storage locations were conducted, focusing largely on community emergency response capabilities and the U.S. Army's organization and procedures for coordination of emergency response activities with community agencies. These assessments point out a number of generic emergency response program components that must be developed or improved. Such program developments and improvements must be quickly implemented. The site-specific emergency response concepts developed in the ERCP will be used as the conceptual basis for development of site-specific emergency response programs, regardless of which disposal option(s) is selected.

L.2.1 SITE-SPECIFIC EMERGENCY RESPONSE

The site-specific emergency response program will establish an adequate level of preparedness in all emergency management component areas. This program involves cooperative interaction between the Army and the appropriate governmental authorities in the area surrounding each of the storage installations. The program provides the organization, relationships, procedures, and systems that will support effective management of emergency response for any potential accidents. Emergency response management includes all functions needed to protect

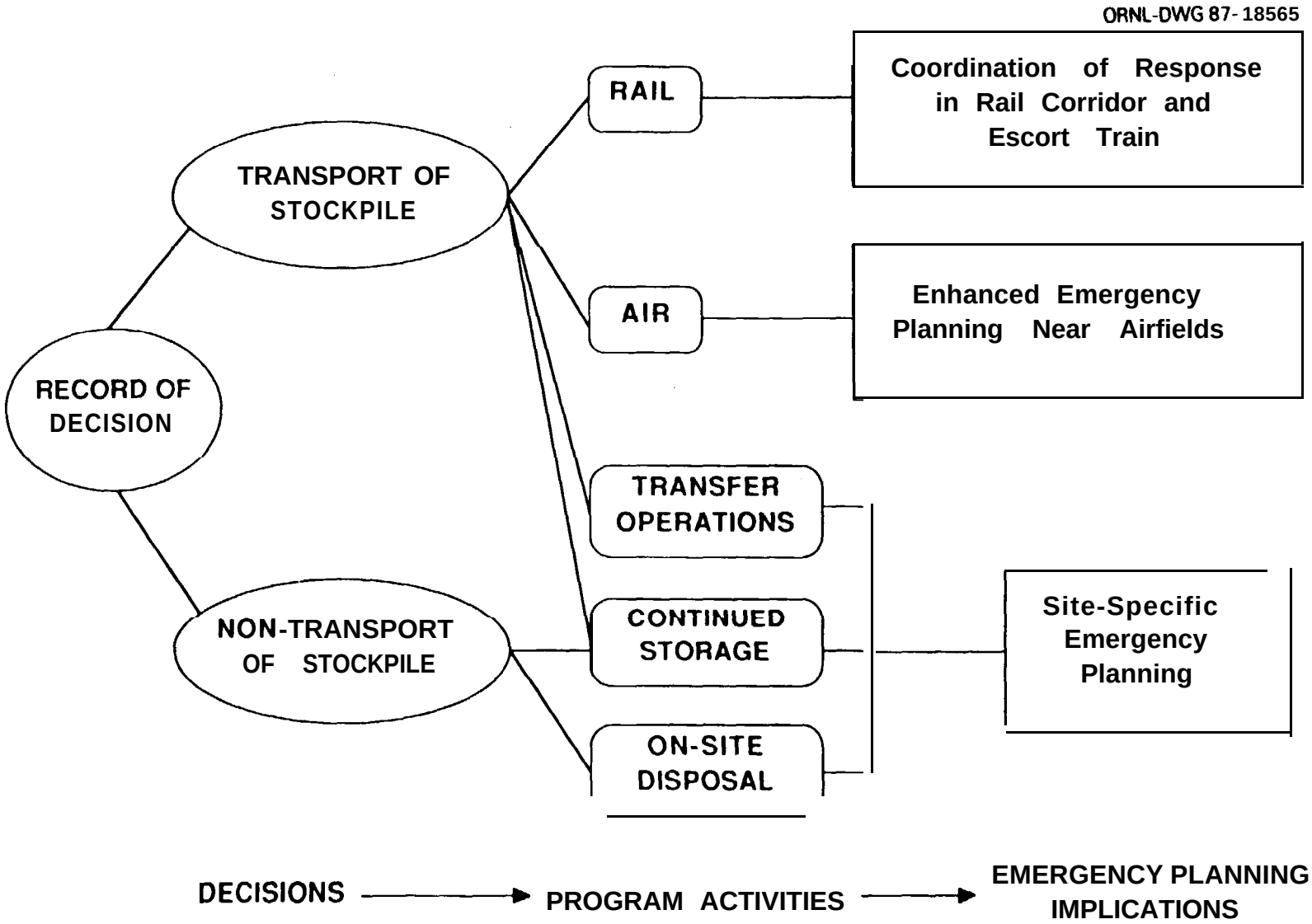


Fig. L.1. Emergency planning implications of disposal alternatives.

the public potentially affected by an accident, such as appropriate communication, command, and control structures to support timely and effective assessment of accidents, as well as cooperative decision-making on the protective actions to be implemented.

The most appropriate public protective action(s) will be analyzed on a site-specific basis. The principal protective action options described in the ERCP are applied selectively to ensure that the proper equipment, supplies, shelter enhancements, information, and training needed for each site are identified. Site-specific plans will identify the special populations and institutions that require unique assistance or protection and the appropriate support mechanisms to be established. An important component of each site-specific program is an emergency warning system capable of alerting people and providing emergency instructions to the public in the event of an accident. Special consideration will be given to the public in closest proximity to the depot, where time to implement a protective action is most limited. The full range of response mechanisms for timely and effective public protection will be established for each site.

The resources required to develop and implement the emergency response plans will be identified from within the Army, the local communities, and other local, state, and federal agencies which have a role in emergency response. Agreements will be coordinated in advance with all applicable groups and agencies. These agreements will include providing resources for transportation, emergency medical services, mass care, security, and protective equipment and other resources to support the full range of potential emergency responses.

Public information programs will be instituted to ensure that community residents understand the nature of the disposal process and the emergency response programs established for their protection. These program also prepare people to react appropriately in the event of an accident. A comprehensive training program for emergency response personnel, including operational exercises, will be established and repeated regularly to ensure that all of the provisions of the emergency response program can be effectively implemented at any time during the chemical stockpile storage and disposal process.

L.2.2 EMERGENCY RESPONSE FOR TRANSPORTATION ALTERNATIVES

Disposal alternatives that involve transportation of chemical agents include: (1) the movement of chemical agents by rail for the regional and national disposal alternatives and (2) the movement of chemical agents by air from Lexington-Blue Grass Army Depot (LBAD) and Aberdeen Proving Ground (APG) to Tooele Army Depot (TEAD) for collocation of the stockpiles. These disposal alternatives pose unique additional requirements for alternative-specific emergency response programs for the principal transportation modes. Although the regional and national disposal alternatives are separate and distinct disposal alternatives, the emergency response concepts that apply to each are similar and they

are considered together under the rail transportation emergency response concepts.

Because on-site storage and disposal activities involve fixed and defined sites, emergency response programs will be quite detailed and specific for the geographic areas that predictably can be impacted by an accident. In the case of the transportation alternatives, the potential area of impact is substantially undefined and it is not possible to have advance knowledge of the specific location of an accident. In the case of movement of chemical agents by air from LBAD and APG to TEAD, however, it is possible to identify an area of high risk. This high-risk area defines an extended zone at either end of the runway at these installations and at emergency landing sites as a definable area for which detailed and specific emergency response planning is practical. For accidents in transit outside of the departure and landing zones for the air transport alternatives, and for the other transportation alternatives, emergency response concepts must be based upon mobile and/or broad-area capabilities for response to accidents.

The emergency response concepts for fixed sites will be implemented based upon the feasibility of detailed site-specific planning. It is not practical to develop such detailed planning for the transportation corridors associated with the various alternatives. For this reason, emergency management concepts for accidental chemical agent releases in transit must be of a more general nature. They must be based either on transporting the appropriate emergency response capability with the stockpile transport or on establishing a broad-area response capability organized at the state level or regionally for the various corridors. The emergency response programs developed for in-transit accidents cannot be as responsive or as complete as those for fixed sites, because of the expansive areas involved and the inability to pinpoint the specific location of an accident prior to its occurrence. Thus the same level of mitigation cannot be provided for in-transit accidents.

L.2.2.1 Rail Transportation

A mobile emergency response capability for public protection is appropriate for alternatives involving rail transportation. An escort train that will accompany the munitions train contains a variety of response capabilities. These capabilities cannot equal the level of preparedness implemented for fixed sites, because of the limitations associated with a mobile vs a fixed capability. A state-level and regional coordination system will be established to support the mobile capability but primarily will provide secondary emergency response capacity.

L.2.2.2 Air Transportation

The limited collocation of the LBAD and APG stockpiles by air transport to TEAD cannot be effectively supported by a mobile response capability. It would be logistically impractical to move sufficient

emergency response resources by air, along with the air shipments, to provide response capabilities at a crash site, should there be an accident in transit. An alternative approach could involve land-based response capabilities stationed at established distances along the air transport route. It is unlikely that such a response capability would be effective for primary response, as it could potentially take an extended period of time to locate and reach a crash site. Emergency landing sites will be designated for air transportation routes; and limited emergency response capabilities will be established at these locations.

L.2.2.3 Summary of Transportation Alternatives

Each of the disposal alternatives involving transportation of chemical agents poses unique emergency response planning situations. Programs that can potentially mitigate the effects of an accident will be established for each alternative. Because of the large geographical areas potentially affected by an accident in transit, the emergency response programs cannot be as effective as those for a fixed, defined site. In *most* cases, emergency response programs may not mitigate the effects of chemical agent accidents in transit. In nearly all cases, emergency responses for in-transit accidents cannot provide a degree of protection which is comparable to that for fixed sites. It should be recognized that emergency landing sites may pose increased risk in that they are designated for use when an aircraft is damaged or otherwise distressed or when munitions leaks occur in transit.

L.3 EMERGENCY PLANNING ZONE CONCEPTS

Emergency planning zones (EPZ) have been developed to support the development of fixed-site and transportation alternative emergency response concepts. Before detailed site-specific or alternative-specific emergency response planning can occur, it is necessary to determine the potentially affected areas. The EPZs establish the areas where the emergency response concepts will be applied. The EPZs are defined generically for the eight sites and for the transportation alternatives. They are developed in consideration of the risk analysis performed for the chemical stockpile disposal program. The critical factors for emergency planning, as identified in the hazard analysis, are considerations of time and distance. The EPZs reflect the differing emergency response requirements associated with the potential rapid onset of an accidental release of agent and the limited amount of time that may be available for warning and response actions. They were developed in recognition of the importance of comprehensive emergency response planning and support systems for rapidly occurring events and the critical nature of such programs in areas nearest the release point. Generic EPZ concepts are summarized in Table L.l.

The EPZs are intended to guide the development of emergency response concepts. Subsequently, they will provide guidance for the development of site-specific and alternative-specific emergency response programs.

Table L.I. Summary of emergency planning concepts, with emphasis by zone

Emergency planning concept description	Emergency planning zone		
	Immediate response zone	Protective action zone	Precautionary zone
Character of zone	Prompt response critical; limited response time (<60 min); heaviest concentrations; public protective measures possible	Primary response evacuation; response time adequate (1-3 h); potentially lethal concentrations; responder protection required	Sheltering for non-lethal exposures; time abundant (3-10 h); agricultural response possible
Recommended implementation area	10 km	IO-35 km	>35 km
Emergency response management			
Coordination	+ + +	+ +	+
Command & control	+ + +	+ +	+
Communications	+ + +	+	
Accident assessment	+ + +	+ +	+
Protective action	+ +	+	
Decision-making	+ + +	+ +	+
Protective actions & responses			
Protective action options	+ + +	+ +	+
Public alert & notification	+ + +	+ +	+
Access & traffic control	+ +	+ +	
Special populations	+ + +	+ +	
Emergency worker protection	+ + +	+ +	
Resource & information management			
Emergency medical services	+	+ +	+ + +
Transportation	+ +	+ + +	
Community resources	+ + +	+ + +	+ +
Public information	+ + +	+ + +	+ + +
Evacuee support		+	+ + +

The +'s in the table are general indicators of the importance each emergency management function has in each zone. Triples (+ + +) indicate the function is critically important in that zone and is characterized by intense program emphasis. Doubles (+ +) indicate more general importance and will be characterized by significant program emphasis, Singles (+) indicate general importance and these functions will receive adequate attention but with limited program focus,

They should not, however, be applied mechanically or inflexibly to a particular site or alternative or to a specific accident scenario. The development of actual site-specific or alternative-specific EPZs should be done in the context of the unique political, social, and geographical characteristics of each site and alternative, as well as agent differences at the various sites. Conceptually, the criteria for establishing the EPZs are applied consistently across the program; however, specific configurations and associated distances may vary from site to site, or with each transportation alternative.

This guidance is programmatic in nature and will be formalized on a site-specific basis. Certain sites may merit substantially different EPZs, due to the nature of the chemical agents in their stockpile. For example, APG and Pueblo Depot Activity (PUDA) currently store only mustard agents; a significantly smaller protective action zone (PAZ) may be appropriate for such installations. The site-specific EPZs will be developed based on local factors, including agent and munition type and local transportation system constraints.

L.3.1 PRECAUTIONARY ZONE (PZ)

For an emergency response program to be effective, the principal concepts that are the basis for the program must be relatively simple. This allows for effective decision-making by emergency program managers without highly detailed local emergency response planning. For planning purposes the usual maximum no-death distance is approximately 35 km (see Sect. 4.2 and Appendix K) for worst-case accidents involving nerve agent releases. Worst-case accidents having lethal effects beyond 35 km provide sufficient warning and response times to preclude the need for detailed local emergency response planning.

The worst-case meteorological condition is a wind speed of 1 m/s (meter per second). The time required for such a worst-case release to reach a distance of 35 km from the stockpile location is nearly 10 h (hours). This time frame is sufficient to implement protective actions without prior comprehensive and detailed local planning efforts. Under conservative most likely (CML) meteorological conditions with a wind speed of 3 m/s, the time required for a chemical agent release to reach 35 km is slightly more than 3 h. However, under these conditions, substantially greater dispersion takes place. The maximum "no-deaths" distance for most accidents would be substantially less than 35 km under CML meteorological conditions. It is prudent to plan various precautionary actions outside 35 km for all disposal alternatives, based upon state or regionally coordinated wide-area information dissemination mechanisms,

Precautionary measures should be considered for areas beyond a 35-km distance from the storage/disposal sites. Given the likelihood of substantial warning and response times for areas beyond 35 km, precautionary measures can be planned and implemented at a state or regional level. An EPZ designated as the precautionary zone (PZ) is

established for this purpose. The PZ is the outermost EPZ and extends to a distance where no adverse impacts to humans would be experienced in the case of a maximum potential release under any conditions. This distance may vary substantially, based upon the circumstances of an accident occurrence, and would be determined on an accident-specific basis. In this EPZ the protective action considerations are limited to precautionary protective actions and actions to mitigate the potential for food-chain contamination as a result of an agent release.

The development of specific protective action for the PZ will be based on site-specific needs and analysis. Sheltering in the PZ is largely a precautionary protective action to reduce the potential for exposure to nonlethal concentrations of chemical agent. Evacuation could also be implemented as a precautionary protective action in this EPZ. The means for implementing the agricultural protection and other precautionary activities appropriate in this EPZ can be based principally on broad-area dissemination of emergency public information at the time of an accidental release of agent. Because of the substantial warning and response time available for implementation of response actions in the PZ, detailed local emergency response planning is not required.

L.3.2 PROTECTIVE ACTION ZONE (PAZ)

Within the PZ, the protective action zone (PAZ) defines an area where the primary emergency response is evacuation. Hence, the available emergency response times and the hazard distances associated with them are sufficiently large to allow most people to respond to an emergency effectively through evacuation. Operationally, a 35-km PAZ is an appropriate basis for emergency response planning. The comprehensive range of programs and support mechanisms described in the ERCP applies within the PAZ.

A comprehensive package of appropriate protective actions and emergency response mechanisms is considered in the PAZ. The principal emergency response, evacuation, must be considered carefully to ensure effective implementation and enhance protection of public health and safety. Evacuation is likely to be the most effective emergency response in the PAZ, as time is sufficient to permit orderly egress. However, evacuation, like other protective actions, requires warning. Because time remains limited in the PAZ, effective warning systems to both alert people to the potential for harm and notify them of the most appropriate actions are required. Available time for protective action varies with agent type, potential accident, and meteorological conditions at the time. These conditions will require careful consideration during site-specific emergency planning.

The capacity to implement emergency response in a timely manner is critical in the PAZ. Some areas, near the immediate response zone (IRZ) (discussed in the next section), will require highly detailed consideration of implementing resources in planning concepts; while

others, near the PZ will require less resource concentration. However, these degrees of needed emergency response capabilities depend on specific agent type, geographical configurations such as transportation routes, choke points such as bridges and available egress, and social characteristics such as the location and type of schools, hospitals, and other institutions.

L.3.3 IMMEDIATE RESPONSE ZONE

Within the PAZ, those areas nearest to the stockpile locations will be given special consideration, because of the potentially very limited warning and response times available within those areas. An IRZ is defined, within the PAZ, to allow for the development of emergency response concepts that are appropriate for immediate response in areas nearest to the site. The three emergency planning zones for a fixed site are graphically shown in Fig. L.2.

The IRZ is defined as an area within the PAZ where prompt and effective response is most critical. Because of the potentially limited warning and response time available in the event of an accidental release of chemical agent, the IRZ extends to a distance of approximately 10 km from the storage/disposal site. This area usually encompasses the no-deaths distances and has less than 1 h response time for most accidents under CML meteorological conditions. This area is most likely impacted by an accidental release of chemical agent. These impacts are within the shortest period of time and are characterized by the heaviest concentrations. Emergency response concepts in the IRZ are developed to provide the most appropriate and effective response possible.

The full range of available protective action options and response mechanisms is considered appropriate for the IRZ. The principal protective actions (sheltering and evacuation) will be considered carefully, along with supplemental protective action options that can significantly enhance the protection of public health and safety. Sheltering may be the most effective principal protective action for the IRZ, because of the potentially short period of time before impact by a released agent. In-place protection is particularly important in areas within the IRZ nearest to the release point. The time may not be available in the IRZ to complete an evacuation. The suitability of sheltering, however, is dependent upon a number of other factors, including the type(s) and concentration(s) of agent(s), expedient or pre-emergency measures taken to enhance the various capacities of buildings to inhibit agent infiltration, the availability of individual protective devices for the general public, the accuracy with which the particular area, time, and duration of impact can be projected, and the ability to communicate instructions to the public in a timely and effective fashion.

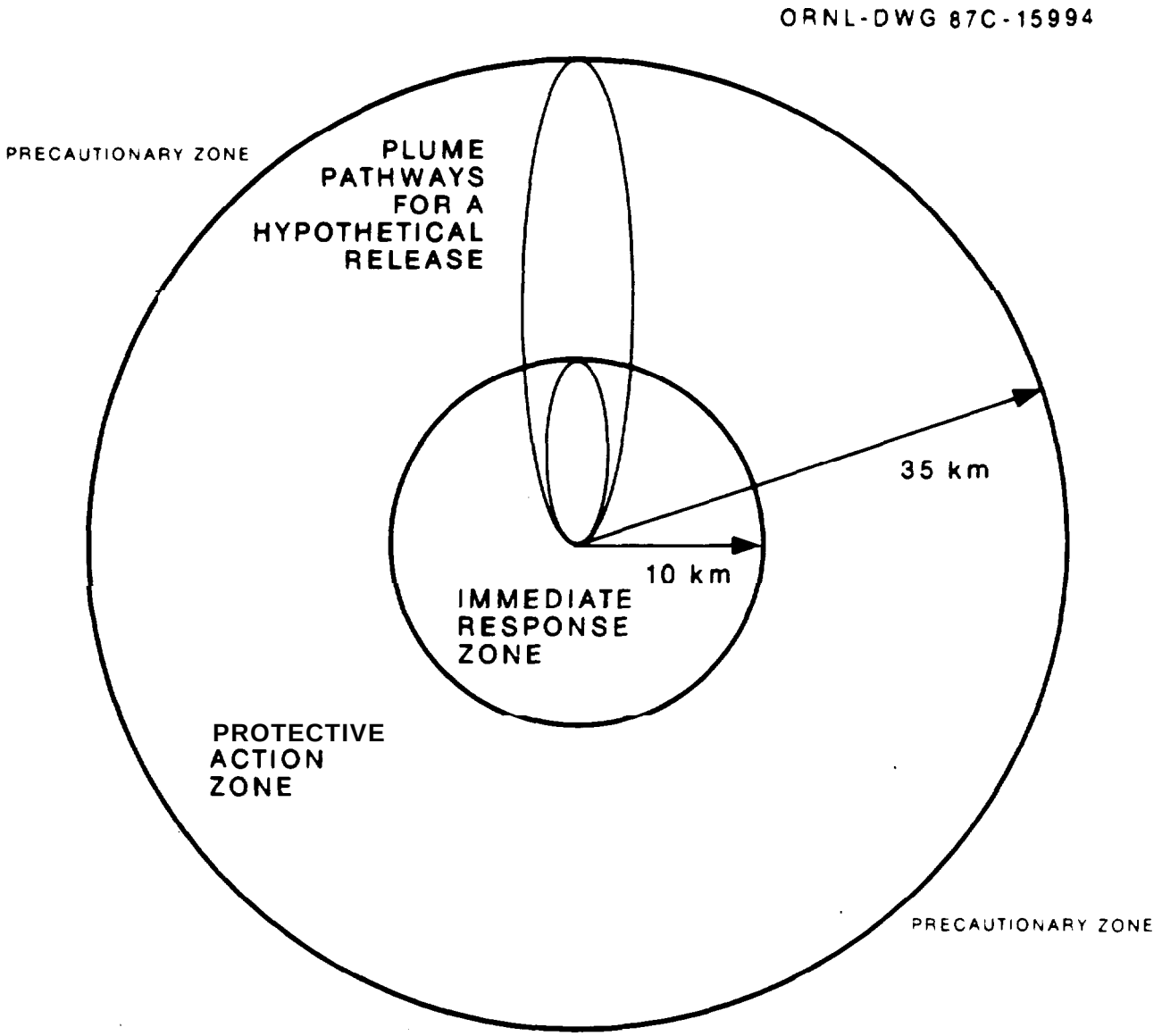


Fig. L.2. Emergency planning zone concepts for chemical agent releases.

The capability to rapidly implement the most appropriate protective action(s) is critical within the IRZ. A thorough analysis of the IRZ specific to each storage/stockpile location will be conducted and a precise methodology for determining the appropriate protective action(s) under various accident scenarios will be established, to ensure that a minimum of decision-making is required at the time of a chemical agent release. This analysis will likely identify certain areas within the IRZ which would implement sheltering under most accident scenarios, with evacuation only available as a precautionary measure. Subzone areas may be defined to accommodate the selective implementation of different protective actions within portions of the IRZ. Given a reasonably effective capability to project the area of impact and predict levels of impact at the time of a release, it may be appropriate to implement sheltering in areas close to the release point within the plume and evacuation in areas not immediately impacted. As an emergency progresses past the initial response phase, concern for IRZ occupants is reduced.

L.3.4 EMERGENCY PLANNING ZONE--RAIL TRANSPORTATION

The EPZ concepts for the disposal alternatives involving transportation of chemical agent by rail include a mobile emergency response capability which will be transported with the rail shipments and a regional response capability, coordinated principally at the state level, which will monitor chemical agent shipments and coordinate the implementation of secondary response activities.

The EPZ concept for rail transportation is described as a moving circular boundary emanating from the munitions train as it moves along the rail transportation route. For the mobile emergency response organization, this EPZ is significant as a fixed zone centered at the point where an accidental agent release takes place. For secondary response organizations, this EPZ is significant as a corridor formed by predetermined boundaries on either side of the rail lines designated. The size of the moving circular IRZ is established as a 10-km distance in all directions from the munitions train for primary response by the mobile emergency response organization. This distance corresponds to the IRZ described for fixed sites and is considered to be the maximum distance for which a mobile emergency response organization, originating from near the release point, can effectively implement primary response functions. The width of the EPZ corridor for secondary response purposes is 35 km on either side of the rail lines, encompassing a 35-km circular boundary emanating from the munitions train at the point where an accidental release occurs. Additional precautionary measures will be developed for implementation outside of the PAZ, as with the PZ described for fixed sites. A graphic description of the EPZ concepts for rail transportation is shown in Fig. L.3.

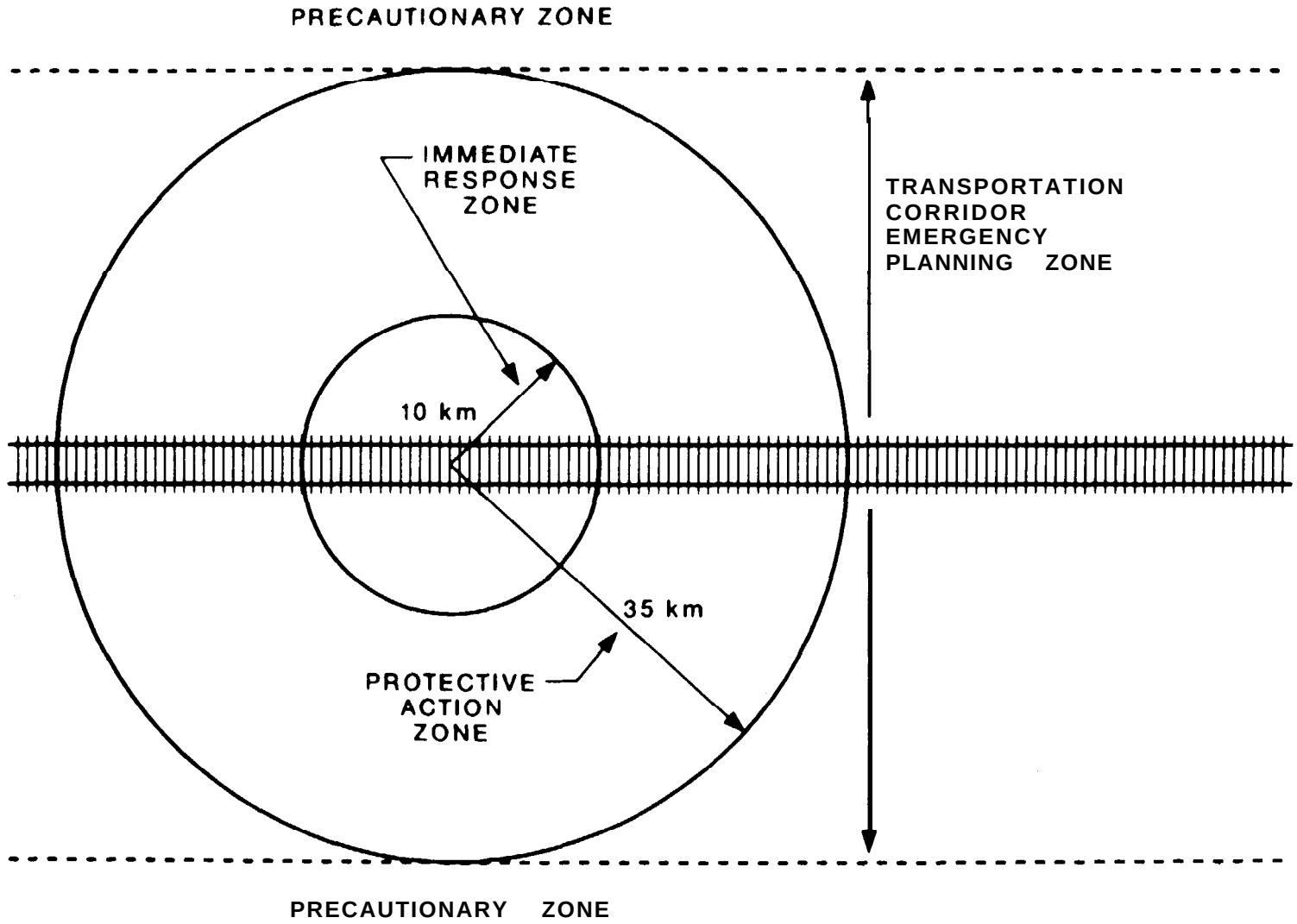


Fig. L.3. Emergency planning zone concepts for rail transportation of chemical agents.

L.3.5 EMERGENCY PLANNING ZONES--AIR TRANSPORTATION

The emergency response concepts for air transportation of chemical agent from LBAD and APG to TEAD involve an expansion of the fixed-site EPZs for those sites to accommodate the increased risk during takeoff and landing operations. The transportation plan describes accident potential zones (APZs) and clear zones for approach and departure that extend to 457.2 m (15,000 ft) on either end of all active runways. In order to develop emergency response programs for accidents occurring in these zones, the EPZs for each of the sites involved are extended in either direction on the assumption that the point source for an accident can be at any point in these zones. The resultant EPZs are elongated and extend for 10 km (IRZ) and 35 km (PAZ) from the storage and handling areas, as well as from any point within the extended accident potential zones on both ends of the runways. These same EPZ configurations are appropriate for any airfields designated as emergency landing sites. In the case of APG, the PAZ and IRZ may be considered as the same 10-km elongated EPZ, since only mustard agent is present at the site.

An air corridor will be designated for air transport operations, and general programs will be developed for broad-area information dissemination and support operations within these corridors. This area will be treated much like the PZ for fixed sites; however, since potential variations in flight paths due to weather conditions and mechanical malfunctions make it extremely difficult to predict all but general areas of potential impact. The emergency planning zone concepts for air transportation of chemical agents are graphically portrayed in Fig. L.4.

L.4 EMERGENCY RESPONSE CONCEPTS

L.4.1 EMERGENCY RESPONSE CONCEPTS FOR FIXED SITES

The emergency response concepts that apply to fixed sites have been analyzed on a programmatic basis. They are summarized here as a general description of the programs that will be put in place at each site to mitigate the effects of an accidental release of chemical agent. The application of these concepts may vary from site to site, in consideration of unique site characteristics and the makeup of the agent stockpile at each site. Emergency response concepts for fixed sites are described in four principal areas: emergency response management, protective actions and responses, emergency resource and information management, and program development and implementation.

L.4.1.1 Emergency Response Management

An emergency response program that clearly delineates authorities and responsibilities for various aspects of emergency response will be established at each stockpile storage site. The Army command for each storage site has authority and responsibility for initial response to accidents that occur on-site and for protection of on-sits personnel and mitigation of accident consequences. It is also responsible for

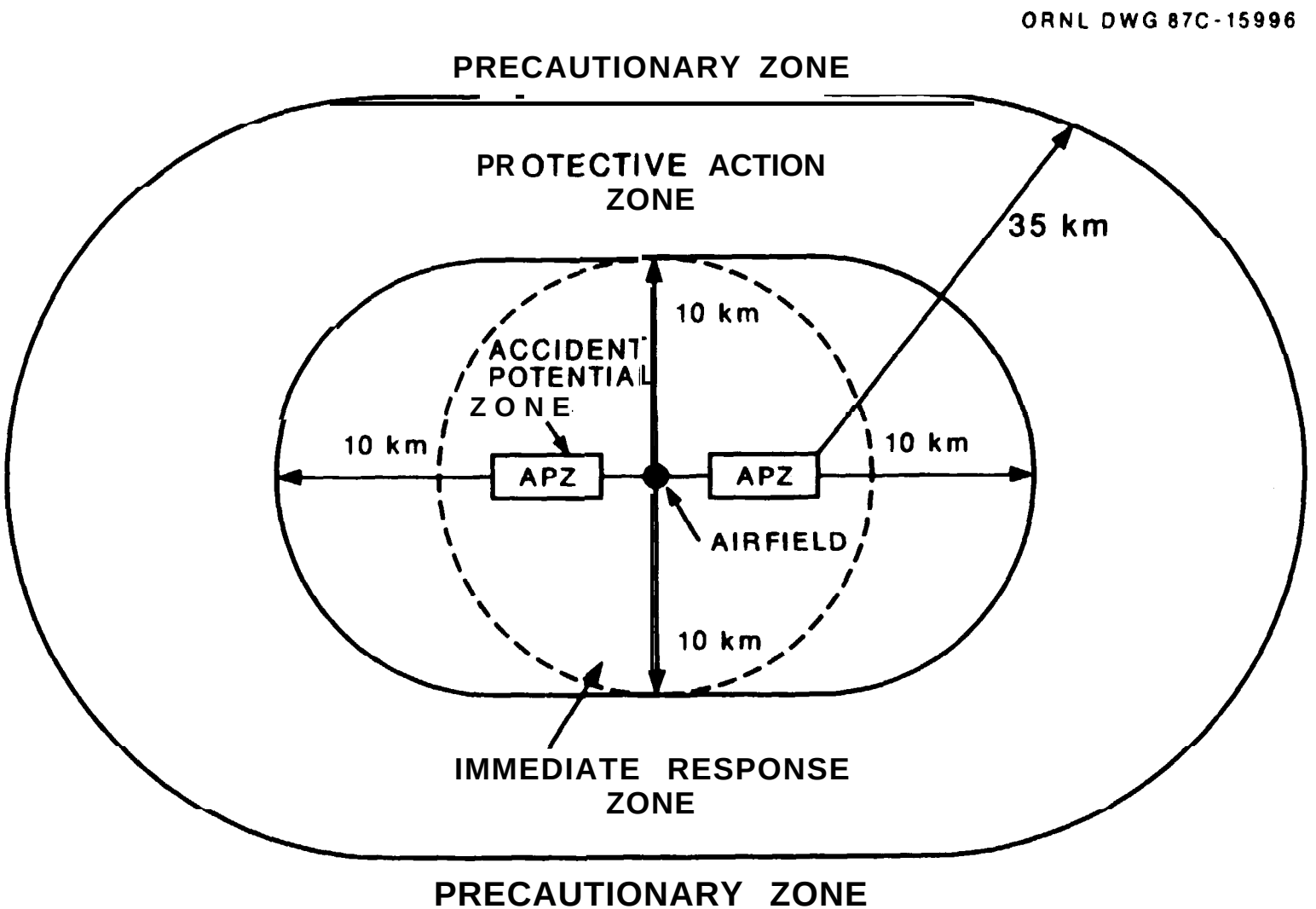


Fig. L.4. Emergency planning zone concepts for air transportation of chemical agents.

performing various actions to alert and inform community officials of the nature of an accident and recommend appropriate measures for protection of the civilian population. Community officials have the authority and responsibility to implement actions for public protection and to coordinate emergency resources and information during all phases of the emergency involving a threat to the civilian population.

The on-site organization of command and control of emergency response is described in the Chemical Accident/Incident Response and Assistance (CAIRA) plans for each of the stockpile storage sites. Although the emergency organizations are currently designated in these plans, some additional provisions will include 24-h coverage, enhanced capabilities to conduct accident assessments, and a command structure for making response decisions rapidly in the event of an accidental release of chemical agents, Off-site response organizations vary from site to site. These variations include different levels of government such as county and municipal, coordination with multiple jurisdictions, and a variety of combinations of volunteer and professional response organizations. The principal emergency management function is the integration of on-site, military incident response with off-site civilian emergency response. Local governments are typically able to respond most effectively to major emergencies, particularly rapidly occurring emergencies, until state and federal resources can become involved in the response.

The emergency response management structure for each storage site will establish the mechanisms and support arrangements for state and federal involvement in emergency response. State involvement will vary from site to site, based upon the established structures, authorities, and capabilities for state response. State and federal response will in most instances involve a few to several hours of mobilization time and will be effective in various support activities, such as medical assistance, evacuee care, and environmental monitoring. The initial response for public protection must be accomplished jointly by the local Army command organization at each stockpile site and the local government agencies in the vicinity of the site. Support and assistance arrangements will be established to ensure the effective utilization of state and federal resources as they are mobilized.

The management of emergency response must be supported by appropriate facilities, communications capabilities, and preestablished mechanisms for accident assessment and decision-making in order to be effective. Emergency operations centers (EOCs), both on-site and off-site, will be established, or upgraded, to provide adequate space, operational displays, communications, and other equipment to support effective emergency response. Emergency facilities currently exist on-site at each storage location and will be upgraded as necessary to support effective emergency response management.

Many of the county governments around the eight stockpile locations have an EOC, although substantial upgrade is necessary in many cases to ensure operational capability. Primary and backup communications systems

will be installed in the on-site and off-site EOCs to allow for reliable notification by the on-site command to all affected local governments of an accidental chemical agent release and to provide follow-up information during the emergency response. In addition, each local government EOC will have the- ability to communicate by radio with all operational response forces in the field in order to coordinate and manage the off-site response.

The on-site EOC will be equipped with computerized dispersion modeling systems, supported by local meteorological stations, that can predict the off-site areas affected and project the impact of a chemical agent release on the civilian population. An accident classification system will be established, specific to the particular stockpile, that will allow for rapid characterization of an accident occurrence and early notification to off-site authorities. Automated real-time agent monitors will be installed as the technology becomes available at each storage site to aid in the early detection of an accidental agent release. Procedures and mechanisms will be established jointly between the on-site and off-site emergency organizations to support timely decision-making on appropriate actions for public protection, including provisions for precautionary actions, automatic actions (for certain types of rapidly occurring events that require immediate public protection), and discretionary actions that involve specifying different protective actions for different locations based upon agent concentrations, meteorological conditions, and response times available.

Capabilities for rapidly detecting and characterizing an accident occurrence, making decisions on the most appropriate action(s) for public protection, and implementing those protective' actions are essential functions of emergency response management. The authority and responsibility for performing these functions are shared jointly by the on-site command and local government authority. While the storage site command is most knowledgeable of potential accident circumstances and impacts, local government authorities are responsible for protection of the civilian population. Close cooperative relationships will be established between the stockpile on-site commands and the surrounding local government authorities, including written agreement, so that appropriate actions for public protection can be implemented quickly and effectively. The emergency program managers, both on-site and off-site, must have in-depth training and regular interaction to maintain an ability to manage the emergency response to a chemical agent accident.

L.4.1.2 Protective Actions and Responses

The emergency response program at each stockpile storage site will establish the systems, resources, and response capabilities needed to implement appropriate actions for protection of the civilian population. Capabilities will be established for three principal methods of public protection: evacuation, sheltering, and individual protection. Evacuation will be implemented when sufficient time is available to move

people without exposing them to significant amounts (concentrations) of chemical agent. Sheltering will be implemented when sufficient time for evacuation is not available. Since sheltering alone does not provide adequate protection from lethal concentrations of chemical agent, additional measures will be taken to provide a high degree of protection for populations in close proximity to the stockpile sites, where evacuation may not be possible because of limited warning and response times. Measures will be taken to improve the sheltering protection of certain buildings that house sensitive populations, such as schools, hospitals, and nursing homes near each site. Protective equipment is recommended as another means of protection to populations in close proximity to the stockpile storage sites. Protective equipment includes civilian face masks and mouthpiece respirators, hooded jackets for young children, and protective infant carriers. Individual protective measures have the added benefit of being useful in combination with other protective actions, such as evacuation or sheltering.

A number of support systems and capabilities will be put in place to ensure that the means for implementing various protective actions are available. Most significant is the installation of a public alert and notification system that is capable of alerting civilian populations to an accident and notifying them of the actions they should take to protect themselves. This system will be established using a number of alerting devices in combination to provide the most effective capability for each site. Outdoor warning sirens and indoor warning devices, including tone-alert radios and automated telephone alerting systems, will be installed and supported by emergency broadcast systems (EBSs), cable television override, and other public information mechanisms to provide good overall coverage in both indoor and outdoor alert and notification. At greater distances, one or more of the alerting systems or mechanisms will be utilized to provide sufficient warning time for the public to take appropriate protective action(s).

Evacuation of the civilian population requires comprehensive planning and a number of support mechanisms. A detailed evacuation time estimate (ETE) study will be conducted for the area surrounding each site, establishing evacuation routes, pinpointing traffic control locations, and providing estimates of evacuation times under various scenarios and weather conditions. The ETE will provide an important basis for protective action decision-making at the time of an accidental release of chemical agent. Detailed traffic and access control plans will be developed to facilitate the implementation of evacuation. Emergency plans will be developed for all special facilities such as schools, hospitals, and nursing homes; and the transportation resources required to evacuate such facilities will be identified and agreements will be obtained for their use in an emergency. So that special assistance can be provided, a survey will be conducted to identify individuals within the general population with special needs, such as the handicapped, hearing and sight impaired, medically disabled, and those without a means of transportation.

Protective equipment will be provided to all civilian emergency workers who have a role in emergency response, to ensure that they are protected from exposure to chemical agent while performing their duties. This will include full protective clothing and respiratory protection, as well as auto injectors for use of agent antidotes should this be necessary. The full range of potential protective actions and responses will be planned for and programs will be implemented at each site to ensure that protective measures can be implemented quickly and effectively.

L.4.1.3 Emergency Resource and Information Management

A significant number of resources will be needed to implement certain protective actions and to provide care to populations affected by an accidental release of agent. The identification and management of these resources for emergencies and the provision of accurate and timely information to the civilian population are important components of the emergency response program for each site.

The principal resources needed to implement protective actions are transportation resources to aid in evacuation of affected populations. This includes buses to move school children and other institutionalized populations that are dependent on others for transportation. Ambulances will be needed to move certain sensitive populations, including some hospital patients, nursing home residents, and the medically disabled. Although many such transportation resources are available in the local areas affected, supplemental resources from outside of the area are likely to be required in the event of a major agent release. Adequate transportation resources from surrounding areas will be identified, agreements will be made, and procedures will be developed to mobilize and deploy these resources in an emergency.

Certain other resources are required to provide care for affected civilian populations during and after an accidental release of chemical agent. Chief among these are the medical resources needed to provide initial and follow-up medical care to exposed individuals. The existing local emergency medical services (EMS) providers will be utilized, but substantial additional resources may be required. Plans will be developed through the statewide and regional EMS organizations that are already in place in most locations to access the required medical resources from among those potentially available. Nontraditional resources will be identified, such as local health department personnel and social service agencies, to aid in the care of affected individuals. In addition, Army personnel who are trained in medical treatment of agent casualties will be identified to support civilian resources. All civilian emergency medical personnel and hospital staffs in the areas involved will be provided training in the treatment of agent casualties. All medical facilities in the vicinity of the stockpile locations will be provided with agent antidotes and other equipment and supplies that would be needed to treat large numbers of chemical agent casualties.

Other facilities, such as schools, nursing homes, and clinics, will be identified to be used as treatment facilities in the event that local hospitals do not have sufficient space to treat all agent casualties.

A myriad of other resources are required to support all aspects of emergency response in the affected communities. Traffic barricades, stretchers, bull horns, and other protective and response equipment -may be required. A detailed resource inventory will be developed and updated for each affected jurisdiction, and resource needs specific to chemical agent accidents will be identified and resolved. Coordination with state-level emergency management agencies will be accomplished during this process to ensure that all available support mechanisms are utilized to provide the resources needed for emergency response to chemical agent accidents.

Public information programs will be implemented to inform the potentially affected populations of the programs established for their protection and to educate them concerning the appropriate measures of self-protection in the event of a chemical agent accident. Printed brochures, public service announcements, and media releases will be utilized to educate the public in regard to the emergency response program. Additional programs will be established to provide public information at the time of an emergency. Pre-scripted emergency announcements for various accident scenarios and protective actions will be provided to the electronic media for use at the time of an emergency. A media center will be established in the vicinity of each stockpile site to provide emergency public information during and after chemical agent accidents. An annual media briefing will be conducted at this facility during the stockpile disposal program, with representation by the site command, the local officials, and the area media organizations to review emergency public information procedures.

L.4.1.4 Program Development and Implementation

The development of site-specific emergency response programs will be accomplished as a cooperative effort involving Army resources, local government officials, and appropriate state and federal agencies and organizations. A set of detailed planning standards and evaluation criteria will be established as a guide to ensure that a consistent level of preparedness is provided at all of the stockpile locations for each of the component areas of emergency response. Sufficient resource support will be provided to state and local emergency planning agencies to ensure that each jurisdiction in the vicinity of a storage site will be able to participate actively in program development and maintain an adequate level of preparedness during the chemical stockpile disposal program.

Program development will be initiated with conferences between the local Army site command and public officials, to describe, explain, and discuss the intended emergency response program. Comprehensive emergency plans will be developed for each local jurisdiction in the vicinity of

each stockpile site and for special. facilities such as schools, hospitals, and nursing homes. Detailed operating procedures will be developed jointly between the site command and local officials for critical activities such. as accident assessment, accident classification, accident notifications, emergency communications, protective action decision-making, public alert and notification, and all other functional areas of emergency response. Evacuation routing, emergency public information programs, and other support activities will be implemented specific to each stockpile site.

Emergency warning systems, communications, facility upgrades, monitoring and assessment capabilities, and other hardware and equipment required to implement the site-specific program will be identified, obtained, and/or installed during the program development process. Protective and other response equipment and supplies will be obtained as needed to support public and emergency-worker protection and response. All other activities associated with identification of resources required for emergency response and coordination of Army, community, county, state, and federal involvement will be accomplished for each site-specific program.

Program implementation activities will include training for all emergency program managers and emergency response personnel in chemical agent accident response. An emergency drill and exercise program will be implemented that involves regular interaction between the on-site emergency organization and the local emergency response organizations. A full scale exercise will be conducted annually during the chemical stockpile disposal program. Table-top exercises for program managers to drill and test systems will be conducted frequently to ensure that each component of the emergency response system is functioning properly and can be fully operational in the event of an accident.

Federal funding sources will be pursued to support personnel and the purchase of equipment for the protection of human health. These funds will provide support for emergency response planning, training, and exercises, as well as public introduction programs, program maintenance, and support studies. Additional funds may be required to provided installation of required warning and communications systems, accident assessment capabilities, emergency protective and response equipment, and facilities upgrading and outfitting.

The development and implementation of a fixed-site emergency response program will provide variable benefits, depending upon the nature of the release (quantity of agent, type of agent, mode of release), the prevailing meteorology, and the nature of the affected area. For scenarios which involve large, instantaneous releases of agent coupled with moderate or high wind speeds, the presence of an emergency response program will have less benefit for those areas closest to the release point. Large instantaneous releases with low wind speeds will allow more time in which to implement emergency actions and will permit a significant reduction in exposure for areas farther from the release point. The critical factor in determining the benefit of a preparedness

program is time. Those scenarios that result in rapid off-site releases of significant agent concentrations will be difficult to mitigate by any emergency response, particularly for areas closest to the release point. Release scenarios that provide for some warning and response time prior to significant off-site releases- can be mitigated to a great extent by emergency preparedness programs. In general, those areas in closest proximity to a fixed site are less likely to benefit from an emergency response program, while areas at greater distances from the site are likely to realize significant benefits from a response program in most release scenarios.

The qualitative benefits that are provided by the development and implementation of fixed-site emergency response programs, for various accident scenarios and meteorological conditions, are presented in Table L.2.

L.5 EMERGENCY RESPONSE CONCEPTS FOR TRANSPORTATION ALTERNATIVES

The emergency response concepts that apply to the chemical stockpile disposal program alternatives involving transportation of munitions have been analyzed for each of the modes of transportation being considered. They are summarized here as a general description of the programs that would be put in place for each alternative. Emergency response concepts are described for two modes of transportation: rail transportation and air transportation.

L.5.1 RAIL TRANSPORTATION

An emergency response program that travels with each rail shipment will be developed for the rail transportation of chemical munitions based on a mobile emergency response escort capability. This capability will be supplemented with state-level emergency response planning by states within the affected rail corridor. The mobile *escort* capability consists of Army personnel and resources for implementing the primary public protective measures that are conducted by community officials in the fixed-site emergency response programs. These activities include public alert and notification, traffic and access control, assistance to affected populations, and initial medical intervention and care. The escort emergency response organization will coordinate its activities with local officials and emergency response personnel at the site of an accident. They must be prepared to conduct all immediate response measures necessary, however, since no substantial detailed local planning or training for chemical agent accidents will be conducted for the rail transportation alternatives. The effectiveness of such a mobile capability is unlikely to approach the level of effectiveness afforded by a fixed-site emergency response program.

Table L.2. Qualitative benefits of fixed-site emergency response programs for nerve agent accidents

Accident scenario"	Meteorological conditions		
	Slow wind speeds (1 m/s)	Moderate wind spuds (3 m/s)	Fast wind spuds (6 m/s)
Moderate instantaneous release (100 kg VX) HO006 VRO04	Fatalities possible to 33 km; potential for multiple fatalities is high within IRZ, lower in PAZ. High reduction in fatalities at all distances	Fatalities possible to IO km; potential for multiple fatalities is high within 2 km, lower to IO km. Low reduction in fatalities to 2 km, higher at greater distances	Fatalities possible to 15 km; potential for multiple fatalities is high within 4 km, lower to 15 km. Low reduction in fatalities to 5 km, higher at greater distances
Small instantaneous release (10 kg VX) HF 003 PO 052	Fatalities possible to 7 km; potential for multiple fatalities is high within 2 km. lower to 7 km. High reduction in fatalities at all distances	Fatalities possible to 4 km; potential fot multiple fatalities is moderate within I km, low to 4 km. Low reduction in fatalities at 2 km	Fatalities possible to 6 km; potential for multiple fatalities is moderate within 2 km, low to 6 km. Low reduction in fatalities to 5 km
Large semicontinuous release (1000 kg VX) SL 004 SL 005 AT 003	Fatalities possible to 100 km; potential for multiple fatalities is very high within IRZ, lower in PAZ and PZ. High reduction in fatalities at all distances	Fatalities possible to 25 km; potential for multiple fatalities is high within IRZ, lower in PAZ. Low reduction in fatalities to 2 km, higher at greater distances	Fatalities possible to 15 km; potential for multiple fatalities is high within IRZ, lower in PAZ. Low reduction in fatalities to 5 km, high at greater distances
Moderate semicontinuous release (100 kg VX) SL 021 PO 033 PO 029 PO 026	Fatalities possible to 45 km; potential for multiple fatalities is very high within IRZ, lower in PAZ, and very low in PZ. High reduction in fatalities at all distances	Fatalities possible to 8 km; potential for multiple fatalities is moderate within 2 km, low to 8 km. Moderate reduction in fatalities at 2 km, greater to 8 km	Fatalities possible to 7 km; potential for multiple fatalities is high within 2 km, lower to 7 km. Low reduction in fatalities at 2 km, moderate to 5 km
Large spill (900 kg GB) VR 006 HF 007 SR 004 PO 009 PO 012	Fatalities possible to I2 km; potential for multiple fatalities is moderate within 3 km, low to 12 km. High reduction in fatalities at all distances	Fatalities possible to 8 km; potential for multiple fatalities is moderate within 2 km, low to IO km. High at all distances	Fatalities possible to 8 km; potential for multiple fatalities is moderate within 2 km, low to 8 km. Moderate reduction in fatalities to 2 km, greater to 8 km

Table L.2 (continued)

Accident scenario'	Meteorological conditions		
	Slow wind speeds (1 m/s)	Moderate wind speeds (3 m/s)	Fast wind speeds (6 m/s)
Moderate instantaneous release (900 kg HD) VR 006	Fatalities possible to 7 km; potential for multiple fatalities is high within 2 km, lower to 7 km. High reduction in fatalities at all distances	Fatalities possible to 2 km; potential for multiple fatalities is moderate within 5 km, low to 2 km. Low reduction in fatalities to 2 km	Fatalities possible to I km; potential for multiple fatalities is low. Potential for precautionary measures is low
Small instantaneous release PO 052 HO 007 HO 01 1	Fatalities possible to 1 km; potential for multiple fatalities is low. High potential for taking precautionary measures	Fatalities possible to 0.2 km; potential for multiple fatalities is very low. Low potential for precautionary measures	Fatalities possible to 0. 15 km; potential for multiple fatalities is very low. Precautionary measures unlikely
Large semicontinuous release (4000 kg HD) SL 004	Fatalities possible to 100 km; potential for multiple fatalities is very high within IRZ, lower in PAZ and PZ. High reduction in fatalities at all distances	Fatalities possible to IO km; potential for multiple fatalities is high within 2 km, lower to IO km. Low reduction in fatalities to 2 km, high at greater distances	Fatalities possible to 7 km; potential for multiple fatalities is high within 2 km, lower to 10 km. Low reduction in fatalities to 5 km, high at greater distances
Moderate semicontinuous release (100 kg HD) PO 001 PO 033	Fatalities possible to 8 km; potential for multiple fatalities is very high within 2 km, lower to 8 km. High reduction in fatalities at all distances	Fatalities possible to 2 km; potential for multiple fatalities is moderate within 5 km, low to 2 km. Moderate reduction in fatalities at 2 km	Fatalities possible to 1.5 km; potential for multiple fatalities is high within 5 km, lower to 1.5 km. Low reduction in fatalities at 1.5 km
Large spill (900 kg HD) vo 003 HA 023	Potential for fatalities is very low. High likelihood of successful precautionary measures	Potential for fatalities is very low. High likelihood of successful precautionary measures	Potential for fatalities is very low. High likelihood of successful precautionary measures

'Accident scenarios are described in Appendix J.

The escort organization will include sufficient personnel to manage the emergency response, coordinate responses with civilian emergency response personnel, and conduct public warning and other immediate public protective actions in the vicinity of the accident. It will be equipped with computerized dispersion modeling and accident assessment capabilities, radio communications, public alerting devices, appropriate transportation capabilities (e.g., all-terrain vehicles), and sufficient protective equipment and medical supplies for escort and local civilian emergency response personnel.

The state-level emergency response planning will focus on providing expedient resource support to the site of a rail transportation accident from state or nearby local government resources. However, such support is likely to require up to a few hours to mobilize and deploy to an accident location. State-coordinated support will be effective for secondary activities (i.e., medical and evacuee care, security, etc.) but is not likely to be effective for the principal response activities needed immediately following an accident.

The limitations associated with deploying all or most of the emergency response resources from a mobile escort capability restrict the potential effectiveness of such a response. Under certain accident scenarios and conditions this capability can be effective in reducing civilian casualties. However, for accident conditions that do not involve easy access to affected populations, **the** mobile emergency response capability cannot be effective in preventing civilian casualties.

L.5.2 AIR TRANSPORTATION

The emergency response programs for the disposal alternatives involving air transportation are based principally on an expansion of the fixed-site programs for the sites involved. The emergency planning zones for these sites are expanded due to the accident potential zones which extend to 457.2 m (15,000 ft) at each end of the runway. In addition, limited fixed-site emergency response programs will be established for any emergency landing sites designated for the air transportation alternative.

L.6 SUMMARY

The emergency response concepts that have been presented for the on-site and transportation alternatives are based on established and accepted principles of emergency management. Sound models for these programs exist in the Radiological Emergency Preparedness Programs currently implemented for fixed nuclear facilities by the Federal Emergency Management Agency and the Nuclear Regulatory Commission, in the Chemical Emergency Preparedness Program of the U.S. Environmental Protection Agency, and in the Hazardous Materials Emergency Planning Guidance of the National Response Team. In the event of an

accident/incident with off-site consequences exceeding the capabilities of state and local authorities, the DOD will assume responsibility for coordinating emergency response activities under the provisions of the National Contigency Plan as prescribed by CERCLA.

The concepts developed for stockpile disposal vary between alternatives in that each poses a unique set of problems and circumstances that must be considered in fashioning appropriate response programs. It is clear that planning for accidents that occur at fixed, defined sites can be accomplished in greater detail and with much greater assurance of an effective response capability than planning for accidents that occur during transportation. But even the fixed-site programs do not provide complete assurance that loss of life can be prevented.

The emergency response concepts presented in the ERCP are intended as a general description of how such programs will be implemented, to support the Chemical Stockpile Disposal Program. An important process of cooperative interaction, involving local, state, and federal agencies and organizations, must be accomplished for these emergency response concepts to be effectively implemented. For fixed-site emergency response planning, the relationship between the U.S. Army command at each stockpile site and the surrounding community organizations is central to development of programs for the protection of the surrounding populations. Further guidance regarding site-specific emergency planning will be developed as part of the site-specific NEPA documentation. For transportation emergency response planning, a broad range of local, state, and federal agencies must be actively involved in planning for accidents that could occur across a potentially expansive area affecting literally thousands of jurisdictions, agencies, and populations.

Detailed guidance for the development of site-specific emergency response plans is currently under development. Detailed plans will be prepared during site-specific NEPA documentation associated with the implementation of specific selected alternatives.

REFERENCES FOR APPENDIX L

Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987. *Emergency Response Concept Plan for the Chemical Stockpile Disposal Program.*

ACRONYMS AND INITIALISMS FOR APPENDIX L

APG	Aberdeen Proving Ground
APZ	accident potential zone
CAIRA	Chemical Accident/Incident Response and Assistance
CML	conservative most likely
DOD	Department of Defense
EBS	emergency broadcast system
EMS	emergency medical system
EOC	emergency operations center
EPZ	emergency planning zone
ERCP	Emergency Response Concept Plan
ETE	evacuation time estimate
IRZ	immediate response zone
LBAD	Lexington-Biue Grass Army Depot
PAZ	protective action zone
PZ	precautionary zone
PUDA	Pueblo Depot Activity
TEAD	Tooele Army Depot

Appendix M

APPROACH FOR SELECTING THE ENVIRONMENTALLY PREFERRED ALTERNATIVE FOR THE CHEMICAL STOCKPILE DISPOSAL PROGRAM FINAL PROGRAMMATIC ENVIRONMENTAL IMPACT STATEMENT

M.1. INTRODUCTION

In compliance with the National Environmental Policy Act (NEPA), the Army is to identify the environmentally preferred alternative(s): if such exists, for disposing of the nation's chemical munitions stockpile in the final programmatic environmental impact statement (FPEIS). The Office of the Program Executive Officer-Program Manager for Chemical Demilitarization (PEO-PM Cml Demil), the Oak Ridge National Laboratory (ORNL), have developed a systematic approach for selecting the environmentally preferred alternative(s) for the FPEIS.

lf.2. OBJECTIVE

The objective of this appendix is to document the approach and methodology used for selecting the environmentally preferred alternative(s). The environmentally preferred alternative resulting from the application of this methodology will be the Army's preferred alternative.

This approach allows the Army as well as other interested and affected groups to: (1) compare the public health and environmental impacts of the various alternatives, for both normal operations and "unplanned" releases of chemical agent (i.e., accidents), for each site; (2) identify the public health and environmental trade-offs associated with each disposal alternative at each site; and (3) reach a decision. As the trade-offs are identified, the approach allows the interested groups to note points of disagreement in the decision process. Therefore, although the interested groups may not agree on the preferred alternative(s), the rationale for selecting the alternative is apparent, points of disagreement are obvious, and the preferences and rationale which groups use to make trade-offs can be clearly defined.

M.3. BACKGROUND

Subsequent to the issuance of the draft programmatic EIS (DPEIS) for the CSDP on 1 July 1986, PEO-PM Cml Demil and ORNL determined that a systematic and consistent approach was needed to identify the environmentally preferred alternative. PEO-PM Cml Demil tasked ORNL to prepare a draft approach and methodology. This was done, and PEO-PM Cml Demil and ORNL agreed that the final approach and methodology should be developed following a number of specified procedures to ensure that the selection methodology would meet the objectives stated above. These included:

1. Review of the draft methodology by various parties, including other ORNL and PEO-PM Cml Demil staff and management, the MITRE Corporation, community study groups [through briefings and, in the case of the Lexington-Blue Grass Army Depot (LBAD) group (whose contract was in place sufficiently early to so permit), review of a draft document), and uninvolved third-party experts. The third-party experts were Professors Howard Kunreuther and Paul Kleindorfer, Department of Decision Sciences, University of Pennsylvania.

2. Modification of methodology, as necessary, based on the comments received from the reviewers.

3. Application of modified approach by PEO-PM Cml Demil and ORNL to select the environmentally preferred alternative(s).

4. Meeting of PEO-PM Cml Demil and ORNL to review the application of the methodology.

5. Selection of environmentally preferred alternative(s) by PEO-PM Cml Demil.

6. Review and approval of the selected environmentally preferred alternative(s) by Army policy-makers.

H.4. LOGIC OF THE SELECTION METHODOLOGY

The methodology is comprised of a number of elements and stages. These include:

1. identification of mitigated alternatives considered;

2. definition, characterization, and ranking of categories of significant impacts;

3. characterization of alternative-specific accident scenarios;

4. establishment of sites (corridors)/alternatives impact pictograms;

5. evaluation and screening of alternatives on the basis of ranked categories of significant impacts;

6. evaluation of risk reduction potential associated with emergency planning and preparedness;

7. preliminary selection of the environmentally preferred alternative(s); and

8. examination of the selected environmentally preferred alternative with respect to each site inventory to ensure that the methodology has not identified an alternative that is obviously incorrect for the installation environments and off-site transportation environments, where relevant.

After describing the basic logic of the methodology, this appendix discusses each of these elements in turn. The final section of the appendix addresses the decision rule employed to determine whether an apparent difference in impacts and risks between and among alternatives is truly significant.

As depicted in Fig. M.l, the methodology is based on the sequential consideration of human health effects and ecosystem and environmental effects of each alternative for both normal operations and accident scenarios (in the case of accident scenarios, the methodology considers multiple probabilistic and consequence measures, as described below).

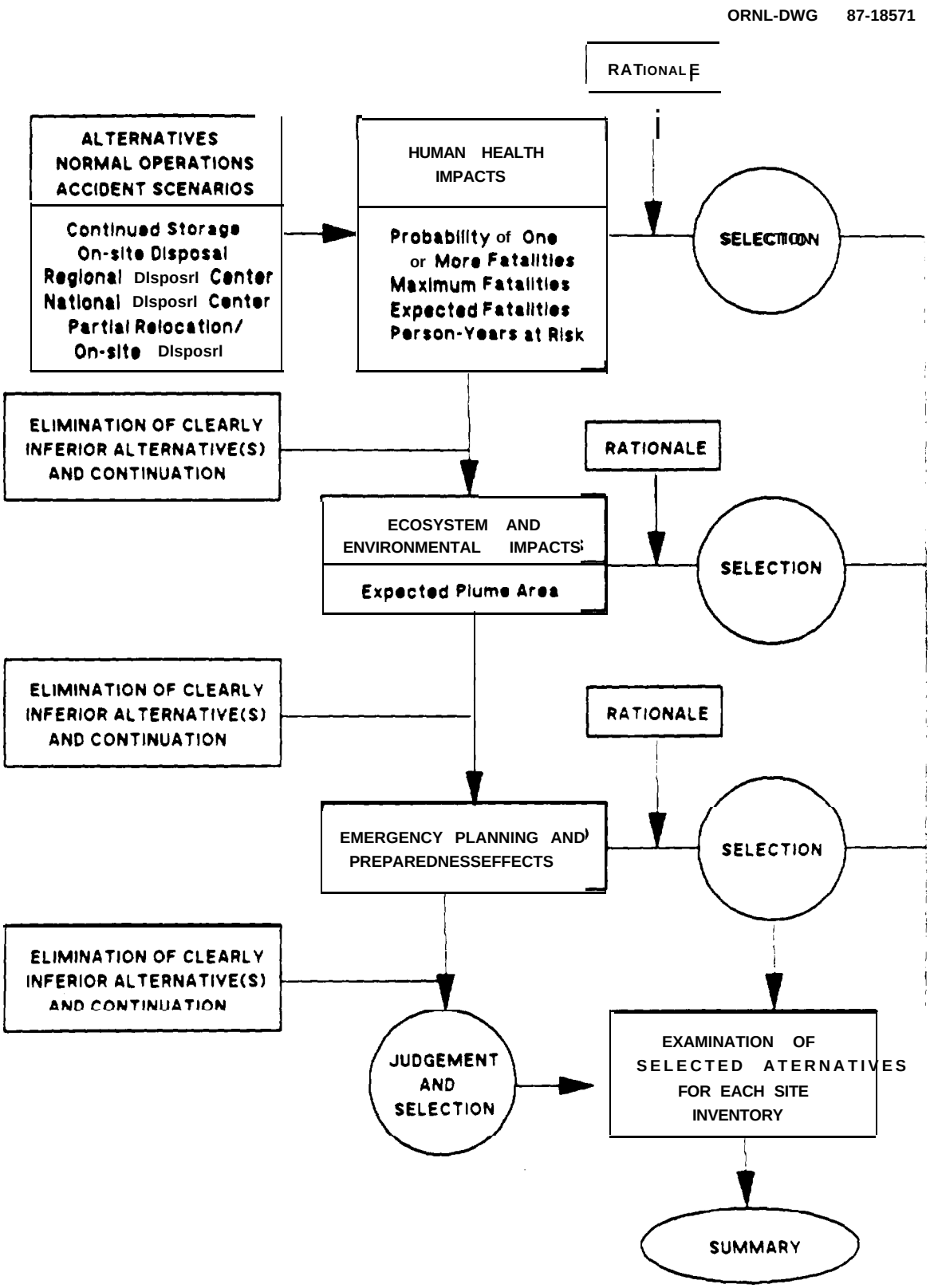


Fig. M.1. Selection methodology.

The selection is then made on the basis of whether an alternative is significantly better than the others. If an alternative is significantly better than others on the basis of human health impacts, that alternative is selected, and the other alternatives are eliminated from further consideration. Similarly, among alternatives judged equivalent on the basis of human health impacts, if an alternative is significantly better on the basis of ecosystem and environmental impacts, it is selected at that stage of the comparison, and the other alternatives are eliminated from consideration.

If these comparisons do not identify a single programmatic environmentally preferred alternative, those alternatives judged equivalent on the basis of impacts on human health and ecosystem and environmental resources are compared on the basis of the feasibility and potential effectiveness of emergency planning and preparedness, the only mitigative measure for which reasonable and defensible quantitative estimates of risk reduction cannot be made. If no clear choice can be made even after considering the value of emergency planning and preparedness, a preliminary judgment is made declaring that more than one alternative is acceptable or identifying the selected environmentally preferred alternative from among those alternatives that have not been eliminated on the basis of human health impacts, ecosystem and environmental impacts, and risk reductions associated with emergency planning and preparedness.

At whatever stage of the analysis a programmatic environmentally preferred alternative is identified (i.e., after consideration of human health effects, ecosystem and environmental effects, or the value of emergency planning and preparedness), that alternative is examined with respect to each installation to ensure that the methodology has not identified an alternative that is obviously incorrect for the installations' inventory. That examination identifies the alternative that would be preferred from the perspective of those persons near the installation. For those situations where the local perspective would indicate a preference for movement of the inventory, the examination also considers the impacts to people and resources along the transportation corridor and at the destination site; it is only by considering impacts to all individuals and resources potentially affected by the alternative (i.e., at the origin site, transportation corridor for that inventory, and destination site) that a programmatic preference regarding the disposal of a particular installation's inventory can be identified. If this examination (by site-specific inventory) reveals that the selection methodology has identified an alternative that is obviously wrong in terms of the net impacts to all potentially affected people and resources, the Army would identify another alternative that corrects that problem without simultaneously adding a significant adverse impact to other affected resources (i.e., transport corridor or destination environments and resources).

M.4.1 ALTERNATIVES CONSIDERED

The FPEIS examines the following alternatives for both normal operations and accident scenarios (parantheticals refer to shorthand designation as appear in the FPEIS):

1. continued storage (Al).

2. on-site disposal (A2).
3. regional disposal centers (A3).
4. national disposal center (A4).
5. partial relocation and on-site disposal (A5) [air movement of Aberdeen Proving Ground (APG) and LBAD inventories to Tooele Army Depot (TEAD) for disposal with remainder of stockpile disposed on-site].

The delayed disposal alternatives identified in the CSDCP Supplement [modified baseline program (A6a), JACADS operational testing program (A6b), and dual technology evaluation program (A6c)] are not specifically analyzed because each could be implemented with any of the basic alternatives (A1-A5), because they involve additional years of storage (which is simply a time-based subset of the continued storage alternative), and because, in the case of selection of an alternate cryofracture technology for the A6c alternative, it would involve the preparation of supplemental NEPA documentation.

Each alternative (A1-A5) is as described in Sect. 2.3 of the FPEIS [i.e., as assessed in the hazard and risk analysis (see Appendix J)]. The quantitative effects of mitigation measures taken for an alternative or class of alternatives [as described (with associated risk reductions) in Sect. 4.5 of the FPEIS] are factored into the final risk profiles for the programmatic alternatives (as shown in Sect. 2.5 of the FPEIS); the risk reductions afforded by emergency planning and preparedness are qualitatively considered at a later stage of the analysis (see above). Thus, the comparison of alternatives and selection of the environmentally preferred alternative in Sect. 2.6 is made on the basis of fully mitigated alternatives. That is conceptualized structurally as follows:

Impacts = impacts of the unmitigated alternatives minus the reductions in impacts due to mitigation.

M.4.2 DEFINITION, CHARACTERIZATION, AND RANKING OF CATEGORIES OF SIGNIFICANT IMPACTS

The decision to consider impacts on human health and ecosystem and environmental resources sequentially is a reflection of the decision made that impacts to human health are considered to be the most important, overriding impact to consider, followed by ecosystem and environmental impacts. Because the Army's primary objective is to dispose of the stockpile without causing any fatalities, regardless of the alternative selected, its plant design, on-site handling activities and procedures, and on-site and off-site transport activities (including the use of on-site and off-site transport packages) have been oriented toward this goal (see Sects. 2.3 and 4.5 of the FPEIS). The ranking also corresponds to the Army's understanding of Congress's concern in its mandate [i.e., P.L. 99-145 (1985)] and to public concerns.

The programmatic environmentally preferred alternative is to be determined on the basis of consideration of the health and environmental impacts of normal operations and relevant accident scenarios of each fully mitigated alternative. There is wide concurrence that health impacts have the highest ranking or priority, followed by ecosystem and environmental impacts (i.e., socioeconomic, ecological. air quality and water resource impacts).

Accident impacts of programmatic alternatives will be identified and assessed quantitatively. Risk can be quantified and, therefore, can be used to compare the risks associated with each alternative. Risk analysis has been widely used in the nuclear and chemical industries to evaluate the risks associated with those industries and to communicate these results to the public and decision makers. Risk analysis is particularly appropriate in the context of this program (i.e., the CSDP), where it is vitally important to account for risks borne by different individuals (i.e., those near the existing sites for all alternatives plus those near transport corridors and destination sites for alternatives requiring off-site transport), depending on the alternative selected. For each storage/disposal site (in Sect. 4.3 of the FPEIS) and transport corridor (in Sect. 4.4 of the FPEIS) and for the program as a whole (Sects. 2.5 and 2.6 of the FPEIS), accidents will be identified by alternative, where each alternative and associated accident sequences will be characterized according to the activities associated with implementing each alternative. The accidents derive from the hazard and risk analysis developed for the CSDP (GA Technologies, Inc. 1987a,b,c, as integrated in Appendix J of the FPEIS, or in MITRE 1987b). Accidents will be characterized by narrative descriptor, source size, mode of release, downwind hazard distance, duration at risk and possible location(s). Because of national security concerns, the probabilities of these accidents will appear only in a SECRET version of the integrated risk analysis prepared by the MITRE Corporation (MITRE 1987a) (discrete probability values can be used to "back calculate" the size of the stockpile, which is classified).

Impacts to human health and the environment will be portrayed according to the two categories of impact significance. According to the priority previously acknowledged, selection of the environmentally preferred alternative will sequentially consider human health impacts and ecosystem and environmental impacts.

Human health impacts will be addressed by storage/disposal site and transportation corridor by alternative in Sects. 4.3 and 4.4, respectively, of the FPEIS and summarized programmatically in Sect. 2 of the FPEIS. Multiple risk measures (i.e., probability of one or more fatalities, maximum fatalities, expected fatalities, and person-years of risk) will be presented to allow for different sensitivities in risk perception of decision makers and publics (e.g., one person may be more sensitive to the probability of a single fatality, while another may be more sensitive to maximum fatalities). The full definition of these measures (including calculation procedures) will be identified in the FPEIS as well as in Appendix J of the FPEIS. Two of these risk measures (i.e., probability of one or more fatalities and expected fatalities) are probabilistic in nature, while the other two are deterministic. For the probabilistic measures, accident impacts will take into account both the probabilities of accidental agent releases and the consequences of those releases by appropriately multiplying those terms (impacts = probability x consequence). The assessment will consider the full set of accident sequences peculiar to an alternative under conservative, most-likely meteorological conditions, as defined in the FPEIS.

Ecosystem and environmental impacts (i.e., impacts to socioeconomic resources, ecological resources, and water resources) will be discussed by storage/disposal site and transportation corridor by alternative in

Sects. 4.3 and 4.4, respectively, of the FPEIS and programmatically summarized by alternative in Sect. 2 of the FPEIS. The site- and corridor-specific assessment will consider ecosystem and environmental resources potentially affected by the alternative-specific accident characterized by the maximum downwind hazard distance. The summary assessment of ecosystem and environmental impacts (to be identified for sites and transport corridors as well as for the program as a whole) will be based on the expected plume area for all appropriate accidents for each alternative for each site and each transport corridor. This probabilistic measure will also be defined fully in the FPEIS and in Appendix J of the FPEIS.

M.4.3 CHARACTERIZATION OF ALTERNATIVE-SPECIFIC ACCIDENT SCENARIOS

As noted previously, each alternative has associated with it a set of probabilistic accidents. The set is comprised of external events over which the Army has little or no control (e.g., airplane crashes, lightning, tornadoes, meteorites) and accidents associated with particular activities (i.e., storage, handling, on-site transport, *off-site* transport, plant operations). All these accidents have been identified by GA Technologies and its subcontractors (GA Technologies, Inc. 1987a,b,c) and integrated by MITRE Corporation (see Appendix J and MITRE 1987a,b). Each accident is characterized by its discrete probability or expected frequency (classified), source size, mode of release, possible location, and duration of time that accident could occur (i.e., the total time during which agent could be released, from *onset* of disposal program to completion of that particular activity).

Using the D2PC downwind hazard distance dispersion code, each accident is also characterized by MITRE Corporation (with input from ORNL on meteorological conditions and population distribution) in terms of consequences (i.e., plume geometries and fatalities).

The concept equations shown in Table M.l provide the conceptual basis for calculating the impacts of each programmatic alternative. These equations have been used by MITRE Corporation to develop a computer program that automatically inputs the results of:

1. the CSDP risk analysis [i.e., site-, munition-, and agent-specific unit frequencies of external events or accidents resulting in agent release; the modes of agent release (spill/evaporation, fire, or detonation); and source terms or strengths) (GA Technologies 1987a,b,c);

2. atmospheric dispersion analysis of the release terms identified in (1) above (using the D2PC atmospheric dispersion code); and

3. population distribution analysis (using the 1980 Census of Population).

The computer program can be used and has been used to sum for all sites and transport corridors the total programmatic impacts for a disposal alternative within any single impact/risk measure (i.e., probability of one or more fatalities, maximum fatalities, expected fatalities, person-years at risk, and expected plume area). Where only qualitative data exist (i.e., for normal operations), they can be used

Table M.l. Concept equations for selection methodology

Continued storage alternative (assumed at 25 years)

Impacts and risks = Sum (at 8 sites) for activities and accidents associated with

1. handling during long-term storage, and
2. long-term storage.

On-site disposal alternative

Impacts and risks = Sum (at 8 sites) for activities and accidents associated with

1. on-site handling away from the disposal facility storage,
2. on-site transportation,
3. handling at the disposal facility, and
4. plant operations.

Regional disposal alternative

Impacts and risks = Sum for activities and accidents associated with

Origin sires (8 sires)

1. handling associated with the rail mode,
2. temporary storage associated with transportation by rail, and
3. on-site transportation associated with transportation by rail,

plus

Transportation corridors (6 corridors)

1. rail transportation

plus

Destination sires (2 sites-Annisron Army Depot and Tooele Army Depot)

1. handling associated with the rail mode,
2. temporary storage associated with transportation by rail,
3. on-site transportation associated with transportation by rail,
4. handling at the disposal facility, and
5. plant operations.

National disposal alternative

Impacts and risks = Sum for activities and accidents associated with

Origin sites (8 sires)

1. handling associated with the rail mode,
2. temporary storage associated with transportation by rail, and

Table M.1 (continued)

3. on-site transportation associated with transportation by rail,

plus

Transportation corridors (7 corridors)

1. rail transportation

plus

Destination site (1 site-Tooele Army Depot)

1. handling associated with the rail mode,
2. temporary storage associated with transportation by rail,
3. on-site transportation associated with transportation by rail,
4. handling at the disposal facility, and
5. plant operations.

Partial relocation and on-site disposal alternative

Impacts and risks = Sum for activities and accidents associated with

Origin sites (5 sires)

1. on-site handling away from the disposal facility storage,
2. on-site transportation,
3. handling at the disposal facility, and
4. plant operations

plus

Origin sires (2 sites-Aberdeen Proving Ground and Lexington-Blue Grass Army Depot)

I. handling associated with the air mode,
2. temporary storage associated with transportation by air,
3. on-site transportation associated with transportation by air, and
4. air transportation (take-offs)

plus

Transportation corridors (2 corridors)

1. air transportation (in flight)

plus

Destination sire (I ***sire-Tooele Army Depot)***

1. air transportation (landing),
2. handling associated with the air mode,
3. on-site transportation associated with the air mode,

Table M.1 (continued)

	4. temporary storage associated with transportation by air, 5. on-site transportation associated with on-site disposal, 6. handling at the disposal facility, and 7. plant operations.

systematically to compare relative differences in the alternatives. By dropping the summation part of the process, the equations can be used to estimate the site-specific risks/impacts of any alternative (terms in the equations specific to other sites are dropped).

H.4.4 ESTABLISHMENT OF SITES/ALTERNATIVES PICTOGRAMS

Site-specific impacts resulting from the set of alternative-specific accidents will be shown in Sects. 4.3 (Storage/Disposal Sites) and 4.4 (Transport Corridors) of the FPEIS. The summary of programmatic impacts by alternative will be shown in Sect. 2.5 of the FPEIS, with the "contribution" from each site inventory also shown. The alternatives will be compared in Sect. 2.6 of the FPEIS on the basis of impacts of normal operations (in qualitative terms) and accident scenarios (quantitative presentation).

M.4.5 QUALITATIVE RISK REDUCTIONS DUE TO EMERGENCY PLANNING AND PREPAREDNESS

Although there is no reasonable and defensible manner by which risk reductions associated with emergency planning and preparedness can be precisely quantified, it is certain that the feasibility and potential effectiveness of emergency planning and preparedness has a variable impact on the different alternatives. This variation in feasibility and effectiveness will be assessed qualitatively in light of the elements of the generic emergency response concept plan (Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987) as the Army plans to implement it on a site- and, as applicable, corridor-specific basis. The assessment will look particularly to whether or not risk reductions or benefits achieved through emergency planning and preparedness for fixed sites are likely to be comparable to reductions or benefits for transportation corridors.

M.4.6 PRELIMINARY SELECTION OF THE ENVIRONMENTALLY PREFERRED ALTERNATIVE

The final step, prior to examining the credibility of the selected alternative against each installation inventory, is to evaluate and rank alternatives for normal operations and accident scenarios sequentially on human health impacts, ecosystem and environmental impacts, and the potential for risk reduction due to emergency planning and preparedness.

The values for each category of human health and ecosystem and environmental impacts will be shown, as described above. If an alternative is clearly preferred on the basis of human health impacts, it is unnecessary to consider other impact categories for the selection of the environmentally preferred alternative(s). If no such clear distinction is observed, all alternatives that are equivalent on the basis of human health effects are compared on the basis of ecosystem and environmental impacts. Finally, all alternatives that are equivalent on the basis of human health and ecosystem and environmental effects are compared on the basis of the potential risk reduction associated with emergency planning and preparedness,

N.4.7 EXAMINING THE PRELIMINARY SELECTED ALTERNATIVE FOR EACH INSTALLATION INVENTORY

The final step in the analysis is to examine the preliminary selected alternative (see above step) against each installation inventory to ensure that the methodology has not identified an alternative that is incorrect for one or more installations' inventory(ies). If the preferred alternative is continued storage (which is legally required to be assessed by the Council in Environmental Quality (CEQ) regulations but which is illegal given the Congressional mandate of Public Law 99-145) or on-site disposal, this will involve comparison of the impacts and risks to be borne by individuals living near the installations to those by individuals living along transport corridors and destination sites as well as the origin sites. These trade-offs will be presented to assist the reader in understanding the rationale for selecting the preferred alternative. In sum, however, for an alternative to stand as the programmatic environmentally preferred alternative, it must have the lowest net adverse impacts and risks to all of society.

M.5. UNCERTAINTIES IN THE SELECTION OF THE ENVIRONMENTALLY PREFERRED ALTERNATIVE

In evaluating alternatives, this methodology may result in the identification of more than one alternative. This may particularly be the case given the uncertainties and potential error associated with the various elements in the analysis. These uncertainties and potential errors are found in both the accident analysis (comprehensiveness of accident or accident class identification, accident frequency, and source term) and the consequence analysis [dispersion analysis, toxicological analysis (dose-response effects), and population distribution].

Although the estimation of uncertainties, itself, is subject to uncertainty, attempts have been made to ensure that the uncertainties and potential for error are consistent and systematic throughout the analysis (i.e., that the uncertainty or error is of approximately the same magnitude and in the same direction for all alternatives). For instance, potential handling accidents that are attributable to human error, regardless of which alternative that particular handling is associated with, were treated consistently; therefore, if the frequency or probability estimate is too high (low), the estimate is consistently high (low) for handling activities for each alternative. Similarly, if the population distribution estimates or the atmospheric dispersion estimates (based on use of the D2PC dispersion code) are too high (low), they are consistently so for each alternative examined. Consequently, even relative differences between alternatives may be significant.

Even though substantial efforts have been made to ensure that uncertainties about the values for the various risk measures are treated consistently and systematically for all alternatives, the values may be in error by as much as a factor of 10 in either direction. This means that a value expressed as 10 might actually be as small as 1 or as large as 100.

The values for the multiple risk measures shown in the risk pictograms (to be shown in the FPEIS for each site and transport corridor in Sects. 4.3 and 4.4, respectively; for each disposal alternative in Sect. 2.5; and for the program as a whole in Sect, 2.6) are ordered on the basis of order-of-magnitude differences (represented by differences in shading; see Appendix J of the FPEIS). The shadings help facilitate understanding of differences in impacts and risks, but they do not allow precise mathematical comparisons, including the relevance of actual uncertainties, of alternatives at each site and for the program as a whole [the categorization of the actual value of each risk measure was necessary to avoid disclosure of classified information (see above)]. Although the classified risk data base can be and has been consulted to ensure that apparent differences (i.e., according to the impact and risk pictograms) are at least an order of magnitude different, the actual discrete results cannot be published. Because the particular calculated numerical values represented by adjoining shading; may not truly represent an order of magnitude difference (e.g., 2×10^{-3} or 0.002 is not an order of magnitude larger than 9×10^{-4} or 0.0009), an additional order of magnitude difference (i.e., over and above the actual uncertainty) among alternatives on any given risk measure is required to represent a significant difference. Thus, it will be necessary to look for differences of two or more orders of magnitude (or two or more differences in shading) in the pictograms to determine whether a significant difference exists.

In light of these apparently rather large uncertainties, a significant part of which is a function solely of the result-presentation format dictated by the SECRET nature of the stockpile, it is important not to rely unduly on the absolute values of the risk measures for the disposal alternatives. Instead, the comparative analysis of the disposal alternatives should be sensitive to relative differences between alternatives on the risk measures. Nevertheless, because of the manner in which the results are presented in the pictograms (i.e., representing a calculated value by a range over an order of magnitude), relative differences must be two or more shadings different to constitute a significant statistical difference.

REFERENCES FOR APPENDIX M

MITRE Corporation 1987a. *Risk* Analysis *Supporting the Chemical Stockpile Disposal Program (CSDP)* (SECRET).

MITRE Corporation 1987b. *Risk Analysis Supporting the Final Environmental Impact Statement for the Chemical Stockpile Disposal Program (CSDP).*

GA Technologies, Inc. 1987a. *Risk Analysis of the Continued Storage of . Chemical Munitions,* GAC 18564.

GA Technologies, Inc. 1987b. *Risk* Analysis *of the Disposal of Chemical Munitions at National or Regional* Sites, GAC 18563.

GA Technologies, Inc. 1987c. *Risk Analysis of the On-Site Disposal of Chemical Munitions,* GAC 18562.

Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987. *Emergency Response Concept Plan for the Chemical Stockpile Disposal Program.*

Public Law 99-145, 1985, Department of Defense Authorization Act, 1986

Appendix N

AQUATIC SPILL MODELING AND ANALYSIS

N.1. INTRODUCTION

In this appendix, a description of the general approach used to assess the potential impacts on surface-water and groundwater quality of a chemical agent accident is given. For the purpose of this assessment, a chemical agent accident can involve a spill and/or an atmospheric release. A spill of liquid agent is the release mode by which the largest concentration of agent might be produced in surface waters or groundwaters.

Surface waters could be contaminated with chemical agent in four different ways (Fig. N.l). First, a spill accident might result in liquid agent directly entering surface water. For example, a spill could occur into a drainage ditch or small tributary of a water body during on-site transportation, or agent could directly enter a river following an accident on a bridge. Second, agent might be deposited from an airborne plume or cloud onto surface water. Such a cloud of agent might form by volatilization from a spill that occurred on land or as a result of an explosion aerosolizing some of the agent. Third, if a heavy rain occurred shortly after an accident, agent could be washed into surface waters in runoff from land that had been contaminated by a spill or deposition. Fourth, groundwater may discharge to surface waters and carry contaminants back to the surface. Chemical agent could reach groundwater if precipitation onto contaminated land carried the chemical down to groundwater. Some groundwater is recharged by surface waters, so agent could reach groundwater from contamination of surface water. Agent in water could reach land surfaces, such as flood plains or the sometimes dry channels of intermittent streams, through flooding or through discharge of agent-bearing groundwater.

The aquatic spill model used to assess the impacts of an accidental release of agent is described below. This model estimates the concentration of agent at different times and distances downstream. It accounts for the movement of the agent plume downstream (advection), the spreading of the plume as it moves (longitudinal dispersion), the breakdown of agent by hydrolysis, and the loss by volatilization to the atmosphere.

The aquatic spill model is used in several ways in assessing the impacts of accidental releases of chemical agents. The most important use of the model is as a tool for organizing and integrating the chemical and environmental information related to the behavior of agents in water. The model helps identify which chemical and environmental factors and processes are likely to be most important in determining the agent concentration in water following an accident. The model permits comparisons among agents in their behavior in water, and allows comparisons among categories of streams and environmental conditions.

Another use of the model is for estimating downstream concentrations at different times for a particular spill scenario. The estimates depend greatly on the specific details of the accident (e.g., the amount of agent reaching the water and the time interval over which the agent

ORNL-OWG 87M-12383

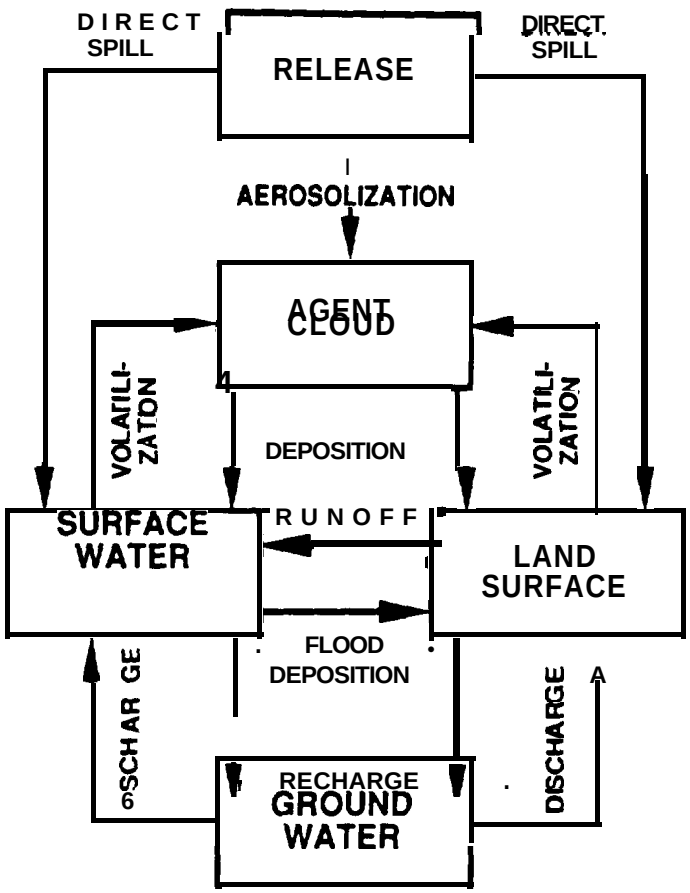


Fig. N.1. Potential pathways for movement of chemical agent to surface water and groundwater following an accidental release.

enters the water) and on the characteristics of the water body at the time of the accident (e.g., the water flow, temperature, and acidity, which all vary seasonally and within seasons).

N.2. AQUATIC SPILL MODEL

Several mathematical models and computer codes have been developed for estimating the downstream concentration of chemicals entering surface waters [see the reviews of Donigian and Brown (1983), Thibodeaux (1979), Neely et al. (1976), Neely and Lutz (1985), and Mills et al. (1982)]. These models differ in the temporal and spatial scales considered (e.g., estimation of concentrations in one, two, or three dimensions along the stream), in the types of chemicals and processes considered, and in the amount of site-specific information required to run the models. Because of the general approach of this assessment, a model was chosen that could be readily applied with a minimum of site-specific information.

An analytical, one-dimensional model (Mills et al. 1982; Eq. IV-139) was used to estimate the downstream concentration profile resulting from a spill of chemical agent into a river [Eq. (N.1)]. This model estimates the concentration of agent at different times and distances downstream, accounting for processes of advection, longitudinal dispersion, hydrolysis, and volatilization to the atmosphere. Additional loss processes, such as microbial degradation of the chemical, can be included as first-order rate constants:

$$C_{x,t} = \frac{M_D}{2A\sqrt{\pi D t}} \exp\left[\frac{-(x - Vt)^2}{4Dt} - k_e t\right] + \left(\frac{k'_v}{k'_v + \sum k_i}\right) \frac{P}{H} [1 - \exp(-k_e t)], \quad \text{(N.1)}$$

where

$C_{x,t}$ = the concentration of chemical agent in the stream at time t and distance x downstream (g/m^3, or ppm);

x = the distance downstream at which the concentration is estimated (m);

t = the time elapsed since the spill (h);

$M_D = M/(1 + K_p S)$ = the effective mass of dissolved chemical agent (g);

M = the mass of chemical agent spilled in the water (g);

K_p = the sediment-partitioning coefficient (L/kg);

S = the concentration of suspended sediment (kg/L);

A = the cross-sectional area of the stream (m^2);

D = the longitudinal dispersion coefficient (m^2/h);

V = the stream velocity (m/h);

$k_e = (k'_v + \sum k_i)/(I + K_p S)$ = the effective first-order decay rate constant for losses of chemical agent (h^{-1});

$k'_v = k_v/Z$ = the volatilization rate constant (h^{-1});

Z = the mean depth of the stream (m);

k_v = the mass-transfer coefficient for volatilization (m/h);

$\sum k_i$ = the sum of first-order rate constants for loss processes, such as hydrolysis and microbial degradation (h^{-1});

P = the partial pressure of the chemical agent in the atmosphere (atm);

H = the Henry's Law constant of the chemical agent $(atm \cdot m^3/g)$.

The equation assumes that a given mass of chemical is released and mixed instantaneously in a plane perpendicular to the flow of the stream (Mills et al. 1982). This assumption allows the model to have a relatively simple mathematical form. This equation is found to give an adequate approximation of the downstream concentration of an injected dye if sufficient time and distance after injection have been allowed for mixing in the vertical and lateral dimensions of the stream (see McQuivey and Keefer 1976a,b; Nusser, Gallagher, and St. John 1978). More-detailed models can do an even better job (see Beer and Young 1983), but they require more-detailed, site-specific information, including data from dye studies for calibration. Near-field models are more useful for predicting the concentration during the initial period following the spill, when vertical and lateral mixing are far from complete; however, such models (McQuivey and Keefer 1976a; Nusser, Gallagher, and St. John 1978; Thibodeaux 1980; Neeley and Lutz. 1965) typically require more specific information about the spill event, which is not known for this analysis.

Equation (N.l) does not apply directly to estuaries or tidal rivers (i.e., rivers in which the flow is influenced by the tides). However, Thomann (1972, pp. 139-145) has used an equation similar to N.l to describe the concentration of a chemical in an estuary following an instantaneous input (e.g., a spill). While Thomann's equation [i.e., Eq. (N.1)] does not describe the back and forth movement of the peak concentration within a tidal cycle, it has proved useful for estimating the tidal dispersion coefficient and for describing the concentration at integer numbers of tidal cycles after the discharge.

N.3. PARAMETER ESTIMATION

N.3.1 STREAM CHARACTERISTICS

N.3.1.1 Steam Size and Flow

Potential surface-water impacts were assessed for a hypothetical river encompassing a wide range of sizes from its headwaters in a small, first-order stream to its discharge into the ocean as a large, tenth-order river. The characteristics of each successive stream order in this river were taken to be the average values estimated by Keup (1985) for U.S. streams of different sizes (Table N.l). The classification of streams into groups, called orders, follows the system of Strahler (1964), in which the smallest streams are classified as first order; the Mississippi River is the only tenth-order stream in the United States (Keup 1985). The Columbia River is representative of the ninth-order segment, the Gila River is representative of the eighth-order segment, and the Allegheny River is representative of the seventh-order segment (Leopold 1962). The flow in a given stream varies during the year according to the pattern of rainfall and the amount of regulation by impoundments; the values in the table are based on mean annual flow. The concentrations of chemical agent in this river at different distances downstream were estimated by using Eq. (N.1), changing the parameters that are influenced by stream size for each successive downstream segment.

The only terms in the spill model that directly represent river size are A, the cross-sectional area; and V, the velocity. Note that the product of AV is the flow or discharge Q. Stream depth, D, increases in larger streams, but enters the model only indirectly; the modeled rate of volatilization of agent from water to the atmosphere is inversely related to mean water depth.

Stream velocity V depends on site-specific factors, including stream gradient and discharge, so that rivers with the same cross-sectional area could have different velocities and discharges. The velocities for each stream order of the hypothetical river represent mean annual flows (Keup 1985). Storm events and changes in reservoir discharge could greatly change the discharge and velocity within a few hours, especially on smaller streams.

N.3.1.2 Water Temperature and pH

The concentration of chemical agent in water following a spill depends greatly on the hydrologic characteristics of the receiving water body, on details of the spill event, and on the water temperature and pH. The range of pH found in natural waters extends from less than 2 to 12 units (Wetzel 1975), though pH is generally from 6 to 9 (Howells 1953). Mabey and Mill (1978) state that a temperature of 20°C, a pH of 7.0, and an ionic strength of 0.00 represent average conditions in most freshwater systems of the United States in non-winter months.

Table N.I. Summary of stream characteristics for U.S. streams by stream order[a]

Stream order	No. of streams[b]	Average length[b] (mile) *L*	Drainage area[b] (mile*) A_d	Mean flow for area drained[c] (cfs)[g] Q	Mean width[d] (ft) *W*	Mean depth[e] (ft) *D*	Mean cross-section area[f] (ft^2) *A*	Mean velocity[d] (ft/s) *V*	Calculated flow[f] (cfs)[g] Q
1	1,570,000	1	1	0.65	4	0.15	0.60	1.0	0.60
2	350,000	2.3	4.7	3.1	10	0.29	2.9	1.3	3.7
3	80,000	5.3	23	15.0	18	0.58	10.	1.5	15.6
4	18.000	12	109	71.0	37	1.10	41.	1.8	73
5	4.200	28	518	340	75	2.20	165	2.3	380
6	950	64	2,500	1,600	160	4.1	660	2.7	1,800
7	200	147	12.000	7,600	320	8.0	2,600	3.3	8,500
8	41	338	56.000	36,000	650	15.0	9,800	3.9	38,000
9	8	777	260,000	171.000	1.300	29.0	38,000	5.6	211,000
10	1	1,800	1,250,000	810.000	2.800	55.0	154,000	5.9	900,000

[a]Data rounded in presentation.
[b]Keup 1985, from Leopold 1962.
[c]A_d X 0.65 cfs = mean discharge/mile^2, or 9 in. of runoff per year.
[d]Figure 1 in Keup 1985.
[e]Calculated $A = W X D$.
[f]Calculated Q = $A X V$.
[g]cfs = cubic feet per second.
Source: Keup 1985, used with permission of the American Water Resources Association.

The persistence of chemical agents in water depends in part on the temperature of the water, due to temperature's influence on the rate of hydrolysis (Mabey and Mill 1978). Figure N.2 shows approximate values for the mean monthly temperature of U.S. surface waters in July and August, generally the time of maximum water temperature (Geraghty et al. 1973). Figure N.3 shows approximate values for the average temperature of shallow, 9 to 18 m (30 to 60 ft) groundwater. The temperature generally increases southward. The temperature of shallow groundwater is determined by the average annual air temperature of the region and seldom varies more than one degree during the year (Geraghty et al. 1973). Within the region encompassing the rail transportation corridors, the average temperature of shallow groundwater ranges from approximately 6°C (42°F) in Wyoming and west-central Colorado to nearly 20°C (67°F) near Montgomery, Alabama.

N.3.1.3 Longitudinal Dispersion

As the pulse of spilled agent moves downstream, it spreads out in the upstream-downstream dimension because of velocity differences and turbulent diffusion (McQuivey and Keefer 1976a). This phenomenon is called longitudinal dispersion and has been studied by **Bansal (1971)**, McQuivey and Keefer **(1976a,b)**, **Bajraktarevic-Dobran (1982)**, and Beer and Young (1983). Beer and Young (1983) suggest dead-zone mixing is the main factor responsible, while McQuivey and Keefer **(1976a,b)** emphasize the effects of vertical differences in stream velocity. The value for longitudinal dispersion *(D)* for the tenth-order stream was taken from Mills et al. (1982), who give a value for the lower Mississippi River (210 m^2/s). For the other stream sizes, *D* was computed using the empirical relationship presented by Hull (1962) in order to account for the observation that dispersion varies with discharge.

$$D = 2.5(QV)^{1/2}, \qquad \text{(N.2)}$$

where *D* is the longitudinal dispersion coefficient (ft^2/s), *Q* is the discharge (cfs or ft^3/s), and *V* is the stream velocity (ft/s). Thomann (1972) reports an average value for **nontidal** rivers of 3 m^2/s (0.1 $mile^2/d$). For the streams characterized in Table N.l, values of *D* estimated using Eq. (N.2) were below 3 m^2/s for those streams listed as order 1 through 4, and above 3 m^2/s for streams listed as order 5 and larger. Thomann specifies the range in *D* for **nontidal** rivers as 0.75 m^2/s (0.025 ❑ ile2/d) to 39 m^2/s (1.3 $mile^2/d$). This range encompasses the estimated *D* values for orders 3 through 7. Because *D* appears inside a square-root sign in the denominator of Eq. **(N.1)**, changes in *D* have a relatively small affect on the estimated concentration. For a stream with Thomann's average value for *D* (3 m^2/s), the estimated concentration would be higher by a factor of 2 if *D were 0.75* m^2/s, or lower by a factor of 3.6 if D were 39 m^2/s. **Thomann** gives a range of 30 to 600 m^2/s

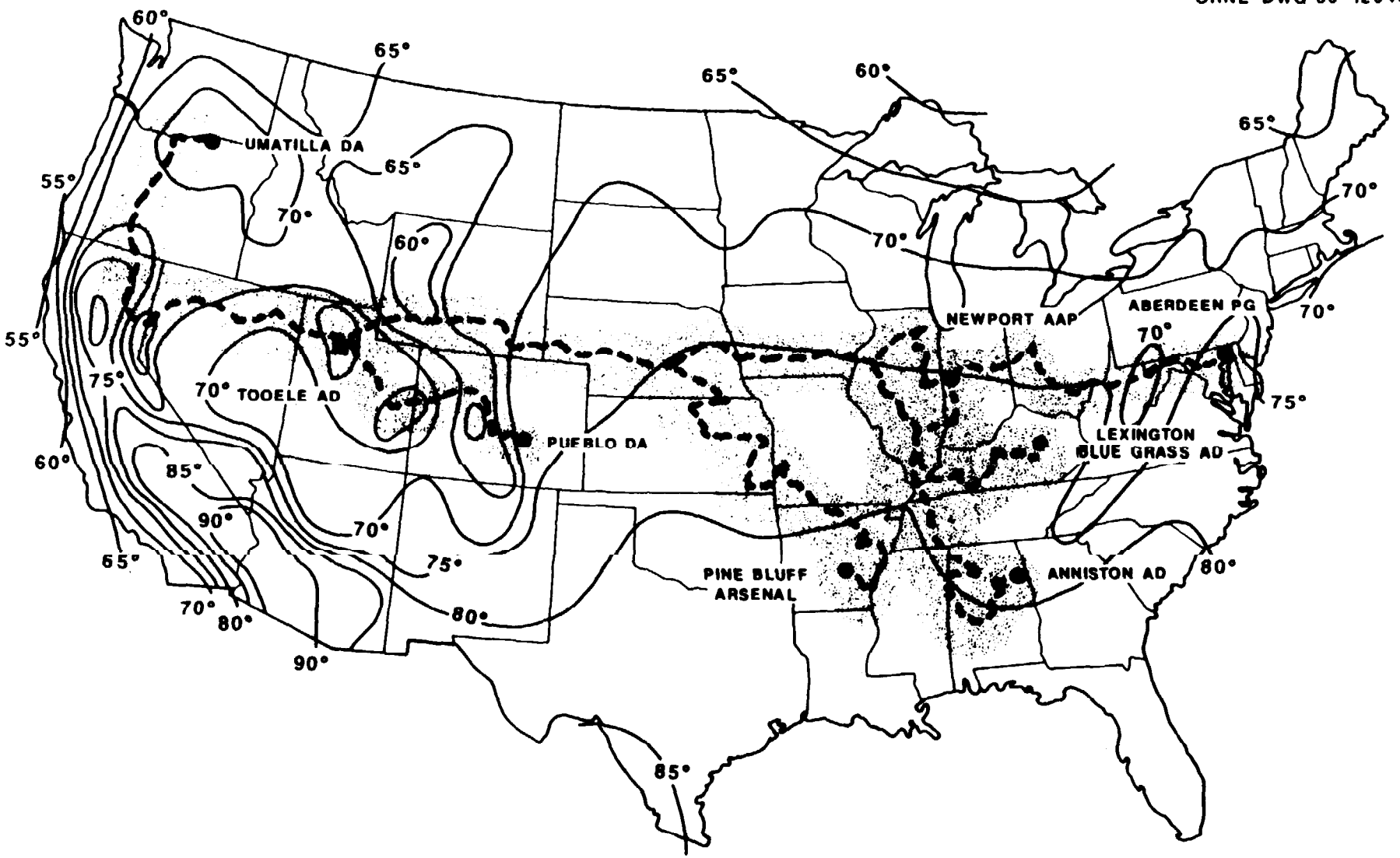


Fig. N.2. Approximate mean monthly temperature (degrees F) of surface water in July and August. The dashed lines indicate the rail transportation routes and the stippled region indicates the area within the 200-km (124-mile) rail transportation corridors from the storage sites to the national and regional disposal facilities. *Source:* Modified from Geraghty, J.J., et al. 1973. *Water Atlas of the United States,* Water Information Center, Port Washington, New York.

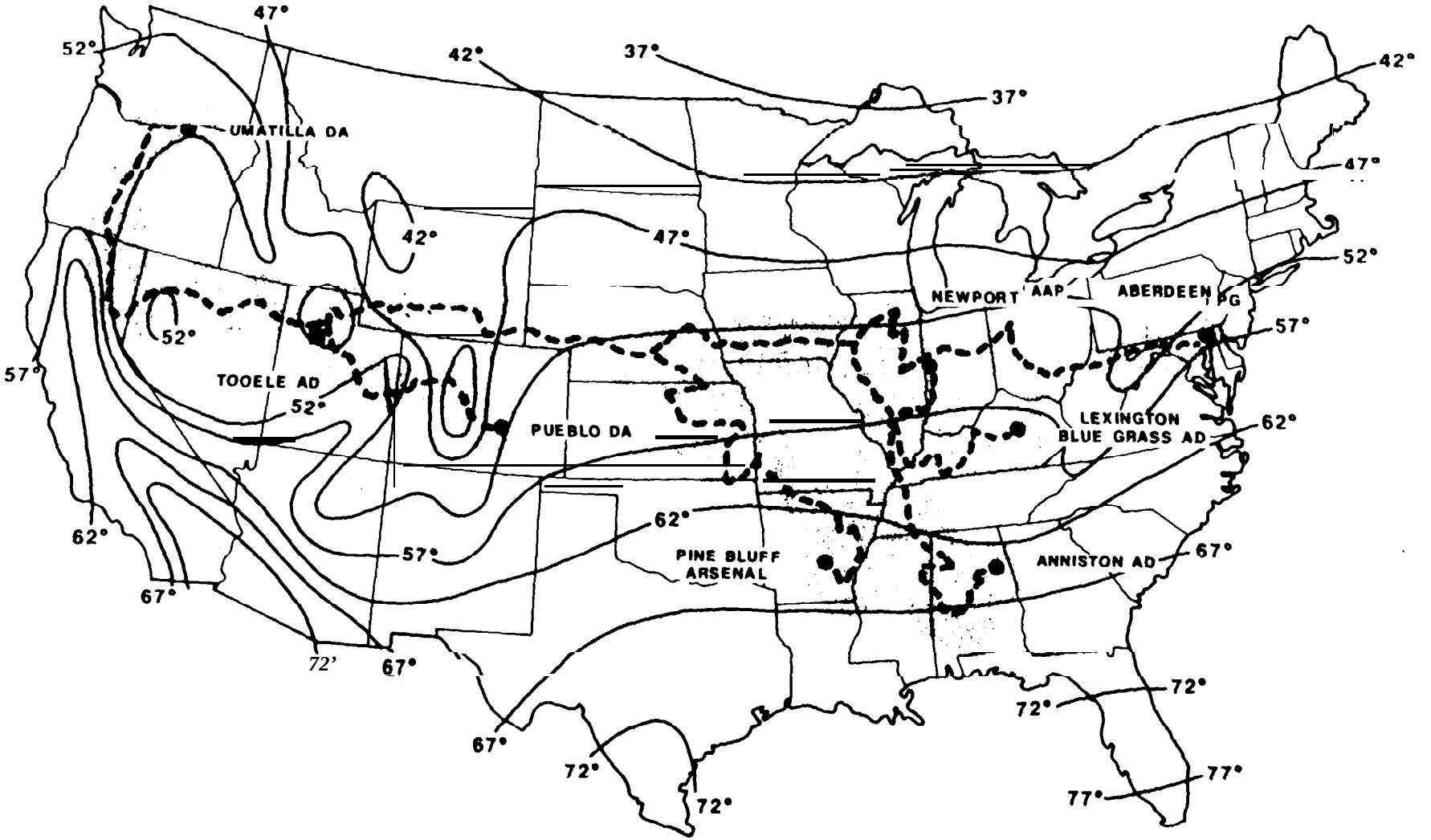


Fig. N.3. Average temperature (degrees F) of shallow [9–18 m (30–60 ft)] groundwater. The dashed lines indicate the rail transportation routes and the stippled region indicates the area within the 200-km (124-mile) rail transportation corridors from the storage sites to the national and regional disposal facilities. *Source:* Modified from Geraghty, J. J., et al. 1973. *Water Atlas of the United States,* Water Information Center, Port Washington, New York.

(1 to 20 $mile^2/d$) for measurements of. longitudinal dispersion in estuarine situations, with 150 m^2/s (5 $mile^2/d$) as the value of D for a typical East Coast estuary. Neely and Lutz (1985) estimated an even larger value [330 m^2/s (11 $mile^2/d$)] for a section of a river in British Columbia that includes rapids. A larger value for longitudinal dispersion would reduce the estimated peak concentration.

N.3.2 PHYSICOCHEMICAL PROPERTIES

Certain model parameters related to the chemical agents themselves were obtained from the open literature and other unclassified sources. Values for a few parameters had to be estimated or assumed (Tables N.2 and N.3). This analysis of aquatic impacts using the aquatic spill model assumes that the total mass of agent from the spill would be released directly into surface waters.

N.3.2.1 Solubility in Water

Bennett et al. (1984) indicate that VX is relatively insoluble at warm temperatures (3% at 25°C) and miscible below 9.4°C. Based on Neely and Lutz (1985), a spill of a compound with this solubility (30 g/L at 25°C) might cause only 20% of the chemical to dissolve immediately, with the remaining 80% going into solution over several days. Similarly, based on reported values for the solubility of distilled mustard agent (HD) [1.0 g/L at 25°C (Small 1984); 0.8 g/L at 20°C (Trapp 1985, p.4); 0.68 g/L at 25°C (U.S. Environmental Protection Agency 1985)], a spill of HD might result in only 5 to 20% of the mustard dissolving immediately. If this slow dissolution occurred with VX or HD, the peak concentration estimated by Eq. (N.l) would be reduced, but a longer exposure to low concentrations would occur as VX and HD went into solution.

Equation (N.l) accounts for sorption of the chemical agent to particles and mass transfer of agent from the atmosphere to the water. However, agents GA and GB are miscible in water, and agent VX is miscible in water at temperatures below 49°F (9.4°C) (Bennett et al. 1984; U.S. EPA 1985). [There is some disagreement about the solubility of GA. One source, Windholz (1983, p. 1297), indicates that GA is miscible, while another source, U.S. Army (1974), indicates a solubility of 98 g/L at 25°C and 72 g/L at 20°C (Table N.3)]. For chemicals that are as soluble in water as these, sorption to particles in water is likely to be insignificant (Mills et al. 1982; Lyman, Reehl, and Rosenblatt 1982). For this analysis, the conservative assumption is made that the vast majority of the chemical will be in the dissolved form, and a negligible fraction is likely to be associated with suspended or sediment particles. This assumption implies that the product K_pS is much less than 1.0 and can be ignored in estimating M_D and k_e for Eq. (N.l). (Any sorption that occurs will reduce the concentration of dissolved agent.) The partial pressure of chemical agent in the atmosphere (P) can be assumed to be zero, further simplifying Eq. (N.l).

Table N.2. Chemical properties of chemical agents HD and L and estimated parameters for tbe aquatic spill model

Property	Symbol	Distilled mustard (HD)	Lewisite (L)
CAS registry number		505-60-2	541-25-3
Chemical formula		$C_4H_8Cl_2S$[a]	$C_2H_2AsCl_3$[a]
Molecular weight	MW	159.08"	207.32[b]
Freezing point		14.45°C[a]	−18 to 0.1°C[a,c]
Vapor pressure	P_{vp}	0.072 mm Hg at 20°C[a] 0.000095 atm at 20°C 0.1 15 mm Hg at 20°C[d]	0.087 mm Hg at 0°C[a] 0.394 mm Hg at 20°C" 0.000518 atm at 20°C
Water solubility	S	1 .0 g/L' 6.3 mol/m³ at 25°C 0.8 g/L at 20°C[d] 0.68 g/L at 25°C[f]	Insoluble[b]; slightly soluble in distilled water'
Liquid density		1.2685 g/cm' at 25°C[a] 1.2741 g/cm³ at 20°C[h]	1.89 g/cm' at 20°C[a]
Estimated maximum solubility	S_{mu}	See s	
Estimated Henry's Law constant	H = P_{vp}/S	2.2 x 10^{-5} atm m³/mol at 25°C	
Estimated nondimensional Henry's Law constant	H' = H/RT	9.2 x IO-'	
Default gas-phase exchange coefficient	k_g	3000 cm/h'	3000 cm/h[i]
Hydrolysis	$t_{1/2}$ (hyd.)	8.5 min in distilled water at 25°C[a] 158 min at 0.6°C[e] 7.4-15.8 min at 20°C 60 min in salt water at 25°C	Rapid for dissolved Lewisite'
Acid dissociation constant	K_a		
Log octanol-water partition coefficient (calculated)	log K_{ow}	1.37[c]	

[a]U.S. Army and Air Force 1975.
[b]U.S. EPA 1985; Windholz 1983, p. 444–445.
<Freezing point depends on purity and isomers present; 0. 1 °C for purified compound.
[d]Trapp 1985, p. 13.
'Small 1984.
[f]U.S. Army 1974.
[g]U.S. EPA 1985.
[h]Windholz 1983, p. 904.
'Thomas 1982.

Table N.3. Chemical properties of chemical agents CA. GB. and VX and estimated parameters for the aquatic spill model

Property	Symbol	Tabun (GA)	Sarin (GB)	VX
CAS registry number		77-81-6	107-44-b	50782-69-9
Chemical formula		$C_5H_{11}N_2O_2P$[a]	$C_4H_{10}FO_2P$[a]	$C_{11}H_{26}NO_2PS$[a]
Molecular weight	MW	162 1 ?	140 10[a]	267 38[a]
Freezing point		−50°C[a]	−56°C[a]	Below − 51°C, calculated to be − 39°C[a]
Vapor pressure	P_{vp}	0 070 mm Hg at 25°C[a] 0 000092 arm at 25°C	2 9 mm Hg at 25°C[a] 0 0038 arm at 25°C I 48 mm Hg at 20°C[a]	0.0007 mm Hg at 25°C 9 2 $\times 10^{-3}$ atm at 25°C 0 0001 I mm Hg at 20°C[d]
Water solubility	s	Miscible[e] 72 g/L at 20°C[a] 98 g/L at 25°C[c]	Miscible[f]	3% at 25°C. 7 5% at 15°C.[c] miscible below 9 4°C[c,f]
Liquid density		I 073 g/cm³ at 25°C[a]	IO887 g/cm³ at 25°C[a] I 10 g/cm³ at 20°C[a]	I.0083 g/cm³ at 25°C[a]
Estimated maximum solubility	S_{max}	661 I mol/m³ at 25°C	7771 mol/m³ at 25°C	3765 mol/m³ at 25°C
Estimated minimum Henry's Law constant	$H_{min} = P_{vp}/S_{max}$	I 4 × 10^{-8} atm m³/mol at 25°C	4 9 × 10^{-7} atm m³/mol at 25°C	2 4 × 10^{-10} arm m³/mol a, 25°C
Estimated minimum nondimensional Henry's Law constant	$H_{min} = H_{min}/RT$	5 ? × 10^{-7}	2 0 × 10^{-5}	I O × 10^{-8}
Default gas-phase exchange coefficient	k_g	3000 cm/h[d]	3000 cm/h[d]	3000 cm/h[d]
Hydrolysis	$t_{1/2}$ (hyd	7 h at 20°C. pH 4 5[a] 8 5 h at 20°C pH 7 0	837 h at 0°C. pH 7 5[a] 453 h at 4°C. pH 7 5[a] 46 h at 20°C. pH 7 5[a] 24 h at 25°C. pH 7 5[a]	I? years at 4°C. pH 7.6[d] 996 h at 25°C. pH 7 0 428 h at 25°C. pH 7 5. 184 h at 25°C. pH 8 0
Acid dissociation constant	K_a			2 5 . 10^{-9}
Log octanol-water partition coefficient (calculated)	log K_{ow}		0 72[f]	2 09[f]

[a] U S Army 1975
[b] Windholz 1983, pp 1297, 1204
[c] U S Army 1974
[d] Trapp 1985
[e] U S EPA 1985, Windholz 1976.
[f] Small 1984
[g] Thomas 1982
[h] Epstein 1974
[i] Epstein, Callahan, and Bauer 1974

N.3.2.2 Hydrolysis

The hydrolysis of chemical agents greatly influences their persistence in water. Agent GB can hydrolyze rapidly at high temperatures and high pH but is quite persistent in water at low temperatures (0 to 4°C) and environmentally realistic pHs (near 7). The dependence of the rate of hydrolysis on both water temperature and pH can be seen in the following equation developed by Epstein (1974) for agent GB:

$$\log t_{1/2} = (5039/T) - 8.035 - \mathrm{pH} , \qquad (N.3)$$

for pH equal to or greater than 6.5, where $t_{1/2}$ is the half-life caused by hydrolysis (h) (the hours required for half of the remaining chemical to be hydrolyzed) and T is the water temperature (degrees Kelvin).

The rate constant for hydrolysis (k_h) is related to the hydrolysis half-life as follows:

$$k_h = (\ln 2)/t_{1/2} . \qquad (N.4)$$

The hydrolysis of GB occurs faster in seawater than in freshwater, primarily due to the increased concentration of magnesium ions in seawater (Michels, Gordon, and Epstein 1973). The hydrolysis products of GB are given in Tables B.19 and B.20 of Appendix B. Epstein et al. (1973, p. 14) indicate that tile organic hydrolysis product of GB has little or no toxicity; rats showed no ill effects after being fed water containing as much as 200 ppm of the hydrolysis products of GB for 3 weeks.

According to the experiments by Epstein, Callahan, and Bauer (1974), at 25°C the hydrolysis rate of VX increases as pH increases above 6. While this behavior is similar to that of GB, the hydrolysis rate of VX tends to be much less: at 25°C and pH 7.5, the hydrolysis half-life of GB is approximately 24 h, while that for VX is approximately 428 h. The hydrolysis half-life of VX in water from rainfall or snowmelt was 28 d at 10°C and 0.4 d at 40°C (Bennett et al. 1984, p. 150). The hydrolysis products of VX are given in Tables B.19 and B.20 of Appendix B. One of the hydrolysis products of VX is highly toxic, approaching the toxicity of VX itself (Epstein et al. 1973, p. 16). This product is much more resistant to hydrolysis than VX. It is estimated that the hydrolysis rate of the toxic hydrolysis product is 1/100 that of VX (Epstein et al. 1973, p. 16); under conditions where the half-life of VX is 428 h, the half-life of the toxic hydrolysis product would be almost 5 years.

The hydrolysis rate of dissolved HD is much faster than that of GB; values of 3.9, 8.5, and 8 min have been given for the hydrolysis half-life of HD in water at 25°C (Small 1984; U.S. Army and U.S. Air Force 1975), and 158 min is the reported value at 0.6°C (Small 1984) (see Tables N.2 and N.3). Mustard hydrolysis is slower in seawater. The reported half-life of mustard in seawater is 15 min at 25°C, 49 min at

15°C, and 175 min at 5°C (Epstein et al. 1973, p. 11). Once mustard agent dissolves, it should hydrolyze relatively rapidly; however, HD does not dissolve easily. The dissolution rate of a droplet of HD is increased by turbulence, such as would occur in most areas of a river or stream. However, Small (1984) has estimated that in quiescent water at 18°C, it would take 15 d for half the mass of a l-cm HD droplet to dissolve. This is enough time for other processes to slow or even halt further dissolution and hydrolysis, such as the formation of a shielding interface layer around the droplet by hydrolysis reaction products, or the formation of the HD linear oligomer or polymer (which are less soluble than HD but still blister-forming) (Small 1984; Trapp 1985, pp. 17-18). Small (1984) notes that HD has been found to persist under water or in soil for years.

The hydrolysis products of HD are given in Tables B.19 and B.20 of Appendix B. The final hydrolysis products of HD, thiodiglycol and hydrochloric acid, are nontoxic materials, as far as was known to Epstein et al. (1973, p. 12). However, one of the intermediate hydrolysis products, beta-chloroethyl hydroxyethyl sulfide, is almost as toxic as mustard itself and has a hydrolysis rate comparable to HD (Epstein et al. 1973, p. 12). Therefore, the half-life for the destruction of total toxic substances by hydrolysis is approximately twice the half-life of HD (Epstein et al. 1973, p. 12).

In some sources (e.g., U. S. Army 1974), Lewisite is described as "slightly soluble" in distilled water. In other sources (Weast 1979, p. C-298; Windholz 1983, p. 445), Lewisite is described as "insoluble" in water; but, "insoluble" is not a precise, quantitative term. Many compounds commonly regarded as insoluble actually dissolve to a slight extent, and conflicting statements about the solubility of chemicals are common in the literature (Hodgman 1960, pp. 761-762). With high turbulence (e.g., shaking in a laboratory flask) Lewisite can dissolve rapidly in large volumes of water and can hydrolyze as rapidly as it dissolves. However, one of the hydrolysis products, chlorovinylarsenous oxide, is a nonvolatile blister-forming solid (the blister-forming properties are destroyed by alkaline hydrolysis) (U.S. Army and U.S. Air Force 1975). Also, because Lewisite is 36% arsenic by weight, even if the compound were broken down, a toxic element would remain. Additional hydrolysis products of Lewisite are given in Tables B.19 and B.20 of Appendix B.

N.3.2.3 Volatilization

Volatilization of chemical agents from water to the atmosphere is another potentially important loss mechanism (Fig. N.l). Because information was not available on the rate of volatilization from water, this rate was estimated with methods suggested by Thomas (1982) and based on the chemical properties of the agents (Tables N.2 and N.3).

Because GA, GB, and VX are miscible in water at low temperatures, their liquid densities set a maximum limit for their solubility in water. Dividing the vapor pressure by this maximum water solubility yields a minimum estimate of the Henry's Law constant; for HD, dividing the vapor pressure by the measured value for water solubility gives an estimate of the Henry's Law constant (Thomas 1982). The vapor pressure of GB, VX, and HD at a particular temperature was computed using Eq. (N.5), from Table A-l of the report, *Transportation of Chemical Munitions at Reduced Temperature* (MITRE 1987):

$$\log P_{vp} = -A/T + B \quad , \qquad \text{(N.5)}$$

where P_{vp} is the vapor pressure (mm Hg), T is the temperature (Kelvin), and A and B are agent-specific coefficients: for GB, A-2478, B-8.725; for VX, A-4747, B-12.63, and for HD, A-3151, B-9.599 (MITRE 1987, Table A-l).

The default value recommended in Thomas (1982) was used for the gas-phase exchange coefficient; the liquid-phase exchange coefficient for each agent was estimated separately for each of the river sizes analyzed (i.e., for each velocity-depth combination in Table N.l). With the Henry's Law constant (converted to its nondimensional form) and the gas- and liquid-phase exchange coefficients, a rate of volatilization was computed for the agents for each of the different stream sizes in Table N.l (Thomas 1982). This estimation technique has greater uncertainties for miscible or very soluble compounds such as GB than for compounds with lower solubility (Sanders and Seiber 1984).

The estimated rate of volatilization differed greatly among the chemical agents, producing the following ranking (from more rapid to very slow): HD > GB > GA > VX. The rates of volatilization of VX and GA were estimated *to* be very low. However, volatilization was estimated to be important for dissolved HD and also for GB in the shallower streams at low stream temperatures, where the hydrolysis rate is low and both hydrolysis and volatilization occur at similar rates. At higher water temperatures, GB hydrolysis is much faster than volatilization, and volatilization is proportionately much less important.

N.3.2.4 Other Loss Processes

Microbial degradation of certain chemicals can contribute significantly to their rate of disappearance from water (Roubal, Horowitz, and Atlas 1979). Because of a lack of information *on* the rate of microbial degradation of chemical agents in water, the conservative assumption was made that this loss rate is zero.

Losses due to photodecomposition are. assumed to be negligible, a conservative assumption, because of the lack of detailed information about this process for chemical agents (Trapp 1985, p. 67).

N.3.3 REFERENCE VALUES

Reference values of 0.0028 ppm for GB and 0.0015 ppm for VX are used in this analysis, based on the recommended maximum permissible concentrations in drinking water used by the public (see Appendix B, Sect. B.6.2.1, Table B.21). Recommended reference values for HD are not yet available. These values are provided as a point of reference for the analysis in this section. The analysis in this section does not attempt in any way to predict or extrapolate to human populations (see Appendix B).

N.4. IMPACTS OF AN AQUATIC SPILL

The impacts of a spill on water would depend on the details of the accident, particularly on how much agent was spilled directly into water or deposited onto water surfaces.

The impacts on drinking water could be greatest on a river of intermediate size. There the dilution capacity is less than that of a larger river, but the likelihood of a public water supply intake is typically greater than that for a smaller river or stream. The number of identified public water supply intakes from surface waters downstream of the chemical agent storage sites is given in Sect. 3. Also presented in Sect. 3 is a figure showing surface water use for public water supplies, as a percent of total water use, for states along the rail corridors.

Figure N.4 shows the estimated maximum concentrations of chemical agents in water if a spill were to occur directly into a headwater (first order) stream for several different combinations of agent, water temperature, pH, and for several sizes of river. For the nerve agents GA, GB, and VX, the peak concentration of the pulse following an instantaneous spill of 4.54 kg (10 lb) (the amount found in a typical burstered munition) in a headwater stream would remain above the reference levels (0.0028 ppm GB, 0.0015 ppm VX) for several hours and many miles downstream. Because the predicted concentration downstream is proportional to the initial amount of agent dissolved in the water, Fig. N.4 can be used to estimate the peak concentration for other spill sizes; a ten-fold larger spill would produce a ten-fold increase in the predicted peak concentration. Note that Fig. N.4 shows the maximum concentration of the pulse as it moves downstream; before and after the peak concentration passes a given location along the river, the concentration will be much less than the value shown in Fig. N.4.

Dilution and longitudinal dispersion are responsible for most of the decrease in peak concentration downstream, especially for VX and for GB at low temperatures (Fig. N.4). These calculated concentrations assume that the longitudinal dispersion coefficient within each river segment

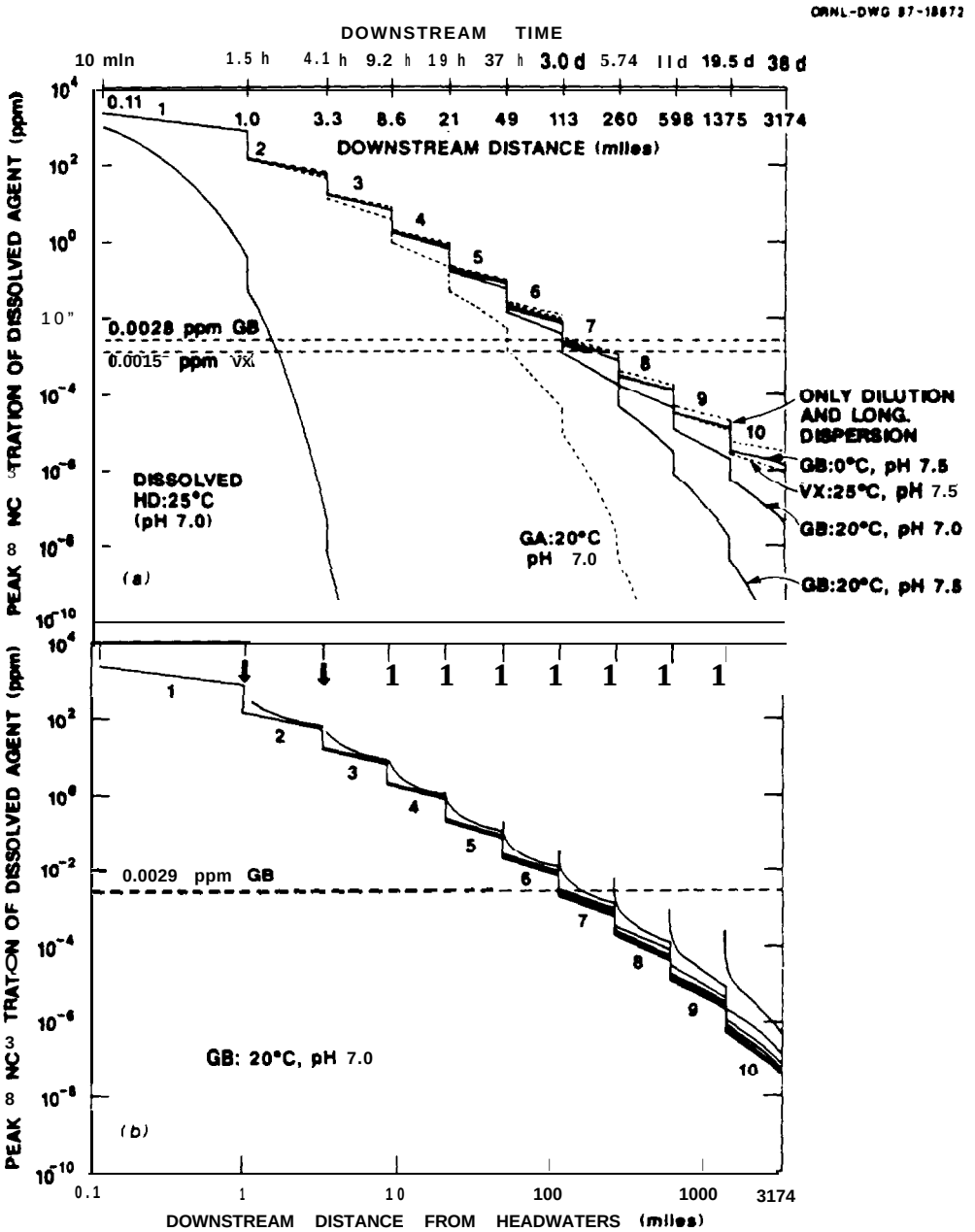


Fig. N.4. Estimated peak concentration of rgent downstream of a spill into a hypothetical river; 10 lb (4.54 kg) of agent are assumed to be spilled and completely dissolved. (a) A spill occurs into one of the river's headwater, first-order streams; each line represents a different agent-temperature-pH combination. Abrupt drops in concentration are due to dilution as the stream merges to form a larger stream. The uppermost concentration line represents the peak concentration for a conservative substance (i.e., the concentration is reduced by dilution and longitudinal dispersion, but not by hydrolysis or volatilization). The downstream times and distances to reach each higher stream order are indicated at the top of the figure. (*b*) A spill of agent GB into water at 20°C and pH 7.0; the lines represent the peak concentrations following spills at different locations (indicated by arrows) on the hypothetical river. Large numbers indicate the stream order (see Table N.l). The reference value (horizontal dashed line) is discussed in Sect. N.3.

is the value estimated by Eq. (N.2) (Hull 1962). If a four-fold lower value were used, the estimated concentrations would be increased by a factor of 2; if a four-fold higher dispersion value were used, the concentrations would be decreased by a factor of 2. For agents HD and L, the concentration of dissolved agent will decrease very rapidly primarily due to rapid hydrolysis of the dissolved agent (though not all of the spilled agent will dissolve, as will be discussed).

If a small spill were to occur in a large river, impacts would be negligible except in the immediate vicinity of the spill because of rapid dilution, even during cold weather. In a large river, even fairly large spills would be rapidly diluted below the reference concentration.

The spill model used here (Sect. N.2) assumes that the mass of chemical agent is instantaneously dissolved in the river or stream--the most conservative assumption. If the chemical agent slowly leaks into the river, then the peak concentration could be greatly reduced but the duration of the plume at any given point would be extended. Because of the time required for vertical and lateral mixing after a spill, the concentrations given in Fig. N.4 are not likely to be good approximations for the actual concentration at a particular point in the (incompletely mixed) plume immediately after a spill, especially in a large river. At the point of the spill, toxicant concentration within the plume may be higher than the values in Fig. N.4, while the concentration may be much less at points outside the plume in the vertical or lateral directions, but at the same distance downstream. McQuivey and Keefer (1976b) measured the dispersion of dye released in the lower Mississippi River and estimated that vertical mixing was nearly complete in about 10 miles [16.1 km, or after about 8 h at a velocity of 1.85 ft/s (0.564 m/s)] and that lateral mixing was nearly complete in about 50 miles [80.4 km, or after about 40 h at 1.85 ft/s (0.564 m/s)]. From these numbers, it is apparent that a public water intake just downstream from a spill site might encounter much lower or much higher concentrations than those shown for the first few minutes following the spill (Fig. N.4), depending on whether the center of the plume misses or passes directly by the water intake. The specific details of the spill event and a more-detailed near-field model (see Nusser, Gallagher, and St. John 1978) would be required to predict the concentration at specified points in the stream during the initial interval after the spill.

The concentration values in Fig. N.4 assume that the agent dissolves completely in water. This is a good assumption for the miscible GB and GA, but it may not be a good assumption for VX, HD (or H or HT), or L. Bennett et al. (1984) characterize VX as relatively insoluble and slightly heavier than water (specific gravity of 1.0083 at 25°C) and suggest that it might sink before gradually dissolving; HD, with a specific gravity of 1.2685 g/cm^3 at 25°C (U.S. Army and U.S. Air Force 1975), would be even more likely to sink. Small globules of VX or HD might remain on the surface, within the water column, or, if the rate of dissolution is very slow, on the bottom. The agent within such globules

would persist until dissolution occurred.

As will be discussed in Sect. N.5, some of the mustard spilled into water would be likely to form a film on the water (Epstein et al. 1973, p. 10). This film would have vesicant (blister-forming) properties, would persist from several hours to several days, and would contaminate the shoreline. Some of the mustard could form oil-like masses on the bottom; the mustard in these masses would be an active vesicant (Epstein et al. 1973, p. 11). These masses would have the potential to persist for several. months, at least, in quiet water, especially at low temperatures. If the accident occurred when the water was cold, mustard would solidify and sink to the bottom. Solid mustard tends to dissolve less slowly than liquid mustard (Epstein et al. 1973, p. 11), so the undissolved agent would tend to persist for a shorter time in cold water. Part af the decontamination effort following a spill would include a search for such masses of mustard, and their removal and decontamination.

If bodies of water used for public water supplies are contaminated by chemical agent, remedial action can be taken at the water utility. However, depending on the type of filtration employed, VX globules may not be completely removed. Emergency treatment to reduce concentrations of specific chemicals (including dissolved chemical agents) in community water supplies is discussed in the American *Water Works* Association *Manual,* Ml9 (Agardy and Ray 1973). The emergency procedures involve superchlorination at a pH of 7, followed by dechlorination and conventional clarification processes. If globules of VX were in the water, they could be resistant to chlorination treatment and might require removal by sand filters.

N.5. DEPOSITION ONTO SURFACE WATERS

An atmospheric release of mustard, such as an aircraft crash into a mustard storage site, would have the potential for significant impacts on surface water quality. Some portion of the mustard released into the atmosphere could deposit onto water surfaces, such as nearby rivers or lakes. Deposition would be greatest close to the accident site (see Sect. 4.2.2.2).

Some of the mustard that deposited on water would farm an iridescent surface film, like a film of oil (Epstein et al. 1973, p. 10; Madema 1986, p. 68). This film would have vesicant properties (Epstein et al. 1973, p. 10). The maximum distance at which deposition from this accident would be able to form a film with vesicant properties is not known at this time (see Sect. 4.2.2.2). In quiet water, the surface film could persist for several days; where there was wind or mechanical agitation of the water surface, the film could probably persist from 24 to 48 h (Epstein et al. 1973, p. 11). The surface film would contaminate the shoreline. If deposited on a stream, the film would likely persist long enough to be transported same distance downstream; the distance downstream that the film might move would depend on the wind, the water

turbulence, and the water velocity at the time of the accident.

This accident could contaminate the bottom (sediment) of bodies of water receiving the mustard. Because HD is more dense than water, droplets of mustard would fall from the water surface to the bottom (Epstein et al. 1973, p. 11). Because of the slow rate at which mustard dissolves, these droplets could persist and retain their vesicant properties for a long time. In quiet water, drops of mustard might persist for months or years. Water currents would move the droplets; mustard would dissolve and break down by hydrolysis more rapidly where there was turbulence, such as would occur in most areas of a river or stream. High flows would resuspend some bottom material and might temporarily increase the concentration of mustard in the water. The turbulence causing the resuspension would also tend to break up and dissolve the mustard droplets from the sediment, increasing mustard breakdown by hydrolysis.

The concentration of mustard in the water between the surface and the bottom probably would be very low several hours after deposition ceased (Epstein et al. 1973, p. 11). Large mustard droplets in this zone would sink to the bottom, fine droplets would dissolve within several hours, and the dissolved mustard would break down by hydrolysis within a few more hours (though it would take longer at low temperatures).

Surface waters could be contaminated by deposition from an airborne release as well as from a spill directly into water, as discussed previously. Table N.4 shows the approximate amount of chemical agent expected to be deposited per square meter between each human-effects isopleth and the water concentrations that would result if the agent were to completely dissolve in a pond 1 m deep. Due to the lack of appropriate values in the literature, no deposition values for blister agents can be calculated. These deposition values for nerve agents were calculated as discussed in Sect. 4.2.2.2. The uncertainty associated with the estimated deposition values is plus or minus one order of magnitude [i.e., actual values might differ by a factor of 10 (Sect. 4.2.2.2)].

The highest agent deposition value listed in Table N.4 is 0.4 mg/m^2, but this is an average value for the area within the 50% isopleth. Dissolved in water 1 m deep, this amount would produce a chemical agent concentration of about 0.0004 ppm, a factor of 7 lower than the reference drinking-water levels of 0.0028 ppm GB, and a factor of about 4 lower than the reference concentration of 0.0015 ppm for VX. Because of the uncertainly associated -with the deposition and water-depth values, the reference drinking-water level might be exceeded in some of the shallow standing water bodies close to the spill site. It is unlikely, however, that shallow, standing water bodies would be used for human ingestion. Agent deposited into flowing water would be diluted to even lower levels than those shown in Table N.4.

Primarily because of the very low -volatility of VX, the concentration of this agent in the atmosphere near the spill site would be negligible (see Sect. 4.2.2.1). Deposition, therefore, would extend a

Table N.4. Potential impacts on water due to deposition from an airborne plume of chemical agent

Assumptions for these calculations are given in the text'

Human-effect isopleth[b]	Deposition (mg/m^2)	Agent concentration in 1-m-deep pond (ppm)
	Agent VX	
50% < x	0.4	0.0004
1% < X < 50%	0.1	0.0001
ND < X < 1%	0.06	0.00006
NE < X < ND	0.003	0.000003
	Agent GB	
50% < x	0.4	0.0004
1% < x < 50%	0.1	0.0001
ND < X < 1%	0.06	0.00006
NE < X < ND	0.003	0.000003

"Concentrations are not listed for mustard because deposition values are not known.

'Human-effect isopleths are discussed in Sect. 4.2.2.1. and deposition of agent is discussed in Sect. 4.2.2.2.

Water concentrations are given in parts per million (ppm) or g/m^3.

negligible distance beyond the spill site (see Table N.4 and Sect. 4.2.2.2) and surface waters would not be affected. Deposition of mustard onto a water body used for drinking water would be of concern due to the potential persistence of the droplets. Any dissolved mustard would, however, hydrolyze rapidly.

N.6 RUNOFF FOLLOWING DEPOSITION ONTO LAND

Contamination of surface waters could be caused by runoff of chemical agent deposited onto land surfaces. Such runoff containing nerve agent might occur following heavy rains, which could mobilize some of the agent deposited from an airborne cloud. Factors influencing the amount of chemical moved to surface waters include (1) the time interval between deposition and the first rainfall; (2) the amount of chemical deposited; (3) the solubility of the chemical [GA and GB are miscible in water; the solubility of VX in water is 3% at 25°C and 100% below 9.4°C; HD and L are much less soluble than VX (Tables N.2 and N.3)]; (4) the slope of the land; (5) the vegetative cover; (6) the amount and intensity of the rainfall; (7) the texture and moisture content of the soil; and (8) the distance between the area of deposition and major bodies of surface water (Merkle and Bovey 1974). Most of the movement of pesticides to surface waters occurs with the first heavy rain following application (Merkle and Bovey 1974), and the same pattern would be expected with these chemical agents. Because GB and HD have greater volatility and a more rapid rate of hydrolysis than VX, they would tend to persist on the soil surface for a shorter period of time than VX (Small 1984). The greater persistence of VX on soil means that, following deposition, there is a longer time interval during which a heavy rain could transport VX to surface waters.

Concentrations resulting from runoff could vary widely among potential accident sites because of the broad range in possible values of the factors influencing runoff. A large or very small fraction of the agent deposited might be dissolved in runoff. A very rough calculation of the concentration in runoff is as follows: If 100% of the GB deposited within the 50%-human-effects isopleth (an average value of 0.4 mg/m^2) were dissolved in a 1-cm rainfall (10 L/m^2), the concentration of GB in the resulting runoff would be 0.04 ppm. A lighter rainfall might produce a higher chemical agent concentration, but a smaller fraction of the rainfall would run off to streams or ponds.

Some mustard deposited onto land could be carried to surface waters by runoff following a heavy rain shortly after the accident. However, in the arid western sites rainfall is infrequent. Though vaporization of mustard from a contaminated mass of soil can be very slow (an estimated 1.8 years to remove 90% from a layer of soil 10 cm deep), vaporization of mustard from a soil surface is estimated to be much faster (an estimated 30 min to remove 50%) (Small 1984, p. 23). Deposition from an atmospheric release would contaminate the soil surface.

N.7. CONTAMINATION OF GROUNDWATER

Chemical agent could reach groundwater from a spill on land, by the leaching of agent deposited onto land surfaces from an airborne cloud, or by recharge from contaminated surface waters (Fig. N.l). Persistence of the chemical agents in soil was discussed in Sect. N.6 and in Sect. 0.3.2.3 in Appendix 0.

In general, the impacts on groundwater from a spill of agent *on* land are likely to be negligible. Decontamination measures following the accident will reduce the concentration of agent in the soil to very low levels, so that the amount reaching an aquifer would be negligible. A discussion of measures for responding to a spill to the ground appears in the section on "Impacts to Soils" in Appendix 0.

In certain unusual situations, spilled agent might enter an aquifer before it could be contained. An example of such an unusual situation would be a case in which the agent could reach an aquifer quickly by moving through a geologic fault, through or along a well, or in which the agent was spilled into surface waters that are directly connected to groundwater. If a significant amount of agent entered the aquifer directly and if the aquifer was a water supply source with little travel time available for breakdown of the agent, serious water supply contamination could result. However, planning and mitigation measures, such as siting facilities to avoid faults, and construction of spill control devices such as dikes and berms, would greatly reduce the chance of such an unusual event occurring at storage or disposal sites.

In areas of karst terrane, where sinkholes, solution cavities, and solution channels have formed in limestone, some of the agent spilled onto ground might move through fractures in the ground and reach groundwater channels before cleanup of the spill was complete. In karst areas, water can move through underground channels and reach springs several miles away within a few hours (Quinlan 1986), so that use of the water downstream of the springs could be affected. Quinlan (1986) suggests some pre-spill actions for karst terranes. These include the identification of significant springs and the delineation and mapping of groundwater basins in the area of concern.

When spilled or deposited onto the ground, chemical agents can be expected to adsorb to soil particles to some degree. The amount of agent retained by the soil can vary greatly and depends on properties of both the soil and the agent spilled. Retention of the spilled material by soil can have two important results: the peak concentration of the agent in groundwater can be reduced, and the agent can be released into a larger volume of water. Soil retention of the spilled agent can reduce the initial peak concentration in groundwater, but also it increases the time required to flush the agent out of the aquifer.

It is difficult to estimate the degree to which the movement of agent through the aquifer is retarded by soil adsorption, or to estimate the rate at which agent adsorbed on soils above the water table is leached down to groundwater. The parameters necessary to make these

estimates have been evaluated for agents HD, VX, and GB by the Army (Small 1984), but the actual retardation and leaching rates can only be estimated for specific soil types. In general, VX is retained in soil approximately twice as much as HD, which is retained approximately twice as much as GB.

The likelihood of contamination of groundwater following an atmospheric release depends greatly on the type of agent involved. Chemical agent would be deposited onto land surfaces following an atmospheric release, and deposition would be greatest close to the accident site. Even though the amount deposited onto each unit area of soil would be small, it could be difficult to decontaminate all of the potentially large area affected. Because GB and HD have greater volatility and a more rapid rate of hydrolysis than VX, they would tend to persist on the soil surface for a shorter period of time than VX (Small 1984). (As discussed previously, if HD is below the surface of the soil, it can persist for long periods.) The greater persistence of VX on soil means that, following deposition, there is a longer time interval during which a heavy rain could occur and transport VX to groundwater. The rate of hydrolysis of agent reaching the soil will depend on the soil pH and temperature (Fig. N.3). The rate of movement through the soil into the groundwater will depend on the type and porosity of the soil and the amount of rainfall.

Because of the relatively rapid hydrolysis rate of GB and the typically slow movement of groundwater, GB that reached groundwater would be likely to decrease to very low concentrations before wells were reached. The hydrolysis products of GB are nontoxic, according to Epstein et al. (1973, p. 14). Further discussion of the toxicity of hydrolysis products of GB and other chemical agents appears in Appendix B.

The impacts of mustard on groundwater are likely to be negligible. The mustard that dissolved would hydrolyze rapidly. Any remaining mustard droplets that reached saturated soil and were moved by the flow of groundwater would be likely to dissolve slowly and then rapidly hydrolyze before reaching wells.

Contamination of groundwater by VX following deposition from a large atmospheric release would be of concern. While only very low concentrations might occur in groundwater due to dilution of the small amount of VX deposited, persistence of toxic materials would be a problem. VX has a very slow rate of breakdown by hydrolysis, and one of the breakdown products of VX is also toxic (see Appendix B) and has an even slower hydrolysis rate than VX.

The requirements for reclamation of an agent-contaminated aquifer will be site-specific. Factors influencing the choice of reclamation measures include the amount and type of agent in the aquifer, the depth to the aquifer, the geologic composition of the aquifer, the uses made of the aquifer (drinking water or irrigation), and the travel time of the groundwater to users. The Army Environmental Hygiene Agency at Aberdeen Proving Ground and the Corps of Engineers are examples of Army

agencies that have expertise In monitoring groundwater for contamination and in cleanup of groundwater.

REFERENCES FOR APPENDIX N

Agardy, F. J., and Ray, A. D. 1973. "Emergency Planning for Water Utility Management," *American Water Works Association Manual,* M19, AWWA, New York.

Bajraktarevic-Dobran, H. 1982. "Dispersion in Mountainous Natural Streams," *J. Environ. Eng. Div.,* Am. Soc. Civil *Eng.* **108**(EE3), 502-14.

Bansal, M. K. 1971. "Dispersion in Natural Streams," *J. Hydraul. Div.,* Am. Soc. Civil *Eng.* **97**(HY11), 1867-86.

Beer, T., and Young, P. C. 1983. "Longitudinal Dispersion in Natural Streams," *J. Environ. Engr.* **109**(5), 1049-67.

Bennett, S. R. et al. 1984. *Environmental Hazards of Chemical Agent Simulants,* CRDC-TR-84055, Environmental Technology Directorate, U.S. Army Armament, Munitions and Chemical Command, Aberdeen Proving Ground, Md.

Donigian, A. S., Jr., and Brown, S. M. 1983. *A Workshop on Water Modeling Needs and Available Techniques for Synfuels Risk Assessment,* prepared for Georgia Institute of Technology, Subcontract No. 1-E-26-630, Anderson-Nichols & Co., Inc., Palo Alto, Ca.

Epstein, J. 1956. "Nerve Gas in Public Water," Public Health Rap. **71**(10), 955-62.

Epstein, J. et al. 1973. *Summary Report on a Data Base for Predicting Consequences of Chemical Disposal Operations,* EASP 1200-12, Headquarters, Edgewood Arsenal, Edgewood Arsenal, Md.

Epstein, J. 1974. "Properties of GB in Water," *J. Am. Water Works Assoc.* **66**(1), 31-37.

Epstein, J., Callahan, J. J., and Bauer, V. E. 1974. "The Kinetics and Mechanisms of Hydrolysis of Phosphonothiolates in Dilute Aqueous Solution," *Phosphorus and the Related Cr. V Elements 4,* 157-63.

Geraghty, J. J., et al. 1973. *Water Atlas of the United States,* Water Information Center, Port Washington, New York.

Hodgman, C. D., ed. in chief, 1960. *Handbook of Chemistry and Physics,* 41st edition, Chemical Rubber Publishing Co., Cleveland.

Howells, G. 1983. "Acid Waters--The Effect of Low pH and Acid Associated Factors on Fisheries," *Adv. Appl. Biol. 9,* 143-255.

Hull, D. E. 1962. "Dispersion and Persistence of Tracer in River Flow Measurements," *Intemat. J. Appl. Radia. and Isotopes 13, 63-73.*

Keup, L. E. 1985. "Flowing Water Resources," Water *Resour. Bull. 21, 291-96.*

Leopold, L. B. 1962. "Rivers," Am. Sci. 50, 511-37.

Lyman, W. J., Reehl, W. F., and Rosenblatt, D. H., eds. 1982. *Handbook of Chemical Property Estimation Methods: Environmental Behavior of Organic Compounds,* McGraw-Hill, New York.

Mabey, W., and T. Mill. 1978. "Critical Review of Hydrolysis of Organic Compounds in Water under Environmental Conditions," *J. Phys. and Chem. Ref. Data 7, 383-415.*

Madema, J. 1986. "Mustard Gas: The Science of H," *Nucl.* Biol. *Chem.* Defense *Technol. Internat.*, 1, 66-71.

McQuivey, R. S., and Keefer, T. N. 1976a. "Convective Model of Longitudinal Dispersion," *J.* Hydraulics *Div., Am. Soc. Civil Eng.* 102(HY10), 1409-24.

McQuivey, R. S., and Keefer, T. N. 1976b. "Dispersion--Mississippi River Below Baton Rouge, La.," *J.* Hydraulics *Div., Am. Soc.* Civil *Eng.* 102(HY10), 1425-37.

Merkle, M. G., and Bovey, R. W. 1974. "Movements of Pesticides in Surface Water," pp. 99-106 in Pesticides in *Soil and* Water, ed. W. D. Guenzi, Soil Science Society of America, Inc., Madison, Wis.

Michels, H. O., Gordon, E. C., and Epstein, J. 1973. "Detection and Estimation of Isopropyl Methylphosphonofluoridate and O-Ethyl S-Diisopropylaminoethylmethylphosphonothioate in Seawater in Parts-per-Trillion Level," *Enviro. Sci. Technol.* 11, 1045-1049.

Mills, M. B. et al. 1982. *Water* Quality *Assessment: A Screening Procedure for Toxic and Conventional* Pollutants, Part *1,* EPA-600/6-82-004a, Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Athens, Ga.

MITRE Corporation 1987. *Transportation of Chemical Munitions at Reduced Temperature,* Report No. SAPEO-CDE-IS-87002, Program Executive Officer--Program Manager for Chemical Demilitarization, Aberdeen Proving Ground, Md.

Neely, W. B., Blau, G. E., and Alfrey, T., Jr. 1976. "Mathematical Models Predict Concentration-Time Profiles Resulting from Chemical Spill in a River," Environ. Sci. *Technol.* 10(1), 72-76.

Neely, W. B., and Lutz, R. W. 1985. "Estimating Exposure from a Chemical Spilled into a River," *J. Hazardous Materials* 10, 33-41.

Nusser, J. A., Gallagher, T. W., and St. John, J. P. 1978. "Mathematical Modeling for Impact and Control of Hazardous Materials Spills," pp. *422-26* in *Control of Hazardous Material Spills,* proceedings of the 1978 National Conference on Control of Hazardous Material Spills, Miami Beach, Fla., Information Transfer, Inc., Rockville, Md.

Quinlan, J. F. 1986. "Recommended Procedure for Evaluating the Effects of Spills of Hazardous Materials on Ground Water Quality in Karst Terranes," pp. 183-196, *Proceedings of the Environmental Problems in Karst Terranes and Their Solutions Conference,* Bowling Green, Ky., 1986, National Water Well Association, Dublin, Ohio.

Roubal, G. E., Horowitz, A., and Atlas, R. M. 1979. "Disappearance of Hydrocarbons Following a Major Gasoline Spill in the Ohio River," pp. 503-507 in *Developments in Industrial* Microbiology, Society for Industrial Microbiology, Arlington, Va.

Sanders, P. F., and Seiber, J. N. 1984. "Organophosphorus Pesticide Volatilization: Model Soil Pits and Evaporation Ponds," pp. 279-295 in Treatment and Disposal of Pesticide Wastes, eds. R. F. Krueger and J. N. Seiber, American Chemical Society, Washington, D.C.

Small, M. J. 1984. *Compounds Formed from the Chemical Decontamination of HD, GB, and VX and Their Environmental Fate,* Technical Report 8304, U.S. Army Medical Bioengineering Research and Development Laboratory, Medical Research and Development Command, Fort Detrick, Frederick, Md.

Strahler, A. N. 1964. "Section 4-II. Geology. Part II. Quantitative Geomorphology of Drainage Basins and Channel Networks," pp. 4-39 to *4-76* in *Handbook of Applied Hydrology,* ed. V. T. Chow, McGraw-Hill, New York.

Thibodeaux, L. J. 1979. *Chemodynamics: Environmental Movement of Chemicals* in Air, *Water, and Soil,* John Wiley & Sons, New York.

Thibodeaux, L. J. 1980. *Spill of Soluble, High Density, Immiscible Chemicals on Water,* DOT/RSPA/DPB-50/80/29, NTIS: PB82-237868, prepared for U.S. Department of Transportation, National Technical Information Service, U.S. Department of Commerce, Springfield, Va.

Thomann, R. V. *1972. Systems Analysis and.Water Quality Management,* McGraw-Hill, New York.

Thomas, R. G. 1982. "Volatilization from Water," Chap. 15 in *Handbook of* Chemical *Property Estimation Methods: Environmental Behavior of Organic Compounds,* eds. W. J. Lyman, W. F. Reehl, and D. H. Rosenblatt, McGraw-Hill, New York.

Trapp, R. 1985. *The Detoxification and Natural Degradation of Chemical Warfare Agents,* Stockholm International Peace Research Institute, Taylor 6 Francis, London and Philadelphia.

U.S. Department of the Army 1974. *Chemical Agent Data Sheets, Vol. 1,* Technical Report EO-SR-74001, Edgewood Arsenal Special Report, Defense Technical Info. Ctr., Alexandria, Va.

U.S. Department of the Army and U.S. Department of the Air Force 1975. *Military Chemistry and Chemical Compounds,* Field *Manual,* Army FM 3-9, Air Force AFR 355-7, U.S. Department of the Army, Headquarters, Washington, D.C.

U.S. Environmental Protection Agency 1985. *EPA Chemical Profile: Mustard Gas,* October 31, 1985.

Weast, R. C., ed. 1979. *CRC Handbook of Chemistry and Physics,* 60th edition, CRC Press, Boca Raton, Fla.

Wetzel, R. G. 1975. *Limnology.* W. B. Saunders Company, Philadelphia.

Windholz, M., ed. 1976. The Merck *Index,* 9th ed., Merck 6 Co., Inc., Rahway, N.J.

Windholt, M., ed. 1983. *The Merck Index,* 10th ed., Merck & Co., Inc., Rahway, N.J.

Appendix 0

TOXICOLOGY AND POTENTIAL IMPACTS OF CHEMICAL AGENTS TO ECOLOGICAL RESOURCES

O.1 INTRODUCTION

This appendix describes the potential impacts that disposal of chemical agents might have on ecological resources. These resources include animals and fish, vegetation, soils, and land use. It also describes two models that were developed to estimate the toxicity of chemical agents to fish and birds.

Other than the destruction of habitat for construction and operation of disposal facilities and the generation of dry process wastes requiring disposal, no impacts are expected from normal operations. Each site would require about 4 ha (11 acres); small animals in this area would be displaced or killed. Since the disposal sites are fenced, no impacts on large animals are expected. For both conventional and nerve agent pollutants, predicted ground-level concentrations from normal operations are, at the most, less than 10% of the standards set by federal and state agencies and DOD (see Table 4.1.3). At these concentrations, no effects of emissions on wildlife, vegetation, or the environment are expected. A recent study (Crook et al. 1983) showed that concentrations of VX that were 625 times greater (0.000005 mg/m^3) than those proposed in the EIS (0.000000008 mg/m^3) caused no effects on mice, rats, guinea pigs, or rabbits over a ten-day period of exposure. Data from McNamara et al. (1975) for mustard gas toxicity showed that exposure of dogs, rabbits, guinea pigs, rats, and mice to a concentration of 0.001 mg/m^3 for 24 h/d, 5 d/week for one year did not produce detectable damage, either systemic, local, pathological, mutagenic, teratologic, or carcinogenic. The dose was equivalent to a concentration of 0.003 mg/m^3 for 8 h/d. This concentration is 150 times greater than the maximum predicted ground-level concentrations for mustard (0.00002 mg/m^3) (see Table 4.1.3).

In general, the impacts of accidents involving chemical agents on aquatic and terrestrial biota are multifaceted and potentially significant. Spills into surface water bodies and aerial deposition cannot be readily contained and could affect aquatic biota. The magnitude of the adverse impacts would depend on (1) the agent involved, (2) the proximity of the accident to a water body, (3) the amount of agent volatilized and subsequently deposited, (4) the volume and flow of the water bodies receiving the agent, (5) the meteorological conditions at the time of the accident, and (6) the existing aquatic biota within the affected waters.

For terrestrial resources, effects in the potential impact zones as the result of aerial dispersion and deposition could be both direct and indirect. Direct effects on animals could result from inhalation; licking; absorption through the skin, nose, and eyes; and ingestion of contaminated vegetation or other foods. Indirect effects result from loss of habitat and loss of food. In addition, the loss of insect pollinators might interfere with crop production.

Numbers of protected ecological resources within 20-, 50-, and 100-km radii of existing sites are given in Table 3.2.7. Resources in the corridors around the transportation routes are given in Table 3.3.4. Accident scenarios for the sites are discussed in Sect. 4 in the appropriate sections for each site and Appendix K. Calculations of toxicity and impacts to various ecological resources are presented in this appendix to provide a means of assessing the impacts of the different alternatives to protected resources and other existing resources in the vicinity of the current storage sites and transportation routes (Sect. 4.3).

0.2 AQUATIC RESOURCES

0.2.1 TOXICITY

A method described in Appendix II-A of the EPA "Blue Book" (NAS/NAE 1973) was used to estimate the effects on aquatic organisms of a pulse of nerve agent moving down a river. This method was originally developed to estimate the impacts on fish from exposure to varying temperatures. The model effectively integrates the concentration-time exposure at a given distance downstream, using information on how median survival time (LT_{50}) depends on concentration. The median survival time, or median lethal time, is the time required to kill half of the organisms exposed to a given concentration of agent. A major assumption of this method is that, at a given exposure level, each time unit of exposure contributes equally toward a lethal exposure. For example, if half the fish exposed to 0.1 ppm GB die in 80 min, then it is assumed that each 10-min period of the exposure contributes equally toward mortality. A second major assumption is that these contributions can be summed across different exposure levels. For example, 50% mortality of the exposed fish would be expected after (1) 40 min of exposure to a concentration with an 80-min LT_{50}, followed by (2) 400 min of exposure to a concentration with an 800-min LT_{50}.

The time when 50% mortality will occur for exposures to variable toxicant concentrations can be estimated given these assumptions and the relationship between LT_{50} and exposure concentrations. This approach was used successfully to predict the time of 50% mortality of brook trout exposed to high water temperatures, based on survival-time experiments with different constant-temperature exposures (Fry et al. 1946). For the spill events analyzed here, the concentrations along the plume of nerve agent passing a given point downstream were estimated with the aquatic-spill model described in Appendix N.

To determine the potential effects of deposition or spills of chemical agents on aquatic organisms, equations for the dependence of the median survival time (LT_{50}) on the concentration of the chemical nerve agents GB and VX in water were developed based on the laboratory toxicity tests of Weiss and Botts (1957) and Weimer et al. (1970), respectively. Median survival time, or median lethal time, is the time

required to kill half the organisms exposed to a given concentration of agent.

For the analysis of aquatic impacts from a spill *or* deposition of nerve agent, it is assumed that the LT_{50} for exposure to GB (at low concentrations) is adequately described by the equation

$$\log(LT_{50}) = a - b \log(C) , \qquad (O.1)$$

where C is the nerve agent concentration (ppm) in water; LT_{50} is the corresponding median survival time, or median lethal exposure (min); and a and b are constants. For goldfish exposure to GB a = 0.84 and $b = 1.42$; for striped bass exposure to VX, a = 1.52 ± 0.06 and $b = 0.63 \pm 0.07$.

The aquatic-spill model (Appendix N and Sect. 4.2.2.3) was used to estimate the average nerve-agent concentration at successive time intervals (e.g., every 0.1 h) for a given distance downstream (x), and Eq. 0.1 was used to estimate the LT_{50} for the average concentration during that interval. These contributions toward a lethal exposure were summed as follows:

$$S_x = \sum_{t=t_1}^{t2} \frac{\Delta t}{(LT_{50} \text{ at } x,t)},$$

where S_X is the dimensionless sum of partial contributions toward a median lethal exposure obtained at distance x, and t is the time interval over which each exposure is assumed to be constant. The summation is done from time $t1$ to $t2$, where tl and $t2$ are chosen to bound the time interval during which the pulse passes the point at distance x.

In this model, if S_X is equal to 1.0, then 50% mortality is expected to occur at distance x in response to the pulse exposure passing that point. If S_X is less than 1, then less than 50% mortality would be expected. Conversely, if S_X is greater than 1, then greater than 50% mortality would be expected. The evaluation distance (x) can then be varied until the distance is found at which S_X is exactly 1.0. The results of these calculations are presented in Appendix 0, Sect. 0.2.2.

Results from the laboratory toxicity tests of Weiss and Botts (1957) using agent GB were used to develop an equation for the dependence of the median survival time (LT_{50}) on the concentration of GB in the water. An equation for VX was developed from information in Weimer et al. (1970).

For a given deposition value, the nerve agent concentration in a 1-m-deep pond was computed. For a spill, the concentration at successive time intervals for a given distance downstream (x) was estimated. Equation (0.1) was used to estimate the LT_{50} at these concentrations for selected aquatic organisms. The toxicity calculations used laboratory data on fish survival time for different concentrations of GB (Weiss and Botts 1957). Existing information (Weiss 1955; McNamara and Leitnaker

1971; Wilber 1954) was used to extrapolate from values calculated for fish species to those for other aquatic species.

0.2.2 IMPACTS TO AQUATIC RESOURCES

Predictions of effects of both deposition and spills on aquatic biota were made, based on the limited information available on the effects of chemical agents on aquatic biota. These predictions were made for different volumes of agent spilled or deposited into water bodies with different sizes/flows.

0.2.2.1 Aerial Deposition

A spill of approximately 5 kg of chemical agent (an amount equivalent to the contents of one M55 rocket) onto the ground under conservative, most-likely meteorological conditions (Sect. 4.2.2) could have an effect on aquatic biota, due to volatilization and transport through the atmosphere, within a downwind distance of 1.0 km. Because of the low volatility of VX (Bennett et al. 1984), downwind effects to aquatic biota from a 5-kg spill onto the ground would be negligible. Under worst-case meteorological conditions (Sect. 4.2.2), deposition of 5 kg of agent aerosolized from a spill could occur for a maximum distance of 3.0 km downwind for GB and a negligible distance downwind for VX. The downwind response distances for GA should be similar to those for GB; downwind response distances for the H-agents (mustard) and lewisite should be similar to those for VX. Aquatic biota within these distances could be adversely affected by volatilization and deposition from a spill.

0.2.2.2 Spill of Agent into Rivers

The estimated maximum concentrations of chemical nerve agents in water, if a spill of approximately 5 kg of agent were to occur into rivers of five different sizes, are given in Tables 0.1 and 0.2. The method used to estimate these concentrations is discussed in Appendix N. Also shown in these tables are median survival times of fathead minnows or striped bass at the concentrations and water temperatures given; these LT_{50} values are computed with the equation and the temperature adjustments described in Sect. 0.2.1.

In a very large river such as the Mississippi (10th-order) (see Appendix N, Table N.1), sufficient dilution of 5 kg of chemical agent would occur after a short time so that fish as sensitive as fathead minnows or striped bass could survive for many days even at the maximum concentration of the moving plume (Tables 0.1 and 0.2). Any impacts on aquatic organisms would likely be confined to the immediate vicinity of the spill or deposition and would be minimal further downstream. Fish would not likely receive a median lethal exposure, except perhaps at the spill or deposition site.

Table 0.1. Estimated maximum concentrations of agent CB in a river after a spill of 5 kg of agent, for three loss rates and for rivers of different sizes

For each temperature and pH combination, the estimated half-life ($t_{1/2}$)[a] caused by losses from hydrolysis and volatilization is given. Also shown is the estimated median survival time (LT_{50}) for fathead minnows at that concentration

River size[b]	Time after spill	0°C, pH 7.5		4°C, pH 7.5		20°C. pH 7.5	
		Concentration (ppm)	Minnow LT_{50}	Concentration (ppm)	Minnow LT_{50}	Concentration (ppm)	Minnow LT_{50}
Very small (1 St-order)	IO min	2200	<0.1 min	2200	<0.1 min	2200	<0. I min
	60 min	860	<0.1 min	860	<0.1 min	850	<0.1 min
	180 min[c]	830	<0.1 min	730	<0.1 min	720	<0.1 min
Small (4th-order)	IO min	9.0	1.2 min	9.0	I.2 min	8.0	0.3 min
	90 min	3.0	4.1 min	3.0	4.1 min	3.0	1.1 min
	120 min	2.0	5.1 min	2.0	5.1 min	2.0	1.3 min
	180 min	1.9	6.4 min	1.9	6.4 min	1.9	I.7 min
	5 h	I.6	8.0 min	1.6	8.0 min	1.5	2.1 min
	7 h	1.3	10.0 min	1.3	10.0 min	1.0	2.8 min
	10 h	1.1	12.5 min	1.1	12.5 min	0.9	3.6 min
Medium (6th-order)	IO min	0.2	78.5 min	0.2	78.5 min	0.2	19.7 min
	108 min	0.06	5.1 h	0.06	5.1 h	0.06	78.8 min
	180 min	0.05	7.2 h	0.05	7.2 h	0.05	1.9 h
	6 h	0.04	10.1 h	0.04	10.1 h	0.03	2.8 h
	11 h	0.03	14.2 h	0.03	14.3 h	0.02	4.2 h
	20 h	0.02	20.2 h	0.02	20.5 h	0.01	6.9 h
	35 h	0.01	28.9 h	0.01	29.7 h	0.009	12.7 h
Large (8th-order)	IO min	0.006	3.1 d	0.006	3.1 d	0.006	18.9 h
	2 h	0.002	13.9 d	0.002	13.9 d	0.002	3.6 d
	5 h	0.001	22.0 d	0.001	22.1 d	0.001	6.0 d
	11 h	0.0007	35.2 d	0.0007	35.5 d	0.0006	10.6 d
	25 h	0.0005	56.6 d	0.0005	57.7 d	0.0004	21.3 d
Very large (10th-order)	10 min	0.0003	118.9 d	0.0003	118.9 d	0.00025	29.8 d
	3 h	0.00006	591.1 d	0.00006	592.4 d	0.00006	154.6 d
	8 h	0.00004	2.9 years	0.00004	2.9 years	0.00003	300.2 d

[a] ($t_{1/2}$) is the estimated time at which only one-half of the original calculated concentration remains.

[b] See Appendix N, Table N.1 for explanation of river sizes.

[c] At this length of time, the plume would be approximately 2.6 km (I mile) downstream of the spill site. This distance would be the estimated maximum distance before confluence with another stream and subsequent dilution.

Table 0.2. Estimated maximum concentrations of agent VX in a river after a spill of 5 kg of agent, for three **loss rates and for rivers of different sizes**

For each temperature and pH combination, the estimated half-life ($t_{½}$)[a] caused by losses from hydrolysis and volatilization is given. The estimated median survival time (LT_{50}) for fathead minnows is also given

River size[b]	Time after spill	4°C, pH 7.5		25°C, pH 7.5	
		Concentration (ppm)	Minnow LT_{50}	Concentration (ppm)	Minnow LT_{50}
Very small (1st-order)	10 min	2200	1.0 min	2200	0.3 min
	60 min	870	1.9 min	870	0.5 min
	108 min	750	2.1 min	740	0.5 min
Small (4th-order)	10 min	8	34.5 min	8	8.6 min
	108 min	3	68.4 min	3	17.1 min
	120 min	2	77.1 min	2	19.3 min
	5 h	1.6	97.9 min	1.6	24.6 min
	7 h	1.3	1.8 h	1.3	27.8 min
	10 h	1.1	2.1 h	I.1	31.4 min
Medium (6th-order)	10 min	0.2	5.8 h	0.2	87.5 min
	108 min	0.07	12.4 h	0.06	3.1 h
	3 h	0.05	14.9 h	0.05	3.7 h
	6 h	0.04	17.9 h	0.04	4.5 h
	11 h	0.03	21.6 h	0.03	5.5 h
	20 h	0.02	26 h	0.02	6.6 h
	35 h	0.015	31.4 h	0.01	8.1 h
Large (8th-order)	10 min	0.006	2.3 d	0.006	13.7 h
	134 min	0.002	5.2 d	0.002	31.2 h
	5 h	0.001	6.7 d	0.001	40.3 h
	11 h	0.0007	8.6 d	0.0007	2.2 d
	25 h	0.0005	11.1 d	0.0005	2.9 d
	57 h	0.0003	14.3 d	0.0003	3.8 d
	127 h	0.0002	18.5 d	0.0002	5.3 d
Very large (10th-order)	10 min	0.0003	17.0 d	0.0003	4.3 d
	166 min	0.00006	41.2 d	0.00006	10.3 d
	8 h	0.00004	56.8 d	0.00004	14.3 d
	21 h	0.00002	78.2 d	0.00002	20.0 d

[a] ($t_{½}$) is the estimated time at which only one-half of the original calculated concentration remains.

[b] See Appendix N, Table N.l for information on river sizes.

In large rivers such as the Arkansas at maximum flow or the Columbia River (8th-order streams, Appendix N, Table N.l) at lo-year low flow, dilution would be enough so that fish could survive for several hours at the maximum concentration of the plume. The plume would be expected to pass a fixed location in a sufficiently short time and *to* have a sufficiently dilute concentration as the result of mixing so that fish would not accumulate a median lethal exposure, even as close *to* the spill site as a few hundred meters downstream (given the assumption of very rapid vertical and horizontal mixing).

A spill of 5 kg of nerve agent GB has the potential to cause fish kills in medium-sized rivers such as the Salt River in Kentucky (6th-order river, Appendix N, Table N.l) and smaller rivers such as Village Creek (4th-order, Appendix N, Table N.l) near Birmingham, Alabama, and the Umatilla River in Oregon (4th-order, Appendix N, Table N.l). As seen in Tables 0.1 and 0.2, fish could survive only a few minutes in a smaller river at the peak concentrations. Minnows in lst- through 6th-order rivers (small to medium-sized) could accumulate a median lethal exposure of GB at a water temperature of 0°C and a pH of 7.5. Sect. 0.2.1 discusses and Table 0.3 shows the estimated point at which 50% of exposed minnows would die. Upstream from this point, more than 50% of exposed minnows would probably die; downstream, less than 50% of exposed minnows would die, according to these calculations. The rate of hydrolysis would affect downstream agent concentrations; at higher temperatures the hydrolysis rate would be faster and the downstream agent concentrations of chemical agent would be lower. However, the warmer temperature also reduces the survival time of the fish (Weiss and Botts 1957); the net result is that minnows could potentially receive a median lethal exposure within the same-order stream. at a greater downstream distance.

If a spill occurred into a river as small as or smaller than 4th-order, fathead minnows could not survive in the peak concentrations of GB for more than a few minutes, and median lethal exposures would be accumulated by minnows at a distance at least as great as 12 km downstream.

Increased flow from tributary input would dilute the concentration of these chemical agents and would tend to mitigate these downstream effects. Similarly, the junction of a smaller river with a larger one would speed the dilution, thereby decreasing the distance (Appendix N) at which toxic effects could occur to aquatic biota. For example, the Arkansas River joins the Mississippi River about 160 km (100 miles) downstream from Pine Bluff Arsenal, and dilution would be accelerated beyond that junction. This simplified analysis assumes that the river shape and flow are uniform downstream. Long residence times are shown merely to indicate the potential for long persistence of the agents in water.

The impact on the aquatic resources could be greatest in rivers up to 6th-order (medium-sized, Appendix N, Table N.l). There, the dilution capacity is less than that of a larger river, but the fish population

Table 0.3. Fraction of tbe fish population killed as tbe result of a spill of S kg of nerve agent into different sized rivers

Stream length (mile)	Stream order	GB		V X	
		4°C, pH = 7	20°C, pH = 7	4°C, pH = 7.5	20°C, pH = 7.5
I	I	> 50%[a]	>50%	>50%	>50%
2.3	2	>50%	> 50%	50% at 0.6 km[c]	>50%
5.3	3	>50%	>50%	<50%	>50%
12	4	>50%	>50%	<50%	50% at 4 km'
28	5	<50%[b]	>50%	<50%	<50%
64	6	<50%	<50%	<50%	<50%
147	7	<50%	<50%	<50%	<50%
338	8	<50%	<50%	<50%	<50%
777	9	<50%	<50%	<50%	<50%
1800	10	<50%	<50%	<50%	<50%

[a] Greater than 50% of the fish population would be killed.

[b] Less than 50% of the fish population would be killed.

[c] Less than 50% mortality closer to the spill site and greater than 50% mortality to the end of this stream order.

size is typically greater than that of smaller rivers or streams.

The results of these calculations indicate that a spill of a chemical nerve agent directly into surface waters has the potential to significantly affect aquatic organisms, especially in 6th-order or smaller rivers (see Table 0.3). Based on data for streams in Kansas (Brunson 1980), the biomass of fish, including all species present, that could be killed by a spill of 5 kg of chemical agent would be 15 kg in a first-order stream, 111 kg in a second-order stream, 252 kg in a third-order stream, and 3,000 kg in a fourth-order stream. From table 0.3 it can be seen that mortality would occur further downstream at higher temperature (20°C) for agent GB and only as far downstream as third-order streams for agent VX. In shallow ponds or lakes within the area receiving the highest deposition of chemical agent from an accident involving an airborne release, one-half or more of the fish that are as sensitive as fathead minnows or striped bass might be killed within approximately one week by dissolved chemical agent from a 5-kg spill.

Under conservative, most-likely meteorological conditions, the maximum downwind distance to the no-effects, human-health isopleth for the chemical agent volatilized from a spill of 500 kg of agent would be less than 10 km for agents GB and VX (see Appendix K). Aquatic biota in surface water bodies receiving deposition of volatilized agent could be adversely affected. The magnitude of the effects would depend on the amount of deposition, the temperature, and the flow of the water body that would determine the exposure to individuals.

During worst-case meteorological conditions, the most severe release studied would adversely affect aquatic biota within a maximum downwind distance of 50 km for agents GB and VX (see Appendix K). As for effects under most-likely meteorological conditions, the distance and duration of the effects to aquatic biota from an accident under worst-case meteorological conditions would depend on the size of the water body receiving the spilled agent and deposition, the water flow and temperature, and the amount of deposition.

The estimated maximum concentrations of agent in water are given in Tables 0.4 and 0.5 for a spill of 500 kg of chemical agent into rivers of five different sizes. The values in these tables provide an approximation of the maximum effect on fish populations; uncertainties such as the rate of agent release and fluctuations in river flow would influence these estimated lethal concentrations. The method used to estimate these concentrations is discussed in Sect. 0.2.1. Also shown in these tables are median survival times of fathead minnows or striped bass if they were to remain at the given peak concentration and water temperature; these LT_{50} values are computed with the equation and temperature adjustments described in Sect. 0.2.1. The toxicity of agents to fathead minnows and striped bass are used as indicators of overall aquatic toxicity in the following discussion.

In very large rivers such as the Mississippi (10th-order), the spilled agent would be so diluted that fish as sensitive as minnows or striped bass would suffer no acute toxic effects after dispersion of the

Table O.4. Estimated maximum concentration of agent GB in a river after a spill of 500 kg of agent, for three loss rates and for rivers of different sizes

For each temperature and pH combination. the estimated half-life ($t_{1/2}$) caused by losses from hydrolysis and volatilization is given. Also shown is the estimated median survival time (LT_{50}) for striped bass at that concentration

River size[a]	Time after spill	0°C. pH 7.5		4°C. pH 7.5		20°C. pH 7.5	
		Concentration (ppm)	Minnow LT_{50}	Concentration (ppm)	Minnow LT,	Concentration (ppm)	Minnow LT_{50}
Very small (Ist-order)	IO min	220.000	<0.1 min	220,000	<0.1 min	220.000	<0.1 min
	72 mm	80.000	<0.1 min	80,000	<0.1 min	79,000	<0.1 min
	84 min	75.000	<0.1 min	75,000	<0.1 min	74.000	CO.1 mm
	108 min	73,000	<0.1 min	73.000	<0.1 min	72,000	<0.1 min
Small (4th-order)	10 min	850	<0.1 min	850	<0.1 min	840	CO 1 mm
	180 min	240	<0.1 mm	240	<0.1 min	280	<0.1 min
	3h	190	<0.1 mm	190	CO.1 min	185	CO.1 min
	5h	160	<0.1 min	160	CO.1 min	150	CO.1 min
	7h	130	CO.1 min	130	0.1 min	120	<0.1 min
	IO h	100	<0.1 min	100	0.1 min	90	CO.1 min
Medium (6th-order)	10 min	21	0.4 min	20	0.4 mm	20.0	0.1 min
	108 min	7	I .6 mm	6	1.6 min	6.0	0.4 min
	3h	5	2.3 min	5	2.3 min	5.0	0.6 mm
	6h	4	3.2 min	4	3.2 min	3.0	0.9 mm
	I I h	3	4.5 min	3	4.5 min	2.0	I.3 min
	20 h	2	6.3 mm	2	6.4 min	I.5	2.2 min
	35 h	I	9.1 min	I	9.4 min	0.9	4.0 min
Large (8th-order)	IO mm	0.6	23.8 min	0.6	23.8 min	0.6	6.0 min
	2 h	0.2	1.7 h	0.2	I.7 h	0.2	21.2 mrn
	5h	0.1	2.8 h	0.1	2.8 h	0.1	45.2 h
	II h	0.07	4.4 h	0.07	4.5 h	0.06	79.8 min
	I d	0.05	7.1 h	0.05	7.3 h	0.03	2.1 h
	2d	0.03	I I.7 h	0.03	12.2 h	0.0 I	1.3 h
	5.6 d	0.02	20.0 h	0.02	22.1 h	0.003	39.3 h
Very large (10th-order)	10 min	0.03	15.0 h	0.03	15.0 h	0.03	3.8 h
	3h	0.006	3.1 d	0.006	3.1 d	0.006	19.5 h
	8 h	0.004	5.6 d	0.004	5.6 d	0.003	37.8 h
	20 h	0.002	I0.I d	0.002	10.2 d	0.002	3.5 d
	60 h	0.001	18.7 d	0.001	19.6 d	0.0006	I2.I d
	7d	0.0007	36.9 d	0.0006	42.0 d	0.00007	127.3 d

[a] ($t_{1/2}$) is the estimated time at which only one-half of the original calculated concentration remains.
[b] See Appendix N, Table N.1 for information on river sizes.

Table 0.5. Estimated maximum concentrations of agent VX in 8 river after 8 spill of 500 kg of agent, for two loss rates and for *rivers* of different sizes

For each temperature and pH combination, the estimated half-life ($t_{1/2}$)[a] caused by losses from hydrolysis and volatilization is given. The estimated median survival time (LT_{50}) for striped bass is also given

River size[b]	Time after spill	4°C, pH 7.5		25°C, pH 7.5	
		Concentration (ppm)	Striped bass LT_{50}	Concentration (ppm)	Striped bass LT_{50}
Very small (lst-order)	10 min	220.000	0.1 min	220,000	<0.1 min
	60 min	87,000	0.1 min	87,000	<0.1 min
	10% min	75,000	0.1 min	74,000	<0.1 min
Small (4th-order)	10 min	850	1.9 min	850	0.5 min
	10% min	290	3.8 min	290	0.9 min
	120 h	240	4.2 min	240	1.1 min
	3h	200	4.8 min	190	1.2 min
	5 h	160	5.4 min	160	1.4 min
	7h	130	6.1 min	130	1.5 min
	10 h	110	6.8 min	110	1.7 min
Medium (6th-order)	10 min	20	19.2 min	20	4.8 min
	10% min	7	40.7 min	7	10.2 min
	3h	5	49.1 min	5	12.3 min
	6h	4	59.1 min	4	14.9 min
	11 h	3	71.3 min	3	18.0 min
	20 h	2	85.8 min	2	21.9 min
	35 h	1	1.7 h	1	26.8 min
Large (%th-order)	10 min	0.6	3.0 h	0.6	45.2 min
	134 min	0.2	6.8 h	0.2	1.7 h
	5 h	0.1	8.8 h	0.1	2.2 h
	11 h	0.07	11.4 h	0.07	2.9 h
	25 h	0.05	14.7 h	0.05	3.8 h
	56 h	0.03	18.9 h	0.03	5.0 h
	127 h	0.02	24.4 h	0.02	6.9 h
Very large (10th-order)	10 min	0.03	22.4 h	0.03	5.6 h
	166 min	0.006	2.3 d	0.006	13.6 h
	8 h	0.004	3.1 d	0.004	18.9 h
	21 h	0.002	4.3 d	0.002	26.4 h

[a] ($t_{1/2}$) is the estimated time at which only one-half of the original calculated concentration remains.
[b] See Appendix N, Table N.l for information on river sizes.

plume. Impacts would likely be limited to the immediate vicinity of the spill or deposition and would be minimal downstream.

For a spill of this volume of chemical agent, effects on aquatic biota would occur in rivers up to and including 8th-order rivers such as the Arkansas at maximum flow; acute toxic effects from exposure to the plume could occur for considerable distances downstream. Depending on the time required for the plume to pass a fixed location, as well as on the concentration of the plume, fish in a river of this size could accumulate a median lethal exposure from GB as far from the spill site as several hundred meters downstream (given the assumption of very rapid vertical and horizontal mixing) (Table N.l).

A spill of 500 kg of agent GB or VX has the potential to cause a large number of fish to be killed over a considerable area in medium-sized and smaller rivers. As seen in Tables 0.4 and 0.5, fish could survive only a few minutes in these rivers at the peak concentrations. The downstream distance at which a median lethal exposure would occur was calculated according to the technique described in Sect. 0.2.1. In a medium-sized river, minnows could accumulate a median lethal exposure to GB at >100 km downstream at a water temperature of 0°C and a pH of 7.5. This represents the estimated exposure at which 50% of the exposed minnows would die (Sect. 0.2.1). This calculation gives an indication of the relative distance over which toxic effects could occur in rivers with little capacity to dilute the agent. Upstream more than 50% of exposed minnows would probably die; downstream, less than 50% of exposed minnows would die, based on these calculations. At 4°C and pH 7.5, the increased hydrolysis rate would reduce concentrations. In warmer water (20°C, pH 7.5), the hydrolysis rate would be even faster and the resulting concentrations of chemical agent downstream from the spill site even lower. However, the warmer temperature also reduces the survival time of the fish (Weiss and Botts 1957). The net result is that minnows could potentially receive a median lethal exposure over 200 km downstream. In the smallest river size analyzed (4th-order), fathead minnows could not survive in the peak concentrations for more than a few minutes, and median lethal exposures would be accumulated by minnows at the downstream end of this stream.

Tables 0.4 and 0.5 give the estimated time to kill 50% of the fish exposed to concentrations that could result from the different releases studied and from deposition of agent from an airborne plume (see Sect. 4.2.2.1 and Appendix K). These median lethal times (LT_{50}) have relatively large (but unknown) uncertainties associated with them; at the lower concentrations they represent extrapolations beyond the lowest concentration measured in laboratory toxicity experiments. It is possible that organisms may be able to adapt to or repair the slight damage caused at very low chemical agent concentrations (Mancini 1983; Connolly 1985; Banas and Sprague 1986), in which case the toxic effects described would be overestimated for very low concentrations. For the purposes of the assessment, conservative assumptions were made that adaptation or repair would not occur and that fish exposed to low

concentrations of agent would exhibit responses proportional to those measured in laboratory studies.

Weiss and Botts (1957) measured the LT_{50} of fathead minnows, green sunfish, and goldfish for GB concentrations ranging from about 0.01 ppm (10 ppb) to 60 ppm. Weimer et al. (1970) exposed blue crabs (*Callinectes* sapidus) to VX concentrations ranging from 10 to 300 ppm, striped bass (*Horone saxatilis*) to concentrations from 0.02 to 5 ppm VX, and white perch (Horone americanus) to concentrations from 0.025 to 10 ppm VX, all at 22°C. The large LT_{50} value measured for striped bass at the lowest VX concentration (i.e., LT_{50} = 17.4 h at 0.02 ppm VX) (Weimer et al, 1970), which was omitted in the regression that yielded the equation in Sect. 0.2.1, suggests that extrapolations beyond the lowest values measured in the laboratory might be too conservative (i.e., the true toxicity at very low concentrations might be much less than these estimates). Further toxicity tests involving long (several-day) exposures to very low concentrations (less than 0.02 ppm VX) are needed to resolve this issue. Nonetheless, these calculations suggest that deposition of chemical agent from the denser areas of an airborne cloud has the potential to cause mortality of at least some fish in shallow standing water (e.g., ponds and shallow lakes).

In toxicity tests with rainbow trout, Marking and Mauck (1975) have shown that the effects of mixtures of organophosphorus pesticides are approximately additive. The impacts of a spill of chemical agent, which are similar in nature and effect, on aquatic organisms could be greater than those described above if the water had recently contained other organophosphate compounds (such as the insecticides malathion, parathion, chlorpyrifos, demeton, and azinphosmethyl) at concentrations that reduce acetylcholinesterase (AChE) levels in the organisms. Coppage and Braidech (1976) measured several organophosphate pesticides from Missouri River water collected downstream from a pesticide manufacturer. The AChE activity of the brain was found to be lower in fish that were caught downstream of the plant than in fish caught upstream. Fish exposed to such compounds (perhaps after a storm that produces agricultural runoff) may have depressed AChE levels for many days (Thirugnanam and Forgash 1977). In that case, the organisms would be more susceptible to the additional effects of chemical nerve agents, and depression of AChE to lethal levels could be caused by lower-than-calculated concentrations of agents in the water.

The toxicity of mustard agents to aquatic organisms is unknown; however, these agents, like VX, are persistent and hydrolyze slowly in water. Bauer et al. (1955) attempted field bioassays of fish with HD but were unsuccessful because "at the low water temperatures, the agents solidified and insufficent quantities dissolved in the water." The dominant hydrolysis product of HD is thiodiglycol (Small 1964); no aquatic toxicity data were found for this chemical--but it has been shown to have a low toxicity to several terrestrial species (Sax 1979). Mustard agents, however, are quite reactive and may form a number of other compounds while decomposing in the environment. The toxicity and

persistence of these other compounds are generally unknown, and some may be as toxic as the parent compound, HD. Until toxicity data on the effects of mustard agents on aquatic biota are available, a conservative assumption must be made that the actions of these agents on aquatic biota are similar to those of VX.

0.3 TERRESTRIAL RESOURCES

0.3.1 TOXICITY TO TERRESTRIAL RESOURCES

Only limited toxicity data are available for effects on laboratory animals, birds, and insects; potential impacts to wildlife must be inferred from assessment of these data. Data on chronic toxicity, potential teratogenicity, and reproductive effects are described in Appendix B. Because so few data are available on the effects of absorption and uptake of agents by plants and subsequent ingestion by animals, these modes of action cannot be quantitatively assessed. Nevertheless, a data base of toxicities (LD_{50}) of pesticides to terrestrial vertebrates was created and subsequently a statistical model was developed that related the toxicity of these materials to birds that may be exposed in the field in the event of a release of chemical agent. The model is based on the premise that organophosphorous chemical agents (e.g., GB and VX) are similar to organophosphorous pesticides in chemical structure, biological activity, or both. A word of caution is appropriate, however; according to Ross et al. (1983), some reports indicate that results obtained with one particular organophosphate may not be applicable to other organophosphates and also that results from organophosphate poisoning may not be parallel to results from chemical agent poisoning.

Tests show that all agents can cause the death of laboratory animals as the result of various modes of uptake. Figure 0.1 illustrates inhalation toxicity data and Table 0.6 lists ingestion toxicity data for the various agents; data for other exposure routes are found in Appendix B. The data suggest that birds and insects may be particularly sensitive. In addition to direct losses of large animals as a result of inhalation and absorption of agents, evidence exists that plants absorb and retain the chemicals and their breakdown products; both may be lethal to animals grazing for sustained periods (Cameron and Schmitz 1973; McNamara 1972; Ross et al. 1983). Hay and corn exposed to agents and subsequently harvested for animal food could also be toxic. Cattle, horses, dogs, and wildlife appear to be less susceptible to ingested VX-contaminated vegetation than sheep (U.S. Congress 1969); however, very little information is available with which to substantiate the statement. There is no evidence that chemical agents are accumulated in food chains. The effects of HD (or H or HT) on wildlife are either unavailable or unknown. Some studies on the effects of mustard agent on laboratory animals, insects, fungi, bacteria, and plants are planned. These studies and other existing information are listed in Tables B.2

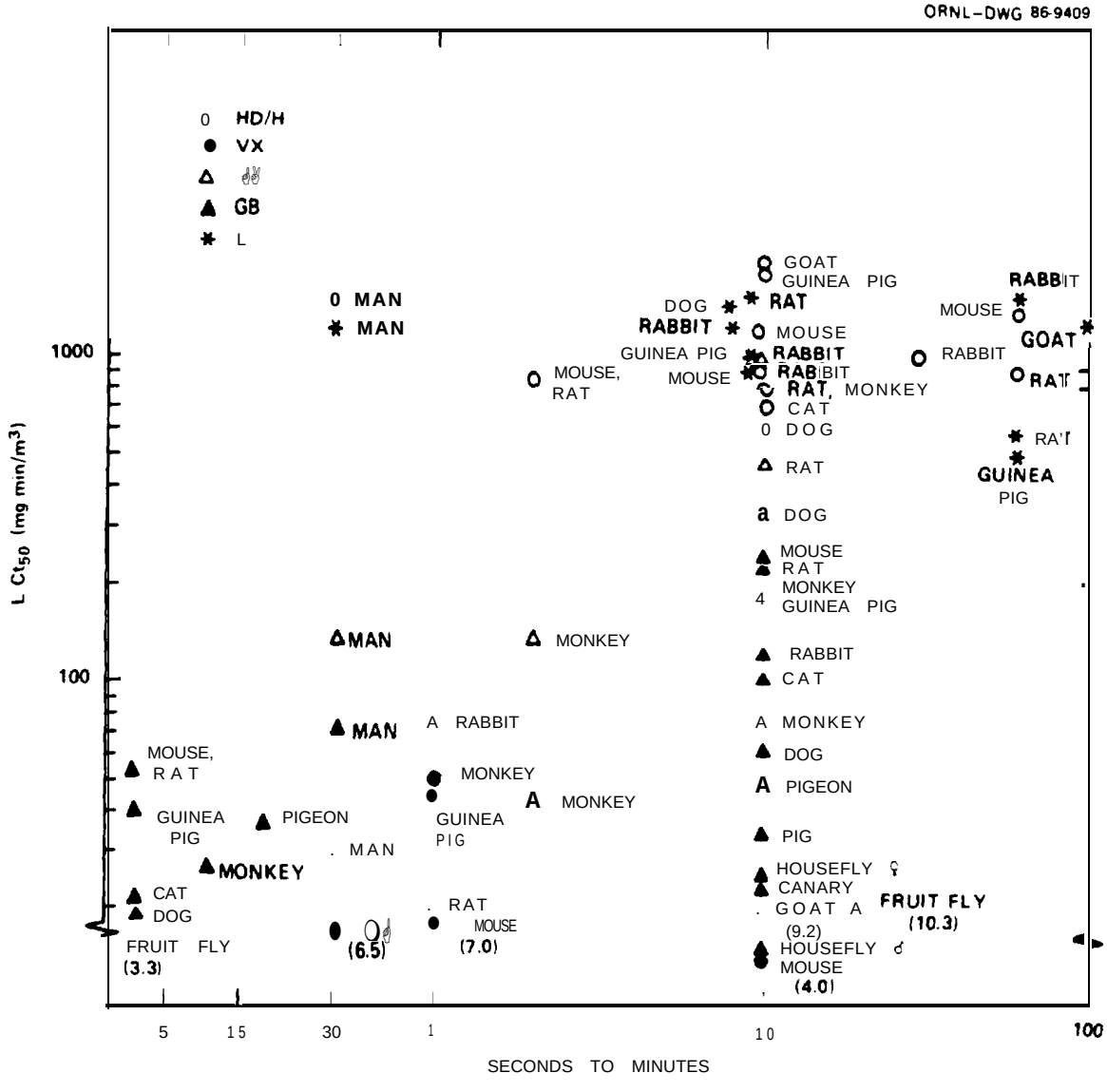


Fig. 0.1. Variation of LCt_{50} inhalation data with exposure time in various species exposed to HD/H, 0; VX, ●; CA, A; GB, ▲; L, *. There were no toxicity data for HT. Values for man are estimates. Source; U.S. Army 1974; McNamara and Leitnaker 1971; Bennett et al. 1984.

Table 0.6. Acute toxicity data (LD_{50})[a] for ingestion of chemical agents by test animals

species	Agents (mg/kg)				
	GB	VX	HD	H	L
Rat	0.8706	0.100[b]	17[c]	16[d]	50[e]
Rabbit	2.50[b]	0.123[c]		40[d]	
Mouse		0.250[f]	92[g]		
Guinea pig				45[d]	
Sheep		0.030[h]			
Young steer		0.026[h]			

[a]Lethal dose in mg of agent per kg of animal weight to 50% of the animals in the test group.
[b]Owens et al. 1973.
[c]Hackett et al. 1985.
[d]Boyland 1944.
[e]U.S. Army 1974.
[f]Ballard et al. 1968.
[g]Small 1983.
*Sutton and Salomon 1975; U.S. Congress 1969.

and B.3 of Appendix B. In most cases, it is impossible to relate the effects seen in laboratory animals to expected impacts on wildlife in natural environments.

Phytotoxicity studies show that plants are susceptible to agents GB and VX. The visible symptoms include flaccid appearance, blue-black color in leaves, and disintegration to jelly-like masses (Worthley 1970). Several studies are available describing the effects of GB on plants. Houle et al. (1972, 1976) showed that bean plants absorbed GB from nutrient solution rapidly and distributed it to all portions of the plant; leaves wilted at estimated GB concentrations of 250 ppm. Based on decomposition rates for GB, these authors estimated that the hazard to indigenous animals eating contaminated plants would last from 1 to 6 d. Schott and Worthley (1974) reported that a by-product of decomposed GB, methylphosphonic acid, was toxic to eight species of flowering plants and algae at concentrations ranging from 1 to 1000 ppm. The relative persistence of GB is less than that of VX; thus, GB has a lower potential for biological uptake and accumulation in plant tissues (U.S. Army 1974).

Studies on the toxicity of VX to plants show that (1) some plants died when exposed to VX concentrations as low as 10 ppm either in soil (Ross et al. 1983) or in aqueous solution (Worthley 1970); (2) P32-VX (or its breakdown products) in aqueous solution was accumulated in leaves, flowers, and fruit (Worthley 1971), but no tests were made of the potential toxicity to animals of the seeds or plants grown from the seeds; and (3) VX did not prevent seed germination (Ross et al. 1983; Worthley 1970). Although it has been suggested that plants may be affected by toxic by-products from decomposition of these agents, vegetation in agent test areas of Dugway Proving Ground in Utah and Aberdeen Proving Ground (APG) showed the same viability and growth as seen in noncontaminated areas (McNamara and Leitnaker 1971).

There is no specific information about the toxicity of HD, HT, H, L, or GA to vegetation. However, Hassett (1963) reported observations from World War I of defoliation of trees and loss of ground cover following the use of H; Perera and Thomas (1986) described contaminated soils and foliage and barren areas resulting from the manufacture and disposal of mustard gas during and after World War II.

Since no data are available to describe the effects that the ingestion of VX- or GB-contaminated food would have on birds, a model was developed to estimate the effects on avian species from data on rats. This can be done using the extrapolation techniques described by Suter et al. (1983). In this case, the extrapolations were performed by regressing avian LD_{50} values for cholinesterase-inhibiting organophosphate pesticides against rat LD_{50} values for the same chemicals. In general, organophosphorus nerve gas agents are similar to organophosphorus pesticides in chemical structure and/or biological activity. Some evidence suggests that combinations of nerve agents and pesticides may have additive toxic effects (Sutton and Salomon 1975); thus, birds and insects in accident areas subject to pesticide use may

be affected by doses of chemical agents lower than the LD_{50} values.

Gaines (1969) was the source of the LD_{50} data on rats, and the avian LD_{50} data were supplied by Hudson et al. (1984). Since there is variance in both the x and y variables, an errors-in-variables regression model, with the ratio of the known variances of x and y, was used (Mandel 1984). Because both x and y are acute oral LD_{50}s, the ratio can be assumed to be 1 (the regression has been found to be sensitive to small differences in the ratio).

The data bases vere sufficient to develop models for only two avian species, the mallard (*Anas platyrynchos*) and ring-necked pheasant *(Phasianus colchicus).* The data used to develop the models and graphic presentations of the regressions are found in Table 0.7 and Figs. 0.2 and 0.3. One of the pesticides is more toxic to rats than is either GB or VX; therefore, the regressions can be applied to the agents without extrapolating beyond the range of the data. The model for mallards is

$$\log \text{mallard } LD_{50} = 1.33(\log \text{ rat } LD_{50}) \cdot 0.58, \quad (r^2 \text{ of } 0.47).$$

The model for pheasants is

$$\log \text{pheasant } LD_{50} = 0.86(\log \text{ rat } LD_{50}) + 0.11, \quad (r^2 \text{ of } 0.38).$$

These models give the results presented in Table 0.8. The mallard is predicted to be considerably more sensitive than the rat, whereas the pheasant is predicted to be slightly less sensitive. The 95% confidence intervals on these estimates are quite wide. This is partly because the laboratory rat is not a particularly good predictor of avian activity and partly because we have used an unconventional confidence interval. Because, in this case, we are interested in predicting the value of future individual observations (specifically, LD_{50} values for GB and VX), the relevant interval is the prediction interval (i.e., the confidence interval *on* the individual observations). The commonly used confidence interval is the interval on the mean, which is applicable when the purpose is to describe some true underlying relationship. Results of the modeling suggest that wild birds are differentially sensitive to VX and GB; thus, generalizations about the toxicity of nerve agents to avian species are not possible.

0.3.2 IMPACTS TO TERRESTRIAL RESOURCES

In the previous section the chemical agents GB, VX, and mustard gas were shown to be toxic to both plants and animals. This section discusses the mode of uptake of chemical agents.

In general, GB, because of its volatility, would result in effects for only a few hours to a few days as the result of inhalation. An accident with GB may affect larger areas than an accident with VX, but the persistence of VX may result in longer-lasting impacts caused by ingestion and absorption. The main uncertainty that applies is the lack of information about the acute and chronic effects of chemical agents and their degradation products on wildlife and vegetation.

Table 0.7. LD_{50} values in log (mg/kg) for organophosphate pesticides in rat, mallard, and pheasant used to develop the extrapolation equations

	LD_{50} log (mg/kg)		
	Rat	Mallard	Pheasant
Carbophenothion	1.23	2.08	2.42
Coumaphos	1.40	1.47	0.89
DEF	2.21	3.46	2.43
Demeton	0.59	0.85	0.91
Diazinon	2.19	0.54	0.63
Dimethoate	1.91	1.71	1.30
Dioxathion	1.49	2.44	2.38
Disulfoton	0.59	0.81	1.07
EPN	1.22	0.66	1.72
Fenitrothion	2.81	3.14	1.74
Fenthion	2.36	0.77	1.25
lmidan	2.12	3.26	2.37
Malathion	3.06	3.17	2.22
Methyl parathion	1.26	1.20	0.91
Mcvinphos	0.67	0.66	0.13
Naled	2.39	1.71	2.01
Oxydemetonmcthyl	1.69	1.73	1.62
Parathion	0.83	0.26	1.09
Phorate	0.20	0.09	0.85
Phosphamidon	1.38	0.58	0.62
Schradan	1.29	1.55	1.27
TEPP	0.04	0.55	0.62
Thionazin	0.67	0.22	0.32
Trichlorfon	2.77	1.56	1.98
Ethion	1.62		3.11
Ronnel	3.26		2.78

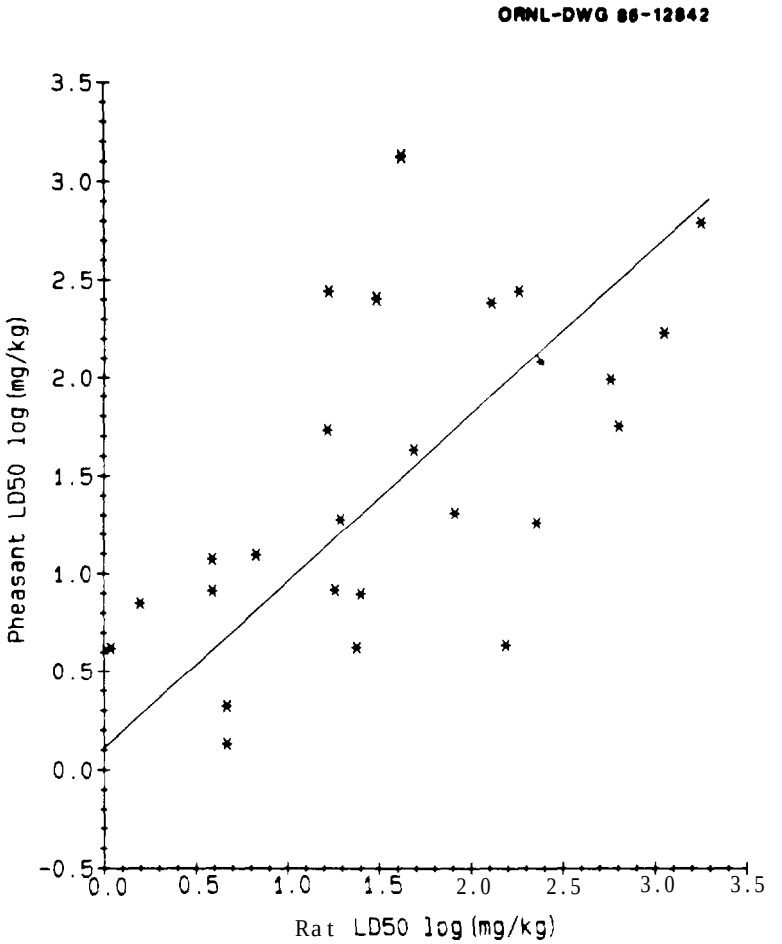


Fig. 0.2. Regression of pheasant LD_{50} log values for cboliaesterase-inhibiting orgraopbosphrte pesticides against rat LD_{50} log values for the same pesticides.

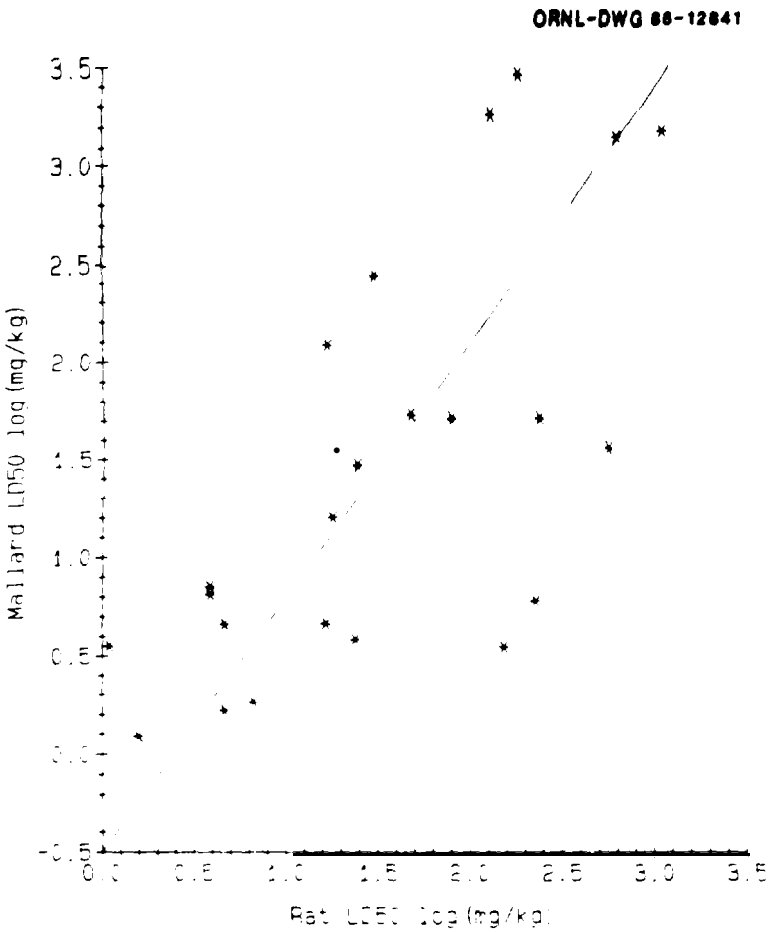


Fig. 0.3. Regression of mallard LD_{50} log values for cholinesterase-inhibiting organophosphate pesticides against rat LD_{50} log values for the same pesticides.

Table 0.8. Avian LD_{50} values in mg/kg estimated from measured rat LD_{50} values

	Rat" LD_{50}	Avian species	Avian LD_{50}	Prediction interval on the avian LD_{50}
GB	1.06	Mallard	0.28	0.0036–22.7
		Pheasant	1.38	0.043–44.0
v x	0.10	Mallard	0.012	0.000090–1.72
		Pheasant	0.18	0.0039-8. I9

"U.S. Army (1974)

0.3.2.1 Impacts to Wildlife

0.3.2.1.1 Effects of inhalation

To compare the effects of inhalation of chemical agents on humans and animals, the available LCt_{50} data (the concentration multiplied by the time that is lethal to SO% of a population) are plotted in Fig. 0.1. (The LCt_{50} values for humans are estimates.) The no-death inhalation values for humans for GB and VX are estimated to be from 6 to 10 and 2 mg-•in/m3, respectively. Thus, in contaminated areas in which no humans die from GB inhalation, it is estimated that SO% of the fruit flies would die. Inhalation effects of VX at the no-effects distances cannot be predicted because VX toxicity data for insects are not available, . Figure 0.1 illustrates that some tested animals are more sensitive than humans and that GB and VX are more toxic than HD/H, GA, or L.

0.3.2.1.2 Effects of ingestion

Data are not available to assess acute or chronic toxicity of chemical agents ingested by wildlife in the field (i.e, mammals, birds, reptiles, amphibians, and insects). However, it is possible to estimate the effects of an accident with VX on a steer in a pasture if some necessary and reasonable assumptions are made. These are (1) that a 272-kg (600-lb) steer consumes about 20 kg of forage per day (Dorn et al. 1973), (2) that the standing crop biomass of pasture grass in the east (i.e., Kentucky and Alabama) is about 0.24 kg per m2 (Shor et al. 1982), and (3) that the deposition of VX due to an accident in the human no-deaths zone is 0.06 mg/m^2. The steer will graze about 83 m^2/d and could receive a dose amounting to 5.0 mg of VX or 0.018 mg VX/kg weight if the agent were evenly dispersed. The available chemical agent LD_{50}s for laboratory animals are given in Table 0.6. The table shows that the LD_{50} for VX for young steers is 0.026 mg/kg. Steers also drink about 110 L (29 gal) of water per day (Dorn et al. 1973), and it is estimated that ponds in the area might contain 0.06 ppb of VX (Carnes et al. 1985), which results in an additional dose of 0.0066 mg of agent per day for a total dose of 0.01801 mg VX/kg. Similar calculations for 50-kg sheep [LD_{50} of 0.030 mg/kg, grass consumption of about 5 kg/d, and water consumption of about 2.7 L/d or 0.7 gal (NAS 1975)] result in a dose of 0.025 mg/kg. Thus, based on the toxicity of VX to steers and sheep, at the no-death distances for humans, some large animals would be expected to die from ingestion of VX-contaminated grass. In addition, there is evidence that a cumulative LD_{50} for a SO-kg sheep is 2.66 mg and results in death in 426 h (Sutton and Salomon 1975), so additional deaths would be expected over time from the persistent VX unless the animals were removed from the area. In the no-effects zone for humans, deposition of VX is estimated to be 0.003 mg/m^2, which results in doses per day of 0.001 and 0.0009 mg/kg for sheep and young steers, respectively. These doses would not be lethal but would depress erythrocyte AChE activity (Sutton and Salomon 1975). For smaller animals, it is reported (Ballard

et al. 1968) that 0.54 mg of VX-contaminated seedlings is lethal to mice. Thus, based on calculations for steers, sheep, and information for small mammals, VX ingestion could be lethal to other grazing animals.

In regard to VX contamination, (1) homogenates of oat seedlings that had absorbed VX from solution were lethal to mice (Ballard et al. 1968); (2) sheep were poisoned at Skull Valley, Utah, from eating VX-contaminated forage as far as 64 km from the point of release--cattle, horses, and dogs were less sensitive (Van Kampen et al. 1969, 1970; Sass et al. 1970); and (3) it took almost a year for swine which foraged on WI-contaminated ground to show no evidence of toxicity as monitored by red-blood-cell cholinesterase levels (Ross et al, 1983).

Although animals may die from eating contaminated vegetation, it is not known if ingestion of dead, agent-contaminated insects or small mammals affects wildlife; however, it is likely that surface-contaminated prey is toxic. There is some evidence that combinations of nerve agents and pesticides may have additive toxic effects (Sutton and Salomon 1975). Based on what is known about other organophosphates in agricultural systems, nerve agents would not show persistence in meat or milk; hence, food-chain effects are not expected. There is usually a spike in concentration that declines rapidly in a matter of days. Meat from an animal that dies of nerve agent exposure should not be eaten by humans or animals because there might be free (unbound) nerve agent in the tissues. Nerve agents bound to animal protein lose their toxicity. However, as described above, nerve agents and /or their degradation products (in particular, VX) do persist in and on vegetation and on dead animals and can pose a problem from ingestion and handling (see also Sect. B.6.1). In two studies (Van Kampen et al. 1970; Oreskovic and Radovic 1972), meat from poisoned sheep and their offspring and poisoned pork and lamb was fed to dogs and hens, and there was no appreciable depression in cholinesterase activities of the dogs and hens. This suggests that chemical agents are not accumulated and would not affect food chains beyond herbivores,

0.3.2.2 Impacts to Vegetation

The extent of damage to natural vegetation from exposure to chemical agents (i.e., dispersion and deposition) cannot be estimated based on the available research. The evidence for impacts to vegetation following use of H is anecdotal and in areas contaminated with nerve agents at APG and Dugway Proving Ground, Utah, there appear to be no impacts to vegetation (Sect. 0.3.1).

Although toxicity data for uptake of agent by vegetation are presented in Sect. 0.3.1, the experimental concentrations (solution culture) are higher than those estimated for soil even in the >50% human-effect isopleth. For instance, given a deposition value of 0.4 mg/m^2 in this zone and 60,000 cm3 of soil (100 cm^2 multiplied by a depth of 6 cm) with an average density of 1.2 g/cm^3 of soil, it is estimated that the concentration of nerve agent available for uptake by plant

roots is 0.006 ppm (60,000 cm^3 multiplied by 1.2 g/cm^3 divided by 0.4 mg of agent). Thus, no impacts to vegetation from deposition are expected. However, crops in the impact zone may not be safe for consumption by humans or other animals, and grasses on grazing land may be lethal to animals due to surface contamination.

0.3.2.3 Impacts to Soils

The persistence of chemical agents in soils depends on the type of agent and the rates of evaporation, hydrolysis, and biodegradation. Some of the additional factors affecting persistence include (1) method and distribution of agents; (2) type, reactivity, moisture content, pH, and temperature of the soil; (3) type and uniformity of vegetation; and (4) meteorological conditions such as wind speed, temperature, and humidity (Rosenblatt et al. 1975). Because persistence is dependent on so many factors and little is known about the agents and their breakdown products in different environments, the following ranking of the relative persistency of the agents is only a rough approximation.

Based on available information (U.S. Army 1974), it appears that the persistency of HD, HT, and H > VX > L > GB > GA. In general, persistency is enhanced at low temperatures (Kurtz 1950) and in soils with high organic matter content (Breazeale et al. 1944a) and low soil moisture (Ward et al. 1944, Crabtree and Sarver 1977). Agents evaporate most readily from sand and least readily from clay (Bouder 1940; Crabtree and Sarver 1977), and calcareous soils may neutralize or bind some agents (e.g., L) but not others (e.g., H) (Breazeale et al. 1944b).

Thus, in relation to the soils described in Sect. 3.2, it is expected that water-soluble agents released during an accident would be hydrolyzed faster in damp eastern soils but the residuals might linger adsorbed to the clay and humus; whereas in the west, the rates of liberation from dry, sandy soils with low organic matter content would be greater. In all cases, low temperatures enhance persistence.

Soil contamination over large areas, which might result from atmospheric deposition of agent after an explosive accident, would not result in the irreversible loss of farmland. Available evidence suggests that effects on vegetation and animals are negligible after one year (Cameron and Schmitz 1973; Ross et al. 1983). However, a spill would result in soil contamination from the release of the chemical agents and also the decontamination solutions (Small 1984) used to neutralize the agents. After decontamination, it might be necessary to dig up and dispose of the soil from the spill area in facility incinerators or suitable landfills. Exact procedures are dependent on, among other things, the agent, the characteristics of the spill area (i.e., topography, soil permeability), and the extent of the spill.

Small (1983) suggests that HD and one of its breakdown products, DCE, may still be present in the soil at Fort McClellan, Alabama, after 9 to 29 years of environmental weathering. Epstein et al. (1973) showed that mustard agent dumped at Edgewood Arsenal in 1941 was still

detectable in 1971. Thus, land heavily contaminated with mustard agents would be unusable for crops or grazing for relatively long periods.

The soil sampling and analysis plan following a spill is found in U.S. Army (1985) and is reproduced here.

> Soil Sampling and Analysis Plan. Soil sampling will be conducted at points where contamination is known to have occurred as the result of a leak or spill. Sampling will be initiated following surface cleanup of a spill site. A sample will be taken in the center of the spill and at four points which designate the horizontal limits of the spill. All samples will be one-inch cores. Sample depth will be determined by visual inspection of initial core (one foot) for evidence of spill (wetness). If the initial core is wet to its full depth, then subsequent cores will be taken until a depth is reached where there is no evidence of wetness. The cores will be analyzed for the presence of the spilled material. If the cores below the area contain contaminants, more cores will be taken until the spill area contamination has been defined. The definition provided by sampling will be used to designate the depth to be cleaned up. The samples will be extracted and the solvent analyzed for appropriate contaminants. In the case of brine spill, water will be used as a solvent.
>
> If the soil sampling indicates that contamination exists, the affected soil will be removed and placed in DOT approved containers. These containers will be removed and properly disposed.

0.3.2.4 Impacts to Land Use

Impacts to land use from accidents with chemical agents depend on the type of agent, the extent of the accident, and local land usage. Estimates of land use within a 20-, 50-, and 100-km radii of existing sites and the 200-km corridor around the rail transportation routes are given in Tables 3.2.7, 3.3.4, and Fig. 3.3.3. Accident scenarios for these distances are discussed in Sect. 4 in the appropriate sections for each site and Appendix K. The potential for impact to crops, pasture, and range is greatest in the Northern Plains and Corn Belt regions, while the potential for impact to natural resources (i.e., national parklands, wilderness areas, threatened and endangered species) is greatest in the Mountain and Pacific regions. Accidents that result in atmospheric deposition of agent off site are not expected to affect future mineral development. Deposition of VX from an explosive accident might make an area unusable for as much as a year; land heavily contaminated with mustard agents might be unusable for longer periods (see Sect. 0.3.2.2). Deposition of GB, which is volatile, would not have lasting effects; contaminated areas would be usable within weeks.

Additional impacts on land use will occur from construction of disposal facilities and from waste disposal (see Sects. 2.5.6.4 and 4.1.2.4). No impacts are expected to mineral development from normal

operations since the sites and waste disposal areas are already closed to mineral development.

0.4 IMPACTS ON FEDERALLY LISTED THREATENED AND ENDANGERED SPECIES

In all cases, federally listed threatened and/or endangered species are found within the impact zones at the existing sites and within the rail transportation corridors. Comparison of protected species within 20-, so-, and 100-km radii around the existing sites (Table 3.2.7) shows that the number of species increases with increasing distance from the site of an accident. Table 3.3.3 shows the numbers of species found in the transportation corridor by region.

Impacts to protected species could range from loss of food to loss of habitat or death. For instance, at Pine Bluff Arsenal, federally listed threatened or endangered bird and bat species may occur within the impact zone. Although no information about the effects of chemical agents on bats is available, the fact that bats congregate in enclosed areas for long periods of time during hibernation makes their populations particularly vulnerable to toxic atmospheric contaminants like GB.

If the Army action involving nerve agents affects listed species or their habitat, then the Army must request in writing, before such action is taken, formal consultation from the U.S. Department of the Interior Fish and Wildlife Service office that has responsibility for the project area (e.g., the Fish and Wildlife Service office in Jackson, Mississippi, for the Arkansas and Alabama transportation areas). See Appendix F for additional information about the consultation process.

REFERENCES FOR APPENDIX O

Banas, W. P., and Sprague, J. B. 1986. "Absence of acclimation to parathion by rainbow trout during sublethal exposure," *Water Research 20,* 1229-1232.

Bauer, V. E., Lindsten, D. C., and Epstein, J. *1955. Field Purification of Water Containing CW Agents with Corps of Engineers* Mobile *Water* Purification *Unit,* MLRR No. 344, Army Chemical Center, Md.

Ballard, T. A., Siegsmund, K., and Owens, E. J. 1968. *The Absorption of VX by the Leaves and Stems of Oat Seedlings,* EATM 113-4, U.S. Department of the Army, Edgewood Arsenal, Md.

Bennett, S. R., et al. 1984. *Environmental Hazards of* Chemical Agent Simulants, CRDC-TR-84055, Environmental Technology Directorate, U.S. Army Armament, Munitions and Chemical Command, Aberdeen Proving Ground, Md.

Bouder, N. M. 1940. *Lewisite (M-l), 1940 Summary of Data,* War Department, Chemical Warfare Service, Edgewood Arsenal, Md.

Boyland, E. 1944. *The* Toxicity *of* Particulate *Clouds of HN-3 and H,* Porton Report No. 2561, Miltary Intelligence Division, United Kingdom.

Breazeale, E. L., et al. 1944a. *The Effect of Organic Matter Decomposition on the Liberation of H,* TRLR 38, Medical Division OC-CWS .

Breazeale, E. L., La Grave, D. R., and Crandell, D. D. 1944b. *The Rate of Liberation of H & L from Some Calcareous Soils: A Preliminary Report,* TRLR 24, Medical Division OC-CWS.

Brunson, *K. 1980. Kansas River Basin, Kansas Stream Survey,* Kansas Fish and Game Commission, Pratt, Kans.

Cameron, B. G., and Schmitz, *G. N. 1973. Extraction of VX from Prairie Grass and Soil,* Suffield Technical Note 297, Defense Research Establishment, Suffield, Ralston, Alberta.

Carnes, S. A., et al. 1985. *Preliminary Assessment of the Health and* Environmental Impacts *of* Continuing to Store *M55 Rockets* at *Lexington-Blue Grass Depot Activity,* Anniston *Army Depot, Vmatilla Depot Activity, Pine Bluff Arsenal, and Tooele Army Depot,* M55-CS-3/ORNL-6196, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Md.

chemical demilitarization operations. These site-specific models can account for the specifics of changes in wind velocity, stability, and specific topographic influences on local meteorological conditions.

The results of these models should be used in the development of daily operating procedures, given the kinds of munitions, amount and type of agent, operations to be performed, and likely meteorological conditions. This preplanning of potential emergency responses provides a daily framework for immediate action should accidents occur. These actions can begin while the site-specific dispersion model is used to predict the expected distribution of the actual release. An on-call meteorologist should be available to interpret the results of the site-specific model as they become available and then determine the operating procedures of the day. Such operating procedures will require updates daily and at other times when changes in meteorological condition warrant.

R.7.3 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

The community study cites concerns regarding emergency preparedness and information release if an accident occurs. Programmatic emergency preparedness procedures and recommendations are found in Appendix L of the FPEIS and in the Emergency *Response* Concept *Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987). Emergency response guidelines are being developed on the basis of the emergency response concepts presented in the *Emergency Response Concept Plan.* A single command center on-site and an emergency operations center off site are currently recommended; however, site-specific circumstances may alter this guideline to an adequate equivalent or better. Part of the site-specific emergency response development is to identify emergency response personnel on-site at the facility and in potentially impacted communities. These emergency response organizations will need to establish working relationships that afford streamlined communication during any emergency should they arise.

R.7.4 ECOLOGICAL RESOURCES

The UMDA community study suggests that toxicological impacts on the agriculture of an area be considered. The assessment of potential impacts in the Umatilla area are discussed in Sect. 4.3.8; however, site-specific impact assessment should include additional impact details regarding toxicological impacts on agriculture and surface and ground waters in the area.

R.7.5 AIRCRAFT ACTIVITY

The UMDA community study suggests that airspace be restricted over the storage and demilitarization facilities plus 4.8 km (3 miles) around UMDA. Furthermore, airspace restriction should begin immediately. Section 3 of the FPEIS now details airspace activity around all storage

Houle, M. J., et al. 1976. The Fate *of Isopropyl Hethylphosphonofluorodiate in Growing Plants,* Chemical Laboratory Division, Dugway Proving Grounds, Dugway, Utah.

Hudson, R. H., Tucker, R. K., and Haegle, M. A. 1984. *Handbook of Toxicology of Pesticides to Wildlife,* Resource Pub. 153, U.S. Fish and Wildlife Service, Washington, D.C.

Kurtz, M.C. 1950. *Literature Search on the Persistency of Mustard* Gas, Technical Services Division, Technical Command, Army Chemical Center, Md.

Mancini, J. L. 1983. "A method for calculating effects, on aquatic organisms, of time varying concentrations," *Water* Research 17, 1355-1362.

Mandel, J. 1984. "Fitting Straight Lines When Both Variables are Subject to Error," *J. Qual. Technol.* 16, l-14.

Marking, L. L., and Mauck, W. L. 1975. *Toxicity of Paired Mixtures of Candidate Forest Insecticides to Rainbow Trout," Bull. *Environ. Contam. Toxicol.* 13, 518-523.

McNamara, B. P. 1972. Toxicological Studies Relating *to* the Cause of *Morbidity and Death in Skull Valley Sheep,* EATR 4627, U.S. Department of the Army, Edgewood Arsenal, Md.

McNamara, B. P., et al. 1975. *Toxicological* Basis *for Controlling Levels of Mustard in the Environment,* Edgewood Arsenal Special Publication EB-SP-74030, U.S. Department of the Army, Edgewood Arsenal, Md.

McNamara, B. P., and Leitnaker, F. C. 1971. *Toxicological Basis for Controlling Emission of GB into the Environment,* EASP 100-98, U.S. Department of the Army, Edgewood Arsenal, Md.

National Academy of Science, National Academy of Engineering (NAS/NAE) 1973. *Water Quality Criteria 1972. A Report of the Committee on Water Quality Criteria, Environmental Studies Board,* EPA-R-73-033, U.S. Government Printing Office, Washington, D.C. 594 pp.

National Academy of Science (NAS) 1975. Nutrient *Requirements of Sheep,* 5th ed., Washington, D.C.

Oreskovic, Pesic V. and Radovic, L. 1972. "On the Possibility of Using Meat of Animals Poisoned with Organophosphates as Food Products," *Vojnosanit. Pregl. 29,* 112-116.

Owens, E. J., et al. 1973. *Toxicity of "Detoxified" GB*, VX, *and HD to Animals and* Aquatic Organisms, EATR 4755, Headquarters, Edgewood Arsenal, Aberdeen Proving Ground, Md.

Perera, J., and Thomas, A. 1986. "Mustard Gas at the Bottom of the Garden," New Sci. 13, 18-19.

Rosenblatt, D. H., et al. 1975. *Problem* Definition Studies *on Potential Environmental Pollutants. I. Toxicology and Ecological Hazards of Sixteen Substances at Rocky Mountain Arsenal,* Technical Report 7508, U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick, Md.

Ross, R. H., et al. 1983. *Problem Definition* Study *of Agent VX*: Demilitarization, *Health Effects, and Toxicology,* ORNL/EIS-200, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Sass, S., *et* al. 1970. *Analysis of Environmental Samples from Area of Sheep Deaths Near Dugway Proving* Ground, Edgewood Arsenal Special Publication EASP 100-69, U.S. Department of the Army, Chemical Research Laboratory, Edgewood Arsenal, Md.

Sax, I. N. 1979. *Dangerous Properties of Industrial Chemicals,* Van Nostrand Reinhold, New York, 1118 pp.

Schott, C. D., and Worthley, E. G. 1974. *The* Toxicity of *Methylphosphonic Acid* to *Some Aquatic Species of Plants,* EB-TM-73011, U.S. Department of the Army, Edgewood Arsenal, Md.

Shor, R. W., Baes, C. F., III, and Sharp, R. D. 1982. *Agricultural Production in the United States by* County:, *A Compilation of Information from the 1974 Census of Agriculture for Use in Terrestrial Food-Chain Transport and Assessment Models,* ORNL-5758, Oak Ridge National Laboratory, Oak Ridge, Tenn.

Small, M. J. 1983. Soil Detection Limits *for Potential Chemical Warfare-Related Contaminants at Fort McClellan, Alabama,* Technical Report 8208, U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick, Md.

Small, M. J. 1984. *Compounds Formed from the Chemical Decontamination of HD, GB, and VX and Their Environmental Fate,* Technical Report 8304, U.S. Army Medical Bioengineering Research and Development Laboratory, Medical Research and Development Command, *Fort* Detrick, Frederick, Md.

Suter, G. W., II, Vaughan, D. S., and Gardner, R. 1983. "Risk Assessment by Analysis of Extrapolation Error: A Demonstration for Effects of Pollutants on Fish," Environ. Toxicol. *Chem. 2, 369-378.*

Sutton, W. W., and Salomon, L. L. 1975. *Development of a* Biological *Monitoring Network--A Test Case: Suitability of Livestock and Wildlife as* Biological Monitors *for* Organophosphorus Contaminants, EPA-680/4-75-003, Office of Research and Monitoring, U.S. Environmental Protection Agency, Washington, D.C.

Thirugnanam, M., and Forgash, A. J. 1977. "Environmental Impact of Mosquito Pesticides: Toxicity and Anticholinesterase Activity of Chlorpyrifos to Fish in a Salt Marsh Habitat," *Arch. Environ. Contam. and Toxicol. 5,* 415-425.

U.S Congress, Subcommittee on Government Operations *1969. Environmental Dangers of Open-Air Testing of Lethal Chemicals,* 91st Cong., 1st sess. (Appendix F). U.S. Government Printing Office, Washington. D.C.

U.S. Department of the Army 1974. *Chemical Agent Data Sheets,* Vol. 1, Technical Report EO-SR-74001, Defense Technical Information Center, Alexandria, Va.

U.S. Department of the Army 1985. *Application for a Research, Development, and Demonstration (RD&D)* Permit *for the Chemical* Demilitarization *Pilot* Plant, document produced by Bechtel National, Inc., San Francisco.

Van Kampen, K. R., et al. 1969. "Organic Phosphate Poisoning of Sheep in Skull Valley, Utah," *J. Am. Vet. Med. Assoc. 154, 623-630.*

Van Kampen, K. R., et al. 1970. "Effects of Nerve Gas Poisoning in Sheep in Skull Valley, Utah," *J. Am. Vet. Med. Assoc.* 156, 1032-1035.

Ward, F. N., Breazeale, E. L., and Crandell, D. D. 1944. *The Effect of* Soil Moisture on Liberation of H, TRLR 31, Medical Division, Edgewood Arsenal, Md.

Weimer, J. T., et al. 1970. *Toxicity of VX and GD* in *Aquatic Animals Indigenous to the Carroll Island Test Area Water,* Edgewood Arsenal Technical Report 4441, U.S. Department of the Army, Edgewood Arsenal, Md.

Weiss, C. M. *1955. The Response of Some Freshwater* Fish *to G Agents in* Water, Edgewood Medical Laboratory Research Report 358, U.S. Department of the Army, Edgewood Arsenal, Md.

Weiss, C. M., and Botts, J. L. 1957. "The Response of Some Freshwater Fish to Isopropyl Methylphosphonofluoridate (Sarin) in Water," *Limnol. Oceanogr. 2, 363-370.*

Wilber, C. G. 1954. "Toxicity of Sarin in Bullfrogs," Science 120, 322.

Worthley, E. *G.* 1970. *The* Toxicity *of VX to Various* Plants, EATM 100-9, U.S. Department of the Army, Edgewood Arsenal, Md.

Worthley, E. G. 1971. Uptake *and* Distribution *of* VX *in Plants,* EATM 100-15, U.S. Department of the Army, Edgewood Arsenal, Md.

Appendix P

CULTURAL AND SOCIOECONOMIC IMPACTS

P.l CULTURAL IMPACTS

This appendix provides the basis for analyzing the cultural and socioeconomic impacts of the alternatives in the chemical stockpile disposal program. In doing so, a generic analysis is utilized to be consistent with the programmatic approach. Factors that may alter the nature *or* the size of impacts are identified, thus enabling the identification of possible variations in impacts by alternative or site.

Although the cultural environment is a somewhat nebulous entity, it has been limited to two more specific areas of concern: the historical and archaeological environment and the quality of life. The latter is divided into three main components: mental health and well-being, social structure, and community well-being.

P.l.l HISTORICAL AND ARCHAEOLOGICAL IMPACTS

Historical and archaeological environments are defined as sites of cultural importance and significance. Importance or significance is determined by a number of factors, including the uniqueness, rarity, age, scholarly value, and public interest of the site. Some sites may attract visitors or users. Sites may also be given formal recognition of their value by being placed on the *National* Register *of* Historic *Places* or by being designated Historic Sites or Monuments.

P.l.l.l Normal Operations

Historical and archaeological impacts are traditionally measured in terms of sites lost or destroyed by an alteration to the physical environment. For this proposed action, such sites would probably not be physically destroyed. The exception would be if a site were located on the land chosen as the site for the demilitarization plant. Access to sites may be denied by traffic delays imposed. by transportation activities. If the public perceived that a site in proximity to chemical weapon storage, transportation, or disposal activities was dangerous due to emissions or accidents, attendance at publicly accessible sites might decrease while the threat exists. Those sites that collect revenues from visitors could lose money if attendance declines.

P.1.1.2 Impacts of Accidents

A release of agent into the environment could cause declines in attendance at nearby sites or interrupt the use of sites. Historic sites could be contaminated if they *were* in the exposure pathway. Fatalities and contamination would result in a decline in use and could denigrate the historic value even after decontamination. If decontamination of a building or structure was not feasible then the site might have to be destroyed by incineration. The use of archaeological sites could be interrupted until decontamination was completed.

P.1.2 QUALITY OF LIFE IMPACTS

Quality of life can be broadly defined as the aggregate effect of all impacts on individuals, families, communities, and other social groupings and on the way in which those groups function. As we define it, the quality of life subsumes what others label as psychological or psychosocial well-being and distributional or satisfactional impacts. We have divided this aspect of environment into three categories:

- Mental health and well-being encompasses changes in the mental states of individuals, including their attitudes, perceptions, and beliefs, as well as the associated psychological and physiological consequences of those changes.

- Social structure encompasses changes in the social organization of families and groups, their collective postures over the impacts, and how impacts affect the cohesion and viability of the group.

- Community well-being encompasses changes in community structure that relate to noneconomic factors such as desirability, social cohesion, livability, attractiveness, and sense of place.

Variation among these facets of quality of life and objective and subjective measures of them might be found from place to place in the various sites and along the various routes that make up the affected environment. The variations may be fairly small for certain items and large for others. For example, based on results from studies of mental health on state-wide or national bases, we would not expect great variations in mental health conditions in people living in impact zones. We could, however, expect variations in family cohesion (divorce rates vary) or in the desirability of a place based on aesthetics or other valued features. No attempt has been made to quantify these measures for two reasons. First, it is difficult to measure quality of life in precise ways. Second, even if a baseline were established, we have no way of predicting how the baseline would change due to implementation of the various alternatives, except in a general and qualitative fashion. The estimation of impact on quality of life is problematic because the events being analyzed do not have a history of occurrence, and our understanding of the phenomena under investigation is not precise. ⁎□ the extent possible we have used findings from analogous situations to bound the problem (Sorensen et al. 1987).

P.1.2.1 Mental Health and Well-Being

P.1.2.1.1 Cognizable impacts under NEPA

Under the National Environmental Policy Act (NEPA), an environmental impact statement (EIS) must consider psychological impacts as well as all other health impacts directly linked with the proposed action. The

U.S. Supreme Court has ruled that psychological effects associated with the perceived risk of an accident should not be considered impacts because risk is not a tangible part of the environment (PANE vs. USNRC 1982). In this ruling the court felt that the causal string of events between risk and mental health was too weak and remote to warrant consideration. The opinion would suggest, however, that the perceived threat of agent exposure from chronic or acute releases of hazardous materials would be cognizable under NEPA. Although research has challenged the scientific accuracy of the ruling that risk is irrelevant to analyzing mental health impacts of hazardous accidents, this analysis will follow the court's guidelines (Sorensen et al. 1987; Hartsough and Savistky 1984). In keeping with the letter of the ruling, the following analysis deals only with mental health impacts of the release of agent into the environment and the perceived threat of the tangible hazard of agent exposure under normal and accident conditions.

P.1.2.1.2 Causes of stress

The major means by which the proposed action will cause negative mental health rests on the assumption that exposure to a risk such as a hazardous waste incinerator or experiencing a disaster is similar in nature to going through a stressful life event (Logue, Melick, and Hansen 1981). The model of life events and mental health suggests three stages of the phenomenon. First, a person is exposed to an initial stressor event. The nature and magnitude of this event and the way in which it is perceived as harmful are important determinants of impacts (Lazarus 1966). The second stage involves exposure to a series of events that may mediate response to the stressors; such events can help reduce stress or, conversely, raise somatic stress levels (Perry and Lindell 1978; Perry 1983). Finally, the increased stress levels may result in both physical and mental impairments. Such impairments are diverse in type and severity (Baum et al. 1980). One major problem in predicting mental-health effects is a weak knowledge of the social and environmental determinants of perceived stress levels and the mechanisms by which stress results in debilitating illness (Sorensen et al. 1983; Perry 1983).

P.1.2.1.3 Stressors

Baum et al. (1980) define stress as a complex set of emotional, mental, behavioral, and biological responses to the threat of being harmed or losing something dear. Some phenomenon or event must, however, produce that threat. Stressors or stress events cover a broad range of incidents that may elevate stress levels. Such stressors occur constantly, yet do not always cause stress-related impairments to mental health.

One way of looking at stress phenomena differentiates between acute and chronic stressors. Acute stressors are short-lived phenomena usually producing a sudden elevation of stress that endures for the length of

time the stressor is present. Chronic stressors either are constantly present or repeat themselves for a long time. In a similar fashion, Lazarus and Cohen (1977) identify cataclysmic and background stressors. The former are defined as strong, sudden, unique, and rare events or groups of events. The latter are defined as ordinary, persistent, or repetitive events.

Frederick (1980) is investigating in more depth the characteristics of cataclysmic stressors that may affect stress levels. He classifies stressors by whether they are natural or man-made, are preventable or non-preventable, have short or long recovery periods, have been prepared for or not, have resulted in widespread evacuation and displacement or not, and have resulted in recoverable or irrevocable damage. Preliminary hypotheses suggest that events with the latter value of each dichotomy are more stressful.

As yet, we do not have a precise empirical model of how such factors determine stress levels or stress-induced debilitating effects, partly because we lack a grounded body of field and experimental research on risk- and disaster-induced stress. This lack of understanding is also attributable to the factors that mediate stress.

P.1.2.1.4 Mediators of stress

A variety of factors related to the individual, the social setting, and the situational context of the threat will mediate stress or heighten it. Individual factors that seem to play major roles in shaping stress response include previous susceptibility to stress (Selye 1956) or psychological instability (Perry 1983); coping ability (Sorensen et al. 1983); coping resources (Lazarus 1966); or coping skills (Perry 1983); perceptions of the hazard and risk (Slovic, Fischhoff, and Lichenstein 1979; Sorensen et al. 1983); individual grief reactions (Perry 1983); perceptions of control (Seligman 1975); and concern with other issues and problems (Sorensen et al. 1983).

Factors related to social setting that influence stress include social support networks (Bolin 1982; Drabek and Key 1984; Perry 1983); community and group ties (Sorensen et al. 1983; Perry and Greene 1982); and crisis intervention (Hartsough 1982; Tierney and Blaisden 1979).

Situational factors that play a role include experience with witnessing death (Perry 1983); experience with evacuation (Frederick 1980); media influence and coverage (Sorensen et al. 1983), and post-disaster relief (Bolin 1982).

Stress problems may be exacerbated when people feel helpless to deal with a stressor--according to theory, when one is exposed to an uncontrollable event and believes that nothing can be done to change the outcome of the event (Seligman 1975; Murphy 1985). The consequences of helplessness include reduction in mental abilities, negative emotional states, maladaptive behavior, and depression (Baum et al. 1980). Thus when a stressor and a feeling of helplessness are both present, the potential for negative mental consequences are increased.

P.1.2.1.5 Stress effects

Distinctions are usually drawn between psychological reactions to *stress* and physiological responses (Baum, Gatchel, and Sohaeffer 1983). Furthermore, it is often desirable to distinguish between direct effects, which are immediate responses to the stressor, and second-order effects, which are longer-term manifestations of stress impacts.

Direct psychological responses are numerous. First reactions often involve a feeling of shock or increased anxiety. Some stress victims may develop apathy and depressive mental states. Others may become irritable and resentful. Often, a feeling of being trapped or of helplessness will accompany the stress.

Direct physiological responses are often associated with increased levels of adrenaline and other catecholamines that increase heart rate and blood pressure and accelerate respiration, perspiration, and other physiological functions. Stress can also cause a numbing reaction in which people experience a stunned or dazed feeling and are unable to respond in a normal fashion. Physical symptoms of these responses include muscular tension, memory lapse, headache, insomnia, tiredness, sweating, dizziness, and general weakness. These are typically referred to as somatic stress effects because of a lack of a specific etiology of their manifestation.

Second-order impacts are divided into coping reactions and behavioral and physical manifestations of stress. Coping is usually defined as behavior employed to reduce *or* eliminate the danger posed by the stressor. Coping may involve conscious and purposeful actions to remove the stressor, to get away from it, or to eliminate the stressor's effects. It also may include actions that are not directed toward the stressor, such as drinking or drug use, or other detrimental effects sometimes called the 'costs of coping' (Cohen et al. 1986). In addition, coping may be solely cognitive; that is, people make mental adaptations, frequently referred to as defense mechanisms, to deal with the stressor. Social support structures such as kinship and friendship ties play an extremely important role in the coping process and may be one of the major ways in which stress is mediated.

Behavioral and physical manifestations of stress can be thought of as longer-term reactions to the first-order symptoms. These manifestations are often caused by chronic experiences with stress or from repeated episodes of acute *stress.* The most *severe* problems are probably produced by repeated catecholamine releases. Such releases exert an influence on various bodily functions, which in turn can lead to cardiovascular problems or coronary heart disease. Other physical problems, such as hypertension, arthritis, ulcers, and arteriosclerosis, have also been associated with stress. In addition, coping responses may also produce negative consequences, such as increased drinking and alcoholism. Excessive drinking can lead to a variety of problems both physical and social. Drug use can also lead to problems including overdose. All of these manifestations can lead to interpersonal

difficulties, causing the loss of friends, divorce, and job-related problems. Furthermore, longer-term psychological manifestations of stress may occur. These manifestations are often not diagnosable as stress impacts, nor are they debilitating. Such problems may include general irritability, antisocial behavior, feelings of worthlessness, impatience, harshness, decreases in analytical abilities, and general contrariness. Such problems, when severe, can lead to clinically diagnosable mental illness.

P.1.2.1.6 Low-level threat and mental health

Few studies have investigated the linkages among being exposed to a potential threat or risk, stress, and stress effects. This shortcoming is most likely caused by methodological difficulties in sorting out the causal effects of various stressors when stress levels are fairly small, It can also be attributed to the lack of any compelling theoretical reasons for such investigations. Studies that have been conducted deal mainly with the response to an ongoing threat after a disaster has occurred. The cumulative record of stress research offers no sound reason to expect that the stress effects of threat are significant unless an anomalous accident or disaster has occurred. More recently, studies have been initiated to look at stress effects of exposure to low levels of toxic materials. These studies provide a better notion of the impacts of threats which can be characterized as low risks.

Empirical evidence for this conclusion is provided by Baum, Gatchel, and Schaeffer (1983). In a study of the stress effects of the accident at the Three Mile Island (TMI) Nuclear Power Plant in 1979, populations at TMI (17 months after the accident) were compared with those (1) at a site with an operating nuclear power plant, (2) at a site with a coal-fired power plant, and (3) at a site without any power plants. Backgrounds and demographic characteristics of each group were comparable. Major stress measures used in the study are presented in Table P.l along with mean scores at each site. The results show that the TMI population is under greater stress than the other populations. No significant difference was found between the population exposed to risk from a nuclear power plant and the two control groups. While this does not mean that the threat in the no-accident site plays no role in determining stress, it suggests that the threat does not elevate stress above normal levels.

Another study by Baum investigated stress associated with the venting of radioactive gases from -the damaged reactor at TMI (Baum, Gatchel, and Schaeffer 1983). The results of this study suggested that the accident at TMI has led to some elevated chronic stress among the population around TMI. The venting of radioactive materials led to elevated acute stress before the release. The venting and post-venting measurements revealed a lowering of the anticipatory preventing stress. In all cases, stress levels were subclinical.

Dew, Bromet, and Schulberg (1987) conducted a comparative study of the stress effects of two types of stressors --the TMI accident and unemployment. They found that levels of subclinical stress symptoms were elevated to a similar degree for each stressor and remained elevated for 2 to 3.5 years.

Using different methods and measures, several recent studies have shown that toxic exposure systematically leads to elevated levels of distress, both specific and global, among individuals (Lebovits, Baum, and Singer 1986). This general finding holds up for a variety of situations, including acute releases of chemicals (Markowitz and Gutter-man 1986), contaminated water supplies (Gibbs 1986), low-level releases of radiation (Davidson et al. 1986), and the presence of a hazardous waste site (Bachrach and Zatura 1986; Levine and Stone 1886).

These studies also found that lack of information and conflicting information exacerbate psychological reactions (Lebovits, Baum, and Singer 1986). Feelings of helplessness, powerlessness, and lack of control also result from heightened distress (Lebovits, Baum, and Singer 1986). The chief implication of this set of studies is that if people believe that they are being exposed to chronic or acute releases of toxic materials, they have a potential *to* develop elevated distress and its various symptoms. This effect is greatest when people do not believe they are receiving accurate information about the problem and feel helpless about gaining some control over the situation.

P.1.2.1.7 Groups vulnerable to lov-level threats

Some evidence exists that children, depending on their age, will be either more or less vulnerable to the threat of chemical'agent exposure. One study suggests that young children (<11 years) may not perceive risks from such activities (Walesa 1977), while adolescents may feel powerlessness and resignation toward the activity (Schwebel and Schwebel 1981). One implication of this finding is that adolescents may be *more* aware of an external stressor and thus more vulnerable to experiencing stress. This also implies that young children may be less aware of external stressors such as a hazardous facility and less likely to experience stress. Family studies, however, show that stress in children can occur due to factors internal to the family and other social networks such as schools. Thus, a child who has parents who are stressed by the external threat may reflect that internal *stress* without being aware of the specific cause. Likewise if the issue disrupts school activities, then stress may also be experienced.

P.1.2.1.8 Effects of stress after a disaster

The effects of stress from experiencing a disaster are a subject of intense debate (Perry 1983; Quarantelli 1979; Sorensen et al. 1983; Hartsough 1982). All agree that stress levels are elevated during a disaster but not on whether those effects are adverse and long-lasting. Some argue that *stress* results in social bonding and rather short-lived

psychological bonding. The differences, however, may come about because of differences in theoretical approaches, methodologies, and the specific disasters investigated (Perry 1983).

Studies have documented numerous types of psychological effects of disaster, including acute grief, anger, anxiety, hostility, resentment, depression, and loss of ambition (Logue, Melick, and Hansen 1981). In the immediate aftermath of disaster, researchers have documented what is commonly called the disaster syndrome (Wallace 1956). This syndrome is described as an absence of emotion, inhibition of activity, docility, indecisiveness, lack of responsiveness, and automatic behavior, together with the physiological manifestation of autonomic arousal (Kinston and Rosser 1974). Some researchers conclude that the disaster syndrome is rare and when it does occur, is relatively short-lived (Quarantelli 1979; Quarantelli and Dynes 1977). Others suggest it is a starting point for enduring psychological problems. While this issue is likely to be further debated, a review of the evidence developed to date on negative effects provides a qualitative but not quantitative assessment of disaster-induced mental health effects.

Melick (1985) provides a comprehensive review of 43 empirical studies on the psychological effects of disasters and offers some generalized conclusions. First, disasters do seem to cause mental health problems in the recovery phase, which may last for several years. Not every study, however, has found long-term effects. Only a few studies have concluded that there is an increase in physical health problems associated with stress. The number of people experiencing health effects and the size of those effects appear to be related to the methods of study and measurement techniques. Clinical asessments find much higher incidents of mental health problems than self-reports or validated mental health scales. Several studies also note that disasters have positive mental health effects.

Perry (1983) discusses factors relevant to the manifestation of mental health impairment from natural disasters. He suggests, [based on Barton (1970)], that duration of impact (defined by long repetitive events or steady manifestation of a condition) is a key factor in determining impacts. He also suggests that geographical extent of the disaster is the second key characteristic of the stressor event. Perry also identifies disaster-specific effects, such as the extent to which friends and kin are killed or injured and the level of personal property loss experienced as determinants of impacts.

P.1.2.1.9 Groups vulnerable to disaster-induced stress

Several studies have focused on the mental health impacts of disasters on vulnerable subgroups of the population, including children and the elderly. Children are considered to be a particularly vulnerable subgroup of the population to disaster-induced psychological impacts (Bolin 1985). Research has shown that following severe disasters, children exhibit separation anxieties, sleep disorders, and phobias. The

persistence and duration of these effects, however, are not well-known. Burke et al. (1986) found that fifth-grade girls experiencing a flood showed increased distress ten months after the event. This research also suggests that girls are more vulnerable, as boys of the same age did not exhibit increased fear depression and anxiety. This is consistent with Dohhrewend's (1979) finding that somatic symptoms of stress such as nightmares or stomachaches were especially common among girls and children in the low grades following the TMI accident. Overall there is growing evidence that disasters cause emotional problems for some children; however, ways in which to ameliorate short- and long-term emotional problems in children are not well understood (Benedek 1985).

Whether the elderly constitute a vulnerable group is still open to some debate, although growing evidence suggests that the elderly do not require special mental health considerations (Bolin 1985). Melick and Logue (1985-86) found that elderly flood victims did not show increased levels of anxiety or depression when compared with non-victims. This contradicts earlier research that defined the elderly as a vulnerable subgroup (Friedsam 1962).

Another vulnerable subgroup is parents, females in particular who have small children or are pregnant. Logue (1968) suggests that women under 65 with lower levels of income and education are a high-risk group. The work of Bromet, Schubert, and Dunn (1982) suggests that people with mental health problems prior to a disaster are more susceptible to disaster-induced problems. Children can either create anxieties or amplify their parents' anxieties. Large families are also more vulnerable to psychological impacts than small families (Bolin 1982).

P.1.2.1.10 Summary of stress impacts

Table P.l presents our best estimates of the type of impacts that would occur under various threat and disaster conditions based on the current literature. To organize this information, we have divided hazards into ones that are dreaded (e.g., nuclear power, chemical spills, plane crash, nerve gas accident) and those that are more common (floods, auto accidents, etc.). We have also distinguished among four situations: threat only, disaster signal, minor disaster, and catastrophic disaster. Threat only is equivalent to exposure to risk. Disaster signal is a situation in which heightened awareness is caused by a near miss or disaster elsewhere. A minor disaster one in which no loss of life occurs. Major or catastrophic disasters include the loss of life.

P.1.2.1.11 Normal operations

A portion of the population could believe that the presence of a chemical agent is a threat to its safety. In the short run, this threat, if accompanied by a belief that one was being exposed to an agent, may elevate stress, particularly in anticipation of activities involving the

Table P.I. Summary of mental health effects

Threat type	Threat only	Disaster signal	Minor disaster	Catastrophic disaster
		Dreaded threat		
Concerns/feelings	Some concern	High concern	Widespread and strong concerns	Extreme trauma
Anxieties	May affect a few individuals	Some people will experience anxiety	Prevalent during some longer-lasting emergencies	High and of lasting duration
Somatic effects/minor etiological effects	None attributable	Some people will experience symptoms	Notably higher for some	Prevalent among many individuals
Clinical impairment	None	None	Few but difficult to attribute	Some likely
		Common threat		
Concerns/feelings	No concern	Minor concern	High concern	Widespread concern; some traumatization
Anxiety	None	Only a few individuals	Prevalent during emergencies	High with some lasting effects
Somatic effects/minor etiological effects	None	Unlikely	Few	High for some individuals
Clinical impairment	None	None	None	Few but difficult to attribute

handling, transport, or disposal of the agent. Once activities begin and as they becomes routine, stress is likely to be reduced. Evidence from the TMI accident research suggests that even if stress were to increase to levels found following the non-damaging accident at TMI, the effects would be subclinical and would not result in any adverse health effects (Baum, Gatchel, and Schaeffer 1983). However, stress would probably-not even reach those levels.

P.1.2.1.12 Accident

In an accident with no acute health impacts, people in the adjacent area may experience elevated stress but are not likely to experience . adverse clinical effects. An accident with offsite exposure and fatalities has a potential for causing long-term clinical effects and adverse mental health problems. The character of an accident involving chemical weapons maximizes the potential for stress to occur because it meets many of the criteria that researchers suggest would maximize mental health problems. These include:

1. it is a technological risk;

2. it is uncontrollable;

3. it will cause evacuation;

4. it will not have elicited much emergency preparation;

5. it will disrupt social and kin networks;

6. it will contaminate property;

7. it will likely result in people seeing others die; and

8. it is a dreaded hazard.

While other factors may mediate against stress, we believe that the above characteristics of an accident will lead to a strong stress response. Whether this will definitely lead to adverse mental impairment is speculative, although the probability is high that some portion of the survivors will experience negative effects.

P.1.2.2 Social Structure Impacts

P.1.2.2.1 Family stress

Stress not only affects individuals but has wider effects on groups and social units such as families, peer networks, and larger social organizations, The impact may arise because the reaction to stress of one group member may affect the behavior of other members in that group.

Alternatively, a force or a situation outside the group may place stress on each member of the group.

A basic model of stress response similar to that for the individual has been formulated for the family by Hill (1949). After many years of research on the family, this model and several variations on the model are still accepted as a basic explanation of the stress-formation process in families (McCubbin, Olson, and Patterson 1983). The model suggests that a stressor interacts with a family's stress-meeting resources and with the group's definition of the situation. The result is labeled a family crisis. The crisis results in a breakdown of unity or cohesion, usually followed by recovery and stabilization of normal interaction and behavioral patterns (Hill and Hansen 1962; Hansen and Hill 1964).

Stress-meeting resources are thought of as both means to cope with and means to eliminate the stressor. For example, resources may include money, marital stability, position in the life cycle, social support networks, and family cohesion (Bolin 1982; Bolin and Bolton, 1986). Definitions of situation are based both on intrafamilial communication patterns and on external communication patterns. In turn, the interaction between resources and definitions of the stressor leads to a coping response. Either the lack of resources or the failure to achieve a consensus definition can lead to social breakdown and associated problems.

P.1.2.2.2 Social conflict

When a number of groups and individuals within a group are concerned about a situation or a perceived problem, a collective social movement may occur to cope with the source of the stress and conflict. For example, following the accident at TMI, a number of citizen protest organizations were formed to intercede in decisions about the future of the reactor site (Walsh 1981). Groups were formed not only to express grievances associated with nuclear power, but also to provide support for resuming operations (Soderstrom et al. 1984). When such groups are at fundamental odds, social conflict can develop, intergroup stresses can mount, and a change or breakdown in social behavior can occur.

P.1.2.2.3 Disasters and family stress

Few empirical investigations have been conducted on how a stressor, such as a disaster, has affected family or group social structure. The most extensive evidence to date has been collected by Bolin (1982) concerning family recovery from tornadoes in Wichita Falls and Vernon, Tex. Evidence suggests that the tornado experience produced emotional effects and family disruption among a portion of the victims. Internal family stress was elevated by the experience and persisted during recovery. The major causes of stress included bereavement, material losses, and continued exposure to disaster-related events associated with the recovery process. Those victims with the greatest losses were

more likely to experience strains in family relationships than those with low or no losses.

P.1.2.2.4 Normal operations

Under normal operations, stress among some families and groups is expected to rise. Minor levels of increased conflict among some group members could result. Such conflict should not, however, cause loss of cohesion or breakup of the group. As the implementation of the alternative continues, stress could fall.

P.1.2.2.5 Accident

An accident that did not have negative health effects would likely have mixed effects on families. For some it could tighten group cohesion because of feelings of relief and avoidance of loss. For others stress may be heightened due to ongoing operations or to family conflict generated by the disposal program. In such instances family cohesion may be disrupted.

If a major accident occurred, negative impacts on group structures would be likely. The nature and magnitude of the impacts cannot be precisely estimated, although the limited data to date suggest that families experiencing the accident undergo emotional upset, disruption, and strain to relationships. These effects persist for several years, particularly in families that lost members or close associates *or* that experienced material losses. Impacts are also determined by the recovery process. Economic strain and reminders of the incident increase family and group disruption.

P.1.2.3 Community Well-Being Impacts

The well-being of a community is largely a subjective concept despite attempts to quantify its dimensions and to attach ratings to them. For the transportation alternative, three dimensions of well-being seem to be relevant: the cohesiveness of the community as a social unit, the quality of the community as defined by inhabitants and outsiders, and public support and opposition. Prior to discussing impacts, a brief perspective on each is offered.

P.1.2.3.1 Cohesiveness and conflict

Cohesiveness within a community context is the ability of groups and organizations to establish and maintain bonds and interactions and to develop solidarity. Conflict, as used in discussing community impacts, is defined as the forces that may alter cohesion and ultimately may result in either various forms of antisocial behavior or an increased bonding. Cohesion and conflict relate to a broader sense of psychological sense of community. In this context, cohesion is defined as people's need to have a desirable community and to be in communion with those around (Glynn 1981). In addition, cohesion relates to

feelings of belonging and identification with a place and to cooperation and bonding with others. The loss of cohesion is associated with many negatives, including feelings of alienation, isolation, loneliness, loss of local autonomy, and inability to maintain social relationships and networks (Glynn 1981).

A number of factors operate within a community to both bolster and erode cohesion. In the context of facing a threatening event or confronting risk, experience with community response to hazardous waste problems provides insight into how cohesion is affected (Sorensen et al. 1983). For example, experience at Love Canal, N.Y., and Wilsonville, Ill., both with serious waste problems, suggests that variability in the type and timing of public involvement, public concern over risks, the role of information, and mitigation closure help determine how cohesion will be affected. At Love Canal, the public was not significantly involved in the issue, credible information was not available, and little was done in a timely way to mitigate concerns and gain closure. Almost the opposite was true at Wilsonville. At Love Canal, much of the area was evacuated or abandoned, and stress contributed to family problems and the loss of a sense of identification. Wilsonville, however, largely preserved its sense of community and cohesion. Thus, an external conflict can result either in enhanced cohesion or loss of cohesion depending on the intervening social processes.

When a severe disaster strikes a community, cohesion and conflict are part of the social response and recovery. While, in general, disasters have been noted to have a positive effect on the cohesion of a community (Fritz 1961; Quarantelli and Dynes 1976), they do at times lead to conflict and the loss of community (Erickson 1976). Table P.2 attempts to summarize some of the major factors thought to influence cohesion and conflict following a disaster.

P.1.2.3.2 Community quality

The analysis of community well-being has taken two approaches in the literature. One has dealt with subjective or perceived facets of well-being'with research concentrating on individuals' satisfaction with various aspects of their lives (Campbell, Converse, and Rodgers 1976; Andrews and Inglehart 1979). Three dimensions of subjective well-being have commonly been tapped (Wasserman and Chua 1980):

1. life variables, such as personal happiness and satisfaction with one's life;

2. specific life-domain variables, such as satisfaction with housing and health; and

3. global life-space variables, such as satisfaction with community or attributes of a place.

Table **P.2. Potential for community conflict following disaster**

Factor	Increased conflict	Decreased conflict
Source of threat	Internal to community	External to community
Nature of threat	Ambiguous	Readily identifiable
Solutions to problems	Division	Consensus
Recognizability of effects	Uncontained and complex	Clearly defined and simple
Temporal dimension	Long-term effect	Short-term effect
Warning	None	Ample to respond
Emergency response	Poor or nonexistent	Well demonstrated
Resources for recovery	Inadequate	Abundant
Control	Response and recovery directed from outside	Response and recovery directed from inside
Social inequities	Distributed impact among group	Equal impact
Group formation	Pre-emergency polarization among groups	No groups

Research shows, however, a high intercorrelation between measures at these three levels (Wasserman and Chua 1380; Andrews and Withey 1976).

A second approach to measuring well-being (Liu 1975; Smith 1973) has used a wide range of objective indicators and reduced them into clusters with multivariate techniques. For example, Golant and McCutcheon (1980) collapsed 92 variables into 11 factors:

1. growth and change (e.g., population change),
2. congestion and crowding (e.g., population density),
3. safety (e.g., crime rate),
4. family welfare (e.g., divorce incidence),
5. economic status (e.g., income),
6. education and professional status (e.g., level of education),
7. availability of services (e.g., indices of various professional and commercial functions),
8. physical health (e.g., death rate),
9. housing stock status (e.g., age of dwelling unit),
10. economic health (e.g., unemployment rate), and
11. mental health (e.g., suicide rate).

A chief criticism of this approach has been that even with an index based on these factors, one still fails to know what they mean in terms of well-being; any interpretation is ultimately subjective.

In light of these problems, several researchers have attempted to compare objective and subjective measures. In general, they have found no overall relationships between the two approaches to rating geographic areas (Schneider 1975). Furthermore, specific variables attempting to measure the objective dimension of well-being are not highly correlated with subjective measures of that same dimension (Wasserman and Chua 1980). This disparity led to considerable skepticism about the utility of objective measures for capturing the human dimension of well-being. Subjective measures, on the other hand, have been attacked on the basis that people tend not to make honest evaluations of their lives when questioned. Research is beginning to dispel this criticism (Atkinson 1982). From this literature we conclude that examining the impacts of any of the proposed alternatives with an objective approach would be difficult. Ultimately, well-being involves the perceptions and Attitudes of local people.

P.1.2.3.3 Public support and opposition

In reviewing public comments and records to date concerning the chemical stockpile disposal program, six themes emerge:

1. Some members of the public support the alternatives involving off-site transport because it would prevent onsite disposal facilities from being built.

2. Some members of the public want in-depth scrutiny of all or some of the alternatives involving offsite transport to determine their acceptability in comparison with the other alternatives.

3. Some members of the public reject the alternatives involving offsite transport because they perceive it to be risky and dangerous.

4. Some members of the public feel that the onsite alternative is acceptable because the technology is safe and risks are negligible or because transporting is risky.

5. Some members of the public want to see the program delayed so that more information and experience with the proposed as well as alternative technology are gained before a decision is made.

6. Some members of the public are ambivalent about which alternative is chosen.

Estimation of which theme dominates public thinking at each storage site or along the proposed transport routes is not feasible. Furthermore, public support and opposition is dynamic and thus will change over the course of the program, and the rationales of public support and opposition vary from site to site,

P.1.2.3.4 Normal operations

Under normal operations, enduring impacts to community well-being will probably not be experienced. The collective stress imposed by each alternative will likely promote cohesion within some communities rather than conflict. Conflict, if it occurs, is unlikely to be manifested in highly visible ways. The well-being of a community may be affected in the short run (during implementation) in subtle ways that would be difficult to isolate and measure. These impacts would most likely involve feelings of isolation and the loss of security and community identity by some people. These feelings would be strong during the beginning of the operation but would dissipate over time. They would not be expected to persist after the operations ceased.

The long-term quality of well-being in communities would not be expected to change significantly during normal operations. The overall

trends of well-being and the factors that determine them would likely outweigh any disposal-related effects.

P.1.2.3.5 Accident

A small accident without public health impacts would likely coalesce people and groups within a community to participate in the decision making process. This could generate an increased or decreased sense of community depending on whether the community had a consensus on how to continue with the program.

A major accident would heavily affect community well-being, the extent of the effects being determined partly by the magnitude of losses from the accident. Based on the assumed characteristics of an accident, the resulting conditions would probably work against a cohesive community response and recovery. A major accident would likely demoralize the community, leading to a sense of isolation, grief, and loss of attachment among the survivors.

Community quality would also be severely affected by an accident. An event of this nature would cause some people to feel that the community is undesirable and not a secure place in which to reside. A stigma associated with an accident may be attached to areas in which losses occur, as well as to the survivors. Such a stigma may depress the area and make development and growth difficult despite the absence of future risks.

If the fatalities were extremely high, the normal structure of a community might be sufficiently destroyed to make a return to normalcy unlikely. While over time a new community structure would be developed, it might never be qualitatively the same. When a severe disaster strikes a community, cohesion and conflict are part of the social response and recovery processes. While, in general, disasters have been noted to have a positive effect on the cohesion of a community they do at times lead to conflict and the loss of community.

P.2 SOCIOECONOMIC IMPACTS

In this section we discuss socioeconomic impacts of the chemical stockpile disposal program alternatives. Categories of impact areas judged to warrant consideration include population, emergency planning, noise, traffic disruption, and local economy.

P.2.1 POPUUTION

The major way in which normal operations could potentially affect population change is through in-migration generated by new jobs or out-migration generated by perceptions of elevated risks from disposal activities.

P.2.1.1 In-migration and Infrastructure

The total number of jobs created by construction and operations, if sufficiently large, could generate in-migration that would affect community Infrastructure such as schools, law enforcement, fire protection, sewer and water supply services, and housing, particularly if the existing population were low. This is referred to as the "boom town effect," where suddenly a low-population area is turned into a rapid-growth area due to the new jobs created by a project. Whether or not in-migration occurs due to new jobs is a function of the total number of jobs created, the timing of the employment in the project schedule, the type of skills required to fill those jobs, the size of the labor market in the impacted area, the unemployment rate, and the number of secondary jobs created by in-migration and increased expenditures. A project that creates a small number of new jobs in relationship to total employment in the area will likely create impacts to infrastructures.

P.2.1.2 Hazards and Mobility

To date, little research has been conducted to investigate the effects of risky activities or disasters on population change. Goldhaber, Houts, and Pisabella (1983) looked at mobility decisions following the TMI accident. This study found that, although people with a greater fear of nuclear power were more likely to move out of the vicinity, they were people who *were* already highly mobile and likely to move in any environment. Thus the study concluded that the presence of a threat such as a damaged reactor that was emitting low levels of radiation did not shape the demographic profile of the area. Palm (1981) examined the role that earthquake risk plays in mobility and found that people rarely consider earthquake hazards in their decision to purchase a house.

Studies by Wright et al. (1979) and Friesema et al. (1979) showed that, in areas in which disasters have occurred, population changes have not taken place. They looked, however, at larger regions (by communities or census tracts) and not at impact areas. Thus, the effects of a disaster could be obscured by the changes in non-impacted areas. Several cases exist where areas contaminated by hazardous materials have largely been abandoned due to voluntary migration and government relocation programs. Both Love Canal, N.Y., and Times Beach, Missouri, experienced large declines in population after toxic wastes were found in their environs. Contamination by a chemical agent, particularly mustards, would likely result in large declines in population in and near the contaminated area until the area was proven safe to occupy.

P.2.1.3 Normal Operations

All of the proposed alternatives will result in the creation of only a small number of construction and plant operation jobs. A disposal plant will require an average labor force of 150 workers to construct (U.S. Army Toxic and Hazardous Materials Agency and U.S. Army Corps of Engineers 1985). The labor force required to operate the disposal facilities varies by site due to the stockpile composition and the number of shifts worked. Transportation of the stockpile off site could generate a small number of jobs for a very limited time at the origin and destination sites. Table P.3 lists the maximum expected labor force by site, employment in the vicinity of each site and unemployment rates for each county. All of the sites can draw from large local labor markets. None of these labor markets has unusually low levels of unemployment that would constrain using local labor to fill the new jobs. It is unlikely that a large percentage of the jobs will require in-migration to be filled. It cannot be assumed, however, that all jobs will be filled locally. Some of the workers will migrate from outside the labor market. Even if all the workers migrated for plant operations, the change in population at each location would be insignificant in comparison with existing populations.

The generation of new jobs by the program activities would have multiplier effects in that the increased income from the new employment would lead to increased spending in other sectors of the economy, which would create some additional new jobs. This could, in turn, lead to additional in-migration. The size of this multiplier effect is dependent on the type of direct employment, the region of the country, and type of community in which the employment occurs. Ranges for new construction activities are 0.35 to 0.9. A normal multiplier for non-construction employment is 0.3.

Out-migration due to the hazards of chemical agent related activities is also expected to be insignificant. Using the TMI situation as an upper bound on impacts from normal operations, we would not expect increased levels of out-migration from implementation of any of the alternatives, although specific moves could be associated with disposal activities. Under normal operations, people living near disposal activities might perceive personal threat from the activity. However, this threat probably would not cause significant migration from the area. Overall, no noticeable population change would occur because of disposal or collocation activities.

P.2.1.4 Accident

Under a major accident scenario, fatalities among the general public could occur. Population would decrease as a result. A major accident would cause evacuation and temporary relocation as well as contamination. A long-term decline in population within the impact area could occur as a result. It is impossible to predict the extent of the decline, as it would be determined by the nature of the accident and

Table P.3. Employment

Site	Construction[a]	Operation[b]	Current employment[c]	Unemployment rate (%)[d]
		On-site		
APG	150	210	118,000	5.0
ANAD	150	308	14,500	8.9
LBAD	150	273	48,500	6.3
NAAP	150	210	13,000	9.5
PBA	150	282	8,500	8.6
PUDA	150	308	13,500	12.1
TEAD	150	518	104,000	6.0
UMDA	150	273	35.500	11.2
		Regional		
ANAD	300	492		
TEAD	450	764		
		National		
TEAD	750	1210		

[a]USATHAMA and USACE, 1985.
[b]USATHAMA, 1986.
[c]U.S. Bureau of Census, 1982a,b,c.
[d]1986 estimates for county in which facility is located.

recovery efforts. Demographic change in a larger regional context, however, is unlikely to occur. Overall impacts would increase as the number of fatalities increased. Impacts to a specific community would increase as the percentage of the community affected increased and, thus, smaller communities would experience greater impact levels.

P.2.2 LAND USE

Land use might be shaped in the long run by the risks in an area. In the short run, major land use changes would not be responsive to changes in the risk profile of an area. This does not preclude microchanges in the use of a specific parcel of land if the current users judge it to be at risk from emissions of agent. Even if such change occurs, it is difficult to automatically attribute the change to the disposal facility, particularly if it follows the prevailing land use changes in the region as a whole.

After a disaster in which property losses and/or number of fatalities are large, land use will typically change (Haas, Kates, and Bowden 1977). Often, this change is positive for the survivors in that it allows community improvement and redevelopment. Unless a disaster renders agricultural lands unsuitable for production, land usually reverts to its original agricultural use after the disaster effects have subsided. If an accident resulted in contamination of, or fatalities to, the public, short-term land use changes would probably occur.

The current Army policy of incineration as the preferred means of decontamination would drastically alter land use. Existing structures and vegetation would be totally destroyed if such a practice were implemented. The land decontaminated in this fashion would be littered with the materials that did not burn. A massive clean-up would likely be needed in order to reclaim the land for productive use.

P.2.2.1 Normal Operations

Patterns of land use around fixed sites and transportation corridors are very unlikely to change in the short run as a result of chemical weapons disposal activities. In the long run, the known existence of a hazardous substance may alter the nature of land use but not in a manner that can be readily predicted.

P.2.2.2 Accident

Agricultural lands would not be used at least until decontamination occurred. Other land use functions would also be abandoned until decontamination had occurred. Longer-term land use change in both urban and rural areas could also occur. Because no reentry criteria exist (see Appendix B), the lands must be assumed to be lost use for an indefinite length of time, or until it can be demonstrated that they are safe. The total amount of land affected would be determined by the nature of the accident. Existing land uses might be abandoned if fatalities occurred,

if the area of impact was perceived to be unsafe, or if a social stigma became associated with the contamination or the facilities,

P.2.3 EMERGENCY PLANNING

The level of emergency planning and response impacts will be determined by the level of emergency planning and response requirements that cannot be met with existing civilian and Army resources and, hence, would require the use of additional state and local resources. Under this emergency planning, we include the development of contingency plans and activities to support planning and implementation. Emergency response includes participating in emergency and security activities. such as exercises, maintaining plans and equipment, training, and responding to a spill or accident.

State and local governments will need to prepare or amend contingency plans and to coordinate their plans with those of the Army. The preparation of plans would likely require technical assistance from the Army, as well as state and local expenditures and staff time. Prior to the beginning of operations, state and local emergency personnel would need to assess the adequacy of their resources for participating in the operation and for responding to a potential emergency. They would also have to procure those resources that are necessary for ensuring public safety.

P.2.3.1 Recommended Levels of Preparedness

The recommended levels of preparedness for alternatives of the disposal program are summarized in Appendix L.

P.2.3.2 Reduction in Impacts from Upgraded Plans

The reduction of impacts from enhanced emergency planning cannot be quantitatively estimated with any degree of reliability. Too many unknown variables exist that cast uncertainties on the estimates. Furthermore, any estimates would reflect an average reduction with extremely large standard deviations. Thus for any given future disaster, the reduction is not predictable.

The reduction in impacts can be defined in qualitative terms by comparing the potential for fatalities without enhanced planning to the potential to reduce fatalities with sound implementation of the recommended response procedures. The results of this comparison are summarized in Tables P.4 and P.5. The reductions have been assessed for different releases under different weather conditions because the amount of reduction in fatalities will vary with scenario. For some situations the reduction are quite large; for others, they are more modest. Indeed, some scenarios are difficult to mitigate with emergency planning.

Table P.4. Qualitative benefits of fixed site emergency response concept
☎✗□□ nerve ● geot accidents)

Accident scenario	Meteorological conditions		
	Slow wind speeds (1 m/s) (approx. downwind distance to .2x no-death concentration in upper corner)	Average wind speeds (3 m/s)	Fast wind speeds (6 m/s)
Moderate instantaneous release (100 kg VX)	33 km Potential for fatalities is high within IRZ;[a] lower in PAZ[b]—high reduction in fatalities at all distances	IO km Potential for fatalities is high within 2 km; lower to IO km-low reduction in fatalities to 2 km; higher at greater distances	15 km Potential for fatalities is high within 4 km; lower to I5 km-low reduction in fatalities to 5 km; higher at greater distances
Small instantaneous release (10 kg VX)	7 km Potential for fatalities is high within 2 km: lower to 7 km-high reduction in fatalities at all distances	4 km Potential for fatalities is moderate within I km; lower to 4 km-low reduction in fatalities to 2 km	6 km Potential for fatalities is moderate within 2 km; lower to 6 km-low reduction in fatalities at 5 km
Large semicontinuous release (1000 kg VX)	100 km Potential for fatalities is very high within IRZ; lower in PAZ and PZ' high reduction in fatalities at all distances	25 km Potential for fatalities is high within IRZ; lower in PAZ-low reduction in fatalities to 2 km; high at greater distances	15 km Potential for fatalities is high within IRZ; lower in PAZ-low reduction in fatalities to 5 km; high at greater distances
Moderate semicontinuous release (100 kg VX)	45 km Potential for fatalities is very high within IRZ; lower in PAZ and PZ- high reduction in fatalities at all distances	8 km Potential for fatalities is moderate within 2 km; lower to 8 km—moderate reduction in fatalities at 2 km; greater to 8	7 km Potential for fatalities is high within 2 km; lower to 7 km-low reduction in fatalities at 2 km; moderate reduction to 5 km
Large spill (900 kg GB)	I2 km Potential for fatalities is moderate within 3 km; lower to I2 km—high reduction in fatalities at all distances	8 km Potential for fatalities is moderate within 2 km; lower to IO km—high reduction in fatalities at all distances	8 km Potential for fatalities is moderate within 2 km; lower to 8 km—moderate reduction in fatalities to 2 km; greater to 8 km

[a]IRZ = immediate response zone.
[b]PAZ = protective action zone.
[c]PZ = precautionary zone.

Table P.S. Qualitative benefits of fixed site emergency response concept
(for mustard agent accidents)

Accident scenario	Meteorological conditions		
	Slow wind speeds (1 m/s) (approx. downwind distance to .2x no-death concentration in upper corner)	Average wind speeds (3 m/s)	Fast wind speeds (6 m/s)
Moderate instantaneous release (900 kg HD)	7 km Potential for fatalities is high within 2 km; lower to 7 km—high reduction in fatalities at all distances	2 km Potential for fatalities is moderate within 0.5 km; lower to 2 km-low reduction in fatalities to 2 km	1 km Potential for fatalities is low—potential for precautionary measures is low
Small instantaneous release (IO kg HD)	1 km Potential for fatalities is low--high potential for taking precautionary measures	0.2 km Potential for fatalities is very low; low potential for precautionary measures	0.15 km Potential for fatalities is very low—precautionary measures unlikely
Large semicontinuous release (4000 kg HD)	100 km Potential for fatalities is very high within IRZ;[a] lower in PAZ" and PZ[c]—high reduction in fatalities at all distances	IO km Potential for fatalities is high within 2 km; lower to 10—low reduction in fatalities to 2 km; high at greater distances	7km Potential for fatalities is high within 2 km; lower to 10 km-low reduction in fatalities to 5 km; high at greater distances
Moderate semicontinuous release (100 kg HD)	8 km Potential for fatalities is high within 2 km; lower to 8 km-high reduction in fatalities at all distances	2 km Potential for fatalities is moderate within 0.5 km; lower to 2 km-moderate reduction in fatalities at 2 km	1.5 km Potential for fatalities is high within 0.5 km; lower to 1.5 km-low reduction in fatalities at 1.5 km
Large spill (900 kg HD)	71 km Fatalities unlikely	71 km Fatalities unlikely	71 km Fatalities unlikely

[a]IRZ = immediate response zone.
[b]PAZ = protective action zone.
[c]PZ = precautionary zone.

P.2.3.3 Normal Operations

P.2.3.3.1 Costs of implementing the ERCP

The impacts of emergency planning on the local socioeconomic structure of communities are substantial at storage and disposal sites, but relatively small along the transportation corridors. The required actions and associated costs of upgrading plans are as follows. The Emergency Response Concept Plan (ERCP) recommends changes in most functional categories of planning and response activities. These changes are presented in detail in the ERCP.

The total costs of implementing fixed site plans are presented in Table P.6 for each site by functional activity. The costs of continued storage, the national or regional disposal alternatives, and partial relocation alternatives represent incremental costs over those estimates for fixed sites. In the case of continued storage, yearly costs would be incurred for a longer duration. For those alternatives involving off-site transport, additional costs are for emergency preparations and coordination activities along the transportation corridors. The additional costs for off-site transportation corridor planning are summarized in Table P.7.

P.2.3.3.2 Costs of temporary relocation during off-site transport

The temporary relocation of population in the rail corridors has been identified as an alternative to development of an emergency response plan. It is difficult to imagine an operation of this size and complexity. The following analysis is an attempt to estimate how much it would cost to implement this type of program.

Several recent evacuations provide fairly good estimates of the cost of temporarily relocating populations at risk. Estimates from TMI and Mississauga indicate that the average daily costs per family were $40 and $58, respectively. This represents transportation costs, lodging, and extra meal expenses. A breakdown of costs suggests that a reasonable assumption is that it will cost about $25 per family for round trip transportation and $40 per day for housing and extra food costs (Sorensen et al. 1987). This, however, assumes that many families would stay with friends and relatives. Thus the actual costs may be twice this amount if people stay overnight at a motel. In addition, this figure does not account for lost income or production.

Additional costs will be incurred for personnel to conduct the evacuations. It is difficult to estimate the magnitude of these costs. It is reasonable to assume that 4 to 8 people would be needed for each mile of the route for an g-hour day at $100 per day.

The average population per kilometer of track in a 10-km (20-km total) corridor is assumed as follows:

Table P.6. Summary cost estimates of CSDP emergency preparedness program, by site[a]

Cost category	Site cost ($ in thousands)							
	APG	ANAD	LBAD	NAAP	PBA	PUDA	TEAD	UMDA
EOC construction/renovation	50	675	I.100	1,000	950	25	450	400
Communications equipment	324	I.133	1,457	1,457	I.295	162	648	486
Pressurized filtration systems for institutional populations	1,095	2.550	4.050	1,425	1,800	95	105	480
Individual protective equipment	1,928	874	I.219	213	312	20	5	189
Public alert systems								
Sirens	1,675	I.675	1,675	I.675	1,675	1,675	1,675	1,675
Telephone alerting system	3,825	I.734	2,419	421	619	39	IO	375
Evacuation time estimate studies	200	350	250	250	250	100	100	150
Emergency worker protective equipment	76	228	179	120	142	59	35	46
Antidote drugs for nerve agents	N/A[b]	1,921	1,279	I.017	I.196	N/A	75	288
Public information materials	145	679	550	372	439	41	45	129
Media center	26	26	26	26	26	26	26	26
Program standards (average)	33	33	33	33	33	33	33	33
Area planning(average)	195	195	195	195	195	195	195	195
Local planning	70	245	315	315	280	35	140	105
State planning	50	50	50	100	50	50	50	100
Army implementation program department (average)	33	33	33	33	33	33	33	33
Army implementation (average)	163	163	163	163	163	163	163	163
Local implementation	350	1,225	1,575	1,575	I.400	175	700	525
State implementation	250	250	250	500	250	250	250	500
Total cost	$10,488	$14,039	$16,788	$10,890	$1 I.108	$3,176	54,738	S5.898
Program total cost $77,135								

[a] These costs are applicable regardless of the alternative selected.

[b] N/A = not applicable; no nerve agent at site.

Source: Jacobs Engineering Group, Inc.. and Schneider EC Planning and Management Services 1987. Emergency *Response Concept Plan for the Chemical Stockpile* Disposal *Program.*

Table P.?. Additional costs for emergency planning due to off-site transportation, s in thousands

Category	Alternative			
	Regional	National	APG air	LBAD air
Communications equipment	565	565	NA[a]	N A
Escort equipment	1.074	1,074	N A	N A
Protective equipment	92	92	N A	N A
Program standards	260	260	N A	N A
Army planning	780	780	N A	N A
State planning	950	1,200	N A	N A
Army program implementation	1,560	1,560	N A	N A
State program implementation	4,750	6,000	N A	N A
Total cost	10,031	11,531	11,511	11,511

WA = data not available.

Source: Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987. ***Emergency Response Concept Plan for the Chemical Stockpile Disposal Program.***

Eastern only routes: 50 per km
Eastern to western routes: 40 per km
Western only routes: 10 per km

The estimated costs are summarized in Table P.8.

P.2.3.3.3 Accident

The impacts of an accident on emergency planning depend on the size and location of the accident and the extent to which plans and response capabilities are upgraded. Three critical situations may differentiate among impacts: (1) an accident that is confined on-site; (2) an accident that causes off-site response; and (3) an accident that results in fatalities. The first situation would not generate significant offsite direct impacts but could lead to additional investments in emergency planning. An accident that involved off-site response would likely necessitate a broad range of state and local emergency responses, including assisting in warning, conducting evacuations of the public and special facilities, maintaining road blocks, providing and maintaining evacuation shelters, procuring and distributing emergency food and water, and assisting in the reentry process.

In addition to these services, if injuries and fatalities occurred, local resources would be needed to assist in transport and medical care of the injured, identify and remove bodies of the victims, assist in search and rescue, and help in the disaster recovery.

The requirements of responding to an emergency would result in increased costs to all governmental entities involved. The level of these costs would depend on the type of accident and the circumstances under which it occurred. These costs could strain, if not exceed, existing state and local resources, and outside assistance would be necessary.

P.2.4 NOISE

P.2.4.1 Normal Operations

Increases in noise will occur from construction and transport activities. On-site noise will not likely affect off-site areas (Carnes et al. 1985a,b). The noise generated from train travel will be approximately equal to that of a normal train trip along the same route or tracks. The total increase in noise over a 24-h period due to the added frequency of train trips will increase the incidence of train-induced noise but will not significantly increase the average noise level over that period of time (Carnes et al. 1985b).

The construction and operations of on-site airfields for partial relocation alternatives could be a significant source of noise pollution in densely populated sites such as Lexington-Blue Grass Army Depot (LBAD) or Aberdeen Proving Ground (APG). At LBAD there is currently no existing air traffic on-site and thus the incremental noise would be

Table P.8. Costs of temporary **evacuation,** s in millions

	Low	High
National disposal option		
Relocation	200	400
Security	76	**152**
Total	**276**	552
Regional disposal option		
Relocation	86	172
Security	3 6	72
Total	122	**244**

substantial. At APG an airfield already exists, but noise would increase because of larger planes and increased frequency of flights.

P.2.4.2 Accident

While an accident may lead to an increase in noise, noise level is not significant in comparison with other impacts of an accident.

P.2.S TRANSPORTATION

P.2.5.1 Normal Operations

Increased use of roads into the disposal sites for construction and operations activities will occur. This could lead to increased traffic congestion, causing inconveniences to existing use. The level of disruption will depend on the existing use/capacity ratio and the increase in traffic trips generated by disposal activity. Because the total number of workers for construction and operations is relatively small, it will be unlikely that the added traffic would strain existing transportation systems such as roads leading to the installations.

Increased train trips may slow or delay existing train traffic along proposed routes but will not cause great disruption or prevent existing use. Train trips will block road traffic at rail crossings for short periods until the train convoy has passed.

Barge activity in the Chesapeake Bay will disrupt commercial and private ship and boat travel in the Bay channel for intermittent periods of time until the barge is loaded and reaches open seas.

P.2.5.2 Accident

Under an accident scenario, traffic in the vicinity of the accident site and in the plume pathway would be disrupted. This disruption would endure for as long as it took to clean up the accident. Post-accident investigations could result in longer delays.

P.2.6 LOCAL ECONOMY

P.2.6.1 Direct and Indirect Impacts

A new industrial facility has several direct impacts on the local economy. The wages and salaries of workers lead to increased spending in the local area. This generates increased sales and other tax revenues for government. The size of the impact will depend on the portion of income workers spend, where they spend it, and on the tax structure of the community and state. In addition, money spent on materials and services during construction and operations will result in increased revenues to businesses providing those materials and services. As discussed in Sect. 1.2.1, these expenditures will have a multiplier effect on the local economies. Table P.9 provides estimates of the total

Table P.9. Economic impacts of demilitarization of stockpile

Site	Operations expenditures			Selected economic multipliers'			Selected major economic impact*			Total	Total wholesale, retail. and services trade' value	Years of construction/ operation	Average annual program impact (% of total value)
	A, Construction	B, Pcrsonncl 0 573'	C, Utilities 0.090'	Total	A_2	B_2	C_2	A_1XA_2	B_1XB_2	C_1XC_2				
On-site disposal costs'														
APG	21.44	*7.09*	I II	12.36	2.312	2 235	I.694	*47.93*	15.84	1.87	65.65	21,298.34	3.3	0.09
ANAD	29 54	34.87	5 44	60.81	I.967	I.916	I.419	58.09	66.83	7.73	132.65	1,358.60	4.1	2.38
I BAD	39 01	13.13	2.05	22.90	2.111	2.028	1.617	82.35	26.63	3.32	I 12.29	4,864.05	2.9	0.80
NAAP	18.03	7.71	I.20	13.45	1.825	I.887	I.586	32.90	14.56	I.91	49.37	1,376.14	3.3	1.09
PBA	12.23	53.16	8.30	*92.70*	1.779	I.931	1.482	21.75	102.64	12.30	136.69	*778.31*	*4.7*	*3.74*
PUDA	31.24	23.60	3.68	41.16	1.909	I.949	I.551	59.64	46.01	5.71	I II.36	*903.78*	3.3	*3.73*
'TEAD	36 75	88.14	13.76	153.71	2.324	2.182	1.895	85.40	192.29	26.08	303.77	11,206.05	6.8	0.40
UMDA	41.36	38.20	5 96	66.6 I	I.864	I.941	1.379	*77.09*	74.14	8.22	159.45	2.026.48	4.3	1.83
Regional disposal alternative costs'														
ANAD	54.98	65.95	10.30	115.00	*I.967*	I.916	I.419	108.12	126.38	14.61	249. I I	I,358.60	4.8	3.82
TEAD	6R 44	148.49	23.18	258.94	2.324	2.182	I.895	159.04	323.93	43.94	526.91	11,206.05	6.1	*0.77*
National disposal alternative costs'														
TEAD	86.1 I	187.67	29.30	*327.26*	2.324	2.182	I.895	200.10	409.40	55.53	665.04	11,206.05	6.1	0.97

"A Costs arc related to military construction of industrial facilities. B Personnel costs arc related primarily to retail trade. C. Utility costs are related io supplies of natural gas. electricity. and water.

'The selected economic impacts are the anticipated major impacts for the local area.

'The proportion of operations expenditures estimated on the basis of M55 rocket disposal proportions devoted io personnel and utilities, respectively.

dSum of wholesale. retail. and services trade in millions of 1982 dollars

'(osts in millions of constant FY 1987 dollars.

Sources

1 Const ruction and operations costs *Chemical Stockpile Disposal* Plan *Supplement* (March 1987). Tables 4.2 io 4.4

2 I conomic multipliers Economic Impact Forecast System (EIFS), Department of Urban and Regional Planning. University of Illinois. Urbana-Champaign, derived from U S. Department of Commerce, Bureau of the Census 1977. *County Business Patterns.* U S. Government Printing Office, Washington, D.C.

3 Wholesale. retail, and services trade U S. Department of Commerce, Bureau of the Census 1982 *Census of Wholesale Trade.* U.S. Government Printing Office. Washington, D C.

could be monitored if evidence came forth that such impacts might be occurring.

P.2.6.4 Disaster Impacts

A disaster produces both direct and secondary losses and revenues to a local economy (Cochrane 1975). Most studies, both empirical (Wright et al. 1979; Friesema et al. 1979) and simulated (Ellson 1984), suggest that commercial and industrial activities recover from disaster, although the recovery time varies with the magnitude. The accident at TMI caused no noticeable impacts on housing prices in the area (Gamble and Downing 1981). A large accident in an isolated area would probably have the greatest impact. A disaster would change the distribution of income loss and gain within the area. It would also affect outside activities that were linked to activities in damaged areas.

P.2.6.5 Normal Operations

Normal operations could cause some disruption to activities in close proximity to the site and transport routes. While this disruption would last for the duration of the operations, it would probably lessen as time passed. Losses in marginal activities might cause them to cease operations.

Property values are unlikely to decline in the long run. Sales of properties at risk may slow down during operations. Disposal activities, including construction, operations, and decommissioning could generate increased direct and indirect expenditures and sales tax income for communities. When disposal alternatives are completed, the increase in revenues generated by the disposal programs would be lost.

P.2.6.6 Accident

If an accident occurred, the impacts would largely depend on the size and location of the accident. It is likely that some short-term disruption would occur. Government relief and compensation could offset some losses. Long-term effects would not be likely unless the accident damaged a large portion of a community.

Total impacts are likely to be greatest in densely populated environments. Impacts to a specific community may be greater as the community size decreases and a greater percentage of the community is affected.

The total costs of an accident are potentially very large. If we use the cost of a catastrophic accident at a nuclear power plant as a rough surrogate of the economic impact of a catastrophic chemical weapons accident, the costs could range between $1 and 10 billion (USGAO 1987).

REFERENCES FOR APPENDIX P

Andrews, F. M., and Inglehart, R. 1979. "The Structure of Subjective Well-Being in Nine Western Societies," *Soc. Indicators Res.* 6(1), 73-90.

Andrews, F. M., and Withey, S. B. 1976. Social *Indicators of Well-Being,* Russell Sage, New York.

Atkinson, T. 1982. "The Stability and Validity of Quality of Life Measures," *Soc. Indicators Res.* 10, 113-32.

Bachrach, K., and Zatura, A. 1986. "Assessing the Impact of Hazardous Waste Facilities," pp. 71-88 in *Exposure to* Hazardous *Substances: Psychological Paramerers,* eds. A. Lebovits, A. Baum, and J. Singer, Lawrence Earlbaum Associates, Hillsdale, N.J.

Barton, A. 1970. Communities *in Disaster* (as cited in Perry 1983).

Baum, A. et al. 1980. *Psychological* Stress *for Alternatives of Decontamination of TMI-2 Reactor Building Atmosphere,* NUREG/CR-1584, U.S. Nuclear Regulatory Commission, Washington, D.C.

Baum, A., Gatchel, R., and Schaeffer, M. 1983. "Emotional, Behavioral, and Physiological Effects of Chronic Stress at Three Mile Island," *J. Consult. Clin. Psychol.* 51, 565-72.

Benedek, E. 1985. "Children and Disasters," *Psychiatric* Annals 15(3), 168-172.

Bolin, R. C. 1982. *Long-Term Family Recovery from Disaster,* Institute of Behavioral Science, University of Colorado, Boulder.

Bolin, R. 1985. "Disaster Characteristics and Psychosocial Impacts," in *Disasters and Mental Health:* Selected *Contemporary Perspectives,* ed. B. Sowder, U.S. Department of Health and Human Services, Washington D.C.

Bolin, R. and Bolton, P. 1986. *Race, Religion and Ethnicity in Disaster Recovery,* Institute of Behavioral Science, University of Colorado, Boulder.

Bromet, E., Schubert, H., and Dunn L. 1982. "Reactions of Psychiatric Patients to the Three Mile Island Nuclear Accident," *Arch. Cen. Psychiatry 39* (June), 725-730.

Burke, J., et al. 1986. "Emotional Distress in Fifth-Grade Children Ten Months after a Natural Disaster," *J. Am. Acad. Child Psychology* 24(4), *536-541.*

Campbell, A., Converse, P., and Rodgers, W. 1976. *The* Quality *of American Life,* Russell Sage, New York.

Carnes, S. A., et al. 1985a. *Preliminary* Assessment *of* the *Health and* Environmental Impacts *of* Incinerating MS5 Rockets Stored at Pine *Bluff Arsenal, Lexington-Blue Grass Depot Activity, and/or Anniston* Army Depot at Pine Bluff Arsenal, M55-CD-13/ORNL-6197, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Md.

Carnes, S. A., et al. 1985b. *Preliminary Assessment of the Health and Environmental Impacts of Transporting M55* Rockets *from Lexington-Blue Grass Depot Activity, Anniston Army Depot, and Umatilla Depot* Activity to Alternative Disposal *Facilities,* M55-CD-12/ORNL-6198, U.S. Army Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, Md.

Cochrane, H. 1975. *Natural Hazards and* Their Distributive *Effects,* NTIS, PB 262-021, Institute of Behavioral Science, University of Colorado, Boulder.

Cohen, S., et al. 1986. *Behavior, Health, and Environmental Stress,* Plenum Press, New York.

Davidson, L., et al. 1986. "Toxic Exposure and Chronic Stress at Three Mile Island," pp. 35-46 in Exposure *to Hazardous Substances: Psychological Parameters,* eds. A. Lebovits, A. Baum, and J. Singer, Lawrence Earlbaum Associates, Hillsdale, N.J.

Dew, M., Bromet, E., and Schulberg, H. 1987. "A Comparative Analysis of Two Community Stressors' Long-Term Mental Health Effects," Am. *J. Community Psychol.* 15(2), 167-184.

Dohhrewend, A. 1979. *Staff Reports to the* President's *Commission on rhe Accident at Three Mile Island,* Report of the Behavioral Effects Task Force, Washington, D.C.

Drabek, T., and Key, W. 1984. Conquering Disaster, Irvington Press, New York,

Ellson, R. 1984. "Measuring the Regional Economic Effects of Earthquakes and Earthquake Predictions," *J. Reg. Sci. 24, 559-79.*

Erickson, K. T. 1976. Everything *in Its Path,* Simon and Schuster, New York.

Frederick, C. 1980. "Effects of Natural vs. Human-Induced Violence Upon Victim," *Evaluation and Change* (Special Issue), 71-75.

Friedsam, H. 1962. "Older Persons in Disaster," pp. 151-184 in *Man and* Societies in *Disaster,* ed. G. Baker, Basic Books, New York.

Friesema, P., et al. 1979. *Aftermath: Communities After Natural Disaster,* Sage, Beverly Hills, Calif.

Fritz, C. 1961. "Disaster," pp. *651-94* in *Contemporary Social Problems,* eds. R. Merton and R. Nisbet, Harcourt, New York.

Gamble, H., and Downing, R. 1981. *Effects of the Accident at Three* Mile *Island on Residential Property Values and Sales,* NUREG/CR-2063, U.S. Nuclear Regulatory Commission, Washington, D.C.

Gamble, H., Downing, R., and Sauerlender, 0. 1979. *Effects of Nuclear Power Plants on Community Growth and Residential Property Values,* NUREG/CR-0454, U.S. Nuclear Regulatory Commission, Washington, D.C.

Gibbs, M. 1986. "Psychopathological Consequences of Exposure to Toxins in the Water Supply," pp. 47-70 in Exposure *to* Hazardous Substances: *Psychological Parameters,* eds. A. Lebovits, A. Baum, and J. Singer, Lawrence Earlbawn Associates, Hillsdale, N.J.

Glynn, T. J. 1981. "Psychological Sense of Community: Measurement and Application," *Hum.* Relations 34, 789-818.

Golant, S., and McCutcheon, A. 1980. "Objective Quality of Life Indicators and the External Validity of Community Research Funding," *Social Indicators Research, 7, 207-235.*

Goldhaber, M., Houts, P., and Pisabella, R. 1983. "Moving after the Crisis: A Prospective Study of the Three Mile Island Population Mobility," Environment *and Behavior* 15, 92-120.

Haas, J., Kates, R., and Bowden, M. 1977. *Reconstruction* Following *Disaster,* Massachusetts Institute of Technology Press, Cambridge.

Hansen, D., and Hill, R. 1964. "Families Under Stress," pp. 782-882 in *Handbook of Marriage and* the *Family,* ed. H. Christensen, Rand McNally, Chicago.

Hartsough, D. 1982. "Planning for Disaster: A New Community Outreach Program for Mental Health Centers," *J. Community Psychology 10* (July), 255-264.

Hartsough, D., and Savitsky, J. 1984. "Three Mile Island: Psychology and Environmental Policy at a Crossroads," Am. *Psychologist 19* (October), 1113-1122.

Hill, R. 1949. Families *Under Stress,* Harper, New York.

Hill, R., and Hansen, D. 1962. "Families in Disaster," pp. 185-221 in *Man and Societies in* Disaster, ed. G. Baker, Basic Books, New York.

Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987. *Emergency Response Concept Plan for the Chemical Stockpile Dispoal Program.*

Kinston, W., and Rosser, R. 1974. "Disaster: Effects on Mental and Physical State," *J. Psychosom. Res. 18, 427-56.*

Lazarus, R. S. *1966. Psychological Stress and the Coping Process,* McGraw-Hill, New York.

Lazarus, R. S., and Cohen, J. 1977. "Environmental Stress," pp. 90-121 in *Human Behavior and Environment, Vol.* 2, eds. I. Altman and J. Wohlwill, Plenum Press, New York.

Lebovits, A., Baum, A., and Singer, J. *1986. Exposure to Hazardous Substances: Psychological Parameters,* Lawrence Earlbaum Associates, Hillsdale, N.J.

Levine, A., and Stone, R. 1986. "Threats to People and What They Value: Residents' Perceptions of the Hazards of Love Canal," in *Exposure to Hazardous Substances: Psychological Parameters,* eds. A. Lebovits, A. Baum, and J. Singer, Lawrence Earlbaum Associates, Hillsdale, N.J.

Liu, Ben-Chieh *1975. Quality of Life Indicators in U.S. Metropolitan Areas,* 1970, U.S. Environmental Protection Agency, Washington, D.C.

Logue, J. N. 1978. "Long-Term Effects of a Major Natural Disaster," Ph.D. dissertation (unpublished), Columbia University, New York.

Logue, J. N., Melick, M. E., and Hansen, H. 1981. "Research Issues and Directions in the Epidemiology of Health Effects of Disasters," *Epidemiol. Rev. 3,* 140-62.

Markowitz, J., and Gutternman, E. 1986. "Predictors of Psychological Stress in the Community Following Two Toxic Chemical Incidents," pp. 89-108 in *Exposure to Hazardous Substances: Psychological* Parameters, eds. A. Lebovits, A. Baum, and J. Singer, Lawrence Earlbaum Associates, Hillsdale, N.J.

McCubbin, H., Olson, D., and Patterson, J. 1983. "Beyond Family Crisis: Family Adaptation," *Int. J. Mass Emergencies and Disasters* 1(1), 73-93.

Melick, M. 1985. "The Health of Post-Disaster Populations: A Review of the Literature and Case Study," pp. 179-210 in *Perspectives on* Disaster *Recovery,* eds. J. Laube and S. Murphy, Appleton-Century-Crofts, Norwalk, Conn.

Melick, M., and Logue, J. 1985-6. "The Effect of Disaster on the Health and Well Being of Older Women," *Int. J.* Aging *and Human Development* 21(1), 27-38.

Montz, B. 1984. "The Floodplain as a Housing Market," oral presentation at the Association of American Geographers Annual Meeting, April 24, 1984, Washington, D.C. (No hard copy available).

Murphy, S. 1985. "The Conceptual Basis for Disaster Research and Intervention," pp. 3-21 in *Perspectives on Disaster Recovery,* eds. J. Laube and S. Murphy, Appleton-Century-Crofts, Norwalk, Conn.

Palm, R. 1981. *Real Estate Agents and Special Studies* Zone *Disclosures,* Institute of Behavioral Science, University of Colorado, Boulder.

PANE v. USNRC 1982. 678 F. 2d 222,228 (D.C. *Cir).*

Perry, R., and Greene, M. 1982. "The Role of Ethnicity in Emergency Decision Making Processes," Sociological *Inquiry* 52(4), *309-334.*

Perry, R. 1983. "Environmental Hazards and Psychopathology: Linking Natural Disasters with Mental Health," *Environ. Manage. 7, 543-52.*

Perry, R. W., and Lindell, M. K. 1978. "The Psychological Consequences *of* Natural Disaster: A Review of Research on American Communities," *Mass Emerg. 3,* 105-17.

Quarantelli, E. L., and Dynes, R. R. 1976. "Community Conflict: Its Absence and Its Presence in Natural Disasters," *Mass Emerg.* 1, 139-52.

Quarantelli, E., and Dynes, R. 1977. "Response to Social Crisis and Disaster," *Annu. Rev. Social. 3, 23-49.*

Schneider, M. 1975. "The Quality of Life in Large American Cities: Objective and Subjective Social Indicators," *Soc.* Indicators *Res. 1, 495-509.*

Schwebel, M., and Schwebel, B. 1981. "Childrens' Reactions to the Threat of Nuclear Power Plant Accidents," *Am. J. Orthopsychiatry* **51(2),** 260-70.

Seligman, *M.* 1975. *Helpleness: On Depression, Development and Death,* Freeman, San Francisco.

Selye, H. 1956. *The Stress of Life,* McGraw-Hill, New York.

Slovic, P., Fischhoff, B., and Lichenstein, S. 1979. "Rating the Risks," *Evironment* **21(3),** 14-39.

Soderstrom, J., et al, 1984. "Risk Perception in an Interest Group Context: An Examination of the **TMI** Restart Issue," *Risk Analysis* **4(3),** 231-244.

Smith, D. M. 1973. *The Geography of* Social Well-Being *in the* United *States,* McGraw-Hill, New York.

Sorensen, J. et al. 1983. *Restarting TMI Unit One:* Social *and Psychological Impacts,* ORNL-5891, Oak Ridge **Natl.** Lab., Oak Ridge, Tenn.

Sorensen, J., et al. *1987. The Impacts of Hazardous Technology,* State University of New York Press, Albany, N.Y.

Tierney, K., and Blaisden, B. 1979. Crisis *Intervention Programs for Disaster* Victims *in Smaller Communities,* National Institute of Mental Health, Rockville, Md.

USATHAMA 1986. *Chemical Stockpile Disposal Plan,* Report **AMXTH-CD-FR-**85047, Aberdeen Proving Ground, Md.

USATHAMA and **USACE** 1985. *Environmental Analysis of Onsite Disposal of M55 Rockets at LBAD*, Report **M55-OD-5,** Aberdeen Proving Ground, Md.

U.S. Bureau of Census 1982a. *Census of Retail Trade,* Washington, DC.

U.S. Bureau of Census 1982b. *Census of Wholesale Trade,* Washington, DC.

U.S. Bureau of Census 1982c. Census *of Service Industries,* Washington, DC.

U.S. Department of the Army 1987. *Chemical Stockpile Disposal Plan Supplement,* Report AMCPM-CD-FR-87109, Program Executive Officer--Program Manager for Chemical Munitions, Aberdeen Proving Ground, Md.

U.S. Department of Commerce, Bureau of the Census 1977. County *Business Patterns,* U.S. Government Printing Office, Washington, D.C.

U.S. Department of Commerce, Bureau of Census 1982. *Census of Wholesale Trade.* U.S. Government Printing Office, Washington, D.C.

U.S. Government Accounting Office (USGAO). 1987. *Nuclear Regulation: A Perspective on Liability Protection* for *a Nuclear Power Plant Accident,* Washington, D.C.

Walsh, E. 1981. "Resource Mobilization and Citizen Protest in Communities Around Three Mile Island," Social *Problems* 29(1), 1-21.

Walesa, C. 1977. "Development of Risk Perceptions in Children and Adolescents," *Pol. Psychol.* Bull. 8(3), 171-76.

Wallace, A. F. *C.* 1956. *Tornado* in *Worcester: An Exploratory Study* of *Individual and Community Behavior in an Extreme Situation,* Publication No. 392, National Research Council Committee on Disaster Studies.

Wasserman, I., and Chua, L. 1980. "Objective and Subjective Social Indicators of the Quality of Life in American SMSA's: A Reanalysis,* *Soc*. Indicators *Res. 8,* 305-81.

Wright, J. D., et al. 1979. *After the Clean-up: Long Range Effects of Natural Disasters,* Sage, Beverly Hills, Calif.

Appendix Q -

SENSITIVITY OF FATALITY ESTIMATES TO DOSE-RESPONSE VALUES, METEOROLOGICAL CONDITIONS, AND DISTRIBUTION OF SENSITIVE POPULATIONS IN AN AFFECTED ENVIRONMENT

Q.l. RATIONALE FOR PERFORMING A SENSITIVITY ANALYSIS

Estimates for fatalities resulting from accidental releases at each chemical agent storage site have been presented in Sect. 4.3. In developing these estimates, atmospheric dispersion analyses from the D2PC computer code (as described in Sect. 4.2.2.1 and Appendix K) were combined with population statistics (as discussed in Sect. 4.2.3.1) to determine the population at risk. Predicted chemical agent dose-responses were then applied to the population at risk in order to develop fatality estimates.

The toxicological data (see Appendix B, Sects. B.2.5 and B.3.5) used in developing these fatality estimates considered only acute lethality for healthy adult males; such data are understood to be appropriate for quantitative evaluation of dose-response. However, the question remains unanswered as to whether consideration of the dose-response of a more precise cross section of the population would result in different fatality estimates. Specifically, infants, children, or elders may die at lower doses than the estimated no-deaths dose. Because the potential inclusion of such revised data might result in significant differences in estimated fatalities, a sensitivity analysis was performed to address these uncertainties.

The specific objective of this sensitivity analysis was to develop new fatality estimates that could be compared with the baseline fatality estimates of Sect. 4.3. These revised fatality estimates were to be based upon consideration of:

1. a more representative population (i.e., one composed of infants, children, adults, and elders);

2. exposure of the sensitive portions of the population (i.e., the infants, children, and elders) to modified doses from accidental chemical agent release; and

3. variations in the meteorological conditions (i.e., primarily wind direction) from those in Sect. 4.3.

Two specific sites, Aberdeen Proving Ground (APG) and Lexington-Blue Grass Army Depot (LBAD), were chosen to be indicative of locations with a high population density and a moderate population density, respectively. Performing a sensitivity analysis for these two sites was intended to produce revised fatality estimates which would demonstrate whether or not there is a significant variation from the baseline fatality estimates of Sects. 4.3.1 and 4.3.3.

4.2 SUMMARY OF THE METHOD USED IN DEVELOPING THE BASELINE FATALITY ESTIMATES

For convenience in understanding the sensitivity analysis, the following discussion summarizes the methodology (see Sects. 4.2.2.1 and 4.2.3.1) used to develop the baseline fatality estimates of Sect. 4.3.

Each specific accident was placed into a consequence category (as defined by downwind kilometer distance to the "no-deaths" dose). One of the two meteorological conditions (conservative most likely or worst case) was used to define a plume geometry. Three separate zones were identified inside each plume as shown in Fig. Q.l. These zones are contained within elliptical contours that overlap at the point of chemical agent release; as described from the outermost zone inward, they are (1) a far-reaching zone, the boundary of which marks the no-deaths dose; (2) a middle zone, the boundary of which marks the 1% lethal dose; and (3) a close-in zone, the boundary of which marks the 50% lethal dose.

The individual plumes, including the three zones, from each specific accident were overlain on a grid of the population distributed about the site of release. For the baseline fatality estimates in Sect. 4.3, the affected population in each zone was assumed to respond to'chemical agent exposure the same as do healthy adult males. Within the close-in zone (from the release point out to the 50% lethal dose boundary), 75% of the population was assumed to be killed; this is the midpoint between the assumed 100% fatalities at the point of release and the 50% fatality rate at the edge of of the zone. In the middle zone (from the 50% to the 1% lethal dose boundary), 25% of the population was assumed to be killed. In the far-reaching zone (between the 1% lethal dose and the no-deaths boundary), 0.5% of the population was assumed to be killed. These percentages were used mathematically as "fatality multipliers" and were applied to the population located within each zone. Acute lethal sensitivity of the affected population in each zone was assumed to approximate that estimated for healthy adult males (i.e., the same response to those doses used in the D2PC code as discussed in Sect. 4.2.2.1).

Because the exact wind direction during any given release would not be known, each plume (which includes all three zones) was rotated about the release site in increments of one compass degree; at each increment the number of fatalities was recomputed from the population contained inside that plume. The largest estimate of fatalities, from among all 360 such increments, is the number tabulated in Sect. 4.3.

4.3 APPROACH TAKEN FOR THE SENSITIVITY ANALYSIS

In performing a sensitivity analysis, two approaches can be taken. In the first, fatality estimates could be recomputed by using the same plume geometries and directions as for the baseline cases. The affected population would then be subject to fatalities, increased in proportion

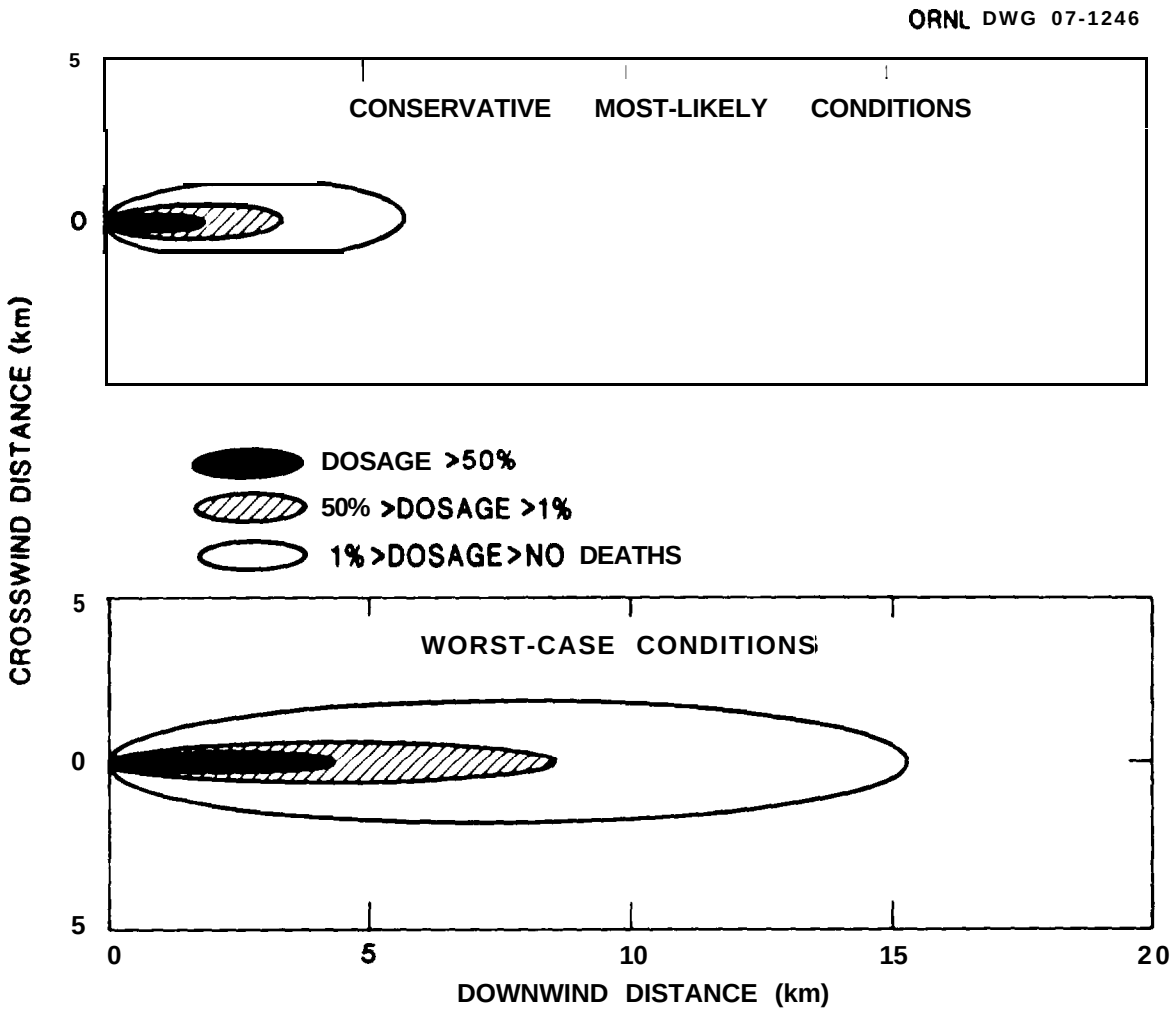


Fig. Q.l. Plume geometries for the same quantity of accidentally released chemical agent under different meteorological conditions.

to the magnitude of preselected dose sensitivity factors for infants, children, and elders. This approach has the advantage that its results can be directly compared with the existing fatality estimates in Sect. 4.3, since the same plumes and populations at risk would be considered. It has the disadvantage that any potential fatalities, among the sensitive population living outside of the no-deaths boundary, would not be included in the revised fatality estimates.

In the second approach, the plume size could be expanded to encompass the population out to a new distance that is related to an increased sensitivity, which corresponds to a fraction of the no-deaths dose. This approach would present problems with predicting plume boundaries corresponding to these scaled no-deaths doses; furthermore, the D2PC calculations for the plume geometries already contain an accuracy within ±50% of the downwind distance. This second approach has the major disadvantage that it is not directly comparable to the existing baseline fatality estimates, because expanded plume boundaries are required and larger populations at risk will be involved. For these reasons, the first approach was taken in this sensitivity analysis.

Q.3.1 DEFINING THE SENSITIVE POPULATION

Three age classes were included in the sensitivity analysis: infants, children, and elders. Infants are defined as those individuals under the age of 5; children are defined as those more than 5 but less than 10 years old; and elders are those persons older than 65 years. Members of the total population who were neither infants, children, nor elders were assumed to respond to chemical agent exposure as healthy adult males.

In the sensitivity analysis, it has been assumed that the geographical distribution of infants, children, and elders is the same as in the general population around both APG and LBAD. For APG, the proportions of the population in Baltimore County 'were taken as being representative of the entire area, because this county has the largest population near the chemical agent storage site. For LBAD, the proportions of Madison County were used. Table Q.1 reports these proportions, as well as those for nearby counties.

Q.3.2 BOUNDING THE SENSITIVITY TO DOSE-RESPONSE

In order to calculate the effects of the sensitivity of the population to chemical agent exposure, it was assumed that the three sensitive groups would have higher rates of death than the rates for the nonsensitive population. The argument has been made (V. Houk, Center for Environmental Health, DHHS, Atlanta, Ga., letter to D. Nydam, OPMCM, Aberdeen Proving Ground, Md., June 1987) that infants, children, or elders might experience fatalities when exposed to chemical agent concentrations that are somewhat less than five times lower than the no-deaths dose for healthy adult males. Recomputing the plume geometries to take this downscaled dose into account was rejected for the reasons

Table Q.l. **Population** distributions for APC **and** LBAD

Region	Sensitive population (%)				Remaining population (%)
	<5 year	5-9 year	>65 year	Total	
APG					
Baltimore County[a]	5.4	6.0	10.6	22.0	78.0
Baltimore City	6.7	6.5	12.8	26.0	74.0
Baltimore MSA	6.3	6.1	10.1	23.1	76.9
Harford Count)	7.0	8.3	6.4	21.7	78.3
LBAD					
Fayette County	6.9	7.0	8.6	22.5	77.5
Lexington MSA	7.3	6.8	9.2	23.3	76.7
Madison County"	6.6	6.1	9.0	21.7	78.3

"Population statistics for these regions were used in the sensitivity analysis for the site-specific fatality estimates.

previously stated. Instead, it was assumed that those individuals sensitive to a dose five times smaller than the no-deaths dose for healthy adult males would die at a rate five times greater than the fatality rate for healthy adult males. This assumed fatality rate would be limited only by the size of the sensitive population, such that no more than 100% of that population could be killed.

In order to bracket the uncertainty in the dose-response of the sensitive populations, the sensitivity analysis included three separate downscaled doses: two times lower than the no-deaths dose (ND/2), five times lower (ND/5), and ten times lower (ND/10). These values effectively increased the sensitivity of the affected population by factors of 2, 5, and 10, respectively. Directly associated with these increases in sensitivity were proportional increases in the assumed fatality rates; for example, the population sensitive to the ND/2 dose was assumed to die at a rate twice as high as the rate for healthy adult males.

Q.3.3 RECOMPUTING FATALITY ESTIMATES

Fatality multipliers for the three zones of each plume are presented in Table 4.2. The fatality multipliers for the sensitive population (as shown in Table 4.2) were generated as the mathematical product of the increased sensitivity (factors of 2, 5, and 10) and the fatality multiplier for the baseline case (healthy adult males); however, this multiplier could obviously never be larger than 100%.

In computing revised fatality estimates for the sensitive population, the fatality multipliers from Table 4.2 were applied within each zone of the plume to the population percentages from Table Q.l for each site. The fatalities in the balance of the population (i.e., those who are neither infants, children, nor elders) were computed using the baseline fatality multipliers. The wind direction for each plume was assumed to be the same as for the corresponding plume used in the baseline fatality estimates for APG in Sect. 4.3.1 and for LBAD in Sect. 4.3.3. In each case, a direct comparison of the revised, estimated fatalities in the total population (including infants, children, and elders) can be made to the baseline fatality estimates. That is, the plume geometries, the wind directions, and the number of persons exposed are the same; only the rates of fatality differ.

For the sake of completeness in analyzing the results from the sensitivity analysis, one other set of fatality estimates were made; each plume direction was varied in increments of one compass degree around the release site, and the largest number of revised fatalities was recomputed. There was a maximum increase of only about 1% in estimated fatalities at both APG and LBAD as compared with those from the "fixed plume direction" analysis. This percentage figure is within the range of the rounding criteria used to present tabular data and is well within the uncertainty associated with the methodology of determining population distributions and plume geometries. Thus,

Table Q.2. Fatality multipliers for sensitive populations

Boundary within plume[a]	(Baseline case)[b] ND/I	Scaled no-deaths dose		
		ND/2	ND/5	ND/10
Release point out to 50% lethal dose	0.75	1.00	1.00	1.00
50% lethal dose out to 1% lethal dose	0.25	0.50	1.00	1.00
1% lethal dose out to no-deaths distance	0.005	0.01	0.025	0.05

[a] See Fig. Q.1.
[b] See Sect. 4.2.3.1.

detailed results from the "variable plume direction" analysis have not been presented in this appendix.

4.4 DISCUSSION OF RESULTS

The revised fatality estimates that result from the sensitivity analysis are presented in Table Q.3 for APG and in Table 4.4 for LEAD. The estimates in these two tables were rounded according to the same criteria as for the tables in Sect. 4.3.1 (for APG) and Sect. 4.3.3 (for LBAD); however, the percentages presented in Tables Q.3 and 4.4 were computed from the unrounded fatality estimates.

In examining Tables Q.3 and Q.4, several trends can be identified immediately. First, the number of estimated fatalities based upon an assumed ten times sensitivity (ND/10) is not measurably different from that number estimated under the assumed five times sensitivity (ND/5). This finding indicates that the range of scaled sensitivity (i.e., ND to ND/5) represents a reasonable bracketing of differential dose sensitivity. Thus, one of the areas of uncertainty for which this analysis was designed has been answered.

Another trend is the tendency of the percentage increases to be higher for the consequence categories larger than 10 km. Those consequence categories under 10 km have smaller total populations that might be affected; the few higher percentage increases for the categories under 10 km result directly from the small number of baseline fatalities on which those percentage increases are based.

The maximum increase in potential fatalities (at ND/10) is about 70% at ND/10 above the baseline figures presented in Sect. 4.3. This value may at first seem to be extremely large and significant; however, it must be tempered by the other uncertainties imposed upon this sensitivity analysis. For example, the atmospheric dispersion calculations predict plume geometries that are accurate to within only ±50% of the downwind distance. Each estimate of fatalities in Tables 4.3 and 4.4 should thus be compared with the baseline estimate in the consequence category immediately below and immediately above the tabulated entry. It can be observed that the baseline fatality estimates for the next highest consequence category (i.e., to the next largest kilometer distance) are, in general, several times higher than those of the previous category; that is, the percentage increase between the estimated fatalities in one consequence category and the next highest is more than 100%. In no case do the fatality estimates for scaled doses in Tables 4.3 and 4.4 fall above the baseline fatality estimate for the next highest consequence category.

Q.4.1 ABERDEEN PROVING GROUND

The maximum potential increase in estimated fatalities for APG is about 70% at ND/10 (see Table 4.3). The distribution of the affected sensitive population for a typical accident consequence category (35 km)

Table 4.3. Revised fatality estimates for Aberdeen Proving Ground

(revision includes sensitivity of selected population to reduced no-deaths dose)

Consequence category[a]	Meteorological conditions	Baseline estimate	Scaled no-deaths dose[b,c]					
			ND/2		ND/5		ND/10	
0.5	Most likely	15	20	(24)	30	(65)	30	(65)
1.0	Most likely	70	75	(10)	80	(21)	80	(21)
2.0	Most likely	100	100	(14)	125	(35)	125	(35)
5.0	Most likely	550	650	(19)	825	(55)	825	(55)
10.0	Most likely	1,550	1,900	(20)	2,500	(59)	2,500	(60)
20.0	Most likely	4,800	5,300	(12)	6,000	(25)	6,000	(26)
35.0	Most likely	17,000	2 1,000	(21)	28,500	(64)	29,000	(68)
50.0	Most likely	92,000	112,000	(22)	152,000	(64)	153,000	(65)
100.0	Most likely	265,000	304,000	(15)	363,000	(37)	364,000	(38)
0.5	Worst case	15	20	(24)	30	(65)	30	(65)
1.0	Worst case	45	45	(7)	45	(7)	45	(7)
2.0	Worst case	85	95	(13)	100	(30)	loo	(30)
5.0	Worst case	215	325	(14)	315	(35)	375	(35)
10.0	Worst case	850	1,000	(22)	1,400	(65)	1,400	(66)
20.0	Worst case	2,300	2,600	(13)	3,000	(29)	3,000	(30)
35.0	Worst case	7,500	9,200	(22)	12,500	(65)	12,500	(66)
50.0	Worst case	42,000	51.000	(22)	69.500	(65)	70,000	(67)
100.0	Worst case	120,000	144.000	(20)	189,000	(57)	189.000	(57)

[a] Kilometers to ND distance.

[b] Fatality estimates have been rounded.

[c] Numbers in parentheses are the percentage increase above baseline estimate and are based on the unrounded values.

Table Q.4. **Revised fatality estimates for Lexington-Blue Grass** Army Depot

(revision includes sensitivity of selected population to reduced **no-deaths** dose)

Consequence category[a]	Meteorological conditions	Baseline estimate	Scaled no-deaths dose[b,c]					
			ND/2		ND/5		ND/10	
0.5	Most likely	0	0		0		0	
1.0	Most likely	0	0		0		0	
2.0	Most likely							
5.0	Most likely	30	35	(24)	50	(66)	50	(69)
10.0	Most likely	1,100	1,300	(22)	1,800	(65)	1,800	(67)
20.0	Most likely	3,500	4,200	(19)	5.400	(54)	5,400	(54)
35.0	Most likely	8,700	9,500	(9)	10,000	(15)	10,000	(15)
50.0	Most likely	10,500	11,500	(9)	12.000	(13)	12.000	(14)
0.5	Worst case	0	0		0		0	
1.0	Worst case	0	0		0		0	
2.0	Worst case							
5.0	Worst case	20	25	(21)	30	(63)	30	(68)
10.0	Worst case	475	575	(22)	775	(65)	775	(67)
20.0	Worst case	1,800	2,200	(21)	2,900	(63)	2,900	(64)
35.0	Worst case	4,600	5,100	(12)	5,800	(26)	5,800	(26)
50.0	Worst case	6,600	7,200	(10)	7,800	(19)	7,800	(19)

[a] Kilometers to ND distance.

[b] Fatality estimates have been rounded.

[c] Numbers in parentheses are the percentage increase above baseline estimate and are based on the unrounded values.

is presented in Fig. 4.2. This figure contains the revised, unrounded fatality estimates. Again, this figure illustrates the small difference between the ND/5 and the ND/10 scaled doses. The largest affected portion of the sensitive population at APG would be those over 65 years of age; this was to be expected, because the data in Table Q.1 show that this age group dominates the sensitive population.

Q.4.2 LEXINGTON-BLUE GRASS ARMY DEPOT

The maximum potential increase in estimated fatalities for LBAD is about 70% at ND/10 (see Table 4.4). The distribution of the affected sensitive population for a typical accident consequence category (20 km) is presented in Fig. 4.3. This figure contains the revised, unrounded fatality estimates. Again, the sensitive population is affected in proportion to the data in Table Q.l; those over 65 years old would be affected the most. Those under 5 years and those 5 to 9 years old would be expected to be affected approximately equally.

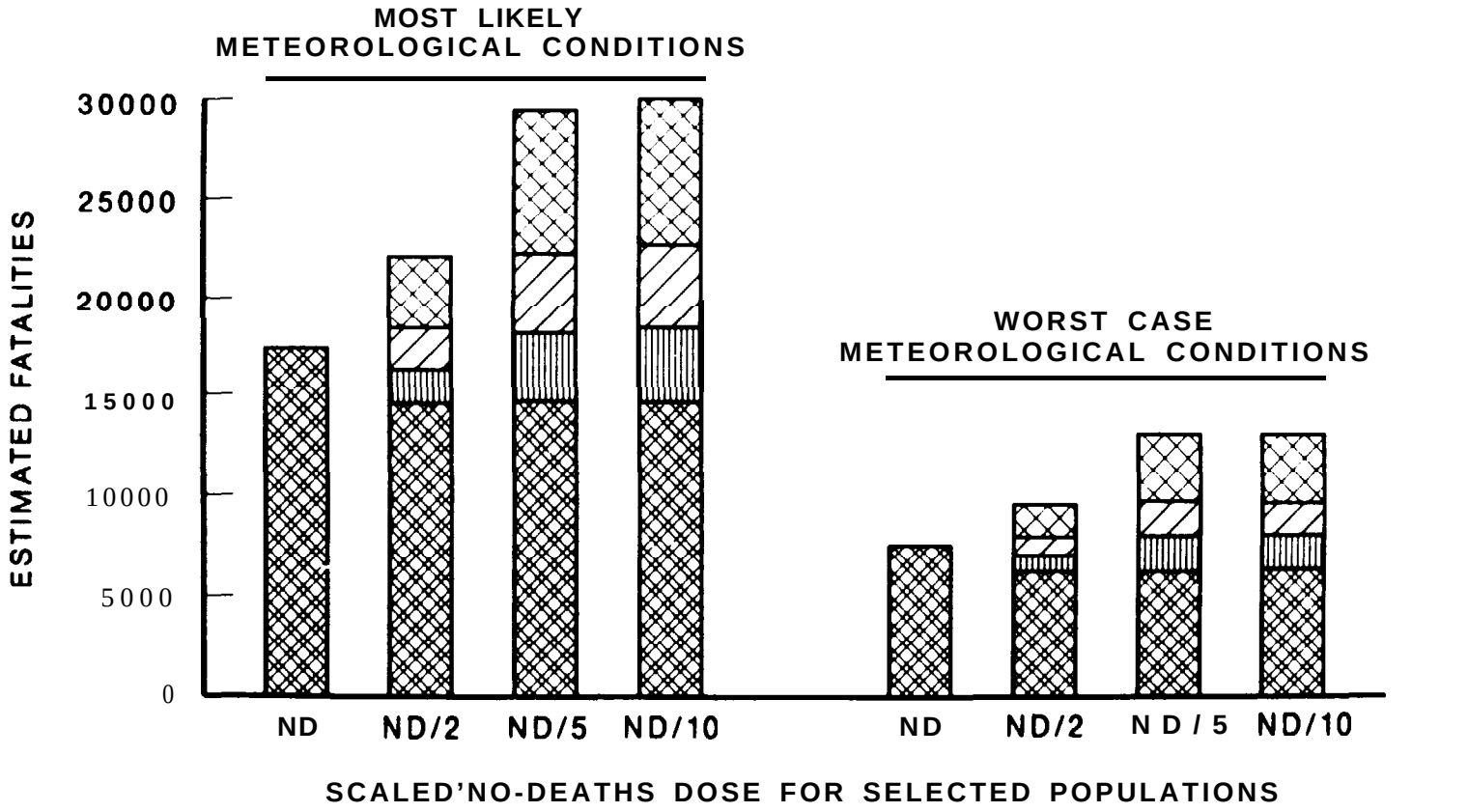


Fig. Q.2. Revised fatality estimates for sensitive populations at APG within the 35-km consequence category.

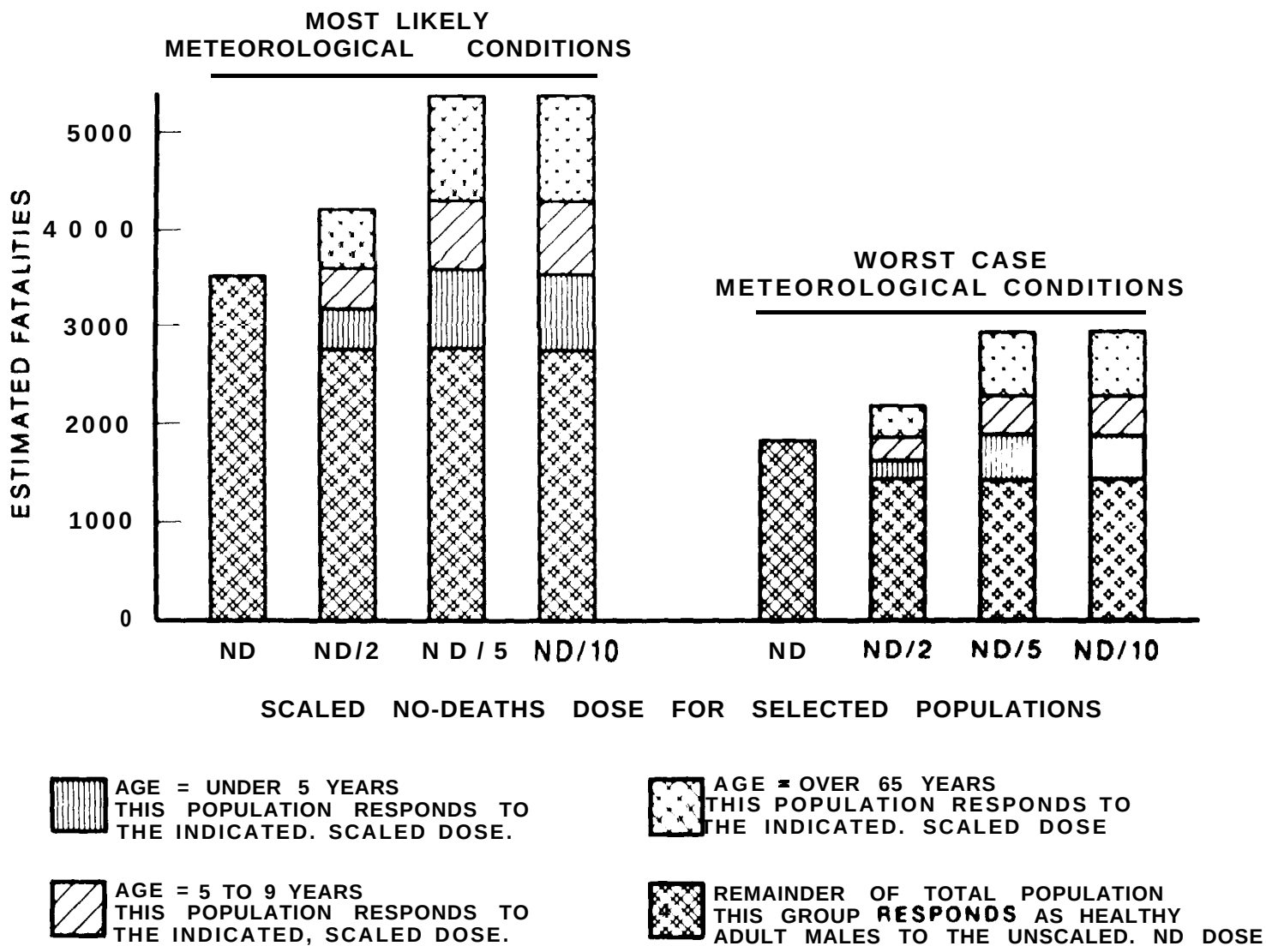


Fig. Q.3. Revised fatality estimates for sensitive populations at LBAD within the 20-km consequence category.

Appendix R

EVALUATION OF COMMUNITY GROUPS' INPUT

R.l BACKGROUND

On August 28, 1987, a public hearing was held in Richmond, Kentucky, regarding the draft programmatic environmental impact statement (DPEIS) for the Chemical Stockpile Disposal Program (CSPP). Among those representing the U.S. Army was Under Secretary James Ambrose. During the question and answer period of the meeting, a local citizen made the following comment to Mr. Ambrose:

> ...I would like to make a request of the Army that they fund the Kentucky Resource Council with $100,000 so that we may be able to conduct some of our own studies which we think might be helpful (Transcript of the CSDP Public Hearing, Thursday, August 28, 1986, p. 88).

Later that evening, Under Secretary Ambrose agreed to sponsor a local citizen study for the Lexington-Blue Grass Army Depot (LBAD) area. Subsequently, the Army also offered local citizens at the seven other sites an opportunity to undertake local studies *(Fed. Reg.* 52:4646, Feb. 13, 1987). Citizen representatives from five sites [LBAD, Aberdeen Proving Ground (APG), Newport Army Ammunition Plant (NAAP), Pine Bluff Arsenal (PBA), and Umatilla Depot Activity (UMDA); see Table R.1] were contracted to write community studies.

R.2 PURPOSE AND CONCERNS

These studies provide another avenue of input for local communities. The community studies generally focused on three objectives: (1) to perform independent evaluation of the DPEIS, (2) to review and comment on ongoing additional studies addressing specific areas of concern, and (3) to perform independent studies as necessary to address areas of concern. The community studies provided this information.

Each citizens' group was required to generate a draft report, The draft was reviewed by the Army, and written comments were provided to each citizens' group. The citizens' groups then screened the Army's comments and provided a final report. Comments were provided by the Army in the area of technical accuracy. No attempt was made to sway the citizens' groups as to the content or tone of their findings. Each group issued a final report (see Table R.2).

This appendix summarizes the concerns raised in the five citizens' groups reports. While doing this, the Army does not attempt to replace the reports with this appendix and encourages any reader interested in an in-depth study of a particular citizens' report to obtain a copy and read it at length. However, this appendix does try to single out the largest or most significant concerns and give the reader an answer as to how the Army is treating each concern. In addition, these studies critiqued the previous DPEIS and draft supporting studies and reiterated criticisms of the program. Those involved in the community studies, however, came to appreciate the program's complexities and recognized

Table R.1. Sites awarded subcontracts

Site	Subcontractor	Date awarded
LBAD	Eastern Kentucky University	Jan. 23, 1987
APG	EA Engineering, Science, and Technology, Inc.	April 28, 1987
NAAP	Concerned Citizens of Vermillion, Parke, Vigo, Fountian, and Tiperanoe counties	April 29, 1987
UMDA	Umatilla County Soil and Water Conservation District	May 4, 1987
PBA	University of Arkansas at Pine Bluff	May 6, 1987

Table R.2. Community study reports

Group	Report title	Army report number
NAAP	*Community Review Report: Disposal of One Ton Containers of VX*	SAPEO-CDE-IS-87015
UMDA	*Evaluation of the DPEIS for the Destruction of Chemical Munitions Stored at UMDA and Other Army Facilities*	SAPEO-CDE-IS-87016
APG	*Community Review Support Study: Aberdeen Area*	SAPEO-CDE-IS-87017
LBAD	*Report of Kentucky Community Study Group*	SAPEO-CDE-IS-87018
PBA		SAPEO-CDE-IS-87019

the detail of effort put into the impact statement and its supporting documents.

A number of general concerns are expressed throughout the community studies. For example, health effects; social, economic, and cultural resources (specifically emergency preparedness); and monitoring are highlighted in all the community studies. The risk analysis, plant design and operations, and selection of alternative methodology are discussed in four of the five community studies. However, specific concerns for various categories may vary from site to site. For example, with health effects, the APG community study (EA Engineering, Science, and Technology, Inc. 1987) expressed concern on health effects beyond lethality issues, including products of incomplete combustion (PICs) and principal organic hazardous constituents (POHCs). For health effects in the LBAD community study (Blackwell et al. 1987), concern was focused on the relationship between risk and health effects. A summary of community concerns by site is depicted in Table R.3.

The format of this appendix is based on a site-by-site discussion of the issues. The general community study point is paraphrased. An Army response is given, and the reader is directed to other studies or specific parts of the FPEIS for further information. Not all information or suggestions resulting from the community studies are relevant to the FPEIS but may be used in the site-specific NEPA documentation. Finally, some comments and suggestions are not relevant to the FPEIS and NEPA process and are therefore not incorporated into the FPEIS.

R.3 APG COMMUNITY STUDY

EA Engineering, Science, and Technology Inc. (1987), conducted the APG community study. The study concluded that transportation of the APG stocks is viable and merits further consideration.

R.3.1 HEALTH EFFECTS

Concern was expressed that except for lethality, other health effects were not addressed. Furthermore, the APG community study questioned whether information is available regarding chronic exposures to chemical agents. As was the case with other community studies, the APG study cited a concern regarding products of incomplete combustion (PICs) and principal organic hazardous constituents (POHCs). Finally, health concerns regarding multiple stack emissions were mentioned.

The FPEIS (Sect. 4.1.2) and Appendix B include comprehensive analyses of the toxicity of all the agents, including mustard, and reviews studies regarding short- and long-term exposure to chemical agents. Long-term chronic health hazards from mustard concentrations during normal operations could result in an annual average exposure between 0.013×10^{-6} mg/m^3 and 0.037×10^{-6} mg/m^3 at the APG boundary (Hunter and Oliverson 1987). Assuming continuous exposure (24 h/d, 5 d/week, for 5 years) at this location, a person's cancer risk wold be between 2×10^{-7} and 8×10^{-8}.

Table R.3. Community concerns

	APG	LBAD	NAAP	PBA	UMDA
Health effects	X	X	X	X	X
PICs and POHCs	X	X			
Multiple stacks		X			
Meteorology	X		X		X
D2PC model	X				
Social, economic, and cultural	X	X	X	X	X
Emergency preparation	X	X	X	X	X
Surface water and groundwater			X		
Ecological resources			X		X
Aircraft activity		X		X	X
Risk analysis	X	X	X		X
Transportation	X	X			X
Low temperature	X	X			
Barge	X			X	
Packaging		X			
Seismic risk					X
Monitoring	X	X	X	X	X
Mitigation		X			
Plant designs/operations	X	X	X	X	X
Selection of alternative	X	X	X	X	
Other	X	X			X

R.3.2 METEOROLOGY

The APG community study expressed concern regarding the use of the D2PC modeling, especially for determining health effects. A detailed description of the D2PC model is located in Appendix K and in Sect. 4.2.2.1 of the FPEIS. This model is used in standard atmospheric dispersion modeling. Data on the type of release, the duration of release, and vapor depletion are used to predict doses of an agent expected at locations downwind of the release. These doses correspond with what could result in human fatalities.

R.3.3 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

The major concern under this category by the APG community study is emergency preparedness. The study calls for site-specific recommendations and Army commitments regarding emergency preparedness. The FPEIS (Appendix L) and the *Emergency Response Concept Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987) pertain to programmatic recommendations regarding emergency planning and response. Important programmatic planning concepts include emergency response management, protective actions and responses, and emergency resource and information management. Specific issues, recommendations, and commitments will be made during the site-specific NEPA analyses.

R.3.4 RISK ANALYSIS

The community study found the risk analysis generally thorough. However, the risk analysis was considered difficult to comprehend. Furthermore, the study contended that the criteria used to screen alternatives was not clear.

The FPEIS is written more clearly, which should help in understanding the risk analysis. In addition, site specific information is given on four measures of risk for each alternative. These four measures of risk include probability of one or more fatalities, maximum number of fatalities, expected fatalities, and person-years of risks over the duration of the CSDP. In addition, a measure of environmental risk (expected plume area) has been considered. Risk information for each site and alternative is also presented in graphic form (see Sects. *2.5, 2.6, 4.3,* 4.4, and Appendix J).

Classified data present problems in describing the risk analysis in a clear fashion. However, each community study group may have a person with a security clearance to look at the data to help others understand the risk analysis.

R.3.5 TRANSPORTATION

A number of different transportation alternatives (e.g., by air) have been studied in detail by the Army. Under Secretary Ambrose decided not to include the barge option in the FPEIS. Three reasons are behind

his decision. First, rail transportation serves as an adequate transportation option to consider for a national programmatic EIS. Lengthy studies would be needed to analyze any potential impacts the barge option may have on the Chesapeake Bay and its tributaries. Finally, a national programmatic decision does not eliminate subsequent consideration of site-specific alternatives at a later date (see Appendix S).

Transporting the chemical agent in low temperatures was suggested in the community study as a way of mitigating transportation risks. Studies demonstrated that the transportation of agent at low temperatures does not mitigate the impacts of every type of accident. Bulk mustard items would be moved at reduced temperature. The discussion of low-temperature transportation is found in Sect. 4.5 of the FPEIS.

R.3.6 MONITORING

Concern was expressed in the APG community study that no monitors were proposed to facilitate public warning during an accident. Monitoring and warning systems are discussed in Appendix L and the *Emergency Response Concept Plan* (Jacobs Engineering Group Inc., and Schneider EC Planning and Management Services 1987).

Scenarios that would create an incident necessary to warn the public immediately would be situations where the incident itself would be a better warning than a monitor. For example, explosives released from a drop or puncture or a fire-generated release will be visually detected without the need for monitors. However, personnel from the area still need to initiate communications and emergency response capabilities (see Sect. 4.5.5.1).

R.3.7 PUNT DESIGN AND OPERATIONS

The community study requested that the FPEIS give more information regarding plant design, incineration, and products of incomplete combustion. Section 2.2.3 of the FPEIS outlines the historical development, technology description and steps involved in the disposal process. (Appendix C of the FPEIS adds further detail.)

Furthermore, the APG community study suggests that more time be allowed to explore and/or test other technological alternatives. Alternatives such as those suggested in the community study are described in Sect. 2.3.6 of the FPEIS.

R.3.8 SELECTION OF PREFERRED ALTERNATIVE

The community study questions whether some important information was left out of the DPEIS that would have changed the preferred alternative. The study questions whether the DPEIS contained enough information to allow one to make an informed decision. Furthermore, the study recommended that hybrid alternatives be considered for the disposal of the chemical agent.

The FPEIS includes more information, including in Sect. 3 site-specific descriptions of the eight storage sites. More detail is also given on the methodology in selecting the preferred alternative (see Sect. 2.6 and Appendix M). Certain hybrid approaches are included in the FPEIS in Sect. 2.3.

The community study also criticized the generic approach. However, the study noted that using such an approach for studying transportation options is considered a legitimate approach. The FPEIS now gives site-specific information for site descriptions (Sect. 3) and impact analysis (Sect. 4). The generic approach is still used for describing and analyzing transportation corridors.

R.3.9 OTHER ISSUES

The APG community study generated much data, noting that the APG community hoped a site-specific assessment would be undertaken. Furthermore, the community group also hoped that data gathered from the community study will be incorporated into the site-specific assessment.

The Army has committed to a site-specific assessment for APG (and the seven other storage sites). Furthermore, data from the community study, where applicable, will be incorporated into the site-specific assessment.

A suggestion was also made that if on-site disposal is chosen, that the incinerator site should be moved to Eagle Point, which is 4.8 to 5.6 km (3 to 3.5 miles) south of the proposed incinerator site. This move would result in a large number of people no longer being within the 10-km-radius emergency planning zone (EPZ) and would solve some emergency preparedness problems. In addition, potential low-level exposure of humans to agent by-products would be reduced. This suggestion will be explored in the site-specific documentation,

R.4 LBAD COMMUNITY STUDY

The LBAD community study was completed through a contract with Eastern Kentucky University (Blackwell et al. 1987). The study recommends that the LBAD stockpile should be flown to Tooele Army Depot (TEAD) for incineration.

R.4.1 HEALTH EFFECTS

The LBAD community study contends that the health effects and risk analysis should have been combined as an integrated epidemiological study. Since changing the approach at this date is not possible, the study suggests that accidents, releases, and process emission be presented in clearer form.

The FPEIS, including the relationship between risk analysis and health effects, is written now in clearer fashion. Relationships between risk and health effects are now clearly explained in Appendix B of the FPEIS. For example, a study by Hunter and Oliverson (1978) shows that

long-term chronic health hazards from mustard concentrations during normal operations could result in an annual average exposure between 0.013 x 10^{-6} ❑ g/m3 and 0.037 x 10^{-6} mg/m^3 at the APG boundary. Assuming continuous exposure (24 h/d, 5 d/week, for 5 years) at this location, a person's cancer risk would be between 2 x 10^{-7} and 8 x 10^{-8}.

Tables illustrating relationships between risks, alternatives, and deaths are now depicted in Sects. 2.5 and 4.2 of the FPEIS. Measures of risk include maximum individual risk, probability of one or more fatalities, maximum number of fatalities, and expected fatalities.

R.4.2 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

Site-specific concerns are raised regarding economic impacts for the LBAD area. The impact analyses (see Sect. 4 and Appendix P) show that no negative economic impacts would occur at any of the sites during normal operations of the incinerator under the on-site disposal option, This finding is consistent with similar studies (see Appendix P).

Because areas around LBAD are heavily populated, the community study group has a number of questions regarding emergency preparedness. Some of their many questions, for example, focus on warning, evacuation, classification of event, disaster management, resources for communities and hospitals, and financial responsibility.

A number of salient concepts are used to devise the programmatic emergency response approach. The EPZs (the immediate response zone, protective action zone, and precautionary zone) are used to devise a general planning strategy. Emergency response concepts play a central role in devising planning. These concepts include emergency response management, protective actions and response, emergency resource and information management, and program development and implementation. The programmatic philosophy regarding emergency preparedness can be found in Appendix L of the FPEIS and in the *Emergency Response Concept Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987).

R.4.3 AIRCRAFT ACTIVITY

The LBAD community study group believes that airspace over LBAD should be restricted. This restriction, they believe, would mitigate many of the high-consequence accidents found in the risk analysis. The FPEIS (see Sect. 3) describes aircraft activity around all the storage sites. The Army will pursue restricted airspace (see Sect. 4.5.1.3).

R.4.4 RISK ANALYSIS

Members of the community study believe that there are a number of errors in the risk analysis. They are especially concerned about the risk data base, the use of different data bases, and the lack of human error factors in the analysis. Furthermore, they contend that more

scrutiny has been given to the on-site disposal option and that perhaps data have been skewed to favor on-site disposal.

Many of these concerns and suggestion have been incorporated into the current risk analysis (see Appendix J). Efforts have been made to make the impacts based upon the risk analysis more understandable to the general reader. In addition, new figures have been devised to present more clearly the risk analysis results (see Sect. 4).

R.4.5 TRANSPORTATION

Transporting the chemical agent in low temperatures was suggested in the community study as a way of mitigating transportation risks. Studies demonstrated that the transportation of agent at low temperatures has limited value, and the FPEIS commits to transport of bulk mustard in a frozen state (see Sect. 4.5.1.3.4).

Packaging during transportation is also a concern. The study group contends that the issue of packaging needs further study. A transportation panel of experts was convened by Army for advice on packaging (and other issues relevant to) the chemical agent. The panel stressed that packaging was the most crucial component in the entire transportation system because (1) it provides containment of the hazard, (2) it affects the extent and manner of handling of the munitions, and (3) it has a significant influence on the choice of equipment used in transport. Packaging should be considered in a system context, taking into account the munition type, bracing and dunnage, overpacking, human factors, and vehicles, The expert panel arrived at four packaging criteria the Army should follow. First, the system should provide redundant protection against agent release during normal transport. Second, the system should prevent agent release into the environment in the event of an accident. Third, the transportation container should be compatible with standard commercial-handling and carrier equipment. Finally, the transportation containers should be equipped with automated agent and temperature monitors and alarms (see Appendix G, Sect. G.5.2).

Also, an overall concern evolves around initial removal of the agent from its storage site for transportation to the incinerator, train, or air transport. The Army's procedures for removing an agent from storage for transport is found in Sect, 2 and Appendix G (Sect. G.5.3). Three operational procedures are central to any type of removal of an agent from storage. They include (1) activities necessary to prepare, package, and move the munitions from the site where they are currently stored to the loading area where off-site transport will begin; (2) procedures for handling munitions or ton containers once they reach the destination site where destruction will occur; and (3) activities involved in preparing and moving munitions and ton containers from the current storage location to a nearby destruction facility on-site.

R.4.6 MONITORING

The main monitoring issue in the community study looks at real-time low-concentration-measuring devices. Other issues on monitoring generally are met with approval.

The specific type of monitoring (real-time low-concentration) and specific monitoring plans have not been developed. Konitoring and warning systems are discussed in more detail in Appendix L and in the ***Emergency Response*** Concept Plan (Jacobs Engineering Group, Inc., and Schneider EC Planning and Management Services 1987).

R.4.7 MITIGATION

The community study is not sure to what degree mitigation procedures are employed in calculating the risk analysis. Furthermore, the group asked for further information on how the Army may mitigate various accidents. Finally, they indicated that the relationship of probabilities, the **D2PC** model, and emission rates needs to be clarified.

The sections on risk analysis and how risk is used to determine impacts have been rewritten in the FPEIS and are now easier to understand. However, writing about risk is not a simple task. The relationships between risk, deaths, and the use for the **D2PC** model should be easier to understand in the FPEIS. Programmatic and some site-specific mitigation recommendations are found in Sect. 4.5.

R.4.8 **PLANT** DESIGN AND OPERATIONS

The LBAD community study cites concerns over the use of a new technology in a heavily populated area. Furthermore, questions arise regarding the types and amounts of emissions, and disposal of waste materials. Appendix C provides a detailed summary of the disposal technology.

R.4.9 SELECTION OF PREFERRED ALTERNATIVE

The community study is concerned with the generic and programmatic approach used in selecting the preferred alternative. The members of the community study group also question what they feel are unclear and unspecific procedures for determining the preferred alternative. Finally, they believe that the computer model developed for selecting a preferred alternative is in the right direction. However, they believe that too many questions and holes exist-to make it a viable tool for decision making.

The FPEIS has more site-specific information incorporated into the methodology used for selecting the preferred alternative than the DPEIS had. Human health is the most important factor in determining the preferred alternative. More information is given on the preferred alternative methodology in Sects. 2.6, 3, and Appendix M.

R.4.10 OTHER ISSUES

The LBAD community study expresses concern regarding trust between the Army and the local community. These people are concerned with the distrust of the Army that the local citizens and public officials have. The FPEIS recognizes this problem. Site-specific responses regarding trust will be in the site-specific documents. A programmatic discussion of the Intergovernmental Consultation and Coordination Board outlines processes and procedures regarding how stronger working relationships can be built and maintained between the Army and local communities during the CSDP.

The study contends that information gaps, incomplete analysis, and logical holes exist in the DPEIS. The FPEIS provides further information and analyses regarding methodologies and conclusions. Furthermore, the FPEIS, we believe, is more readable.

The community report requests detailed procedures for disposal of incineration by-products. The members of the study group feel that the handling of waste materials is neglected. The FPEIS deals with the programmatic nature of waste disposal, including Resource Conservation and Recovery Act (RCRA) permitting. A detailed description of waste management will be found in the site-specific documents.

R.5 NAAP COMMUNITY STUDY

The NAAP community study was conducted by the Concerned Citizens of Vermillion, Parke, Vigo, Fountain, and Tippecanoe counties (Heiser et al. 1987). The study group recommended that the whole continental United States (CONUS) stockpile be transported by rail to a national disposal site characterized by a profound remoteness from human habitation, animal population, or vegetation. The Great Salt Desert was given as one possible location.

R.5.1 HEALTH EFFECTS

Concern was expressed regarding the long-term, low-level chronic effects of Vx. Specifically, the study says that chronic sublethal dosages of VX, which is similar to other closely related methylphosphonates, could lead to permanent CNS effects. These concerns are addressed in an extended, in-depth discussion of low-level chronic sublethal dosages of VX (see Appendix B, Sect. B.2.3 and especially B.2.3.3).

R.5.2 METEOROLOGY

The suggestion was made that site-specific dispersion modeling be used with meteorological data specific to the area surrounding NAAP. If possible, local topography should be incorporated into the model.

The type of modeling requested is generally available only on mainframe computers. However, the Army will investigate any breakthroughs regarding dispersion models for PCs. Any new developments

will be presented to the public. The advantages and liabilities of various dispersion models can be found in Appendix K.

R.5.3 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

The community study criticizes the generic approach for describing populations. A whole section of the community report was devoted to issues regarding emergency response. The main point is that local emergency preparedness and response for a VX accident is inadequate.

Section 3 of the FPEIS no longer uses generic categories for describing the eight storage sites. Rather, a concise description of key social resources is given. These data are used to determine impacts (see Sect. 4.2 and Appendix P).

The FPEIS (Appendix L) and *Emergency Response Concept Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987) address programmatic recommendations regarding emergency planning and response. Important programmatic planning concepts include emergency response management, protective actions and responses, and emergency resource and information management. Specific issues, recommendations, and commitments will be treated in the site-specific analyses.

Psychological stress caused by the CSDP was noted in the community study. The issue of psychological stress from the chemical stockpile disposal program or a possible accident is addressed in Appendix L.

R.5.4 SURFACE WATERS AND GROUNDWATERS

Concern is expressed in the community study on potential groundwater contamination of the Wabash River. This river is the source of drinking water for many surrounding communities. A site-specific analysis of water impacts is found in Sect. 4.3 and Appendix N. Any major accident could have severe consequences for the Wabash River. (see Sect. 4.3.4). Spillage of agent during on-site transport could also affect surface waters and groundwaters. Site-specific measures will be addressed (see Sect. 4.5.2.1).

The community study also raises questions regarding *current* contamination of deep wells from depositing items contaminated with VX in the past. This is *not* an appropriate issue for this FPEIS.

R.S.5 ECOLOGICAL RESOURCES

The community study presents a well-documented inventory of ecological resources in the NAAP area. The study has uncovered some information that was previously unavailable, especially regarding endangered species. The amount of information requested is too voluminous for the FPEIS. This level of detail is useful and, where appropriate, will be incorporated into the site-specific document for NAAP.

R.5.6 RISK ANALYSIS

The community study raises concerns over the risk analysis and associated assumptions regarding probabilities of risks. For example, the community study members believe that a lightning strike on the container building could be more hazardous and more probable than a tornado or earthquake striking the area.

The methods and assumptions of the risk analysis are detailed in the risk analysis report (Appendix J). The programmatic concerns of the community study are generally answered in this report. Site-specific concerns and the methods for interpreting probabilities of risk analysis, however, are not explicitly covered in Appendix J.

R.S.7 MONITORING

The community study group expressed concerns that appropriate monitors are not currently installed at and around the NAAP area. The members of the community study group contend that monitoring would allow monitoring of long-term health impacts and improve emergency response in the case of an accident. Monitoring and warning systems are discussed in Appendix L and in the *Emergency Response Concept Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Services 1987).

R.5.8 PLANT DESIGN AND OPERATIONS

The citizen study asks for further research on the filtering system. More detailed comments are also made on the incinerator, furnace, air pollution equipment, and brine dryer. The community study group also analyzes other methods for disposing of the chemical agents. These include liquid-injection incineration, rotary-kiln incineration, starved-air combustion, and other thermal modes; biological methods; irradiation; chemical oxidation; and dilution for use as a pesticide. The study's analysis concludes that other incineration methods, such as plasma technology, should be considered.

Although some of these alternatives have merit, the Army has decided to use the chemical agent munitions disposal system/Johnston Atoll chemical agent disposal system (CAMDS/JACADS) process of incineration. Section 2.2.3 of the FPEIS explains the selection of the incineration plant design over other technologies. Reasons for this technology include its strong recommendation from the National Academy of Science and its development and success of disposal since 1972 at Rocky Mountain Arsenal.

Finally, the NAAP community study expresses concerns over the training and management of incinerator personnel. The procedures that the Army plans to use to maintain a high standard of training, management, and operations are found in Sect. 2.3.2.6 of the FPEIS. These include drawing upon contractors with successful experience in managing similar programs; internal and external documentation of

organizational activities; and inspections assessing disposal operations, safety, security, surety, and chemical accident response and assistance.

R.5.9 SELECTION OF PREFERRED ALTERNATIVE

The community study questions the DPEIS's preferred alternative of on-site incineration at the eight sites. Rather, these people propose that a national disposal facility be built at a remote site in Utah. The logic behind choosing from three different disposal alternative (on-site, regional, national) is found in Sect. 2. The criteria used for the preferred alternative is found in Sect. 2.6 and Appendix L. The most important factor in determining the preferred alternative is human health.

R.6 PBA COMMUNITY STUDY

The PBA community study was contracted through the University of Arkansas, Pine Bluff, Arkansas (Demecs et al. 1987). They recommend on-site disposal as the environmentally preferred decision.

This study has included a number of findings and recommendations. Many of these are pertinent to the program. However, these findings and recommendations are more relevant to site-specific considerations. Therefore, in this section only those issues that are of a programmatic nature or point out why some of the other major recommendations (e.g., emergency preparedness) are more suited for site-specific documentation are discussed.

R.6.1 HEALTH EFFECTS

The community study concludes that more information is needed on the effects of chemical agents and that generalizing from animal studies to human effects should be a starting place. The study group suggests that other recent potential data sources on chemical warfare (i.e., Afghanistan, Iran) should be explored.

Appendix B of the FPEIS presents a thorough overview of health effects, including the few studies available on how chemical agents effect humans. Reviews of studies on acute (see Sect. B.2.2) and chronic effects (see Sect. B.2.3) can be found in Appendix B. However, extrapolating chronic health effects from animal data to human effectiveness is a problem basic to evaluating therapies for nerve agent poisoning from nerve agents (see Appendix B, p. B-92).

R.6.2 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

Two general concerns about social, economic, and cultural resources are addressed here. The first is that more site-specific information should be gathered for all socioeconomic resources. Second, the community study expresser both concerns and opportunities with emergency planning. These include, for example, using the CSDP as a way of

expanding national civil defense.

The site-specific assessment of the eight storage sites will all have more information. Data cited in the community study may be useful in the site-specific NEPA document.

The community study's general concerns about emergency preparedness are addressed in Appendix L and the *Emergency Response Concept Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987). However, opportunities such as using the CSDP as a way to improve the nation's civil defense are not within the realm of this program or NEPA.

R.6.3 ECOLOGICAL RESOURCES

Concerns of the disposal program effecting biota are expressed in the community report. The report states that more site-specific information is needed and that the on-site incineration option would have the least impacts on biota.

Further information about biota should be found in site-specific documents. The importance of biota and endangered species in the disposal selection process can be found in Sect. 2 of the FPEIS. However, other issues, such as human health, have a higher priority than issues, such as endangered species, in determining the preferred alternative.

R.6.4 MONITORING

The community study group expressed concerns that appropriate monitors are not installed and will not be installed outside the fences of PBA. The community study contends that off-site monitoring would facilitate the determination of long-term chronic health effects and contamination outside the PBA fences. A major theme within the community study is to develop a environmental data baseline for determining any possible effects during normal operations or storage. Monitoring and warning systems are discussed in Appendix L and in the *Emergency Response Concept Plan* (Jacobs Engineering Group Inc., and Schneider EC Planning and Management Services 1987).

Small chronic leaks that are not immediately recognized by visual means will be detected by sensitive perimeter monitors. At this time, off-site monitoring is not being considered by the Army. The monitoring report (U.S. Army 1987a), a support study to the FPEIS, details the Army's philosophy toward monitoring.

R.6.5 AIRCRAFT ACTIVITY

The community study recommends that in order to lower the risk of a large agent release, airspace over chemical storage areas should be restricted. The FPEIS (see Sect. 3) describes aircraft activity around each of the storage sites. The Army will pursue the restriction of airspace (see Sect. 4.5.1.3.1).

R.6.6 PLANT DESIGN AND OPERATIONS

The community study suggests that a Dual Technology Evaluation Program be considered. This includes an extension of the 1994 deadline to dispose of the chemical agent. The community study group recommended that cryofracture be considered as a possible disposal mechanism. The reasoning for eliminating cryofracture is detailed in Sect. 2 of the DPEIS.

Further consideration of this option is now described in Sect. 2.3 of the FPEIS. The principal disadvantage of the option is an approximate eight-year delay in the completion of this program with its inherent additional risks of storage.

Finally, the community study suggested that the Government-Owned/Contractor Operated (GO/CO) method of operations should not specifically go to the lowest bidder and that mechanisms should be put into the system to ensure safety. Contracts for this program will be based on considerations other than cost alone. Section 2.3 describes the management processes that will be used.

R.6.7 SELECTION OF PREFERRED ALTERNATIVE

Questions were raised throughout the community study regarding the generic approach for the selection of the preferred alternative in the DPEIS. In the FPEIS, the generic approach is no longer used for site-specific information and analysis. The study also recommends that community involvement continue during the CSDP. The role of the community and local officials is discussed in Sect. 4 regarding the Governmental Coordination Board.,

This is the only community study that concurred generally with the DPEIS preferred alternatives. However, the study stated that the barge option for APG should be given full consideration.

R.7 UMDA COMMUNITY STUDY

The Umatilla community study (Umatilla County Soil and Water Conservation District 1987) was conducted by the Umatilla County Soil and Water Conservation District. It recommends that the on-site incineration alternative be chosen for disposal of the chemical stockpile at UMDA. The study points out that the incineration permit is thorough and should be approved. The on-site incineration of the UMDA stockpile is the preferred disposal alternative. However, the study raises a series of concerns that are addressed in the subsequent sections.

R.7.1 HEALTH EFFECTS

The community members would like to see analyses of how chemical agents affect reproduction, genes, and cancer. Information concerning chronic health effects, including PICs and POHCs, should be considered in the development of the risk analysis. The environmentally preferred

alternative takes into consideration the potential impacts of normal operations and accidental releases. A detailed summary of chronic health effects of chemical agent, the effects of PICs and POHCs, and related topics are presented in Appendix B. Several measures of risk are discussed in Appendix J and Sect. 4.3. The environmentally preferred alternative (see Appendix M for discussion of methodology) is presented in Sect. 2.6.

Control limits outlined in Sect. 4.1 (Table 4.2) have been established by the Surgeon General's panel and will be in effect during normal operations of agent disposal procedures. All available information indicates that exposure to nerve agents at concentrations much greater than these do not cause mutations or cancer, do not damage the fetus, and do not cause other reproductive problems (see Sect. 4.1 and Appendix B). While chronic impacts on biological organisms, and particularly among people, have been observed only after acute exposures and while technological monitors can detect agent levels far below these levels, biological monitors are recommended to account for any potential cumulative effect. In addition, elaborate pollution abatement systems are incorporated in the incineration facility design to collect potential combustion products and to ensure that their emissions do not exceed federally regulated standards.

R.7.2 METEOROLOGY

A number of interrelated concerns about meteorology are listed in the UMDA community study. First, they recommend that a full-time atmospheric scientist be employed during the day shift or at the storage/disposal sites. More specific comments pertain to the D2PC model. Overall, the study members felt that the model is inadequate for predicting far-downwind concentrations of agent. Second, material accidentally released during incineration should be input into the D2PC model. Finally, the report states that a whole spectrum of releases using a revised D2PC model be employed.

The D2PC model is described in Appendix K and Sect. 4.2.2.1. The model is used to model atmospheric dispersion of accidental releases. The type and duration of release, and a vapor-depletion technique are used to predict doses of agent expected at locations downwind of the release. These doses correspond with what could result in human fatalities. The transport and diffusion portion of the model is a Gaussian-plume model, which is the standard analytical technique. The Gaussian-plume model has some restrictions that must be recognized to avoid the introduction of substantial error. Approaches discussed in Appendix K are used to bound these problems.

While D2PC does not account for the impacts of topography or changes in wind velocity or atmospheric stability with time and location, the resulting biases are systematic. Hence, the D2PC results provide an adequate meteorological basis for making programmatic decisions. Site-specific meteorological models will be required prior to start of

chemical demilitarization operations. These site-specific models can account for the specifics of changes in wind velocity, stability, and specific topographic influences on local meteorological conditions.

The results of these models should be used in the development of daily operating procedures, given the kinds of munitions, amount and type of agent, operations to be performed, and likely meteorological conditions. This preplanning of potential emergency responses provides a daily framework for immediate action should accidents occur. These actions can begin while the site-specific dispersion model is used to predict the expected distribution of the actual release. An on-call meteorologist should be available to interpret the results of the site-specific model as they become available and then determine the operating procedures of the day. Such operating procedures will require updates daily and at other times when changes in meteorological condition warrant.

R.7.3 SOCIAL, ECONOMIC, AND CULTURAL RESOURCES

The community study cites concerns regarding emergency preparedness and information release if an accident occurs. Programmatic emergency preparedness procedures and recommendations are found in Appendix L of the FPEIS and in the Emergency *Response* Concept *Plan* (Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987). Emergency response guidelines are being developed on the basis of the emergency response concepts presented in the *Emergency Response Concept Plan.* A single command center on-site and an emergency operations center off site are currently recommended; however, site-specific circumstances may alter this guideline to an adequate equivalent or better. Part of the site-specific emergency response development is to identify emergency response personnel on-site at the facility and in potentially impacted communities. These emergency response organizations will need to establish working relationships that afford streamlined communication during any emergency should they arise.

R.7.4 ECOLOGICAL RESOURCES

The UMDA community study suggests that toxicological impacts on the agriculture of an area be considered. The assessment of potential impacts in the Umatilla area are discussed in Sect. 4.3.8; however, site-specific impact assessment should include additional impact details regarding toxicological impacts on agriculture and surface and ground waters in the area.

R.7.5 AIRCRAFT ACTIVITY

The UMDA community study suggests that airspace be restricted over the storage and demilitarization facilities plus 4.8 km (3 miles) around UMDA. Furthermore, airspace restriction should begin immediately. Section 3 of the FPEIS now details airspace activity around all storage

sites. The Army will pursue restricted airspace (see Sect. 4.5.1.3.1).

R.7.6 RISK ANALYSIS

Concern is expressed regarding internal events (e.g., spontaneous firing of M55 rockets) being included as a serious accident in the risk analysis. The problem of destabilization of rocket propellant in M55 rockets resulted in an Independent Evaluation/Assessment of rocket [115 mm: Chemical Agent (GB or VX),] M55 (U.S. Army 1985). A series of programs were initiated to assess the destabilization problem and continue to monitor the situation (Science Applications International Corporation 1985).

This assessment, which included sampling and testing of a significant number of rockets at each site, concluded that continued storage of the M55 rockets is not an immediate hazard. A second round of testing is currently in progress.

The community study suggests that the use of conditional probabilities frequencies be developed on a per-munitions basis to determine accident probabilities. This per-munitions basis was used in conjunction with nonexternal events of the CSDP risk analysis. This includes accidents during handling, processing, and transportation. However, external accidents, such as earthquakes and airplane crashes, are treated on an annual basis.

R.7.7 SEISMIC RISK

The community notes some concern about earthquakes in the UMDA area. To prevent accidents resulting from earthquakes, the study suggests mitigative measures (e.g., redesign tanks, transfer lines) be taken. Section 3 of the FPEIS describes the earthquake risk for each site. Appendix H and the mitigation study (U.S. Army 1987b) give information on mitigative actions for the earthquake risk.

R.7.8 PLANT DESIGN AND OPERATIONS

The community study concludes that a single document should describe the overall operational command and control requirements. Currently, command and control are described in Sect. 2 of the FPEIS. More detail on operational command and control are currently under development based upon the CAMDS experience.

The community study suggests a three-year delay of on-site operations until the Tooele and JACADS systems are tested. A failure during this time, the study suggests, would put the program on hold. The delay-disposal option suggested above is described in Sect. 2 of the FPEIS.

R.7.9 OTHER ISSUES

The community study suggests that the Army issue a monthly newsletter summarizing nonclassified information regarding the CSDP. New items would include successes, events, incidents, and accidents. Such information dissemination would help prevent the beginning and circulation of rumors regarding the storage disposal of the chemical agents. In addition, the community study reports that ways to include community input should be devised. The Intergovernmental Consultation and Coordination Board, detailed in Sect. 4.5.6 of the FPEIS outline procedures, such as those described above, to allow information flow and community input and participation into the CSDP.

The **UMDA** community study also suggests that assumptions for all models and methodologies be clearly delineated. The appendices of the FPEIS describe in great detail the assumptions. For example, Appendix K describes the logic behind the **D2PC** code used for dispersion modeling; Appendix L outlines assumptions used for the socioeconomic impact analysis.

REFERENCES FOR APPENDIX R

Blackwell, O., et al. 1987. *Report of Kentucky Community Study Group,* Eastern Kentucky University, Richmond, Ky. (SAPEO-CDE-IS-87018).

Demecs, D., et al. 1987. *Findings and Recommendations: A Systematic Approach to the CSDP DPEIS and the Reports to Drafts Tiered to It (Draft),* University of Arkansas, Pine Bluff, Ark. (SAPEO-CDE-IS-87019).

EA Engineering, Science, and Technology, Inc. 1987. *Community* Review *Support Study: Aberdeen Area,* Sparks, Md. (SAPEO-CDE-IS-87017).

Federal Register Feb. 13, 1987. 52:4646.

Heiser, C., et al. 1987. *Community Review Final Report: Disposal of Ton Containers of VX,* Concerned Citizens of Vermillion, Parke, Vigo, Fountain, and Tippecanoe counties, Newport, Ind. (SAPEO-CDE-IS-87015).

Hunter, C. H., and Oliverson, F. W. 1987. *Air* Pollution *Consultation #43-21-1229-87, Evaluation of Multiple Incinerator* Air *Quality Impacts, Edgewood Area, Aberdeen Proving Ground, Md., May 1987,* U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, Md.

Jacobs Engineering Groups, Inc., and Schneider EC Planning and Management Service 1987. *Emergency Response Concept Plan.*

Science Applications International Corporation 1985. *M55 Rocket Disposal Program* Study: *Probabilities of Selected Hazards* in *Disposition of M55 Rockets,* La Jolla, Calif.

Umatilla County Soil and Water Conservation District 1987. *Evaluation of the Draft Programmatic Environmental Impact Statement for the* Destruction *of Chemical Munitions Stored at the Umatilla Army Depot and Other* Army Facilities 1987, Pendleton, Oreg. (SAPEO-CDE-IS-87016).

U.S. Department of the Army 1985. *Independent Evaluation/Assessment of Rocket, 115MM: Chemical Agent (GB or VX), M55.* U.S. Army Material Systems Analysis Activity, Aberdeen Proving Ground, Md.

U.S. Department of the Army 1987a. *Chemical* Stockpile *Disposal* Program *Monitoring Concept Study,* Program Executive Officer--Program Manager for Chemical Demilitarization, Aberdeen Proving Ground, Md.

U.S. Department of the Army 1987b. *Mitigation of Public Safety Risks of the* Chemical Stockpile *Disposal Program,* Program Executive Officer--Program Manager for Chemical Demilitarization, Aberdeen Proving Ground, Md.

Appendix S

OCEAN TRANSPORT OF ABERDEEN PROVING GROUND STOCKPILE: BASIS FOR ELIMINATING THIS ALTERNATIVE

DEPARTMENT OF THE ARMY
OFFICE OF THE UNDER SECRETARY
WASHINGTON, D.C. 20310-0103

October 21, 1987

MEMORANDUM FOR PROGRAM MANAGER FOR CHEMICAL DEMILITARIZATION

SUBJECT: Ocean Transport of Aberdeen Proving Ground Stockpile--INFORMATION MEMORANDUM

I have decided that, for the purpose of making a final determination of the programmatic alternative for the national disposal of obsolete chemical munitions, further consideration of the alternative to transport ton containers of bulk mustard chemical agents by ocean barge from Aberdeen Proving Ground, Maryland, is not reasonable. The reasons for this decision are as follows:

--Given our Congressional mandate to destroy the stockpile by 1994, and the public interest in destroying the already leaking munitions as quickly as possible, the overall programmatic decision, which has already been delayed by a year, should not be further delayed for an indefinite period by necessary studies of a complex transportation scheme coupled with a multiplicity of different environmental areas. The word "areas," as used in this paragraph, refers only to the waterway systems and contiguous land areas which lie within the jurisdiction of the continental United States.

--Rail transportation as an alternative to on-site disposal provides adequate consideration of transportation for the purposes of a national programmatic EIS. Analysis of this method of transportation will be accomplished in sufficient detail to provide reasonable comparison of the alternatives of removal from site versus disposal on site.

--Adequate consideration of the range of possible environmental impacts on the ecology of complex water systems such as the Chesapeake Bay and its tributaries would entail, in all likelihood, lengthy and extensive studies.

--A national programmatic decision does not foreclose subsequent consideration of site-specific alternatives at a later date.

James R. Ambrose
Under Secretary of the Army

cc:
Assistant Secretary of the Army
(Installations and Logistics)
The General Counsel
Chief of Legislative Liaison
Chief of Public Affairs (SAPA-PP)
Deputy Chief of Staff for Operations
and Plans (DAMO-SW)
Commanding General, U. S. Army
Materiel Command (AMCCN)
Chairman, Council on Environmental
Quality

Appendix **S**

OCEAN TRANSPORT OF ABERDEEN PROVING GROUND STOCKPILE: BASIS FOR **ELIMINATING** THIS ALTERNATIVE

DEPARTMENT OF THE ARMY
OFFICE OF THE UNDER [illegible]
WASHINGTON D. C. 20310-0102

October 21, 1987

MEMORANDUM *FOR* PROGRAM MANAGER FOR CHEMICAL DEMILITARIZATION

SUBJECT: Ocean Transport **of** Aberdeen Proving Ground Stockpile-- INFORMATION MEMORANDUM

I have decided that, for the purpose of making **a** final determination of the programmatic alternative *for* the national disposal of obsolete chemical munitions, further consideration of **the** alternative to transport ton containers of bulk mustard chemical agents by ocean barge from Aberdeen Proving Ground, Maryland, is not reasonable. The reasons **for** this decision are **as** follows:

--Given *our* Congressional mandate to destroy the stockpile by 1994, and the public interest in destroying the already leaking munitions **as** quickly as possible, the overall programmatic decision, which has already been delayed by a year, should not be further delayed for an indefinite period **by** necessary studies of a complex transportation scheme coupled with a multiplicity of different environmental areas. The word "areas," **as** used in this **paragraph,** refers only to the waterway systems and contiguous land areas which lie within the jurisdiction **of** the continental United States.

--Rail transportation **as** an alternative to on-site disposal provides **adequate** consideration of transportation for the purposes of **a** national programmatic EXS. Analysis of this method of transportation will **be** accomplished in sufficient detail to provide **reasonable** comparison of the alternatives of removal **from** site versus disposal on site.

--Adequate consideration of the range of possible environmental impacts on the ecology of complex water systems such as the Chesapeake Bay and its tributaries would entail, in all likelihood, lengthy and extensive studies.

--A national programmatic decision does not foreclose subsequent consideration of site-specific alternatives at a later date.

James R. Ambrose
Under Secretary of the Army

cc:
Assistant Secretary of the Army
(Installations and Logistics)
The General Counsel
Chief of Legislative Liaison
Chief of Public Affairs (SAPA-PP)
Deputy Chief of Staff for Operations
and Plans (DAMO-SW)
Commanding General, U. S. Army
Materiel Command (AMCCN)
Chairman, Council on Environmental
Quality