TABLE OF CONTENTS

Section		Page
EXECUTIVE SUMMARY		ES-1
1.0	INTRODUCTION	1-1
1.1	OVERVIEW	1-1
1.2	STUDY AREA BACKGROUND	1-2
1.2.1	RSA FACILITY	1-2
1.2.1.1	General Description	1-2
1.2.1.2	Manufacturing Operations and Waste Generation at RSA	1-3
1.2.2	AREA F (RSA 49)	1-5
1.2.3	AREA G (RSA 48)	1-6
1.2.4	AREA R (RSA 59)	1-6
1.2.5	AREAS S AND T (RSA 55/54)	1-6
1.2.6	SUMMARY OF PREVIOUS INVESTIGATIONS AT RSA	1-7
1.3	GENERAL SAMPLING LOCATIONS AND MEDIA	1-10
1.3.1	AREA F (RSA 49)	1-10
1.3.1.1	Test Pits	1-11
1.3.1.2	Shallow Surface Soil Sampling	1-11
1.3.1.3	Soil Borings	1-12
1.3.1.4	Sediment Sampling	1-13
1.3.1.5	Surface Water Sampling	1-13
1.3.1.6	Groundwater Sampling	1-13
1.3.2	AREA G (RSA 48)	1-15
1.3.2.1	Test Pits	1-15
1.3.2.2	Soil Borings	1-16
1.3.2.3	Sediment Sampling	1-16
1.3.2.4	Surface Water Sampling	1-16
1.3.2.5	Groundwater Sampling	1-17

TABLE OF CONTENTS
(Continued, Page 2 of 10)

Section				Page
		1.3.3	AREA R (RSA 59)	1-18
		1.3.3.1	Test Pits	1-18
		1.3.3.2	Shallow Soil Sampling	1-18
		1.3.3.3	Soil Borings	1-19
		1.3.3.4	Sediment Sampling	1-19
		1.3.3.5	Surface Water Sampling	1-19
		1.3.3.6	Monitor Well Installation and Groundwater Sampling	1-20
		1.3.4	AREAS S AND T (RSA 55/54)	1-20
		1.3.4.1	Test Pits	1-20
		1.3.4.2	Shallow Surface Soil Sampling	1-21
		1.3.4.3	Soil Borings	1-21
		1.3.4.4	Groundwater	1-22
	1.4		REPORT ORGANIZATION	1-25
		1.4.1	DESCRIPTION OF BRA PROCESS	1-25
		1.4.2	CONCEPTUAL SITE MODEL FOR BRA 1	1-26
2.0			HAZARD IDENTIFICATION/IDENTIFICATION OF COPCs	2-1
	2.1		GENERAL DATA COLLECTION CONSIDERATIONS	2-1
	2.2		GENERAL DATA EVALUATION CONSIDERATIONS	2-2
		2.2.1	FLAGGED DATA	2-2
		2.2.2	BLANK CONTAMINATION	2-3
		2.2.3	BACKGROUND LEVELS	2-3
	2.3		STUDY AREA-SPECIFIC RESULTS	2-4
		2.3.1	AREA F (RSA 49)	2-5
		2.3.1.1	Sampling Results	2-5
		2.3.1.2	Nature and Extent of Contamination	2-5

TABLE OF CONTENTS
(Continued, Page 3 of 10)

Section				Page
		2.3.1.3	Soil Concentrations Versus Background Levels	2-7
	2.3.2	AREA G (RSA 48)		2-8
		2.3.2.1	Sampling Results	2-8
		2.3.2.2	Nature and Extent of Contamination	2-8
		2.3.2.3	Soil Concentrations Versus Background Levels	2-10
	2.3.3	AREA R (RSA 59)		2-10
		2.3.3.1	Sampling Results	2-10
		2.3.3.2	Nature and Extent of Contamination	2-11
		2.3.3.3	Soil Concentrations Versus Background Levels	2-12
	2.3.4	AREAS S AND T (RSA 55/54)		2-13
		2.3.4.1	Sampling Results	2-13
		2.3.4.2	Nature and Extent of Contamination	2-13
		2.3.4.3	Soil Concentrations Versus Background Levels	2-15
	2.4	GENERAL DATA UNCERTAINTY		2-16
	2.5	SUMMARY OF COPCs		2-16
3.0	EXPOSURE ASSESSMENT			3-1
	3.1	CHARACTERIZATION OF EXPOSURE SETTING		3-1
	3.1.1	PHYSICAL SETTING		3-1
		3.1.1.1	Climate	3-1
		3.1.1.2	Topography	3-2
		3.1.1.3	Regional Geology	3-2
		3.1.1.4	Surface Hydrology	3-3

TABLE OF CONTENTS
(Continued, Page 4 of 10)

Section			Page
	3.1.1.5	Groundwater Hydrology	3-3
	3.1.1.6	Vegetation and Habitat	3-5
3.1.2	POTENTIALLY EXPOSED HUMAN POPULATIONS		3-7
	3.1.2.1	Demography of Huntsville, Alabama	3-7
	3.1.2.2	Current RSA Land Use	3-8
	3.1.2.3	Potential Alternate Future Uses	3-9
	3.1.2.4	RSA and City of Huntsville Groundwater Use	3-10
	3.1.2.5	RSA Surface Water Use	3-11
	3.1.2.6	City of Madison Groundwater Use	3-12
	3.1.2.7	Subpopulations of Potential Concern	3-12
3.1.3	POTENTIALLY EXPOSED ECOLOGICAL POPULATIONS		3-13
	3.1.3.1	Proximity of Ecological Receptors with Respect to Study Areas	3-13
	3.1.3.2	Aquatic and Terrestrial Habitats	3-13
	3.1.3.3	Aquatic and Terrestrial Communities	3-16
	3.1.3.4	Subpopulations of Potential Concern	3-16
	3.1.3.5	Endangered or Threatened Fauna	3-16
	3.1.3.6	Nonhuman Representative Receptors	3-16
3.2	IDENTIFICATION OF POTENTIAL EXPOSURE PATHWAYS		3-21
3.2.1	SOURCES AND RECEIVING MEDIA		3-21
	3.2.1.1	Area F	3-22
	3.2.1.2	Area G	3-22
	3.2.1.3	Area R	3-23
	3.2.1.4	Areas S and T	3-23

TABLE OF CONTENTS
(Continued, Page 5 of 10)

Section			Page
3.2.2	FATE AND TRANSPORT IN RELEASE MEDIA		3-24
	3.2.2.1	Physical and Chemical Properties	3-25
	3.2.2.2	Environmental Fate Processes	3-27
	3.2.2.3	Contaminant Migration Pathways	3-33
	3.2.2.4	Study-Area-Specific Discussions of Contamination Migration Pathways	3-36
3.2.3	HUMAN EXPOSURE POINTS AND EXPOSURE ROUTES		3-38
	3.2.3.1	Area F	3-39
	3.2.3.2	Area G	3-39
	3.2.3.3	Area R	3-40
	3.2.3.4	Areas S and T	3-40
3.2.4	POTENTIAL HUMAN RECEPTORS AND SCENARIOS		3-40
	3.2.4.1	Current Worker	3-40
	3.2.4.2	Future Worker	3-41
	3.2.4.3	Recreational User	3-41
	3.2.4.4	Future Construction Worker	3-42
	3.2.4.5	Current Resident	3-42
	3.2.4.6	Future Resident	3-43
3.2.5	SUMMARY OF COMPLETED HUMAN EXPOSURE PATHWAYS TO BE QUANTIFIED IN BRA		3-43
	3.2.5.1	Groundwater Ingestion Pathway	3-43
	3.2.5.2	Groundwater Dermal Contact Pathway	3-44
	3.2.5.3	Groundwater Inhalation Pathway	3-44

TABLE OF CONTENTS
(Continued, Page 6 of 10)

Section				Page
		3.2.5.4	Soil Dermal Contact Pathway	3-45
		3.2.5.5	Soil Ingestion Pathway	3-45
		3.2.5.6	Soil Inhalation Pathway	3-45
		3.2.5.7	Sediment/Surface Water Dermal Contact Pathway	3-46
		3.2.5.8	Sediment/Surface Water Ingestion Pathway	3-46
		3.2.5.9	Fish Ingestion Pathway	3-46
	3.2.6		IDENTIFICATION OF POTENTIAL ECOLOGICAL EXPOSURE PATHWAYS	3-46
	3.3		QUANTIFICATION OF EXPOSURE	3-49
	3.3.1		EXPOSURE CONCENTRATIONS	3-49
	3.3.2		CHEMICAL INTAKES FOR HUMAN EXPOSURES	3-51
	3.3.3		ECOLOGICAL RECEPTOR INTAKES	3-52
	3.4		IDENTIFICATION OF UNCERTAINTIES IN EXPOSURE ASSESSMENT	3-55
	3.4.1		HUMAN EXPOSURE ASSESSMENT	3-55
	3.4.2		ECOLOGICAL EXPOSURE ASSESSMENT	3-55
4.0			TOXICITY ASSESSMENT	4-1
	4.1		TOXICITY INFORMATION FOR NONCARCINOGENIC EFFECTS	4-2
	4.2		TOXICITY INFORMATION FOR CARCINOGENIC EFFECTS	4-2
	4.3		CHEMICALS WITH UNAVAILABLE EPA TOXICITY VALUES	4-3
	4.3.1		CARCINOGENIC CHEMICALS WITH NO ESTABLISHED CSF	4-3
	4.3.2		CHEMICALS WITH NO ESTABLISHED RFDs	4-4
	4.4		ECOTOXICITY BENCHMARKS	4-5
	4.5		UNCERTAINTIES RELATED TO TOXICITY INFORMATION	4-6
	4.6		TOXICITY INFORMATION SUMMARY	4-6

TABLE OF CONTENTS
(Continued, Page 7 of 10)

Section		Page
5.0	RISK CHARACTERIZATION	5-1
5.1	METHODS FOR HUMAN RISK CHARACTERIZATION	5-1
5.1.1	CARCINOGENIC RISK	5-1
5.1.2	NONCARCINOGENIC HI	5-3
5.2	STUDY-AREA-SPECIFIC RESULTS OF HUMAN RISK CHARACTERIZATION FOR CURRENT AND FUTURE LAND USE	5-4
5.2.1	AREA F	5-4
5.2.1.1	Current Land Use	5-4
5.2.1.2	Future Land Use	5-5
5.2.2	AREA G	5-5
5.2.2.1	Current Land Use	5-5
5.2.2.2	Future Land Use	5-6
5.2.3	AREA R	5-7
5.2.3.1	Current Land Use	5-7
5.2.3.2	Future Land Use	5-7
5.2.4	AREAS S AND T	5-8
5.2.4.1	Current Land Use	5-8
5.2.4.2	Future Land Use	5-8
5.3	METHODS FOR ECOLOGICAL RISK CHARACTERIZATION	5-9
5.4	STUDY-AREA-SPECIFIC ECOLOGICAL RISK CHARACTERIZATION	5-10
5.4.1	AREA F	5-11
5.4.1.1	Terrestrial	5-11
5.4.1.2	Aquatic	5-11

TABLE OF CONTENTS
(Continued, Page 8 of 10)

Section			Page
	5.4.2	AREA G	5-11
	5.4.2.1	Terrestrial	5-11
	5.4.2.2	Aquatic	5-11
	5.4.3	AREA R	5-11
	5.4.3.1	Terrestrial	5-11
	5.4.3.2	Aquatic	5-12
	5.4.4	AREAS S AND T	5-12
	5.4.4.1	Terrestrial	5-12
	5.4.4.2	Aquatic	5-12
5.5	SUMMARY OF UNCERTAINTIES ASSOCIATED WITH THE BRA		5-12
	5.5.1	UNCERTAINTIES ASSOCIATED WITH HAZARD IDENTIFICATION	5-13
	5.5.1.1	Uncertainties Associated with Data Collection	5-13
	5.5.1.2	Uncertainties Associated with Data Evaluation	5-16
	5.5.2	UNCERTAINTY ASSOCIATED WITH EXPOSURE ASSESSMENT	5-17
	5.5.2.1	Human Exposure Uncertainties	5-17
	5.5.2.2	Ecological Exposure Uncertainties	5-19
	5.5.3	UNCERTAINTY ASSOCIATED WITH TOXICITY ASSESSMENT	5-20
	5.5.3.1	Uncertainty Associated with the Human Toxicity Assessment	5-20
	5.5.3.2	Uncertainty Associated with the Ecological Toxicity Assessment	5-21

TABLE OF CONTENTS
(Continued, Page 9 of 10)

Section		Page
	5.5.4 UNCERTAINTY ASSOCIATED WITH RISK CHARACTERIZATION	5-22
6.0	DEVELOPMENT OF REMEDIAL GOAL OPTIONS	6-1
	6.1 DEVELOPMENT OF RGOs	6-1
	6.1.1 RGOs FOR GROUNDWATER	6-3
	6.1.1.1 Development of RGOs for Individual Groundwater Contaminants Exceeding 1 x 10^{-6} Risk or an HI of 1	6-3
	6.1.2 RGOs BASED ON ECOLOGICAL RISKS	6-5
7.0	SUMMARY AND CONCLUSIONS	7-1
	7.1 OBJECTIVES OF THE BRA	7-1
	7.2 ORGANIZATION OF THE RISK ASSESSMENT REPORT	7-1
	7.2.1 OVERVIEW OF HAZARD IDENTIFICATION	7-2
	7.2.2 OVERVIEW OF THE EXPOSURE ASSESSMENT	7-3
	7.2.3 OVERVIEW OF TOXICITY ASSESSMENT	7-4
	7.2.4 RISK CHARACTERIZATION	7-5
	7.2.4.1 Human Risk Characterization	7-5
	7.2.4.2 Ecological Risk Characterization	7-6
	7.2.5 RGOs	7-7
REFERENCES		

TABLE OF CONTENTS
(Continued, Page 10 of 10)

Section | Page

APPENDICES

APPENDIX A--ANALYTICAL SUMMARY TABLES
AREAS F, G, R, S/T, PHASE I
AND PHASE II
APPENDIX B--EXPOSURE CONCENTRATIONS
APPENDIX C--INTAKE ESTIMATION METHODOLOGY
APPENDIX D--DAILY INTAKES
APPENDIX E--TOXICOLOGICAL PROFILES
APPENDIX F--HUMAN RISKS AND HAZARD INDICES
APPENDIX G--SPECIES LISTS FOR ENDANGERED, THREATENED,
AND SPECIAL CONCERN BIOTA AT REDSTONE
ARSENAL
APPENDIX H--NRMP FLORA AND FAUNA SURVEY

LIST OF TABLES

Table	
1.1-1	Sites to be Evaluated in the Baseline Risk Assessments at RSA
2.2-1	Summary of Lab Qualifiers for Samples Collected at Area F
2.2-2	Summary of Lab Qualifiers for Samples Collected at Area G
2.2-3	Summary of Lab Qualifiers for Samples Collected at Area R
2.2-4	Summary of Lab Qualifiers for Samples Collected at Areas S and T
2.3-1	Comparison of Onsite RFI Phase I Soil Sample Results at Area F to Background Concentrations
2.3-2	Comparison of Onsite RFI Phase I Soil Sample Results at Area G to Background Concentrations
2.3-3	Comparison of Onsite RFI Phase I Soil Sample Results at Area R to Background Concentrations
2.3-4	Comparison of Onsite RFI Phase I Soil Sample Results at Areas S and T to Background Concentrations
2.5-1	Summary of COPCs at Areas F, G, R, and S/T
3.1-1	Average Monthly Precipitation, Temperature, and Windspeed Data for RSA
3.1-2	Water Well and Spring Survey for RSA
3.1-3	Endangered or Threatened Fauna Potentially Found at RSA
3.2-1	PAHs Detected in the Study Areas
3.2-2	VOCs, Phthalate, and Pesticides Detected in the Study Areas
3.2-3	Metals Detected in the Study Areas
3.2-4	Physicochemical Properties of PAHs
3.2-5	Physicochemical Properties of VOCs, SVOCs, and Pesticides

LIST OF TABLES
(Continued, Page 2 of 3)

Table

3.2-6 Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T

3.3-1 Soil Intake Values for Terrestrial Indicator Species

4.0-1 Chronic Dose-Response Toxicity Constants for the COPCs at RSA Sites F, G, R, and S/T

4.0-2 Weight-of-Evidence Categories for Potential Carcinogens

4.4-1 Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T

4.4-2 Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T

5.2-1 Summary of Human Health Risks and HIs for Areas F, G, R, and S/T

5.2-2 Summary of Media and COPCs Exceeding Risks Based on the Human Risk Characterization

5.2-3 Summary of Media and COPCs Exceeding an HQ of 0.1 or HI of 1 Based on the Human Noncarcinogenic Risk Characterization

5.4-1 Chemical Intakes and EQs for the Terrestrial Indicator Species at Areas F, G, R, and S/T

5.4-2 Ecotoxicity Quotients (EQs) for Potential Aquatic Indicator Species

5.4-3 Comparison of COPC Concentrations in Surface Water to Ambient Water Quality Criteria

5.5-1 Comparison of Normal and Lognormal Distribution-Based UCL95s for Groundwater at Area F

5.5-2 Intakes and RMEHIs for the Two Largest Observed Differences in UCL95s for Groundwater at Area F

5.6-1 Uncertainty Associated with the RA Process

LIST OF TABLES
(Continued, Page 3 of 3)

Table

6.1-1 Preliminary RGOs Associated with Future Worker Oral and Inhalation Exposure to Contaminants in Groundwater at RSA Study Areas F, G, R, and S/T

6.1-2 Preliminary RGOs Associated with Future Worker Exposure at RSA Study Areas F, G, R, and S/T Based on Cumulative Risk of 1E-4 or HI of 1

LIST OF FIGURES

Figure	
1.1-1	Location of Redstone Arsenal, Alabama
1.1-2	Location of 16 Study Areas Included in Baseline Risk Assessments at Redstone Arsenal, Alabama
1.2-1	Area F - Closed Disposal Ponds
1.2-2	Area G - Closed Landfill
1.2-3	Area R - Closed Landfill
1.2-4	Area S/T - Closed Disposal Trenches
1.3-1	Area F - Closed Disposal Ponds - Excavation and Soil Sampling Locations
1.3-2	Area F - Closed Disposal Ponds - Groundwater and Surface Water Sampling Locations
1.3-3	Area G - Closed Landfill - Excavation and Soil Sampling Locations
1.3-4	Area G - Closed Landfill - Groundwater and Surface Water Sampling Locations
1.3-5	Area R - Closed Landfill - Excavation and Soil Sampling Locations
1.3-6	Area R - Closed Landfill - Groundwater and Surface Water Sampling Locations
1.3-7	Area S and T - Closed Disposal Trenches - Excavation and Soil Sampling Locations
1.3-8	Area S and T - Closed Disposal Trenches - Groundwater and Surface Water Sampling Locations
1.4-1	Conceptual Site Model for Area F
1.4-2	Conceptual Site Model for Area G
1.4-3	Conceptual Site Model for Area R
1.4-4	Conceptual Site Model for Area S/T
3.1-1	Location of Water Wells, Springs, and Raw Water Sources In and Around Redstone Arsenal

LIST OF ACRONYMS AND ABBREVIATIONS

AAAP	Alabama Army Ammunition Plant
AEHA	U.S. Army Environmental Hygiene Agency
AOC	areas of concern
ARAR	applicable and relevant or appropriate requirement
atm-m^3/mole	atmospheres per cubic meter per mole
AWQC	ambient water quality criteria
B2EHP	bis2-ethylhexyl)phthalate
BCF	bioconcentration factor
BNA	base-neutral and acid extractable
BOD_5	5-day bio-chemical oxygen demand
BRA	baseline risk assessment
BRAC	Base Realignment and Closure
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CFR	Code of Federal Regulations
CFS	cancer slope factors
cm/sec	centimeters per second
CMS	Corrective Measures Study
COC	chemicals of concern
COD	chemical oxygen demand
COPC	chemicals of potential concern
CSF	cancer slope factor
df	degree of freedom
°F	degrees Fahrenheit
f_{oc}	soil organic carbon fraction
EPA	U.S. Environmental Protection Agency
EQ	ecotoxicity quotient
ESE	Environmental Science & Engineering, Inc.
FS	feasibility study
ft/year	feet per year
ft	foot
ft-amsl	feet above mean sea level

ft-bls	feet below land surface
FY	fiscal year
g	grams
g/mole	grams per mole
g/day	grams/day
G&M	Geraghty & Miller, Inc.
GOCO	government-owned/contractor-operated
ha	hectare
HEA	Health and Environmental Assessment
HEAST	Health Effects Assessment Summary Tables
HI	hazard index
H_o	hypothesis
HQ	hazard quotient
ID	identification
IRA	interim remedial action
IRIS	Integrated Risk Information System
Kd	organic partition coefficient
kg	kilogram
kg/day	kilograms per day
km^2	square kilometers
K_{oc}	organic carbon partition coefficient
L/day	liters per day
LOAEL	lowest-observed-adverse-effect-level
MICOM	U.S. Army Missile Command
MCL	maximum contaminant level
MDC	maximum detected concentration
mg/kg	milligrams per kilogram
mg/kg/day	milligrams per kilogram per day
mg/L	milligram per liter
mi^2	square miles
mL/g	milliliter per gram

mmHg	millimeter of mercury
MRL	minimum risk level
MSFC	Marshall Space Flight Center
NASA	National Aeronautics and Space Administration
NCP	National Contingency Plan
nm	nanometers
NOAEL	no-observed-adverse-effect-level
NPL	National Priority List
NRMP	Natural Resources Management Plan
PAH	polycyclic aromatic hydrocarbon
PbB	blood-lead
PELA	P.E. LaMoreaux & Associates, Inc.
PRG	Preliminary Remedial Goal
QA/QC	quality assurance/quality control
QCSR	Quality Control and Summary Report
RAGS	Risk Assessment Guidance for Superfund
R&D	research and development
RCRA	Resource Conservation and Recovery Act
RD	remedial design
RfD	reference dose
RFI	RCRA Facility Investigation
RGO	remedial goal option
RME	reasonable maximum exposure
RSA	Redstone Arsenal
SARA	Superfund Amendments and Reauthorization Act of 1986
SCS	Soil Conservation Service
SDWA	Safe Drinking Water Act
STP 4	Sewage Treatment Plant 4
SVOC	semivolatile organic compound
SWMU	Solid Waste Management Unit

TEF	toxicity equivalency factor
TIC	tentatively identified compound
TVA	Tennessee Valley Authority
UCL	upper confidence limit
UCL95	upper 95th confidence limit of the mean
μg/kg	micrograms per kilogram
μg/L	micrograms per liter
UR	unit risk
USACE	U.S. Army Corps of Engineers
USATHAMA	U.S. Army Toxic and Hazardous Materials Agency
USDA	U.S. Department of Agriculture's
VOC	volatile organic compound
WNWR	Wheeler National Wildlife Refuge
WoE	weight-of-evidence
WTP1	Water Treatment Plant 1
WTP3	Water Treatment Plant 3
WWII	World War II

EXECUTIVE SUMMARY

Environmental Science & Engineering, Inc. (ESE), under contract with the U.S. Army Corps of Engineers (USACE), Huntsville Division, is conducting human and ecological baseline risk assessments (BRAs) to address contamination detected at 10 study areas, comprising 16 study areas, at the Redstone Arsenal (RSA) in Madison County, Alabama. The purpose of the BRAs are to determine the human health and ecological risks associated with the no-action alternative at the 10 areas to determine which areas, media, and contaminants require further evaluation in the feasibility study (FS).

To make the most effective use of the available information and to expedite the remedial response process, the 16 study areas are evaluated in three separate BRAs based on several factors including geographical proximity, similarities in study area contaminants, and similarities in human and/or ecological receptors. This BRA addresses 5 of the 16 study areas including the following:

1.	Area F	RSA 49	Former Arsenic Ponds
2.	Area G	RSA 48	Former Sanitary and Industrial Landfill
3.	Area R	RSA 59	Former Industrial Landfill
4/5.	Areas S and T	RSA 55/54	Former Industrial and Sanitary Landfill

The remaining areas will be addressed in two subsequent BRAs; one to address Unit 1 (RSA 10), Area Q3 (RSA 53), and Area Q4 (RSA 60); and one to address Unit 2 (RSA 12, 13, 14, 131, and 133); Area X1 (RSA 66), and Area Z (RSA 68).

STUDY AREA HISTORY

RSA is comprised of three separate military facilities originally established in 1941. These included the Redstone Ordnance Plant (later RSA), the Huntsville Arsenal, and the Gulf Chemical Warfare Depot. These three facilities worked together to produce conventional and chemical munitions for use during World War II from 1942 to 1945.

Numerous chemical manufacturing plants were operated at the three facilities to produce raw materials for toxic agents as well as to manufacture the agents themselves (Department of the Army, 1977). Immediately after World War II, the Gulf Chemical Depot acted as the center

for receipt and storage of surplus chemical munitions and agents as well as captured German chemical agents. Between 1945 and 1949, all three military units reduced activities to standby status levels. In 1947, the functions of the Gulf Chemical Depot were incorporated into those of the Huntsville Arsenal and subsequently the Huntsville Arsenal was declared surplus. In 1949, the Army designated RSA as caretaker of the Huntsville Arsenal properties. RSA was reactivated in 1949 to perform basic research and development (R&D) in rocketry and guided missile systems. As research activities increased, the need for additional land led the Army to formally incorporate all lands previously used by the Huntsville Arsenal and the Gulf Chemical Depot into the complex that forms the current boundaries of RSA.

REGULATORY HISTORY

RSA has conducted studies of past hazardous waste management practices at its facility in accordance with the requirements of the Resource Conservation and Recovery Act of 1976, with Amendments (RCRA). These studies included a RCRA Facility Investigation (RFI) and Health and Environmental Assessment (HEA) [Geraghty & Miller, Inc. (G&M); 1991, 1992] in which historical and analytical data were collected and evaluated in two phases (Phase I and Phase II), to determine the nature of contamination at the 16 solid waste management units (SWMUs). In addition, a RCRA Corrective Measures Study (CMS) (ESE, 1993) was completed to evaluate corrective measures to be implemented at study areas where contaminants exceeded generic residential health criteria as presented in the RFI HEA (G&M, 1991; 1992).

While the draft CMS report was being completed for an April 1993 submittal to the U.S. Environmental Protection Agency (EPA), RSA was proposed to be listed on the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priority List (NPL). On May 31, 1994, RSA was formally proposed for NPL status in the Federal Register, with actual promulgation to occur on June 30, 1994. As a result of the RSA NPL nomination, EPA Region IV requested that RSA revise both the RCRA HEA (conducted as part of the RFI (G&M, 1991, 1992) and the RCRA CMS (ESE, 1993) to meet the requirements of the equivalent documents under CERCLA and the National Contingency Plan (NCP). At the time of the request, EPA indicated to RSA that the information developed during the RCRA studies was to be used, to the greatest extent practical, in preparation of the equivalent CERCLA documents. EPA, ADEM, and RSA agree that

inorganics (excluding a number of metals because they were representative of background conditions), volatile organics, semivolatile organics, and several pesticides. While EPA risk assessment guidance allows for a reduction in the number of chemicals that must undergo complete evaluation in the risk assessment (i.e., focus the risk assessment on those chemicals that are most toxic), computers enable the risk assessor to completely evaluate all pertinent data. Thus, the only data screening that was performed for this BRA was for inclusion of all contaminants that were thought to be study area-related, and contaminants that were detected at levels significantly above background.

Exposure Assessment

The exposure assessment is the cornerstone of the risk assessment process as this step accomplishes the identification of significant human and ecological exposure pathways and population(s) based on the physical and chemical characteristics of the study area; determines the exposure concentrations to potential receptors; and estimates the magnitude, duration, and frequency of exposure for each receptor (or receptor group). For the five areas of concern, the primary human exposure pathways included in this BRA include the following:

Human Exposure Pathways	Ecological Exposure Pathways
Current Recreational	Aquatic
Current Worker	Terrestrial
Future Worker	

Human exposure addresses potential exposure to surface soil, groundwater, surface water, and sediment. Terrestrial exposure addresses potential exposure to surface soil, while aquatic exposure addresses potential exposure to surface water and sediment.

Groundwater will be evaluated as an aquatic exposure pathway only if habitats (i.e., caves) are discovered near the five areas of concern. To date, no such habitats have been documented.

Toxicity Assessment

The primary purpose of the toxicity assessment is to summarize the toxicological properties of the COPCs and identify concentration levels that are not expected to produce adverse effects. A literature and database search was conducted to obtain the toxicological properties of the COPCs, including pharmacokinetics, metabolism, acute and chronic toxicity, carcinogenic and noncarcinogenic effects on human receptors, wildlife, and aquatic species.

The primary sources of toxicological data were from EPA-verified references. When this information was not identified an appropriate toxicological constant, current literature was reviewed to find appropriate toxicological data, which were used to calculate dose-response values using the methodologies outlined in EPA guidance documents.

Risk Characterization

The study area-specific human carcinogenic and noncarcinogenic risk estimates are determined using the exposure concentrations and factors presented in the exposure assessment along with the dose-response information developed in the toxicity assessment. The potential carcinogenic risks are compared with the EPA target cumulative risk range. Acceptable exposure levels are the contaminant concentration levels that present an excess cancer risk of 10^{-6} (1 in 1,000,000) to 10^{-4} (1 in 10,000) to the exposed population, [NCP, 40 Code of Federal Regulations (CFR) 300, 430:62].

When a cumulative carcinogenic risk (risk associated with exposure to a mixture of chemicals) to an individual receptor under the assumed exposure conditions at a Superfund study area exceeds 10^{-4}, CERCLA generally requires remedial action at the study area (EPA, 1991a). If the cumulative risk is less than 10^{-4}, action generally is not required but may be warranted if a chemical-specific standard that is risk based [e.g., the maximum contaminant level (MCL) or an ambient water quality criterion (AWQC)] is violated. A risk-based remedial decision could be superseded by the presence of noncarcinogenic impact or environmental impact at the study area as indicated by a hazard index greater the 1 for human noncarcinogenic exposures or an exceedance of an ecological quotient (EQ) of 1 for aquatic or terrestrial exposures.

Human Risk Characterization Results

The results of the human BRA indicate that the following scenarios exceed either a cumulative risk of 10^{-4} or an HI of 1:

Area F	Groundwater	Future worker	$> 1 \times 10^{-4}$	Arsenic, carbon tetrachloride, chloroform
			HI > 1	Arsenic, mercury, carbon tetrachloride
Area G	Groundwater	Future worker	HI > 1	Arsenic, barium, chromium, and cadmium

Area R	Groundwater	Future worker	HI > 1	Arsenic, cadmium, chromium,
Areas S and T	Groundwater	Future worker	HI > 1	Benzene, chlorobenzene

In addition, the MCL for lead of 15 μg/L was exceeded for lead at Areas G, R, S, and T.

Ecological Risk Characterization Results

The results of the ecological BRA indicate that none of the exposure scenarios resulted in an EQ greater than 1 or an exceedance of an AWQC.

RGOs

The Superfund Amendments and Reauthorization Act of 1986 (SARA) requires that remedial actions attain a degree of contaminant cleanup that ensures protection of public health and the environment. Thus, the risk characterization results are used to identify whether study area COPCs need to be reduced to acceptable health-based levels. The acceptable health-based levels are referred to as RGOs which are chemical-specific concentration goals for individual chemicals for specific medium and reasonable land use combinations.

Based on the results of the risk characterization, all five areas, Areas F, G, R, and S/T resulted in excess risk or HI based on the future worker exposure scenario to groundwater. Thus, this scenario was the basis for developing RGOs for groundwater at these areas. Ecological risk characterization results indicated that none of the exposure scenarios resulted in EQs of more than 1, or exceedance of AWQCs. Thus, RGOs were developed for groundwater based on the human risk results. In summary, the RGOs are developed for the following chemicals to provide risk managers with the maximum risk-related media level options on which to develop remediation aspects of the FS:

Area	Medium	Organics	Inorganics
F	Groundwater	Carbon tetrachloride Chloroform	Arsenic Mercury

G	Groundwater	Not applicable	Arsenic Barium Chromium Cadmium Lead
R	Groundwater	Not applicable	Arsenic Cadmium Chromium Lead
S and T	Groundwater	Benzene Chlorobenzene	Lead

1.0 INTRODUCTION

1.1 OVERVIEW

The U.S. Army Corps of Engineers (USACE) has contracted with Environmental Science & Engineering, Inc. (ESE) to conduct human and ecological baseline risk assessments (BRAs) to address contamination detected at 16 study areas at the Redstone Arsenal (RSA) in Madison County, Alabama (Figure 1.1-1). The BRA is performed by ESE under contract USACE, Delivery Order 0010, Contract Number: RSA-DACA-87-92-D-0018. The purpose of a BRA is to determine if remedial action is required at the study areas by assessing the potential health risks to humans and ecological receptors that may be posed by these areas.

1.1.1 RSA has conducted studies of past hazardous waste management practices at its facility in accordance with the requirements of the Resource Conservation and Recovery Act of 1976, with Amendments (RCRA). These studies included a RCRA Facility Investigation (RFI) and Health and Environmental Assessment (HEA) [Geraghty & Miller, Inc. (G&M); 1991a, 1992a] in which historical and analytical data were collected and evaluated in two phases (Phase I and Phase II) to determine the nature of contamination at the 16 SWMUs. In addition, a Draft RCRA Corrective Measures Study (CMS) (ESE, 1993) was completed to evaluate corrective measures to be implemented at study areas where contaminants exceeded generic residential health criteria as presented in the RFI HEA (G&M, 1991a; 1992a). These studies were conducted on the 16 SWMUs listed in Table 1.1-1 and shown on Figure 1.1-2.

1.1.2 While the draft CMS report was being completed for an April 1993 submittal to the U.S. Environmental Protection Agency (EPA), RSA was proposed to be listed on the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) National Priority List (NPL). On May 31, 1994, RSA was formally proposed for NPL status in the Federal Register, with promulgation expected to occur on June 30, 1994. As a result of the RSA NPL nomination, EPA Region IV requested that RSA revise both the RCRA HEA (conducted as part of the RFI (G&M, 1991a, 1992a) and the RCRA CMS (ESE, 1993) to meet the requirements for their equivalent documents under CERCLA and the National Contingency Plan (NCP). At the time of the request, EPA indicated to RSA that the information developed during the RCRA studies was to be used, to the greatest extent practical, in the preparation of the equivalent CERCLA documents. To achieve the EPA

request, RSA has used substantial portions of the RFI report, indicating any additional investigations to be performed during remedial design to ensure validity of determined risks, appropriateness of remedial designs, and conformance to regulatory requirements.

1.1.3 To make the most effective use of the available information, and to expedite the remedial response process, the 16 study areas (formerly referred to as SWMUs under RCRA) are evaluated in three separate BRAs based on several factors including geographical proximity (see Table 1.1-1 and Figure 1.1-2), similarities in study area contaminants, and similarities in human and/or ecological receptors. This BRA addresses the following 5 of the 16 study areas:

Area F	RSA 49	Former Arsenic Ponds
Area G	RSA 48	Former Sanitary and Industrial Landfill
Area R	RSA 59	Former Industrial Landfill
Areas S and T	RSA 55/54	Former Industrial and Sanitary Landfills

1.1.4 The purpose of this BRA is to summarize and interpret the RFI results (G&M, 1991a and 1992) to determine if remedial action is required at these areas based on an evaluation of the potential risks posed to human and ecological receptors, in addition, the BRA will determine remedial design data needs to reduce uncertainty in risk characterizations. The remaining six areas will be addressed in two separate BRAs.

1.2 STUDY AREA BACKGROUND

To provide a general understanding of the study area history and conditions, summaries of the area-wide operations and history of RSA are presented. These are followed by more detailed summaries of the history and operations conducted at the five study areas under consideration in this BRA. This information was obtained from the G&M Phase I and Phase II RFI reports dated October 1991 and October 1992, respectively.

1.2.1 RSA FACILITY

1.2.1.1 General Description

The land area of the current RSA included three separate military facilities originally established in 1941; (1) the Redstone Ordnance Plant (later Redstone Arsenal), (2) the Huntsville Arsenal, and (3) the Gulf Chemical Warfare Depot. These three facilities worked together to produce conventional and chemical munitions for use during World War II

(WWII) from 1942 to 1945. The responsibilities for weapon production were separated as follows:

1. Huntsville Arsenal produced toxic agents and incendiary materials;
2. Redstone Ordnance Plant assembled the munitions; and
3. Gulf Chemical Warfare Depot stored and shipped the munitions as well as bulk chemicals and equipment associated with decontamination.

1.2.1.1.1 Numerous chemical manufacturing plants were operated at the three facilities to produce raw materials for toxic agents as well as to manufacture the agents themselves (Department of the Army, 1977).

1.2.1.1.2 Immediately after WWII, the Gulf Chemical Depot acted as the center for receipt and storage of surplus chemical munitions and agents as well as captured German chemical agents. Between 1945 and 1949, all three military units reduced activities to standby status levels. In 1947, the functions of the Gulf Chemical Depot were incorporated into those of the Huntsville Arsenal and subsequently the Huntsville Arsenal was declared surplus. In 1949, the Army designated Redstone Ordnance Plant as caretaker of the Huntsville Arsenal properties. RSA was reactivated in 1949 to perform basic research and development (R&D) in rocketry and guided missile systems. As research activities increased, the need for additional land led the Army to formally incorporate all lands previously used by the Huntsville Arsenal and the Gulf Chemical Depot into a complex that forms the current boundaries of RSA.

1.2.1.1.3 The U.S. Army Ballistic Missile Agency was established in 1956. In 1960, the civilian rocketry and missile activities conducted by the Army were transferred to the National Aeronautics and Space Administration (NASA). Marshall Space Flight Center (MSFC) of NASA is a tenant activity at RSA that currently develops, tests, and manufactures space vehicles and components.

1.2.1.2 <u>Manufacturing Operations and Waste Generation at RSA</u>

In 1942, six mustard gas manufacturing plants were constructed at the facility and were in operation until 1943. Materials that were used or produced in substantial quantities as part of the mustard gas operations included sulfur monochloride, ethylene, brine, caustic soda, liquid

caustic, chlorine, and thionyl chloride. The two chlorine plants that were constructed to support the mustard gas generation activities were in operation until 1945. These plants were located in the central part of RSA north of Huntsville Spring Branch (Figure 1.1-2). Mustard gas products and wastes were stored and disposed of onsite at various locations throughout the arsenal.

1.2.1.2.1 Lewisite, a chemical warfare agent containing arsenic, was manufactured in five plants located in the central part of RSA in 1942 and 1943. Arsenic-containing wastes, generated from the Lewisite manufacturing process, were disposed of in shallow ponds (including Area F) at RSA.

1.2.1.2.2 Subsequent to World War II, the chemical manufacturing facilities used to produce bulk chemicals were leased by the Army to numerous private firms for production of commercial chemicals and pesticides. The manufacturing of pesticides, including DDT, was begun in 1947 and terminated in 1971. The manufacturing of DDT and other pesticides resulted in the production of significant amounts of DDT-contaminated wastes. Thousands of pounds of these wastes were buried at landfills throughout RSA, including those at Area Q3, Area Q4, and Areas S and T. In addition, large quantities of wastewater containing DDT were discharged to Huntsville Spring Branch. In July of 1979, the U.S. Army initiated an extensive DDT Migration Abatement Program. From July 1979 to August 1982, DDT wastes, including highly contaminated soils and sediments, were excavated from the DDT manufacturing area, the DDT drainage ditch, and former DDT disposal sites throughout RSA, and disposed of in a specified DDT Waste Soils Landfill located in Unit 1. In 1983, Olin Corporation (principal DDT manufacturer) began DDT cleanup procedures under a U.S. Justice Department Consent Decree. Their activities included cleanup of DDT contamination in Huntsville Spring Branch, erosion control and earth work at the site of the former DDT facility, diversion and reconstruction of Huntsville Spring Branch, covering of the contaminated creek and adjacent areas, capping and revegetation of the remediated areas, and initiation of a groundwater and surface water monitoring program to be continued until the year 2007.

1.2.1.2.3 The military R&D efforts at RSA have been continually supported since 1949 by civilian contractors. The first government contracts were issued in 1949 to Rohm and Haas

Company and the Thiokol Corporation to develop various types of rocket propellants. Chemical manufacturing facilities were constructed for the government contractors to provide materials for these R&D activities.

1.2.1.2.4 Thiokol currently operates a rocket motor manufacturing plant that utilizes organic nitrogen propellants based on RDX/HMX compounds, classified as 1.1 propellants, and ammonium perchlorate-based mixtures that are classified as 1.3 propellants. According to facility representatives, the Thiokol missile production facility is currently responsible for the generation of approximately 75 percent of the wastes generated at RSA. These wastes include propellants, propellant-contaminated shipping materials, propellant-contaminated solvents (principally methylene chloride), as well as small amounts of Freon 113, solvents, and waste oils. The propellants, explosives, propellant-contaminated wastes, and propellant-contaminated solvents are thermally treated at Unit 2, the open burn/open detonation area.

1.2.1.2.5 Ordnance, including shells, grenades, and smoke pots have also been manufactured and demilitarized at RSA. Large areas on the facility continue to be used for testing propellants, explosives, and ordnance, as well as for troop training.

1.2.1.2.6 RSA has a Part B permit for the Hazardous Waste Storage Unit, which includes 13 storage igloos set aside for hazardous waste storage. RSA has a Part A permit for Unit 2, the open burn/open detonation area, which is classified as a hazardous waste thermal treatment unit.

1.2.2 AREA F (RSA 49)

Area F (RSA-49) occupies approximately 5 acres of land and consists of three closed ponds used in the 1940s for the disposal of arsenic-contaminated waste from chemical manufacturing facilities (Figure 1.2-1). Area F is located in the north-central part of RSA and is bounded by a small drainage ditch on the east, open pasture to the north, and an area of pine trees on the west. The drainage area on the east is planned for rerouting as part of an interim remedial action (IRA). The IRA is expected to be completed by the beginning of the third quarter of fiscal year (FY) 1995 and will entail removing existing vegetation at the study area, constructing a clay cap, and installing a new chain link fence. The former Lewisite

manufacturing facility, where the arsenic waste was generated, was located less than 1,000 feet (ft) south of the arsenic ponds. Waste ash, rubble, and industrial waste were disposed of in the ponds subsequent to the disposal of arsenic waste. The source of the rubble and industrial waste was the demilitarization of sumps and buildings from the former Lewisite manufacturing facility and other onsite facilities. The ponds were closed, covered, and capped in 1977 and planted with grass and pine trees.

1.2.3 AREA G (RSA 48)

Area G (RSA 48) is located in the east-central portion of RSA and occupies approximately 5.5 acres (Figure 1.2-2). Area G is bounded by McDonald Creek on the east, a drainage ditch and RSA Sewage Treatment Plant 4 (STP 4) on the north, pasture lands on the west, and railroad tracks on the south. Area G is a former sanitary and industrial landfill. The unlined, uncapped earthen landfill was active from 1947 to 1950 and received rubble from construction materials and sanitary waste, but no hazardous materials. Currently, refuse is exposed on the north side of the landfill, along the south bank of the north drainage creek. A thin layer of soil, grasses, small trees, and brush cover the area.

1.2.4 AREA R (RSA 59)

Area R (RSA 59), a former industrial landfill located in the central part of RSA occupies approximately 8 acres and is bounded on the north, south, and east by woods and wetlands (Figure 1.2-3). Area R was active from 1972 to 1976 and received rubble from construction activities. Rubble, metal debris, railroad ties, and concrete slabs were observed around the landfill boundaries, particularly along the south and east boundaries. Area R may have been a borrow area before being used as a rubble fill. There is a wetland on the western edge of the rubble fill. The shallow subsurface consists essentially of concrete rubble. Examination of 1950s aerial photographs indicates that the area may have been a discharge basin or drainage for the adjacent liquid caustic plant.

1.2.5 AREAS S AND T (RSA 55/54)

Area S (RSA 55) comprises a single landfill which occupies approximately 5 acres and is located in the central portion of RSA (Figure 1.2-4). Area T is to the north and occupies approximately 13 acres. Both areas were used during the 1960s for disposal of household, administrative, and industrial waste [P.E. LaMoreaux & Associates, Inc. (PELA), 1988a,

1989]. Trenches were used to dispose of waste and then covered with a thin layer of soil. DDT waste was buried at various locations at Areas S and T between 1968 and 1973.

1.2.5.1 Areas S and T are bounded on the west by Mills Road, on the north by Martin Road, and on the east by McMorrow Labs (Building 5400). The landfill is intersected by Fowler Road which was constructed after the landfill was operable. Until recently, Area S, on the south side of Fowler Road, was covered with thick vegetation including tall pine trees and associated undergrowth. Between the performance of the Phase I RFI and Phase II RFI, a large part of Area S was clearcut. Area T, on the north side of Fowler Road, is covered by woods, briars, and grasses. The east central portion of Area T is maintained as an open area and was at one time used as a construction staging and storage site for Harbert Construction Company. Although the landfill areas have been revegetated, remnants of disposal trenches are still visible.

1.2.6 SUMMARY OF PREVIOUS INVESTIGATIONS AT RSA

In 1977, a comprehensive environmental review of RSA operations was conducted by the Office of the Project Manager for Chemical Demilitarization and Installation Restoration at the request of the U.S. Army Missile Materiel Readiness Command. The final report, entitled "Installation Assessment of Redstone Arsenal, Alabama" (Department of the Army, 1977), included a review of current environmental resources, current and historical waste disposal practices, potential for contaminant migration, and conclusions/recommendations to improve environmental quality.

1.2.6.1 Several reports document the DDT cleanup activities. During the late 1970s, a study was conducted by the U.S. Army Environmental Hygiene Agency (AEHA) to investigate impacts of DDT disposal practices to the local population and environment. This report is entitled "Special Study No. 24-0044-77, Evaluation of Environmental Degradation from Prior DDT Waste Disposal at Redstone Arsenal, Alabama," (AEHA, 1977). In 1983, a summary report on an environmental survey and the DDT abatement programs conducted by the U.S. Army Toxic and Hazardous Materials Agency was prepared for Redstone Arsenal (Water and Air Research, Inc., 1983). The Environmental Affairs Department of the Olin Chemicals Group has prepared several semi-annual and annual reports documenting their DDT investigations and long-term monitoring program (Olin Chemicals Group, 1983, 1987, 1989).

In 1986, the final environmental impact statement for the Olin remedial action project was issued by the USACE, Nashville District (USACE, 1986). In 1979, the U.S. Army Missile Command of Redstone Arsenal prepared a "Summary of DDT Contamination at Redstone Arsenal, Alabama" to document the DDT disposal activities and mitigation program. This report was updated in 1981 and again in 1989 (U.S. Army Missile Command, 1989).

1.2.6.2 General environmental reports concerning RSA include "Installation Environmental Assessment" (Department of the Army, 1977) and "Analytical/Environmental Assessment Report, Redstone Arsenal, Alabama" (Harland Bartholomew & Associates, Inc., 1986). These environmental assessments outline the cultural, ecological, socio-economic, and utility features of RSA.

1.2.6.3 In September 1989, G&M was contracted by USACE to perform an RFI at RSA. The purpose of the RFI was as follows:

1. Confirm the presence or absence of contamination at each of 10 study areas;
2. Determine the extent and degree of contamination;
3. Identify and characterize the sources of contamination;
4. Assess the potential for migration, and identify exposure pathways and receptor populations; and
5. Identify health and environmental risks.

1.2.6.4 Previous investigations at Unit 1, Unit 2, and the Unit 3 have generated the following reports:

Phase I Report RCRA Facility Investigations at Unit 1, Unit 2, and Selected Unit 3 Areas. Redstone Arsenal, Alabama. Prepared for the U.S. Army Corps of Engineers, Huntsville Division. Contract No. DACA87-89-C-0075. Prepared by Geraghty & Miller, Inc.

Phase II Addendum RCRA Facility Investigations at Unit 1, Unit 2, and Selected Unit 3 Areas. Redstone Arsenal, Alabama. Prepared for the U.S. Army Corps of Engineers, Huntsville Division. Contract No. DACA87-89-C-0075. Prepared by Geraghty & Miller, Inc.

Natural Resource Management Plan for Redstone Arsenal. RSA. June 1992.

Identification and Evaluation of Potential Solid Waste Management Units (SWMUs) and Areas of Concern (AOCs) at Redstone Arsenal, Alabama. Geraghty and Miller,

Inc. (G&M). 1991. Tampa, FL. February, 1991. Contract No. DACA87-89-C-0075. Prepared for the U.S. Army Corps of Engineers, Huntsville, Alabama.

Preliminary Site Inspection Report for Redstone Arsenal. Site Inspection Report No. 91031. Prepared for the U.S. Army Corps of Engineers, Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD. Prepared by: Advanced Sciences, Inc. Oak Ridge, TN. 1992.

Final Environmental Impact Statement for Regulatory Actions Associated with the Olin Corporation, Remedial Action Plan to Isolate DDT from the People and the Environment in the Huntsville Spring Branch-Indian Creek System, Wheeler Reservoir, Alabama. U.S. Army Corps of Engineers (USACE). 1986. Nashville District. Cooperating Agencies are the U.S. Environmental Protection Agency, the U.S. Fish and Wildlife Service, and the Tennessee Valley Authority.

Installation Assessment of Redstone Arsenal, Alabama, Record Evaluation Report No. 118, Department of the Army, December, 1977.

Installation Environmental Assessment. U.S. Army Missile Command. September 1979.

Interim RCRA Facility Assessment Report of the Redstone Arsenal Huntsville, Alabama, EPA ID No. AL 721 002 0742, A.T. Kearney, Inc., September, 1989.

Interim RCRA Facility Assessment Report of the Marshall Space Flight Center, Huntsville, Alabama, EPA ID No. AL1 800 013 863, A.T. Kearney, Inc. 1989.

Master Plan Narrative for Redstone Arsenal, Alabama, USACOE, Mobile District, Kimley-Horn and Associates, Inc., December, 1989.

Huntsville Spring Branch-Indian Creek, Long-Term Monitoring Program, Annual Report No. 4, Olin Corp. TN. May 1992.

Environmental Effects of Army Actions, Army Regulations, Environmental Quality, December 1988, HQ Dept. of the Army, Washington D.C.

Final Remedial Action Plans for Redstone Arsenal, Alabama, Unit 1 DDT and Sanitary Landfill and Unit 2 Open Burn/Open Demolition Area, September 1988, P.E. LaMoreaux & Associates, Inc. (PELA) Tuscaloosa, Alabama, for U.S. Army Engineering Division, Huntsville AL.

Installation Restoration Program Cost Estimate, September 1991, DOD, Office of the Deputy Assistant Secretary of Defense (Environmental).

Final Remedial Investigation Engineering Report Redstone Arsenal, Alabama, Unit 1 DDT and Sanitary Landfills and Unit 2 Open Burn/Open Demolition Area, Volume I Text, September 1988, P.E. LaMoreaux & Associates, Inc. (PELA) Tuscaloosa, Alabama, for U.S. Army Engineering Division, Huntsville AL.

Final Confirmation Report Unit 3 Investigations Redstone Arsenal, Alabama, Volume I Text, July 1988, P.E. LaMoreaux & Associates, Inc. (PELA) Tuscaloosa, Alabama, for U.S. Army Engineering Division, Huntsville AL.

Final Upgrade Confirmation Report and Assessment of Remedial Alternatives for Selected Unit 3 Sites Redstone Arsenal, Alabama, April 1989, P.E. LaMoreaux & Associates, Inc. (PELA) Tuscaloosa, Alabama, for U.S. Army Engineering Division, Huntsville AL.

Interim RCRA Facility Assessment Report of the Redstone Arsenal, Huntsville, Alabama, September 1989, A.T. Kearney, Inc. Alexandria VA for U.S. Environmental Protection Agency, Atlanta, GA.

Cultural Resource Management Plan for RSA. Prepared by Mobile District USACE, January 1992.

Management of Cultural Resources at RSA, Alabama. Prepared for: U.S. Army Missile Command. Prepared by: Charles M. Hubbert., University of Alabama. March 1989.

Remedial Action Decision Document for the DDT Contaminated Areas. Redstone Arsenal.. January 1988.

Hazardous Waste Study No. 37-26-05450-87 Investigation of Soil Contamination at the Contaminated Waste Burning Pits and the Open Burning Area, U.S. Army Environmental Hygiene Agency 1986, Redstone Arsenal, Alabama, 7-14 January 1986. (AEHA, 1986)

1.3 GENERAL SAMPLING LOCATIONS AND MEDIA

The sample identifications (IDs), collection dates, and other relevant information used for the BRA were obtained directly from the G&M Phase I and II reports and the CMS report. Level III data were used. The sample locations for each area are discussed separately and two maps (soil and groundwater sampling locations) are provided for each area. The exposure concentrations were obtained from the RFI and were based on the intervals of the samples collected at that time (G&M, 1990 and 1991), which include surface depths designated as 0-2 ft. The exposure concentrations for soil in this document are assumed to be at the surface (the first 12 inches) within the sample interval. The ground cover present at all study areas of concern is not conducive to suspended airborne dust as it is vegetated and the contaminant sources are subsurface sources such as landfills.

1.3.1 AREA F (RSA 49)

As part of the Phase I and Phase II RFIs, test pits, soils, groundwater, surface water, and sediment were sampled at this area. Figure 1.3-1 shows the location of the test pits, sediment, and soil samples, Figure 1.3-2 shows the monitor well and surface water sampling locations at Area F.

1.3.1.1 Test Pits

To better define the waste characteristics of the ponds, three test pits (FTP-1, FTP-2, and FTP-3) were excavated during Phase I in November 1990. The test pits were located inside the former pond areas. The test pits were sited within the former impoundments based on the results of geophysical investigations, study area observations (stressed vegetation), and aerial photos from which former pond boundaries can be identified. Six soil and waste samples were collected and analyzed for volatile organic compounds (VOCs), base-neutral and acid extractables (BNAs), and total metals (Appendix A). No test pits were excavated during the Phase II RFI.

1.3.1.1.1 As stated in the Phase II RFI report (G&M, 1991a; 1992a), in addition to test pit samples, several shallow soil samples taken from the closed arsenic lagoons during the second phase were also categorized as waste samples. These shallow soil samples (FSB-1S, FSB-7A, FSB-8A, FSB-8C, and FSB-9C) were collected from soil borings and analyzed for contamination.

1.3.1.1.2 Air monitoring was conducted in association with the test pit excavation. During the excavation of all of the test pits, air samples were collected and analyzed for VOCs in the field using portable gas chromatograph equipment. Field logs for the air monitoring conducted at Area F are presented in the Phase I RFI.

1.3.1.2 Shallow Surface Soil Sampling

One shallow soil sample (FS-7) was collected south of Area F during Phase I to investigate the potential for additional contamination sources near the former chemical manufacturing plant located southwest of the closed arsenic ponds. The soil sample was analyzed for VOCs, BNAs, and total metals (Appendix A, Table F-2). The soil sampling log is included in the Phase I RFI.

1.3.1.2.1 During the Phase II RFI in April 1992, seven shallow soil samples (FS-14, FS-16, FS-17, FS-18, FS-19, FS-20, and FS-21) were collected around the perimeter of Area F to determine the areal extent of impacted surface soils associated with Area F. Two additional shallow soil samples (FS-22 and FS-23) were collected on the west side of Area F to help determine the concentration of selected constituents in the shallow soils within Area F but

outside the closed disposal ponds. All of the shallow soil samples were analyzed for polycyclic aromatic hydrocarbons (PAHs), arsenic, mercury, and cadmium (Appendix A) Field descriptions of the soils are included in Attachment III of the Phase II Quality Control and Summary Report (QCSR) (G&M, 1992b).

1.3.1.3 Soil Borings

Soil boring FSB-1 was drilled in the west central portion of Area F during Phase I. The purpose of the boring was to evaluate the subsurface geology and potential soil contamination within the closed arsenic ponds of Area F. Another soil boring, FSB-2, was located to the northwest, outside and upgradient of Area F. This boring was used to collect background soil samples in an area presumed not to have been impacted by study area activities. Field geologic logs for soil borings drilled in Area F are included in the RFI. Two soil samples were collected from each of the borings and analyzed for VOCs, BNAs, and total metals (Appendix A).

1.3.1.3.1 Seven soil borings were drilled and sampled during the Phase II RFI at Area F. Two of the soil borings (FSB-3 and FSB-4) were drilled on the west and north sides of Area F, respectively. Both were drilled to determine the surface and subsurface extent of impacted soils within Area F but outside of the closed disposal ponds. The remaining five soil borings (FSB-5, FSB-6, FSB-7, FSB-8, and FSB-9) were drilled within the suspected boundaries of the closed disposal ponds to help determine the surface and subsurface extent of constituents associated with the disposal of wastes to the ponds in Area F. Field geologic logs for soil borings in Area F are included in the Phase II RFI.

1.3.1.3.2 An average of two soil samples were collected from each of the borings. One 2-ft composite shallow subsurface soil sample from intervals between 0 and 8 feet-below land surface (ft-bls) and one 2-ft deep subsurface soil sample from an interval between 10 and 16 ft-bls was collected from each soil boring (Appendix A). The soil boring samples were analyzed for PAHs, arsenic, mercury, and cadmium. Samples from five sampling intervals (1SB-7A, 1SB-8A, 1SB-8C, and 1SB-9C) were categorized as waste samples based on visual assessment of the soils. The waste samples were analyzed for the constituents listed previously with the exception of 1SB-8C and 1SB-9C which were not analyzed for PAHs. Soil sampling logs are included in Attachment III of the Phase II QCSR (G&M, 1992b). In

addition, geotechnical tests, including grain size distribution, Atterberg limits, and moisture content were obtained from three of the soil borings (FSB-4, FSB-7, and FSB-9).

1.3.1.4 Sediment Sampling

Four sediment samples were collected at and near Area F during Phase I. One sediment sample (FS-6) was collected south of Area F to investigate the potential for additional contamination sources near the former chemical manufacturing plant located southwest of closed arsenic ponds. Sediment samples (FS-5, FS-8, and FS-9) were collected in the surface drainage to the east of Area F to evaluate the potential for contaminant migration due to erosion and seepage from the closed arsenic ponds. The sediment samples collected were analyzed for VOCs, BNAs, and total metals (Appendix A, Table F-5). Sediment sampling logs are included in the RFI.

1.3.1.4.1 Phase II of the RFI included collection of five sediment samples (FS-10, FS-11, FS-12, FS-13, and FS-15) from natural drainage ditches east and south of Area F. These samples were collected to evaluate the extent of impacted sediment due to erosion of wastes and soils from Area F. The sediment samples collected were analyzed for PAHs, arsenic, mercury, and cadmium (Appendix A). Sediment sampling logs are included in the Phase II RFI (G&M, 1992a).

1.3.1.5 Surface Water Sampling

One surface-water sample (SW-F-1) was collected in conjunction with the sediment sample (FS-9) during Phase I sampling. The surface-water sample was analyzed for VOCs and total metals (Appendix A). The water sampling log is included in the Phase I RFI. No surface water samples were collected during the Phase II sampling effort.

1.3.1.6 Groundwater Sampling

Phase I

Seven new monitor wells were installed in November and December 1990 to supplement the four existing wells at Area F. The purpose of the monitor wells was to assist in characterizing the geology, defining the direction of groundwater flow, and mapping the extent of groundwater contamination.

1.3.1.6.1 Four overburden wells screened in the water-bearing zone at the base of the overburden were installed. Three bedrock wells were also installed, including one in the upper bedrock and two in the deeper bedrock. Monitor well construction details for the seven newly installed monitor wells and the four previously installed monitor wells are summarized in the RFI, as are the field geologic logs and well construction details. The seven new monitor wells were installed, developed, and tested in accordance with the RFI Work Plan and as described in the RFI. Two proposed overburden monitor wells were not installed as the overburden at the proposed locations was dry at the time of drilling.

1.3.1.6.2 The four overburden wells (RS257, RS260, RS262, and RS263) were located around the study area perimeter to supplement existing wells. These wells were used to assist in determining the direction of groundwater flow in the overburden in addition to evaluating the areal extent of contamination at Area F. The overburden well (RS263) located south of Area F, was installed to investigate the potential for contamination originating near the former chemical manufacturing plant. The three bedrock wells (RS256, RS259, and RS261) were installed to assist in determining the vertical extent of groundwater contamination and in mapping groundwater flow direction in the bedrock.

1.3.1.6.3 Samples from the seven new monitor wells and from the four existing overburden wells were sampled and analyzed for VOCs and total and dissolved metals (Appendix A). All wells were sampled between January and February 1991. Water sampling logs are included in the RFI report.

1.3.1.6.4 Water levels were measured in all of the new and existing wells to assist in mapping the groundwater flow directions and gradients in the overburden and bedrock. In situ permeability tests were conducted in each of the seven new wells in accordance with the methods described in the RFI.

Phase II

1.3.1.6.5 One new deep overburden monitor well (RS258) was installed in April 1992 to supplement the eleven existing wells at Area F. The purpose of the monitor well was to assist in characterizing the geology, defining the direction of groundwater flow, and evaluating the concentration of constituents of concern in the deep overburden within Area F.

1.3.1.6.6 The overburden well was screened in the water-bearing zone at the base of the overburden. Monitor well construction details for RS258, the eleven previously installed monitor wells, the field geologic logs, and well construction details for RS258 are summarized in the RFI. Monitor well RS258 was installed, developed, and tested in accordance with the RFI Work Plan and as described in the Phase II RFI. Water levels were measured in all of the new and existing wells to assist in mapping the groundwater flow directions and gradients in the overburden and bedrock. In situ permeability tests were conducted in the new monitor well in accordance with the methods described in the Phase II RFI.

1.3.1.6.7 Groundwater samples from the new monitor well were collected and analyzed for VOCs, PAHs, and total and dissolved metals (Appendix A). In addition, seven existing bedrock and overburden wells were sampled and analyzed for PAHs to help determine whether or not the PAHs previously detected in the soils have migrated into the groundwater. All the monitor wells were sampled in June and July 1992. Water sampling logs are included in Phase II RFI.

1.3.2 AREA G (RSA 48)

As part of the Phase I and Phase II RFIs, test pits, soils, groundwater, surface water, and sediment were sampled at this area. Figure 1.3-3 shows the location of the test pits, sediment, and soil samples and Figure 1.3-4 shows the monitor well and surface water locations at Area G.

1.3.2.1 Test Pits

During Phase I, three test pits GTP-1, GTP-2, and GTP-3 were excavated (Figure 1.3-3). The test pits were sited in the former disposal areas based on the geophysical investigations, aerial photographs, and study area observations. Test pits were excavated to assist in characterizing the wastes disposed of in the landfill. Descriptions of the waste and stratigraphy encountered in each test pit in Area G, as well as photographs of test pits, wastes and soils samples, are included in Phase I RFI. One to three samples were collected from each test pit for chemical analyses of VOCs, BNAs, and total metals (Appendix A). Soil sampling logs are included in the RFI.

1.3.2.1.1 Air monitoring was conducted in association with the test pit excavation. During excavation of the three test pits, air samples were collected and analyzed in the field for volatiles using portable gas chromatograph equipment. Field logs for the air monitoring conducted at Area G are presented in the RFI. No samples of the test pits were taken in Area G during Phase II sampling efforts.

1.3.2.2 Soil Borings

Based on the results of the geophysical investigations, previous sampling, and study area observations, three soil borings (GSB-1, GSB-2, and GSB-3) were drilled to 15 ft-bls and sampled in September 1990 (Phase I). The soil borings were located around the perimeter of the disposal site of Area G to assist in evaluating the extent of soil contamination associated with study area activities at Area G. One soil boring, GSB-2, was located on a presumably unimpacted area upgradient of the disposal trenches of Area G. Field geologic logs for soil borings drilled in Area G are included in the RFI. Two samples were collected from each soil boring for analyses of VOCs, BNAs, and total metals (Appendix A). Soil sampling logs are included in the RFI. No soil samples were taken during Phase II sampling efforts.

1.3.2.3 Sediment Sampling

Five sediment samples were collected at the study area during Phase I. Two sediment samples (GSS-4 and GSS-5) were collected from seeps and the drainage ditch located north of Area G, and three samples (GSS-6, GSS-7, and GSS-8) were collected from McDonald Creek east of Area G. The samples were collected to help evaluate potential contaminant migration by erosion of wastes and contaminated soils as well as by leachate directly discharging into the creeks. The samples were analyzed for VOCs and total metals (Appendix A). Sediment sampling logs are included in the Phase I RFI. No sediment samples were taken during Phase II.

1.3.2.4 Surface Water Sampling

During Phase I, three surface-water samples (SW-G-3, SW-G-4, and SW-G-5) were collected for the analysis of VOCs and total metals as shown in Appendix A). The water sampling logs are included in the Phase I RFI. The samples were used in conjunction with the sediment samples to help determine water quality of McDonald Creek and the contamination impacts of erosion and leachate discharge to the surface waters by the landfill.

1.3.2.4.1 Seven surface-water samples (SW-G-4II, SW-G-5II, SW-G-6, SW-G-7, SW-G-8, SW-G-9, and SW-G-10) were collected in April 1992 (Phase II) for the analysis of VOCs as shown in Appendix A. The water sampling logs for each surface water sample are included in the Phase II RFI. These samples were used to evaluate the water quality of McDonald Creek with respect to VOCs and establish whether or not Area G was the source of the VOCs detected in the creek during the Phase I RFI. To meet this objective, two of the surface-water samples (SW-G-4II and SW-G-5II) were collected at two of the Phase I sampling sites (SW-G-4 and SW-G-5) to confirm the presence of trichloroethene as indicated by the Phase I RFI results. The five remaining samples were located along McDonald Creek several hundred yards upstream and downstream of the study area, and in the drainage ditch north of the study area in an effort to establish background conditions around the study area.

1.3.2.5 <u>Groundwater Sampling</u>

Five new monitor wells were installed in December 1990 (Phase I) to supplement existing wells at Area G. The purpose of the monitor wells was to assist in characterizing the geology, determining direction of groundwater flow, and mapping the areal extent of groundwater contamination. Monitor well construction details for the five newly installed monitor wells and the four previously installed monitor wells, the field geologic logs, and well construction details are summarized in the RFI. The five new monitor wells were installed, developed, and tested in accordance with the RFI Work Plan and as described in the Phase I RFI.

1.3.2.5.1 Two monitor wells (RS264 and RS267) were screened in a perched water-bearing overburden zone, and two wells (RS266 and RS268) were screened in the deeper basal portion of the overburden. The overburden wells are located within the disposal area and downgradient of Area G. One of the shallow overburden wells (RS264) was installed in a perched zone adjacent to an existing deeper overburden well to determine if contamination occurs in the perched zone where contamination previously had not been identified in the deeper well. One bedrock well (RS265) was installed within the disposal area to assist in determining the vertical extent of contamination.

1.3.2.5.2 Samples from the five new monitor wells and from the four pre-existing overburden wells were sampled and analyzed for VOCs and total and dissolved metals

(Appendix A). All of the wells were sampled in February 1991. Water sampling logs are included in the RFI.

Water levels were measured in all of the wells to assist in mapping groundwater flow directions and gradients in the overburden and in the bedrock. In situ permeability tests were conducted on each of the five new wells in accordance with the methods described in the RFI as are the geotechnical tests conducted on four formation samples from three boreholes associated with the new monitor wells.

1.3.3 AREA R (RSA 59)

The following sections describe the monitoring that took place during Phase I of the investigation. No additional sampling was performed in Area R during Phase II. As part of the Phase I RFI, test pits, soils, groundwater, surface water, and sediment were sampled at this area. Figure 1.3-5 shows the location of the test pits, sediment, and soil samples, Figure 1.3-6 shows the monitor well and surface water locations at Area R.

1.3.3.1 Test Pits

Three test pits (RTP-1, RTP-2, and RTP-3) were excavated during Phase I to assist in identifying and characterizing potential contamination sources present within Area R. The characteristics of the rubble fill (large concrete slabs) and metal piping necessitated several attempts before the backhoe could penetrate between the rubble pieces. The locations of the test pits were based on the results of the geophysical surveying, study area observations, and access limitations. One sample was collected from each test pit and analyzed for VOCs, BNAs, and total metals (Appendix A). Soil/waste sampling logs are included in the RFI.

1.3.3.1.1 Air monitoring was conducted in association with the test pit excavations. During the excavation of all of the test pits, air samples were collected and analyzed in the field for volatile organics using a portable gas chromatograph. Field logs for the air monitoring conducted at Area R are presented in the RFI.

1.3.3.2 Shallow Soil Sampling

Two shallow soil samples (RS-11 and RS-12) were collected west and southwest of Area R. The data generated by these samples were used to identify possible additional sources of

contamination to characterize the background chemical composition of the soil. Shallow soil samples were analyzed for VOCs, BNAs, and total metals (Appendix A). The soil sampling logs are included in the RFI.

1.3.3.3 Soil Borings

Soil borings were drilled to assist in characterizing soil/waste in Area R and assist in determining the extent of soil contamination. The locations of the soil borings were based on the results of previous investigations, the geophysical surveying, study area observations, and access limitations. One soil boring (RSB-1) was drilled to help identify and characterize potential sources of contamination present within Area R. One additional soil boring (RSB-2) was positioned in a presumably unimpacted area northwest of Area R. Field geologic logs for the soil borings are included in the RFI. Two samples were collected from each soil boring and analyzed for VOCs, BNAs, and total metals (Appendix A). The soil sampling logs are included in the RFI.

1.3.3.4 Sediment Sampling

Four sediment sampling sites were located in creeks and wetlands that border Area R. The samples from the drainage ditch to the north of the study area and the wetlands east of the study area (RS-7, RS-8, and RS-9) were collected to evaluate the potential for contaminant migration to the surface waters by erosion. Sediment samples RS-8 and RS-9 were collected at the same location as the two surface-water samples, but prior to the collection of the surface-water samples. The sediment sample from the drainage ditch southwest of Area R (RS-10) was collected to evaluate erosion impacts. Sediment samples were analyzed for VOCs, BNAs, and total metals (Appendix A). The sediment sampling logs are included in the RFI.

1.3.3.5 Surface Water Sampling

Collection of two surface water samples from the wetlands on the east side of Area R at the same locations as two of the sediments was originally proposed. In July 1990, when surface water samples were collected from the other RFI units/areas, the wetlands east of Area R had receded and there was no surface water to collect. Surface water samples were collected from the east side of Area R in May 1991, after extensive rains resulted in re-encroachment of the wetlands and flooding. The data from these samples were used to evaluate contaminant

migration from disposal areas to the wetlands via erosion and potentially contaminated groundwater. The surface water samples were analyzed for VOCs, BNAs, and total metals (Appendix A). The water sampling logs are included in the RFI.

1.3.3.6 Monitor Well Installation and Groundwater Sampling

Three overburden monitor wells (RS293, RS294, and RS295) were installed in December 1990 at Area R to supplement data from existing overburden wells. Monitor well construction details for the three new monitor wells and the three previously installed monitor wells, field geologic logs, and well construction details are summarized in the RFI. The three wells were installed, developed, and tested in accordance with the RFI Work Plan and as described in the RFI. Four monitor wells were originally proposed for Area R. Access limitations, including the concrete rubble and unstable fill, as well as the wetlands, limited drilling on the east side of Area R. As a result, two of the proposed wells (RS294 and RS296 in the RFI Work Plan) were consolidated into one centrally located well.

1.3.3.6.1 Groundwater samples from all of the monitor wells were analyzed for VOCs and total and dissolved metals. In addition, samples from the new wells were analyzed for BNAs (Appendix A). The water sampling logs are included in the RFI.

1.3.3.6.2 Water levels were measured in all of the new and existing wells to assist in mapping groundwater flow directions and gradients in the overburden. In situ permeability tests were conducted on each of the four new wells in accordance with the methods described in the RFI, as are the geotechnical tests conducted on two formation samples from boreholes associated with the monitor wells.

1.3.4 AREAS S AND T (RSA 55/54)

As part of the Phase I and Phase II RFI, test pits, soils, groundwater, and sediment were sampled at this area. Figure 1.3-7 shows the location of the test pits, sediment, and soil samples; Figure 1.3-8 shows the monitor well locations at Areas S and T.

1.3.4.1 Test Pits

Seven test pits (TTP-1, TTP-2, TTP-3, and TTP-4 in Area T, and STP-1, STP-2, and STP-3 in Area S) were excavated in October and November 1990 (Phase I). The purpose of the test

pits was to assist in determining the chemical and physical characteristics of the wastes in the disposal areas. The test pits were sited in landfill areas based on the results of geophysical investigations, study area observations, and aerial photographs from which former disposal trenches can be identified. Descriptions of the waste and stratigraphy encountered in each test pit in Areas S and T, together with photographs of test pits, waste, and samples are included in the RFI, as are the soil/waste sampling logs. Thirteen soil/waste samples from the test pits were analyzed for VOCs, BNAs, total metals, and pesticides (Appendix A).

1.3.4.1.1 Air monitoring was conducted in association with the test pit excavation. During the excavation of all of the test pits, air samples were collected and analyzed in the field for volatiles using portable gas chromatograph equipment. During the excavation of test pit TTP-4, air samples were collected and analyzed for volatiles, semi-volatiles, and particulates. Field logs for the air monitoring conducted at Areas S and T are presented in the RFI.

1.3.4.2 Shallow Surface Soil Sampling

A total of four shallow soil samples, TS-3, TS-4, and TS-5 in Area T and SS-3 in Area S, were collected in July and August 1990, within and near the disposal trenches of Areas S and T. One of the shallow surface soil samples (SS-3) was collected within the apparent former disposal area. The remaining three shallow soil samples (TS-3, TS-4, and TS-5) were collected west and north of Area T along Martin Road to investigate areas exhibiting high organic soil vapor that were not previously suspected of containing source material. The four soil samples were analyzed for VOCs, total metals, and pesticides as shown in Appendix A, Table S/T-2. Shallow soil sample SS-3 was also analyzed for BNAs. The soil sampling logs are included in the RFI.

1.3.4.3 Soil Borings

Phase I

A total of eight soil borings were drilled and sampled in August 1990. Five soil borings were drilled in Area T (TSB-1 through TSB-5) and three were drilled in Area S (SSB-1 through SSB-3). Two of the soil borings (TSB-1 and TSB-3) were drilled within apparent former waste disposal areas. Four soil borings, TSB-5, SSB-1, SSB-2, and SSB-3, were drilled outside the area of former disposal activities to assist in delineating the extent of soil contamination. Two soil borings (TSB-2 and TSB-4), one west of Area T and one southeast

of Area T, were drilled to investigate areas exhibiting high organic soil vapor that were not previously suspected of containing source material. Field geologic logs for soil borings drilled in Areas S and T are included in the RFI.

1.3.4.3.1 Two soil samples, one shallow (0 to 2 ft-bls) and one deep (10 to 15 ft-bls) were collected from each boring for laboratory analysis. Soil sampling logs are included in the RFI. The samples from the soil borings were analyzed for VOCs, BNAs, total metals, and pesticides (Appendix A).

Phase II

1.3.4.3.2 Two soil borings were drilled and sampled in April 1992 for the Phase II RFI. Both soil borings (TSB-6 and TSB-7) were drilled at the north end of Area T to confirm the northernmost extent of disposal trenches.

1.3.4.3.3 Two soil samples, one shallow (2 to 4 ft-bls) and one deep (14 to 16 ft-bls) were collected from each boring for laboratory analysis. Soil sampling logs are included in the Phase II RFI. The samples from the soil borings were analyzed for VOCs, PAHs, and organochlorine pesticides (Appendix A).

1.3.4.4 <u>Groundwater</u>

Phase I

Fifteen new monitor wells were installed in October and November 1990 to supplement existing wells at Areas S and T. The purpose of the monitor wells was to assist in characterizing the geology, defining the direction of groundwater flow, and mapping the extent of groundwater contamination.

1.3.4.4.1 Eleven overburden wells were installed including four shallow overburden wells screened in the perched water-bearing zone of the overburden, and seven deep overburden wells screened in the water-bearing zone at the base of the overburden. The shallower perched zone wells were installed only where drilling revealed the existence of a perched zone. A total of four bedrock wells were installed, including two bedrock wells at locations where existing overburden wells previously showed contamination. Monitor well construction details for the 15 newly installed monitor wells and the 14 previously installed monitor wells,

the field geologic logs, and well construction details are summarized in the RFI. The ten new monitor wells were installed, developed, and tested in accordance with the RFI Work Plan and as described in the RFI.

1.3.4.4.2 Two shallow overburden monitor wells (RS297 and RS303), screened in the uppermost water-bearing zone of the overburden, three deep overburden monitor wells (RS298, RS299, and RS305), screened in the basal overburden, and three bedrock monitor wells (RS300, RS301, and RS302), screened in the limestone bedrock, were installed north of Fowler Road (Area T). The data from these and existing wells were used to help establish the lateral extent of contamination as well as the direction of groundwater flow in the northern section of Areas S and T. One shallow overburden monitor well (RS309), two deep overburden monitor wells (RS308 and RS310), and one bedrock monitor well (RS311), screened as described previously, were installed around the south perimeter of Areas S and T south of Fowler Road. The data from these wells and existing wells were used to establish the groundwater flow direction in the southern part of the study area, establish the areal extent of groundwater contamination in the shallow and deep overburden, as well as evaluate the vertical extent of contamination near the study area perimeter. A single shallow overburden monitor well (RS306), located south of Fowler Road in the center of Areas S and T, was installed to provide data to estimate the highest levels of contamination in the shallow overburden beneath this area of the landfill. Two deep overburden monitor wells (RS312 and RS313) installed to the southeast of Areas S and T, provided data concerning the direction of groundwater flow in the overburden as well as establish the areal extent of the contamination previously detected in monitor wells RS038 and RS039.

1.3.4.4.3 The fifteen new monitor wells and the fourteen existing monitor wells were sampled and analyzed for the parameters included in Appendix A. With the exception of RS311, sampled in February 1991, all of the wells were sampled in December 1990. The water sampling logs are included in Attachment III of the QCSR. The eight newly installed monitor wells (RS297, RS298, RS299, RS300, RS301, RS302, RS303, and RS305) and ten existing monitor wells (RS034, RS035, RS036, RS36A, RS037, RS165, RS200, RS202, RS203, and RS223) located to the north of Fowler Road were sampled and analyzed for VOCs, total metals, and dissolved metals. In addition, samples collected from monitor wells near former DDT burial sites, including overburden monitor wells and RS034, RS035,

RS036, RS036A, RS165, RS223, and RS303, were analyzed for pesticides. The seven newly installed monitor wells (RS306, RS308, RS309, RS310, RS311, RS312, and RS313) and the four existing overburden monitor wells (RS038, RS039, RS166, and RS201) south of Fowler Road were sampled for VOCs, total metals, and dissolved metals. Monitor wells near former DDT burial sites, including monitor wells RS038, RS039, RS306, and RS309, were also analyzed for pesticides.

1.3.4.4.4 Water levels were measured in all of the new and existing wells to assist in mapping groundwater flow directions and gradients in the shallow and deep overburden and in the shallow and deep bedrock. In situ permeability tests were conducted on each of the fifteen new wells in accordance with the methods described in the RFI report, which also provided methods for the geotechnical tests conducted on ten samples from seven of the fifteen boreholes associated with the monitor wells.

Phase II

1.3.4.4.5 Four new overburden monitor wells (RS304, RS307, RS384, and RS385) were installed in May and July 1992 for the Phase II RFI to supplement existing wells at Areas S and T. The purpose of the monitor wells was to assist in characterizing the geology, defining the direction of groundwater flow, and to further delineate the extent of groundwater contamination in the perched and basal overburden west of Areas S and T.

1.3.4.4.6 The overburden wells included two shallow overburden wells screened in the perched water-bearing zone of the overburden (RS384 and RS385), and two deeper overburden wells screened in the water-bearing zone at the base of the overburden (RS304 and RS307). Monitor well construction details for the four newly installed monitor wells and the 29 previously installed monitor wells are summarized in the RFI as are the field geologic logs and well construction details. The four new monitor wells were installed, developed, sampled, and tested in accordance with the RFI Work Plan and as recommended in the Phase I RFI.

1.3.4.4.7 The four new monitor wells were sampled and analyzed for VOCs (Appendix A). Ten existing wells (RS306, RS310, RS037, RS036A, RS036, RS034, RS309, RS308, RS035, RS303) were also sampled and analyzed for PAHs. These wells were sampled to help

determine whether or not the PAHs detected in the soils/wastes had migrated to groundwater in the overburden.

1.3.4.4.8 In addition to completing the contamination assessment, the Phase II field investigation presented an opportunity to collect readily obtainable data that may be needed for the development of corrective measures. During the Phase II field investigations, two composite groundwater samples were prepared in the field. One from monitor wells RS036A and RS039, and a second from RS201 and RS305. Each of the two composite samples were analyzed for conventional water-quality parameters including total suspended solids, total dissolved solids, total hardness, calcium hardness, 5-day bio-chemical oxygen demand (BOD_5), chemical oxygen demand (COD), alkalinity, total organic carbon, chloride, sulfate, iron, manganese, magnesium, sodium, potassium, and calcium.

1.3.4.4.9 Water levels were measured in all of the new and existing wells to assist in establishing groundwater flow directions and gradients in the shallow and deep overburden and in the shallow and deep bedrock. Geotechnical tests were conducted on four samples from two of the four boreholes associated with the monitor wells installed during Phase II. In situ permeability tests were conducted on each of the four new wells in accordance with the methods described in the Phase II RFI.

1.4 REPORT ORGANIZATION

1.4.1 DESCRIPTION OF BRA PROCESS

Typically, BRAs conducted at Superfund sites are complex, involving extensive data evaluation and multiple exposure pathway analysis at the site. The BRA process consists of the following primary components:

1. Hazard identification/selection of chemicals of potential concern (COPCs),
2. Exposure assessment,
3. Toxicity assessment,
4. Risk characterization, and
5. Development of remedial goal objectives.

1.4.1.1 The BRA is conducted based on the EPA manual, Risk Assessment Guidance for Superfund (RAGS), Volume I, Part A, Human Health Evaluation Manual (EPA, 1989a); EPA

Region IV; and published supplements to this manual as well as relevant EPA and EPA Region IV guidance regarding exposure and toxicity assessments. The ecological impacts are evaluated based on RAGS, Volume II, Environmental Evaluation Manual (EPA, 1989b); Ecological Assessment of Hazardous Waste Sites (EPA, 1989c); ECO Updates (issued intermittently by EPA to supplement RAGS) as well as relevant EPA guidance on exposure and toxicity assessments.

1.4.1.2 BRAs begin with an introduction to the study area, also known as a site characterization, which presents an overview of historical information and site monitoring data to gain an understanding of the history of the operations and dynamics of the study area and surrounding environment (Section 1.0). This information is used to develop a conceptual site model. Hazard Identification involves the evaluation of the site monitoring data to ascertain which chemicals are site related and should be evaluated further in the BRA to assess potential human health and ecological impacts (Section 2.0). The exposure assessment for the five study areas was conducted to estimate the magnitude, frequency, and pathways of actual and/or potential exposures to human and ecological receptor populations (Section 3.0). Fate and transport of the study area related chemicals at the five areas is evaluated and included as part of Section 3.0. Section 4.0, toxicity assessment, summarizes the potential health and ecological impacts and the dose-response information for the COPCs identified at the study area. The last step of a BRA, risk characterization (Section 5.0), integrates the information obtained in the exposure and toxicity assessment sections to provide an estimate of health risk to human and ecological receptor populations. The results of the risk characterization are used to identify which contaminants and environmental media need to be addressed in the feasibility study (FS), thus, Section 6.0 provides a summary of the preliminary remedial cleanup goals developed to serve as remedial action objectives to be considered in the FS. A summary of each BRA component is presented in Section 7.0, along with the uncertainties inherent in the BRA.

1.4.2 CONCEPTUAL SITE MODEL FOR BRA 1

Based on a review of the study area operations and history, as well as a preliminary evaluation of the site monitoring data, a conceptual site model was prepared for each of the five areas addressed in this BRA (see Figures 1.4-1 through 1.4-4). Due to the close proximity of Areas S and T, these two areas are presented in one CSM. The models provide

a graphical representation of the environmental concerns for each area. The model shows the environmental systems and the biological, physical, and chemical processes (i.e., chemical release mechanisms) that determine the transport of contaminants from sources (i.e., primary and secondary sources) at each study area to potential receptors within the system.

1.4.2.1 The primary contaminant sources of each area are the locations where contaminants were disposed (i.e., pond or landfill), the secondary sources are environmental media that have received contamination from the primary source (i.e., underlying groundwater). Contaminant release mechanisms are processes that may affect the fate and transport of contaminants (i.e., volatilization or leaching from soil); and potential exposure pathways to receptors is the method by which a human or ecological receptor may come into contact with a contaminant (i.e., fish exposed to groundwater contaminants as a result of groundwater discharge to surface water; or a worker exposed to soils as a result of incidental direct contact with soil during work activities). The conceptual site model serves as the foundation from which to generate the BRA, which further evaluates this information in a quantitative manner.

2.0 HAZARD IDENTIFICATION/ IDENTIFICATION OF COPCs

COPCs are the study area-related chemicals that may pose the most critical health concerns to human health and/or environmental receptors. According to EPA methods for Superfund risk assessments (EPA, 1989a), if adequate computing facilities are not available, analytical data may be screened to limit the number of chemicals that are carried through the risk assessment process to those that are study area related and pose the most significant health concern. This screening uses chemical classes, frequency of detection, essential nutrient information, and a concentration-toxicity screen to reduce the number of COPCs. Such a screening would normally result in a list of COPCs that is limited to contaminants at a study area believed to be the most toxic to humans and/or biota, most mobile, and/or most frequently detected. However, this screening method is often no longer necessary due to the ability to include all relevant constituents in the risk assessment process through the use of automated data management systems. Instead of limiting the number of chemicals that undergo complete evaluation to those that are most likely to be toxic or harmful, computers enable the risk assessor to completely evaluate all pertinent data. Since EPA (1989a) recommends that COPCs not be further reduced if sufficient computing facilities are available, the only data screening that was performed for this BRA was the elimination of contaminants that apparently are not study area related (laboratory contaminants were eliminated), and contaminants that were not detected at levels significantly above background.

2.0.1 To understand the process by which COPCs for this BRA were determined, the data collection and evaluation procedures will be discussed in general terms before the analytical data are presented and discussed. The analytical results, presented in tabular form, are followed by a discussion of the nature and extent of contamination at each area, and a comparison of soil concentrations versus background levels.

2.1 GENERAL DATA COLLECTION CONSIDERATIONS

The source of all data to be used in this BRA is the data set presented in the RFI reports (G&M, 1991a; 1992a). According to the HEA presented in the RFI reports (G&M, 1991a; 1992a), data evaluation was carried out in accordance with EPA guidance (EPA, 1989). To most effectively use available resources, the data collected during the RFI efforts are the data upon which this BRA is based. The summarized and compiled data from the RFI reports

were directly incorporated into the calculations found in this BRA. The discussion of which parameters were considered during the initial sampling efforts can be found in the G&M 1991 and 1992 RFI reports.

2.1.1 The available data include surface soil, subsurface soil, groundwater, surface water, and sediment analytical results and are presented in Appendix A. The summarized data compiled for Areas F (RSA 49), G (RSA 48), R (RSA 59), and S/T (RSA 55 and 54) are presented in Apppendix A.

2.2 GENERAL DATA EVALUATION CONSIDERATIONS

Steps that must be taken during the data evaluation process include the evaluation of analytical methods; the determination of data quality with respect to sample quantitation limits, qualifiers, codes and blanks, including quality assurance/quality control (QA/QC) methods; and the evaluation of tentatively identified compounds (TICs). Data evaluation also includes study area-specific data considerations to include comparing potential study area-related contamination with study area background concentrations; and reviewing historical information and onsite data collection results to identify study area-related chemicals.

2.2.1 FLAGGED DATA

Although a chemical may be detected, its quantitative concentration value may be uncertain for several reasons. These reasons are discussed in the QA/QC methods for evaluating analytical results, and the specific data points that are affected are denoted by a flagging code (G&M, 1991a, 1992a). The type of flagging code used is based on the nature of the uncertainty. Flagging codes for the analytical results are also described in the QA/QC report (G&M, 1991a, 1992a) and are also presented in the data summary table (Appendix A). The flagging code identified throughout the data set for this BRA is the flagging code "J." This flagging code is the most commonly encountered data qualifier in Superfund data packages (EPA, 1989), and indicates a qualitative value is presented because the value is below laboratory reporting limits but above the instrument detection limit. A J value indicates uncertainty in the reported concentration but not in its assigned identity. Thus, based on EPA Region IV and EPA region-wide guidance, the J-qualified data should be used the same way as positive data that do not have this qualifier to ensure that a study area-related chemical is not overlooked. Use of J values ensures that potential exposures are conservatively addressed

to ensure the protection of public health. A summary of the J-qualified data is presented in Tables 2.2-1 through 2.2-4 for Areas F, G, R, and S/T, respectively.

2.2.2 BLANK CONTAMINATION

According to RAGS (EPA, 1989a), to prevent inclusion of nonstudy-area-related contaminants in the risk assessment, the concentrations of chemicals detected in blanks must be compared with concentrations of the same chemicals detected in study area samples. According to the data evaluation presented in the RFI (G&M, 1992a), constituents that were classified as undetected due to contamination in laboratory or field blanks [i.e., acetone, methylene chloride and bis(2-ethylhexyl)phthalate] were included in the HEA because the levels detected in study area samples were similar to those levels detected in the blanks.

2.2.2.1 Typically, if the study area sample concentration exceeds ten times the maximum amount detected in any blank, the study area concentration should be considered a positive result (EPA, 1989a). Otherwise the sample value should be evaluated as if the reported level was less than the level reported for the blank. However, to provide a conservative estimate of the risk associated with the constituents associated with laboratory contamination, the concentrations used in the HEA are equal to the qualified value. Based on a review of the data and history information, common laboratory contaminants such as acetone, methylene chloride, and bis(2-ethylhexyl)phthalate could be associated with the study area and were identified in most media sampled at all study areas. Thus, as with the HEA, to ensure the protection of human and ecological health, the common laboratory contaminants were included in the BRA risk characterization process. In the event the study area risks are driven by potential laboratory contaminants, the risks will be re-evaluated to account for the introduction of laboratory contaminants in study area samples.

2.2.3 BACKGROUND LEVELS

The presence of inorganics in onsite samples may or may not be associated with study area activities. To determine if inorganics are associated with study area activities, the onsite concentrations are compared to inorganic concentrations detected offsite near the installation. The concentrations of inorganics detected offsite are referred to as background concentrations. EPA Region IV guidance recommends that maximum onsite concentrations be compared with two times the average background concentrations. However, only ranges of background

concentrations were presented in the RFI (G&M, 1991). As a result, average onsite concentrations were compared with two times the maximum background concentrations. Thus, EPA Region IV's guidance was applied based on the data presented in the RFI. Section 2.4.2 discusses the potential impacts of not being able to use average background concentrations in this comparison.

2.2.3.1 Study area-specific background samples were collected at RSA (G&M, 1991) and were used to compare against concentrations of inorganics detected at each of the five study areas. Information on background chemicals may also be obtained from other sources such as the EPA Hazardous Waste Land Treatment Guidance (EPA, 1983).

2.2.3.2 If the COPCs are present in background samples, the origin of the COPCs and whether they are naturally occurring or anthropogenic should be established. Anthropogenic chemicals should not be eliminated from the risk analysis. If the study area-related levels of inorganic chemicals are similar to the naturally occurring levels, they are eliminated from quantitative risk analysis. To determine whether the inorganics present are study area related or are representative of background concentrations, the average study area-specific background concentrations are compared to the average concentrations of inorganics detected in soil at each study area. According to EPA Region IV guidance, inorganics should be included as COPCs if they are detected at concentrations significantly above background samples (the criteria for determining significance should generally be two times the background concentration) (EPA, 1991).

2.3 STUDY-AREA-SPECIFIC RESULTS

The following sections present a summary of the data evaluation for each study area to include a description of the specific constituents and the general nature and extent of the contamination that was detected at each of the five areas. Following these discussions the levels of inorganic contaminants that were detected in soils are compared to background soil levels for that area.

2.3.1 AREA F (RSA 49)

2.3.1.1 Sampling Results

A variety of constituents were detected in the test pit, shallow surface soil, subsurface soil, sediment, surface water, and groundwater samples collected within and near the former Area F arsenic ponds. Selected media from Area F were collected for analysis of BNAs, VOCs, and metals. Appendix A summarizes the analytical results from Phase I and Phase II field investigations. Table 2.2-1 presents a summary of the J-qualified data. A J value indicates uncertainty in the reported concentration, but not in its assigned identity. Thus, inclusion of the results are important in risk characterization to ensure the protection of human and environmental health.

2.3.1.2 Nature and Extent of Contamination

Potential sources of contamination at Area F (RSA 49) include the closed arsenic waste ponds and the media at the location of the former lewisite manufacturing plant previously located south of the ponds. The principal constituents found at Area F were metals (arsenic, cadmium, and mercury) and PAHs in the soils; and arsenic, mercury, and chlorinated hydrocarbons (primarily carbon tetrachloride) in the groundwater. A summary of the nature and extent of the contamination is presented in the following paragraphs, focusing on the principal constituents that were detected in study area media and the overall horizontal and vertical extent of the contamination.

Soil Contamination

2.3.1.2.1 Soils in Area F (RSA 49) were sampled during both Phase I and II of the RFI investigation. The majority of the impacted soils at Area F appear to be within the closed disposal ponds. To a lesser extent, soils and sediments containing arsenic and PAHs have been detected south of Area F, which is north of the former lewisite manufacturing plant. PAHs appear to be restricted to very shallow soils (less than 5 ft-bls) and sediment at selected study areas within and outside Area F. The PAHs are likely related to the rubble and construction debris that were disposed of in the ponds (asphalt and roofing materials, for example). The highest concentrations of arsenic occur in soils and waste within the former disposal ponds at depths that are generally less than 10 ft-bls. At depths greater than 10 ft, relatively clean native soils, with lower levels of arsenic, were found. The dense clays in the native soils apparently have prevented the migration of significant quantities of arsenic

through the soils. Boring and sediment samples from the Phase I and Phase II field investigations taken from areas outside of the study area boundary generally contained levels of arsenic within the range for natural soils. Exceptions to this finding were FSB-3 and some of the sediments in ditches south of Area F that had elevated levels of arsenic.

2.3.1.2.2 Based on both the chemical data and field observations, the vertical extent of significantly impacted soils/waste appears to be restricted to soils from 0 to 11 ft-bls. Construction debris and a grey-white clayey material characteristic of arsenic-contaminated wastes were detected in several of the test pits. Native soils were generally encountered below 11 ft. The chemical data support the field observations that were made during Phase I and Phase II of the RFI.

2.3.1.2.3 The horizontal extent of impacted soils appears to be limited to the area within the boundary of Area F, and sediments contained in the drainage pathways located south of Area F. The elevated arsenic levels south of the study area boundary may be attributed to runoff or may indicate contamination related to the former lewisite manufacturing plant located south of Area F.

Groundwater Contamination

2.3.1.2.4 Sampling data indicate that groundwater is impacted by significant concentrations of mercury in the immediate vicinity of RS258 and that migration of substantial quantities of the metal has not occurred. In addition, the reduction in concentration from the total to the dissolved results suggest that the majority of the metal is bound to the soils and not available for transport.

2.3.1.2.5 The remaining groundwater data indicate that arsenic and chlorinated hydrocarbons were the most widely distributed constituents of concern at Area F. During Phase I and II, the most significant concentrations of these constituents in the groundwater occurred in the center and south of Area F. Monitor well RS052 (35 ft deep), located north of the closed ponds, is the only well installed in a shallow (perched) aquifer. Arsenic was detected at a concentration of 57 micrograms per liter (μg/L) in this shallow well during one sampling event.

2.3.1.2.6 Several groundwater samples were collected during Phase II for analysis of PAHs to determine whether or not PAHs, which had been detected at significant concentrations in the soil during Phase I field investigations, had migrated to the groundwater. No PAHs were detected in the groundwater sampled from Area F.

2.3.1.2.7 Regional groundwater flow direction in the deep overburden (approximately 40 to 60 ft-bls) is southerly toward the Tennessee River. Groundwater direction flow maps for Area F are not present in the RFI. The highest concentrations of arsenic and chlorinated hydrocarbons (primarily carbon tetrachloride) were reported in samples from wells installed south of the closed disposal ponds of Area F. The highest dissolved arsenic level was detected at RS263 (99.8 μg/L). Elevated concentrations of arsenic (68 μg/L) were also detected in a well installed in the center of the closed ponds. The RFI reported that the extent of contamination in the deep overburden appears to be defined on the east, southwest, and west by low levels of dissolved arsenic. The same pattern exists for the chlorinated hydrocarbons (primarily carbon tetrachloride).

2.3.1.2.8 Groundwater contamination in the upper bedrock was found south, but not north of the study area. Arsenic contamination in well RS261 may result from leakage from the deep overburden as there does not appear to be a confining unit between the deep overburden and the upper bedrock. No constituents of concern were detected in groundwater samples collected from the deep bedrock monitor well at RS259 (134 ft deep) installed in the center of the Area F disposal ponds.

Surface Water Contamination

2.3.1.2.9 Surface water sample SW-F-1, collected during Phase I field investigations, was free from the constituents for which the sample was analyzed and that appear to be associated with the study area.

2.3.1.3 Soil Concentrations Versus Background Levels

Phase I

Subsurface soil samples that were collected from the deep part of the soil borings from Area F showed relatively low levels of metals. Table 2.3-1 presents the ranges expected for natural soils (EPA, 1983) and the background for RSA, as well as the average concentrations

of metals detected in soil/waste, surface soil, and subsurface soil samples. The average concentration of arsenic, cadmium, and mercury in the soil/waste and shallow samples was more than twice the concentration observed in either the range for natural soils and/or RSA background soils; therefore, these three inorganics are included for further soil risk analysis for Area F.

Phase II

2.3.1.3.1 Waste analyses results indicate that arsenic, cadmium, and mercury were well above the range expected for natural soils and background soils at RSA. The results of the subsurface analysis indicate that arsenic and mercury exceed the range expected for natural soils and background soils at RSA. Phase II soil results confirm that arsenic, cadmium, and mercury must be further evaluated in the risk characterization of soils at Area F.

2.3.2 AREA G (RSA 48)

2.3.2.1 Sampling Results

A variety of constituents were detected in the wastes, surface soil, subsurface soil, sediment, surface water, and groundwater samples collected within and near the former landfill and rubble fill of Area G. Selected media were sampled for analysis of BNAs, VOCs, and metals during the Phase I RFI, but only surface water was sampled as part of the Phase II RFI at this area. Appendix A summarizes the analytical results from the Phase I and Phase II field investigations. A summary of the J-qualified values is presented in Table 2.2-2.

2.3.2.2 Nature and Extent of Contamination

The potential sources of contamination associated with Area G are the disposal trenches where construction debris and sanitary wastes were disposed of. The principal constituents found at Area G were PAHs in the soil and trichloroethene in the surface water. The following discussion of the nature and extent of soils and groundwater contamination at Area G have been excerpted from the Phase I RFI Report (G&M, 1992a), as no new sampling in these media was conducted during Phase II. The discussion of the nature and extent of surface water contamination is based on the results of both the Phase I and Phase II investigations.

2.3.2.2.1 A summary of the nature and extent of the contamination is presented in the following paragraphs, focusing on the principal constituents that were detected in study area media and the overall horizontal and vertical extent of the contamination.

Soil and Sediment Contamination

2.3.2.2.2 Detectable levels of PAH concentrations in the soil sampled in September and October 1990, occur exclusively within the test pits located within the boundaries of Area G. Outside the boundaries, all soil borings were free of PAHs. Sediment samples taken outside the boundaries were not analyzed for PAHs. Although low levels of metals were detected in sediment, the samples were free of VOCs (except for laboratory artifacts). The overall nature of the soil contamination at Area G is similar to that expected for a sanitary landfill and is not indicative or representative of substantial chemical/hazardous waste disposal.

2.3.2.2.3 The vertical and horizontal extent of solid debris in the landfill area has been characterized. Solid debris was encountered to depths of 8 to 12 ft-bls within the boundaries of Area G. All soil borings and monitor wells installed outside the limits of Area G encountered only native soils to their total depths. The areal extent of solid waste is outlined by the boundary of Area G, except for the southwestern edge. No debris was encountered during the installation of monitor well RS111, and the topography indicates that the southwestern limit of solid waste probably exists between RS111 and test pits GTP-1 and GTP-3.

Groundwater Contamination

2.3.2.2.4 Groundwater flow has been divided into the following three zones at Area G: (1) the shallow overburden (perched), (2) the deep overburden, and (3) the bedrock. Groundwater direction flow maps for Area G are not available in the RFI. One well (RS265) was installed in the upper bedrock to a depth of 85 ft-bls. Three wells (RS112, RS264, and RS267) were installed in localized perched zones and may share a hydraulic connection. The remaining wells were installed in the deep overburden where groundwater flow direction appears to be east-northeast toward McDonald Creek.

2.3.2.2.5 Acetone was the only VOC detected in groundwater at Area G. Sampling efforts indicated that arsenic, barium, cadmium, chromium, and lead are present in groundwater.

Several of the total metal concentrations in the unfiltered groundwater samples exceeded MCLs.

Surface Water Contamination

2.3.2.2.6 Low levels of metals including barium, lead, and mercury were detected in surface water adjacent to Area G. However, lead and mercury levels do not appear to be site related because samples upstream from the site had detections of lead and mercury, but samples downstream from the site were below detection and surface soils at the site were within background levels for lead and mercury. In addition, the area is not prone to flooding, so there is no release mechanism for lead and mercury. Trichloroethene is considered to be the primary constituent of concern in this medium since it was detected in all five of the surface water sampling locations in McDonald Creek on the eastern edge of the Area G landfill. Two of the surface water samples containing trichloroethene were collected from locations upstream of Area G. In addition, the trichloroethene is inconsistent with the general contamination found onsite (PAHs in the soils, no trichloroethene contamination in the groundwater). It is unlikely that Area G is the source for the trichloroethene. A source of trichloroethene contamination upstream (north) of Area G is likely impacting the water quality in McDonald Creek.

2.3.2.3 Soil Concentrations Versus Background Levels

Metals analyses indicated low levels of inorganics in soil/waste samples. As presented in Table 2.3-2, the average concentrations of metals in the soil/waste, surface soil, and subsurface soil samples were within the range expected for natural soils (EPA, 1983) and the background soils for RSA. Based on this comparison, inorganics were not included as COPCs in soil for Area G.

2.3.3 AREA R (RSA 59)

2.3.3.1 Sampling Results

Sampling in Area R (RSA 59) was only conducted during Phase I. A variety of constituents were detected in the surface soil, subsurface soil, sediment, surface water, and groundwater samples collected within and near the rubble fill of Area R. Selected media were sampled for analysis of BNAs, VOCs, and metals. Appendix A summarizes the analytical results from

Phase I and Phase II field investigations. A summary of the J-qualified values is presented in Table 2.2-3.

2.3.3.2 Nature and Extent of Contamination

The potential sources of contamination for Area R include: (1) the construction debris/rubble/ industrial landfill into which a variety of undocumented industrial waste was disposed; and (2) past discharges from the former chlorine plant adjacent to Area R. The principal constituents of concern found at Area R were PAHs in the soils and chlorinated hydrocarbons in the ground and surface waters. The following discussion has been excerpted from the Phase I RFI Report (G&M, 1992a). No sampling was done at Area R during Phase II investigations.

2.3.3.2.1 A summary of the nature and extent of the contamination is presented in the following paragraphs, focusing on the principal constituents that were detected in study area media and the overall horizontal and vertical extent of the contamination.

Soil Contamination

2.3.3.2.2 Low levels of several metals (arsenic, barium, chromium, lead, mercury, and selenium) were detected in shallow and subsurface soil samples. PAHs were detected in shallow soil samples, sediment samples, and the shallow portions of the soil borings and test pits. The highest levels occurred in the shallow sample from soil boring RSB-1 [16,270 micrograms per kilogram (μg/kg)] and sediment samples RS-7 (7,400 μg/kg) and RS-9 (1,820 μg/kg). The overall nature of the soil contamination at Area R was found to be similar to that expected for ordinary rubble, asphalt, and construction debris.

2.3.3.2.3 The horizontal extent of solid debris in the landfill can be observed visually, but was also defined by geophysical survey, soil borings, and test pits. Borings at locations within the boundary lines all encountered construction debris and rubble fill. The vertical extent of solid debris in the landfill area was also defined by the soil borings and test pits. Solid wastes were generally found no deeper than 3 to 6 ft-bls, and the horizontal limits appear to agree with the boundary of Area R.

2.3.3.2.4 All of the sites where PAHs were detected were located within or immediately adjacent to the landfill. Sampling results indicate that PAH contamination is limited to the landfill and that the PAHs have not migrated to the underlying native soils.

Groundwater Contamination

2.3.3.2.5 Monitor wells were installed exclusively in the deep overburden at Area R. The thickness of the overburden is inconsistent, varying from approximately 17 ft-bls at RS222 to 49 ft-bls at RS221. The groundwater flow direction appears to trend toward the wetlands east of the study area. The RFI does not have groundwater direction flow maps for Area R.

2.3.3.2.6 Arsenic, barium, cadmium, chromium, and lead were detected in groundwater at Area R. Several of the total metal concentrations exceeded established MCLs, although dissolved metal concentrations did not exceed individual MCLs. Low-level chlorinated hydrocarbon contamination was detected randomly in the groundwater in February 1991. Chloroform was detected southeast of Area R and is presumably upgradient of Area R. The chloroform could be a consequence of past discharges of sodium hypochlorite from the former chlorine plant adjacent to Area R followed by reaction of the hypochlorite with natural organic compounds to produce chloroform. None of the PAHs detected in the soil/waste samples from the soil borings, test pits, and sediment samples were detected in the groundwater samples.

Surface Water Contamination

2.3.3.2.7 Of the two surface-water samples collected at Area R, only samples from SW-R-2, located east and downgradient of Area R operations, contained constituents of concern (primarily chloroform at 8 μg/L). Neither surface water sample contained detectable PAHs. Several inorganics detected at low levels included arsenic, barium, and silver. Because silver was detected only once and was not detected in any waste, soil, or groundwater samples, silver was excluded from further analysis as it does not appear to be site related.

2.3.3.3 <u>Soil Concentrations Versus Background Levels</u>

Metals analyses indicated low levels of arsenic, barium, chromium, lead, mercury, and selenium (Table 2.3-3). The average concentrations of metals detected in waste, surface soil, and subsurface soil samples were generally within the range expected for natural soils and

RSA background soils. Because the average concentrations of inorganics detected at Area R are less than twice the background concentrations, inorganics in soil were not included for further evaluation in the risk analysis of soils at Area R.

2.3.4 AREAS S AND T (RSA 55 AND 54)

2.3.4.1 Sampling Results

A variety of constituents were detected in the test pit, shallow surface soil, subsurface soil, and groundwater samples collected within and near the former sanitary and industrial landfill disposal sites of Areas S and T. Selected media were sampled for analysis of BNAs, VOCs, metals, and pesticides. Appendix A summarizes the analytical results from the Phase I and Phase II field investigations. A summary of the J-qualified values is presented in Table 2.2-4.

2.3.4.2 Nature and Extent of Contamination

The apparent source of contamination at Areas S and T is the network of closed disposal trenches. The principal constituents found at Areas S and T are PAHs, chlorobenzene, and acetone in the soils; and PAHs, chlorobenzene, and other chlorinated hydrocarbons in the groundwater. The most significant concentrations of PAHs, chlorobenzene, and DDT are found in the soils in the region defining the area of closed disposal trenches in both Areas S and T. The most significant concentrations of chlorobenzene and other chlorinated hydrocarbons in the groundwater appear to concentrate in the southwestern quarter of Area T. PAHs in the groundwater were restricted to monitor wells within and immediately adjacent to the disposal trenches in Areas S and T.

2.3.4.2.1 A summary of the nature and extent of the contamination is presented in the following paragraphs, focusing on the principal constituents that were detected in study area media and the overall horizontal and vertical extent of the contamination.

Soil Contamination

2.3.4.2.2 The only organics detected in soil include pesticides and organics that are suspected laboratory contaminants. Numerous samples were reported to contain detectable levels of the chlorinated pesticide 4,4'-DDT and/or its degradation products 4,4'-DDE and 4,4'-DDD. Although the majority of the positive results indicate the presence of trace levels of

constituents, samples from soil boring TSB-4 located in the southeast corner of Area T contained substantially elevated levels of 4,4'-DDT.

2.3.4.2.3 The only BNAs and VOCs detected in the deep soil boring samples were common laboratory contaminants bis(2-ethylhexyl)phthalate, acetone, and methylene chloride. Two of the eight deep soil boring samples collected from Areas S and T contained 4,4'-DDT and/or its metabolites. Another organochlorine pesticide detected was alpha-BHC. Though barium and chromium were found in all deep soil boring samples collected from Areas S and T, the concentrations were only slightly elevated. Traces of arsenic, and lead were detected in individual samples. No PAHs, chlorobenzene, or pesticides were detected in samples from the soil borings collected north of the area, thereby defining both the extent of the disposal trenches and the lateral extent of contamination north of the disposal trenches.

Groundwater Contamination

2.3.4.2.4 Four water-bearing zones have been defined at Areas S and T; the shallow overburden (perched), the deep overburden (basal), the upper bedrock, and the deep bedrock. Previous investigations did not indicate groundwater contamination in the upper bedrock in Areas S and T with one exception, well RS203. For the most part, the upper bedrock is competent, and acts more as an aquitard than as a means for groundwater migration. Groundwater flow directions in the shallow overburden zone, deep overburden zone, and deep bedrock zone for Area S/T (RSA 55/54) are shown in RFI Figures IX-5, IX-6, and IX-7, respectively.

2.3.4.2.5 Groundwater flow in the perched zone is north-northeast near Area T and south-southwest near Area S. VOC contamination in this zone appears concentrated in the southwest quarter of Area T as reflected in concentrations of 3,000 μg/L chlorobenzene at well RS037. Diminishing concentrations of chlorobenzene are found along the lines of northward groundwater flow. The extent of contamination is defined by the relative absence of constituents detected to the north in shallow overburden wells RS297 and RS200, to the east in wells RS223 and RS038, to the south in wells RS309 and RS039, and to the west in RS385 and RS384.

2.3.4.2.6 Groundwater in the deep overburden flows northwest in the northern section of the study area and northeast in the southern portion of Areas S and T. Contamination in this zone is derived primarily from vertical leakage of chlorobenzene from the trenches in the southwest corner of Areas S and T. The highest contamination concentrations derived from this source are reflected in chlorobenzene concentrations and other chlorinated hydrocarbons found at wells RS201 (100 μg/L total chlorinated hydrocarbon) and RS308 (285 μg/L total chlorinated hydrocarbon), and in lower concentrations downgradient at RS036A (87 μg/L chlorobenzene). The chlorobenzene plume is defined by the absence of concentrations to the north in wells RS298, RS202, RS165, and RS299, to the southeast at well RS312, to the south at well RS166, and to the west at wells RS304 and RS305. The total chlorinated hydrocarbon plume is generally similar to the chlorobenzene plume, except that it extends southeast and southwest at nominal levels in wells RS313 (13 μg/L) and RS307 (13 μg/L).

2.3.4.2.7 Groundwater flow in the deep bedrock zone during Phase II was north. No significant contamination was detected in this water zone that typically was encountered between 460 and 470 feet above mean sea level (ft-amsl) (130 and 150 ft-bls). Well RS311, drilled to 250 ft-bls, did not encounter a significant water bearing unit and was left as an open hole in bedrock without a discrete-screened interval. Subsequently, the 33 μg/L of total chlorinated hydrocarbons detected in the sample may be due to downward leakage of contaminants from the deep overburden into the upper portions of the bedrock near this well.

2.3.4.3 Soil Concentrations Versus Background Levels

Except for silver detected in waste samples, the average concentrations of metals in shallow soil and subsurface soil samples were within the range expected for natural soils (EPA, 1983) and the background for RSA (Table 2.3-4). Because silver was detected at concentrations higher than twice the background concentrations, silver was included for further risk analysis of soils at Areas S and T. Additional soil sampling was conducted as part of the Phase II sampling activities at Areas S and T; however, these samples were not analyzed for metals. While elevated levels of lead were detected in test pit samples [i.e., 548 milligrams per kilogram (mg/kg) in TTP-1A; 648 mg/kg in TTP-1B], the average concentration was within two times background for surface soil. To ensure protection of human health as a result of potential impacts of subsurface soil to groundwater, lead was considered a COPC for groundwater risk characterization. Although exposure to subsurface soil and buried waste is

an incomplete exposure pathway, lead has been selected as a COPC in soil for this site, based on its presence in other media (groundwater and sediment).

2.4 GENERAL DATA UNCERTAINTY

As with most NPL sites, the data used for this BRA were collected over time and by different contractors using different QA/QC procedures. This accounts for the variation in the analytical methods used and detection limits reported. Only Level 3 data [U.S. Army Toxic and Hazardous Materials Agency (USATHAMA), 1992] was used in performing this risk assessment. The potential uncertainty is reduced by using automated methods for statistical analysis of the data. The air sampling collected during test pit excavations were included for evaluation of potential inhalation exposure to vapors. The uncertainty associated with the vapors released during the excavations is high, as the field screening measurements are not compound specific. Further discussion of the uncertainty is presented in Section 5.0.

2.4.1 While blank contamination may result in inclusion of nonstudy-area-related contaminants in the risk assessment, the concentrations of chemicals detected in blanks were included in the BRA as many of the blank contaminants could be related to the study area and their inclusion provides a conservative estimate of the risk associated with those constituents.

2.4.2 Although average background inorganic concentrations in soil were not available for comparison since the data were adopted from the RFI (G&M, 1991), the deviation from EPA Region IV's recommended use should not greatly impact the outcome of this analysis. Since a majority of the inorganics were COPCs for groundwater at all five areas (Table 2.5-1), these inorganics were carried through the risk assessment by groundwater although most inorganics were not COPCs for soil.

2.5 SUMMARY OF COPCs

According to RAGS (EPA, 1989) quantitative evaluation of all available data is recommended; however, for study areas with a large number of potential COPCs, an optional process to shorten the list of COPCs can be performed. However, due to the availability of an automated data management system, all the chemicals, except for those that are representative of background conditions or are associated with common laboratory

contaminants, are processed through the quantitative risk evaluation for the BRA. A summary list of the COPCs for this BRA are presented in Table 2.5-1.

Table 2.5-1. Summary of COPCs at Areas F, G, R, and S/T

Parameter	Area F				Area G				Area R				Area S/T			
	SOIL	GW	SW	SED	SOIL	GW	SW	SED	SOIL	GW	SW	SED	SOIL	GW	SW	SED
Inorganic Chemicals																
Arsenic	√	√		√		√		√		√	√	√				
Barium		√	√	√		√	√	√		√	√	√		√		
Cadmium	√	√		√		√				√		√		√		
Chromium		√		√		√		√		√		√		√		
Lead		√		√		√				√		√		√		
Mercury	√	√		√								√		√		
Selenium														√		
PAHs*	√			√					√			√		√		
Pesticides																
DDT													√			
DDE													√			
Semivolatile Organic Chemicals																
Bis(2-ethylhexyl)phthalate	√								√	√	√	√	√			
Bis(2-chloroethyl)ether										√						
Butylbenzylphthalate									√							
VOCs																
Acetone	√	√		√	√	√	√	√	√	√		√	√	√		
Benzene														√		
Carbon tetrachloride		√		√								√				
Chlorobenzene										√				√		
Chloroform		√								√	√			√		
Dichloroethane, 1,1-							√					√				
Dichloroethane, 1,2-														√		
Dichloroethenes, 1,2-,total														√		
Ethylbenzene														√		
Methylene chloride	√			√	√		√	√	√	√		√	√	√		
Tetrachloroethene														√		
Toluene														√		
Trichloroethane, 1,1,1-														√		
Trichloroethene		√					√							√		
Vinyl chloride														√		
Xylenes, total							√							√		

* Includes one or more of the following: acenaphthene; anthracene; benz(a)anthracene; benzo(a)pyrene; benzo(b)fluoranthene; benzo(g,h,i)perylene; chrysene; dibenz(a,h)anthracene; fluoranthene; fluorene; indeno(1,2,3-cd)pyrene; naphthalene; phenanthrene; pyrene

Source: ESE.

3.0 EXPOSURE ASSESSMENT

Exposure is defined as the contact of an organism with a chemical or a physical agent. The exposure assessment is the determination or estimation of the magnitude, frequency, duration, and exposure route. An exposure assessment provides a systematic analysis of the potential exposure mechanism by which a receptor may be exposed to chemical or physical agents at or originating from a study area. The objectives of an exposure assessment are as follows:

1. Characterization of exposure setting,
2. Identification of potential exposure pathways, and
3. Quantification of exposure.

3.0.1 Factors influencing the contaminant migration were also considered during exposure pathway assessment.

3.1 CHARACTERIZATION OF EXPOSURE SETTING

This section discusses the study area physical conditions as well as potential human and ecological populations in relation to the nature and extent of contamination described earlier in Section 2.3. This evaluation will provide insight for assessment of the fate and transport of COPCs so that exposure pathways can be identified.

3.1.1 PHYSICAL SETTING

3.1.1.1 Climate

The climate at RSA is mild and temperate with an average annual temperature of 62 degrees Fahrenheit (°F). The summer temperature averages 77°F and the average winter temperature is 47°F. The mean annual snowfall is 3 inches and the mean annual rainfall is 48 inches. Total monthly precipitation is usually highest in March (5.6 inches) and lowest in October (2.7 inches) (see Table 3.1-1). Moderately dry conditions generally prevail throughout autumn.

3.1.1.1.1 The amount of precipitation at RSA was unusually high during the Phase I RFI field investigations, particularly during groundwater sampling activities. Total monthly precipitation from December 1990 through May 1991 averaged 9.7 inches.

3.1.1.1.2 Floods are common from mid-December to mid-April, although extensive flooding is infrequent. The 100-year flood level of the Tennessee River is at an elevation of 572.5 ft-msl. With the exception of Area G (RSA 48), the remaining four areas are outside the 100-year floodplain.

3.1.1.2 Topography

RSA is diverse in topography and vegetation. Elevations range from bottomlands at approximately 560 ft-msl to mountains higher than 1,200 ft. According to the 1988 RSA forest inventory, 15,665 acres are covered in forest. The figure is divided into approximately 6,200 acres of pine, 8,300 acres of hard wood, 60 acres of mixed pine-hardwood, 440 acres of cedar, and 600 acres of tupelo gum swamps. Tennessee Valley Authority (TVA) land and Wheeler Refuge land inside arsenal boundaries is not included in these figures [U.S. Army Missile Command (MICOM) 1992].

3.1.1.2.1 The boundary between the Cumberland Plateau section of the Appalachian Plateau physiographic province and the Highland Rim section of the Interior Low Plateau physiographic province is in central Madison County. RSA is within the Highland Rim province. The topography of RSA is gently rolling and generally slopes from north to south toward the Tennessee River. Topographically high areas are Weeden and Madkin Mountains. Topographically low areas include valleys and flood plains of the Tennessee River and its tributaries.

3.1.1.3 Regional Geology

Overburden residual soils and the Tuscumbia Limestone (bedrock) underlie most of the area. The residual soil overburden, derived from the limestone, typically consists of sandy clay and clay with chert and weathered limestone fragments (Unit 1 and Areas F, G, Q3, Q4, R, and S and T). Generally, chert and limestone fragments are more abundant near the base of the overburden. Alluvial deposits compose the overburden near the Tennessee River (Unit 2 and Areas X1 and Z).

3.1.1.3.1 The Tuscumbia Limestone has an average thickness of 150 ft and consists of gray, massive, fossiliferous limestone with interbedded cherty layers. The limestone is karstic, containing enlarged openings that have developed along joints, fractures, bedding planes, and

faults. Dissolution of limestone has formed caves and cavities, contributing to the formation of sinkholes and depressions in the land surface.

3.1.1.3.2 The Tuscumbia Limestone is successively underlain by the Fort Payne Chert, the Chattanooga Shale, and older geologic units. The Fort Payne Chert is 155 to 185 ft thick and consists of gray, cherty limestone. The Chattanooga Shale is about 10 ft thick and consists of dark gray to black shale.

3.1.1.4 Surface Hydrology

The Tennessee River, which marks the southern boundary of RSA, flows west. Huntsville Spring Branch, McDonald Creek, and Indian Creek are major tributaries that flow generally southward, discharging to the Tennessee River. Approximately 90 percent of the drainage passes through Wheeler Reservoir, located in the central and southwest portion of RSA, before entering the Tennessee River. Wetlands are associated with the creeks and tributaries as well as the Tennessee River (see Section 3.1.1.6).

3.1.1.5 Groundwater Hydrology

Principal hydrogeologic units include the overburden, Tuscumbia Limestone, Fort Payne Chert, and Chattanooga Shale. The Tuscumbia Limestone and the Fort Payne Chert together make up the primary aquifer in the area. In most areas, water in the limestone aquifer occurs under confined conditions with the overburden, in part, as well as the Chattanooga Shale, acting as confining units. The majority of water in the limestone aquifer is stored and transmitted through openings along bedding planes, fractures, and solution-enlarged cavities which are more common in the upper 100 ft of the limestone. In many areas, the top of the limestone is highly weathered and appears hydraulically connected to the overlying basal overburden.

3.1.1.5.1 Water-bearing units in the overburden consist of laterally discontinuous clayey sand lenses that occur as perched water zones in the upper portions of the overburden. The lower part of the overburden, which exhibits either abundant fragments of chert and limestone, or alluvial deposits of sand and gravel near the Tennessee River, is typically the most permeable zone in the overburden. Depending on the location, groundwater in the deeper overburden zones may occur under either unconfined or confined conditions. The deeper overburden

zones appear hydraulically connected with the top of limestone, but the overall relationship between water-bearing units in the overburden and the bedrock is complex and varies with location.

3.1.1.5.2 The groundwater flow rate in each of the zones varies with the hydraulic conductivity and hydraulic gradient.

3.1.1.5.3 Hydraulic conductivities in the sandy clay and chert rubble zone of the basal overburden in the central part of RSA (Unit 1 and Areas F, G, Q3, Q4, R, and S/T) vary with the amounts of sand, clay, chert, and limestone fragments. Hydraulic conductivities in this zone range from 1.1×10^{-2} to 3.5×10^{-6} centimeters per second (cm/sec). Using this range in hydraulic conductivities, a typical hydraulic gradient of 0.007, and an estimated effective porosity of 0.3, the groundwater flow velocity in the basal overburden rubble zone is calculated to range from 0.1 to 265 feet per year (ft/year).

3.1.1.5.4 Hydraulic conductivities in the perched overburden are also dependent on the amounts of sandy clay and clay. Conductivities are typically lower, however, than in the basal overburden, ranging from 5.1×10^{-3} to 1.0×10^{-6} cm/sec. Because the perched zones are typically discrete units, establishing gradients and flow rates is difficult.

3.1.1.5.6 The hydraulic conductivity of the alluvial overburden on the south side of the facility (Unit 2 and Areas X1 and Z) is dependent on the amounts of sand, clay, and gravel present. Hydraulic conductivities for this zone range from 1.3×10^{-2} to 2.8×10^{-5} cm/sec. Using this range in hydraulic conductivities, a typical hydraulic gradient of 0.007, and an estimated effective porosity of 0.3, the groundwater flow velocity in the alluvial overburden is calculated to range from less than 1 to more than 300 ft/year.

3.1.1.5.7 The hydraulic conductivities in the limestone vary greatly and depend on the presence of fractures and solution features. Where the limestone is "tight" and has no such features, the hydraulic conductivity is small (10^{-6} cm/sec). Where fractures and solution features are present, hydraulic conductivities can be very high (10^{-1} cm/sec). Using these ranges in hydraulic conductivities, a typical hydraulic gradient of 0.004, and an estimated

effective porosity of 0.3, the groundwater flow velocity in the bedrock limestone can be expected to range from 0.01 to 1,400 ft/yr.

3.1.1.6 Vegetation and Habitat

Most vegetation on RSA consists of trees and native or seeded grass cover. Wetlands on RSA occur in the form of beaver ponds, sloughs, marshes, tupelo swamps, backwater, and overflow hardwood bottomlands (MICOM, 1992). Approximately 1,250 acres in the southern half of the reservation are under water. This area is identified as a part of the Wheeler Reservoir. An additional 14,560 acres are classified as being in the 100-year flood zone. The mountain area consists of approximately 2,800 acres. Appendix H provides the fauna and flora survey that was conducted by qualified personnel as part of the RSA National Resources Management Plan (NRMP) in June 1992.

3.1.1.6.1 Hay and pastureland cover 5,117 acres. Rights-of-way, test areas, training areas, old field situations (abandoned open areas) in various stages of plant succession, and developed areas provide further diversity. Creeks, sloughs, and ponds are scattered throughout the arsenal. These various habitats have the potential for great diversity in wildlife species (MICOM, 1992). A description of the forest types found at RSA are described in the following paragraphs.

3.1.1.6.2 There are three types of forests at RSA including the upland, floodplain, and bottomland swamp forests (USACE, 1986). The upland forests are primarily dry throughout the year, but the floodplain and bottomland swamp are inundated with water at regular intervals. The floodplain and bottomland swamp forests are differentiated by frequency and depth of inundation. The swamp forest is flooded up to a 2-ft depth for several months at a time. This induces the characteristic buttressing of the bases of swampland trees. The floodplain forest is usually flooded only at times of stormwater surges. Since floodplain topography is not uniform, gradations between these two extremes exist.

3.1.1.6.3 The bottomland hardwood swamp was found to be the least diverse vegetative association at RSA. No ground cover was observed under the 12- to 18-inch-deep black, standing water. Although a good habitat for some aquatic organisms and birds, this association does not offer much useable habitat for terrestrial animals.

3.1.1.6.4 The floodplain forest is drier than the bottomland swamp for longer periods of time during the year. This combination of wet and dry conditions is suitable for certain types of both terrestrial and aquatic vegetation, resulting in a diverse forest community. The floodplain forest was the most diverse type of forest found at RSA (ESE, 1993). This vegetative diversity provides prime habitat for many different terrestrial and avian species. The lack of a continuous cover of water, however, would restrict permanent residence by many of the aquatic species.

3.1.1.6.5 In addition to bottomland swamp and floodplain forests, there are three types of upland forest associations encountered at RSA: planted pine forest, mixed pine and deciduous hardwood forest, and deciduous hardwood forest. The mixed forest generally occurs on higher ground relative to the deciduous forest, which sometimes exists between a floodplain forest and a mixed forest. Planted pines occur also on the higher ground. Each of these have varying degrees of value to wildlife, but many wildlife species make use of all the different forest types. All three associations are scattered throughout RSA.

3.1.1.6.6 Planted pine forests probably have the lowest tree species diversity of the upland forests at RSA. Where the planted pine forest approaches maturity, the canopy coverage approaches 100 percent, which, coupled with the very acidic nature of fallen needles, precludes all but a few other species from surviving. The younger forests provide moderate wildlife habitat, and the more mature pine plantations maintain lower wildlife potential.

3.1.1.6.7 The mixed pine and deciduous hardwood forest has a much higher diversity than the planted pine forest and is somewhat drier than the deciduous hardwood forest. The mixed forest usually has a more open canopy, resulting in more shrub and ground cover. This is excellent wildlife habitat, providing mast-producing trees, fruit-producing vines, and cover for roosting or nesting.

3.1.1.6.8 RSA is located on the north bank of the Tennessee River about 46 miles above Wheeler Dam and 17 miles downstream of Guntersville Dam. RSA encompasses 38,235 acres, of which approximately 10,400 acres are affected by high stages of the Tennessee River and other tributary streams. Huntsville Spring Branch, with a drainage area of 86 square miles (mi^2), flows south through the City of Huntsville and enters the northeast corner

of RSA. From this point, the stream flows southwest to join Indian Creek, a tributary of the Tennessee River. Indian Creek, which enters the western edge of RSA, drains an area of 143 mi^2 and joins the Tennessee River at mile 321. The normal pool of Wheeler Lake at elevation 556 backs into the reservation to form permanent pools having areas of 680 and 575 acres, respectively, at the lower end of these streams. Within the installation boundaries, Indian Creek drains approximately 12,000 acres; McDonald Creek, a short tributary of the Huntsville Spring Branch, drains approximately 6,000 acres; and Huntsville Spring Branch drains approximately 5,000 acres. The southern portion of the reservation drains directly into the Tennessee River through smaller channels. Approximately 2,000 acres, located south of Madkin Mountain, drain into a low area with appropriate drainage outlets constructed as a result of the construction of Fowler Road. Slope exposures are variable with the overall exposure to the south.

3.1.2 POTENTIALLY EXPOSED HUMAN POPULATIONS

3.1.2.1 Demography of Huntsville, Alabama

Based on available information and the ESE site visit, a number of points exist at RSA where human receptors may potentially come into contact with the chemicals released as a result of activities at Areas F, G, R, and S/T. Potential receptors were identified by considering the types of chemicals released, accessibility to the study area, the physical characteristics of the study area that may affect chemical migration, demographics, and current and expected land uses at or near the installation.

3.1.2.1.1 The city of Huntsville, Alabama, is a major metropolitan area that surrounds RSA on the east, north, and portions of the west. The population of the city is 159,789, and the population of all of Madison County (which includes Huntsville) is 233,912. Huntsville encompasses approximately 154 mi^2 of the total 806 mi^2 comprising Madison County. The area surrounding RSA is predominantly urban and densely populated with a large industrial base. Huntsville was originally a textile center in the late 1800s, and large amounts of cotton were grown in the surrounding areas. With the location of RSA in Huntsville in 1941, the city emerged as a leading science and technology center. Since 1950, the population of Huntsville has increased from 15,400 to nearly 160,000. With the exception of a growing number of industrial parks, the areas outside the Huntsville city limits are rural and have a highly agricultural employment base (Advanced Sciences, Inc., 1992).

3.1.2.1.2 As reported by Advanced Sciences (1992), the Huntsville school system has 26 elementary schools, 9 middle schools, and 5 high schools with a student population of more than 25,000. The Madison County school system provides additional education resources for more than 12,000 students. Fifty-five private and parochial schools and kindergartens and more than 14 vocational-technical schools are located in the area, and 4 colleges/universities also serve the area. Because of the proximity of RSA to Huntsville, many of these educational facilities are located near RSA for the convenience of RSA employees and employees at nearby industrial parks.

3.1.2.2 Current RSA Land Use

The majority of the land at RSA (85 percent) is either forest or open land, including land outleased for agriculture. Approximately 10 percent of the land has already been developed, and the remaining 5 percent is either covered by water or brush. The land that has already been developed is used for research and development facilities, administrative buildings, storage facilities, family and troop housing, and community facilities (credit union, hospital, fire station, libraries, etc.) All of the 10,233 acres of outleased agricultural land have been classified by the U.S. Department of Agriculture's (USDA) Soil Conservation Service (SCS) as being prime farmland or lands of state importance (Advanced Sciences, 1992). Prime farmland is defined as the most productive agriculturally, with the fewest impacts and adverse environmental effects. The ownership of RSA is divided as follows:

U.S. Army	
Permitted to MSFC	1,826 acres
Government-owned/contractor-operated (GOCO) to Thiokol Corporation	1, 172 acres
Total U.S. Army owned	31,245 acres
Wheeler National Wildlife Refuge (WNWR)	
Permitted to U.S. Army	4,085 acres
Tennessee Valley Authority (TVA)	
Permitted to U.S. Army	2,905 acres
Total Land on RSA	38,235 acres

3.1.2.2.1 The five study areas discussed in this BRA are closed or abandoned disposal sites. The study areas are all heavily vegetated and are used for containment of waste or contaminated materials.

3.1.2.2.2 Outside the boundaries of RSA, much of the adjacent area is metropolitan with single-and multi-family residential, shopping, and industrialized areas. Because the City of Huntsville borders RSA on three sides, the more rural areas are those that border RSA to the southwest and southeast and across the Tennessee River to the south (Advanced Sciences, 1992). In these areas, as well as a few of the other outlying areas of Madison County, agriculture (e.g., soybeans, cotton, livestock) remains an economic mainstay.

3.1.2.3 <u>Potential Alternate Future Uses</u>

Future uses of RSA are anticipated to be the same as the current use (industrial), because the Army does not have plans to change the overall current mission of RSA. The Army has no plans to develop or use the five study areas under discussion for any purpose. These areas of abandoned or closed landfills will remain unused for the foreseeable future.

3.1.2.3.1 Based on the planning constraints and physical features of RSA, an analysis of the buildable areas was conducted (MICOM, 1992). Buildable areas are those where proposed major facilities with concentrations of personnel can be located. All land within one or more of the areas in the following list was considered unbuildable (MICOM, 1992).

1. Steep slopes (20 percent or more);
2. Flood plains (100-year floodline);
3. Explosive clearance zones;
4. Test areas;
5. Contaminated areas (i.e., toxic substance manufacturing, munitions drop areas, demolition areas, mustard gas or lewisite dumping and/or storage, DDT dumping areas, arsenic ponds);
6. Sanitary landfill areas (past and current);
7. Airfield clear zones; and
8. Potential airfield accident zones.

3.1.2.3.2 These development constraints indicate that only about 30 percent of the more than 38,000 acres at the installation are buildable (MICOM, 1992).

3.1.2.4 <u>RSA and City of Huntsville Groundwater Use</u>

Local municipal water supplies rely on groundwater and surface water resources from wells, springs, and surface waters that surround RSA near the installation boundaries (G&M, 1992). Figure 3.1-1 provides the locations of the withdrawal points of these water resources. Specific data for springs, wells, and surface water resources in and around RSA are provided in Table 3.1-2. Water resource withdrawal points labeled on Figure 3-1.1, and described in the following text, refer to the identification numbers on this table.

3.1.2.4.1 There are a number of private wells located east and west of RSA and to the south, across the Tennessee River (see Figure 3.1-1 and Table 3.1-2). The City of Huntsville is supplied by four wells upgradient of RSA and by two plants drawing from Wheeler Reservoir. As shown in Table 3.1-3, the four wells include the following:

1. Brahan Spring at Huntsville (Location 2 on Table 3.1-2)--Natural spring with well casings set below the surface of spring intake. Services approximately 60,000 customers, in network with other water sources, for potable water supply in Huntsville.
2. Well X-44 (Location 12 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
3. Well P-68 (Location 18 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
4. Well Q-79 (Location 23 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
5. Lincoln/Dallas wells (Location 39 on Table 3.1-2)--Wells used to supply, in network with other sources, potable water for approximately 60,000 customers in the City of Huntsville.

3.1.2.4.2 In addition to the four well or well clusters providing potable water to the City of Huntsville, there are two surface water intakes that also supply potable water. As shown in Table 3.1-2, the surface water intakes for the City of Huntsville include:

1. East Huntsville Water Treatment Plant--Raw water treatment facility for the City of Huntsville. In network with other sources, provides potable water to approximately 60,000 customers in the City of Huntsville. Water intake is directly from the Tennessee River.
2. West Huntsville Treatment Plant--Raw water treatment facility for the City of Huntsville. In network with other sources, provides potable water to approximately 60,000 customers in the City of Huntsville. Water intake is directly from the Tennessee River.

3.1.2.4.3 In the past, RSA was supplied potable water from three wells including the well at the Visitor's Center, one on Martin Road near Test Area 6, and one near Building 6103 at Test Area 3, north of Martin Road, and east of Anderson Road. These three wells, however, are no longer in service, as the source of drinking water on RSA is the Tennessee River, which is treated at two water treatment plants. As shown in Table 3.1-2, the surface water intakes for RSA include:

1. Water Treatment Plant 1 (WTP1)--Raw water treatment facility for RSA. Has separate units for treatment of potable and industrial water. In network with WTP3, provides potable water for approximately 14,000 customers on RSA. Water intake is directly from the Tennessee River.
2. Water Treatment Plant 3 (WTP3)--Raw water treatment facility for RSA. This plant receives partially treated (clarified) water from the industrial water distribution system supplied by WTP1, via a pipeline. In network with WTP1, provides potable water to approximately 14,000 customers on RSA.

3.1.2.4.4 Future use of groundwater resources for potable supplies is not anticipated, since the Tennessee River provides the area with a much more accessible high-quality alternative source.

3.1.2.5 <u>RSA Surface Water Use</u>

The main water source at RSA is the Tennessee River, which forms Wheeler Reservoir, located at the southern boundary of RSA. Two segregated systems provide potable and industrial water to RSA developed areas. Raw water is obtained from Wheeler Reservoir at two locations and is treated for domestic and industrial purposes. One treatment plant

produces industrial and potable water. Another produces only industrial water, while the third plant produces potable water from water obtained from the industrial distribution system.

3.1.2.6 City of Madison Groundwater Use

The Nolan Drake Well (Location 40) produces 4,500,000 gallons per day of pre-treated raw water for the City of Madison municipal water system. The well is set into a spring located upgradient of RSA.

3.1.2.7 Subpopulations of Potential Concern

Human receptors of primary interest at the five areas evaluated in this BRA include current workers (persons working adjacent to or maintaining the grounds near the study areas), potential future workers (persons conducting activities associated with the mission of RSA), and recreational users of the study areas and associated surface waters. Recreational users of the study areas may use the grounds for hunting and the surface waters for fishing (i.e., McDonald Creek adjacent to Area G).

3.1.2.7.1 RSA and residents near RSA are supplied with potable drinking water through surface water intakes in the Tennessee River. In addition, three groundwater production wells at RSA are used for industrial purposes only. Several residential wells located along the western and southern borders of RSA could potentially be affected by contaminant migration from RSA. However, it is not possible to trace groundwater contaminants to a particular disposal area on RSA because many of the landfill units contain similar types of waste. The only potential exposure associated with groundwater at the study area is a future worker exposure, which assumes a worker uses groundwater at the study area for potable purposes.

3.1.2.7.2 Because this BRA addresses only 5 of more than 200 areas of concern at RSA, risks associated with consumption of offsite groundwater cannot be addressed until all potential onsite source areas have been characterized.

3.1.2.7.3 In addition, although some of the landfills have been transected during installation of pipes or utility lines, future construction workers are not considered a subpopulation of potential concern. RSA is designated as an NPL site, and as such, any construction or excavation activities in known or suspected contaminated areas would be performed by OSHA

hazardous-waste-trained personnel. Due to the personal protective equipment required during excavation activities in contaminated areas (i.e., chemical-resistant gloves and suits, respirators), none of the typical exposure pathways (oral, dermal, and inhalation exposure to soils and inhalation of vapors) would be complete. Therefore, future construction workers would not be a subpopulation of potential concern.

3.1.3 POTENTIALLY EXPOSED ECOLOGICAL POPULATIONS

3.1.3.1 Proximity of Ecological Receptors with Respect to Study Areas

The five study areas under discussion in this BRA are heavily vegetated with unrestricted access for wildlife. Each area is either surrounded by a forest stand or has a forest stand directly on top the area (Area S). The numbers and types of wildlife near or on the study areas have not been determined, but signs of wildlife are abundant at or adjacent to all of the areas.

3.1.3.1.1 An appreciation for the types of wildlife present at RSA can be gained through the analysis of habitats and community structures found at the study areas. The following sections describe the habitats and communities that have been documented at RSA. After the communities are defined, subpopulations of potential concern and a final list of representative species that were evaluated further in this BRA are presented.

3.1.3.2 Aquatic and Terrestrial Habitats

Aquatic

Of the five study areas discussed in this BRA, the only potentially exposed aquatic habitats are intermittent streams at Area F, surface water bodies adjacent to Area G, and wetland systems next to Area R. Although the surface water of Area F is intermittent, it was included for risk evaluation. The most significant aquatic communities are in McDonald Creek, which is adjacent to Area G. The surface water in the wetland system adjacent to Area R appeared to be very shallow at the time of the ESE visit (May and June 1994).

Terrestrial - Upland

3.1.3.2.1 Areas F, G, and R were all overgrown with thick brush and high grasses. These vegetative conditions provide wildlife with suitable cover, but do not provide abundant food supplies. The forests surrounding each of the study areas would be suitable habitat for many

types of wildlife, but the type of habitat directly on these study areas suggests that only small mammals may live within the study area boundaries.

3.1.3.2.2 Area S is an upland forest with abundant cover and suitable food supplies for many species of wildlife. Area T is an open field with high grass and is directly adjacent to a heavily industrialized area. Although an upland forest borders Area T on the north and west perimeter, the study area itself is likely to only provide habitat for small mammals in the high grass. The ground was marshy in places, a poor habitat for nesting birds. The grass in Area T appears to be maintained occasionally, which would also indicate that animal communities would not use this study area as prime habitat.

3.1.3.2.3 The upland forested areas in Area S and surrounding Areas F, G, R, and T include deciduous hardwood forest, mixed pine and hardwood forest, and pine plantations. Each of these have varying degrees of value to wildlife, but many wildlife species make use of all the different forest types.

3.1.3.2.4 The deciduous hardwood forest is excellent wildlife habitat due to the diversity of mast-producing tree species. Members of the oak family found here probably produce more edible biomass for a greater number of wildlife species than any other type of vegetation. Acorns, twigs, buds, and bark produced by the oaks are used by a variety of avian and mammal species. The inhabitants of this forest type include deer, turkey, fox, gray squirrel, and numerous woodland birds.

3.1.3.2.5 Signs of deer, raccoon, and wild birds were observed during the ESE site visit (May and June 1994). Signs of wildlife were particularly abundant in the area adjacent to Area G. Animal tracks were observed at the edge of McDonald Creek (in the hardwood forest next to Area G). Wild berries were also observed directly outside the east side of the Area G perimeter fence. Berry patches such as these serve as attractant food sources for many types of wildlife.

3.1.3.2.6 The mixed pine and hardwood forests (Area F, R, and T perimeters) may exhibit a lower rate of mast production than the deciduous hardwood forest, but the open canopy allows for a more pronounced growth of berry-producing vines, shrubs, and trees which are also

widely used by wildlife species. In addition to this excellent trophic support of wildlife, these forests provide much cover needed for nesting and resting.

3.1.3.2.7 The pine plantations are the least diverse of the forest types. Older plantations have a low potential for wildlife support due to a scant understory of blackberry and bracken fern, which provide suboptimal food and cover. Younger plantations, however, are somewhat more diverse and have a more dense understory yielding a moderate value to wildlife.

Wetlands

3.1.3.2.8 Wetlands on RSA occur in the form of beaver ponds, sloughs, marshes, tupelo swamps, backwater, and overflow hardwood bottomlands. Wetlands were once considered wastelands, but are now known to serve many valuable functions. Among these, wetlands filter excess nutrients and reduce sedimentation, which helps to maintain the quality of downgradient water systems. Wetlands help to keep erosion under control and promote groundwater storage. They also provide critical habitat for many species of fish and wildlife. Of the five areas discussed in this BRA, the most significant wetland is the system located adjacent to Area R.

Caves and Water-Filled Void Spaces

3.1.3.2.9 Two caves that are not in Areas F, G, R, or S/T have been identified at RSA: Mathews Cave and Ravine, which is on the northwest border of RSA (USACE, 1992); and Bobcat Cave, which is 3 miles north of the confluence of Indian Creek and Huntsville Spring Branch (USACE, 1986). However, to date, it has not been determined that caves and water-filled void spaces are present under Areas F, G, R, or S/T. Until such a determination is made, groundwater will not be considered to be a direct exposure pathway for ecological receptors. MICOM is currently working with the U.S. Fish and Wildlife Service (USFWS) to determine if cave and water-filled void space habitats exist under Areas F, G, R, and S/T. Results of the MICOM/USFWS efforts should be available by the end of this year. If such habitats are found near the five areas of concern, then risks to aquatic life will be addressed. The results of the cave and water-filled void space habitat assessments will be presented in an addendum to the RSA BRA1.

3.1.3.3 Aquatic and Terrestrial Communities

Aquatic communities in the surface water near Area G include aquatic invertebrates, and all trophic levels of fish, turtles, and amphibians. The wetlands near Area R had only shallow water at the time of the ESE visit (May and June 1994). These wetland surface waters are not expected to sustain fish much larger than *Gambusia* (mosquitofish).

3.1.3.3.1 Small animals expected to be found at the study areas include mice, rabbits, skunks, and raccoons. Several game species that have been introduced to RSA since 1954 include quail, wild turkey, and white-tailed deer. These populations are quite prosperous at RSA. Some of the successful terrestrial communities on RSA that have been documented as being nuisance populations include blackbird flocks, beavers, ground hogs, coyotes, and skunks (MICOM, 1992). In addition to these species, bluebird and wood duck populations are being actively managed so that appropriate nesting habitat is available at RSA. A complete list of species believed to inhabit RSA can be found in the NRMP (MICOM, 1992) in Appendix H.

3.1.3.4 Subpopulations of Potential Concern

As with the identification of human populations, a similar evaluation of release and exposure pathways and study area characteristics was performed to identify the primary ecological populations of concern. Potential receptors were identified by considering the types of chemicals released, the physical characteristics of the study area that may affect chemical migration, current and expected land uses, and habitat type (i.e., wetland, 100-year floodplain, proximity to surface water body).

3.1.3.5 Endangered or Threatened Fauna

A complete list of potential endangered, threatened, or species of special concern is provided in Appendix G. Table 3.1-3 lists endangered or threatened fauna.

3.1.3.6 Nonhuman Representative Receptors

Due to the number and diversity of nonhuman receptors present at the study areas, it is not feasible to evaluate each species present. EPA guidance indicates that biological receptors (ecosystem components expected to reflect adverse effects of pollutant stress) and endpoints (type of actual or potential impact due to contaminant exposure by a receptor) be selected to

represent indicators of any potential adverse effects to all ecosystem components (EPA, 1988a). Initial screening was done to identify receptors present at the study areas. EPA guidance also indicates that consideration be given to rare, threatened, and endangered species, and to species of commercial or sport value (EPA, 1989). Sensitive populations or subpopulations that could be more adversely affected by the contaminants of concern, such as juveniles and species with predicted higher-than-average exposure rates, should also be considered.

3.1.3.6.1 Representative receptor species and sensitive subpopulations for the study areas were selected using the following criteria:

1. Species observed onsite during site visits;
2. Species observed offsite during site visits;
3. Species observed within the study area as noted by other studies and agencies;
4. Species that are an important component of the food chain, thus providing a source of exposure to higher trophic levels or representing an important forage component that may alter ecosystem stability if the population is adversely affected;
5. Rare, threatened, or endangered species known to occur onsite that may occur offsite as well;
6. Potentially affected commercial or sport species;
7. Species of high public interest;
8. Species with known sensitivity to RSA contaminants of concern;
9. Species with behaviors, life history, or physiological aspects which may increase exposure or uptake, or change its toxic effects threshold; and
10. Species with available ecotoxicity data.

3.1.3.6.2 Although the habitat located within the perimeters of the five areas may be considered suitable for several of the endangered or threatened species previously mentioned, little toxicological and/or exposure parameter information exists in the literature for most of these species. The primary fauna receptors at the areas were observed to be small mammals. No listed federal or state threatened or endangered small mammal species are listed. Similarly, habitat requirements suggest that none of the listed fish, amphibians, or reptiles would be found at the study areas. Although one or more of the listed avian species may

infrequently visit the areas, their foraging ranges would be very large relative to the size of the study areas, and thus exposure would be expected to be minimal. However, one of these species will be considered at the screening level to represent a predator, and qualitatively evaluate the potential for bioaccumulation. Many assumptions need to be made during the analysis of risks to ecological receptors. It is prudent to choose receptors for which there are well documented and well researched benchmark exposure parameters and toxicological data. The representative species that were chosen for this ecological risk assessment have been found at RSA and there is sufficient information available to allow for a quantitative evaluation of health risks. The selected representative nonhuman receptors are the mouse, raccoon, common bobwhite quail, green frog, red-shouldered hawk, and the white-tailed deer. In addition, surface water concentrations are compared to water quality criteria to assess potential risks to aquatic life at Areas F, G, and R. A description of each of the six representative indicator species is provided in the following paragraphs.

Mouse

3.1.3.6.3 Mice, particularly of the genus *Peromyscus*, are primary and secondary consumers (USDA, 1986). They are ground-dwelling, nocturnal organisms that potentially contact contaminated soils during burrowing activities, as well as during feeding and grooming. Their primary food sources are seeds, nuts, and berries, but they also feed on invertebrates, fungi, and other items encountered during foraging (USDA, 1986; Caras, 1967). Their home ranges are fairly small ranging from 0.2 to 0.6 hectare (ha) if habitat quality is high, and 2 to 4 ha if habitat quality is low (Caras, 1967). *Peromyscus* specimens are thought to be present throughout the study areas.

Raccoon

3.1.3.6.4 Raccoons (*Procyon lotor*) are omnivorous animals that spend much of their time around wetlands. They eat crayfish, molluscs, terrestrial invertebrates, vegetable matter, berries, nuts, fish, small birds, and mammals (USDA, 1986). Raccoons represent a secondary consumer trophic level. Dens are usually in large hollow trees. Raccoons tracks were observed at the study areas and may be wide-ranging.

Common Bobwhite Quail

3.1.3.6.5 Quail are relatively common at RSA since they were introduced as a game species and because many parts of RSA provide good habitat. The common bobwhite quail (*Colinus virginianus L.*) was chosen to represent birds at RSA because of the available database concerning exposure parameters and chemical toxicities to this species. The mandatory cover types used by bobwhites (Johnsgard, 1973) are grasslands (used for spring and summer nesting and feeding cover), croplands (used for summer feeding and resting cover), and brushy habitat (used year-round for escape and roosting cover). These birds are primary and secondary consumers that feed mostly on seeds but may also occasionally consume beetles and other invertebrates (USDA, 1986; Johnsgard, 1973). Bobwhite are ground nesters and nonmigratory, year-round residents. Population densities are fairly small (1 to 4 ha per quail) with a covey (10 to 15 birds) requiring at least 5 ha (Johnsgard, 1973). The home ranges in Alabama are reported to be small, ranging from 3.2 to 7.2 ha (reviewed in Johnsgard, 1973).

White-Tailed Deer

3.1.3.6.7 White-tailed deer are abundant throughout RSA, with sightings common and evidence, like scat and trails, numerous. White-tail deer are primary consumers that feed mainly on twigs, bark, leaves, grasses, nuts, fruit, roots, and mushrooms (USDA, 1986; Caras, 1967). Many areas of RSA provide excellent browsing habitat for the deer. The extensive range of these animals [2.5 square kilometers (km^2) as reported in Caras, 1967] can potentially expose them to many contaminants throughout the study area via terrestrial and aquatic exposure pathways.

Red-Shouldered Hawk

3.1.3.6.8 Numerous predatory birds are found at RSA. The Red-shouldered hawk (*Buteo lineatus*) has been chosen to represent a predatory wildlife species at the Arsenal. Although not endangered, the red-shouldered hawk is a species of special concern in Alabama. This bird prefers moist mixed woodlands and feeds primarily on snakes, frogs, crayfish, mice, and some small birds (EPA, 1993). The home range size of the closely related red-tailed hawk varies from a few hundred to over 1,500 ha depending on the habitat quality (EPA, 1993a). Population densities of red-tailed hawks are generally lower than 0.005 pairs per ha (EPA, 1993a).

Aquatic Indicator Species

3.1.3.6.9 Because intermittent, as well as perennial water bodies, exist near Areas F, G, and R, these areas were included in the ecological risk characterization. The indicator species for the aquatic exposure and risk assessment were selected based on the likelihood of common organisms typically present in such environments. The indicator species were also chosen based on their capability to withstand periods of desiccation. The species evaluated for potential exposures to surface water and sediments include a primary producer (*Scenedesmus* sp.), a planktonic invertebrate (*Daphnia magna*), and a benthic invertebrate (*Chironimus* sp.).

Green Frog

3.1.3.6.10 The green frog (*Rana clamitans*) is a common amphibian in the eastern United States, and is usually found near shallow fresh water waterbodies which may be permanent or semipermanent. One of the two subspecies of green frog (*Rana clamitans melanota*) is commonly found at RSA (U.S. Army Missile Command, 1992). Juveniles are aquatic, but adult green frogs are terrestrial feeders among shoreline vegetation. Their diet consists primarily of insects, worms, small fish, crayfish and other crustaceans, newts, spiders, small frogs, and molluscs (EPA, 1992a).

3.1.3.6.11 For this assessment, it has been assumed that green frogs are exposed to surface waters only. This assumption is based upon the intermittent nature of the waters which would encourage adults to use them only seasonally. It is unlikely that these waters would be important breeding sites given their temporary nature. If early life stages are present, eggs would be exposed to surface waters since green frogs deposit egg massess so that they float (EPA, 1993a).

3.1.3.6.12 To characterize potential risks to aquatic vertebrates exposed to surface water contamination at the study areas, the detected contaminant concentrations were compared to ambient water quality criteria (AWQC) for the protection of freshwater organisms or ecological benchmarks in the absence of AWQCs.

3.2 IDENTIFICATION OF POTENTIAL EXPOSURE PATHWAYS

To determine the potential health risks associated with onsite COPCs, significant potential exposure pathways need to be identified. The exposure pathway describes a mechanism by which the receptor may be potentially exposed to a chemical or physical agent at or originating from a study area.

3.2.0.1 An exposure pathway is complete if it results in a receptor coming into contact with a COPC. For an exposure pathway to be complete, the following four elements must be present:

1. A source or a release from a source (Section 3.2.1);
2. A probable environmental migration pathway (i.e., leaching, volatilization or partitioning from one medium to another) of a study area-related chemical (Section 3.2.2);
3. An exposure point where receptors may come in contact with a study area-related chemical (i.e., a source area or environmental medium) (Section 3.2.3); and
4. A route by which a study area-related chemical may enter a potential receptor's body (i.e., ingestion, inhalation of dust or vapors, and dermal contact).

3.2.0.2 If any of these four elements is not present, the exposure pathway is considered incomplete and is not expected to contribute to the total exposure from the study area. A screening of each exposure pathway element was conducted for each area of interest to identify significant completed exposure pathways. This screening ensures that the risk characterization focuses only on the completed exposure pathways and eliminates from further consideration those pathways that are incomplete. Each of the four components of the potentially completed exposure pathways is discussed in the following sections.

3.2.1 SOURCES AND RECEIVING MEDIA

In general, potential exposure to different media includes exposure to soil, groundwater, surface water, air, and biota living on contaminated media. Exposure routes to soil include ingestion, inhalation, direct dermal contact, and consumption of biota living on the contaminated soils. Exposure to the surface water could occur by direct dermal contact and consumption of biota living in the contaminated surface water, sediments, and surrounding contaminated soil. Environmental monitoring activities performed at the five areas have

confirmed the presence of COPCs in soil, groundwater, surface water, and sediment. The study area-specific contaminants and the affected media are discussed in the following sections.

3.2.1.1 Area F

Area F (RSA 49) is approximately 5 acres in size and has 3 impoundments or ponds that have been covered, capped, and planted with grass and pine trees. The unit was used in the 1940s for the disposal of arsenic-contaminated wastes from lewisite manufacturing facilities that were located south of Area F. Lewisite is a chemical warfare agent containing arsenic trichloride. General industrial wastes were also disposed of in the ponds. The unit was reportedly closed in 1977. Most of the organic and metallic contaminants at this study area were detected in the waste samples and shallow soils near the ponds. Some of the contaminants were detected in the sediments in the drainage ditches and groundwater.

3.2.1.1.1 The results of the Phase I and II data indicate that the primary source of contamination is soil. Contaminant migration has occurred, as indicated by the presence of COPCs in drainage ditches and groundwater. The principle constituents found in the soils at Area F were metals (arsenic, cadmium, and mercury) and PAHs. The principle contaminants in the groundwater at this study area are arsenic, mercury, and chlorinated hydrocarbons (primarily carbon tetrachloride).

3.2.1.2 Area G

Area G (RSA 48) is a former sanitary and industrial landfill that was active from 1947 through 1950. Construction materials and sanitary wastes from the RSA installation were buried here. According to facility representatives, this unit did not receive hazardous materials. Currently, the study area is covered by a thin layer of soil, grasses, small trees, and brush. Soils are the source of concern due to the potential for contaminant migration to groundwater and/or to surface water in the adjacent creek.

3.2.1.2.1 The COPCs are benzo(a)pyrene (detected in the waste) and trichloroethene, lead, and mercury (detected in the surface water). However, it appears that these compounds are from an upgradient source. Several metals (arsenic, barium, cadmium, chromium, and lead)

were found in the groundwater at Area G. The only VOC detected in the groundwater was acetone.

3.2.1.3 Area R

Area R (RSA 59) is a former landfill that received rubble, construction debris, asphalt, and undocumented industrial waste during the 1970s. Rubble, metal debris, railroad ties, and concrete slabs have been observed at the surface and around the edges of the landfill. Prior to its being used as a rubble fill, the area may have been a borrow area. Examination of 1950s aerial photos indicate the area may have been used as a discharge basin or drainage for activities at the adjacent (to the west) liquid caustic plant (RSA-117).

3.2.1.3.1 Today, the study area is overgrown with grass and briars. During the spring, approximately one-third of the study area is covered with water. This tendency to flood may increase the risk of offsite contaminant migration into surrounding wetlands. The principle constituents found at Area R were PAHs in the soils and chlorinated hydrocarbons in the ground and surface waters. Some of the metals in groundwater exceed MCLs, but the metal concentrations in the soils at this study area were generally low.

3.2.1.4 Areas S and T

Areas S and T (RSA 55/54) contain an 18-acre landfill used in the 1960s and early 1970s for the disposal of household, administrative, industrial wastes, and DDT. Fowler Road was constructed across the middle of the landfill. Area S occupies approximately 5 acres, and Area T occupies the northernmost 13 acres. Area S has been vegetated with pine trees (which have grown to heights of approximately 20 ft). Approximately one-half of Area T is cleared and vegetated with grass; a large portion of this cleared area is used as a staging area for BAMSI, contractors for RSA. The other half of Area T is low-lying and vegetated with grasses, briars, and pines. Although heavy rainfall occasionally results in water pooling in low-lying areas, surface water is not typically present in Areas S and T. This is a terrestrial site with low probability of supporting aquatic populations.

3.2.1.4.1 The apparent source of contamination at Areas S and T is the network of closed disposal trenches and surrounding soils. Areas S and T were included in the DDT Migration Abatement Program conducted from 1977 to 1982. DDT residues buried in the northeast

corner of Area S and in Area T between 1968 and 1973 were excavated and reburied in the DDT Waste Soils Landfill Q1 (RSA-107).

3.2.1.4.2 Organic contaminants, including volatile aromatic compounds and chlorinated benzene compounds, were detected in groundwater samples collected from the wells in this area during previous investigations (PELA, 1988a and PELA, 1989). Metals detected included arsenic, barium, and lead. DDT was not detected in any of the wells. VOCs, metals, and DDT were detected in soil samples collected from the area (PELA, 1988a and PELA, 1989).

3.2.2 FATE AND TRANSPORT IN RELEASE MEDIA

A qualitative environmental fate and transport analysis for the COPCs is provided in this section. The contaminants of concern include seven PAHs, six VOCs, one semivolatile organic compound (SVOC), and five metals. As summarized in Table 3.2-1, all the PAHs were detected in Area F. Only benzo(a)anthracene was found in Area G. In Area R, both benzo(a)anthracene and benzo(a)pyrene were detected. In addition to these two PAHs, dibenzo(a,h)anthracene was also detected in Areas S and T. Table 3.2-2 lists the VOCs, SVOC, and pesticide detected in the five areas. Five VOCs including trichloroethane, tetrachloroethane, vinyl chloride, benzene, and chlorobenzene were present in Areas S and T. VOCs were not detected in Area R. Only trichloroethene was found in Area G. Both carbon tetrachloride and trichloroethene were present in Area F. The only SVOC detected was bis(2-ethylhexyl)phthalate (B2EHP), in Area G. The pesticide 4,4'-DDT, along with its two transformation products, 4-4'-DDD and 4,4'-DDE, was only present in Areas S and T. This chlorinated pesticide was not present in the other samples.

3.2.2.0.1 As shown in Table 3.2-3, the metals present in the five study areas are arsenic, cadmium, mercury, lead, and chromium. In Area F, only arsenic, cadmium, and mercury were detected. In Areas S and T, only lead was detected. Mercury and lead were found in Area G and lead in Area R. Chromium was detected at all five study areas.

3.2.2.0.2 A general review of the basic physicochemical properties of these COPCs will be presented in Section 3.2.2.1, followed by a discussion of the environmental fate processes in

Section 3.2.2.2. The different contaminant migration pathways are described in Section 3.2.2.3.

3.2.2.1 Physical and Chemical Properties

The fate and transport processes of the contaminants detected in the five areas are strongly influenced by their physicochemical properties. These properties relate to the environmental partitioning and mobility of the chemicals. Some of these properties also affect the chemical behavior of the compounds and their susceptibilities to degradation induced by physical and biological agents.

PAHs

3.2.2.1.1 Table 3.2-4 summarizes important physicochemical properties of PAHs detected in the five areas. The properties include molecular weight, solubility in water, vapor pressure, Henry's Law Constant, and organic carbon partition coefficient. These PAHs are typically composed of four to five benzene rings with molecular weights of over 200 grams per mole (g/mole). Their solubilities do not exceed 0.01 milligram per liter (mg/L) of mercury. The vapor pressure values, which range from 3.0×10^{-4} to 5.6×10^{-9} millimeter of mercury (mmHg), suggest that these compounds are not volatile. The Henry's Law Constants are generally lower than 1×10^{-3} atmospheres per cubic meter per mole (atm-m^3/mole) indicating that these compounds have little tendency to volatize from surface waters. The organic carbon partition coefficient (Koc) values are higher than 1.0×10^5 milliliter per gram (mL/g), which indicate that these PAHs would be significantly adsorbed by organic materials in the aquatic and subsurface environments.

VOCs

3.2.2.1.2 The physicochemical properties of the six VOCs found at the five areas are presented in Table 3.2-5. Two of the VOCs are aromatic compounds: benzene and chlorobenzene. The remaining four are chlorinated ethenes and methanes. Compared to PAHs, these VOCs are more soluble in water. The water solubilities range from 1.5 E+2 mg/L for tetrachloroethane to 2.7 E+3 mg/L for vinyl chloride. The VOCs have much higher vapor pressures than PAHs. The vapor pressures vary from 9.0 E+1 mmHg for carbon tetrachloride to 2.66 E+3 mmHg for vinyl chloride. All the VOCs have greater tendencies to escape from the aqueous phase as indicated by the values of Henry's Law

Constants which are greater than 1 x 10^{-3} atm-m^3/mole. With the exception of chlorobenzene, all the remaining VOCs would be expected to be weakly adsorbed by organic carbon present in the aquifer and surface water sediments, based on the low values of Koc. All the Koc values are below 400 mL/g, except chlorobenzene with a value of 930 mL/g. Thus, chlorobenzene would be the most significantly adsorbed of all the six VOCs.

SVOCs

3.2.2.1.3 The physicochemical properties of bis(2-ethylhexyl)phthalate, the only SVOC detected, are shown in Table 3.2-5. Bis(2-ethylhexyl)phthalate is a high molecular-weight phthalate which is not very soluble in water. Its vapor pressure and Henry's Law Constant are low, indicating that this compound would not volatize rapidly from open water bodies. Its high Koc of 1.0 E+4 indicates that this chemical would be significantly adsorbed by organic matter in the aquatic and terrestrial environments.

Pesticides

3.2.2.1.4 The physicochemical properties of 4,4'-DDT and its two transformation products 4,4'-DDD and 4,4'-DDE are summarized in Table 3.2-5. The three compounds contain two chlorinated benzene rings with molecular weights ranging from 318 to 354 g/mole. They are not very soluble in water, as indicated by solubilities of less than 0.1 mg/L. The vapor pressures and Henry's Law Constants are also low, suggesting that loss of these chemicals from the aqueous phase to air would not be significant. The high Koc values of 2.38 E+4 to 5.01 E+4 indicate strong adsorption of these compounds to sediments of streams and aquifer materials which have appreciable quantities of organic matter.

Metals

3.2.2.1.5 Compared to organic contaminants, the specific physicochemical properties of the metals detected in the five study areas vary considerably, depending on the existing environmental conditions and predominant species of each metal. Since information concerning the specific species of each metal present in RSA is limited, and difficult to deduce, important physicochemical properties of the five metals such as solubility, vapor pressure, Henry's Law Constants, and adsorption coefficient could not be provided. However, general and qualitative properties of the metals are described in the following paragraphs.

3.2.2.1.6 The halide salts (chloride, bromide, etc.) of arsenic, cadmium, mercury, lead, and arsenic tend to be more soluble than carbonate and hydroxide compounds of these metals. For instance, $PbCl_2$ is more soluble than $PbCO_3$. The compounds of these five metals dissolve in water by forming ions that do not escape into the atmosphere. In some instances, certain species of the dissolved metal ions may be converted into forms that can leave an aquatic system and escape into the atmosphere. Redox-sensitive metals such as arsenic, mercury, and lead may exist in alkylated species and become gaseous. For example, dimethyl arsine, methyl mercury, and tetramethyl lead can be produced under anaerobic or anoxic environments. For these metal forms, vapor pressures and Henry's Law Constants may be applied. Cadmium is not believed to occur in organically bound species for which volatile behavior is typically discussed.

3.2.2.1.7 The adsorptive properties of the metals are generally described in terms of Kd or the distribution coefficient in contrast with Koc for organic compounds. The Kd is influenced by the predominant species of the metal present in the aqueous phase, nature or properties of the adsorbent, and chemistry of the aqueous solutions. Thus, Kd of a metal can vary by orders of magnitude from one study area to another. In some instances, interpretation of Kd can be complicated by solubility and precipitation equilibria involving the metal of concern.

3.2.2.2 <u>Environmental Fate Processes</u>

Several fate processes occur in environmental media and influence the transport and concentration of the organic and metallic contaminants. One process may be closely linked to another because the phase transfer of a chemical in one medium enhances the transformation process in another different environmental matrix.

3.2.2.2.1 This process can be illustrated when a contaminant adsorbed on a solid matrix moves into the aqueous phase where chemical or biological degradation of the contaminant is favored. Important fate mechanisms that can significantly affect the contaminants in the five study areas include microbial degradation, or biotransformation, volatilization, and photolysis. Other fate processes that can influence metals include chemical speciation, sorption, and precipitation. Over time, all these processes reduce the chemical concentrations in each medium. For conservative risk assessment applications (to be most protective of human health and the environment), the environmental fate processes occurring at the five study areas

were not factored into calculating chemical exposures at the site. Thus, the concentrations represent a conservative estimate of exposure as the concentrations were not reduced to account for biodegradation; biotransformation; volatilization; photolysis; or metal speciation, sorption, and precipitation. This approach ensures that risks are most likely overestimated rather than underestimated.

3.2.2.2.1 Microbial Degradation/Transformation

Microorganisms such as bacteria and fungi are known to be present in the surface and subsurface environments. They participate in the transformation and biogeochemical cycling of organic compounds and metals. The degradation can take place under either aerobic or anaerobic conditions.

PAHs

3.2.2.2.1.1 Biodegradation of a PAH with three or more aromatic rings such as those detected in media at the five study areas is reported to be slower (Perwak et al., 1982). The rate and extent of degradation is variable. The mechanism appears to be the removal of one cyclic unit at a time. Some studies showed that PAHs can be microbially metabolized and transformed into other compounds. Under aerobic conditions, benzo(a)anthracene was transformed by *Cunninghanella elegans* to 3,4-, 8,9-, and 10,11- dihydrols (Kobayashi and Rittman, 1982). A bacterial strain of *Beijerincka* was reported to metabolize benzo(a)anthracene to form a polycyclic aromatic acid (Mahaffey, et al., 1988). In addition, both *Beijerincka* and *Cunninghanella elegans* have been demonstrated to biotransform benzo(a)pyrene (Gibson, 1976; Kobashi and Rittman, 1982). Negligible degradation was observed by McKenna and Heath (1976) for dibenz(a,h)anthracene in shake flask experiments.

VOCs

3.2.2.2.1.2 Microorganisms can also metabolize chlorinated methane, ethenes, benzene, and chlorobenzene. Parsons and Lage (1985) reported that microbiota degraded tetrachloroethane and trichloroethane via a reductive dehalogenation pathway under conditions of neutral to acidic pH, and an absence of oxygen and light in a simulated groundwater environment. Results of laboratory studies of Vogel and McCarty (1985) confirmed that tetrachloroethane can undergo reductive dehalogenation under anaerobic conditions. Both tetrachloroethane and trichloroethane are converted to dichloroethenes and then to vinyl chloride, which can be

eventually mineralized to carbon dioxide. Thus, it is possible that the vinyl chloride detected could have resulted from the tetrachloroethane or trichloroethane biodegradation. Carbon tetrachloride can also undergo reductive dechlorination and form chloroform as one of the degradation products (Smith and Dragun, 1984; Galli and McCarty, 1989).

3.2.2.2.1.3 Benzene and chlorobenzene are also known to be metabolized by microbes. A mutant of *Pseudomonas putida* can transform benzene to catechol (Dagley, 1972). Bacterial dioxygenases can in turn cleave catechol to yield acid and aldehyde (Chapman, 1972). For chlorobenzene, unacclimated aerobic river die-away tests indicate that aqueous biodegradation is possible.

<u>SVOCs</u>

3.2.2.2.1.4 Bis(2-ethylhexyl)phthalate can be biotransformed in the environment. Johnson and Lulves (1975) reported on the aerobic biodegradation of bis(2-ethylhexyl)phthalate in freshwater hydrosoil. Biodegradation of this ester under aerobic conditions in river or lake water and water/sediment systems is also expected to occur. In water/sediment mixtures under anaerobic conditions, biodegradation may be slow or not occur at all.

<u>Pesticides</u>

3.2.2.2.1.5 4,4'-DDT can be biotransformed to different products by different microorganisms. Fries (1972) reported that *Aerobacter aerogenes* degraded 4,4'-DDT under aerobic conditions to yield several products that included 4,4'-DDD and 4,4'-DDE. The white rot fungus *Phanerchaete chrysosporium* can also degrade 4,4'-DDT to produce several metabolites such as 4,4'-DDD and dicofol (Bumpus and Aust, 1987). In soils, 4,4'-DDT is slowly converted to 4,4'-DDE under aerobic conditions, and rapidly converted to 4,4'-DDD under anaerobic conditions (Kearney and Kaufman, 1976). Other degradation products may be also produced under aerobic and anaerobic conditions by various microbes (Kobayashi and Rittman, 1982).

3.2.2.2.1.6 Both 4,4'-DDD and 4,4'-DDE may also undergo biotransformation. Several compounds were reported by Fries (1972) when 4,4'-DDD is degraded in aerobic and anaerobic conditions. Indigenous microbes in lake sediments were able to degrade 4,4'-DDD to derivatives of ethane and ethanol under reducing conditions (Leland <u>et al</u>., 1973). 4,4'-

DDE was also reported to degrade further to an acid by loss of two more molecules of HCl (Pesticide Dictionary, 1976).

Metals

3.2.2.2.1.7 Bacteria, yeast, and fungi can catalyze the modification of metals in the environment. These transformations from one form of a metal or element to another affect the concentration and distributions of the metallic pollutants. The microbial transformation processes generally observed include redox, alkylation, dealklylation, and sulfide precipitation. Of the metals detected in the five study areas, arsenic and mercury are affected by the microbially mediated redox process. A yeast of the genus *Cryptococcus*, along with *Escheria coli* and streams of *Pseudomonas sp.*, was found to be capable of converting mercuric chloride to elemental mercury (Alexander, 1974; Brunker and Bott, 1974). Arsenic, in the form of As (III) can be oxidized to As (V) in soil (Quastel and Scholefield, 1953).

3.2.2.2.1.8 Alkylation or methylation of mercury, arsenic, lead, and cadmium can be catalyzed by microorganisms as part of a detoxification mechanism. Several studies have shown that mercury can be methylated by both anaerobic and aerobic bacterial species (Bechert et al., 1974; D'Itri, 1972; Hamdy and Noyes, 1975). Arsenic can be methylated by bacteria and fungi to yield dimethyl and trimethylarsines (McBride and Wolfe, 1971; Saxena and Howard, 1977; Thayer and Brinckman, 1982). Formation of tetramethyl lead by microorganisms in anaerobic sediments was demonstrated in incubation experiments of Wong et al. (1975). Methylation of cadmium to organic species by *Pseudamonas sp.* has been also reported. The reverse process, dealkylation is also possible and has been studied in systems involving arsenic and mercury.

3.2.2.2.1.9 Sulfide precipitation mediated by sulfate-reducing bacteria generally affects lead and silver. H_2S resulting from reduction of sulfate and cleavage of some organic molecules due to microbial action reacts with lead to form sulfide compounds.

3.2.2.2.2 Volatilization

Volatilization is a process of mass transfer of the contaminants from the aqueous phase to the air or atmosphere. As described in Section 3.2.2.1, chemicals with Henry's Law Constant of greater than 1×10^{-3} atm-m^3/mole have a greater tendency to volatilize.

PAHs

3.2.2.2.2.1 Due to the low Henry's Law Constants and vapor pressures of the seven detected PAHs, these are not expected to escape readily from solution to the air. Thus, volatilization is considered to be slow and less important for this group of compounds.

VOCs

3.2.2.2.2.2 Volatilization is an important fate process for the six detected VOCs. The rate of mass transfer or loss from an open water body would be affected by surface and bulk agitation of the liquid medium, wind velocity, and temperature. The presence of suspended matter and particulates in the water, to which the chemicals can sorb, will influence volatilization. Chlorobenzene and tetrachloroethane with Koc values of 930 and 364 mL/g will tend to be strongly adsorbed compared to the other VOCs. Consequently, suspended organic materials can potentially retard the volatilization of these two VOCs.

SVOCs and Pesticides

3.2.2.2.2.3 Bis(2-ethylhexyl)phthalate and 4,4'-DDT, along with its transformation products, 4,4'-DDD and 4,4'-DDE, would not be expected to volatilize readily owing to the low Henry's Law Constants. In an aquatic environment, these compounds will tend to be preferentially adsorbed, thus remaining in only small quantities in the aqueous phase.

Metals

3.2.2.2.2.4 The inorganic forms of arsenic, lead, and cadmium present in the aqueous phase are not expected to volatilize. Only Hg^0 is known to be volatile. However, the microbial methylation process described previously can transform arsenic, lead, and cadmium into volatile species under certain environmental conditions. For instance, arsenic can be converted to di- and trimethylarsines that escape from natural waters. The rate of volatilization may be retarded by other processes such as adsorption and chemical degradation during the upward diffusion of the volatile species from the sediments and underlying water column to the air.

3.2.2.2.3 Photolysis

Chemicals present on the soil surface and natural waters can undergo chemical degradation by absorbing solar energy. The energy can excite the molecule of the contaminants leading to bond cleavage and formation of photolysis products.

PAHs

3.2.2.2.3.1 The PAHs detected in the five study areas have four to five aromatic rings that are susceptible to photolytic degradation. One of the most widely studied compounds is benzo(a)pyrene which has been reported to undergo photolysis by light and yield mixture of quinones. The fate process is believed to be mediated by single oxygen molecules that are formed through energy transfer from the electronically excited aromatic molecule in its triplet state (Andelman and Suess, 1970; NAS, 1972, Neff, 1979). The photolysis rate can be affected by the presence of natural organic and inorganic substances in solution. The photolytic degradation of benzo(a)pyrene was found to be inhibited in natural waters with humic acids (Smith et al., 1988). Inhibition of photolysis was also observed when benzo(a)pyrene was adsorbed onto kaolinite clay.

3.2.2.2.3.2 Other PAHs would be expected to exhibit photolytic behavior comparable to that of benzo(a)pyrene. Chrysene was reported to be potentially susceptible to photolysis and yield quinones based on studies involving structurally related compounds such as benzo(a)pyrene (EPA, 1985).

VOCs

3.2.2.2.3.3 Based on the review of Callahan et al. (1979), photolysis is considered too slow to be a significant factor in influencing the fate of VOCs. No data were found to suggest that tetrachloroethene and trichloroethene undergo photodegradation. Although direct oxidation of benzene in environmental waters is unlikely, cloud chamber data indicate that it may be photooxidized rapidly in the atmosphere (EPA, 1985).

SVOCs

3.2.2.2.3.4 No information was found to assess directly the photolytic stability of bis(2-ethylhexyl)phthalate. According to the study conducted by Wolfe et al. (1980), on the

aqueous photolysis for dimethylphthalate, it is possible that bis(2-ethylhexyl)phthalate can be photolyzed with half-lives ranging from months to years.

Pesticides

3.2.2.2.3.5 Photolysis may not be a significant fate process for 4,4'-DDT and 4,4'-DDD. No data were found to directly evaluate the photolytic stability of 4,4'-DDD. Degradation of DDT was not observed in distilled water solutions under sunlight for 7 days (Callahan et al., 1979). For 4,4'-DDE, photolysis may be a significant process based on the measured rate constants for 4,4'-DDE eradicated at more than 290 nanometers (nm) with artificial sunlight in water solution (Zepp and Cline, 1977; Callahan, 1979).

Metals

3.2.2.2.3.6 No information was found during a literature survey to indicate that photolysis is an important fate process affecting the concentration and distribution of the six detected metals in the environment.

3.2.2.3 Contaminant Migration Pathways

The organic compounds and metals present in the waste and soils at RSA have the potential to migrate to other environmental media and eventually to move toward downgradient locations. The six possible migration pathways are as follows:

1. Soil-to-groundwater,
2. Soil-to-surface water,
3. Soil-to-air,
4. Surface water-to-air,
5. Groundwater-to-air, and
6. Groundwater-to-surface water.

3.2.2.0.1 Some of these contaminant migration pathways may be important in certain areas depending on the nature of contamination. The rate and extent of migration of the contaminants will be strongly influenced by their physicochemical properties and existing onsite environmental conditions. In this section, the different migration pathways are described followed by a qualitative analysis of the expected contaminant migration at each area.

3.2.2.3.1 Soil-to-Groundwater Pathway

During and after a rainfall event, water moves on the soil surface as surface runoff and infiltrates the soil. As leachate percolates the subsurface environment, the water carries with it dissolved organic chemicals, soluble metallic species, and compounds adsorbed to suspended matter in the aqueous phase. Those chemicals strongly sorbed to suspended matter, such as those with high adsorption coefficients (Koc or Kd), would be expected to be retained by the upper few inches or feet of the surficial soil. The dissolved forms can continue migrating downward. However, only a certain fraction of the total dissolved compounds is expected to reach the groundwater because adsorption, and other dissipation mechanisms may still continue as the solution passes through the vadose zone and finally reaches the aquifer. The extent of adsorption will be affected by the chemical structure, charge of metallic species, presence of competing species, and hydrogeological factors such as porosity, soil texture, depth to water table, and presence of layers with low hydraulic conductivity. In some instances, solution chemistry factors such as pH, redox potential, and ionic strength may exert significant influence on leaching.

3.2.2.3.2 Soil-to-Surface Water Pathway

Part of the rain water that does not percolate can move over the surficial soil to low-lying areas as surface runoff. This runoff can transport the dissolved chemicals and metallic species from the source areas to drainage systems and nearby surface water bodies such as streams and creeks. In addition, metals and organics sorbed onto particulates or coated onto moving soil particles can be transported with the surface runoff. As the surface water moves or stagnates in certain locations, some speciation and transformation of metals can occur depending on the pH, presence of oxidizing and reducing agents, levels of organic matter and inorganic ligands picked up during the transport. Organic compounds in standing water runoff can potentially undergo photolysis and volatilization.

3.2.2.3.3 Soil-to-Air Pathway

Organic contaminants and metals in the ground surface and near the surficial soils can be released into the atmosphere either by volatilization, fugitive dust, or particulate emission. Compounds such as VOCs would be expected to be lost by volatilization. For compounds with low vapor pressure and high Koc, volatilization is considered to be of lesser importance. However, these compounds, along with metallic species sorbed onto fine soil particulates,

may be released to the air via dust or particulate emission due to wind-driven currents or other mechanical disturbances of the surface soil (i.e., through human activities). This mechanism of contaminant transport, however, may not become a significant pathway if the contaminated surface soil is covered by grass and other barriers.

3.2.2.3.4 Surface Water-to-Air Pathway

Contaminants that eventually reach surface water bodes such as streams and creeks have the potential to migrate to the atmosphere. These contaminants are usually VOCs that have relatively high vapor pressures and Henry's Law Constants (more than 1×10^{-3} atm-m^3/mole). The volatilization loss rate would be influenced by surface and bulk agitation, wind current, temperature, and presence of materials in the aqueous phase that inhibit the actual mass transfer to the atmosphere. Volatile contaminants, such as VOCs, reaching the atmosphere can undergo fairly rapid photooxidation with hydroxyl radicals.

3.2.2.3.5 Groundwater-to-Air Pathway

Volatile contaminants in the groundwater can diffuse through the soil pore spaces and finally reach the soil surface and surrounding air. This subsurface volatilization pathway is affected by depth to the water table, moisture content of the soil column, and soil texture of the vadose zone. When the moisture content is low, the water table is shallow, and the soil is predominantly sandy, volatilization from the groundwater is highly favored to occur. When the air porosity is low, water table is deep, and moist silt and clay abound in the unsaturated soil column, the groundwater-to-air pathway would not be significant.

3.2.2.3.6 Groundwater to Surface Water

The hydrogeology in the vicinity of RSA may permit the migration of chemicals from groundwater to nearby surface water bodies. Areas having nearby surface bodies include:

- Area F Surface water drainage ditch along the east, north, and west boundary;
- Area G McDonald Creek and a drainage ditch adjacent to the unit; and
- Area R Wetlands surround the north, south, and east sides of the unit.

Hydrogeologic investigations suggest that groundwater in the overburden is controlled primarily by topography and surface water drainage (G&M, 1991). Following these observations, groundwater may tend to flow toward streams, rivers, and low-lying areas on

and near the site. The concentrations of any chemical discharging from groundwater to surface water is expected to be reduced significantly through dilution, adsorption, settlement, volatilization, photolysis, and/or biodegradation (EPA, 1979). The site illustrates the reduction of groundwater chemical concentrations upon reaching nearby surface waters as indicated by the analytical data. For example, at Area F, many inorganics and organics were detected in groundwater; however, they were below detection in surface water. For Areas G and R, both inorganics and organics were detected in groundwater, while surface water concentrations were detected at lower concentrations or below detection. At Area G, concentrations of trichloroethene, an organic, were detected in the upstream surface water sample, but were below detection in groundwater, indicating an upgradient source(s) or organic chemicals.

3.2.2.4 Study-Area-Specific Discussions of Contaminant Migration Pathways

Area F

An analysis of the nature and extent of contamination and environmental conditions at Area F suggest soil-to-groundwater, soil-to-air, and soil-to-surface water are the three important pathways that affect the contaminant transport and distribution. The soil-to-groundwater pathway applies to arsenic and mercury. These two metals were detected in waste samples and have leached to the aquifer. Both metals were found in the groundwater. Despite the presence of the PAHs in the waste samples and shallow soils, these compounds were not detected in the groundwater probably due to the strong adsorption in the vadose zone. Some of the PAHs such as benzo(a)anthracene and benzo(a)pyrene, along with arsenic, were detected in the sediments of the drainage ditch, suggesting soil-to-surface water migration in which the two PAHs and arsenic eventually became trapped in the surface water sediments.

3.2.2.4.1 PAHs and arsenic were also found in shallow soils. They can be released to the surrounding air during potential mechanical disturbance of the soil via soil-to-air contaminant migration pathway. PAHs and arsenic sorbed to soil particles are more likely to become airborne due to dust or particulate emission rather than through volatilization.

Area G

3.2.2.4.2 Based on the evaluation of the environmental monitoring data from Area G, soil-to-surface water and surface water-to-air are the significant contaminant migration pathways.

The soil-to-surface water pathway is applicable to benzo(a)pyrene. This PAH may be transported to nearby land surface and eventually reach McDonald Creek and the northern drainage creek. The mode of transport is likely to be via surface water runoff carrying benzo(a)pyrene adsorbed to suspended matter or soil particles. The surface runoff will tend to move toward McDonald Creek and the northern drainage creek due to the topography of the area. The other pathway, surface water-to-air, is important for trichloroethylene, which was detected at several sampling points along McDonald Creek. Volatilization of trichloroethene is expected because this compound has a high vapor pressure and a high Henry's Law coefficient as discussed in Section 3.2.2.1.

Area R

3.2.2.4.3 An assessment of the environmental monitoring data from Area R indicates that the soil-to-air and soil-to-surface water pathways are important in the contaminant migration analysis. Benzo(a)anthracene and benzo(a)pyrene can be transported to the surrounding air by mechanical disturbance of the shallow soil such as excavation. This soil-to-air pathway can be enhanced when air currents are strong and favor emission of soil particles to which the two PAHs are adsorbed. Volatilization of the two PAHs is not likely because the two compounds have low vapor pressures and Henry's Law constants. The other pathway which affects benzo(a)anthracene and benzo(a)pyrene is the soil-to-surface water pathway. The transport mechanism is likely to be via surface runoff carrying suspended materials and particulates with sorbed PAHs. These sorbed PAHs would finally reside in the beds of nearby drainage ditches or creeks in the eastern and southeastern portion of the study area. Environmental monitoring data confirm the presence of the two PAHs in surface water sediments. The surface water-to-air pathway is not considered significant for bis(2-ethylhexyl)phthalate. This phthalate has a low Henry's Law Constant and is not expected to volatilize from the drainage ditch surface water.

Areas S and T

3.2.2.4.4 Based on the environmental monitoring data reviewed for Areas S and T, the important contaminant migration pathways are soil-to-groundwater and soil-to-air. The soil-to-groundwater pathway would involve chlorobenzene which is associated with the production and disposal of DDT. The other contaminants, such as the three PAHs, lead, and 4,4'-DDT and its two transformation products, may not be readily leached to the aquifer because these

chemicals can be strongly adsorbed in the vadose zone. Chlorobenzene is expected to have a relatively faster percolation rate due to its higher water solubility and lower Koc compared to the other contaminants in the waste samples and soils. Chlorobenzene has been already detected in the groundwater and is considered the principal VOC contaminant. The other four VOCs also found in the groundwater may have come from the waste materials present in the landfill. The other pathway, soil-to-air, becomes significant when mechanical disturbance such as excavation leads to particulate emission. The three PAHs, lead, and 4,4'-DDT and its transformation products can be carried to the air via airborne soil or waste particles to which these contaminants are adsorbed. Volatilization and fugitive dust emission of these contaminants are not likely because the study area is currently covered with vegetation.

3.2.2.4.5 The concentrations of both the organic and metallic contaminants that may be transported to the different environmental media through the different contaminant migration pathways would be expected to decrease if biodegradation and photolysis can occur in any of the five study areas. The rate and extent of these dissipation mechanisms, however, are difficult to evaluate due to limited information. For instance, data on groundwater chemistry such as pH, redox potential, ionic strength, and presence of inhibitory substances are needed to determine if microorganisms can exist and metabolize the contaminants. Consequently, an assessment of the potential impact of the degradation processes is difficult to perform, even in a qualitative fashion.

3.2.3 HUMAN EXPOSURE POINTS AND EXPOSURE ROUTES

An exposure point and exposure route are the third and fourth elements, respectively, of an exposure pathway. Through the identification of chemical sources and receiving media (Section 3.2.1) and the analysis of the movement of these chemicals from the source (Section 3.2.2), exposure points (a location where receptors can come into contact with study area-related compounds) and exposure routes (the route by which a contaminant enters the body) can be identified as defined in Section 3.2.

3.2.3.0.1 In general, potential exposure to different media includes exposure to soil, groundwater, surface water, air, and biota contacting the contaminated media. Exposure routes to soil include ingestion, inhalation, direct dermal contact, and consumption of biota living on the contaminated soils. Exposure routes to groundwater include ingestion, dermal

contact, and inhalation of vapors volatized from groundwater (i.e., while showering). Exposure to the surface water and sediment could occur by ingestion, direct dermal contact, and consumption of biota living in the contaminated surface water, sediments, and surrounding contaminated soil.

3.2.3.0.2 Although there are currently no human receptors exposed to groundwater under the study areas, a full assessment of the potential risks to future workers was conducted for exposure to groundwater at each study area. Oral, dermal, and inhalation pathways of exposure were evaluated in the BRA. Based on the study area-specific information, other relevant media, exposure routes, and potential receptors were selected for the potential exposure at each study area as discussed in the following sections.

3.2.3.1 Area F

The most likely point of contact with contaminants at the study area is surface soil, either directly onsite, or in the drainage ditch that runs along the southern border of the study area. The drainage ditch is dry except during periods of heavy precipitation; thus, samples from the ditch are evaluated as soils. Potential exposure routes for surficial soil contamination at this study area include direct dermal contact, incidental ingestion, and inhalation of soil/dust particles. There are no perennial surface water bodies at this study area; therefore, no surface water or sediment exposure points or routes need to be addressed for Area F.

3.2.3.1.1 Ingestion, dermal contact, and inhalation exposure to soils and groundwater are potentially complete future pathways and are evaluated in this BRA.

3.2.3.2 Area G

Area G is the only area of the five at which a permanent recreational surface water body occurs (near the perimeter of the closed landfill). Persons frequenting the vicinity of McDonald Creek may be exposed to surface water and sediments via dermal contact or incidental ingestion. Ingestion of fish caught in the creek is another potential exposure route.

3.2.3.2.1 Potential pathways of future exposure at this study area include exposure to surface water and sediments, as stated previously, as well as incidental ingestion, dermal contact, and inhalation of contaminants in groundwater and soil.

3.2.3.3 Area R

No current exposures to site contaminants are evident. However, human receptors may be exposed to soil and groundwater contaminants at this area via ingestion, dermal contact, and inhalation.

3.2.3.4 Areas S and T

Current exposure to site soils may be possible via ingestion, direct contact, and inhalation. In addition, human receptors may be exposed to soil and groundwater contaminants at these areas in the future via ingestion, dermal contact, and inhalation.

3.2.4 POTENTIAL HUMAN RECEPTORS AND SCENARIOS

3.2.4.1 Current Worker

As described in Section 3.1.2.3, the current industrial use of the installation will continue into the future as the Army does not have plans to change the current mission of RSA. Thus, the current worker exposure scenario is the primary expected exposure scenario, which includes maintenance activities. While a trespasser may traverse the study area on an occasional basis, the worker scenario is a more frequent exposure to assess human health risks.

3.2.4.1.1 Because all of the study areas are closed or abandoned landfills, there are no buildings located directly on these areas, as landfills would not be foundationally sound to support large structures (i.e., homes, warehouses, offices). A small sewage lift station is located adjacent to the southwest corner of Area R. However, as this structure is not situated on the rubble area and is occupied on an infrequent basis, no current worker exposure to study area COPCs is anticipated. In addition, the area beneath the power lines adjacent to Area G is maintained regularly; however, since the power lines are not located on the old fill area, no exposure to COPCs is anticipated for a worker cutting grass adjacent to the study area.

3.2.4.1.2 The only workers who may potentially be exposed to the COPCs associated with the five study areas are those who work in the office building adjacent to Area F and those who maintain the grounds at Area T. Although Areas F and T are covered, capped, and vegetated, surficial soil samples indicated the presence of residual contamination. Therefore, the current worker scenario is considered valid for these two areas and current exposure to

soil is evaluated. The exposure assumptions for the current workers at each of these areas are listed in Appendix C. Because RSA obtains potable water from the Tennessee River and treats the water at three treatment plants, groundwater beneath RSA is not currently used.

3.2.4.2 Future Worker

The possibility for future industrial use of Areas F, G, R, and S/T is unlikely because the study areas are closed landfills or waste pits regulated under CERCLA. However, to provide a perspective on the level of contamination at each area, future industrial use is evaluated in this BRA to illustrate the level of risk that would be associated with regular exposure to the groundwater and soils at these study areas. A future worker may contact soils through incidental ingestion, inhalation of fugitive dusts, or dermal contact. All three of these pathways are considered complete for the future worker and are evaluated in this BRA. Although unlikely, groundwater beneath RSA could be developed as a potable source; thus, ingestion, direct contact, and inhalation of groundwater chemicals is evaluated for a future worker. Since McDonald Creek, a perennial water body, is located adjacent to Area G, the potential for future workers to contact surface water and sediment during breaks may exist. Therefore, incidental oral and dermal exposure to surface water and sediment is evaluated for future workers.

3.2.4.3 Recreational User

The study areas addressed in this BRA are open or forested and the potential exists for current and future recreational use. Access to RSA as a whole is restricted; however, Area F is fenced but does not have a gate; Areas G, S, and T do not have fences that significantly inhibit access to the study areas; and Area R does not have a perimeter fence. Hunting and fishing are allowed on RSA property and it is possible that these recreational users of RSA will trespass onto the study areas occasionally. In addition, a cemetery is located adjacent to the southeast corner of Area S. As the cemetery is not situated on the study area and may be visited by family members on an infrequent basis, no current recreational exposure to COPCs is anticipated.

3.2.4.3.1 A future recreational user would be exposed to the same media via the same pathways as the future worker. Although the recreational user scenario is valid, it evaluates the exposure for an individual who is only exposed to study area soils for a small number of

days each year. The future worker scenario evaluates the risks associated with exposure to study area soils for 250 days per year. Therefore, future worker exposure to soils represents a conservative estimate of recreational soil exposure as well. The potential risks posed to the trespasser will be discussed in the risk characterization as they compare to the potential risks posed to future workers.

3.2.4.3.2 In addition to soils, recreational exposure to surface water and sediment may occur at McDonald Creek adjacent to Area G. Persons fishing or wading in the creek may be exposed to surface water and sediments via dermal contact or incidental ingestion. Ingestion of fish caught in the creek is another potential exposure route and is evaluated in this BRA. Surface water concentrations in McDonald Creek were used to model the concentrations of COPCs in fish tissue. The intake of fish was then estimated, and the associated risks were determined. Since current and future recreational exposure patterns are expected to be equivalent, only current recreational exposure is evaluated.

3.2.4.4 Future Construction Worker

The probability that construction activities will take place directly onsite at the five areas is unlikely. The areas are closed waste sites or landfills and the land is considered unsuitable for building. Uneven settling of the ground overlying the landfills would preclude any large-scale construction activities. The only activities that may intermittently occur would be the maintenance or installation of underground wiring or plumbing which may disrupt the surface. These activities are expected to occur infrequently. Additionally, the exposure to the construction worker who may be at a site for several days is expected to be much less than the exposure received by a future worker who would be exposed 250 days per year for 25 years. For these reasons, it was concluded that the construction worker scenario is superfluous and it is not quantitatively evaluated in this BRA.

3.2.4.5 Current Resident

There are currently no residents located either on or directly adjacent to any of the five study areas assessed in this BRA. Local groundwater is not used as a potable water source on RSA, and risks to offsite residents who may be exposed to regional surface and/or groundwater as a drinking water source cannot be evaluated in this BRA, as this BRA addresses only five of more than 200 potential sources of groundwater contamination at RSA.

3.2.4.6 Future Resident

The potential for a future resident living directly on one of the study areas is unlikely. Future land use will be in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA in which there will be development in accordance with RSA remaining as an Army-based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo Base Realignment and Closure (BRAC), which further supports that the current mission will continue into the future. Thus, this information supports the conclusion that a future residential scenario is unrealistic. In addition, landfill sites would have limited future uses as they would not be foundationally sound to support residential development.

3.2.4.6.1 If the study areas were to be scheduled for such land use in the future, the risks associated with such activity would need to be evaluated at that time. It is not useful to evaluate the risks for a future resident at this time because chemical concentrations in various media are expected to change considerably over time.

3.2.5 SUMMARY OF COMPLETED HUMAN EXPOSURE PATHWAYS TO BE QUANTIFIED IN BRA

A summary of the human exposure pathways quantified in this BRA are presented in Table 3.2-6 and are discussed in the following sections.

3.2.5.1 Groundwater Ingestion Pathway

Domestic/industrial consumption of groundwater is a pathway of concern when humans use wells that tap into groundwater flowing beneath a potentially contaminated site. Exposure will occur as a result of direct ingestion of water and beverages and foods made with water, as well as incidental ingestion during various activities, such as showering or washing cars. This pathway is not complete for current residents or workers because there are currently no drinking water wells near Areas F, G, R, or S/T. Although there are private wells located in Huntsville, none of these wells are close enough to the study areas that groundwater contamination could be attributed to the subject areas.

3.2.5.1.1 The groundwater consumption pathway is considered complete only for future workers and is evaluated for this receptor at all five of the study areas. The scenario of having a potable water well on one of the disposal sites is unlikely, but the scenario is

included in the BRA so that the potential risks associated with groundwater contamination at the study areas may be quantitatively evaluated. As per EPA region-wide (1989b) and Region IV (1992c) guidance, the quantitative evaluation of groundwater exposure is based only on unfiltered samples. Filtered samples may be discussed qualitatively, where appropriate.

3.2.5.2 Groundwater Dermal Contact Pathway

Domestic direct contact with groundwater may occur during various activities, including showering, cooking, and washing cars, dishes, and laundry. Dermal exposure to groundwater in an industrial setting may result from certain work-related processes, as well as showering. As with the groundwater ingestion pathway, this pathway is not complete for current residents or workers because there are currently no wells near Areas F, G, R, or S/T.

3.2.5.2.1 The groundwater dermal contact pathway is considered complete only for future workers and is evaluated for this receptor at all five of the areas. The future worker scenario assumes that a worker will have dermal contact with the water during showering. The scenario of having a potable water well on one of the disposal sites is unlikely, but as with groundwater ingestion, the scenario is included in the BRA so that the potential risks associated with groundwater contamination at the study areas may be quantitatively evaluated.

3.2.5.3 Groundwater Inhalation Pathway

Inhalation exposure to groundwater VOCs may occur as these chemicals volatilize from groundwater to indoor air. Similar to direct contact, domestic inhalation of groundwater chemicals may occur during various activities, including showering, cooking, and washing cars, dishes, and laundry. Inhalation exposure to groundwater VOCs in an industrial setting may result from certain work-related processes, as well as showering. The groundwater inhalation pathway is not complete for current residents or workers because there are currently no wells near Areas F, G, R, or S/T.

3.2.5.3.1 As with groundwater ingestion and direct contact, the groundwater inhalation pathway is considered complete only for future workers and is evaluated for this receptor at all five of the areas. The future worker scenario assumes that a worker will inhale groundwater VOCs while showering. Although unlikely, this scenario is included in the BRA

so that the potential risks associated with groundwater VOC contamination at the study areas may be quantitatively evaluated.

3.2.5.4 Soil Dermal Contact Pathway

Although soil contamination at the five study areas has been detected deep below the surface in many cases, certain COPCs have been found in surficial samples. The pathway of direct dermal contact with soil at the study areas must therefore be considered complete for areas with potential human receptors. The dermal exposure route for soil is evaluated for a person currently working at the building adjacent to Area F, as well as a maintenance worker at Area T. Direct soil exposure is evaluated for a future worker at all five study areas.

3.2.5.5 Soil Ingestion Pathway

The soil ingestion pathway considers incidental direct ingestion of contaminated soils. This pathway may occur during hand-to-mouth activities, such as smoking or eating after coming in contact with soil. The direct ingestion pathway is considered complete for areas with potential human receptors due to the presence of COPCs in surficial soils at all five study areas. Incidental ingestion of soil is also evaluated for a person currently working at the building adjacent to Area F and a maintenance worker at Area T.

3.2.5.6 Soil Inhalation Pathway

Inhalation of COPCs in soils can occur from 2 sources: (1) inhalation of chemicals that have volatilized from soil to ambient air, and (2) exposure to fugitive dusts generated from surficial soils due to wind or study area activities. Pathway number 1 is usually only of concern where there are buildings located on top of the contaminated media and inhalation exposure to indoor air may occur. VOC concentrations in outdoor ambient air are typically reduced by wind and atmospheric breakdown processes to undetectable levels, which is supported by the finding that no VOCs were detected by field teams during sampling efforts (G&M, 1991a, 1992a). Consequently, inhalation of ambient chemical vapors has not been quantified.

3.2.5.6.1 Inhalation of fugitive dusts is considered a complete pathway and is evaluated for study areas F, G, R, and S/T, as all of these study areas, although heavily vegetated, are not paved.

3.2.5.7 Sediment/Surface Water Dermal Contact Pathway

Incidental dermal contact with McDonald Creek sediments and surface water adjacent to Area G is evaluated for current recreational (while fishing) and future worker (during breaks) exposures. Study areas other than Area G either did not have sediment or surface water samples or the sediments and surface water were part of a wetland system.

3.2.5.8 Sediment/Surface Water Ingestion Pathway

As with the sediment/surface water dermal exposure pathway, incidental ingestion of sediment and surface water is evaluated only for current recreational and future worker exposures to McDonald Creek adjacent to Area G.

3.2.5.9 Fish Ingestion Pathway

Persons recreating at McDonald Creek adjacent to Area G may potentially be exposed to COPCs if they ingest fish caught from the creek. No samples of actual fish tissue are available; therefore, fish concentrations were modeled using the surface water levels found in McDonald Creek. The recreational fish ingestion pathway is considered to be complete for Area G and is quantitatively evaluated in this BRA.

3.2.6 IDENTIFICATION OF POTENTIAL ECOLOGICAL EXPOSURE PATHWAYS

Animals may be present in or on the soil and, depending on their physiological capabilities and behavior, may migrate or burrow between various contaminated soil layers. They may also migrate between various contaminated areas. Potential exposure pathways for animals from contaminated soils may include the following:

1. Dermal contact by burrowing animals;
2. Ingestion of contaminated soils;
3. Inhalation of contaminated wind-borne dusts;
4. Dermal exposure from contaminated soil particles adhering to skin, fur, or feathers;
5. Inadvertent consumption of soils via digging and burrowing activities;
6. Ingestion of animals or plants on which contaminated soils adhere; and
7. Ingestion of contaminated prey items, plants, and animals by resident and nonresident consumers. This pathway would be most applicable to predatory animals for COPCs that are significant bioaccumulators.

3.2.6.1 At Area G, limited contact with surface water may provide an exposure pathway to terrestrial organisms. Terrestrial animals may be exposed to a contaminant from surface water as follows:

1. Dermal exposure and absorption to animals washing, wading, swimming, or feeding in contaminated water;
2. Ingestion of contaminants in solution or suspension by animals;
3. Ingestion of contaminants retained on skin, fur, or feathers via preening; and
4. Ingestion of contaminated prey organisms (due to the extremely limited nature of the surface water, consumption of aquatic prey is unlikely).

3.2.6.2 Aquatic species would be exposed via uptake across cellular membranes (algae) and digestive and/or gill surfaces (invertebrates or vertebrates).

3.2.6.3 Sediments near Area G, particularly fine-grained sediments in depositional environments, often act as a sink for contaminants. These bottom sediments may provide an exposure pathway to terrestrial animals near Area G, especially since these are generally exposed (e.g., not covered by water). Benthic-dwelling organisms would have constant exposure. Potential exposure pathways via bottom sediments may include the following:

1. Dermal exposure to wading animals, bank inhabitants, and benthic species;
2. Ingestion of contaminants retained on skin, fur, or feathers via preening and feeding activities;
3. Consumption of contaminated prey or food items; and
4. Ingestion of contaminated porewater via digestive system and/or across gill surfaces by benthic dwelling species.

3.2.6.4 Plants are also ecological receptors at RSA. However, observations from the site visits revealed the following:

1. Significant portions of the areas evaluated are pine plantation or maintained fields of characteristically low diversity, and
2. Natural areas appeared to consist of diverse native plant communities with composition and relative dominants not distinguishable from reference sites.
3. The areas under study are man-made disturbances such as constructed landfills, and thus are not conducive to providing habitat for native plant species.

3.2.6.5 For the purposes of this BRA, screening evaluations as to the risks to ecological receptors refer to risks to fauna only with potential impacts to plants discussed only qualitatively. The risks to terrestrial animals, birds, and aquatic species are discussed later in this BRA.

3.2.6.6 Of the many potential exposure pathways for terrestrial ecological receptors that are discussed previously, only the ingestion of contaminants in surficial soils was quantified in this BRA. The dose of contaminants received from direct ingestion of soil in the diet is expected to account for the most significant portion of the exposure, especially since the primary exposed population is small mammals. Uptake of contaminants into plant structures is possible, but is limited for many contaminants due to the physiological structure of the root system. In any case, observations at the site have shown that the onsite vegetation, while providing cover, is low in food value. Therefore, the exposure to COPCs through the ingestion of plants is considered to be minimal compared to the direct ingestion of contaminants in soil.

3.2.6.7 The methods for the estimation of dermal absorption of contaminants into animal tissue are currently under development for use in risk assessment. This is also the case for the quantification of risks due to the inhalation of particles. Site-specific observations have led to the conclusion that small mammals (herbivores) represent the primary exposed population at the study areas. Food chain pathways have not been modeled for this screening evaluation because the small size of the study areas relative to the foraging ranges of potential predators suggests that the contribution of food from a study area to the diet of a predator would be very small. For example, it is estimated that Areas F, G, R, and S/T would support 0.01, 0.01, 0.02, and 0.04 red-tailed hawk breeding pairs, respectively. With their large home ranges, any resident hawks would have to obtain most of their food offsite. In addition, the COPCs included for further quantitative analysis are not strong bioaccumulators. Nevertheless, the potential for bioaccumulation will be qualitatively discussed. The quantification of risks to terrestrial and avian species is therefore limited to the evaluation of risks due to the ingestion of contaminated soils.

3.2.6.8 The evaluation of risks to aquatic species is accomplished through the comparison of surface water concentrations with water quality standards and ecotoxicity benchmarks.

Surface water quality standards are designed to be protective of even the more sensitive species that may be present in the ecosystem. This evaluation of surface water concentrations occurs in the ecological risk characterization section.

3.3 QUANTIFICATION OF EXPOSURE

Quantifying the magnitude of exposure and assessing the frequency and duration of exposure to the population, involves two stages: (1) exposure concentration estimation, and (2) pathway-specific intake estimation. The following sections present the application of these two stages as they pertain to RSA.

3.3.1 EXPOSURE CONCENTRATIONS

Exposure concentrations are the contaminant concentrations that a receptor may come in contact with at a study area. Exposure concentrations, either the maximum concentration or upper 95th confidence limit of the mean (UCL_{95}), were previously calculated for all exposure media as part of the Phase I and Phase II RFI reports. According to the RFI reports, these exposure concentrations were estimated based on RAGS methods, thus, they were obtained directly from the G&M RFI reports and used in this BRA for evaluating both human and ecological risks. With the exception of Area R where only Phase I samples were collected, all UCL_{95} concentrations were based on combining relevant data from both the Phase I and Phase II investigations. The UCL_{95} concentrations were calculated as follows (G&M, 1992):

$$UCL_{95} = \bar{x} + t_{\alpha}(s/\sqrt{n}) \qquad (3\text{-}1)$$

where: $\bar{X}$ = Sample arithmetic mean $= \frac{1}{n}\sum_{i=1}^{n} x_i$

t = "t" values are from Student's t distribution and is based on (n-1) degrees of freedom (df).

α = The probability of a Type I error which is equivalent to rejecting the null hypothesis (H_o) given that H_o is true; in this case, $\alpha = 0.95$ which is equivalent to a one-tailed confidence interval with a probability of a Type I error at 0.05.

s = Population standard deviation $= \sqrt{s^2}$

$$s^2 = \text{Population variance} = \frac{1}{n} \sum_{i=1}^{n} (x_i - \bar{x})^2$$

$$n = \text{number of samples}$$

3.3.1.1 For those constituents with concentrations below detection limits, one-half of the detection limit was used in the calculation. Because the upper control limit (UCL) calculation uses values for undetected constituents that are equal to one-half the reported detection limit, the calculated UCL can be significantly affected by samples where matrix interferences have resulted in sample dilution and significantly elevated detection limits have been reported. In some instances, these elevated detection limits result in an UCL exceeding the maximum concentration of the constituent detected. In these cases, the maximum detected concentration (MDC) instead of the UCL was used for quantifying health risks (Appendix B).

3.3.1.2 Constituents that were classified as undetected due to contamination in laboratory or field blanks were included in the risk analysis. The concentrations used were equal to the qualified value rather than one-half the undetected values to provide a conservative estimate of the risk associated with those constituents whose presence or absence could not be firmly established.

3.3.1.3 A summary of the UCL_{95} concentrations obtained from the RFI reports and subsequently used in this risk assessment are presented in Appendix B. Because the ingestion of fish caught near Area G was not evaluated as part of the RFIs (G&M, 1991; 1992), a single set of exposure concentrations were calculated. The fish tissue concentrations were calculated by multiplying a bioconcentration factor (BCF) by the UCL_{95} in surface water. BCFs are listed in Table 3.3-1.

3.3.1.4 G&M (1991 and 1992) calculated the normal distribution-based UCLs under RCRA RFI guidance. However, current CERCLA BRA guidance recommends lognormal distribution-based UCLs for most environmental data (EPA, 1992a). Section 5.5.2, Uncertainty Associated with Exposure Assessment, provides examples that illustrate the impact of the use of normal versus lognormal distribution-based UCL equations.

3.3.2 CHEMICAL INTAKES FOR HUMAN EXPOSURES

Chemical intake is the amount of the chemical contaminant entering the receptor's body. To estimate a reasonable maximum exposure (RME), upperbound (upper 90th or 95th) percentile exposure factor values were used, where available. The exposure concentrations from the study area were estimated using the UCL_{95} data from the reports which were completed for RSA. The formulas used to calculate human pathway-specific chemical intakes were based on the generic intake equation presented in RAGS (EPA, 1989) and can be presented as follows:

$$I = \frac{C \times CR \times EF \times ED}{BW \times AT} = (mg/kg/day) \qquad (3\text{-}2)$$

where: I = intake, the amount of chemical at the exchange boundary;

C = chemical concentration, the average concentration contacted over the exposure period (e.g., mg/L for groundwater);

CR = contact rate, the amount of contaminated medium contacted per unit time or event [e.g., liters per day (L/day)];

EF = exposure frequency (days/year);

ED = exposure duration;

BW = body weight, the average body weight of the exposed individual [kilogram (kg)]; and

AT = averaging time, period over which exposure is averaged (days).

3.3.2.1 Because the exposure conditions differ for each receptor location, study-area-specific exposure factors, as well as standard default exposure factors, were incorporated into the generic formula for each exposure pathway to produce a set of chemical intake formulas for each study area. When applying study-area-specific exposure factors, best professional judgement was used to derive these values. For example, based on an evaluation of the meteorological data for the study area, exposure factors for recreational exposure were derived (i.e., exposure frequency and duration, surface area of skin available for contact). The study area-specific and chemical-specific intake formulas, along with the intake factors used for the intake estimations, are described in detail in Appendix C. Appendix D presents the human intake estimates.

3.3.3 ECOLOGICAL RECEPTOR INTAKES

The process for evaluating potential health risks to terrestrial and avian wildlife is similar to the procedure for evaluating health risks to humans. The exposure points, exposure concentrations, and potential uptake routes are identified, the chemical intakes are calculated, and the health risks are determined. The main differences between evaluating ecological and human health risks are that: (1) while the objective of the human health risk assessment is to protect the individual, the objective of the ecological risk assessment is to protect the population, and (2) although acceptable human exposure levels are available for many chemicals, acceptable doses have not been determined for the majority of ecological receptors.

3.3.3.1 For most of the terrestrial organisms found at the study areas, home ranges are large relative to the extent of contamination; however, for an upperbound estimate of risk, the home range of mice, raccoons, deer, and bobwhites was assumed to be the size of the contaminated area. While this assumption is overly conservative, if no risk is exceeded, then more site-specific studies are not warranted (i.e., food chain, biomagnification studies, site-specific exposure analysis). The exposure concentrations calculated for human exposure (Section 3.3.2) were also used for evaluating ecological exposure. Although mean concentrations are thought to be more relevant due to the extent of the populations and the mobility of individuals, for this BRA the conservative assumption was made that exposure point concentrations for terrestrial and aquatic receptors were assumed to be the lesser of the UCL_{95} or maximum observed concentrations. (Exposure concentration values for each medium at each site are provided in Appendix B.)

3.3.3.2 The basic intake formula for ecological exposure to soil is identical to the formula for human intake presented in Equation 3-2 (Sec. 3.3.2) with the "CR" or "IR" term given in units of kg/day, the "C" term expressed in mg/kg, and the "ED" term expressed in years. Because the actual exposure (including entry route, duration, and concentration) for terrestrial organisms at the study areas is unknown, exposure pathways were developed to estimate exposure doses that could then be compared with ecological benchmark concentrations. For this assessment, accumulation via the food web will not be quantified for terrestrial exposure. Because the COPCs at this site are not strong bioaccumulators, the concentrations of study area contaminants in prey items will be much less than surficial soil concentrations.

Therefore, the exposure of terrestrial organisms to soils is evaluated with a conservative intake versus dose scenario.

3.3.3.3 Most ecotoxicity benchmarks for vertebrates are based on doses administered in feed or as a dose per kilogram of body weight. For these organisms, scenarios were developed that assumed a portion of the ration was actually from soil ingested during foraging, burrowing, grooming, and related activities. This portion of the ration was included in the numerator of the intake equation (Equation 3-3). Intake parameters for each indicator species are presented in Table 3.3-2 and are described in the following sections. The soil intake values for each indicator species presented in Table 3.3-2 are multiplied by the chemical exposure concentrations to yield the receptor-specific chemical intakes presented in Table 5.4-1.

$$I = \frac{C * IR * EF * EC * soil\ fraction}{BW * AT} \qquad 3\text{-}3$$

3.3.3.4 Mouse--The pertinent exposure factors from EPA guidance (EPA, 1993) for the deer mouse (*Peromyscus maniculatus*) are as follows:

- Average adult body weight = 0.019 kilograms (kg)
- Average food intake = 0.0038 kilograms/day (kg/day)
- Mean natural life-span = 1 year
- Amount of soil ingested in diet = 2 percent

3.3.3.5 It was conservatively assumed that the mouse receives all of its diet from contaminated areas at RSA and that the mouse is exposed to the soils every day of its life. These assumptions, although conservative, should allow for the determination of an upper-end risk estimate.

3.3.3.6 Raccoon--Pertinent exposure factors from EPA and other guidance for the raccoon (*Procyon lotor*) are as follows:

- Average adult body weight = 5.7 kg (EPA, 1993)
- Average food intake = 0.350 kg/day (Spector, 1956)
- Mean natural life-span = 3 years (EPA, 1993)
- Amount of soil ingested in diet = 9.4 percent (EPA, 1993).

3.3.3.7 It was conservatively assumed that the raccoon eats from contaminated areas at RSA every day and that 100 percent of its soil intake is contaminated.

3.3.3.8 Deer--Pertinent exposure factors obtained from various sources for white-tailed deer (*Odocoileus virginianus*) include the following:

- Average adult body weight = 54 kg [calculated using data from a survey performed at RSA (MICOM, 1992), where a weighted average was taken for adult deer between the ages of 1.5 and 3.5+ years old, N = 378].
- Average food intake = 0.972 kg of grass per day [estimated from the known ingestion rate for a cow (ESE, 1992) which was used to extrapolate to a 54 kg deer].
- Mean natural life-span = 5 years [but the majority of adult deer that were killed during hunting activities at RSA were between 1.5 and 2.5 years old (USACE, 1989].
- Amount of soil ingested in diet = 6.3 percent [extrapolated from another grazing herbivorous species, the jackrabbit based upon EPA (1993) exposure factors for jackrabbits].

3.3.3.9 The ingestion of vegetation and soil was conservatively assumed to occur every day of the deer's life. The entire diet was assumed to come from areas with contaminated soils.

3.3.3.10 Bobwhite Quail--The pertinent exposure factors from EPA guidance (EPA, 1993) for the bobwhite quail (*Colinus virginianus*) are as follows:

- Average adult body weight = 0.174 kg (EPA, 1993).
- Average food intake = 0.014 kg food/day (EPA, 1993).
- Amount of soil ingested in diet = 1 percent (based on feeding behaviors published by EPA, 1993, and study area-specific conditions that would preclude significant grit ingestion for such seed foraging birds).
- Mean natural lifespan = 10 months (EPA, 1993).

3.3.3.11 For this ecological RA it was conservatively assumed that the entire diet of the quail for this 10-month period is derived from areas with contaminated soils at RSA.

3.3.3.12 For aquatic organisms, chemical intake was not calculated. Surface water exposure concentrations were compared directly to an acceptable level (i.e., ecological benchmark).

3.4 IDENTIFICATION OF UNCERTAINTIES IN EXPOSURE ASSESSMENT

3.4.1 HUMAN EXPOSURE ASSESSMENT

The most significant uncertainty in the RA is due to the exposure assumptions used to quantify the intake. Exposure pathways assumed at each of the five study areas are future projected scenarios, except for the current worker exposure scenario at Areas F and T and the current recreational exposure scenario at Area G. The duration of time spent at each source area is overestimated; for example, there are no known onsite workers who frequently come in contact with the contaminated portions of the study areas. A more detailed description of uncertainties associated with exposure pathways is presented in Section 5.2.

3.4.1.1 Because the UCL_{95} calculations use values for undetected chemicals that are equal to one-half the reported detection limit, the calculated UCL can be significantly affected by samples where matrix interferences have resulted in sample dilution and significantly elevated detection limits. In some instances, these elevated detection limits result in an UCL_{95} exceeding the maximum detected concentration of the chemical detected. In these cases, the maximum detected concentration was used as the RME concentration to evaluate study area risk instead of the UCL_{95}.

3.4.1.2 In addition, inclusion of several subsurface samples [samples collected in soil borings designated as shallow (SB-A) at a depth of 2 to 4 ft] in the UCL_{95} calculations for surface soil at Areas F and S/T may overestimate risk. These samples were included based on the assumption that the concentrations observed within the sample are present at 2 ft and not dispersed throughout the 2 to 4 ft horizon.

3.4.2 ECOLOGICAL EXPOSURE ASSESSMENT

Because the actual exposure (including entry route, duration, and concentration) for organisms at the study area is unknown, scenarios have been developed to estimate exposure doses. These estimated exposure doses can then be compared to benchmark concentrations, which are literature-derived values for the lowest concentration or dose of chemical causing an ecologically interpretable negative response to a test organism. Ecologically interpretable

negative responses may be either acute or chronic. They are responses such as lowered reproductive rates, lowered survival rates, etc. When possible, it is important to consider exposures to early life stages since in many species they are more susceptible to contaminated effects than adults.

3.4.2.1 For surface water, the assumptions are that the organism is exposed to the surface water exposure concentration for a length of time corresponding to the benchmark test conditions. However, exposure is unlikely to be continuous for all organisms for several reasons as follows:

1. Many aquatic organisms can swim into and out of contaminated zones,
2. Exposure concentrations will fluctuate with changing water levels and other factors, and
3. Many aquatic organisms such as chironomids spend only the larval portions of their life cycles in aquatic habitat.

3.4.2.2 Such conditions and the continuous exposure assumption justify reduction of exposure durations in the exposure dose estimation step. However, because this scenario does not estimate potential uptake from sediments, pore water, or prey items, the exposure duration at a contaminated area was not reduced. It is assumed that these negate each other.

3.4.2.3 Some data are now available in EPA publications concerning the amounts of soil that may inadvertently be ingested by animals during feeding, preening, burrowing, and other activities. The exposure scenarios developed for soil exposure to terrestrial organisms, which assume certain percentages of soil in the diet come primarily from EPA guidance (EPA, 1993). The uncertainty involved with estimating the amount of soil in the diet is high, but the exposure factors are designed to over, rather than underestimate the intake of contaminants.

Table 3.2-3. Metals Detected in the Study Areas

Metal	Area F	Area G	Area R	Area S/T
Arsenic	X	--	--	--
Cadmium	X	--	--	--
Mercury	X	X	--	--
Lead	--	X	X	X
Chromium	X	X	X	X

Source: ESE.

Table 3.3-1. Soil Intake Values for Terrestrial Indicator Species

Species	Soil Intake (kg soil/kg-BW/day)	Intake Factors			Source / Comments
Mouse	4E-03	IR	0.0038	kg/day	(0.2 g/g-day * 19 grams)
		EF	365	days/year	
		ED	1	year	EPA Factor
		%soil	0.02		EPA factor
		BW	0.019	kg	average of six mean values from EPA doc. (Millar, Fordham, Dewsbury)
		AT	365	days	
Raccoon	6E-03	IR	0.35	kg/day	based on Spector (1956)
		EF	365	days/year	
		ED	3	years	
		%soil	0.094		EPA factor
		BW	5.7	kg	Adult weight based on EPA factors (Sanderson, Nagel, Johnson)
		AT	1095	days	EPA factor (Johnson)
Deer	1E-03	IR	0.972	kg/day	based on g/kg BW value for cattle from LAAP report
		EF	365	days/year	
		ED	5	years	
		%soil	0.063		factor from jackrabbit EPA factor
		BW	54	kg	RSA Natural Resources document
		AT	1825	days	RSA Natural Resources Document - NUJ estimation
Bobwhite	7E-04	IR	0.014	kg/day	based on EPA factor of .07775 x BW (avg. from Koerth&Guthery data)
		EF	300	days/year	total lifespan - days
		ED	0.8333333333	years	EPA Factor
		%soil	0.01		based on EPA factor value for American woodcock
		BW	0.174	kg	EPA Factor (avg. from Robel, Roseberry & Klimstra, Guthery)
		AT	304.16666667	days	

Source: ESE.

4.0 TOXICITY ASSESSMENT

This section of the BRA weighs the available evidence regarding the potential for a particular chemical to cause adverse effects in exposed individuals, and provides an estimate of the extent of exposure and possible severity of adverse effects. The assessments used to develop toxicity values consist of two steps: (1) hazard identification, and (2) dose-response assessment. The hazard identification determines the potential adverse effects associated with exposure to a chemical as well as the types of potential health effects involved. In the dose-response assessment, quantitation of the toxicity values and estimation of reference dose values is performed.

4.0.1 Since most of the COPCs detected at Areas F, G, R, and S/T are well studied, toxicological assessments and water-quality criteria technical documents prepared by EPA served as the primary information sources on pharmacokinetics, human health effects, and effects on fish and wildlife. Toxicity factors [cancer slope factors (CSFs) for carcinogens and reference doses (RfDs) for noncarcinogens] presented in this section reflect current toxicological information available from EPA [Integrated Risk Information System (IRIS), 1995; Health Effects Assessment Summary Tables (HEAST) EPA, 1994] and other sources. These factors are used to estimate risk and HI values in the risk characterization.

4.0.2 Except for lead, the exposure levels of chemicals observed at Areas F, G, R, and S/T are more relevant to a chronic exposure scenario, as none of the identified contaminants are at high enough levels to warrant an acute or a subchronic toxicity criteria application. Lead is an exception, as some critical effects can be manifested at subchronic exposure levels, as a result, EPA has yet to establish RfDs or CSFs for lead. Thus, for risk characterization, lead was evaluated qualitatively (i.e., comparing to health-based guidance levels). A list of all criteria used for the relative risk calculations is included in Table 4.0-1. The RfDs and CSFs presented in this table are the values provided in IRIS (1995), HEAST (EPA, 1994), and other sources and have been rounded to two significant figures. A description of carcinogenic weight-of-evidence (WoE) classifications for potential carcinogens is presented as Table 4.0-2.

4.1 TOXICITY INFORMATION FOR NONCARCINOGENIC EFFECTS

An RfD is an estimate (with uncertainty spanning approximately an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects if experienced continuously during a lifetime. RfD is the toxicity value most often used to evaluate the noncarcinogenic impacts from exposure to chemicals. RfDs are specific to the route of exposure (i.e., an inhalation RfD is used for inhalation exposure), critical effect (developmental or systemic), and the length of exposure evaluated. Chronic RfDs are specifically developed to be protective against long-term exposure to a chemical. Subchronic RfDs are developed to characterize potential noncarcinogenic effects associated with shorter-term exposures. The derivation procedure for an RfD can be found in RAGS, Part A (EPA, 1989a) or other technical guidance documents for criteria development.

4.1.1 The list of COPCs at Areas F, G, R, and S/T and their respective RfDs are presented in Table 4.0-1. The RfDs listed are the chronic RfDs, as Superfund guidance requires use of chronic exposure dose (RfD) levels. Chronic RfDs are applicable because: (1) the contaminant concentrations typically found at Superfund sites are low, and (2) the expected intake rate of contaminants is similar to the chronic dose levels administered to experimental animals in chronic toxicity studies.

4.2 TOXICITY INFORMATION FOR CARCINOGENIC EFFECTS

A CSF and the accompanying WoE determination are the toxicity data most commonly used to evaluate potential human carcinogenic risks. The methods used by EPA to derive CSFs or unit risks are described in RAGS, Part A (EPA, 1989a). For carcinogens, EPA usually assumes a nonthreshold response, or that at every dose level of a carcinogen some amount of adverse response occurs; no dose is believed to be risk-free. For carcinogens, EPA uses a two-part evaluation, determination of a WoE classification and calculation of a CSF.

4.2.1 Generally, a CSF is a plausible upperbound estimate of the probability of a response per unit intake of a chemical over a lifetime. Toxicity to carcinogens can be expressed in several ways. The CSF is usually the UCL_{95} of the slope of the dose-response curve and is expressed as per milligrams per kilogram per day $(mg/kg/day)^{-1}$. Toxicity values for

carcinogenic effects can also be expressed as risk per unit concentration of the substance in the medium of exposure, referred to as unit risks.

4.2.2 Toxicity profiles for the final COCs (those chemicals associated with a HI $\geq$ 1 or a risk $\geq$ 1E-6 via any exposure pathway) are presented in Appendix E.

4.3 CHEMICALS WITH UNAVAILABLE EPA TOXICITY VALUES

4.3.1 CARCINOGENIC CHEMICALS WITH NO ESTABLISHED CSF

Several of the organic chemicals found at Areas F, G, R, and S/T are either known, suspected, or possible human carcinogens. A list of CSFs and WoE classifications for the carcinogens identified at Areas F, G, R, and S/T is presented in Table 4.0-1.

4.3.1.1 Of the organic chemicals detected at Areas F, G, R, and S/T, butylbenzyl phthalate and 1,1-dichloroethane are classified as Group C possible human carcinogens. These organic chemicals do not have EPA-determined CSF values; therefore, only the noncarcinogenic hazards associated with exposure to these organic chemicals are evaluated. In addition, no CSFs have been established for the potentially carcinogenic PAHs benz(a)anthracene, benzo(b)fluoranthene, chrysene, dibenz(ah)anthracene, and indeno(1,2,3-cd)pyrene. However, oral and inhalation CSFs can be derived based on the oral and inhalation values for benzo(a)pyrene and using the Toxicity Equivalency Factor (TEF) Methodology proposed by EPA Region IV (EPA, 1992) and presented in the EPA Region III Risk-Based Concentration Table, January to June 1995 (EPA, 1995). According to this methodology, the toxicity of potentially carcinogenic PAHs can be compared to that of benzo(a)pyrene using the following TEFs: 1.0 for dibenz(ah)anthracene; 0.1 for benz(a)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene; and 0.001 for chrysene. Therefore, multiplying the oral and inhalation CSFs for benzo(a)pyrene by the TEFs for the respective PAHs yields the following oral and inhalation CSFs: 7.3 and 6.1 $(mg/kg/day)^{-1}$ for dibenz(ah)anthracene; 0.73 and 0.61 $(mg/kg/day)^{-1}$ for benz(a)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene; and 0.0073 and 0.0061 $(mg/kg/day)^{-1}$ for chrysene.

4.3.1.2 The oral and inhalation CSFs for tetrachloroethene and trichloroethene have been removed from IRIS (1995) and HEAST (EPA, 1994) pending further evaluation of their carcinogenic potential in humans. The CSF values presented for tetrachloroethene in

Table 4.0-1 are from the 1991 Annual Update to HEAST (EPA, 1991a), while the values for trichloroethene are presented in an EPA Environmental Criteria Assessment Office (ECAO) Risk Assessment Issue Paper and have been adopted by EPA Region III (EPA, 1995).

4.3.1.3 EPA has not developed an inhalation unit risk (UR) or CSF for p,p'-DDE (IRIS, 1994; EPA, 1994a). To evaluate potential risks associated with inhalation of this pesticide, the inhalation CSF for its parent compound (p,p'-DDT) is used. The parent compound is known to be transformed to p,p-DDE upon intake by living things (ATSDR, 1989a).

4.3.1.4 Of the inorganic COPCs at Study Areas F, G, R, and S/T, lead is currently classified as a Group B2 suspect human carcinogen. However, no CSF values are available for this metal and the potential carcinogenicity associated with exposure to this metal cannot be evaluated. Only the potential noncarcinogenic hazards associated with exposure to lead were evaluated.

4.3.2 CHEMICALS WITH NO ESTABLISHED RFDs

Noncarcinogenic effects were evaluated for all of the organic COPCs identified at Areas F, G, R, and S/T, including potentially carcinogenic chemicals. Most of the chemicals detected at Areas F, G, R, and S/T and considered as COPCs have final or interim toxicity values developed by EPA.

4.3.2.1 The oral RfDs for benzene, 1,2-dichloroethane, and trichloroethene and the inhalation RfDs for benzene and 1,2-dichloroethane are interim values provided by ECAO and presented in EPA Region III's Risk-Based Concentration Table, January to June 1995 (EPA, 1995). The oral RfD for vinyl chloride is a minimum risk level (MRL) provided in ATSDR's Toxicological Profile for Vinyl Chloride (1989).

4.3.2.2 In addition, no human health-based values are available for bis(2-chloroethyl) ether; therefore, a provisional health-based value was developed. The oral value of 0.00075 mg/kg/day for bis(2-chloroethyl) ether is based on an acute oral LD_{50} for rats of 75 mg/kg (RTECS, 1995), an uncertainty factor of 10,000 (10 for sensitive human subpopulations, 10 for animal-to-human extrapolation, 10 for acute-to-chronic extrapolation, and 10 to account

for a study endpoint other than a NOAEL) and a modifying factor of 10 (accounts for the lethal endpoint of the study).

4.3.2.3 For comparative purposes, the oral RfD for the PAH pyrene (0.03 mg/kg/day) was used to evaluate potential noncarcinogenic hazards associated with exposure to the PAHs benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(ghi)perylene, chrysene, dibenz(ah)anthracene, indeno(1,2,3-cd)pyrene, and phenanthrene. The oral RfD for p,p'-DDT was used to evaluate exposure to p,p'-DDE.

4.3.2.4 EPA has determined that the inhalation RfD for 1,1,1-trichloroethane is not verifiable and has removed this value from IRIS (1995) and HEAST (EPA, 1994). To evaluate potential hazards associated with inhalation of this volatile chemical, the inhalation RfD presented in the 1991 Annual Update to HEAST (EPA, 1991a) is used.

4.3.2.5 In addition, no RfDs are available for the heavy metal lead. Due to the high toxicity of this metal in children, EPA recommends using a biokinetic uptake model (EPA, 1991b) on a site-by-site basis to evaluate blood-lead (PbB) levels instead of a RfD approach.

4.4 ECOTOXICITY BENCHMARKS

Risks to ecological receptors are quantitatively evaluated by comparing the chemical intake of a receptor to an assumed acceptable dose, or adjusted benchmark, for that chemical in the specific receptor. Adjusted benchmarks are derived from raw ecological benchmarks using the following equation:

$$\textit{Adjusted Benchmark} = \frac{\textit{Raw Benchmark}}{\textit{Applicable Uncertainty Factors}}$$

4.4.1 Selected raw benchmarks for the COPCs at Areas F, G, R, and S/T were obtained from the available literature and are presented in Table 4.4-1. Raw benchmarks were chosen based on the following considerations:

1. Including acute and chronic effects,
2. Choosing results of tests using organisms as closely related taxonomically to representative receptors as possible,
3. Choosing tests with ecologically relevant endpoints, and

4. Choosing tests conducted with an ecologically relevant exposure pathway.

4.4.2 The preferred value sought was a chronic lowest-observed-adverse-effect level (LOAEL) in the indicator species or related organism. An uncertainty factor (UF) of 10 was applied to extrapolate toxicity data between species. For chemicals with no available chronic LOAEL, other values (e.g., an LD_{50}, or concentration lethal to 50 percent of a study population) were used to derive an adjusted benchmark. A UF of 10 was applied to data for which the endpoint is from a lethal study. Because individuals may comprise a significant fraction of the population of rare, threatened, or endangered species, UFs for sensitive individuals are not applied for ecological risk characterization. Therefore, the maximum possible uncertainty factor used to develop an adjusted benchmark was 100. The adjusted benchmarks are provided in Table 4.4-2.

4.5 UNCERTAINTIES RELATED TO TOXICITY INFORMATION

The quantitative uncertainty (uncertainty factor and modifying factor) associated with each toxicity value is listed in Table 4.0-1. The greater the uncertainty factor/modifying factor, the greater the uncertainty behind applicability of the value to the environmental exposure conditions. In addition, while the method used for the development of CSFs assumes a nonthreshold approach, experimental evidence indicates that some of the potential carcinogens have dose-response curves that suggest a response threshold (Casarett and Doull, 1991). Values derived from sources other than IRIS are presumed to exhibit greater uncertainty because they have not been subjected to the same level of peer review.

4.6 TOXICITY INFORMATION SUMMARY

Table 4.0-1 summarizes the toxicity information for all of the COPCs (all organic chemicals detected in all media; all inorganic chemicals detected in groundwater, surface water, and sediment; inorganic chemicals detected above background levels in soil) at Areas F, G, R, and S/T. Table 4.0-2 presents a description of WoEs for potential carcinogens.

5.0 RISK CHARACTERIZATION

The objective of this risk characterization is to integrate information developed in the exposure assessment (Section 3.0) and the toxicity assessment (Section 4.0) into a complete evaluation of the potential and actual human health and ecological risks associated with contaminants at the areas of concern. The BRA evaluates the nature and degree of risk to the potential receptor populations described in Section 3.0. Wherever possible, risk estimates are derived for individual source areas as well as for the total contaminant contribution from the five study areas to aid in developing priorities for remedial action planning.

5.0.1 The methods used in this risk characterization are based on those presented in EPA risk assessment guidance for human and ecological exposures (EPA, 1989a; EPA, 1989b; EPA, 1991); Guidance on Ecological Assessment of Hazardous Waste Sites (EPA, 1989c), ECO Updates (issued intermittently by EPA to supplement RAGS), and EPA Region IV guidance. Uncertainties associated with each of the analyses are presented along with relevant calculations. These uncertainties may be attributed to several input factors such as lack of monitoring data, incomplete understanding of the mechanisms involved in contaminant transport, assumptions used in the RA, or lack of toxicological information for a particular contaminant.

5.1 METHODS FOR HUMAN RISK CHARACTERIZATION

5.1.1 CARCINOGENIC RISK

The potential risks associated with exposure to individual carcinogens are calculated using CSFs from IRIS and HEAST as presented in Section 4.0. The risk is the chemical intake value multiplied by the CSF.

$$Risk = I \times CSF \qquad (5\text{-}1)$$

where: Risk = probability for an individual developing cancer under the assumed exposure conditions (unitless),

I = daily chemical intake averaged over a lifetime of 70 years (mg/kg/day), and

CSF = carcinogenic slope factor, expressed in $(mg/kg/day)^{-1}$

5.1.1.1 The formulas and factors used to calculate the intakes are included in Appendix C. The intake values used for different pathways are included in Appendix D. The combined risk from exposure to multiple chemicals at a study area is evaluated by addition of resultant risks from different chemicals.

$$Risk_T = \Sigma Risk_i \qquad (5\text{-}2)$$

where: $Risk_T$ = the sum of individual chemical risks, unitless probability; and
$Risk_i$ = the risk estimate for the ith chemical.

5.1.1.2 Risks are also added across the pathways if the multiple exposures are to the same individual [e.g, a person working with the soil onsite could be exposed by both the potential exposure pathways (namely oral and dermal) and, if relevant, inhalation]; therefore, the pathways are additive.

Risk from exposure to soil = Risk (exposure $pathway_1$) + Risk (exposure $pathway_2$) + Risk (exposure $pathway_3$)+....+ Risk (exposure $pathway_i$)

5.1.1.3 The study-area-specific carcinogenic risk estimates are determined using the UCL95 concentrations and the exposure factors presented in Section 3.0. The potential risks resulting from exposure to the study area contaminants are compared with the EPA target cumulative risk levels. Acceptable exposure levels are the contaminant concentration levels that present an excess cancer risk of 10^{-6} (1 in 1,000,000) to 10^{-4} (1 in 10,000) to the exposed population, based on the dose-response information for each carcinogenic COPC (NCP) 40 Code of Federal Regulations (CFR) 300, 430:62].

5.1.1.4 When a cumulative carcinogenic risk (risk associated with exposure to a mixture of chemicals) to an individual receptor under the assumed exposure conditions at a Superfund site exceeds 10^{-4} (1 in 10,000 excess cancer risk), CERCLA generally requires remedial action at the site (EPA, 1991a). If the cumulative risk is less than 10^{-4}, action generally is not required but may be warranted if a chemical-specific standard that is risk-based [e.g., maximum contaminant level (MCL)] is violated. A risk-based remedial decision could be superseded by the presence of noncarcinogenic impact or environmental impact requiring

action at the study area. Therefore, based on EPA Superfund guidance (EPA, 1991a), the individual risks are compared to the lower bound of the acceptable risk range, 10^{-6}, while the cumulative site-related carcinogenic risks at each area of concern are compared with the upper limit of permissible risk 10^{-4} (1 in 10,000).

5.1.2 NONCARCINOGENIC HI

Noncarcinogenic health risks are estimated by comparing actual or expected exposure levels to acceptable concentrations. This is accomplished by calculating a noncarcinogenic hazard quotient (HQ). An HQ is the ratio of chronic daily intake of the study area contaminant and the RfD of the contaminant and is calculated as follows:

$$HQ = \frac{I}{RfD} \tag{5-3}$$

where: I = intake of contaminant (mg/kg/day), and
RfD = reference dose of contaminant (mg/kg/day).

5.1.2.1 The impact from the presence of multiple chemicals at a study area is considered additive of impacts from individual contaminants. Thus, the HI is equal to the sum of the HQs:

$$HI = \frac{I_1}{RfD_1} + \frac{I_2}{RfD_2} + \ldots + \frac{I_i}{RfD_i} \tag{5-4}$$

where: I_i = Intake for the ith toxicant (mg/kg/day), and
RfD_i = reference dose for the ith toxicant (mg/kg/day).

5.1.2.2 I and RfD are expressed in the same units and represent the same exposure period (i.e., chronic, subchronic, or shorter term). When the HI exceeds unity (1.0), there may be concern for potential health effects. An HI or HQ equal to or less than 1.0 indicates that it is unlikely for even sensitive populations to experience adverse health effects (EPA, 1989). While any single chemical with an exposure level greater than the toxicity value will cause the

HI to exceed unity, for multiple chemicals the HI can also exceed unity due to additivity of multiple chemical HQs.

5.1.2.3 The HI is estimated for both carcinogens and noncarcinogens to obtain an assessment for the overall potential for noncarcinogenic health effects. The total HI is estimated for each COPC and is provided in Appendix F. The study area-specific risk characterization for Areas F, G, R, and S/T are presented in the following sections.

5.2 STUDY-AREA-SPECIFIC RESULTS OF HUMAN RISK CHARACTERIZATION FOR CURRENT AND FUTURE LAND USE

Using the risk evaluation methods described previously, the exposure concentrations for the RME concentrations risk estimates were obtained for the noncarcinogenic and carcinogenic contaminants identified at each study area. The HI values for the noncarcinogens are presented separately from the risk values for the carcinogens. A discussion of the human HI and risk results are presented for each study area in the following sections and are summarized in Table 5.2-1. Tables 5.2-2 and 5.2-3 present the COPCs that are contributing significantly to excess risks at each area. Appendix F summarizes all the noncarcinogenic HIs and cancer risks at each study area.

5.2.1 AREA F

5.2.1.1 Current Land Use

The current exposure scenario that exists at Area F is for a worker at the building adjacent to the study area who may be exposed to contaminants in surface soil. Based on the exposure assumptions evaluated, the current potential cumulative cancer risk associated with exposure to soil is approximately 5×10^{-6}, which is below the lower limit of the permissible cumulative risk of 10^{-6}. The cumulative HI for noncarcinogenic effects is 7×10^{-4}, which is well below the target HI of 1.

5.2.1.1.1 These results indicate that Area F does not pose adverse health effects or risks to current workers based on the exposure information evaluated. In addition, the risk and HI associated with current worker exposure may be overestimated as several subsurface samples [samples collected in soil borings designated as shallow (SB-A) at a depth of 2 to 4 ft] were included in the UCL_{95} calculations for surface soil. These samples were included based on

the assumption that the concentrations observed within the sample are present at 2 ft and not dispersed throughout the 2- to 4-ft horizon.

5.2.1.2 Future Land Use

The future exposure scenario evaluated at Area F is for a worker who may be exposed to contaminants in surface soil and groundwater. Based on the exposure assumptions evaluated, the future potential cumulative cancer risk associated with exposure to groundwater, 3×10^{-4}, exceeds the upperbound acceptable risk limit of 10^{-4} (Table 5.2-1). Ingestion of arsenic comprises 96 percent of the groundwater risk while combined ingestion and inhalation of carbon tetrachloride and chloroform contribute 2.6 and 1.4 percent, respectively, to the total risk. Future worker exposure to soil at Area F may result in an increased cancer risk of 5×10^{-6}, which is within the permissible cumulative risk range of 10^{-6} to 10^{-4}. The soil cancer risk is due primarily to oral and dermal exposure to PAHs (64 and 34 percent, respectively).

5.2.1.2.1 For noncarcinogenic effects, the cumulative HI associated with exposure to groundwater is 12, which exceeds the target HI of 1 (Table 5.2-3). This HI is due primarily to ingestion of mercury (approximately 90 percent) and arsenic (approximately 10 percent). The cumulative HI associated with future worker exposure to soil, 7×10^{-4}, is well below the target HI of 1.

5.2.1.2.2 These future risk and HI results indicate that groundwater at Area F may pose adverse health effects or risks to future workers based on the exposure assumptions evaluated (i.e., ingesting 1 L/day of water).

5.2.2 AREA G

5.2.2.1 Current Land Use

The current exposure scenarios that exist at Area G are for recreational exposure (i.e., boating and fishing activities) to the surface water and sediments in McDonald Creek adjacent to the study area, as well as ingestion of fish caught during the recreational activities. Based on the exposure assumptions evaluated, current potential increased cancer risks associated with recreational exposure to fish, sediment, and surface water are 9×10^{-9}, 5×10^{-10}, and 4×10^{-8}, respectively, which are below the lower acceptable risk limit of 10^{-6} (Table 5.2-1). The cumulative HIs for noncarcinogenic effects from exposure to fish, sediment, and surface

water are 4×10^{-4}, 2×10^{-6} and 2×10^{-3}, respectively, for an adult and 8×10^{-4}, 2×10^{-5} and 5×10^{-3}, respectively, for a child. All of these HIs are below the target HI of 1.

5.2.2.1.1 These results indicate that Area G does not pose adverse health effects or risks to current recreational receptors based on the exposure information evaluated.

5.2.2.2 Future Land Use

The future exposure scenario that exists at Area G is for a future worker who may be exposed to contaminants in surface soil, groundwater, surface water, and sediment. Based on the exposure assumptions evaluated, the future potential cumulative cancer risk associated with exposure to all four media is 5×10^{-5}, which is below the upper cumulative permissible risk of 10^{-4}. The cumulative cancer risk associated with exposure to groundwater, which accounts for greater than 99 percent of the total study area risk, exceeds the lower permissible risk limit of 10^{-6} and is due to ingestion of arsenic (Table 5.2-2).

5.2.2.2.1 For noncarcinogenic effects, the cumulative HI associated with groundwater exposure, 1.8, exceeds the target HI of 1 due to ingestion of cadmium (35 percent), chromium (35 percent), barium (16 percent), and arsenic (14 percent) (Table 5.2-2). The HIs associated with exposure to surface soil, surface water, and sediment are 3×10^{-6}, 2×10^{-4}, and 2×10^{-6}, respectively. All of these HIs are below the target HI of 1.

5.2.2.2.2 Although no RfD or CSF has been established for lead, the groundwater concentrations can be compared to the EPA drinking water action level of 15 μg/L established under the Safe Drinking Water Act (SDWA). The groundwater exposure concentration of 139 μg/L indicates that the potential for adverse impacts to human health from lead may result if the groundwater were to be used for potable purposes.

5.2.2.2.3 The risk and HI results indicate that groundwater at Area G may pose adverse health effects or risks to future workers based on the exposure information evaluated.

5.2.3 AREA R

5.2.3.1 Current Land Use

No completed exposure pathways are evident for persons working near Area R. As the sewage lift station located adjacent to the study area is not situated on the rubble area, persons working in the building on an intermittent basis or maintaining the area around the building are not expected to contact study area chemicals. Therefore, this area was not evaluated for current exposure scenarios in the risk characterization.

5.2.3.2 Future Land Use

The future exposure scenario evaluated for Area R is for a worker who may be exposed to contaminants in surface soil and groundwater. Based on the exposure assumptions evaluated, the future potential cumulative cancer risks associated with exposure to groundwater and soil are 7×10^{-5} and 3×10^{-6}, respectively, both of which are below the upper permissible cumulative risk of 10^{-4} but exceed the lower limit of 10^{-6} (Tables 5.2-1 and 5.2-2). The groundwater risk is due primarily to ingestion of arsenic (51 percent) and bis(2-chloroethyl) ether (32 percent) and inhalation of chloroform (14 percent), while greater than 99 percent of the soil cancer risk is due to oral and dermal exposure to PAHs.

5.2.3.2.1 For noncarcinogenic effects, the cumulative HI associated with groundwater exposure, 1.3, exceeds the target HI of 1 due to ingestion of chromium (45 percent), cadmium (18 percent), arsenic (15 percent), barium (8 percent), bis(2-chloroethyl) ether (6 percent), and chloroform (3 percent) and inhalation of chloroform (4 percent) (Table 5.2-2). The HI associated with exposure to surface soil is 4×10^{-4}, which is below the target HI of 1.

5.2.3.2.2 Although no RfD or CSF has been established for lead, the groundwater concentrations can be compared to the EPA drinking water action level of 15 μg/L established under the SDWA. The groundwater exposure concentration of 50 μg/L indicates that the potential for adverse impacts to human health from lead may result if the groundwater were to be used for potable purposes.

5.2.3.2.3 The risk and HI results indicate that groundwater at Area R may pose adverse health effects or risks to future workers based on the exposure information evaluated.

5.2.4 AREAS S AND T

5.2.4.1 Current Land Use

The current exposure scenario that exists at Areas S and T is for a maintenance worker at Area T who may be exposed to contaminants in surface soil while cutting grass. Based on the exposure assumptions evaluated, the current potential cumulative cancer risk associated with exposure to soil is approximately 2×10^{-9}, which is below the lower limit of permissible cumulative risk of 10^{-6}. The cumulative HI for noncarcinogenic effects is 3×10^{-5}, which is well below the target HI of 1.

5.2.4.1.1 These results indicate that Area F does not pose adverse health effects or risks to current workers based on the exposure information evaluated. In addition, the risk and HI associated with current worker exposure may be overestimated as several subsurface samples [samples collected in soil borings designated as shallow (SB-A) at a depth of 2 to 4 ft] were included in the UCL_{95} calculations for surface soil. These samples were included based on the assumption that the concentrations observed within the sample are present at 2 ft and not dispersed throughout the 2- to 4-ft horizon.

5.2.4.2 Future Land Use

The future exposure scenario evaluated at Areas S and T is for a worker who may be exposed to contaminants in surface soil and groundwater. Based on the exposure assumptions evaluated, the future potential cumulative cancer risk associated with exposure to groundwater, 8×10^{-5}, exceeds the lowerbound acceptable risk limit of 10^{-6} (Table 5.2-1); however, this risk is below the cumulative permissible risk of 10^{-4} for a single medium. This groundwater risk is due to oral, inhalation, and dermal exposure to several VOCs, including vinyl chloride (92 percent), chloroform (3 percent), 1,2-dichloroethane (3 percent), and benzene (2 percent). Future worker exposure to soil at Areas S and T may result in an increased cancer risk of 4×10^{-9}, which is below the lowerbound acceptable risk limit of 10^{-6}

5.2.4.2.1 For noncarcinogenic effects, the cumulative HI associated with exposure to groundwater is 2, which exceeds the target HI of 1 (Table 5.2-1). This HI is due primarily to inhalation of benzene (55 percent) and chlorobenzene (34 percent), as well as ingestion of chlorobenzene (4 percent), vinyl chloride (2 percent), and cadmium (1 percent) (Table 5.2-3).

The cumulative HI associated with future worker exposure to soil is 6×10^{-5}, which is well below the target HI of 1.

5.2.4.2.2 In addition, the exposure concentration for lead, 21.6 μg/L, exceeds the EPA drinking water action level of 15 μg/L, indicating that the potential for adverse health effects may exist if the groundwater is used for potable purposes.

5.2.4.2.3 These future risk and HI results indicate that groundwater at Areas S and T may pose adverse health effects or risks to future workers based on the exposure assumptions evaluated (i.e., ingesting 1 L/day of water).

5.3 METHODS FOR ECOLOGICAL RISK CHARACTERIZATION

Methods are only now being developed to quantify risks to aquatic and terrestrial ecosystems. Variability in population dynamics and other parameters is often great even under natural conditions. Data on study area ecological systems are seldom sufficient to perform a comprehensive analysis; however, comparisons of potential chemical intakes to ecotoxicological benchmarks can be achieved. Ecologists then evaluate the results to estimate the potential for adverse effects to the natural systems present at the site. As stated in Section 3.3.3, one important difference between ecological and human health evaluations is that the emphasis in nonhuman systems is placed on populations, communities, and ecosystems (unless the study area evaluation must include one or more rare or endangered species). Therefore, the potential effects predicted for individuals must be extrapolated to populations and interacting populations to evaluate the potential for measurable adverse impacts to the ecosystem.

5.3.1 To evaluate potential risks to ecological receptors, the chemical intake for a particular indicator species (see Section 3.3.3) is compared to a chemical-specific adjusted benchmark derived for that species (see Section 4.4). The ratio of chemical intake to adjusted benchmark is known as the ecotoxicity quotient (EQ) and is calculated as follows:

$$EQ = \frac{\textit{Chemical Intake}}{\textit{Adjusted Benchmark}}$$

5.3.2 EQs less than 1 suggest that the benchmark effect is unlikely to occur in the individual; EQs greater than 1 require further evaluation. Although these EQs may indicate some potential for adverse effects to individuals, at this point, the potential for adverse effects to populations or ecosystems is qualified. Although the EQ method does not provide an estimate of uncertainty and is not an estimation of risk, it is commonly used for screening the potential for ecological effects from exposure to hazardous chemicals (EPA, 1988a).

5.3.3 The appropriate media considered for these assessments are surface water, for evaluation of aquatic receptors; and surficial soil for evaluation of terrestrial receptors. Sediment exposure and bioaccumulation are evaluated qualitatively.

5.3.4 For this assessment, exposure concentrations as calculated for human exposure (Appendix B) were also used for evaluating ecological exposure. To evaluate potential impacts to benthic (sediment-dwelling) organisms, aquatic benchmark values were compared to sediment pore water concentrations as estimated from sediment concentrations and from either: (1) organic partition coefficients (Kds) for inorganic contaminants, or (2) K_{oc} / organic carbon fraction (f_{oc}) for organic chemicals (Table 5.4-2).

The formula used to estimate porewater concentrations was:

$$C_{porewater} = \frac{C_{sediment}}{Kd} \text{ or } \frac{C_{sediment}}{K_{OC} * foc} \qquad (5\text{-}1)$$

5.3.5 For the initial screening, EQs are calculated without regard for receptor home ranges; therefore, the initial EQs are conservative because they assume that the receptor is continuously in a contaminated area. Comparisons considering intermittent exposure due to the movement of animals through contaminated areas will reduce the EQs.

5.4 STUDY-AREA-SPECIFIC ECOLOGICAL RISK CHARACTERIZATION

The calculated EQs for terrestrial organisms are presented in Table 5.4-1. The calculated EQs for aquatic organisms are presented in Table 5.4-2. A comparison of study area surface water concentrations to AWQC is presented in Table 5.4-3.

5.4.1 AREA F

5.4.1.1 Terrestrial

The terrestrial risk characterization for the indicator species at Area F indicate that none of the COPCs exceed an EQ of 1. EQs less than 1 suggest that the benchmark effect is unlikely to occur to the individual under the exposure assumptions evaluated. EQs for COPCs that may bioaccumulate are significantly less than one. Since this pathway is expected to be small relative to the one estimated, no adverse effects are anticipated.

5.4.1.2 Aquatic

All of the EQs for sediment- and water-dwelling aquatic organisms at Area F are less than one, which indicates that there is low potential for adverse effects to these aquatic organisms. EQs for COPCs that may bioaccumulate are significantly less than one. Since this pathway is expected to be small relative to the one estimated, no adverse effects are anticipated. No AWQC are available for barium, the only COPC detected in surface water.

5.4.2 AREA G

5.4.2.1 Terrestrial

The terrestrial risk characterization for the indicator species at Area G indicate that none of the COPCs exceed an EQ of 1. EQs that are less than 1 suggest that the benchmark effect is unlikely to occur to the individual under the exposure assumptions evaluated. The COPCs present in soils do not represent a significant bioaccumulation potential.

5.4.2.2 Aquatic

The aquatic risk characterization for the indicator species at Area G indicate that none of the COPCs exceed an EQ of 1 for sediment or surface water. In addition, no exceedance of available AWQCs was observed.

5.4.3 AREA R

5.4.3.1 Terrestrial

The terrestrial risk characterization for the indicator species at Area R indicate that none of the COPCs exceed an EQ of 1. EQs that are less than 1 suggest that the benchmark effect is unlikely to occur to the individual under the exposure assumptions evaluated. EQs for

COPCs that may bioaccumulate are significantly less than one. Since this pathway is expected to be small relative to the one estimated, no adverse effects are anticipated.

5.4.3.2 Aquatic

The aquatic risk characterization for the indicator species at Area R indicates that none of the COPCs exceed an EQ of 1 for surface water or sediment. EQs that are less than 1 suggest that the benchmark effect is unlikely to occur to the individual under the exposure assumptions evaluated.

5.4.4 AREAS S AND T

5.4.4.1 Terrestrial

The terrestrial risk characterization for the indicator species at Areas S and T indicate that none of the COPCs exceed an EQ of 1. EQs that are less than 1 suggest that the benchmark effect is unlikely to occur to the individual under the exposure assumptions evaluated. EQs for COPCs that may bioaccumulate are significantly less than one. Since this pathway is expected to be small relative to the one estimated, no adverse effects are anticipated.

5.4.4.2 Aquatic

Substantial amounts of water may pool at Areas S and T during periods of heavy precipitation; however, surface water is not typically present. An aquatic risk characterization was not performed as wetland species are not present in this area.

5.5 SUMMARY OF UNCERTAINTIES ASSOCIATED WITH THE BRA

The risk measurements used in Superfund study area RAs are not full probability estimates of risk but are conditional estimates given a set of assumptions about exposure and toxicity. Therefore, it is important to fully specify the assumptions and uncertainties inherent in the RA to place the risk estimates in proper perspective (EPA, 1989). Uncertainty analysis is also essential for an FS.

5.5.0.1 A qualitative uncertainty analysis of each RA component is sufficient for most study areas [RAGS (EPA, 1989)]. Table 5.6-1 presents the potential uncertainties inherent in the

RA process. A study-area-specific discussion of these individual components is summarized in the following sections.

5.5.1 UNCERTAINTIES ASSOCIATED WITH HAZARD IDENTIFICATION

5.5.1.1 Uncertainties Associated with Data Collection

Groundwater is the only media for which an unacceptable risk level occurs in Areas F, G, R, and S/T. The purpose of this section is to identify uncertainties in the groundwater data collected during the RFI. Selection and design of the appropriate remedial alternatives to address these unacceptable risks will be performed during the FS and remedial design (RD) phase. The RD process will require adequate data characterization for the following:

1. Horizontal and vertical extent of contamination;
2. Groundwater flow direction and gradients; and
3. Hydrogeologic characteristics of the multi-zone aquifer system and aquifer parameters such as transmissivity, specific yield, and storage coefficient.

5.5.1.1.1 To fulfill these data requirements, additional data collection will be performed during the RD process.

5.5.1.1.2 The following sections address specific groundwater data uncertainties for each of the five study areas and identify the anticipated impact these uncertainties may have on the calculated risk levels.

Area F

5.5.1.1.3 An unacceptable risk level is caused by elevated levels of arsenic, carbon tetrachloride, and mercury in site groundwater. The three water bearing zones at Area F are the shallow (perched), the overburden, and the bedrock (deep) aquifers. Some uncertainty remains in establishing the direction of groundwater flow in both the overburden and bedrock aquifers at the site. The regional direction of groundwater flow in the overburden zone is south, toward the Tennessee River, however the RFI determined that seasonal conditions in the overburden zone may create a northeasterly direction of flow during some periods. In addition, the RFI findings noted that the groundwater flow direction in the bedrock zone could not yet be determined. Therefore, data collection during the RD phase will confirm the groundwater flow direction at the Area F.

5.5.1.1.4 The horizontal extent of contamination in the shallow aquifer will also be a focus during RD data collection efforts. Only one of the 12 wells installed at Area F is a shallow aquifer well. This well is located immediately north of Area F and arsenic was the only COPC detected above acceptable risk levels. Conclusions about the extent and distribution of arsenic in the shallow aquifer cannot be made from a single data point. The impact of this data uncertainty on the calculated risks at Area F is the potential for an undercalculation of risk. It is possible that higher concentrations of arsenic may be present in the shallow aquifer downgradient of the site, therefore, this data uncertainty may cause an underestimation in the calculated risk at Area F.

5.5.1.1.5 The overburden aquifer has been also been impacted by past activities at the area. The highest concentrations of all three COPCs were detected in the overburden aquifer at Area F. The RFI has determined that the extent of contamination has been defined on the east, southwest, and west sides of the site by low levels of arsenic and carbon tetrachloride (G&M, 1992a). Additional data collected during the RD may include definition of the extent of contamination on the south and southeast sides of the site, where it is currently undefined. As stated previously, for the case of the shallow aquifer, the impact of this data uncertainty on the calculated risks at Area F is the potential for an undercalculation of risk. Undefined extent of contamination data on the presumed downgradient side of the site may underestimate the actual COPC concentrations used in the risk calculation and thus underestimate the risk.

Area G

5.5.1.1.6 Elevated levels of barium, cadmium, lead, and chromium are present in groundwater at Area G that pose an unacceptable risk level. The same 3-layer aquifer system described for Area F is also present at Area G. Water levels in the shallow and overburden aquifer indicate an easterly groundwater flow direction toward the creek (G&M, 1992a). The RFI also noted that the groundwater flow in the bedrock zone was not determined. Data collection during RD may involve additional groundwater level measurements to confirm RFI findings for the shallow and overburden zones.

5.5.1.1.7 The shallow aquifer data for Area G consists of three wells, all located in close proximity on the eastern side of the site. Only one of these three wells had COPC concentrations at levels that pose an unacceptable risk. The data collected during the RD

phase may include further delineation of the extent of contamination in both the shallow and overburden aquifers.

5.5.1.1.8 Four of the five overburden wells had COPC concentrations at levels that pose an unacceptable risk. While the highest concentrations were found in the overburden well near the center of Area G, the most easterly (downgradient) overburden well had cadmium and chromium levels in the unacceptable risk range. The downgradient extent of contamination for these metals will be defined during the RD phase and may include collection of additional data north, south, and west of the property where there are currently no data points. The impact of this data uncertainty on the calculated groundwater risks at Area G is the potential for an undercalculation of risks. Incomplete extent of contamination data may underestimate the actual COPC concentrations used in the risk calculation and thus underestimate the risk.

Area R

5.5.1.1.9 Elevated groundwater concentrations of cadmium and chromium at Area R pose an unacceptable risk level. One water-bearing zone, the overburden aquifer, was investigated at Area R during the RFI. The groundwater flow direction in the overburden zone is reported in the RI to be easterly. Data collection efforts during the RD phase will include confirmation of the reported groundwater flow direction in the overburden aquifer.

5.5.1.1.10 Three of the six overburden wells at Area R had cadmium and chromium concentrations above an acceptable risk level. While the highest concentrations were found in the overburden well near the center of Area R, the most easterly (downgradient) overburden well had cadmium and chromium levels in the unacceptable risk range. The downgradient extent of contamination will be defined during the RD phase and may include collection of additional data north, east, and west of the site. The impact of this data uncertainty on the calculated groundwater risks at Area R is the potential for an undercalculation of risks. Incomplete extent of contamination data may underestimate the actual COPC concentrations used in the risk calculation and thus underestimate the risk.

Areas S and T

5.5.1.1.11 An unacceptable risk level exists in groundwater at Areas S and T due to elevated levels of benzene, vinyl chloride, lead and chlorobenzene. Three water-bearing zones were

investigated during the RFI including the shallow (perched), overburden, and bedrock aquifers.

5.5.1.1.12 Some uncertainty remains in determining the groundwater flow direction in the overburden and bedrock zones. The regional groundwater flow direction in the overburden zone is southerly, toward the Tennessee River, however the RFI determined that seasonal conditions in the overburden zone may create a northerly direction of flow during some periods (G&M, 1992a). In addition, the groundwater flow direction in the bedrock zone exhibited seasonal reversal. Therefore, data collection during the RD phase will confirm the groundwater flow direction at this area.

5.5.1.1.13 The higher levels of chlorobenzene and benzene at Areas S and T are in the shallow perched water-bearing zone. The RFI determined that the extent of the contamination in the perched zone remains undefined on the west side of Areas S and T (G&M, 1992a). Also, the extent of contamination in the deeper zone remains to be completely defined by additional data (G&M, 1992a). Therefore, data collection during the RD phase will focus on these data uncertainties. The overall impact of these uncertainties in the actual extent and distribution of groundwater contamination is a potential underestimation of the actual risks because higher COPC concentrations may be present at the site, but are not included in the current risk calculation.

5.5.1.2 <u>Uncertainties Associated with Data Evaluation</u>

As with most NPL sites, the data used for this BRA were collected over time and by different contractors using different QA/QC procedures outlined in regulator-approved work plans. This accounts for the variation in the analytical methods used and detection limits reported. The air sampling collected during test pit excavations were included for evaluation of potential inhalation exposure to vapors. The uncertainty associated with the vapors released during the excavations is high, as the field screening measurements are not compound specific.

5.5.1.2.1 Automation of the data evaluation reduces associated uncertainty as detection limits are automatically halved and the comparison of the UCL_{95} to the maximum concentration is automated to reduce human error in making these numerous comparisons.

5.5.1.2.2 There is a possibility that a COPC or a toxic metabolite was not identified through the sampling and analytical process or that the results are not an accurate representation of the concentrations that occur onsite. However, this uncertainty is greatly reduced by including all positively detected organic chemicals as COPCs in the exposure and risk analysis, as is the case for this evaluation. Another potential source of uncertainty associated with the data set used in this evaluation is that the sampling was conducted over different time periods by different contractors using different approved work plans. Such samples were analyzed using different analytical methods and detection limits, which contribute to the uncertainty in the chemicals detected and their concentrations.

5.5.1.2.3 Another source of uncertainty resulting in overestimates of risk is due to extraneous contamination introduced during sampling or analysis. For example, bis(2-ethylhexyl)phthalate, acetone, and methylene chloride were frequently detected and were included as COPCs, although these compounds could be laboratory artifacts and not study area-related.

5.5.2 UNCERTAINTY ASSOCIATED WITH EXPOSURE ASSESSMENT

5.5.2.1 Human Exposure Uncertainties

The applied exposure concentration estimates may result in overestimates of risk, if contaminant levels at receptor points are decreasing with time. The exposure scenarios developed in Section 3.0 are based on information available from previous reports and from a site visit conducted by ESE in May and June of 1994. The surface water-use exposure scenarios developed in the exposure and risk analysis for Area G incorporated a variety of exposure routes based on visual observations as well as historical meteorological information. Some of the exposure scenarios are currently not complete, but were included as conservative estimates of future exposure such as an 8-hour work day at all five areas to include use of groundwater for potable purposes (i.e., ingestion and washing).

5.5.2.1.1 Considerable uncertainty is associated with the quantification of exposure. The exposure factors used most frequently are those default assumptions from EPA sources. When necessary, study-area-specific information is incorporated to reduce the uncertainty. However, for air contaminant concentration and fish contaminant concentration estimations, the parameters used were conservative estimates. For instance, methods to estimate inhalation

exposure to volatiles, suspended particulates, or dust may overestimate intake and risk. In addition, the contaminants in fish are assumed to be distributed equally throughout the whole body. Also, the realistic intake concentrations from the consumption of fish will be lower than the estimated values due to cooking. The monitored lipid content in fish is well below the assumed or modeled fish lipid content, which is important for BCF calculations.

5.5.2.1.2 Reduced exposure by any or all routes would be expected if the future worker was not spending the entire exposure time at the contaminated portion of the area and did not use the groundwater at that area for potable purposes. As described in the exposure assessment (Section 3.0), potentially exposed populations at Area G include RSA personnel and their families, as well as other persons using the surface water near the study area. Certain individuals may be more sensitive to the adverse effects of contaminant exposure because of poor health, age, or other factors. The contaminants of interest in this area, however, are not known to specifically impact certain segments of the population, such as young children. The degree of conservatism in the assumptions and analyses for surface-water-use pathways is anticipated to reasonably protect potential receptor populations.

5.5.2.1.3 Exposure concentrations used for groundwater are based on unfiltered groundwater samples, as per EPA regionwide (1989) and Region IV (1991) guidance. However, risks to future groundwater users would be much lower if exposure was based on filtered groundwater. For example, the dissolved metals concentrations at Areas G and R were significantly lower than the total metals concentrations. This indicates that future groundwater users could be protected from an unacceptable risk simply by filtering the groundwater prior to use.

5.5.2.1.4 As mentioned in Section 3.3.1.4, the exposure concentrations used in this BRA were adopted from the normal distribution-based UCL_{95}s as calculated for the RFI under RCRA. Table 5.5-1 provides an example of the impact of using normal distribution-based UCL_{95} equations instead of lognormal distribution-based UCL_{95} equations for groundwater exposure at Area F. If the lognormal distribution-based UCL_{95} value exceeded the maximum concentration, the maximum concentration was used in calculating percent differences from the normal distribution-based UCL_{95} as calculated by G&M.

5.5.2.1.4.1 As seen in Table 5.5-1, the largest observed lognormal or maximum to normal ratios were for arsenic and mercury at 2.74 and 4.29, respectively. Note that these two metals were the only constituents from the groundwater at Area F where the lognormal distribution-based UCL_{95} equation produced a value which exceeded the maximum concentrations.

5.5.2.1.4.2 Of the ten analytes' concentration data, only arsenic fits the lognormal distribution. However, the lognormal distribution was a better fit than the normal distribution in all cases except for cadmium.

5.5.2.1.4.3 Table 5.5-2 provides intake and reasonable maximum exposure hazard index (RMEHI) values for arsenic and mercury. The RMEHI for oral exposure to arsenic was 4.7E+0 using the maximum concentration compared to 1.3E+0 using the normal distribution-based UCL_{95} equation. The RMEHI for dermal ingestion of arsenic was 1.7E-2 using the maximum concentration compared to 4.8E-3 using the normal distribution-based UCL_{95} equation. The RMEHI for oral exposure to mercury was 4.6E+1 using the maximum concentration compared to 1.1E+1 using the normal distribution-based UCL_{95} equation. The RMEHI for oral ingestion of mercury was 1.7E-1 using the maximum concentration compared to 3.9E-2 using the normal distribution-based UCL_{95} equation. The differences between RMEHI values are the same order of magnitude for oral exposure and one order of magnitude for dermal exposure. Since the most extreme examples for groundwater at Area F result in no more than one order of magnitude difference, it is likely that the use of the normal distribution-based UCL_{95} equation over the lognormal distribution-based UCL_{95} equation did not have a great impact on the outcome of this risk assessment.

5.5.2.2 Ecological Exposure Uncertainties

Because the actual exposure for ecological receptors is unknown, scenarios are developed to estimate exposure doses that can then be compared with ecological benchmark concentrations or doses. Ecological benchmark concentrations are literature-derived values for the lowest concentration or dose of chemical causing an ecologically interpretable negative response to a test organism. Ecologically interpretable negative responses may be either acute or chronic. These responses include lowered reproductive rates, lowered survival rates, etc.

5.5.2.2.1 For surface water, the assumptions are that the organism is exposed to the surface water exposure concentration for a length of time corresponding to the benchmark test conditions. Exposure is unlikely to be continuous for all organisms for several reasons as follows:

1. Many aquatic organisms can swim into and out of contaminated zones,
2. Exposure concentrations will fluctuate with changing water levels and other factors, and
3. Many aquatic organisms such as chironomids spend only the larval stage of their life cycles in an aquatic habitat.

5.5.2.2.2 Assuming continuous exposure may overestimate the potential for adverse effects. However, this scenario does not estimate potential uptake from sediments, pore water, or prey items. The majority of the study area contaminants, except mercury, do not strongly bioaccumulate; therefore, uptake from food or incidental ingestion of sediments is not likely to be a major pathway (except possibly for benthic organisms). In addition, this pathway is not considered significant, as the home range for most animals at the study area is much greater than the area of contamination. Thus, an organism is not expected to spend 100 percent of its time on the contaminated area.

5.5.2.2.3 The uncertainty involved with estimating the amount of soil in the diet is high, but the exposure factors are designed to over, rather than underestimate the intake of contaminants. The exposure scenarios developed for soil exposure to terrestrial organisms, which assume certain percentages of soil in the diet, are derived primarily from EPA guidance (EPA, 1993).

5.5.3 UNCERTAINTY ASSOCIATED WITH TOXICITY ASSESSMENT

5.5.3.1 Uncertainty Associated with the Human Toxicity Assessment

A majority of the uncertainty in an RA is associated with the use of dose-response data that have been generated under experimental laboratory conditions (using non-human mammals) and extrapolating these results for comparison to (i.e., RfD) human exposure under a different environmental exposure scenario. To extrapolate the experimental evidence from animals to humans, a series of uncertainty factors and modifying factors, which have been derived by

EPA, are applied. These uncertainty factors and modifying factors are the quantitative uncertainty associated with the value in question.

5.5.3.1.1 As with the noncarcinogenic dose-response assessment, the carcinogenic dose-response assessment includes the following:

1. Selection of the appropriate data sets;
2. Derivation of estimates at low doses from experimental data at high doses, using an appropriate extrapolation model (extrapolation is ordinarily carried out first by fitting a mathematical model to the observed data and then by extending the model from the observed range down toward risks expected at low exposure) (IRIS, 1994);
3. Selection of an equivalent human dose when animal data sets are used;
4. Introduction of additional assumptions, with corresponding additional uncertainties, for route-to-route extrapolation when only one route has been tested in animals or evaluated in humans.

5.5.3.1.2 The level of confidence associated with the CSFs from EPA can be obtained from the literature from which the dose-response studies for the carcinogenic COCs were obtained. The uncertainty factors associated with each toxicity factor used for the RA are included in Section 4.0.

5.5.3.1.3 There is considerable uncertainty in estimates of toxicity values. Critical toxicity values are subject to change as new evidence becomes available. This may result in overestimates or underestimates of risk. For some chemical classes (i.e., PAHs), in the absence of toxicity values, the CSF or RfD of a highly toxic class member (i.e., benzo(a)pyrene for cancer risks) is commonly adopted. This approach may overestimate risks.

5.5.3.2 <u>Uncertainty Associated with the Ecological Toxicity Assessment</u>

The derivation of an ecological benchmark for different chemicals and different indicator species was done using laboratory toxicity data that were available in the literature. To address the considerable amount of uncertainty associated with inter- and intraspecies extrapolation, several uncertainty factors have been applied. This approach may overestimate

risks, however, the approach allows for the quantitation of ecological risks for many species which do not have benchmarks. These uncertainty factors are the quantitative uncertainty associated with the value in question.

5.5.3.2.1 The preferred value that was sought for use as an ecological benchmark was a chronic LOAEL dose. An uncertainty factor of 10 was applied to all values that were extrapolated from one species to another species.

5.5.3.2.2 When there was no chronic LOAEL value available for a chemical in the literature, other values such as LD_{50}s were used to derive a safe dose. An uncertainty factor of 10 was used to account for the extrapolation from an acute study to a chronic effect.

5.5.4 UNCERTAINTY ASSOCIATED WITH RISK CHARACTERIZATION

The uncertainties of the risk characterization include the uncertainties associated with the previous three steps of the RA process. In most cases, the uncertainties are more than compensated for by inclusion of upperbound exposure concentrations, upperbound exposure factors (Section 3.0), uncertainty factors and modifying factors in developing RfDs and CSFs (Section 4.0), and incorporating conservative assumptions in estimated future risks by assuming that contaminant degradation does not occur (Section 3.2.2.2). Incorporation of the factors and variables to account for uncertainty in each step of the RA process presents a reasonable upperbound estimate of the risks and impacts scenario on which to calculate risks. This procedure ensures the protection of public health, because if the upperbound risk estimate represents an acceptable risk, then there is a high level of confidence that an adverse impact will not occur. Most of the cumulative carcinogenic risk estimated for each of the study areas is attributable to Category B2 (probable human) carcinogens. Thus, it is not known whether these chemicals actually cause cancer in humans, particularly at the low intake levels estimated for the study area.

Table 5.2-1. Summary of Human Health Risks and HIs for Areas F, G, R, and S/T (Page 1 of 2)

Area	Scenario	Medium	Exposure Route	Hazard Index Adult	Hazard Index Child	Risk
F	Current Worker	Soil	Ingestion	< 0.1	NA	4E-06
			Dermal	< 0.1	NA	2E-06
			Inhalation	< 0.1	NA	3E-08
			TOTAL	< 0.1	NA	5E-06
	Future Worker	Groundwater	Ingestion	12	NA	3E-04
			Dermal	< 0.1	NA	1E-06
			Inhalation	0.3	NA	7E-06
			Subtotal	13	NA	3E-04
		Soil	Ingestion	< 0.1	NA	4E-06
			Dermal	< 0.1	NA	2E-06
			Inhalation	< 0.1	NA	3E-08
			Subtotal	< 0.1	NA	5E-06
			TOTAL	13	NA	3E-04
G	Current Recreational	Fish	Ingestion	< 0.1	< 0.1	9E-09
			Subtotal	< 0.1	< 0.1	9E-09
		Sediment	Ingestion	< 0.1	< 0.1	4E-10
			Dermal	< 0.1	< 0.1	4E-11
			Subtotal	< 0.1	< 0.1	5E-10
		Surface Water	Ingestion	< 0.1	< 0.1	1E-08
			Dermal	< 0.1	< 0.1	3E-08
			Subtotal	< 0.1	< 0.1	4E-08
			TOTAL	< 0.1	< 0.1	5E-08
	Future Worker	Groundwater	Ingestion	1.8	NA	5E-05
			Dermal	< 0.1	NA	2E-07
			Inhalation	--	NA	--
			Subtotal	1.8	NA	5E-05
		Sediment	Ingestion	< 0.1	NA	2E-10
			Dermal	< 0.1	NA	9E-12
			Subtotal	< 0.1	NA	2E-10
		Soil	Ingestion	< 0.1	NA	2E-11
			Dermal	< 0.1	NA	8E-12
			Inhalation	< 0.1	NA	4E-14
			Subtotal	< 0.1	NA	2E-11
		Surface Water	Ingestion	< 0.1	NA	6E-10
			Dermal	< 0.1	NA	2E-09
			Subtotal	< 0.1	NA	2E-09
			TOTAL	1.8	NA	5E-05

Table 5.2-1. Summary of Human Health Risks and HIs for Areas F, G, R, and S/T (Continued, Page 2 of 2)

Area	Scenario	Medium	Exposure Route	Hazard Index Adult	Hazard Index Child	Risk
R	Future Worker	Groundwater	Ingestion	1.3	NA	6E-05
			Dermal	< 0.1	NA	4E-07
			Inhalation	< 0.1	NA	1E-05
			Subtotal	1.3	NA	7E-05
		Soil	Ingestion	< 0.1	NA	2E-06
			Dermal	< 0.1	NA	1E-06
			Inhalation	< 0.1	NA	2E-08
			Subtotal	< 0.1	NA	3E-06
			TOTAL	1.3	NA	8E-05
S/T	Current Worker	Soil	Ingestion	< 0.1	NA	2E-09
			Dermal	< 0.1	NA	8E-11
			Inhalation	< 0.1	NA	7E-13
			TOTAL	< 0.1	NA	2E-09
	Future Worker	Groundwater	Ingestion	0.59	NA	6E-05
			Dermal	< 0.1	NA	2E-06
			Inhalation	1.4	NA	2E-05
			Subtotal	2	NA	8E-05
		Soil	Ingestion	< 0.1	NA	2E-09
			Dermal	< 0.1	NA	1E-09
			Inhalation	< 0.1	NA	2E-11
			Subtotal	< 0.1	NA	4E-09
			TOTAL	2	NA	8E-05

Note: NA = not applicable.
-- = no volatiles detected in groundwater.

Source: ESE.

Table 5.2-2. Summary of Media and COPCs Exceeding Risks* Based on the Human Risk Characterization

Area	Scenario	Medium	Exposure Route	COPC	Risk
F	Future Worker	Soil	Ingestion	Benzo(a)pyrene	2E-06
				Dibenz(a,h)anthracene	1E-06
		TOTAL FOR SOIL			3E-06
		Groundwater	Ingestion	Arsenic	3E-04
				Carbon tetrachloride	4E-06
				Subtotal	3E-04
			Inhalation	Chloroform	4E-06
				Carbon tetrachloride	3E-06
				Subtotal	7E-06
		TOTAL FOR GROUNDWATER			3E-04
	TOTAL FOR GROUNDWATER AND SOIL				ERR
G	Future Worker	Groundwater	Ingestion	Arsenic	5E-05
		TOTAL FOR GROUNDWATER			5E-05
R	Future Worker	Soil	Ingestion	Benzo(a)pyrene	1E-06
		TOTAL FOR SOIL			1E-06
		Groundwater	Ingestion	Arsenic	4E-05
				Bis(2-chloroethyl) ether	2E-05
				Subtotal	6E-05
			Inhalation	Chloroform	1E-05
				Subtotal	1E-05
		TOTAL FOR GROUNDWATER			7E-05
	TOTAL FOR GROUNDWATER AND SOIL				7E-05
S/T	Future Worker	Groundwater	Ingestion	Vinyl chloride	6E-05
				Subtotal	6E-05
			Inhalation	Vinyl chloride	2E-05
				Chloroform	3E-06
				1,2-Dichloroethane	2E-06
				Benzene	1E-06
				Subtotal	3E-05
			Dermal	Vinyl chloride	2E-06
				Subtotal	2E-06
	TOTAL FOR GROUNDWATER				9E-05

*Chemicals are listed if an individual chemical exceeds 1E-06 risk within a medium.

Source: ESE.

Table 5.4-2. Ecotoxicity Quotients (EQs) for Potential Aquatic Indicator Species (Page 1 of 2)

Receptor	Chemical	Exposure Concentration (ppm)*	Estimated Porewater Conc. (mg/L)	(Kd) or (Koc / foc)	Benchmark		EQ
					Value*	Source	
Area F -- Surface Water							
Daphnia magna	Barium	3.21E-02	--	--	5.00E+01 (a)	EPA, 1986	6.42E-04
Rana clamitans	Barium	3.21E-02	--	--	5.00E+01 (a)	EPA, 1986	6.42E-04
Scenedesmus sp.	Barium	3.21E-02	--	--	5.00E+01 (a)	EPA, 1986	6.42E-04
Area F -- Sediment							
Chironomus sp.	Acetone	5.25E-02	3.50E+02 (b)	1.50E-04	NA		
	Benz(a)anthracene	3.29E-01	1.65E-04 (b)	2.00E+03	NA		
	Benzo(a)pyrene	3.40E-01	6.18E-06 (b)	5.50E+04	NA		
	Benzo(b)fluoranthene	3.03E-01	5.51E-05 (b)	5.50E+03	NA		
	Carbon tetrachloride	3.00E-03	2.73E-03 (b)	1.10E+00	3.52E+01 (c)	IRIS, 1994	7.75E-05
	Chrysene	3.25E-01	1.63E-04 (b)	2.00E+03	NA		
	Fluoranthene	2.92E-01	7.68E-04 (b)	3.80E+02	3.98E+00	IRIS, 1994	1.93E-04
	Methylene chloride	2.26E-02	2.57E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	2.33E-02
	Phenanthrene	3.63E-01	2.59E-03 (b)	1.40E+02	6.30E-03	IRIS, 1994	4.12E-01
	Pyrene	3.03E-01	7.97E-04 (b)	3.80E+02	NA		
	Arsenic	4.05E-02	2.03E-04 (d)	2.00E+02	1.90E-01	40 CFR 141,142	1.07E-03
	Barium	2.33E-01	3.88E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	7.77E-05
	Cadmium	6.80E-04	1.05E-04 (d)	6.50E+00	6.60E-01 (e)	EPA, 1986	1.59E-04
	Chromium	1.05E-01	1.24E-04 (d)	8.50E+02	2.10E-01	40 CFR 141,142	5.88E-04
	Lead	1.07E-01	1.19E-04 (d)	9.00E+02	2.58E-01 (f)	EPA, 1980c	4.61E-04
	Mercury	3.98E-03	3.98E-04 (d)	1.00E+01	2.00E-02 (g)	Rehwoldt et al., 1973	1.99E-02
Area G -- Surface Water							
Daphnia magna	Acetone	1.59E-02	--	--	1.00E-01 (h)	Verscheuren, 1983	1.59E-01
	1,1-Dichloroethane	2.90E-03	--	--	NA		
	Methylene chloride	5.70E-03	--	--	2.24E+02 (i)	EPA, 1982	2.54E-05
	Trichloroethene	1.59E-02	--	--	6.40E+01 (j)	Canton and Adema, 1978	2.48E-04
	Xylenes	3.50E-03	--	--	1.00E+00 (h)	Verscheuren, 1983	3.50E-03
	Barium	8.07E-02	--	--	5.00E+01 (a)	EPA, 1986	1.61E-03
Rana clamitans	Acetone	1.59E-02	--	--	2.40E+00 (i,n)	HazardText, 1995	6.63E-03
	1,1-Dichloroethane	2.90E-03	--	--	2.02E+00 (o,t)	HazardText, 1995	1.44E-03
	Methylene chloride	5.70E-03	--	--	2.94E+00 (o,u)	HazardText, 1995	1.94E-03
	Trichloroethene	1.59E-02	--	--	4.50E+00 (i,n)	HazardText, 1995	3.53E-03
	Xylenes	3.50E-03	--	--	1.69E-01 (p,v)	HazardText, 1995	2.07E-02
	Barium	8.07E-02	--	--	5.00E+01 (a)	EPA, 1986	1.61E-03
Scenedesmus sp.	Acetone	1.59E-02	--	--	NA		
	1,1-Dichloroethane	2.90E-03	--	--	NA		
	Methylene chloride	5.70E-03	--	--	1.10E+01 (c)	IRIS, 1994	5.18E-04
	Trichloroethene	1.59E-02	--	--	2.19E+01 (a)	EPA, 1986	7.26E-04
	Xylenes	3.50E-03	--	--	NA		
	Barium	8.07E-02	--	--	5.00E+01 (a)	EPA, 1986	1.61E-03
Area G -- Sediment							
Chironomus sp.	Acetone	1.13E-01	7.53E+02 (b)	1.50E-04	NA		
	Methylene chloride	2.12E-02	2.41E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	2.19E-02
	Arsenic	4.25E-03	2.13E-05 (d)	2.00E+02	1.90E-01	40 CFR 141,142	1.12E-04
	Barium	7.23E-02	1.21E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	2.41E-05
	Chromium	5.44E-02	6.40E-05 (d)	8.50E+02	2.10E-01	40 CFR 141,142	3.05E-04
Area R -- Surface Water							
Daphnia magna	Bis(2-ethylhexyl) phthalate	1.69E-02	--	--	5.00E+00 (i)	EPA, 1980b	3.38E-03
	Chloroform	1.75E-02	--	--	2.89E+01 (k)	EPA, 1980 (AWQC)	6.06E-04
	Arsenic	1.05E-02	--	--	9.12E-01	EPA, 1980a	1.15E-02
	Barium	1.30E-01	--	--	5.00E+01 (a)	EPA, 1986	2.60E-03
Rana clamitans	Bis(2-ethylhexyl) phthalate	8.07E-02	--	--	2.00E-01 (q)	Wams, 1987	4.04E-01
	Chloroform		--	--	3.51E+00 (r)	Birge et al., 1980	0.00E+00
	Arsenic	0.00E+00	--	--	4.00E+01 (s)	USFWS, 1988	0.00E+00
	Barium	0.00E+00	--	--	5.00E+01 (a)	EPA, 1986	0.00E+00
Scenedesmus sp.	Bis(2-ethylhexyl) phthalate	1.69E-02	--	--	3.60E-01 (l)	IRIS, 1994	4.69E-02
	Chloroform	1.75E-02	--	--	1.24E+00 (m)	IRIS, 1994	1.41E-02
	Arsenic	1.05E-02	--	--	2.32E+00	EPA, 1980a	4.53E-03
	Barium	1.30E-01	--	--	5.00E+01 (a)	EPA, 1986	2.60E-03

Table 5.4-2. Ecotoxicity Quotients (EQs) for Potential Aquatic Indicator Species (Continued, Page 2 of 2)

Receptor	Chemical	Exposure Concentration (ppm)*	Estimated Porewater Conc. (mg/L)	(Kd) or (Koc / foc)	Benchmark		EQ
					Value*	Source	
Area R -- Sediment							
Chironomus sp.	Acetone	3.34E-02	2.23E+02 (b)	1.50E-04	NA		
	Benz(a)anthracene	7.75E-01	3.88E-04 (b)	2.00E+03	NA		
	Benzo(a)pyrene	8.80E-01	1.60E-05 (b)	5.50E+04	NA		
	Benzo(b)fluoranthene	1.81E+00	3.29E-04 (b)	5.50E+03	NA		
	Benzo(ghi)perylene	9.20E-01	5.75E-05 (b)	1.60E+04	NA		
	Bis(2-ethylhexyl) phthalate	7.85E-01	2.14E-03 (b)	3.66E+02	1.80E+01 (i)	EPA, 1980b	1.19E-04
	Carbon tetrachloride	1.07E-02	9.73E-03 (b)	1.10E+00	3.52E+01 (c)	IRIS, 1994	2.76E-04
	Chrysene	8.26E-01	4.13E-04 (b)	2.00E+03	NA		
	1,1-Dichloroethane	1.67E-02	2.88E-02 (b)	5.80E-01	NA		
	Fluoranthene	5.11E-01	1.34E-03 (b)	3.80E+02	3.98E+00	IRIS, 1994	3.38E-04
	Indeno(1,2,3-cd)pyrene	9.26E-01	5.79E-05 (b)	1.60E+04	NA		
	Methylene chloride	3.76E-02	4.27E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	3.88E-02
	Pyrene	7.02E-01	1.85E-03 (b)	3.80E+02	NA		
	Arsenic	1.77E-02	8.85E-05 (d)	2.00E+02	1.90E-01	40 CFR 141,142	4.66E-04
	Barium	1.41E-01	2.35E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	4.70E-05
	Cadmium	6.12E-03	9.42E-04 (d)	6.50E+00	6.60E-01 (e)	EPA, 1986	1.43E-03
	Chromium	3.90E-02	4.59E-05 (d)	8.50E+02	2.10E-01	40 CFR 141,142	2.18E-04
	Lead	5.90E-02	6.56E-05 (d)	9.00E+02	2.58E-01 (f)	EPA, 1980c	2.54E-04
	Mercury	1.07E-03	1.07E-04 (d)	1.00E+01	2.00E-02 (g)	Rehwoldt et al., 1973	5.35E-03

Note:
NA = benchmark not located in available literature.
conc. = concentration.
-- = not applicable.
sp. = species.
mg/kg = milligram per kilogram.
ppm = part per million.
mg/L = milligram per liter.
Kd = organic partition coefficient.
Koc = organic carbon partition coefficient.
foc = soil organic carbon fraction.

(a) Value available in EPA's Quality Criteria for Water (1986), but not a criteria value.
(b) Porewater conc. = Sediment conc. / (Koc * foc). foc of 0.01 is used for site soils (Rosenblatt and Small, 1981).
(c) Acute lowest effect concentration (LEC).
(d) Porewater conc. = Sediment conc. / Kd (Tetra Tech, 1986).
(e) Hardness-dependent value for freshwater organisms based on 50 mg/L calcium carbonate ($CaCO_3$).
(f) 10-day LC50 (concentration lethal to 50 percent of organisms), hardness = 47 mg/L.
(g) Acute LC50/EC50 for mercuric nitrate, as mercury. EC50 is the effective concentration for 50% of organisms.
(h) 24-hour median tolerance limit (TLm).
(i) 48-hour LC50.
(j) Species acute value.
(k) 48-hour static bioassay.
(l) Chronic ambient water quality criteria (AWQC).
(m) Chronic LEC.
(n) Adjusted value for clawed toad.
(o) Adjusted value for the guppy.
(p) Adjusted value for the goldfish.
(q) Adjusted value for Xenopus laevis (toad).
(r) Adjusted value for toads/frogs.
(s) Adjusted value for narrow-mouthed toad.
(t) 7-day LC50.
(u) 14-day LC50.
(v) 96-hour LC50.

* Surface water and porewater concentrations are in mg/L; sediment concentrations are in mg/kg.

Source: ESE.

Table 5.5-1. Comparison of Normal and Lognormal Distribution-Based UCL95s for Groundwater at Area F

Constituent	Normal UCL95 (b)	Lognormal UCL95 (c)	Maximum Conc'n (d)	Smaller of Lognormal UCL95 and Maximum (e)	Diff. (f)	Ratio of Lognormal or Maximum to Normal (g)	Normal Prob<W (h)	Lognormal Prob<W (i)	Distribution	Distribution with Better Fit
Volatile Organics (mg/L) (a)										
Acetone	3.21E-02	4.70E-02	8.40E-02	4.70E-02 (UCL95)	1.49E-02	1.46	0.0001	0.0001	Unknown	Lognormal
Chloroform	5.96E-03	5.49E-03	1.60E-02	5.49E-03 (UCL95)	-4.65E-04	0.92	0.0001	0.0001	Unknown	Lognormal
Carbon tetrachloride	7.97E-03	8.71E-03	1.70E-02	8.71E-03 (UCL95)	7.39E-04	1.09	0.0002	0.0023	Unknown	Lognormal
Trichloroethene	3.29E-03	3.28E-03	5.00E-03	3.28E-03 (UCL95)	-1.35E-05	1.00	0.0001	0.0001	Unknown	Lognormal
Metals (Total, mg/L)										
Arsenic	4.02E-02	1.43E-01	1.10E-01	1.10E-01 (Maximum)	6.98E-02	2.74	0.0004	0.0865*	Lognormal	Lognormal
Barium	9.13E-02	8.56E-02	3.20E-01	8.56E-02 (UCL95)	-5.70E-03	0.94	0.0001	0.0051	Unknown	Lognormal
Cadmium	3.69E-03	3.47E-03	7.60E-03	3.47E-03 (UCL95)	-2.18E-04	0.94	0.0001	0.0001	Unknown	Same
Chromium	6.86E-03	7.19E-03	1.61E-02	7.19E-03 (UCL95)	3.29E-04	1.05	0.0001	0.0004	Unknown	Lognormal
Lead	1.28E-02	1.00E-02	5.24E-02	1.00E-02 (UCL95)	-2.72E-03	0.79	0.0001	0.0001	Unknown	Lognormal
Mercury	3.26E-01	3.53E+00	1.40E+00	1.40E+00 (Maximum)	1.07E+00	4.29	0.0001	0.0001	Unknown	Lognormal

Notes:

(a) mg/L = milligrams per liter

(b) Normal UCL95 is the upper 95% confidence limit of the arithmetic mean for a normal distribution

(c) Lognormal UCL95 is the upper 95% confidence limit of the arithmetic mean for a lognormal distribution

(d) Maximum concentration is listed because the maximum concentration should be used as the exposure concentration if the UCL95 is greater than the maximum concentration

(e) Maximum concentration should be used as the exposure concentration if the UCL95 is greater than the maximum concentration

(f) *If lognormal UCL95 is less than the maximum concentration, then Difference = Lognormal UCL95 - Normal UCL95.*
If lognormal UCL95 is greater than the maximum concentration, then Difference = Lognormal UCL95 - Maximum Concentration.

(g) If lognormal UCL95 is less than the maximum concentration, then Ratio = Lognormal UCL95 / Normal UCL95.
If lognormal UCL95 is greater than the maximum concentration, then Ratio = Maximum Concentration / Normal UCL95.

(h) The Shapiro-Wilks test (aka W-test) statistic was used to determine if the nontransformed data come from a normal distribution.
Values greater than or equal to 0.05 indicate that the data come from a normal distribution (note that none of the concentrations are normally distributed)

(i) The W-test statistic was used to determine if the natural logarithm-transformed data come from a normal distribution.
Values greater than or equal to 0.05 indicate that the data come from a lognormal distribution (note that only arsenic concentrations are lognormally distributed)

Sources: G&M, 1991 and 1992; ESE, 1995.

Table 5.5-2. Intakes and RMEHIs for the Two Largest Observed Differences in UCL95s for Groundwater at Area F

Analyte	Pathway	Normal Distribution-Based UCL_{95} Equation		Maximum Concentration (Less Than Lognormal Distribution-Based UCL_{95} Equation)	
		Intake	RMEHI	Intake	RMEHI
Arsenic	Oral	3.9E-4	1.3E+0	1.4E-3	4.7E+0
	Dermal	1.4E-6	4.8E-3	5.1E-6	1.7E-2
Mercury	Oral	3.2E-3	1.1E+1	1.4E-2	4.6E+1
	Dermal	1.2E-5	3.9E-2	5.0E-5	1.7E-1

Table 5.6-1. Uncertainty Associated with the RA Process

BRA Component	Potential for Uncertainty
Hazard Identification	. Initial selection of COPCs . Tentatively identified compounds . Limited chemical monitoring data . Extraneous contamination introduced during sampling or analysis . Use of inappropriate analytical methods or method detection limits
Exposure Assessment	. Selection of site-specific exposure pathways . Estimation of exposure concentrations with limited monitoring data . Estimation of exposure to multiple substances . Estimation of inhalation exposure to volatiles, suspended dust . Estimation of exposure factors and parameters . Use of current data for future exposure conditions . Use of normal distribution-based UCL_{95} equation instead of lognormal distribution-based UCL_{95} equation
Toxicity Assessment	. Selection of toxicity values . Factors used to derive toxicity values including interspecies extrapolation . Weight of evidence for human toxicity . Derivation of carcinogenic slope factors . Extrapolation of less-than-lifetime exposure to lifetime cancer risks . Interaction of multiple substances . Use of high to low dose extrapolation models
Risk Characterization	. Addition of risks across multiple exposure pathways . Addition of risks from multiple substances . For PAHs, in the absence of CSFs for most of the PAHs, the CSF of a highly toxic PAH is adopted for all PAHs in the class

Source: ESE.

6.0 REMEDIAL GOAL OPTIONS (RGOs)

The purpose of the BRA for RSA Areas F, G, R, and S/T was to assess the potential human health and the ecological impacts associated with the study-area-related chemicals under current and future land-use conditions, assuming that no remedial action is occurring at the study area. Those chemicals that pose unacceptable health risks in the baseline RA also referred to as the final chemicals of concern (COCs), are further evaluated in the FS to evaluate a reasonable range of remedial alternatives for migration control and source control measures to reduce study area contaminants to acceptable levels. Thus, RA input is required in the FS to ensure that the overall remediation goal for the study area, which is to protect human health and the environment by preventing or reducing contaminant release or migration by implementing appropriate remedial actions, is achieved.

6.0.1 The Superfund Amendments and Reauthorization Act (SARA) requires that remedial actions attain a degree of contaminant cleanup that ensures protection of public health and the environment. Thus, in the event that the baseline RA of a study area indicates the need to reduce study area COCs to acceptable health-based levels, RGOs must be developed. RGOs are chemical-specific concentration goals for individual chemicals for specific medium and land use combinations at CERCLA study areas (EPA, 1991b/RAGS B). There are two sources of RGOs: (1) concentrations based on applicable and relevant or appropriate requirements (ARARs), and (2) concentrations based on risk assessment (risk-based concentrations under specific exposure conditions) (EPA, 1991b/RAGs B).

6.0.2 A chemical-specific ARAR may not always be protective if the chemical exists in a mixture; in addition, chemical-specific ARARs have not been established for many chemicals, particularly for chemicals detected in soil. Thus, in the absence of an ARAR or, in instances where an ARAR is not protective, RGOs can be developed based on the BRA to ensure that they meet the threshold criteria of (1) protection of human health and the environment, and, (2) compliance with ARARs (EPA, 1991b/RAGS B).

6.1 DEVELOPMENT OF RGOs

RGOs for RSA were established based on the most current EPA guidance on the role of the baseline RA in the remedy selection process (EPA, 1991C), as well as guidance regarding the

development of RGOs (EPA, 1991/RAGS B; Region IV Supplemental Guidance, 1994). As stated in EPA guidance (EPA, 1991C), action is generally not warranted at a Superfund study area if the following occurs:

1. The cumulative carcinogenic study area risk to an individual based on reasonable maximum exposure for both current and future land use is less than 10^{-4},
2. The noncarcinogenic hazard quotient is less than 1,
3. No adverse environmental impacts exist, and
4. ARARs (i.e., MCLs, non-zero MCLGs) are not exceeded.

6.1.0.1 RGOs may not be required for a COC in the event that the above four criteria are met. This is consistent with EPA guidance on development of Preliminary Remedial Goals (PRGs) (EPA, 1991\RAGS B), which states that RGOs must be maintained for contaminants in a medium when any of the previously listed criteria are exceeded. According to EPA Region IV Supplemental RAGS guidance, RGOs should be identified/developed for those chemicals addressed in the BRA that meet the following criteria:

1. Contribute to a pathway that exceeds a 10^{-4} risk (or whatever risk level is chosen as the remediation "trigger" by the risk manager), or
2. A HI of 1 or greater, or
3. A COPC exceeds a state of federal chemical-specific ARAR.

6.1.0.2 According to EPA Region IV Supplemental guidance (EPA, 1994), chemicals need not be included for RGO development if their individual carcinogenic risk contribution to a pathway is less than 10^{-6} or their noncarcinogenic HQ is less than 0.1.

6.1.0.3 Thus, based on EPA risk assessment guidance and the results of the BRA, a list of media cleanup levels was established for each COC in each land-use scenario that met the following combined region-wide and region-specific criteria:

1. Contributes to a pathway that exceeds a 10^{-4} risk,
2. The individual HQ contribution to a pathway is greater than 0.1 for a pathway that results in a cumulative HI greater than 1,
3. An EQ exceeds 1, and
4. A COPC exceeds a state or federal chemical-specific ARAR.

6.1.0.4 Except for future worker groundwater exposure at Area F, all cumulative risks at the study area are within the permissible cumulative risk range of 1×10^{-6} to 1×10^{-4}. In addition, a cumulative HI of 1 was exceeded at all five areas. Therefore, RGOs were developed for groundwater for all five areas.

6.1.1 RGOs FOR GROUNDWATER

The results of the risk characterization at RSA Areas F, G, R, and S/T indicate that cumulative risks associated with future worker RME potential exposures to groundwater at these areas are within the EPA cumulative risk range of 1×10^{-6} to 1×10^{-4} for all areas except Area F. Area F cumulative risk associated with exposure to groundwater is 2.6×10^{-4}, which slightly exceeds 1×10^{-4}. These risk results suggest that remediation for the study area-related contaminants may not be required at Area G, R, and S/T, as the cumulative risk of 1×10^{-4} is not exceeded for this medium. However, because this cumulative risk level has not yet been selected by the risk manager, all chemicals contributing to an individual risk of 1×10^{-6} within a pathway were included in the RGO table for risk management decision making. The noncarcinogenic HQ and HI analysis indicates that all five study areas result in cumulative HIs that exceed the target HI of 1. Thus, RGOs were developed for those compounds contributing 0.1 or greater to the HI.

6.1.1.1 Development of RGOs for Individual Groundwater Contaminants Exceeding 1×10^{-6} Risk or an HI of 1

The RGOs for potential carcinogens were developed by combining the receptor intake levels to each chemical posing a risk greater than 1×10^{-6} for a pathway contributing a risk in excess of 1×10^{-4} or an HQ greater than 0.1. The RGOs were developed from all appropriate routes of exposure, (i.e., inhalation, ingestion, and dermal exposure) within a pathway and by rearranging the study-area-specific chemical intake equations used in the exposure assessment (see Appendix C) to solve for the concentration term.

6.1.1.1.1 Because the dermal exposure pathway did not contribute significantly to the overall groundwater risk, the two primary exposure routes considered when developing RGOs for groundwater at RSA included incidental ingestion and inhalation exposure. These two pathways were evaluated as a single algorithm to develop a RGO that is protective for all of the potential significant pathways of exposure to groundwater. RGOs for potential

carcinogenic and noncarcinogenic effects were calculated using the following algorithms, Equation 2 and Equation 3, respectively.

$$C_{gw} = \frac{TR \times BW \times AT_{carc} \times 365\ days/year}{EF \times ED \times [(CSF_o \times IR_{gw}) + (CSF_i \times IR_{as} \times FC_{as} \times ET_{as})]} \qquad (6\text{-}1)$$

where:

- TR = target risk (unitless).
- BW = body weight (kg).
- AT_{carc} = averaging time for carcinogenic exposures (years).
- EF = exposure frequency (days/year).
- ED = exposure duration (years).
- IR_{gw} = intake rate for groundwater (L/day).
- CSF_o = oral cancer slope factor [(mg/kg/day)$^{-1}$].
- CSF_i = inhalation cancer slope factor [(mg/kg/day)$^{-1}$]
- IR_{as} = intake rate for shower air (m^3/hour).
- FC_{as} = factor to convert groundwater concentration to shower air concentration (L/m^3).
- ET_{as} = exposure time for shower air (hours/day).

Equation 3 for noncarcinogenic effects:

$$C_{gw} = \frac{THI \times BW \times AT_{noncarc} \times 365\ days/year}{(ED \times EF) \times [(1/RfD_o \times IR_{gw}) + (1/RfD_i \times FC_{as} \times IR_{as} \times ET_{as})]} \qquad (6\text{-}2)$$

where:

- THI = target risk (unitless).
- BW = body weight (kg).
- $AT_{noncarc}$ = averaging time for noncarcinogenic exposures (years).
- EF = exposure frequency (days/year).
- ED = exposure duration (years).
- IR_{gw} = intake rate for groundwater (L/day).
- RfD_o = oral reference dose (mg/kg/day).
- RfD_i = inhalation reference dose (mg/kg/day).
- IR_{as} = intake rate for shower air (m^3/hour).

FC_{sa} = factor to convert groundwater concentration to shower air concentration (L/m^3).

ET_{sa} = exposure time for shower air (hours/day).

6.1.1.1.2 The list of the exposure factors used to calculate chemical intakes and the sources used to develop the exposure factors are presented in Appendix C. Table 6.1-1 lists the RGOs for groundwater based on potential carcinogenic and noncarcinogenic risk results along with federal MCLs, if available. A summary of the exposure concentrations and RGOs are presented in Table 6.1-2. As shown in this table, the majority of groundwater risk is driven by noncarcinogenic effects.

6.1.2 RGOs BASED ON ECOLOGICAL RISKS

The EQ analysis indicates none of the aquatic and terrestrial indicator species evaluated at each study area resulted in EQs exceeding 1. Thus, RGOs were not developed based on ecological exposure.

Table 6.1-1. Preliminary RGOs Associated with Future Worker Oral and Inhalation Exposure to Contaminants in Groundwater at Areas F, G, R, and S/T

Constituent	Applicable Sites	Carcinogenic RGOs (mg/L)									Noncarcinogenic RGOs (mg/L)									MCL
		Oral			Inhalation			Oral + Inhalation			Oral			Inhalation			Oral + Inhalation			
		1E-06	1E-05	1E-04	1E-06	1E-05	1E-04	1E-06	1E-05	1E-04	0.1	1	10	0.1	1	10	0.1	1	10	
Potential Carcinogens																				
Arsenic	F, G, R	2E-04	2E-03	2E-02	na	na	na	2E-04	2E-03	2E-02	3.1E-03	3.1E-02	3.07E-01	na	na	na	3.1E-03	3.1E-02	3.1E-01	5.0E-02 (a)
Benzene	S/T	1E-02	1E-01	1E+00	5E-03	5E-02	5E-01	3E-03	3E-02	3E-01	3.1E-03	3.1E-02	3.07E-01	2.7E-04	2.7E-03	2.7E-02	2.5E-04	2.5E-03	2.5E-02	5.0E-03
Carbon tetrachloride	F	2E-03	2E-02	2E-01	2E-03	2E-02	2E-01	1E-03	1E-02	1E-01	7.2E-03	7.2E-02	7.15E-01	nd	nd	nd	7.2E-03	7.2E-02	7.2E-01	5.0E-03
Chloroform	F	5E-02	5E-01	5E+00	2E-03	2E-02	2E-01	2E-03	2E-02	2E-01	1.0E-01	1.0E+00	1.02E+01	nd	nd	nd	1.0E-01	1.0E+00	1.0E+01	1.0E-01 (b)
Lead	G, R, S/T	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.5E-02 (c)
Noncarcinogens																				
Barium	G	--	--	--	--	--	--	--	--	--	7.2E-01	7.2E+00	7.15E+01	na	na	na	7.2E-01	7.2E+00	7.2E+01	2.0E+00
Cadmium	G, R	--	--	--	--	--	--	--	--	--	5.1E-03	5.1E-02	5.11E-01	na	na	na	5.1E-03	5.1E-02	5.1E-01	5.0E-03
Chlorobenzene	S/T	--	--	--	--	--	--	--	--	--	2.0E-01	2.0E+00	2.04E+01	2.7E-02	2.7E-01	2.7E+00	2.4E-02	2.4E-01	2.4E+00	1.0E-01
Chromium	G, R	--	--	--	--	--	--	--	--	--	5.1E-02	5.1E-01	5.11E+00	na	na	na	5.1E-02	5.1E-01	5.1E+00	1.0E-01 (d)
Mercury	F	--	--	--	--	--	--	--	--	--	3.1E-03	3.1E-02	3.07E-01	na	na	na	3.1E-03	3.1E-02	3.1E-01	2.0E-03

Note: na = inhalation of metals from groundwater is not applicable.
nd = no inhalation CSF and/or RfD determined.

(a) National Interim Primary Drinking Water Regulation.
(b) Value for total trihalomethanes.
(c) EPA drinking water action level.
(d) Value based on total chromium.

Source: ESE.

7.0 SUMMARY AND CONCLUSIONS

7.1 OBJECTIVES OF THE BRA

The purpose of this BRA is to determine the human health and ecological risks associated with the no-action alternative at Areas F, G, R, and S/T, to determine which areas, media, and contaminants require further evaluation in the FS. Specific objectives of the process were to:

1. Provide an analysis of baseline risks to assist in determining the need for action at these areas,
2. Provide a basis for recommendations of RGOs.
3. Provide a consistent method for evaluating and documenting human health or ecological threats at these areas.

7.2 ORGANIZATION OF THE RISK ASSESSMENT REPORT

This BRA was conducted in accordance with RAGS, Vols. I and II (EPA, 1989, EPA 1991a); RAGS updates and supplements, as well as current Region IV RAGS, in the following five phases:

1. Hazard identification/selection of COPCs,
2. Exposure assessment,
3. Toxicity assessment,
4. Risk characterization, and
5. Development of RGOs.

7.2.0.1 Identification of COPCs involves analyzing the site monitoring data by focusing on human health and the environmental impact of the chemical(s) under consideration. This step requires development of a data set to be carried through the quantitative risk evaluation (Section 2.0). The exposure assessment is conducted to estimate the magnitude, frequency, and the pathways of actual and/or potential exposure to human and nonhuman receptor populations (Section 3.0). The toxicity assessment (Section 4.0) summarizes the available human health and ecological impacts data as described in published literature, and identifies the dose-response information to be used to calculate health risks. The last step in the BRA, risk characterization (Section 5.0), integrates the information obtained in the exposure and

toxicity assessment sections to determine the human and ecological health risks associated with potential exposure to site-related contaminants. In addition, a summary of the uncertainties associated with each component of the BRA is presented in Section 5.0. At the completion of risk characterization, the chemicals and media that pose unacceptable risks are identified and health-based remediation goals, also referred to as RGOs are developed (Section 6.0). The purpose of RGOs is to provide the FS with preliminary goals to be met by the recommended remedial alternative.

7.2.1 OVERVIEW OF HAZARD IDENTIFICATION

The hazard identification identified the primary site-related COPCs at the study areas. Based on past site operations and disposal activities at the five areas of concern, the COPCs evaluated in this BRA included a subset of inorganics (excluding a number of metals because they were representative of background conditions), volatile organics, semivolatile organics, and several pesticides. While EPA risk assessment guidance allows for a reduction in the number of chemicals that must undergo complete evaluation in the risk assessment (i.e., focus the risk assessment on those chemicals that are most toxic), computers enable the risk assessor to completely evaluate all pertinent data. Thus, the only data screening that was performed for this BRA for the inclusion of all contaminants that were thought to be site-related, and contaminants that were detected at levels significantly above background. The COPCs for the study areas were identified based on the results of the chemical selection and screening activities and included the following:

VOCs

- Acetone
- Benzene
- Carbon tetrachloride
- Chlorobenzene
- Chloroform
- Dichloroethane, 1,1-
- Dichloroethane, 1,2-
- Dichloroethenes, 1,2-,total
- Ethylbenzene
- Methylene Chloride
- Tetrachloroethene
- Trichloroethene
- Vinyl chloride
- Xylenes, total

SVOCs

Miscellaneous SVOCs

- Bis(2-chloroethyl)ether
- Bis(2-ethylhexyl)phthalate
- Butlybenzyl phthalate

PAHs

Acenapthene
Anthracene
Benz(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(ghi)perylene
Chrysene
Fluoranthene
Flourene
Indeno(1,2,3-cd)pyrene
Phenanthrene
Pyrene

Pesticides

DDE
DDT

Inorganic Chemicals

Arsenic
Barium
Cadmium
Chromium
Lead
Mercury
Selenium

7.2.2 OVERVIEW OF THE EXPOSURE ASSESSMENT

An exposure pathway describes the course a chemical takes from the source to the exposed receptor. An exposure pathway analysis links the sources, locations, and types of releases with population locations and physical activity patterns to determine the significant exposure pathways of human and nonhuman exposure.

7.2.2.1 The primary human exposure pathways quantified in the BRA for different study areas are as follows:

Exposure Pathway	Medium	Area
Current Recreational	Surface water/Sediment	G
	Fish ingestion	G
Current Worker	Soil	F, S and T
Future Worker	Soil and Groundwater	F, G, R, S and T
	Surface water/Sediment	G

7.2.2.2 Current onsite worker exposure assumes that a worker may incidentally come into direct contact with soil. Soil contaminants can be incidentally ingested, absorbed through the skin or inhaled in the form of suspended dust. Current recreational exposure assumes that an

individual may incidentally come into direct contact with surface water and sediment while fishing near Area G. In addition, if fish are caught, risks are evaluated for ingestion of fish caught from this area.

7.2.2.3 Future onsite worker exposure assumes that a worker may incidentally come into direct contact with soil, surface water, and sediment. Soil contaminants can be incidentally ingested, absorbed through the skin or inhaled in the form of suspended dust. Groundwater is evaluated in the event that groundwater under the area is used for potable purposes.

7.2.2.4 While additional human exposure pathways exist, they were not included for quantification (i.e., future recreational activities at all of the study areas). Potential risks due to these pathways are indirectly evaluated in the future worker scenario where exposure duration is much greater.

7.2.2.5 The primary ecological exposure pathways quantified in the BRA for different study areas are as follows:

Exposure Pathway	Medium	Area
Aquatic Exposure (freshwater fish and aquatic invertebrates)	Surface water/Sediment	G,R
Terrestrial Exposure (biota, birds, and mammals)	Soil	F, G, R, S/T

7.2.3 OVERVIEW OF TOXICITY ASSESSMENT

The toxicity assessment summarizes and weighs the available evidence for the potential of a COPC to cause adverse effects in exposed individuals (humans and ecological receptors) and to provide, where possible, an estimate of the relationship between the extent of exposure to a chemical and the increased likelihood and/or severity of adverse effects. The toxicity assessment involves developing profiles for each COPC and summarizes the available toxicological information to include human health and ecological effects, as well as summarize criteria and standards. The human health and ecological health risks from potential exposure to site contaminants is assessed based on the available toxicity information.

7.2.4 RISK CHARACTERIZATION

7.2.4.1 Human Risk Characterization

The risk characterization process defines the nature and degree of adverse human or ecological health effects that may occur from exposure to site-related contaminants under the conditions described in the exposure assessment. In addition, the risk characterization attempts to identify the contaminant source areas associated with the most significant risk so that remedial actions can be targeted in the most cost-effective manner to reduce health risks to acceptable levels. Health risks are determined by integrating the information developed in the three previous steps. For potential human exposures, the potential for noncarcinogenic hazards and carcinogenic risks are calculated separately for each exposure route (i.e., ingestion, dermal absorption) and are reported either as a ratio noncarcinogenic HI or as a probability (cancer risks).

7.2.4.1.1 The site-specific carcinogenic risk estimates are determined using the UCL_{95} concentrations and the exposure factors presented in Section 3.0. The potential risks resulting from exposure to the site contaminants are compared with the EPA target cumulative risk levels. Acceptable exposure levels are the contaminant concentration levels that present an excess cancer risk of 10^{-6} (1 in 1,000,000) to 10^{-4} (1 in 10,000) to the exposed population, based on the dose-response information for each carcinogenic COPC, (NCP 40 CFR 300, 430:62). When a cumulative carcinogenic risk within a medium (i.e., groundwater) exceeds 10^{-4} (1 in 10,000 excess cancer risk), CERCLA generally requires remedial action at the site (EPA, 1991a). If the cumulative risk is less than 10^{-4}, action generally is not required but may be warranted if a chemical-specific standard that is risk-based (e.g., MCL) is violated. A risk-based remedial decision could be superseded by the presence of noncarcinogenic impact or environmental impact requiring action at the site. Therefore, based on the latest EPA Superfund guidance (EPA, 1991a), the cumulative risks are compared to 10^{-4}, while the cumulative noncarcinogenic HI are compared to the target HI of 1.

7.2.4.1.2 To calculate the total risk to a receptor, risks were added across the pathways if the same individual was assumed to be exposed via multiple pathways (e.g., a person working with the soil onsite could be exposed via ingestion, dermal, and inhalation pathways).

7.2.4.1.3 The results of the human BRA indicate that the following scenarios exceed either a cumulative risk of 10^{-4} or an HI of 1:

Area	Medium	Exposure Pathway	Risk	COPC
F	Groundwater	Future worker	1×10^{-4}	Arsenic, carbon tetrachloride, chloroform
			HI > 1	Arsenic, mercury, carbon tetrachloride
G	Groundwater	Future worker	HI > 1	Arsenic, barium, cadmium, chromium
R	Groundwater	Future worker	HI > 1	Arsenic, cadmium, chromium
S/T	Groundwater	Future worker	HI > 1	Benzene, chlorobenzene

In addition, the MCL for lead was exceeded at Areas G, R, and S/T.

7.2.4.2 Ecological Risk Characterization

Methods are still being developed to quantify risks to aquatic and terrestrial ecosystems. Variability in population dynamics and other parameters is often great even under natural conditions. Sufficient data on a site's ecosystem(s) are seldom available for comprehensive analysis. However, comparisons of ecotoxicological benchmarks and potential exposure concentrations resulting in EQs can be calculated to screen for potential problems (much like HIs are calculated for human exposure). One important difference between ecological and human health evaluations is that the emphasis is placed on populations, communities, and ecosystems in nonhuman systems (unless the site evaluation must include one or more rare or endangered species). Therefore, the potential effects predicted for individuals must then be extrapolated to populations and communities to evaluate the potential for measurable adverse impacts to the ecosystem.

7.2.4.2.1 Toxicity benchmarks for terrestrial wildlife were compared directly to the RME exposure concentrations at each study area, as were benchmarks for aquatic receptors. Concentrations of contaminants in the sediments were converted to porewater concentrations via standard equilibrium partitioning methods. Porewater concentrations were then compared

to toxicity information for chironomids, when available, or to ambient water quality criteria for the protection of freshwater aquatic life when chironomid data were unavailable.

7.2.4.2.2 EQs that are less than one suggest that there is a low probability of an adverse effect occurring to the individual receptor. EQs greater than one indicate that there is some probability of an adverse effect occurring to the individual receptor. The higher the EQ, the greater the probability of an adverse effect. The results of the risk analysis indicate that none of the EQs exceed 1, nor do the surface water concentrations exceed AWQCs, indicating that Areas F, G, R, and S/T do not pose unacceptable risks to terrestrial or aquatic receptors based on the exposure assumptions evaluated.

7.2.5 RGOs

The SARA requires that remedial actions attain a degree of contaminant cleanup that ensures the protection of public health and the environment. Thus, the risk characterization results are used to identify whether site COPCs need to be reduced to acceptable health-based levels. The acceptable health-based levels are referred to as RGOs, which are chemical-specific concentration goals for individual chemicals for specific medium and reasonable land use combinations.

7.2.5.1 Based on the results of the risk characterization, all five areas, Areas F, G, R, and S/T resulted in excess risk and/or HI based on the future worker exposure scenario to groundwater, thus, this scenario was the basis for developing RGOs at these areas. In summary, the RGOs are developed for the following chemicals to provide risk managers with the maximum risk-related media level options on which to develop remediation aspects of the FS:

Area	Medium	COPCs Requiring RGO Development	
F	Groundwater	Organics:	Carbon tetrachloride, chloroform
		Inorganics:	Arsenic, mercury
G	Groundwater	Inorganics:	Arsenic, barium, chromium, cadmium, lead
R	Groundwater	Inorganics:	Arsenic, cadmium, chromium, lead
S/T	Groundwater	Organics:	Benzene, chlorobenzene
		Inorganics:	Lead

REFERENCES

Advanced Sciences, Inc. 1992. Preliminary Site Inspection for Redstone Arsenal. Site Inspection Report No. 91031. Final. January 1992. Contract No. DAAA15-90-D-0001, Task 9. Oak Ridge, TN.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989a. Toxicological Profile for DDT, DDE, and DDD. Prepared by Clement Associates. U.S. Public Health Service, Atlanta, GA. NTIS No. PB90-182171.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989b. Toxicological Profile for 1,2-Dichloroethane. Prepared by Clement Associates. U.S. Public Health Service, Atlanta, GA. NTIS. No. PB90-171422.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989c. Toxicological Profile for Vinyl Chloride. Prepared by Syracuse Research Corporation. U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/25. NTIS. No. PB90-103870.

Andelman, J.B. and Suess, M.J. 1970. Polynuclear Aromatic Hydrocarbons in the Water Environment. Bull. World Health Org. *43*: 479-508.

Bechert, W.F., Moghissi, A.A., Au, F.H.F., Bretthauer, E.W. and McFarlane, J.C. 1974. Formation of Methyl-mercury in a Terrestrial Environment. Nature. 249: 674-675.

Brunker, R.L. and Bott, T.L. 1974. Reduction of Mercury to the Elemental State by a Yeast. Applied Microbiol. *27*: 870-73.

Bumpus, J.A. and Aust., S.D. 1987. Biodegradation of DDT [1,1,1-Trichloro-2,2 bis (4-chlorophenyl) ethene] by the White Pot Fungus *Phanerochaete chrysosporium*. Applied Env. Microbiol. *53*(9): 2001-2008.

Callahan, M.A., Slimark, M.S., Gabel, N.W., May, I.P., Fowler, C.F., Freed, J.R., Jennings, P., Durfee, R.L., Whitmore, F.C., Maestri, B., Mabey, W.R., Holt, B.R. and Gould, C. 1979. Water-related Environmental Fate of 129 Priority Pollutants. U.S. EPA, Washington, DC.

Canton, H. and D.M. Adema. 1978. Reproducibility of Short-term and Reproduction Toxicity Experiments with *Daphnia magna* and Comparison of the Sensitivity of *Daphnia magna* with *Daphnia pulex* and *Daphnia cucullata* in Short-term Experiments. Hydrobiologia. 59:135.

Caras, R.A. 1967. North American Mammals. 1st Ed. Meredith Press, New York, NY.

Casarett and Doull, J. 1991. Casarett and Doull's Toxicology, the Basic Science of Poisons. Fourth Ed. Eds. M.O. Andur, J. Doull, and C.D. Klaassen. McGraw Hill, NY.

Chapman, P.J. 1972. An Outline of Reaction Sequences Used for the Bacterial Degradation of Synthetic Organic Molecules in the Biosphere: Natural, Pesticidal, and Various Other Man-Made Components. Nat'l. Acad. Sci., Washington, DC.

D'Itri, F.M. 1972. Biological Methylation of Mercury in the Environment. *In*: The Environmental Mercury Problem. CRC Press, Cleveland, OH.

Dagley, S. 1972. Microbial Degradation of Stable Chemical Structures. General Features of Metabolic Pathways. *In*: Degradation of Synthetic Organic Molecules in the Biosphere: Natural, Pesticidal, and Various Other Man-Made Compounds. Nat'l. Acad. Sci., Washington, DC.

Department of the Army. 1977. Installation Assessment of Redstone Arsenal, Alabama, Record Evaluation Report No. 118, December 1977.

Environmental Science & Engineering (ESE). 1992. Supplemental Remedial Investigation/Feasibility Study for Soils in Area A, Alabama Army Ammunition Plant, Final Baseline Risk Assessment. Contract No. DAAA15-90-D-0017.

Environmental Science & Engineering (ESE). 1993. Corrective Measure Studies for Redstone Arsenal, Alabama. Prepared for the U.S. Army Corps of Engineers (USACE), Huntsville Division. Contract No. DACA87-92-D-0018.

Fries, G.F. 1972. Degradation of Chlorinated Hydrocarbons Under Anaerobic Conditions. *In*: Fate of Organic Pesticides in the Aquatic Environment, Advance in Chemistry Series (R.F. Gould, ed.), American Chemical Society, Washington, DC.

Galli, R. and McCarty, P.L. 1989. Biotransformation of 1,1,1-Trichloroethane, Trichloromethane, and Tetrachloromethane by a *Clostridium sp*. Applied Env. Microbiol., *55*(4): 837-844.

Geraghty and Miller (G&M). 1991a. Phase I RCRA Facility Investigations at Unit 1, Unit 2, and Selected Unit 3 Areas. Redstone Arsenal, Alabama. Prepared for the U.S. Army Corps of Engineers, Huntsville Division. Contract No. DACA87-89-C-0075.

Geraghty and Miller (G&M). 1991b. Final Identification and Evaluation of Potential Solid Waste Management Units and Areas of Concern, Redstone Arsenal, Alabama, Prepared for U.S. Army Corps of Engineers, February 1991.

Geraghty and Miller (G&M). 1992a. Phase II Addendum RCRA Facility Investigations at Unit 1, Unit 2, and Selected Unit 3 Areas. Redstone Arsenal, Alabama. Prepared for the U.S. Army Corps of Engineers, Huntsville Division. Contract No. DACA87-89-C-0075.

Geraghty and Miller (G&M). 1992b. Quality Control Summary Report, Phase II RCRA Facility Investigations at Unit 1, Unit 2, and Selected Unit 3 Areas, Redstone Arsenal, Alabama. Report prepared for the U.S. Army Corps of Engineers, October 1992.

Gibson, D.T. 1976. Microbial Degradation of Polycyclic Aromatic Hydrocarbons. Proceedings of the Third International Biodegradation Symposium: 56-65.

Gibson, D.T. 1976. Microbial Degradation of Polycyclic Aromatic Hydrocarbons. As cited by Verschueren, K. (1983) Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Company, Inc., New York.

Hamdy, M.K. and Noyes, O.R. 1975. Formation of MethylMercury by Bacteria. Applied Microbiol. *30*:424-432.

Harland, Bartholomew & Associates, Inc. 1986. Analytical/Environmental Assessment Report, Redstone Arsenal, Alabama, May 1986.

HazardText(TM) Hazard Management. 1995. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Englewood, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Johnsgard, P.A. 1973. Grouse and Quails of North America. Univ. Nebraska Press, Lincoln, NE.

Johnson, B.T. and Lulves, W. 1975. Biodegrodation of Dibutylphthalate and Di-2-ethylhexylphthalate in Freshwater Hydrosoil. J. Fish Res. Blard Can. *32*(3): 333-337.

Kearney, P.C. and Kaufman, D.D. 1976. Herbicides: Chemistry, Degradation and Mode of Action. Marcel Dekker, Inc., New York. 1036p.

Kobayashi, H. and Rittman, B.E. 1982. Microbiol Removal of Hazardous Organic Compounds. Environ. Sci. Technol. *16*(3): 170A-183A.

LaMoreaux, P.E. and Associates, Inc. (PELA). 1988a. Confirmation Report, Unit 3 Investigations, Redstone Arsenal, Alabama. Volumes I through VI. Report Prepared for U.S. Army Corps of Engineers, July 1988.

LaMoreaux, P.E. and Associates, Inc. (PELA). 1988b. Remedial Investigation Engineering Report, Redstone Arsenal, Alabama, Unit 1 DDT and Sanitary Landfills and Unit 2 Open Burn/Open Demolition Area. Volumes I through VII. Consulting Report Prepared for U.S. Army Corps of Engineers, September 1988.

LaMoreaux, P.E. and Associates, Inc. (PELA). 1989. Upgrade Confirmation Report and Assessment of Remedial Alternatives for Selected Unit 3 Sites, Redstone Arsenal, Alabama. Volumes I through V. Report Prepared for U.S. Army Corps of Engineers, April 1989.

Leland, H.V., Bruce, W.M., and N.F. Shimp. 1973. Chlorinated Hydrocarbon Insecticides in Sediments in Southern Lake Michigan. Env. Sci. Technol. *7*(9): 833-838.

Mahaffey, W.R., Gibson, D.T., and Cerniglia, C.E. 1988. Bacterial Oxidation of Chemical Carcinogen: Formation of Polycyclic Aromatic Acids from Benz[a]anthracene. Applied Env. Microbiol. *54*(10). 2415-2423.

McBride, B.C. and Wolfe, R.S. 1971. Biosynthesis of Dimethylarsine by Methanobacterium. Biochemistry. *10*: 4312-4317.

McKenna, E.J. and Heath, R.D. 1976. Biodegradation of Polynuclear Aromatic Hydrocarbon Pollutants. Project No. A-073-I11. Univ. of Illinois at Urbana-Champaign.

National Academy of Sciences (NAS). 1972. Particulate Polycyclic Organic Matter. Washington, DC.

Neff, J.M. 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. Applied Science Publishers, London.

Olin Chemicals Group, Environmental Affairs Department. 1983. Huntsville Groundwater Report, Redstone Arsenal, Huntsville, Alabama. November 1983.

Olin Chemicals Group, Environmental Affairs Department. 1987. DDT Investigation, Huntsville, Alabama. Semi-Annual Report Number 3. September 1, 1987.

Olin Chemicals Group - Environmental Affairs Department. 1989. Huntsville Spring Branch - Indian Creek, Long-Term Monitoring Program, Annual Report No. 1, April 15, 1989.

Orenshaw, D.C. 1993, Personal Communication, Memorandum of July 13, 1993 to Georgette Myers, Environmental Management Division, Letterkenny Army Depot, re: EPA Region III Comments on the Final Draft of the Letterkenny Baseline Risk Assessment for the Southeast Area. Remedial Project Manager, U.S. Environmental Protection Agency, Region III.

Parsons, F. and Lage, G.B. 1985. Chlorinated Organics in Simulated Groundwater Environments. Journal of the American Water Works Association, 77:52-59.

Perwak, J., Byrne, M., Coons, S., Guyer, M., and Harris, J. 1982. An Exposure and Risk Assessment for Benzo(a)pyrene and Polycyclic Aromatic Hydrocarbons: Vol. IV. U.S. Environmental Protection Agency, Washington, DC.

Pesticide Dictionary, 1976. Farm Chemicals. Meister Publishing Co., Willouhby, OH.

Quastel, J.H. and Scholefield, P.G. 1953. Arsenite Oxidation in Soil. Soil Sci. *75*: 279-285.

Rehwoldt, R., Lawrence, L., Shaw, C., and E. Wirhowski. 1973. The Acute Toxicity of Some Heavy Metal Ions Toward Benthic Organisms. Bull. Environ. Contam. Toxicol. 10:291-294.

Registry of Toxic Effects of Chemical Substances (RTECS). 1995. National Institute of Occupational Safety and Health (NIOSH). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Rosenblatt, D.H. and M.J. Small. 1981. Preliminary Pollutant Limit Values for Alabama Army Ammunition Plant. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, MD.

Saxena, J. and Howard, P.H. 1977. Environmental Transformation of Alkylated and Inorganic Forms of Certain Metals. Adv. Applied Microbiol. *21*: 195-226.

Sims, R.C. and Overcash, R. 1983. Fate of Polynuclear Aromatic Compounds (PNAs) in Soil-plant Systems. Residue Rev. 88:1-68.

Smith, L.R. and Dragun, J. 1984. Degradation of Volatile Chlorinated Aliphatic Priority Pollutants in Groundwater. Environ. Int. 10:291-298.

Spector, W.S. 1956. Handbook of Biological Data. W.B. Saunders Co., London, England.

Thayer, J.S. and Brinckman, F.E., 1982. The Biological Methylation of Metals and Metalloids. Adv. Organometallic Chem. *20*: 313-356.

U.S. Army Corps of Engineers (USACE). 1986. Nashville District, U.S. Environmental Protection Agency, U.S. Fish and Wildlife Service, and the Tennessee Valley Authority 1986, Final Environmental Impact Statement for Regulatory Actions Associated with the Olin Corporation, Remedial Action Plan to Isolate DDT from the People and the Environment in the Huntsville Spring Branch - Indian Creek System, Wheeler Reservoir, Alabama.

U.S. Army Corps of Engineers (USACE). 1989. Master Plan Narrative for Redstone Arsenal, Alabama. Prepared by Kimley-Horn and Associates, Inc. under the Direction of U.S. Army Engineer District, Mobile. Mobile, Alabama. Contract No. DACA01-87-D-0031.

U.S. Army Corps of Engineers (USACE). 1992. Preliminary Site Inspection for Redstone Arsenal. Site Inspection Report No. 91031. Toxic and Hazardous Materials Agency, Aberdeen Proving Ground, MD.

U.S. Army Environmental Hygiene Agency (AEHA). 1977. Special Study No. 24-0044-77, Evaluation of Environmental Degradation from Prior DDT Waste Disposal at Redstone Arsenal, Alabama. Letter Report for Commander, USA Material Development and Readiness Command.

U.S. Army Missile Command. 1989. Summary of DDT Contamination at Redstone Arsenal, Alabama.

U.S. Army Missile Command. 1992. Natural Resources Management Plan for Redstone Arsenal. Parts I, II, III, IV, V. June 1992. Redstone Arsenal, Alabama.

U.S. Department of Agriculture (USDA). 1986. New England Wildlife: Habitat, Natural History, and Distribution. Forest Service, NE Forest Experiment Station. General Technical Report NE-108.

U.S. Environmental Protection Agency (EPA). 1979. Water-related Environmental Fate of 129 Priority Pollutants. Office of Water Planning and Standards. EPA-44014-79-0296. NTIS 80-204381. Washington, DC.

U.S. Environmental Protection Agency (EPA). 1985a. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Office of Waste Programs Enforcement, EPA, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1986. Quality Criteria for Water. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 44/5-86-001.

U.S. Environmental Protection Agency (EPA). 1988a. Review of Ecological Risk Assessment Methods. Prepared by ICF Inc. Washington, DC. EPA 230-10-88-041.

U.S. Environmental Protection Agency (EPA). 1988b. Superfund Exposure Assessment Manual. Office of Remedial Response. Washington, DC. EPA/540/1-88/001.

U.S. Environmental Protection Agency (EPA). 1989. Mouse Oral Subchronic Toxicity Study. Prepared by Toxicity Research Laboratories, Ltd., Muskegon, MI. Office of Solid Waste, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1989a. Risk Assessment Guidance for Superfund (RAGS), Vol. I, Human Health Evaluation Manual (Part A). EPA/540/1-89/002.

U.S. Environmental Protection Agency (EPA). 1989b. Risk Assessment Guidance for Superfund (RAGS), Vol. II, Environmental Evaluation Manual. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/001.

U.S. Environmental Protection Agency (EPA). 1989c. Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory References. Environmental Research Laboratory, Corvalis, OR. EPA/600/3-89/013.

U.S. Environmental Protection Agency (EPA). 1991. Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors. OSWER Directive 9285.6-03. Office of Solid Waste and Emergency Response (Mar. 25, 1991).

U.S. Environmental Protection Agency (EPA). 1991a. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1991. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. OERR 9200.6-303 (91-1). NTIS No. PB91-921199.

U.S. Environmental Protection Agency (EPA). 1991b. Users Guide for Lead: A PC Software Application of the Uptake/Biokinetic Model. Version 0.50. Environmental Criteria and Assessment Office and Office of Health and Environmental Assessment, Cincinnati, OH.

U.S. Environmental Protection Agency (EPA). 1992. Addendum to the Region IV Supplement to the Risk Assessment Guidance for Superfund (RAGS). Region IV, Atlanta, GA. February 10, 1992.

U.S. Environmental Protection Agency (EPA). 1992a. Supplemental Guidance to RAGS: Calculating the Concentration Term. Office of Solid Waste and Emergency Response, Washington, DC. Publication 9285.7-081. May 1992.

U.S. Environmental Protection Agency (EPA). 1993a. Wildlife Exposure Factors Handbook. Vol. I of II. Office of Research and Development, Washington, DC. EPA/600/R-93/187a.

U.S. Environmental Protection Agency (EPA). 1993b. Wildlife Exposure Factors Handbook. Vol. II of II. Office of Research and Development, Washington, DC. EPA/600/R-93/187b.

U.S. Environmental Protection Agency (EPA). 1993c. Risk Assessment Issue Paper for Derivation of a Provisional Oral RfD for Benzene (CASRN 71-43-2). Superfund Technical Support Center, Environmental Criteria and Assessment Office (ECAO), Cincinnati, OH. No. 93-27.

U.S. Environmental Protection Agency (EPA). 1993d. Risk Assessment Issue Paper for Derivation of a Provisional Oral RfD for 1,2-Dichloroethane (CASRN 107-06-2). Superfund Technical Support Center, Environmental Criteria and Assessment Office (ECAO), Cincinnati, OH. No. 93-22.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1995. Risk-Based Concentration Table, January to June 1995. Technical Support Section, Region III, Philadelphia, PA. March 7, 1995.

U.S. Fish and Wildlife Service (USFWS). 1988. Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Washington, DC. Biological Report 85(1.12).

Vogel, T.M. and McCarty, P.L. 1985. Biotransformation of Tetrachloroethylene to Trichloroethylene, Dichloroethylene, Vinyl Chloride, and Carbon Dioxide under Methanogenic Conditions. Applied Env. Microbiol. *49*(5): 1080-1083.

Wams, T.J. 1987. Diethylhexylphthalate as an Environmental Contaminant--A Review. Sci. Tot. Environ. 66:1-16.

Water and Air Research, Inc. 1983. Redstone Arsenal Installation Restoration Summary, Volumes I, II, and III, Consulting Report Prepared for U.S. Army Toxic and Hazardous Materials Agency, March 1983.

Wong, P.T.S., Chau, Y.K. and Luxon, P.L. 1975. Methylation of Lead in the Environment. Nature. *253*: 263-264.

Wolfe, N.L., Paris, D.F., Steen, W.C., and Baughman, G.L. 1980. Correlation of Microbial Degradation Rates with Chemical Structure. Environ. Sci. Technol. 14:1143-1144.

Zepp, R.G. and Cline, D.M. 1977. Rates of Direct Photolysis in Aquatic Environment. Env. Sci. Technol. *11*: 359-366.

LIST OF TABLES

Table	
1.1-1	Study Areas to be Evaluated in the Baseline Risk Assessments at RSA
2.2-1	Summary of Lab Qualifiers for Samples Collected at Area F
2.2-2	Summary of Lab Qualifiers for Samples Collected at Area G
2.2-3	Summary of Lab Qualifiers for Samples Collected at Area R
2.2-4	Summary of Lab Qualifiers for Samples Collected at Areas S and T
2.3-1	Comparison of Onsite RFI Phase I Soil Sample Results at Area F to Background Concentrations
2.3-2	Comparison of Onsite RFI Phase I Soil Sample Results at Area G to Background Concentrations
2.3-3	Comparison of Onsite RFI Phase I Soil Sample Results at Area R to Background Concentrations
2.3-4	Comparison of Onsite RFI Phase I Soil Sample Results at Areas S and T to Background Concentrations
2.5-1	Summary of COPCs at Areas F, G, R, and S/T
3.1-1	Average Monthly Precipitation, Temperature, and Windspeed Data for RSA
3.1-2	Endangered or Threatened Fauna Potentially Found at RSA
3.1-3	Water Well and Spring Survey for RSA
3.2-1	PAHs Detected in the Study Areas
3.2-2	VOCs, Phthalate, and Pesticides Detected in the Study Areas
3.2-3	Metals Detected in the Study Areas
3.2-4	Physicochemical Properties of PAHs
3.2-5	Physicochemical Properties of VOCs, SVOCs, and Pesticides

LIST OF TABLES
(Continued, Page 2 of 2)

Table	
3.2-6	Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T
3.3-1	Soil Intake Values for Terrestrial Indicator Species
4.0-1	Chronic Dose-Response Toxicity Constants for the COPCs at RSA Study Areas F, G, R, and S/T
4.0-2	Weight-of-Evidence Categories for Potential Carcinogens
4.4-1	Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T
4.4-2	Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T
5.2-1	Summary of Human Health Risks and HIs for Areas F, G, R, and S/T
5.2-2	Summary of Media and COPCs Exceeding Risks Based on the Human Risk Characterization
5.2-3	Summary of Media and COPCs Exceeding an HQ of 0.1 or HI of 1 Based on the Human Noncarcinogenic Risk Characterization
5.4-1	Surface Soil Intakes and EQs for the Terrestrial Indicator Species at Areas F, G, R, and S/T
5.4-2	EQs for Potential Aquatic Indicator Species
5.4-3	Comparison of COPC Concentrations in Surface Water to Ambient Water Quality Criteria
5.6-1	Uncertainty Associated with the RA Process
6.1-1	Preliminary RGOs Associated with Future Worker Oral and Inhalation Exposure to Contaminants in Groundwater at RSA Study Areas F, G, R, and S/T
6.1-2	Preliminary RGOs Associated with Future Worker Exposure at RSA Study Areas F, G, R, and S/T Based on Cumulative Risk of 1E-4 or HI of 1

Table 1.1-1. Sites to be Evaluated in the Baseline Risk Assessments at RSA

Site	RSA Area	RSA No.	BRA No.	Unit Description
Unit 1		10	2	Active Inert and Closed Landfill - Closed Disposal Trenches, Closed Waste Oil Pits
Unit 2				Active Open Burn/Open Detonation Area
		12	3	Open Burn Pans
		13	3	Unlined Open Burn Areas
		14	3	Waste Burn Trenches
		131	3	Open Detonation Area
		132	3	Former Popping Furnace
		133	3	Former Rocket Washout Pad
Unit 3	F	49	1	Former Arsenic Ponds
	G	48	1	Former Sanitary and Industrial Landfill
	Q3	53	2	Former Sanitary and Industrial Landfill
	Q4	60	2	Former Sanitary and Industrial Landfill
	R	59	1	Former Industrial Landfill
	S/T	55/54	1	Former Industrial and Sanitary Landfill
	X1	66	3	Former Ash Disposal and Demolition Area
	Z	68	3	Former Industrial Waste and Ordnance Disposal and Demolition Area

Source: ESE.

Table 2.2-1. Summary of Lab Qualifiers for Samples Collected at Area F

Area	Medium	COPC	Lab Qualifier (frequency)
F (Phase I)	Surface Soil	Acetone	J (1/1)
	Soil Boring (shallow)	Barium	J (2/2)
	Sediment	PAHs	J (2/4)
		Cadmium	J (1/4)
		Barium	J (1/4)
		Carbon tetrachloride	J (1/4)
	Surface Water	Barium	J (1/1)
	Groundwater	Carbon tetrachloride	J (3/11)
		Trichloroethene	J (2/11)
		Arsenic	J (3/11)
		Barium	J (10/11)
		Chromium	J (2/11)
		Lead	J (3/11)
		Selenium	J (4/11)
Area F (Phase II)	Surface Soil	PAHs	J (4/9)
	Soil Boring (shallow)	PAHs	J (3/4)
	Sediment	PAHs	J (2/5)
	Groundwater	Cadmium	J (1/1)
		Mercury	J (1/1)

Table 2.2-2. Summary of Lab Qualifiers for Samples Collected at Area G

Area	Medium	COPC	Lab Qualifier (frequency)
G (Phase I)	Soil Boring (shallow)	Methylene chloride	J (1/3)
		Barium	J (1/3)
	Sediment	Acetone	J (1/5)
		Arsenic	J (2/5)
		Barium	J (2/5)
	Surface Water	Barium	J (1/3)
	Groundwater	Acetone	J (1/9)
		Arsenic	J (4/9)
		Barium	J (3/9)
		Chromium	J (1/9)
		Lead	J (1/9)
Area G (Phase II)	Surface water	Acetone	J (1/7)
		Dichloroethene, 1,1-	J (1/7)

Table 2.2-3. Summary of Lab Qualifiers for Samples Collected at Area R

Area	Medium	COPC	Lab Qualifier (frequency)
R (Phase I)	Surface Soil	Methylene chloride	J (2/2)
	Soil Boring (shallow)	PAHs	J (2/2)
		Barium	J (1/2)
		Selenium	J (2/2)
	Sediment	PAHs	J (2/4)
		Acetone	J (3/4)
		Dichloroethane, 1,1-	J (1/4)
		Barium	J (1/4)
	Surface water	Bis(2-ethylhexyl)phthalate	J (2/2)
		Arsenic	J (1/2)
		Barium	J (2/2)
		Silver	J (1/2)
	Groundwater	Bis(2-chloroethyl)ether	J (1/6)
		Bis(2-ethylhexyl)phthalate	J (1/6)
		Acetone	J (2/6)
		Chloroform	J (1/6)
		Methylene chloride	J (1/6)
		Arsenic	J (2/6)
		Barium	J (3/6)
		Lead	J (1/6)

Table 2.2-4. Summary of Lab Qualifiers for Samples Collected at Area S and T

Area	Medium	COPC	Lab Qualifier (frequency)
Area S and T (Phase I)	Surface Soil	Bis(2-ethylhexyl)phthalate	J (1/1)
		Acetone	J (1/4)
		Barium	J (2/4)
		DDT, 4,4'-	J (1/1)
	Soil boring (shallow)	Bis(2-ethylhexyl)phthalate	J (3/7)
		Barium	J (6/7)
		DDE, 4,4'-	J (5/7)
		DDT, 4,4'-	J (3/7)
	Groundwater	Acetone	J (6/29)
		Chloroform	J (3/29)
		Dichloroethane, 1,2-	J (1/29)
		Ethylbenzene	J (2/29)
		Tetrachloroethene	J (2/29)
		Trichloroethene	J (1/29)
		Trichloroethane	J (1/29)
		Barium	J (16/29)
		Cadmium	J (1/29)
		Chromium	J (5/29)
		Lead	J (1/29)
		Selenium	J (10/29)
Area S and T (Phase II)	Soil Boring (shallow)	Acetone	J (1/4)
		Methylene chloride	J (2/4)
	Groundwater	Chlorobenzene	J (1/4)
		Methylene chloride	J (1/4)
		PAHs	J (4/9)

Table 2.3-1. Comparison of Onsite RFI Phase I Soil Sample Results at Area F to Background Concentrations

Element	Expected Range for Natural Soils* (mg/kg)	Background Range for Soil at RSA** (mg/kg)	Area F, Average Concentration for Soil/ Wastes (mg/kg)	Area F, Average Concentration for Shallow Soils (mg/kg)	Area F, Average Concentration for Subsurface Soils (mg/kg)
Phase I RFI					
Arsenic	<1 - 73	3.3 - 8.8	9237.8	4275	5.3
Barium	10 - 1,500	5.7 - 172	62.2	73.8	15.1
Cadmium	0.010 - .10	<0.5	27.3	66.7	ND
Chromium III	1 - 1,000	11.2 - 138	23.9	24.1	52.9
Lead	<1 - 300	5.5 - 87.4	25.1	24.6	7.8
Mercury	0.01 - 3.4	<0.05 - 0.57	2.0	66.6	1.9
Selenium	0.1 - 2	<0.22 - 0.94	ND	0.28	ND
Silver	--	<0.05	ND	ND	ND
Phase II RFI					
Arsenic	<1 - 73	3.3 - 8.8	6,870	12.1	124
Cadmium	0.010 - 0.10	<0.5	35.8	ND	ND
Mercury	0.01 - 3.4	<0.05 - 0.57	247	1.7	4.3

Note: mg/kg = milligrams per kilogram.
ND = not detected.
-- = not available.

*Source: EPA Office of Solid Waste and Emergency Response, Hazardous Waste Land Treatment, SW-874 (April, 1983) Page 273, Table 6.45.
**Based on analytical results of background soil boring and shallow soil samples collected at selected sites on RSA during the 1990/1991 field investigations.

Source: G&M, 1991; 1992.

Table 2.3-2. Comparison of Onsite RFI Phase I Soil Sample Results at Area G to Background Concentrations

Element	Expected Range for Natural Soils* (mg/kg)	Background Range for Soils at RSA** (mg/kg)	Area G, Average Concentration for Soil/Waste Samples (mg/kg)	Area G, Average Concentration for Shallow Soil Samples (mg/kg)	Area G, Average Concentration for Subsurface Soil Samples (mg/kg)
Arsenic	<1 - 73	3.3 - 8.8	5.1	5.9	4.7
Barium	10 - 1,500	5.7 - 172	85.2	79.6	21.2
Cadmium	0.010 - .10	<0.5	0.6	ND	0.8
Chromium III	1 - 1,000	11.2 - 138	32.2	73.6	41.4
Lead	<1 - 300	5.5 - 87.4	66.8	15.0	10.5
Mercury	0.01 - 3.4	<0.05 - 0.57	0.9	0.2	ND
Selenium	0.1 - 2	<0.22 - 0.94	ND	ND	0.4
Silver	--	<0.5	ND	ND	ND

Note: mg/kg = milligrams per kilogram.
ND = not detected.
-- = not available.

*Source: EPA Office of Solid Waste and Emergency Response, Hazardous Waste Land Treatment, SW-874 (April, 1983) Page 273, Table 6.45.
**Based on analytical results of background soil boring and shallow soil samples collected at selected sites on RSA during RFI 1990/1991 field investigations.

Source: G&M, 1991.

Table 2.3-3. Comparison of Onsite RFI Phase I Soil Sample Results at Area R to Background Concentrations

Element	Expected Range for Natural Soils* (mg/kg)	Background Range for Soils at RSA** (mg/kg)	Area R, Average Concentration for Soil/Waste Samples (mg/kg)	Area R, Average Concentration for Shallow Soil Samples (mg/kg)	Area R, Average Concentration for Subsurface Soil Samples (mg/kg)
Arsenic	<1 - 73	3.3 - 8.8	1.8	7.0	9.6
Barium	10 - 1,500	5.7 - 172	55.1	87.90	13.4
Cadmium	0.010 - .10	<0.5	ND	ND	ND
Chromium III	1 - 1,000	11.2 - 138	9.1	26.8	79.9
Lead	<1 - 300	5.5 - 87.4	6.6	72.9	8.1
Mercury	0.01 - 3.4	<0.05 - 0.57	0.10	0.9	0.13
Selenium	0.1 - 2	<0.22 - 0.94	ND	0.51	1.07
Silver	--	<0.5	ND	ND	ND

Note: mg/kg = milligrams per kilogram.
ND = not detected.
-- = not available.

*Source: EPA Office of Solid Waste and Emergency Response, Hazardous Waste Land Treatment, SW-874 (April, 1983) Page 273, Table 6.45.
**Based on analytical results of background soil boring and shallow soil samples collected at selected sites on RSA during 1990/1991 field investigations.

Source: G&M, 1991.

Table 2.3-4. Comparison of Onsite RFI Phase I Soil Sample Results at Areas S and T to Background Concentrations

Element	Expected Range for Natural Soils* (mg/kg)	Background Range for Soils at RSA** (mg/kg)	Area S/T, Average Concentration for Soil/Waste Samples (mg/kg)	Area S/T, Average Concentration for Shallow Soil Samples (mg/kg)	Area S/T, Average Concentration for Subsurface Soil Samples (mg/kg)
Arsenic	<1 - 73	3.3 - 8.8	4.2	6.5	5.9
Barium	10 - 1,500	5.7 - 172	70.1	37.4	11.7
Cadmium	0.010 - .10	<0.5	0.5	ND	ND
Chromium III	1 - 1,000	11.2 - 138	34.9	43.7	58.8
Lead	<1 - 300	5.5 - 87.4	125	9.9	6.3
Mercury	0.01 - 3.4	<0.05 - 0.57	0.9	0.1	ND
Selenium	0.1 - 2	<0.22 - 0.94	ND	ND	ND
Silver	--	<0.5	2.4	ND	ND

Note: mg/kg = milligrams per kilogram.
ND = not detected.
-- = not available.

*Source: EPA Office of Solid Waste and Emergency Response, Hazardous Waste Land Treatment, SW-874 (April, 1983) Page 273, Table 6.45.

**Based on analytical results of background soil borings and shallow soil samples collected at selected sites on RSA during the 1990/1991 field investigations.

Source: G&M, 1991.

Table 2.5-1. Summary of COPCs at Areas F, G, R, and S/T

Parameter	Area F				Area G				Area R				Area S/T			
	SOIL	GW	SW	SED	SOIL	GW	SW	SED	SOIL	GW	SW	SED	SOIL	GW	SW	SED
Inorganic Chemicals																
Arsenic	√	√		√		√		√		√	√	√				
Barium		√	√	√		√	√	√		√	√	√		√		
Cadmium	√	√		√		√				√		√		√		
Chromium		√		√		√		√		√		√		√		
Lead		√		√		√				√		√		√		
Mercury	√	√		√								√		√		
Selenium											√	√				
PAHs*	√			√					√			√		√		
Pesticides																
DDT													√			
DDE													√			
Semivolatile Organic Chemicals																
Bis(2-ethylhexyl)phthalate	√								√	√	√	√	√			
Bis(2-chloroethyl)ether										√						
Butylbenzylphthalate									√							
VOCs																
Acetone	√	√		√	√	√	√	√	√	√		√	√	√		
Benzene		√		√										√		
Carbon tetrachloride												√				
Chlorobenzene		√								√				√		
Chloroform							√			√	√			√		
Dichloroethane, 1,1-												√				
Dichloroethane, 1,2-														√		
Dichloroethenes, 1,2-,total														√		
Ethylbenzene														√		
Methylene chloride	√			√	√		√	√	√	√		√	√	√		
Tetrachloroethene														√		
Toluene														√		
Trichloroethane, 1,1,1-														√		
Trichloroethene		√					√									
Vinyl chloride														√		
Xylenes, total														√		

* Includes one or more of the following: acenaphthene; anthracene; benz(a)anthracene; benzo(a)pyrene; benzo(b)fluoranthene; benzo(g,h,i)perylene; chrysene; dibenz(a,h)anthracene; fluoranthene; fluorene; indeno(1,2,3-cd)pyrene; naphthalene; phenanthrene; pyrene

Source: ESE.

Table 3.1-1. Average Monthly Precipitation, Temperature, and Windspeed Data for RSA*

Month	Average Temperature (°F)	Average Rainfall (Inches)	Average Wind Speed (Knots)
January	43	3.15	4
February	46	4.53	4
March	51	6.64	5
April	60	4.64	4
May	69	5.21	3
June	75	3.70	3
July	80	4.44	2
August	77	3.18	2
September	71	4.20	3
October	60	2.08	3
November	49	4.51	4
December	44	6.54	5
Annual Average	60	4.40	4

*Based on meteorological summaries for Redstone Arsenal for 1991, 1992, 1993.

Source: Department of the Army, Atmospheric Sciences Laboratory Redstone Meteorological Team.

Table 3.1-2. Water Well and Spring Survey for RSA

ID No.	Water Source	Owner	Description/Comments
1	Big Spring of Huntsville	City of Huntsville	Major spring in Huntsville. Formerly used for water supply. No longer in use for potable supply.
2	Brahan Spring at Huntsville	City of Huntsville	Natural spring serving approximately 60,000 customers in network with other water sources for potable water supply in Huntsville.
3	West Huntsville	Not available	Spring, no known water use.
4	Boiling Spring at Huntsville	University of Alabama	Spring, no known water use.
5	Boiling Spring at Madison	Not available	Spring, no known water use.
6	Lowe Spring near Madison and adjacent well #P-73	Estate Lowe	Spring, no known water use Well, no current water use.
7	X-37 Town of Triana	Not available	Well, no known water use.
8	X-45 A.G. Air Company	Not available	No known water use.
9	X-38 W. Lehman	Not available	No known water use.
10	P-28	U.S. Army	Well, no current water use.
11	Williams Spring and Lady Ann Well (P-27)	U.S. Army and City of Madison	Spring, no known water use. Well currently not in use. Previously used for City of Madison.
12	X-44 Williams Well	City of Huntsville	Well is used, in network, to supply potable water for approximately 60,000 customers (individuals or families) in the City of Huntsville.
13	MT-126 I.W. Grayson	Not available	Well, no known water use.
14	P-78 RSA	U.S. Army	Well previously used to supply Test Area 6 at RSA. No known water usage currently exists.
15	MT-130 Ragland Estate	Not available	Used as a test well. No water use.

Table 3.1-2. Water Well and Spring Survey for RSA (Continued Page 2 of 4)

ID No.	Water Source	Owner	Description/Comments
16	MT-129 Ragland Estate	Not available	Probably a test well. No known water use.
17	MT-111 University of Alabama	Not available	Well is used as a test well. No water use.
18	P-69 Jane Lowe	City of Huntsville	Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
19	CT 61	Hunstville Industrial Sites	Probably a test well. No known water use.
20	MT-171	Not available	Probably a test well. No known water use.
21	MT-170	Not available	Probably a test well. No known water use.
22	CT-74 J. Blue Co.	Not available	No known water use.
23	Q 79 Lowe Mill Well	City of Huntsville	Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
24	Q 200 West Huntsville Spring	West Huntsville Warehouse	Spring, no current water use.
25	Mad 1	City of Huntsville	No current water use.
26	WTP1	U.S. Army	Raw water treatment facility for RSA. Has separate units for treatment of potable and industrial water. In network with WTP3, serves to provide potable water for approximately 14,000 customers on the Arsenal. Water intake is directly from the Tennessee River.
27	WTP2	U.S. Army	Raw surface water (from Tennessee River) treatment facility. WTP2 supplies industrial use (nonpotable) water to RSA.

Table 3.1-2. Water Well and Spring Survey for RSA (Continued, Page 3 of 4)

ID No.	Water Source	Owner	Description/Comments
28	WTP3	U.S. Army	Raw water treatment facility for RSA. This plant receives partially treated (clarified) water from the industrial water distribution system supplied by WTP1 via a pipeline. In network with WTP1 serves to provide potable water for approximately 14,000 customers on the Arsenal.
29	East Huntsville Water Treatment Plant	City of Huntsville	Raw water treatment facility for the City of Huntsville. In network with other sources, provides potable water to approximately 60,000 customers (individuals or families) in the City of Huntsville. Water intake is directly from the Tennessee River.
30	West Huntsville Water Treatment Plant	City of Huntsville	Raw water treatment facility for the City of Huntsville. In network with other sources, provides potable water to approximately 60,000 customers (individuals or families) in the City of Huntsville. Water intake is directly from the Tennessee River.
31	Well at Visitor's Center	U.S. Army	Well previously used to provide potable water to Visitor's Center at RSA. Pumping capacity of 730 gph is available; however, water use is restricted to sewage plumbing. No potable water use currently exists.
32	Well at Center of Test Area 3	U.S. Army	Well previously used to provide potable water to server Test Area 3. No known water use currently exists.
33	Rock Spring	Not available	Spring, no known water use.
34	Lenox Spring	Not available	Spring, no known water use.
35	Mascus Spring	Not available	Spring, no known water use.
36	Bowers Spring	Not available	Spring, no known water use.
37	Airfield Runway Well	U.S. Army	Well that existed at time of purchase of RSA by the Government in 1941. It is not currently in service.
38	Well on North Side of Test Area 3	U.S. Army	Not currently in service.

Table 3.1-2. Water Well and Spring Survey for RSA (Continued, Page 4 of 4)

ID No.	Water Source	Owner	Description/Comments
39	Lincoln and Dallas Wells	City of Huntsville	Wells used to supply, in network with other sources, potable water for approximately 60,000 customers in the City of Huntsville.
40	Nolan Drake Well	Billy Drake	Well in a spring located upgradient of RSA. Well is used to supply potable water to the City of Madison. Water is pretreated (aerator) to remove TCE above MCLs. In winter, it is projected to serve approximately 18,000 customers at a projected output rate of 4,500,000 gpd.
41	Northeast Morgan County	Morgan County	Raw water intake from the Tennessee River for northeast Morgan County.

Source: G&M, 1992; MICOM, 1994.

Table 3.1-3. Endangered or Threatened Fauna Potentially Found at RSA

Animal Type	Common Name
Mammals	Florida Panther Eastern Cougar Southeastern Shrew Indiana Bat Hoary Bat Grey Bat Black Bear
Birds	Red-Cockaded Woodpecker American Peregrine Falcon Arctic Peregrine Falcon Sharp Skinned Hawk Golden Eagle Bald Eagle Osprey Bewick's Wren Buchman's Warbler
Reptiles/Amphibians	American Alligator Tennessee Cave Salamander
Fishes	Tuscumbia Darter Southern Cavefish Flame Chub
Invertebrates	Alabama Cave Shrimp (Listed as Endangered)

Source: Engineering-Science, 1994.

Table 3.2-1. PAHs Detected in the Study Areas

PAH	Area F	Area G	Area R	Area S/T
Benzo(a)anthracene	X	X	X	X
Benzo(a)pyrene	X	--	X	X
Benzo(b)fluoranthene	X	--	--	--
Benzo(k)fluoranthene	X	--	--	--
Dibenzo(a,h)anthracene	X	--	--	X
Indeno(1,2,3-cd)pyrene	X	--	--	--
Chrysene	X	--	--	--

Source: ESE.

Table 3.2-2. VOCs, Phthalate, and Pesticides Detected in the Study Areas

Compound	Area F	Area G	Area R	Area S/T
VOCs				
Carbon Tetrachloride	X	--	--	--
Trichloroethene	X	X	--	X
Tetrachloroethene	--	--	--	X
Vinyl Chloride	--	--	--	X
Benzene	--	--	--	X
Chlorobenzene	--	--	--	X
SVOCs				
Bis(2-ethylhexyl)phthalate	--	--	X	--
Pesticides				
4,4'-DDT	--	--	--	X
4,4'-DDD	--	--	--	X
4,4'-DDE	--	--	--	X

Source: ESE.

Table 3.2-3. Metals Detected in the Study Areas

Metal	Area F	Area G	Area R	Area S/T
Arsenic	X	--	--	--
Cadmium	X	--	--	--
Mercury	X	X	--	--
Lead	--	X	X	X

Source: ESE.

Table 3.2-4. Physicochemical Properties of PAHs

PAH	MW (g/M)	Water Solubility (mg/L)	Vapor Pressure (mmHg)	Henry's Law Constant (atm-m^3/mole)	Organic Carbon Partition Coefficient (mL/g)
Benzo(a)anthracene	228.3	9.0 E-3	2.2 E-8	7.3 E-7	1.4 E+6
Benzo(a)pyrene	252.2	3.8 E-3	5.6 E-9	4.9 E-7	5.5 E+6
Benzo(b)fluoranthene	252.3	1.0 E-3	5.0 E-7	1.7 E-4	5.5 E+5
Benzo(k)fluoranthene	252.3	5.5 E-4	5.0 E-7	3.0 E-4	5.5 E+5
Dibenzo(a,h)anthracene	278.4	5.0 E-4	1.0 E-10	7.3 E-8	1.7 E+6
Indeno(1,2,3-cd)pyrene	276.3	5.0 E-4	1.0 E-10	7.3 E-8	1.6 E+6
Chrysene	228.3	1.8 E-3	6.3 E-9	1.1 E-6	2.0 E+5

Note: atm-m^3/mole = atmospheres per cubic meter per mole.
g/M = gram per mole.
mg/L = milligrams per liter.
mL/g = milliliters per gram.
mmHg = millimeters of mercury.
MW = molecular weight.

Source: ESE.

Table 3.2-5. Physicochemical Properties of VOCs, SVOCs, and Pesticides

	MW (g/mole)	Solubility (mg/L)	Water Vapor Pressure (mmHg)	Henry's Law Constant (atm-m^3/mole)	Organic Carbon Partition Coefficient (mL/g)
VOCs					
Carbon Tetrachloride	154	7.57 E+2	9.00 E+1	2.41 E-2	110
Trichloroethene	131	1.10 E+3	5.79 E+1	9.07 E-3	126
Tetrachloroethene	166	1.50 E+2	1.78 E+1	2.59 E-2	364
Vinyl Chloride	63	2.67 E+3	2.66 E+3	8.26 E-2	57
Benzene	78	1.78 E+3	9.50 E+1	5.48 E-3	83
Chlorobenzene	113	4.66 E+2	1.17 E+1	3.72 E-3	930
SVOCs					
Bis(2-ethylhexyl)phthalate	391	3.0 E-1	6.45 E-6	1.11 E-5	1.0 E+4
Pesticides					
4,4'-DDT	354.5	5.5 E-3	7.3 E-7	6.2 E-5	2.38 E+4
4,4'-DDD	320.0	2.0 E-2	1.0 E-6	2.1 E-5	3.75 E+4
4,4'-DDE	318.0	1.4 E-2	6.5 E-6	1.9 E-4	5.01 E+4

Source: ESE.

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Page 1 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Current Land Use			
Onsite Worker	Incidental ingestion, direct dermal contact, and inhalation of airborne soil particulates while working at the study areas	Yes	Persons working in the building adjacent to Area F may be exposed to site COPCs; onpost workers access Area T occasionally for maintenance-related activities
Recreational	Incidental ingestion and dermal contact with surface water and sediment in McDonald Creek adjacent to Area G; ingestion of fish caught in McDonald Creek	Yes	Site observations suggest that McDonald Creek is used for fushing in the vicinity of Area G
Future Land Use			
Onsite Worker	Use of the site for future industrial purposes; assumes a worker will have the same soil exposure pathways as a current worker but spends longer periods of time at the site; also uses onsite groundwater for drinking (ingestion) and showering (dermal and inhalation) (as required by regulatory agencies)	Yes	Although RSA does not plan to change the existing land use of the site, for conservative risk estimates, such assumptions are analyzed for all five areas

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Continued, Page 2 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Future Land Use, Cont.			
Residential	Use of the site for future residential purposes	No	For this BRA, the analytical data and the exposure assumptions were evaluated based on an industrial land use. This projected future land use is in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA to be developed in accordance with RSA remaining as an Army-based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo BRAC, which further supports that the current mission will continue into the future. Thus, this information supports that the future residential scenario is unrealistic.

Source: ESE.

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Page 1 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Current Land Use			
Onsite Worker	Incidental ingestion, direct dermal contact, and inhalation of airborne soil particulates while working at the study areas	Yes	Persons working in the building adjacent to Area F may be exposed to site COPCs; onpost workers access Area T occasionally for maintenance-related activities
Recreational	Incidental ingestion and dermal contact with surface water and sediment in McDonald Creek adjacent to Area G; ingestion of fish caught in McDonald Creek	Yes	Site observations suggest that McDonald Creek is used for fushing in the vicinity of Area G
Future Land Use			
Onsite Worker	Use of the site for future industrial purposes; assumes a worker will have the same soil exposure pathways as a current worker but spends longer periods of time at the site; also uses onsite groundwater for drinking (ingestion) and showering (dermal and inhalation) (as required by regulatory agencies)	Yes	Although RSA does not plan to change the existing land use of the site, for conservative risk estimates, such assumptions are analyzed for all five areas

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Continued, Page 2 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Future Land Use, Cont.			
Residential	Use of the site for future residential purposes	No	According to the RSA Master Plan (1989), the Army does not plan to change the existing land use conditions at the base

Source: ESE.

Table 3.3-1. Soil Intake Values for Terrestrial Indicator Species

Species	Intake (kg/kg/day)	Intake Factors			Source / Comments
Mouse	4E-03	IR	0.0038	kg/day	(0.2 g/g-day * 19 grams)
		EF	365	days/year	
		ED	1	year	EPA Factor
		%soil	0.02		EPA factor
		BW	0.019	kg	average of six mean values from EPA doc. (Millar, Fordham, Dewsbury)
		AT	365	days	
Raccoon	6E-03	IR	0.35	kg/day	based on Spector (1956)
		EF	365	days/year	
		ED	3	years	
		%soil	0.094		EPA factor
		BW	5.7	kg	Adult weight based on EPA factors (Sanderson, Nagel, Johnson)
		AT	1095	days	EPA factor (Johnson)
Deer	1E-03	IR	0.972	kg/day	based on g/kg BW value for cattle from LAAP report
		EF	365	days/year	
		ED	5	years	
		%soil	0.063		factor from jackrabbit EPA factor
		BW	54	kg	RSA Natural Resources document
		AT	1825	days	RSA Natural Resources Document - NUJ estimation
Bobwhite	7E-04	IR	0.014	kg/day	based on EPA factor of .07775 x BW (avg. from Koerth&Guthery data)
		EF	300	days/year	total lifespan - days
		ED	0.8333333333	years	EPA Factor
		%soil	0.01		based on EPA factor value for American woodcock
		BW	0.174	kg	EPA Factor (avg. from Robel, Roseberry & Klimstra, Guthery)
		AT	304.16666667	days	

Source: ESE.

Table 4.0-1. Chronic Dose-Response Toxicity Constants for the COPCs at RSA Sites F, G, R, and S/T (Page 1 of 4)

Chemical	Oral RfD (UF)*	Inhal RfD (UF)**	Oral CSF*	Oral WoE*	Inhal CSF***	Inhal WoE*
Inorganic Chemicals (IOC)						
Arsenic	3.0E-04 (3)	--	1.8E+00[I1]	A	5.0E+01#	A
Barium	7.0E-02 (3)	1.4E-04# (1,000)	--		--	
Cadmium (aqueous matrix)	5.0E-04 (10)	--	--		--	
Cadmium (solid matrix)	1.0E-03 (10)	--	--		6.3E+00	B1
Chromium, total[I2]	5.0E-03 (500)	nd[I3]	--		4.2E+01	A
Lead	--[I4]	--	nd[I5]	B2	nd[I5]	B2
Mercury	3.0E-04# (1,000)	8.6E-05# (30)	--		--	
Selenium	5.0E-03 (3)	--	--		--	
Polycyclic Aromatic Hydrocarbons (PAHs)						
Acenaphthene	6.0E-02 (3,000)	--	--		--	
Anthracene	3.0E-01 (3,000)	--	--		--	
Benz(a)anthracene	3.0E-02[H1] (3,000)	--	7.3E-01[H2]	B2	6.1E-01[H2]	B2
Benzo(a)pyrene	3.0E-02[H1] (3,000)	--	7.3E+00	B2	6.1E+00	B2
Benzo(b)fluoranthene	3.0E-02[H1] (3,000)	--	7.3E-01[H2]	B2	6.1E-01[H2]	B2
Benzo(ghi)perylene	3.0E-02[H1] (3,000)	--	--		--	
Chrysene	3.0E-02[H1] (3,000)	--	7.3E-03[H2]	B2	6.1E-03[H2]	B2
Dibenz(ah)anthracene	3.0E-02[H1] (3,000)	--	7.3E+00[H2]	B2	6.1E+00[H2]	B2
Fluoranthene	4.0E-02 (3,000)	--	--		--	
Fluorene	4.0E-02 (3,000)	--	--		--	
Indeno(1,2,3-cd)pyrene	3.0E-02[H1] (3,000)	--	7.3E-01[H2]	B2	6.1E-01[H2]	B2
Naphthalene	4.0E-02 (1,000)	--	--		--	
Phenanthrene	3.0E-02[H1] (3,000)	--	--		--	
Pyrene	3.0E-02 (3,000)	--	--		--	
Pesticides						
DDE, p,p'-	5.0E-04[P1] (100)	--	3.4E-01	B2	3.4E-01[P2]	B2
DDT, p,p'-	5.0E-04 (100)	--	3.4E-01	B2	3.4E-01	B2

Table 4.0-1. Chronic Dose-Response Toxicity Constants for the COPCs at RSA Sites F, G, R, and S/T (Continued, Page 2 of 4)

Chemical	Oral RfD (UF)*	Inhal RfD (UF)**	Oral CSF*	Oral WoE*	Inhal CSF***	Inhal WoE*
Miscellaneous Semivolatile Organic Chemicals (SOCs)						
Bis(2-chloroethyl) ether	7.5E-04[S1] (100,000)	--	1.1E+00	B2	1.2E+00	B2
Bis(2-ethylhexyl) phthalate	2.0E-02 (1,000)	--	1.4E-02	B2	nd[S2]	B2
Butylbenzyl phthalate	2.0E-01 (1,000)	--	nd[S3]	C	nd[S4]	C
Miscellaneous Volatile Organic Chemicals (VOC)						
Acetone	1.0E-01 (1,000)	--	--		--	
Benzene	3.0E-04[V1] (3,000)	1.7E-03[V2] (na)	2.9E-02	A	2.9E-02	A
Carbon tetrachloride	7.0E-04 (1,000)	--	1.3E-01	B2	5.3E-02	B2
Chlorobenzene	2.0E-02 (1,000)	5.7E-03# (10,000)	--		--	
Chloroform	1.0E-02 (1,000)	--	6.1E-03	B2	8.1E-02	B2
Dichloroethane, 1,1-	1.0E-01# (1,000)	1.4E-01# (1,000)	nd[V3]	C	nd[V3]	C
Dichloroethane, 1,2-	3.0E-02[V4] (1,000)	2.9E-03[V2] (na)	9.1E-02	B2	9.1E-02	B2
Dichloroethenes, 1,2-, total	9.0E-03# (1,000)	--	--		--	
Ethylbenzene	1.0E-01 (1,000)	2.9E-01 (300)	--		--	
Methylene chloride	6.0E-02 (100)	8.6E-01# (100)	7.5E-03	B2	1.6E-03	B2
Tetrachloroethene	1.0E-02 (1,000)	--	5.1E-02[V5]	B2[V5]	1.8E-03[V5]	B2[V5]
Toluene	2.0E-01 (1,000)	1.1E-01 (300)	--		--	
Trichloroethane, 1,1,1-	9.0E-02# (1,000)	2.9E-01[V6] (1,000)	--		--	
Trichloroethene	6.0E-03[V7] (na)	--	1.1E-02[V5]	B2[V5]	6.0E-03[V8]	B2[V5]
Vinyl chloride	1.3E-03[V9] (na)	--	1.9E+00#	A	3.0E-01#	A
Xylenes, total	2.0E+00 (100)	--	--		--	

Note:
RfD = reference dose [mg/kg/day].
UF = uncertainty factor.
MF = modifying factor.
CSF = cancer slope factor [(mg/kg/day)$^{-1}$].
WoE = weight of evidence for ranking as a human carcinogen (see Table 4.0-2).

Table 4.0-1. Chronic Dose-Response Toxicity Constants for the COPCs at RSA Sites F, G, R, and S/T (Continued, Page 3 of 4)

inhal = inhalation.
na = not applicable.
nd = not determined.
p,p'-DDE = 1,1-dichloro-2,2-bis-(p-chlorophenyl)ethene.
p,p'-DDT = 1,1,1-trichloro-2,2-bis-(p-chlorophenyl)ethane.
MCL = EPA Maximum Contaminant Level.
NOAEL = no observed adverse effect level.
LD_{50} = dose resulting in death in 50 percent of a study population.
MRL = minimum risk level.
mg/kg/day = milligrams per kilogram per day.
mg/L = milligrams per liter.
µg/L = micrograms per liter.
L/day = liters per day.

(H1) No RfD is available for this PAH; the lowest non-naphthalene value (pyrene) is used for comparison, only.
(H2) CSF for this potentially carcinogenic PAH is an interim value from EPA ECAO and listed in EPA Region III's Risk-Based Concentration Table, Third Quarter 1994 (EPA, 1994b). The value is based on the CSF for benzo(a)pyrene and the following Toxicity Equivalency Factors: benz(a)anthracene, 0.1; benzo(b)fluoranthene, 0.1; benzo(k)fluoranthene, 0.01; chrysene, 0.001; dibenz(ah)anthracene, 1.0; and indeno(1,2,3-cd)pyrene, 0.1.
(I1) CSF for arsenic based on unit cancer risk of 5×10^{-5} $(\mu g/L)^{-1}$ proposed by Risk Assessment Forum.
(I2) All values are for hexavalent chromium; a less conservative oral RfD of 1E+00 mg/kg/day for trivalent chromium is also available.
(I3) Inhalation RfD for chromium has been withdrawn from IRIS pending further EPA review.
(I4) EPA's RfD Work Group considers it inappropriate to develop an RfD for inorganic lead and prefers to use the Uptake/Biokinetic (UBK) Model (EPA, 1991b) to evaluate total lead exposure on a site-specific basis.
(I5) Although EPA has classified lead as a Group B2 suspect human carcinogen via ingestion and inhalation, no CSF has been developed for either of these exposure pathways.
(P1) No RfD is available for p,p'-DDD or p,p'-DDE. Listed value is for parent compound p,p'-DDT.
(P2) No inhalation CSF is available for p,p'-DDE; listed value is for parent compound p,p'-DDT.

Table 4.0-1. Chronic Dose-Response Toxicity Constants for the COPCs at RSA Sites F, G, R, and S/T (Continued, Page 4 of 4)

(S1) Oral RfD for bis(2-chloroethyl) ether based on an acute oral LD_{50} for rats of 75 mg/kg (RTECS, 1994), an uncertainty factor of 10,000 (10X for sensitive human subpopulations, 10X for animal-to-human extrapolation, 10X for acute-to-chronic extrapolation, and 10X for LD_{50}-to-NOAEL extrapolation), and a modifying factor of 10 (accounts for the lethal endpoint of the study).
(S2) Although EPA has classified this SOC as a Group B2 suspect human carcinogen via inhalation, no CSF has been developed for this exposure pathway.
(S3) Although EPA has classified this SOC as a Group C possible human carcinogen via ingestion, no CSF has been developed for this exposure pathway.
(S4) Although EPA has classified this SOC as a Group C possible human carcinogen via inhalation, no CSF has been developed for this exposure pathway.
(V1) Oral RfD for benzene from an EPA Environmental Criteria and Assessment Office (ECAO) Risk Assessment Issue Paper (EPA, 1993).
(V2) Inhalation RfD from ECAO (EPA, 1995).
(V3) Although EPA has classified 1,2-dichloroethane as a Group B2 suspect human carcinogen via ingestion and inhalation, no CSF has been developed for either of these exposure pathways.
(V4) Oral RfD for 1,2-dichloroethane from an ECAO Risk Assessment Issue Paper (EPA, 1993).
(V5) CSFs and WoEs for this VOC have been withdrawn from IRIS pending further review; values are from HEAST (EPA, 1991a).
(V6) EPA has determined the inhalation RfD for this VOC to be not verifiable; the listed value is from HEAST (EPA, 1991a).
(V7) Oral RfD for trichloroethene from an ECAO Risk Assessment Paper (EPA, 1995).
(V8) Inhalation CSF for trichloroethene from ECAO (EPA, 1995).
(V9) Oral RfD for vinyl chloride is MRL from ATSDR's Toxicological Profile for Vinyl Chloride (1989).

*All oral RfDs, oral CSFs, and WoEs are available in IRIS (1995), unless otherwise noted.
**Inhalation RfDs are based on the inhalation RfC available in IRIS (1995) and assume that a healthy 70-kilogram adult inhales 20 m^3/day air, unless otherwise noted.
***Inhalation CSFs are based on the inhalation UR available in IRIS (1995) and assume that a healthy 70-kilogram adult inhales 20 m^3/day air, unless otherwise noted.
#Value available in HEAST (EPA, 1994a).

Source: ESE.

Table 4.0-2. Weight-of-Evidence Categories for Potential Carcinogens

EPA Category	Group Description	Evidence Description
Group A	Human carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer
Group B1	Probable human carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies
Group B2	Probable human carcinogen	Sufficient evidence of carcinogenicity in animals but inadequate data in humans
Group C	Possible human carcinogen	Limited evidence of carcinogenicity in animals
Group D	Not classified	Inadequate evidence of carcinogenicity in animals
Group E	No evidence of carcinogenicity in humans	No evidence of carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies

Source: IRIS, 1994.

Table 4.4-1. Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T (Page 1 of 4)

17 Feb 1995 14:00

COC	Receptor	Test	Effect	Concentration	Units	Exposure Type	Duration	Reference
Arsenic	Duckling	Chronic		6.00E+02	ppm	Oral	4 weeks	no citation in tox profile
	Mouse	Acute	LOEC	4.00E+01	mg/kg-bw/day	Oral	3 days	EPA, 1984
	Whitetailed deer			9.23E+02	mg			ESE, 1989
	Wild rabbit			1.05E+01	mg/kg-bw			ESE, 1989
Cadmium	Mouse	Acute	LD50	8.90E+02	mg/kg			RTECS, 1994
	Rabbit	Acute	LDLo	7.00E+01	mg/kg			RTECS, 1994
Mercury	Japanese quail	Acute	LD50	3.11E+01	mg/kg	Oral	14 days	Hill and Soares, 1984
	Rat	Chronic	NOAEL	3.20E-01	mg/kg/day	Oral	7 days/week, 2 years	Fitzhugh et al., 1950 (ATSDR, 1989)
Selenium	Rat	Acute	LD50	6.70E+03	mg/kg	Oral		RTECS, 1994
Acenaphthene	Mouse	Subchronic	NOAEL	1.75E+02	mg/kg/day	Gavage	90 days	EPA, 1989 (IRIS, 1994)
Anthracene	Mouse	Subchronic	NOEL	1.00E+03	mg/kg/day	Gavage	90 days	EPA, 1989 (IRIS, 1994)
Benz(a)anthracene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Benzo(a)pyrene	Mouse	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Benzo(b)fluoranthene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Benzo(ghi)perylene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Chrysene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Dibenz(ah)anthracene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)

Table 4.4-1. Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T (Continued, Page 2 of 4)

17 Feb 1995 14:00

COC	Receptor	Test	Effect	Concentration	Units	Exposure Type	Duration	Reference
Fluoranthene	Rodent	Acute	LD50	2.00E+03	mg/kg-bw	Oral		Sims and Overcash, 1983
Fluorene	Mouse	Subchronic	NOAEL	1.25E+02	mg/kg/day	Gavage	13 weeks	EPA, 1989 (IRIS, 1994)
Indeno(1,2,3-cd)pyrene	Mouse *	Chronic	NOEL	1.33E+01	mg/kg/day	Oral	110 days	Neal and Rigdon, 1967 (ATSDR, 1990)
Naphthalene	Cat	Acute	LDLo	1.00E+03	mg/kg	Oral		RTECS, 1994
	Dog	Acute	LDLo	4.00E+02	mg/kg	Oral		RTECS, 1994
	Guinea Pig	Acute	LD50	1.20E+03	mg/kg	Oral		RTECS, 1994
	Mouse	Acute	NOAEL	5.30E+01	mg/kg		14 days	EPA, 1990 (HSDB, 1994)
	Mouse	Chronic	NOAEL	2.00E+02	mg/kg	Gavage	5 days/week, 13 weeks	EPA, 1990 (HSDB, 1994)
	Mouse		LD50	5.33E+02	mg/kg	Gavage		EPA, 1990 (HSDB, 1994)
	Mouse		LD50	7.10E+02	mg/kg	Gavage		EPA, 1990 (HSDB, 1994)
	Mouse	Acute	LD50	5.33E+02	mg/kg	Oral		RTECS, 1994
	Rabbit	Acute	LDLo	3.00E+03	mg/kg	Oral		RTECS, 1994
	Rat	Chronic	NOAEL	5.00E+01	mg/kg	Gavage	5 days/week, 13 weeks	EPA, 1990 (HSDB, 1994)
	Rat	Chronic	NOAEL	4.10E+01	mg/kg	Oral	700 days	EPA, 1990 (HSDB, 1994)
	Rat		LD50	2.60E+03	mg/kg	Oral		Papciak and Mallory, 1990 (HSDB, 1994)
	Rat		LD50	2.20E+03	mg/kg	Oral		EPA, 1990 (HSDB, 1994)
	Rat		LD50	2.40E+03	mg/kg	Oral		EPA, 1990 (HSDB, 1994)
	Rat	Acute	LD50	4.90E+02	mg/kg	Oral		RTECS, 1994
Phenanthrene	Rodent	Acute	LD50	7.00E+02	mg/kg-bw	Oral		Sims and Overcash, 1983
Pyrene	Rodent	Chronic		9.90E+01	mg/kg-bw	Oral		Sims and Overcash, 1983
DDE	Bobwhite quail	Acute	LC50	8.25E+02	ppm	Oral	5 days	USFWS, 1975 (HSDB, 1994)
	Hamster	Acute	LD50	> 5.00E+03	mg/kg	Oral		RTECS, 1994
	Japanese quail	Acute	LC50	1.36E+03	ppm	Oral	5 days	USFWS, 1975 (HSDB, 1994)
	Japanese quail	Acute	LC50	8.59E+02	ppm	Oral	5 days	USFWS, 1986 (HSDB, 1994)
	Mallard duck	Acute	LC50	3.57E+03	ppm	Oral	5 days	USFWS, 1975 (HSDB, 1994)
	a Mouse	Acute	LD50	7.00E+02	mg/kg	Oral		RTECS, 1994
	a Rat	Acute	LD50	8.80E+02	mg/kg	Oral		RTECS, 1994
	Ring-necked pheasant	Acute	LC50	8.29E+02	ppm	Oral	5 days	USFWS, 1975 (HSDB, 1994)
	a Small Mammal Average			7.90E+02	ppm			

Table 4.4-1. Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T (Continued, Page 3 of 4)

17 Feb 1995 14:00

COC	Receptor	Test	Effect	Concentration	Units	Exposure Type	Duration	Reference
DDT	Bobwhite quail	Acute	LC50	6.11E+02	ppm	Oral	5 days	USFWS, 1972 (HSDB, 1994)
	Calf		LOAEL	2.50E+02	mg/kg	Oral		Osweiler et al., 1985
	California quail		LD50	5.95E+02	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Cat	Acute	LDLo	2.50E+02	mg/kg	Oral		RTECS, 1994
	Chicken	Acute	LDLo	3.00E+02	mg/kg	Oral		RTECS, 1994
	Dog		LD50	7.50E+02	mg/kg	Oral		Hartley and Kidd, 1987 (HSDB, 1994)
	Dog	Acute	LD50	1.50E+02	mg/kg	Oral		RTECS, 1994
	Goat		LD50	> 1.00E+03	mg/kg	Oral		Hartley and Kidd, 1987 (HSDB, 1994)
	Goat	Acute	LDLo	3.00E+02	mg/kg	Oral		RTECS, 1994
	b Guinea pig	Acute	LD50	1.50E+02	mg/kg	Oral		RTECS, 1994
	Hamster	Acute	LD50	> 5.00E+03	mg/kg	Oral		RTECS, 1994
	Japanese quail		LD50	8.41E+02	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Japanese quail	Acute	LC50	5.68E+02	ppm	Oral	5 days	USFWS, 1972 (HSDB, 1994)
	Japanese quail	Acute	LC50	4.16E+02	ppm	Oral	5 days	USFWS, 1986 (HSDB, 1994)
	Mallard duck		LD50	> 2.24E+03	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Mallard duck	Acute	LC50	1.87E+03	ppm	Oral	5 days	USFWS, 1972 (HSDB, 1994)
	b Mouse		LD50	3.00E+02	mg/kg	Oral		Hartley and Kidd, 1987 (HSDB, 1994)
	b Mouse	Acute	LD50	1.35E+02	mg/kg	Oral		RTECS, 1994
	b Rabbit		LD50	3.00E+02	mg/kg	Oral		Hartley and Kidd, 1987 (HSDB, 1994)
	b Rabbit	Acute	LD50	2.50E+02	mg/kg	Oral		RTECS, 1994
	b Rat	Acute	LD50	1.18E+02	mg/kg	Oral		Worthing and Walker, 1987 (HSDB, 1994)
	b Rat	Acute	LD50	8.70E+01	mg/kg	Oral		RTECS, 1994
	Ring-necked pheasant		LD50	1.33E+03	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Ring-necked pheasant	Acute	LC50	3.11E+02	ppm	Oral	5 days	USFWS, 1986 (HSDB, 1994)
	Rock dove		LD50	5.95E+02	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Sandhill crane		LD50	> 1.20E+03	mg/kg	Oral		USFWS, 1984 (HSDB, 1994)
	Sheep		LD50	> 1.00E+03	mg/kg	Oral		Hartley and Kidd, 1987 (HSDB, 1994)
	b Small Mammal Average			1.91E+02	ppm			
Bis(2-ethylhexyl) phthalate	c Guinea pig		LD50	2.63E+04	mg/kg	Oral		Krauskopf, 1973
	c Mouse		LD50	3.35E+04	mg/kg	Oral		Krauskopf, 1973
	c Mouse		LD50	4.90E+04	mg/kg	Oral		Yamada, 1974
	c Mouse		LD50	2.60E+04	mg/kg	Oral		Patty, 1967
	c Rabbit		LD50	3.39E+04	mg/kg	Oral		Shaffer et al., 1945
	c Rat		LD50	2.60E+04	mg/kg	Oral		Patty, 1967
	c Rat (Wistar, male)		LD50	3.06E+04	mg/kg	Oral		Shaffer et al., 1945
	c Rat (Wistar, male)		LD50	> 3.40E+04	mg/kg	Oral		Hodge, 1943
	c Small Mammal Average			3.24E+04	mg/kg			

Table 4.4-1. Unadjusted Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T (Continued, Page 4 of 4)

17 Feb 1995 14:00

COC	Receptor	Test	Effect	Concentration	Units	Exposure Type	Duration	Reference
Butylbenzyl phthalate	d Guinea pig	Acute	LD50	1.38E+04	mg/kg			RTECS, 1994
	d Mouse	Acute	LD50	4.17E+03	mg/kg			RTECS, 1994
	d Rat	Acute	LD50	2.33E+03	mg/kg			RTECS, 1994
	d Small Mammal Average			6.75E+03	ppm			
Acetone	Dog		LDLo	8.00E+03	mg/kg	Oral		RTECS, 1994
	Japanese quail		LC50	> 4.00E+04	ppm	Oral		USFWS, 1986 (HSDB, 1994)
	e Mouse		LD50	3.00E+03	mg/kg	Oral		RTECS, 1994
	e Rabbit		LD50	5.34E+03	mg/kg	Oral		RTECS, 1994
	e Rat		LD50	5.80E+03	mg/kg	Oral		RTECS, 1994
	Ring-necked pheasant		LC50	> 4.00E+04	ppm	Oral		USFWS, 1975 (HSDB, 1994)
	e Small Mammal Average			4.71E+03	ppm			
Methylene chloride	Mouse	Acute	LD	> 1.70E+04	mg/kg	Oral		RTECS, 1994
	Mouse	Chronic	LOEL	5.80E+02	mg/kg	Oral	90 days	Kirschman et al., 1986
	Rat		LD50	1.60E+03	mg/kg	Oral		Verschueren, 1983
	Rat		LOEL	1.90E+02	mg/kg	Oral		Kirschman et al., 1986

*Value for benzo(a)pyrene is used for all carcinogenic PAHs.

Source: ESE.

Table 4.4-2. Ecological Benchmarks for Soil COPCs at Areas F, G, R, and S/T*

17 Feb 1995 16:00

Chemical	Unadjusted Benchmark Value						Adjusted Benchmark Value **			
	Terrestrial			Avian						
	Value	Species	Type	Value	Species	Type	Mouse	Raccoon	Deer	Bobwhite
Metals										
Arsenic	4.00E+01	Mouse	Acute LOEC	--	--	--	4.00E+01	4.00E+00	4.00E+00	4.00E+00
Cadmium	8.90E+02	Mouse	Acute LD50	--	--	--	8.90E+01	8.90E+00	8.90E+00	8.90E+00
Mercury	3.20E-01	Rat	Chronic NOAEL	3.11E+01	Japanese quail	Acute LC50	3.20E-01	3.20E-02	3.20E-02	3.11E-01
Selenium	6.70E+03	Rat	Acute LD50	--	--	--	6.70E+02	6.70E+01	6.70E+01	6.70E+01
PAHs										
Acenaphthene	1.75E+02	Mouse	Subchronic NOAEL	--	--	--	1.75E+02	1.75E+01	1.75E+01	1.75E+01
Anthracene	1.00E+03	Mouse	Subchronic NOEL	--	--	--	1.00E+03	1.00E+02	1.00E+02	1.00E+02
Benz(a)anthracene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Benzo(b)fluoranthene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Benzo(ghi)perylene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Benzo(a)pyrene	1.33E+01	Mouse	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Chrysene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Dibenz(ah)anthracene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Fluoranthene	2.00E+03	Rodent	Acute LD50	--	--	--	2.00E+01	2.00E+01	2.00E+01	2.00E+01
Fluorene	1.25E+02	Mouse	Subchronic NOAEL	--	--	--	1.25E+02	1.25E+01	1.25E+01	1.25E+01
Indeno(1,2,3-cd)pyrene	1.33E+01	Mouse (BaP)	Chronic NOEL	--	--	--	1.33E+01	1.33E+00	1.33E+00	1.33E+00
Naphthalene	2.00E+02	Mouse	Chronic NOAEL	--	--	--	2.00E+02	2.00E+01	2.00E+01	2.00E+01
Phenanthrene	7.00E+02	Rodent	Acute LD50	--	--	--	7.00E+00	7.00E+00	7.00E+00	7.00E+00
Pyrene	9.90E+01	Rodent	Chronic Unspecified	--	--	--	9.90E-01	9.90E-01	9.90E-01	9.90E-01
Pesticides										
DDE, p,p'-	7.90E+02	Small Mammal Average	Acute LD50	8.25E+02	Bobwhite Quail	Acute LC50	7.90E+00	7.90E+00	7.90E+00	8.25E+01
DDT, p,p'-	1.91E+02	Small Mammal Average	Acute LD50	6.11E+02	Bobwhite Quail	Acute LC50	1.91E+00	1.91E+00	1.91E+00	6.11E+01
Miscellaneous SOCs										
Bis(2-ethylhexyl)phthalate	3.24E+04	Small Mammal Average	Acute LD50	--	--	--	3.24E+02	3.24E+02	3.24E+02	3.24E+02
Butylbenzyl phthalate	6.75E+03	Small Mammal Average	Acute LD50	--	--	--	6.75E+01	6.75E+01	6.75E+01	6.75E+01
VOCs										
Acetone	4.71E+03	Small Mammal Average	Acute LD50	4.00E+04	Japanese quail	Acute LC50	4.71E+01	4.71E+01	4.71E+01	4.00E+02
Methylene chloride	5.80E+02	Mouse	Chronic LOEL	--	--	--	5.80E+02	5.80E+01	5.80E+01	5.80E+01

*All units are mg/kg/day.

** Adjusted Benchmark Value = Unadjusted benchmark value / applicable uncertainty factors.

10 to account for an observed lethal endpoint.

10 to extrapolate from one species to another.

Source: ESE.

Table 5.2-1. Summary of Human Health Risks and HIs for Areas F, G, R, and S/T (Page 1 of 2)

Area	Scenario	Medium	Exposure Route	HI Adult	HI Child	Risk
F	Current Worker	Soil	Ingestion	< 0.1	NA	4E-07
			Dermal	< 0.1	NA	4E-07
			Inhalation	< 0.1	NA	4E-08
			TOTAL	< 0.1	NA	9E-07
	Future Worker	Groundwater	Ingestion	12	NA	3E-04
			Dermal	< 0.1	NA	1E-06
			Inhalation	< 0.1	NA	7E-06
			Subtotal	12	NA	3E-04
		Soil	Ingestion	< 0.1	NA	4E-06
			Dermal	< 0.1	NA	2E-06
			Inhalation	< 0.1	NA	4E-09
			Subtotal	< 0.1	NA	5E-06
			TOTAL	12	NA	3E-04
G	Current Recreational	Fish	Ingestion	< 0.1	< 0.1	9E-09
			Subtotal	< 0.1	< 0.1	9E-09
		Sediment	Ingestion	< 0.1	< 0.1	9E-10
			Dermal	< 0.1	< 0.1	4E-11
			Subtotal	< 0.1	< 0.1	9E-10
		Surface water	Ingestion	< 0.1	< 0.1	1E-08
			Dermal	< 0.1	< 0.1	3E-08
			Subtotal	< 0.1	< 0.1	4E-08
			TOTAL	< 0.1	< 0.1	5E-08
	Future worker	Groundwater	Ingestion	1.8	NA	5E-05
			Dermal	< 0.1	NA	2E-07
			Inhalation	< 0.1	NA	--
			Subtotal	1.8	NA	5E-05
		Sediment	Ingestion	< 0.1	NA	2E-10
			Dermal	< 0.1	NA	9E-12
			Subtotal	< 0.1	NA	2E-10
		Soil	Ingestion	< 0.1	NA	2E-11
			Dermal	< 0.1	NA	8E-12
			Inhalation	< 0.1	NA	4E-14
			Subtotal	< 0.1	NA	2E-11
		Surface water	Ingestion	< 0.1	NA	6E-10
			Dermal	< 0.1	NA	2E-09
			Subtotal	< 0.1	NA	2E-09
			TOTAL	1.8	NA	5E-05

Table 5.2-1. Summary of Human Health Risks and HIs for Areas F, G, R, and S/T (Continued, Page 2 of 2)

Area	Scenario	Medium	Exposure Route	HI Adult	HI Child	Risk
R	Future worker	Groundwater	Ingestion	1.3	NA	6E-05
			Dermal	< 0.1	NA	4E-07
			Inhalation	< 0.1	NA	1E-05
			Subtotal	1.3	NA	7E-05
		Soil	Ingestion	< 0.1	NA	2E-06
			Dermal	< 0.1	NA	1E-06
			Inhalation	< 0.1	NA	2E-08
			Subtotal	< 0.1	NA	3E-06
			TOTAL	1.3	NA	8E-05
S/T	Current worker	Soil	Ingestion	< 0.1	NA	9E-11
			Dermal	< 0.1	NA	1E-10
			Inhalation	< 0.1	NA	7E-13
			TOTAL	< 0.1	NA	2E-10
	Future worker	Groundwater	Ingestion	0.37	NA	6E-05
			Dermal	< 0.1	NA	2E-06
			Inhalation	3.5	NA	2E-05
			Subtotal	4	NA	8E-05
		Soil	Ingestion	< 0.1	NA	2E-09
			Dermal	< 0.1	NA	1E-09
			Inhalation	< 0.1	NA	2E-11
			Subtotal	< 0.1	NA	4E-09
			TOTAL	4	NA	8E-05

Note: NA = not applicable.
-- = no volatiles detected in groundwater.

Source: ESE.

Table 5.2-2. Summary of Media and COPCs Exceeding Risks* Based on the Human Risk Characterization

Area	Scenario	Medium	Exposure Route	COPC	Risk
F	Future Worker	Groundwater	Ingestion	Arsenic Carbon tetrachloride	3E-04 4E-06
				Subtotal	3E-04
			Inhalation	Chloroform Carbon tetrachloride	4E-06 3E-06
				Subtotal	7E-06
		TOTAL FOR GROUNDWATER			3E-04

*Chemicals are listed if an individual chemical exceeds 1E-06 risk within a medium which exceeds a cumulative risk a 1E-04.

Source: ESE.

Table 5.2-3. Summary of Media and COPCs Exceeding an HQ of 0.1 or HI of 1 Based on the Human Noncarcinogenic Risk Characterization

Area	Scenario	Medium	Exposure Route	COPC	HI Adult	HI Child
F	Future Worker	Groundwater	Ingestion	Mercury	1.1E+01	NA
				Arsenic	1.3E+00	NA
				Carbon Tetrachloride	1.1E-01	NA
				TOTAL HI	1.2E+01	NA
G	Future Worker	Groundwater	Ingestion	Cadmium	6.2E-01	NA
				Chromium	6.2E-01	NA
				Barium	2.9E-01	NA
				Arsenic	2.5E-01	NA
				TOTAL HI	1.8E+00	NA
R	Future Worker	Groundwater	Ingestion	Chromium	5.9E-01	NA
				Cadmium	2.4E-01	NA
				Arsenic	2.0E-01	NA
				TOTAL HI	1.0E+00	NA
S/T	Future Worker	Groundwater	Inhalation	Chlorobenzene	1.3E+00	NA
				Subtotal	1.3E+00	NA
			Ingestion	Benzene	1.9E-01	NA
				Chlorobenzene	1.7E-01	NA
				Subtotal	3.6E-01	NA
				TOTAL HI	1.7E+00	NA

Note: NA = not applicable, scenario not evaluated.

Source: ESE.

Table 5.4-1. Chemical Intakes and EQs for the Terrestrial Indicator Species at Areas F, G, R, and S/T

Site	Analyte	Cs (mg/kg)	Chemical Intake (mg/kg/day)				EQ			
			Mouse	Raccoon	Deer	Bobwhite	Mouse	Raccoon	Deer	Bobwhite
F	Acenaphthene	9.14E-01	3.66E-03	5.48E-03	9.14E-04	6.40E-04	2.09E-05	3.13E-04	5.22E-05	3.66E-05
	Acetone	8.00E-02	3.20E-04	4.80E-04	8.00E-05	5.60E-05	6.79E-06	1.02E-05	1.70E-06	1.40E-07
	Anthracene	8.97E-01	3.59E-03	5.38E-03	8.97E-04	6.28E-04	3.59E-06	5.38E-05	8.97E-06	6.28E-06
	Arsenic	7.06E-02	2.82E-04	4.24E-04	7.06E-05	4.94E-05	7.06E-06	1.06E-04	1.77E-05	1.24E-05
	Benz(a)anthracene	1.95E+00	7.80E-03	1.17E-02	1.95E-03	1.37E-03	5.86E-04	8.80E-03	1.47E-03	1.03E-03
	Benzo(a)pyrene	1.36E+00	5.44E-03	8.16E-03	1.36E-03	9.52E-04	4.09E-04	6.14E-03	1.02E-03	7.16E-04
	Benzo(b)fluoranthene	1.98E+00	7.92E-03	1.19E-02	1.98E-03	1.39E-03	5.95E-04	8.93E-03	1.49E-03	1.04E-03
	Benzo(ghi)perylene	1.02E+00	4.08E-03	6.12E-03	1.02E-03	7.14E-04	3.07E-04	4.60E-03	7.67E-04	5.37E-04
	Bis(2-ethylhexyl) phthalate	3.12E+00	1.25E-02	1.87E-02	3.12E-03	2.18E-03	3.85E-05	5.78E-05	9.63E-06	6.74E-06
	Cadmium	4.60E-04	1.84E-06	2.76E-06	4.60E-07	3.22E-07	2.07E-08	3.10E-07	5.17E-08	3.62E-08
	Chrysene	1.63E+00	6.52E-03	9.78E-03	1.63E-03	1.14E-03	4.90E-04	7.35E-03	1.23E-03	8.58E-04
	Dibenz(ah)anthracene	8.54E-01	3.42E-03	5.12E-03	8.54E-04	5.98E-04	2.57E-04	3.85E-03	6.42E-04	4.49E-04
	Fluoranthene	3.35E+00	1.34E-02	2.01E-02	3.35E-03	2.35E-03	6.70E-04	1.01E-03	1.68E-04	1.17E-04
	Fluorene	9.54E-01	3.82E-03	5.72E-03	9.54E-04	6.68E-04	3.05E-05	4.58E-04	7.63E-05	5.34E-05
	Indeno(1,2,3-cd)pyrene	1.10E+00	4.40E-03	6.60E-03	1.10E-03	7.70E-04	3.31E-04	4.96E-03	8.27E-04	5.79E-04
	Mercury	3.57E-03	1.43E-05	2.14E-05	3.57E-06	2.50E-06	4.46E-05	6.69E-04	1.12E-04	8.04E-06
	Methylene chloride	1.71E-02	6.84E-05	1.03E-04	1.71E-05	1.20E-05	1.18E-07	1.77E-06	2.95E-07	2.06E-07
	Naphthalene	7.83E-01	3.13E-03	4.70E-03	7.83E-04	5.48E-04	1.57E-05	2.35E-04	3.92E-05	2.74E-05
	Phenanthrene	2.20E+00	8.80E-03	1.32E-02	2.20E-03	1.54E-03	1.26E-03	1.89E-03	3.14E-04	2.20E-04
	Pyrene	2.99E+00	1.20E-02	1.79E-02	2.99E-03	2.09E-03	1.21E-02	1.81E-02	3.02E-03	2.11E-03
G	Acetone	1.54E-01	6.16E-04	9.24E-04	1.54E-04	1.08E-04	1.31E-05	1.96E-05	3.27E-06	2.69E-08
	Methylene chloride	1.20E-02	4.80E-05	7.20E-05	1.20E-05	8.40E-06	8.28E-08	1.24E-06	2.07E-07	1.45E-07
R	Acenaphthene	3.93E-01	1.57E-03	2.36E-03	3.93E-04	2.75E-04	8.98E-06	1.35E-04	2.25E-05	1.57E-05
	Acetone	1.60E-01	6.40E-04	9.60E-04	1.60E-04	1.12E-04	1.36E-05	2.04E-05	3.40E-06	2.80E-07
	Anthracene	4.60E-01	1.84E-03	2.76E-03	4.60E-04	3.22E-04	1.84E-06	2.76E-05	4.60E-06	3.22E-06
	Benz(a)anthracene	1.37E+00	5.48E-03	8.22E-03	1.37E-03	9.59E-04	4.12E-04	6.18E-03	1.03E-03	7.21E-04
	Benzo(a)pyrene	9.96E-01	3.98E-03	5.98E-03	9.96E-04	6.97E-04	3.00E-04	4.49E-03	7.49E-04	5.24E-04
	Benzo(b)fluoranthene	1.83E+00	7.32E-03	1.10E-02	1.83E-03	1.28E-03	5.50E-04	8.26E-03	1.38E-03	9.63E-04
	Benzo(ghi)perylene	6.69E-01	2.68E-03	4.01E-03	6.69E-04	4.68E-04	2.01E-04	3.02E-03	5.03E-04	3.52E-04
	Bis(2-ethylhexyl) phthalate	7.80E-01	3.12E-03	4.68E-03	7.80E-04	5.46E-04	9.63E-06	1.44E-05	2.41E-06	1.69E-06
	Butylbenzyl phthalate	7.80E-01	3.12E-03	4.68E-03	7.80E-04	5.46E-04	4.62E-05	6.93E-05	1.16E-05	8.09E-06
	Chrysene	9.96E-01	3.98E-03	5.98E-03	9.96E-04	6.97E-04	3.00E-04	4.49E-03	7.49E-04	5.24E-04
	Fluoranthene	2.21E+00	8.84E-03	1.33E-02	2.21E-03	1.55E-03	4.42E-04	6.63E-04	1.11E-04	7.74E-05
	Fluorene	3.93E-01	1.57E-03	2.36E-03	3.93E-04	2.75E-04	1.26E-05	1.89E-04	3.14E-05	2.20E-05
	Indeno(1,2,3-cd)pyrene	6.69E-01	2.68E-03	4.01E-03	6.69E-04	4.68E-04	2.01E-04	3.02E-03	5.03E-04	3.52E-04
	Methylene chloride	8.93E-03	3.57E-05	5.36E-05	8.93E-06	6.25E-06	6.16E-08	9.24E-07	1.54E-07	1.08E-07
	Phenanthrene	1.60E+00	6.40E-03	9.60E-03	1.60E-03	1.12E-03	9.14E-04	1.37E-03	2.29E-04	1.60E-04
	Pyrene	2.13E+00	8.52E-03	1.28E-02	2.13E-03	1.49E-03	8.61E-03	1.29E-02	2.15E-03	1.51E-03
S/T	Acetone	4.66E-01	1.86E-03	2.80E-03	4.66E-04	3.26E-04	3.96E-05	5.94E-05	9.89E-06	8.16E-07
	Bis(2-ethylhexyl) phthalate	1.97E-01	7.88E-04	1.18E-03	1.97E-04	1.38E-04	2.43E-06	3.65E-06	6.08E-07	4.26E-07
	DDE, p,p'-	4.33E-03	1.73E-05	2.60E-05	4.33E-06	3.03E-06	2.19E-06	3.29E-06	5.48E-07	3.67E-08
	DDT, p,p'-	2.76E-02	1.10E-04	1.66E-04	2.76E-05	1.93E-05	5.78E-05	8.67E-05	1.45E-05	3.16E-07
	Methylene chloride	1.88E-02	7.52E-05	1.13E-04	1.88E-05	1.32E-05	1.30E-07	1.94E-06	3.24E-07	2.27E-07

Note: Cs = Surface soil exposure concentration.
EQ = Chemical Intake [Soil Intake (Table 3.3-2) * Soil Exposure Concentration] / Adjusted Benchmark (Table 4.4-2)

Source: ESE.

Table 5.4-2. Ecotoxicity Quotients (EQs) for Potential Aquatic Indicator Species (Page 1 of 2)

Receptor	Chemical	Exposure Concentration (ppm)*	Estimated Porewater Conc. (mg/L)	(Kd) or (Koc / foc)	Benchmark Value*	Benchmark Source	EQ
Area F – Surface Water							
Daphnia magna	Barium	3.21E-02	–	–	5.00E+01 (a)	EPA, 1986	6.42E-04
Rana clamitans	Barium	3.21E-02	–	–	5.00E+01 (a)	EPA, 1986	6.42E-04
Scenedesmus sp.	Barium	3.21E-02	–	–	5.00E+01 (a)	EPA, 1986	6.42E-04
Area F – Sediment							
Chironomus sp.	Acetone	5.25E-02	3.50E+02 (b)	1.50E-04	NA		
	Benz(a)anthracene	3.29E-01	1.65E-04 (b)	2.00E+03	NA		
	Benzo(a)pyrene	3.40E-01	6.18E-06 (b)	5.50E+04	NA		
	Benzo(b)fluoranthene	3.03E-01	5.51E-05 (b)	5.50E+03	NA		
	Carbon tetrachloride	3.00E-03	2.73E-03 (b)	1.10E+00	3.52E+01 (c)	IRIS, 1994	7.75E-05
	Chrysene	3.25E-01	1.63E-04 (b)	2.00E+03	NA		
	Fluoranthene	2.92E-01	7.68E-04 (b)	3.80E+02	3.98E+00	IRIS, 1994	1.93E-04
	Methylene chloride	2.26E-02	2.57E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	2.33E-02
	Phenanthrene	3.63E-01	2.59E-03 (b)	1.40E+02	6.30E-03	IRIS, 1994	4.12E-01
	Pyrene	3.03E-01	7.97E-04 (b)	3.80E+02	NA		
	Arsenic	4.05E-02	2.03E-04 (d)	2.00E+02	1.90E-01	40 CFR 141,142	1.07E-03
	Barium	2.33E-01	3.88E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	7.77E-05
	Cadmium	6.80E-04	1.05E-04 (d)	6.50E+00	6.60E-01 (e)	EPA, 1986	1.59E-04
	Chromium	1.05E-01	1.24E-04 (d)	8.50E+02	2.10E-01	40 CFR 141,142	5.88E-04
	Lead	1.07E-01	1.19E-04 (d)	9.00E+02	2.58E-01 (f)	EPA, 1980c	4.61E-04
	Mercury	3.98E-03	3.98E-04 (d)	1.00E+01	2.00E-02 (g)	Rehwoldt et al., 1973	1.99E-02
Area G – Surface Water							
Daphnia magna	Acetone	1.59E-02	–	–	1.00E-01 (h)	Verscheuren, 1983	1.59E-01
	1,1-Dichloroethane	2.90E-03	–	–	NA		
	Methylene chloride	5.70E-03	–	–	2.24E+02 (i)	EPA, 1982	2.54E-05
	Trichloroethene	1.59E-02	–	–	6.40E+01 (j)	Canton and Adema, 1978	2.48E-04
	Xylenes	3.50E-03	–	–	1.00E+00 (h)	Verscheuren, 1983	3.50E-03
	Barium	8.07E-02	–	–	5.00E+01 (a)	EPA, 1986	1.61E-03
Rana clamitans	Acetone	1.59E-02	–	–	2.40E+00 (i,n)	HazardText, 1995	6.63E-03
	1,1-Dichloroethane	2.90E-03	–	–	2.02E+00 (o,t)	HazardText, 1995	1.44E-03
	Methylene chloride	5.70E-03	–	–	2.94E+00 (o,u)	HazardText, 1995	1.94E-03
	Trichloroethene	1.59E-02	–	–	4.50E+00 (i,n)	HazardText, 1995	3.53E-03
	Xylenes	3.50E-03	–	–	1.69E-01 (p,v)	HazardText, 1995	2.07E-02
	Barium	8.07E-02	–	–	5.00E+01 (a)	EPA, 1986	1.61E-03
Scenedesmus sp.	Acetone	1.59E-02	–	–	NA		
	1,1-Dichloroethane	2.90E-03	–	–	NA		
	Methylene chloride	5.70E-03	–	–	1.10E+01 (c)	IRIS, 1994	5.18E-04
	Trichloroethene	1.59E-02	–	–	2.19E+01 (a)	EPA, 1986	7.26E-04
	Xylenes	3.50E-03	–	–	NA		
	Barium	8.07E-02	–	–	5.00E+01 (a)	EPA, 1986	1.61E-03
Area G – Sediment							
Chironomus sp.	Acetone	1.13E-01	7.53E+02 (b)	1.50E-04	NA		
	Methylene chloride	2.12E-02	2.41E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	2.19E-02
	Arsenic	4.25E-03	2.13E-05 (d)	2.00E+02	1.90E-01	40 CFR 141,142	1.12E-04
	Barium	7.23E-02	1.21E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	2.41E-05
	Chromium	5.44E-02	6.40E-05 (d)	8.50E+02	2.10E-01	40 CFR 141,142	3.05E-04
Area R – Surface Water							
Daphnia magna	Bis(2-ethylhexyl) phthalate	1.69E-02	–	–	5.00E+00 (i)	EPA, 1980b	3.38E-03
	Chloroform	1.75E-02	–	–	2.89E+01 (k)	EPA, 1980 (AWQC)	6.06E-04
	Arsenic	1.05E-02	–	–	9.12E-01	EPA, 1980a	1.15E-02
	Barium	1.30E-01	–	–	5.00E+01 (a)	EPA, 1986	2.60E-03
Rana clamitans	Bis(2-ethylhexyl) phthalate	1.69E-02	–	–	2.00E-01 (q)	Wams, 1987	8.45E-02
	Chloroform	1.75E-02	–	–	3.51E+00 (r)	Birge et al., 1980	4.99E-03
	Arsenic	1.05E-02	–	–	4.00E+01 (s)	USFWS, 1988	2.63E-04
	Barium	1.30E-01	–	–	5.00E+01 (a)	EPA, 1986	2.60E-03
Scenedesmus sp.	Bis(2-ethylhexyl) phthalate	1.69E-02	–	–	3.60E-01 (l)	IRIS, 1994	4.69E-02
	Chloroform	1.75E-02	–	–	1.24E+00 (m)	IRIS, 1994	1.41E-02
	Arsenic	1.05E-02	–	–	2.32E+00	EPA, 1980a	4.53E-03
	Barium	1.30E-01	–	–	5.00E+01 (a)	EPA, 1986	2.60E-03

Table 5.4-2. Ecotoxicity Quotients (EQs) for Potential Aquatic Indicator Species (Continued, Page 2 of 2)

Receptor	Chemical	Exposure Concentration (ppm)*	Estimated Porewater Conc. (mg/L)	(Kd) or (Koc / foc)	Benchmark		EQ
					Value*	Source	
Area R – Sediment							
Chironomus sp.	Acetone	3.34E-02	2.23E+02 (b)	1.50E-04	NA		
	Benz(a)anthracene	7.75E-01	3.88E-04 (b)	2.00E+03	NA		
	Benzo(a)pyrene	8.80E-01	1.60E-05 (b)	5.50E+04	NA		
	Benzo(b)fluoranthene	1.81E+00	3.29E-04 (b)	5.50E+03	NA		
	Benzo(ghi)perylene	9.20E-01	5.75E-05 (b)	1.60E+04	NA		
	Bis(2-ethylhexyl) phthalate	7.85E-01	2.14E-03 (b)	3.66E+02	1.80E+01 (i)	EPA, 1980b	1.19E-04
	Carbon tetrachloride	1.07E-02	9.73E-03 (b)	1.10E+00	3.52E+01 (c)	IRIS, 1994	2.76E-04
	Chrysene	8.26E-01	4.13E-04 (b)	2.00E+03	NA		
	1,1-Dichloroethane	1.67E-02	2.88E-02 (b)	5.80E-01	NA		
	Fluoranthene	5.11E-01	1.34E-03 (b)	3.80E+02	3.98E+00	IRIS, 1994	3.38E-04
	Indeno(1,2,3-cd)pyrene	9.26E-01	5.79E-05 (b)	1.60E+04	NA		
	Methylene chloride	3.76E-02	4.27E-01 (b)	8.80E-02	1.10E+01 (c)	IRIS, 1994	3.88E-02
	Pyrene	7.02E-01	1.85E-03 (b)	3.80E+02	NA		
	Arsenic	1.77E-02	8.85E-05 (d)	2.00E+02	1.90E-01	40 CFR 141,142	4.66E-04
	Barium	1.41E-01	2.35E-03 (d)	6.00E+01	5.00E+01 (a)	EPA, 1986	4.70E-05
	Cadmium	6.12E-03	9.42E-04 (d)	6.50E+00	6.60E-01 (e)	EPA, 1986	1.43E-03
	Chromium	3.90E-02	4.59E-05 (d)	8.50E+02	2.10E-01	40 CFR 141,142	2.18E-04
	Lead	5.90E-02	6.56E-05 (d)	9.00E+02	2.58E-01 (f)	EPA, 1980c	2.54E-04
	Mercury	1.07E-03	1.07E-04 (d)	1.00E+01	2.00E-02 (g)	Reifwoldt et al., 1973	5.35E-03

Note:
NA = benchmark not located in available literature.
conc. = concentration.
– = not applicable.
sp. = species.
mg/kg = milligram per kilogram.
ppm = part per million.
mg/L = milligram per liter.
Kd = organic partition coefficient.
Koc = organic carbon partition coefficient.
foc = soil organic carbon fraction.

(a) Value available in EPA's Quality Criteria for Water (1986), but not a criteria value.
(b) Porewater conc. = Sediment conc. / (Koc * foc). foc of 0.01 is used for site soils (Rosenblatt and Small, 1981).
(c) Acute lowest effect concentration (LEC).
(d) Porewater conc. = Sediment conc. / Kd (Tetra Tech, 1986).
(e) Hardness-dependent value for freshwater organisms based on 50 mg/L calcium carbonate ($CaCO_3$).
(f) 10-day LC50 (concentration lethal to 50 percent of organisms), hardness = 47 mg/L.
(g) Acute LC50/EC50 for mercuric nitrate, as mercury. EC50 is the effective concentration for 50% of organisms.
(h) 24-hour median tolerance limit (TLm).
(i) 48-hour LC50.
(j) Species acute value.
(k) 48-hour static bioassay.
(l) Chronic ambient water quality criteria (AWQC).
(m) Chronic LEC.
(n) Adjusted value for clawed toad.
(o) Adjusted value for the guppy.
(p) Adjusted value for the goldfish.
(q) Adjusted value for Xenopus laevis (toad).
(r) Adjusted value for toads/frogs.
(s) Adjusted value for narrow-mouthed toad.
(t) 7-day LC50.
(u) 14-day LC50.
(v) 96-hour LC50.

* Surface water and porewater concentrations are in mg/L; sediment concentrations are in mg/kg.

Source: ESE

Table 5.4-3. Comparison of COPC Concentrations in Surface Water to Ambient Water Quality Criteria

Site	Chemical	Exposure Concentration (EC) (mg/L)	Chronic AWQC (mg/L)	Does EC Exceed AWQC?
F	Barium	3.21E-02	NA	--
G	Acetone	1.59E-02	NA	--
	1,1-Dichloroethane	2.90E-03	NA	--
	Methylene chloride	5.70E-03	1.10E+01 (a)	No
	Trichloroethene	1.59E-02	2.19E+01	No
	Xylenes	3.50E-03	NA	--
	Barium	8.07E-02	NA	--
R	Bis(2-ethylhexyl) phthalate	1.69E-02	NA	--
	Chloroform	1.75E-02	1.24E+00	No
	Arsenic	1.05E-02	1.80E+00	No
	Barium	1.30E-01	NA	--

Note: NA = not available.
-- = cannot be calculated due to the lack of an AWQC.

(a) No chronic value available; given value is the acute AWQC.

Source: ESE.

Table 5.5-1. Comparison of Normal and Lognormal Distribution-Based UCL95s for Groundwater at Area F

Constituent	Normal UCL95 (b)	Lognormal UCL95 (c)	Maximum Conc'n (d)	Smaller of Lognormal UCL95 and Maximum (e)	Diff. (f)	Ratio of Lognormal or Maximum to Normal (g)	Normal Prob<W (h)	Lognormal Prob<W (i)	Distribution	Distribution with Better Fit
Volatile Organics (mg/L) (a)										
Acetone	3.21E-02	4.70E-02	8.40E-02	4.70E-02 (UCL95)	1.49E-02	1.46	0.0001	0.0001	Unknown	Lognormal
Chloroform	5.96E-03	5.49E-03	1.60E-02	5.49E-03 (UCL95)	-4.65E-04	0.92	0.0001	0.0001	Unknown	Lognormal
Carbon tetrachloride	7.97E-03	8.71E-03	1.70E-02	8.71E-03 (UCL95)	7.39E-04	1.09	0.0002	0.0023	Unknown	Lognormal
Trichloroethene	3.29E-03	3.28E-03	5.00E-03	3.28E-03 (UCL95)	-1.35E-05	1.00	0.0001	0.0001	Unknown	Lognormal
Metals (Total, mg/L)										
Arsenic	4.02E-02	1.43E-01	1.10E-01	1.10E-01 (Maximum)	6.98E-02	2.74	0.0004	0.0865*	Lognormal	Lognormal
Barium	9.13E-02	8.56E-02	3.20E-01	8.56E-02 (UCL95)	-5.70E-03	0.94	0.0001	0.0051	Unknown	Lognormal
Cadmium	3.69E-03	3.47E-03	7.60E-03	3.47E-03 (UCL95)	-2.18E-04	0.94	0.0001	0.0001	Unknown	Same
Chromium	6.86E-03	7.19E-03	1.61E-02	7.19E-03 (UCL95)	3.29E-04	1.05	0.0001	0.0004	Unknown	Lognormal
Lead	1.28E-02	1.00E-02	5.24E-02	1.00E-02 (UCL95)	-2.72E-03	0.79	0.0001	0.0001	Unknown	Lognormal
Mercury	3.26E-01	3.53E+00	1.40E+00	1.40E+00 (Maximum)	1.07E+00	4.29	0.0001	0.0001	Unknown	Lognormal

Notes:

(a) mg/L = milligrams per liter

(b) Normal UCL95 is the upper 95% confidence limit of the arithmetic mean for a normal distribution

(c) Lognormal UCL95 is the upper 95% confidence limit of the arithmetic mean for a lognormal distribution

(d) Maximum concentration is listed because the maximum concentration should be used as the exposure concentration if the UCL95 is greater than the maximum concentration

(e) Maximum concentration should be used as the exposure concentration if the UCL95 is greater than the maximum concentration

(f) If lognormal UCL95 is less than the maximum concentration, then Difference = Lognormal UCL95 - Normal UCL95.
If lognormal UCL95 is greater than the maximum concentration, then Difference = Lognormal UCL95 - Maximum Concentration.

(g) If lognormal UCL95 is less than the maximum concentration, then Ratio = Lognormal UCL95 / Normal UCL95.
If lognormal UCL95 is greater than the maximum concentration, then Ratio = Maximum Concentration / Normal UCL95.

(h) The Shapiro-Wilks test (aka W-test) statistic was used to determine if the nontransformed data come from a normal distribution.
Values greater than or equal to 0.05 indicate that the data come from a normal distribution (note that none of the concentrations are normally distributed)

(i) The W-test statistic was used to determine if the natural logarithm-transformed data come from a normal distribution.
Values greater than or equal to 0.05 indicate that the data come from a lognormal distribution (note that only arsenic concentrations are lognormally distributed)

Sources: G&M, 1991 and 1992; ESE, 1995.

Table 5.5-2. Intakes and RMEHIs for the Two Largest Observed Differences in UCL95s for Groundwater at Area F

		Normal Distribution-Based UCL_{95} Equation		Maximum Concentration (Less Than Lognormal Distribution-Based UCL_{95} Equation)	
Analyte	Pathway	Intake	RMEHI	Intake	RMEHI
Arsenic	Oral	3.9E-4	1.3E+0	1.4E-3	4.7E+0
	Dermal	1.4E-6	4.8E-3	5.1E-6	1.7E-2
Mercury	Oral	3.2E-3	1.1E+1	1.4E-2	4.6E+1
	Dermal	1.2E-5	3.9E-2	5.0E-5	1.7E-1

Table 5.6-1. Uncertainty Associated with the RA Process

BRA Component	Potential for Uncertainty
Hazard Identification	. Initial selection of COPCs . Tentatively identified compounds . Limited chemical monitoring data . Extraneous contamination introduced during sampling or analysis . Use of inappropriate analytical methods or method detection limits
Exposure Assessment	. Selection of site-specific exposure pathways . Estimation of exposure concentrations with limited monitoring data . Estimation of exposure to multiple substances . Estimation of inhalation exposure to volatiles, suspended dust . Estimation of exposure factors and parameters . Use of current data for future exposure conditions . Use of normal distribution-based UCL_{95} equation instead of lognormal distribution-based UCL_{95} equation
Toxicity Assessment	. Selection of toxicity values . Factors used to derive toxicity values including interspecies extrapolation . Weight of evidence for human toxicity . Derivation of carcinogenic slope factors . Extrapolation of less-than-lifetime exposure to lifetime cancer risks . Interaction of multiple substances . Use of high to low dose extrapolation models
Risk Characterization	. Addition of risks across multiple exposure pathways . Addition of risks from multiple substances . For PAHs, in the absence of CSFs for most of the PAHs, the CSF of a highly toxic PAH is adopted for all PAHs in the class

Source: ESE.

Table 6.1-1. Preliminary RGOs Associated with Future Worker Oral and Inhalation Exposure to Contaminants in Groundwater at Areas F, G, R, and S/T

Constituent	Applicable Sites	Carcinogenic RGOs (mg/L)									Noncarcinogenic RGOs (mg/L)									MCL
		Oral			Inhalation			Oral + Inhalation			Oral			Inhalation			Oral + Inhalation			
		1E-06	1E-05	1E-04	1E-06	1E-05	1E-04	1E-06	1E-05	1E-04	0.1	1	10	0.1	1	10	0.1	1	10	
Potential Carcinogens																				
Arsenic	F, G, R	2E-04	2E-03	2E-02	na	na	na	2E-04	2E-03	2E-02	3.1E-03	3.1E-02	3.1E-01	na	na	na	3.1E-03	3.1E-02	3.1E-01	5.0E-02 (a)
Benzene	S/T	1E-02	1E-01	1E+00	5E-03	5E-02	5E-01	3E-03	3E-02	3E-01	2.0E-01	2.0E+00	2.0E+01	2.7E-04	2.7E-03	2.7E-02	2.7E-04	2.7E-03	2.7E-02	5.0E-03
Carbon tetrachloride	F	2E-03	2E-02	2E-01	2E-03	2E-02	2E-01	1E-03	1E-02	1E-01	7.2E-03	7.2E-02	7.2E-01	nd	nd	nd	7.2E-03	7.2E-02	7.2E-01	5.0E-03
Chloroform	F	5E-02	5E-01	5E+00	2E-03	2E-02	2E-01	2E-03	2E-02	2E-01	1.0E-01	1.0E+00	1.0E+01	nd	nd	nd	1.0E-01	1.0E+00	1.0E+01	1.0E-01 (b)
Lead	G, R, S/T	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	1.5E-02 (c)
Noncarcinogens																				
Barium	G	--	--	--	--	--	--	--	--	--	7.2E-01	7.2E+00	7.2E+01	na	na	na	7.2E-01	7.2E+00	7.2E+01	2.0E+00
Cadmium	G, R	--	--	--	--	--	--	--	--	--	5.1E-03	5.1E-02	5.1E-01	na	na	na	5.1E-03	5.1E-02	5.1E-01	5.0E-03
Chlorobenzene	S/T	--	--	--	--	--	--	--	--	--	2.0E-01	2.0E+00	2.0E+01	2.7E-02	2.7E-01	2.7E+00	2.4E-02	2.4E-01	2.4E+00	1.0E-01
Chromium	G, R	--	--	--	--	--	--	--	--	--	5.1E-02	5.1E-01	5.1E+00	na	na	na	5.1E-02	5.1E-01	5.1E+00	1.0E-01 (d)
Mercury	F	--	--	--	--	--	--	--	--	--	3.1E-03	3.1E-02	3.1E-01	na	na	na	3.1E-03	3.1E-02	3.1E-01	2.0E-03

Note: na = inhalation of metals from groundwater is not applicable.
nd = no inhalation CSF and/or RfD determined.

(a) National Interim Primary Drinking Water Regulation.
(b) Value for total trihalomethanes.
(c) EPA drinking water action level.
(d) Value based on total chromium.

Source: ESE.

Table 6.1-2. Preliminary RGOs Associated with Future Worker Exposure at Areas F, G, R, and S/T Based on Cumulative Risk of 1E-4 or HI of 1

Constituent	RGO; Oral + Inhalation * (mg/L)		MCL (mg/L)	Exposure Concentration (mg/L)	Does Exposure Concentration Exceed Indicated Criterion?		
	Carcinogenic **	Noncarcinogenic			Risk-based RGO	HI-based RGO	MCL
Area F							
Potential Carcinogens							
Arsenic	2E-02	3.1E-02	5.0E-02 (a)	4.02E-02	Yes	Yes	No
Carbon tetrachloride	1E-01	7.2E-02	5.0E-03	8.00E-03	No (e)	No	Yes
Chloroform	2E-01	1.0E+00	1.0E-01 (b)	6.00E-03	No (e)	No	No
Noncarcinogens							
Mercury	--	3.1E-02	2.0E-03	3.26E-01	--	Yes	Yes
Area G							
Potential Carcinogens							
Arsenic	2E-02	3.1E-02	5.0E-02 (a)	7.60E-03	No (e)	No (f)	No
Lead	--	--	1.50E-02 (c)	1.39E-01	--	--	Yes
Noncarcinogens							
Barium	--	7.2E+00	2.0E+00	2.07E+00	--	No (f)	Yes
Cadmium	--	5.1E-02	5.0E-03	3.19E-02	--	No (f)	Yes
Chromium	--	5.1E-01	1.0E-01 (d)	3.19E-01	--	No (f)	Yes
Area R							
Potential Carcinogens							
Arsenic	2E-02	3.1E-02	5.0E-02 (a)	6.04E-03	No (e)	No (f)	No
Lead	--	--	1.50E-02 (c)	5.00E-02	--	--	Yes
Noncarcinogens							
Cadmium	--	5.1E-02	5.0E-03	1.24E-02	--	No (f)	Yes
Chromium	--	5.1E-01	1.0E-01 (d)	3.00E-01	--	No (f)	Yes
Area S/T							
Potential Carcinogens							
Benzene	3E-01	2.5E-03	5.0E-03	5.79E-03	No (e)	Yes	Yes
Lead	--	--	1.50E-02 (c)	2.10E-02	--	--	Yes
Noncarcinogens							
Chlorobenzene	--	2.4E-01	1.0E-01	3.48E-01	--	Yes	Yes

Note: na = inhalation of metals from groundwater is not applicable.
nd = no inhalation CSF/RfD determined.

(a) National Interim Primary Drinking Water Regulation.
(b) Value for total trihalomethanes.
(c) EPA drinking water action level.
(d) Value based on total chromium.
(e) Individual risk is > 1E-6 but the cumulative risk for the medium is < 1E-4.
(f) Individual HQ is < 1 but the cumulative HI for the medium is > 1.

* RGO for metals based on oral exposure, only.
** RGO based on cumulative risk of 1E-4.

Source: ESE.

LIST OF FIGURES

Figure	
1.1-1	Location of Redstone Arsenal, Alabama
1.1-2	Location of 16 Study Areas Included in Baseline Risk Assessments at Redstone Arsenal, Alabama
1.2-1	Unit 3/Area F - Closed Disposal Ponds - Study Area Location
1.2-2	Unit 3/Area G - Closed Landfill - Study Area Location
1.2-3	Unit 3/Area R - Closed Landfill - Study Area Location
1.2-4	Unit 3/Area S and T - Closed Disposal Trenches - Study Area Location
1.3-1	Unit 3/Area F - Closed Disposal Ponds - Excavation and Soil Sampling Locations
1.3-2	Unit 3/Area F - Closed Disposal Ponds - Groundwater and Surface Water Sampling Locations
1.3-3	Unit 3/Area G - Closed Landfill - Excavation and Soil Sampling Locations
1.3-4	Unit 3/Area G - Closed Landfill - Groundwater and Surface Water Sampling Locations
1.3-5	Unit 3/Area R - Closed Landfill - Excavation and Soil Sampling Locations
1.3-6	Unit 3/Area R - Closed Landfill - Groundwater and Surface Water Sampling Locations
1.3-7	Unit 3/Area S and T - Closed Disposal Trenches - Excavation and Soil Sampling Locations
1.3-8	Unit 3/Area S and T - Closed Disposal Trenches - Groundwater and Surface Water Sampling Locations
1.4-1	Conceptual Study Area Model for RSA Study Area F

LIST OF FIGURES
(Continued, Page 2 of 2)

Figure	
1.4-2	Conceptual Study Area Model for RSA Study Area G
1.4-3	Conceptual Study Area Model for RSA Study Area R
1.4-4	Conceptual Study Area Model for RSA Study Area S and T
3.1-1	Location of Offsite Wells

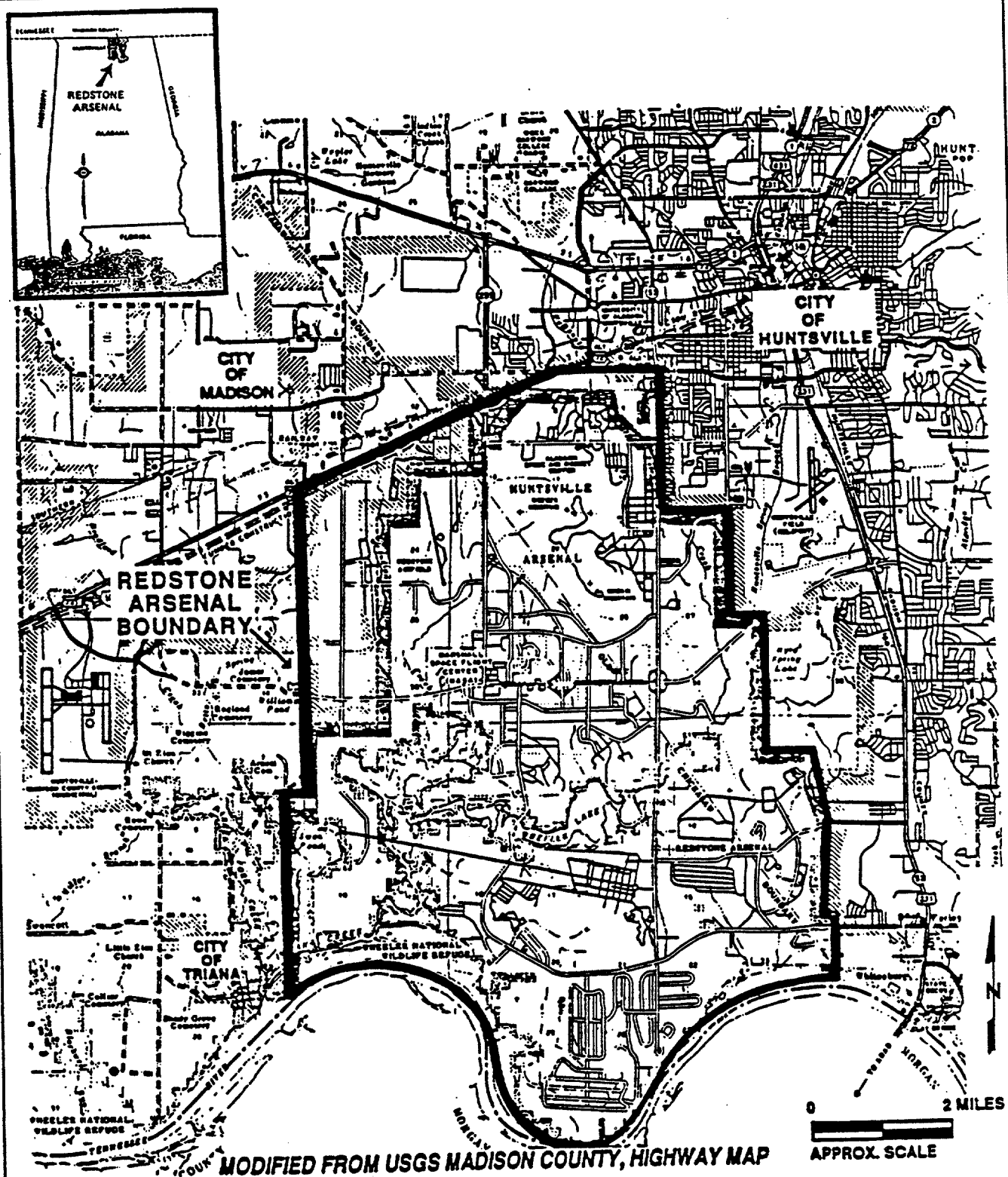


Figure 1.1-1
LOCATION OF REDSTONE ARSENAL, ALABAMA

SOURCE: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

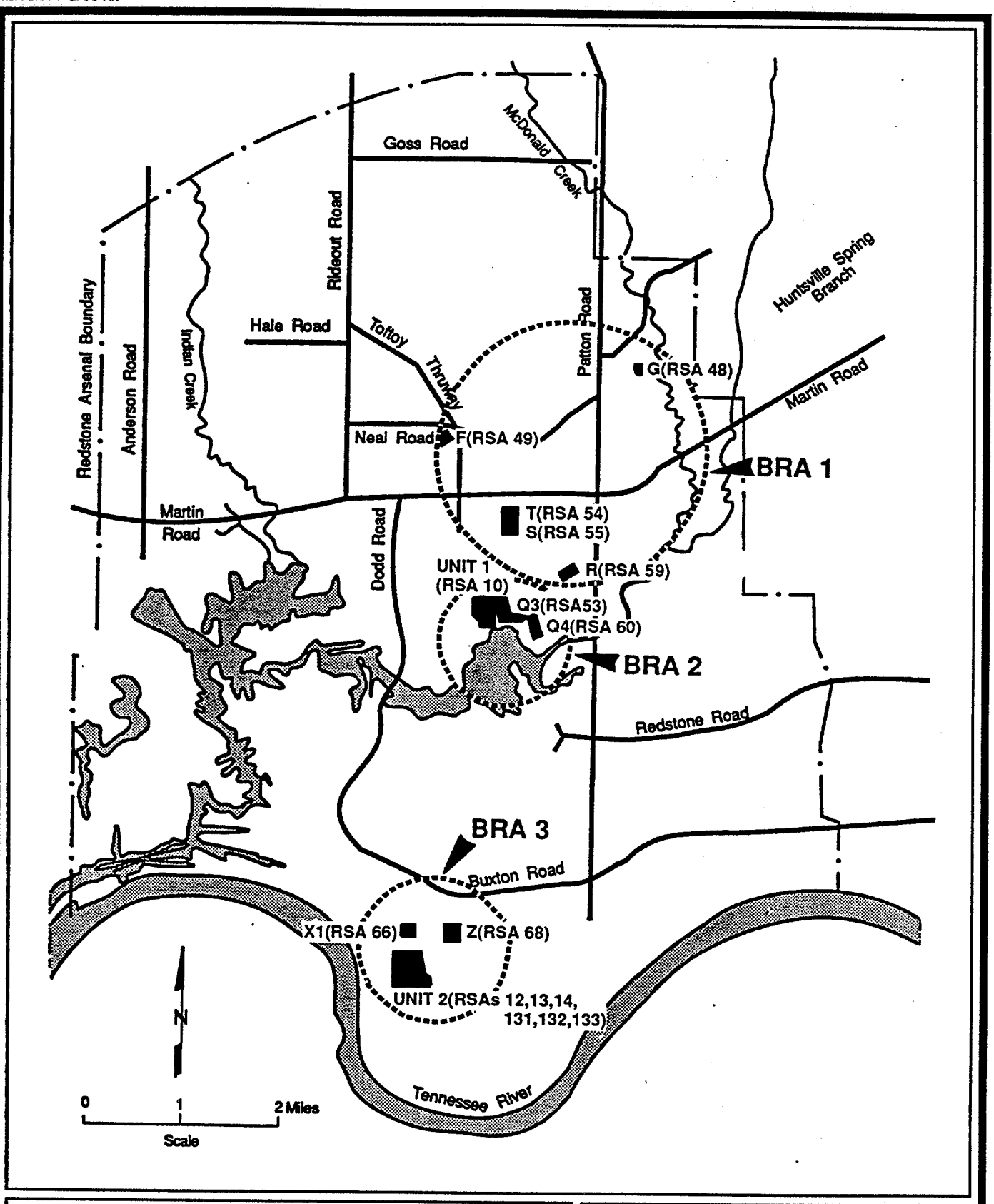


Figure 1.1-2
LOCATION OF 16 STUDY AREAS INCLUDED IN BASELINE RISK ASSESSMENTS AT REDSTONE ARSENAL, ALABAMA

SOURCES: G&M, 1991; ESE.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

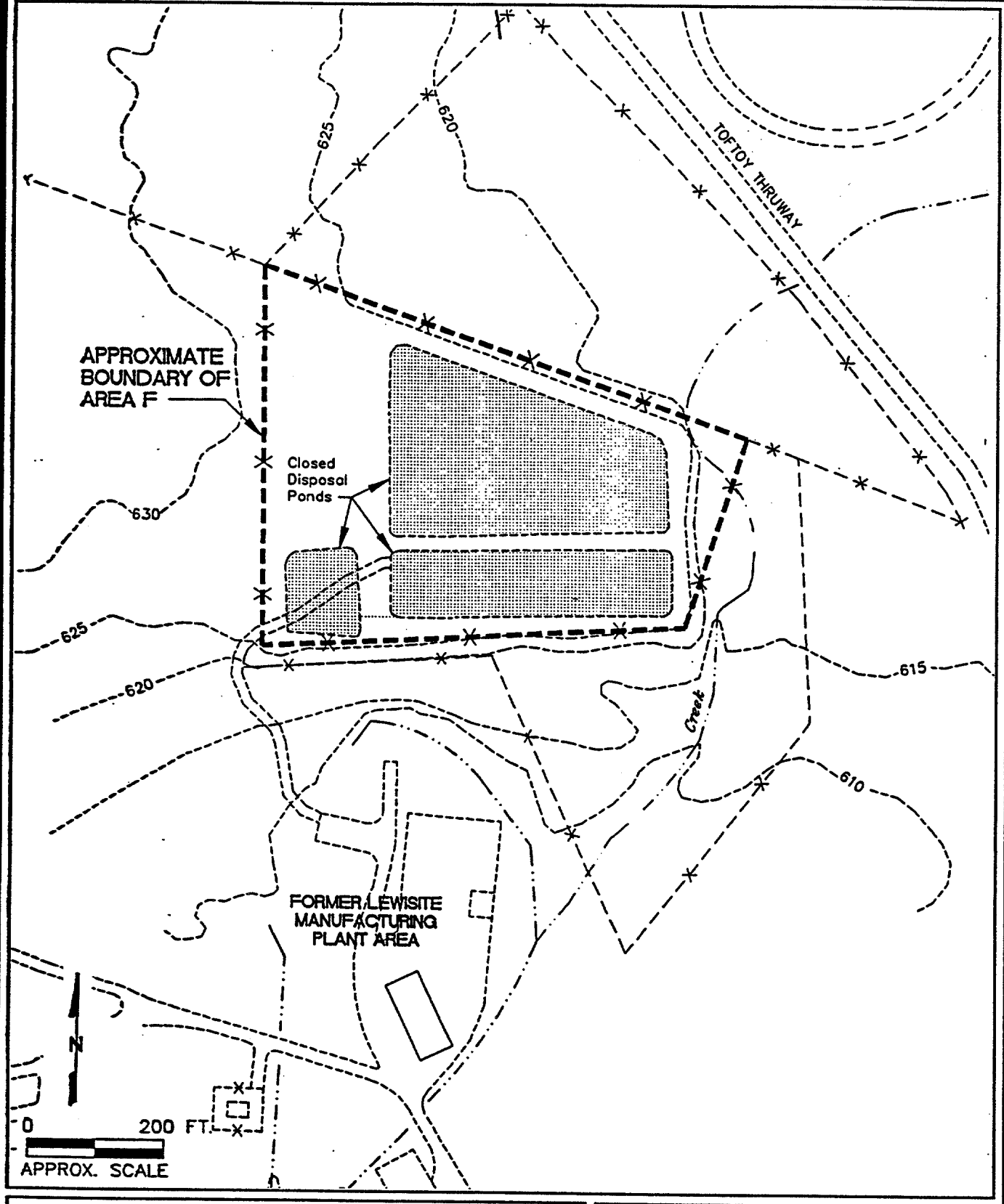


Figure 1.2-1
AREA F - CLOSED DISPOSAL PONDS

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

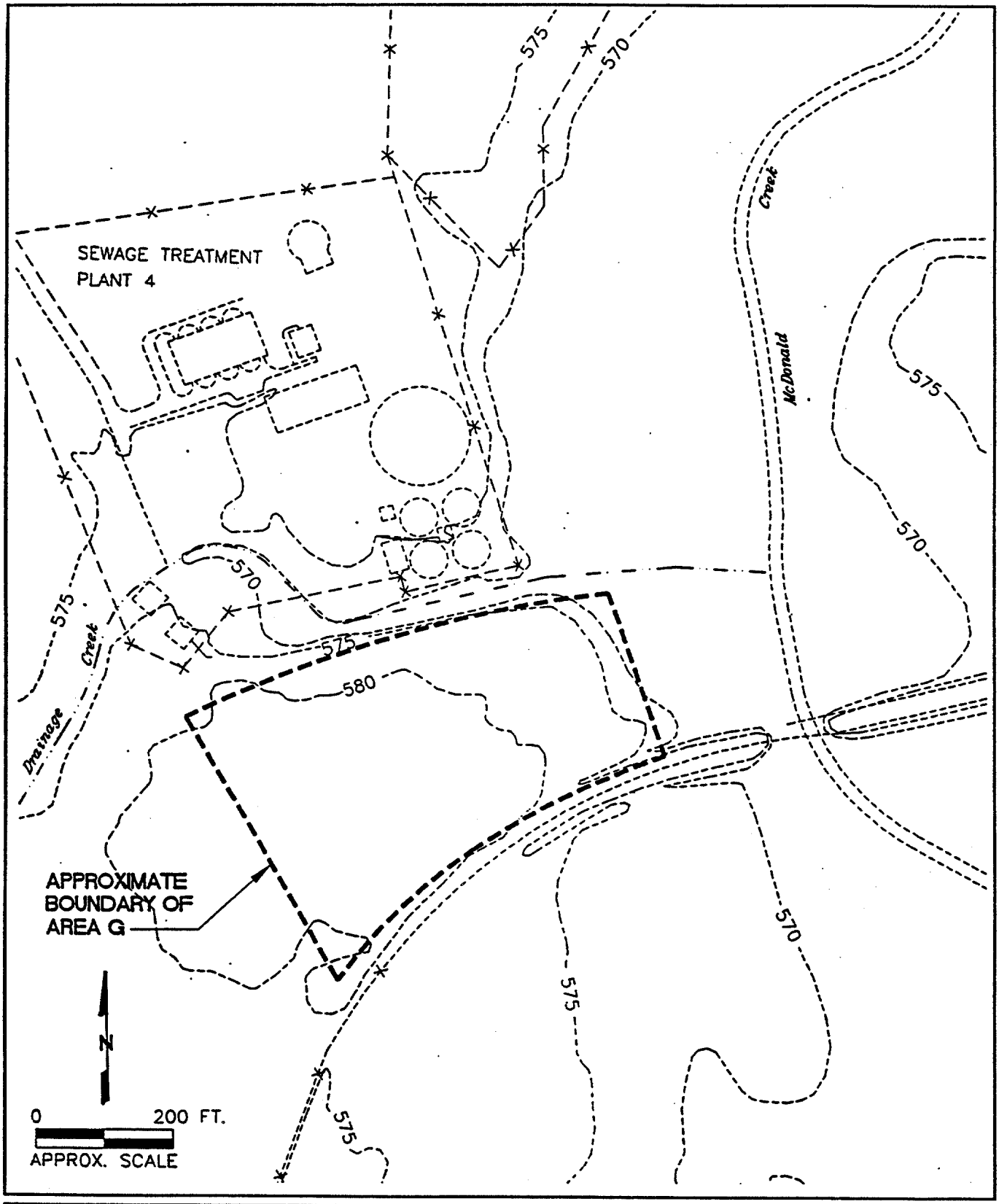


Figure 1.2-2
AREA G - CLOSED LANDFILL

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

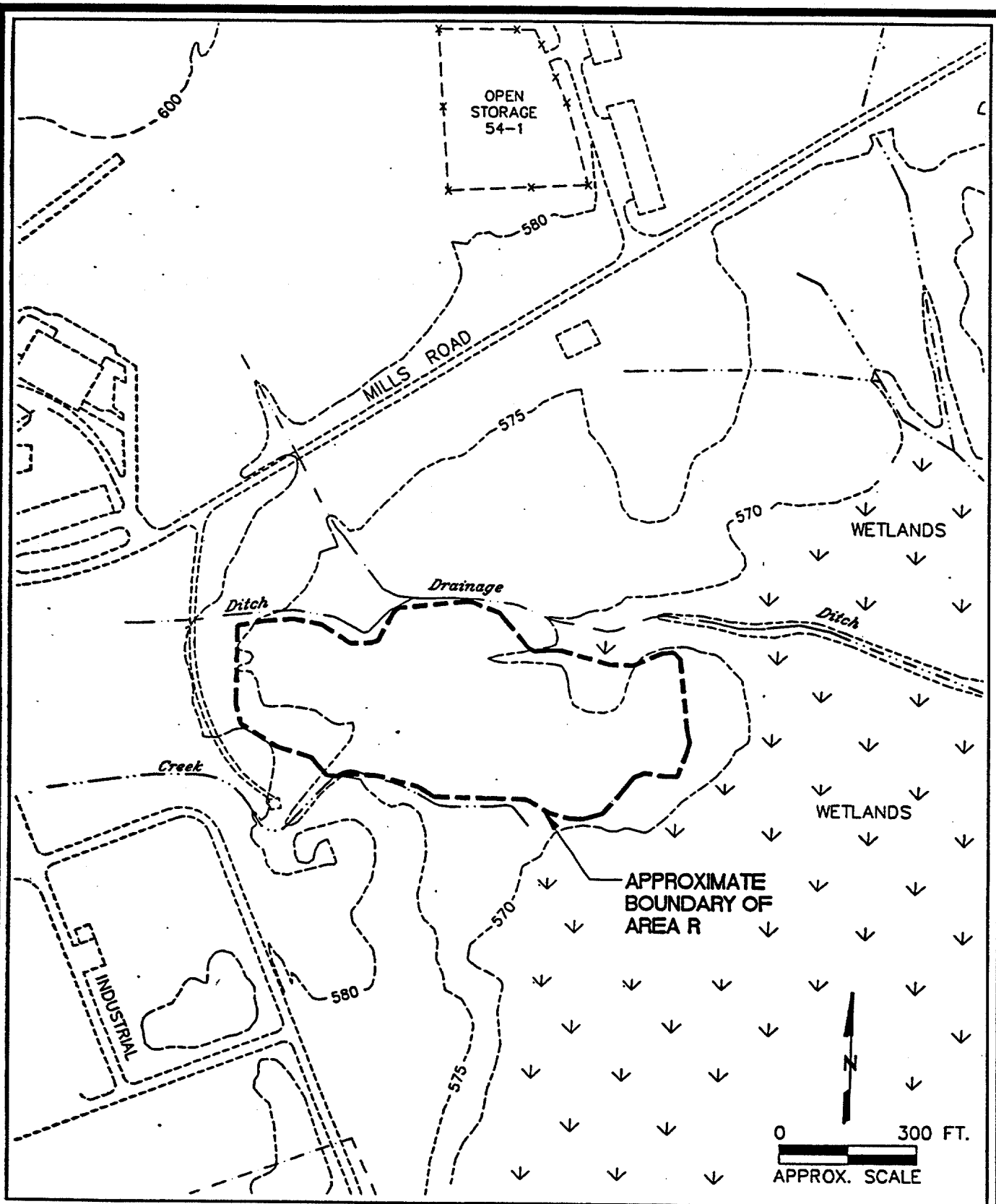


Figure 1.2-3
AREA R - CLOSED LANDFILL

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

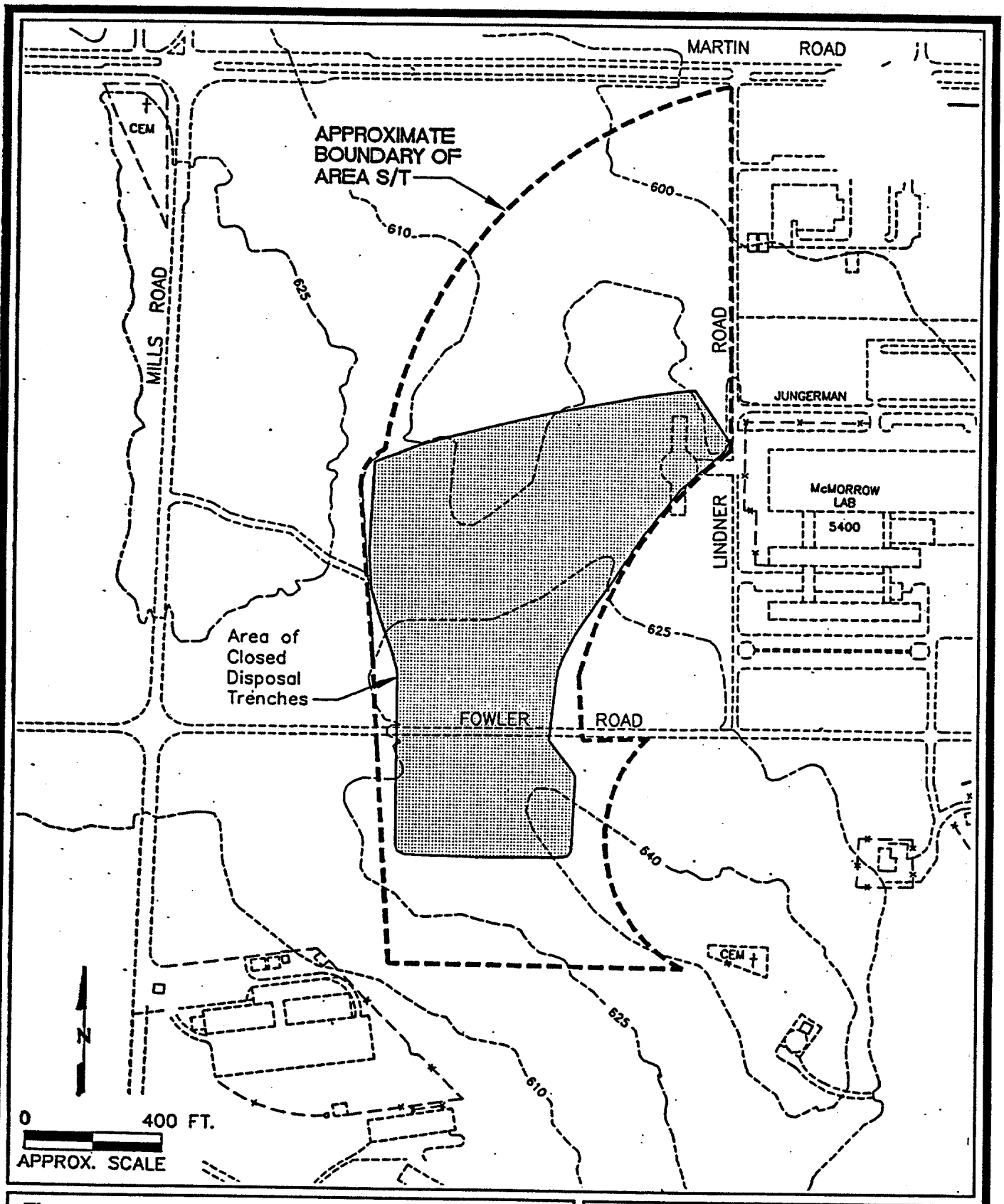


Figure 1.2-4
AREA S/T - CLOSED DISPOSAL TRENCHES

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

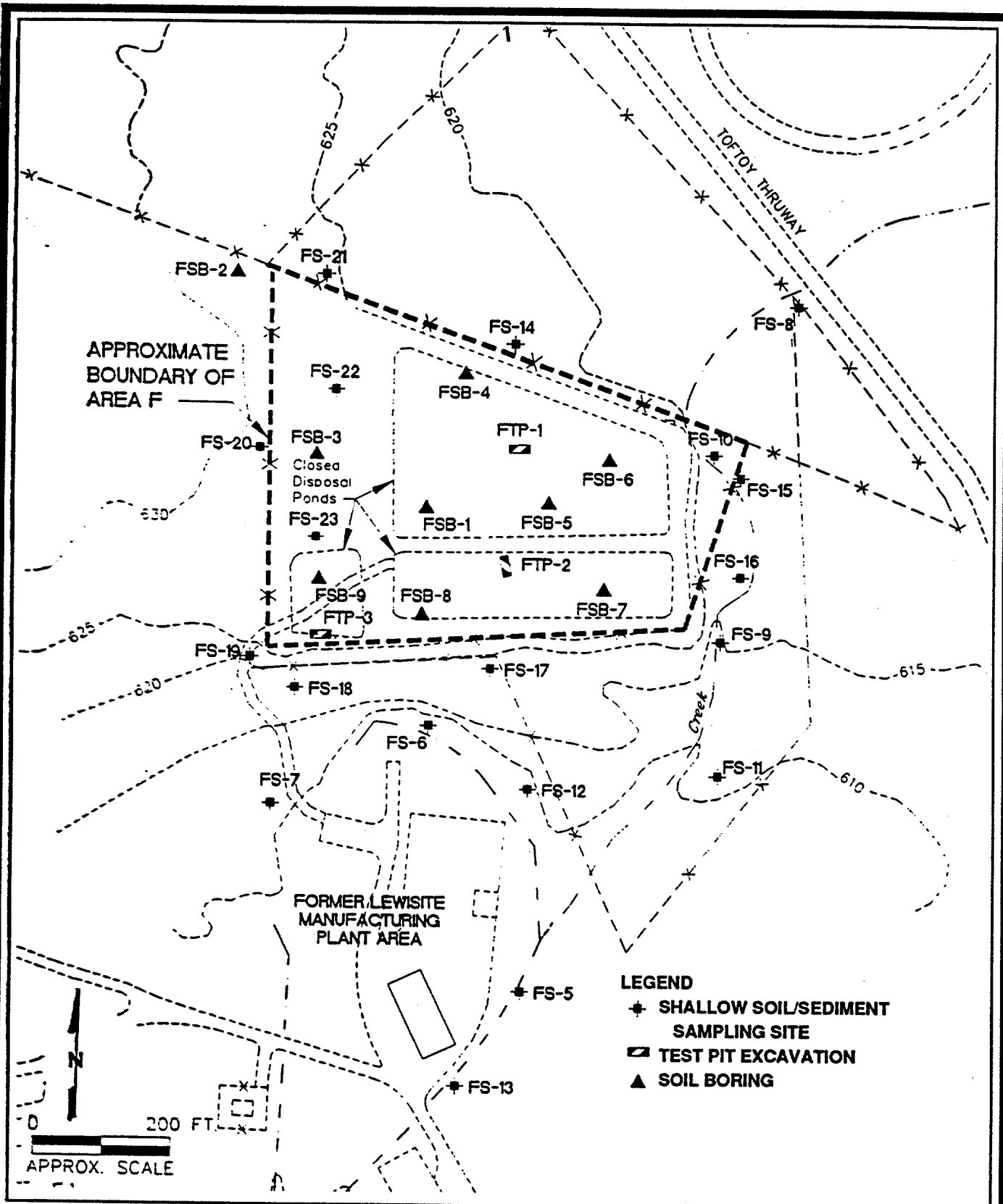


Figure 1.3-1
AREA F - CLOSED DISPOSAL PONDS - EXCAVATION AND SOIL SAMPLING LOCATIONS

SOURCES: G&M, 1992.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

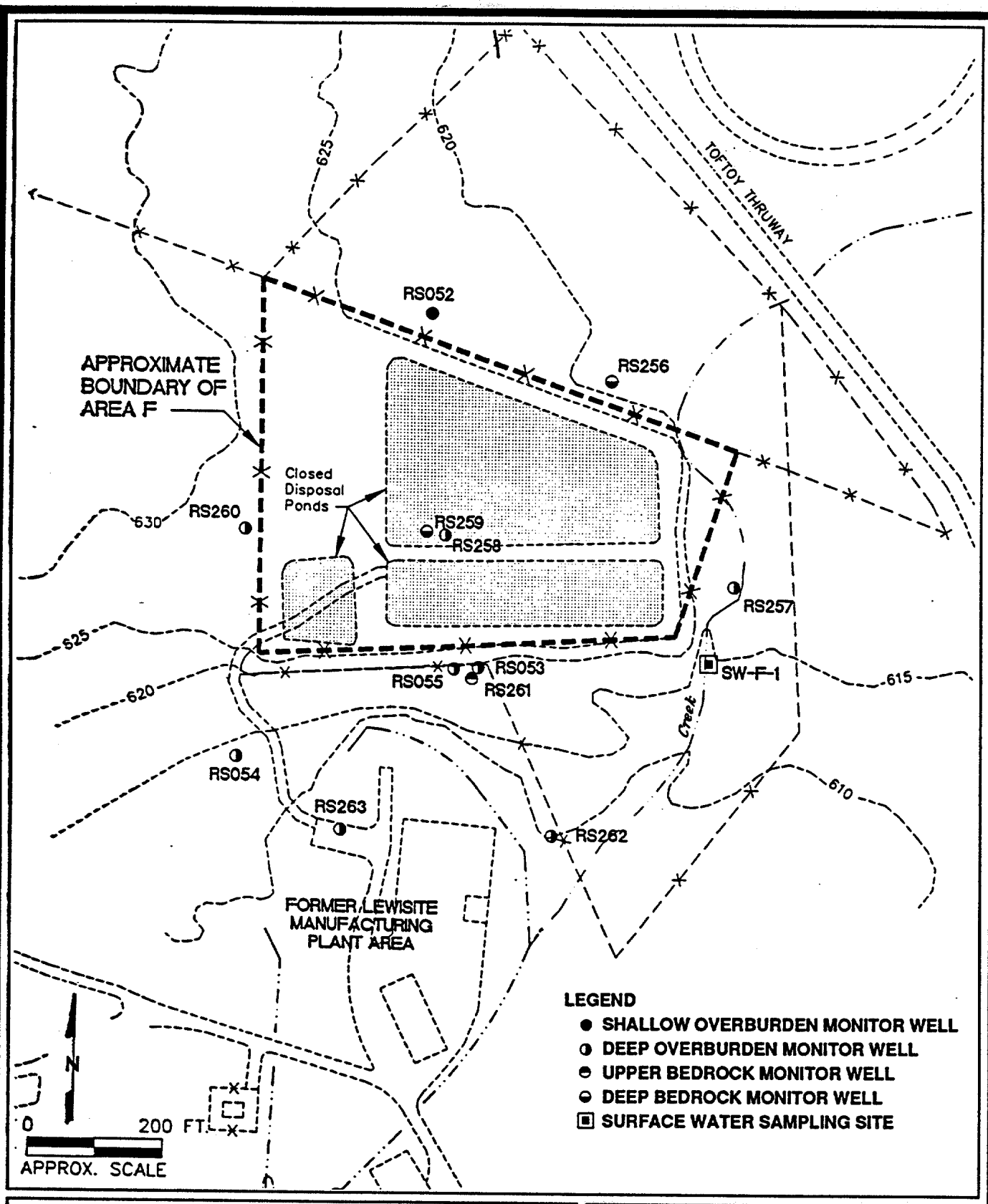


Figure 1.3-2
AREA F - CLOSED DISPOSAL PONDS - GROUNDWATER AND SURFACE WATER SAMPLING LOCATIONS

SOURCES: G&M, 1992.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS HUNTSVILLE DIVISION

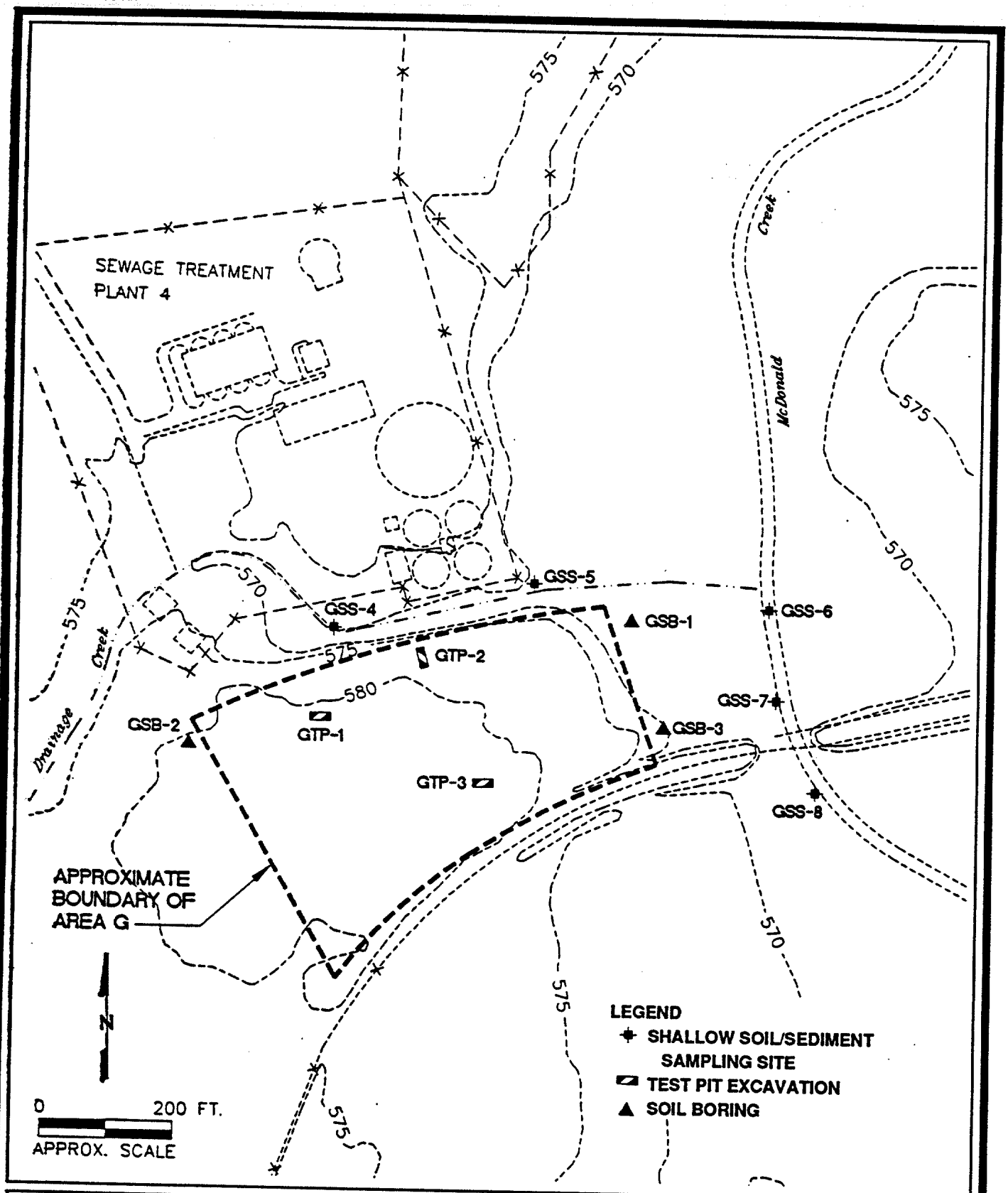


Figure 1.3-3
AREA G - CLOSED LANDFILL - EXCAVATION AND SOIL SAMPLING LOCATIONS

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

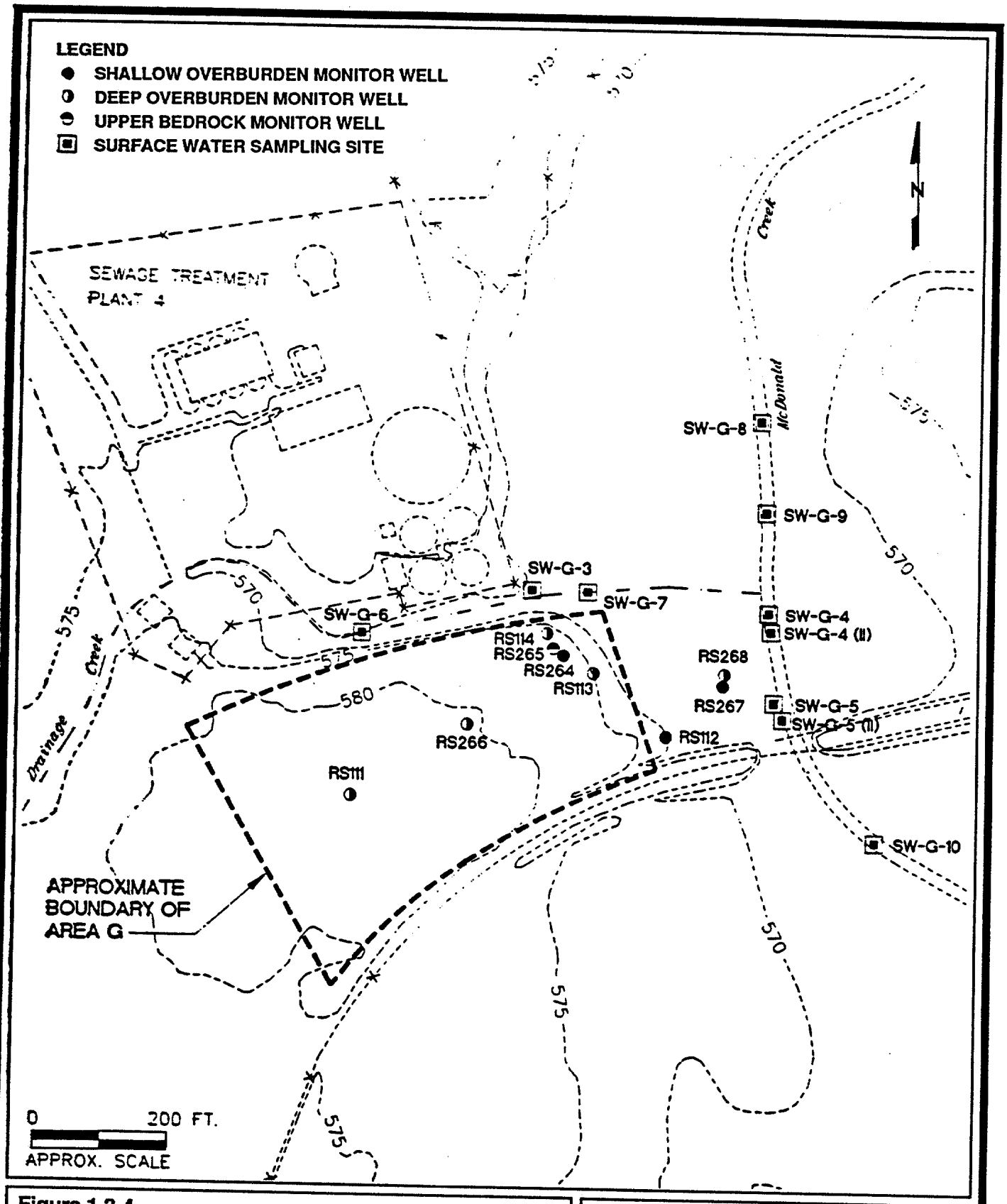


Figure 1.3-4
AREA G - CLOSED LANDFILL - GROUNDWATER AND SURFACE SAMPLING LOCATIONS

SOURCES: G&M, 1992.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

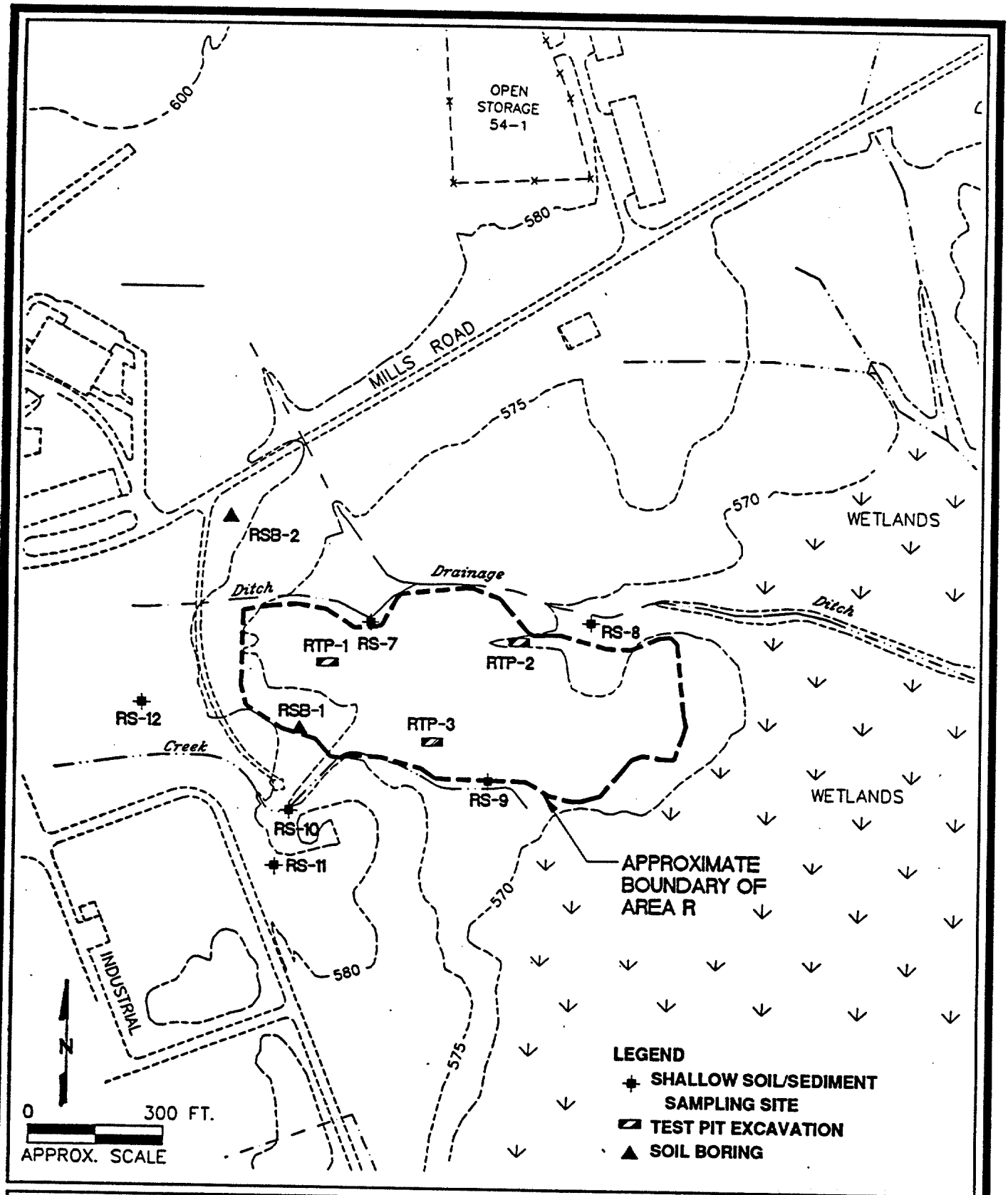


Figure 1.3-5
AREA R - CLOSED LANDFILL - EXCAVATION AND SOIL SAMPLING LOCATIONS

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

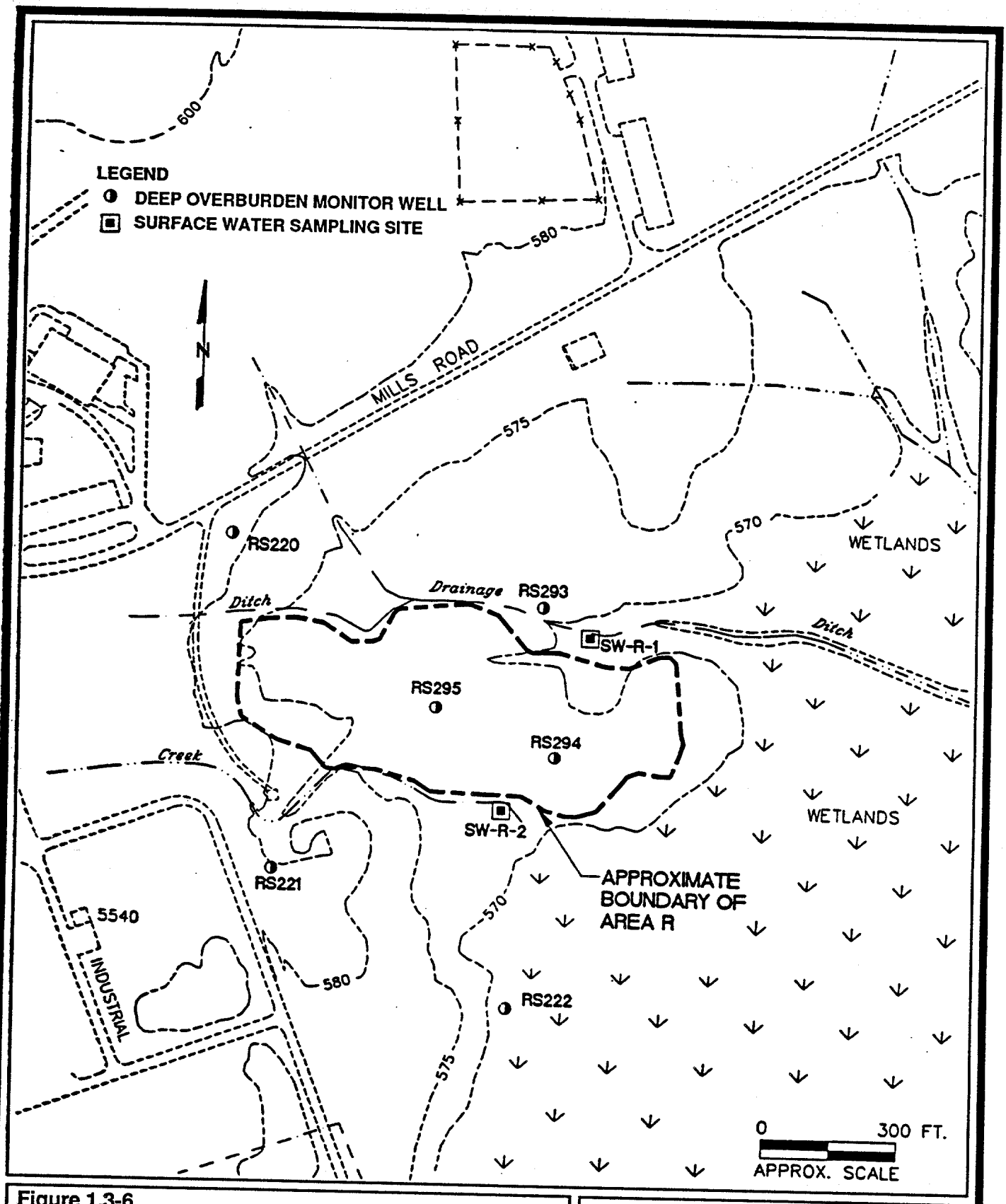


Figure 1.3-6
AREA R - CLOSED LANDFILL - GROUNDWATER AND SURFACE WATER SAMPLING LOCATIONS

SOURCES: G&M, 1991.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

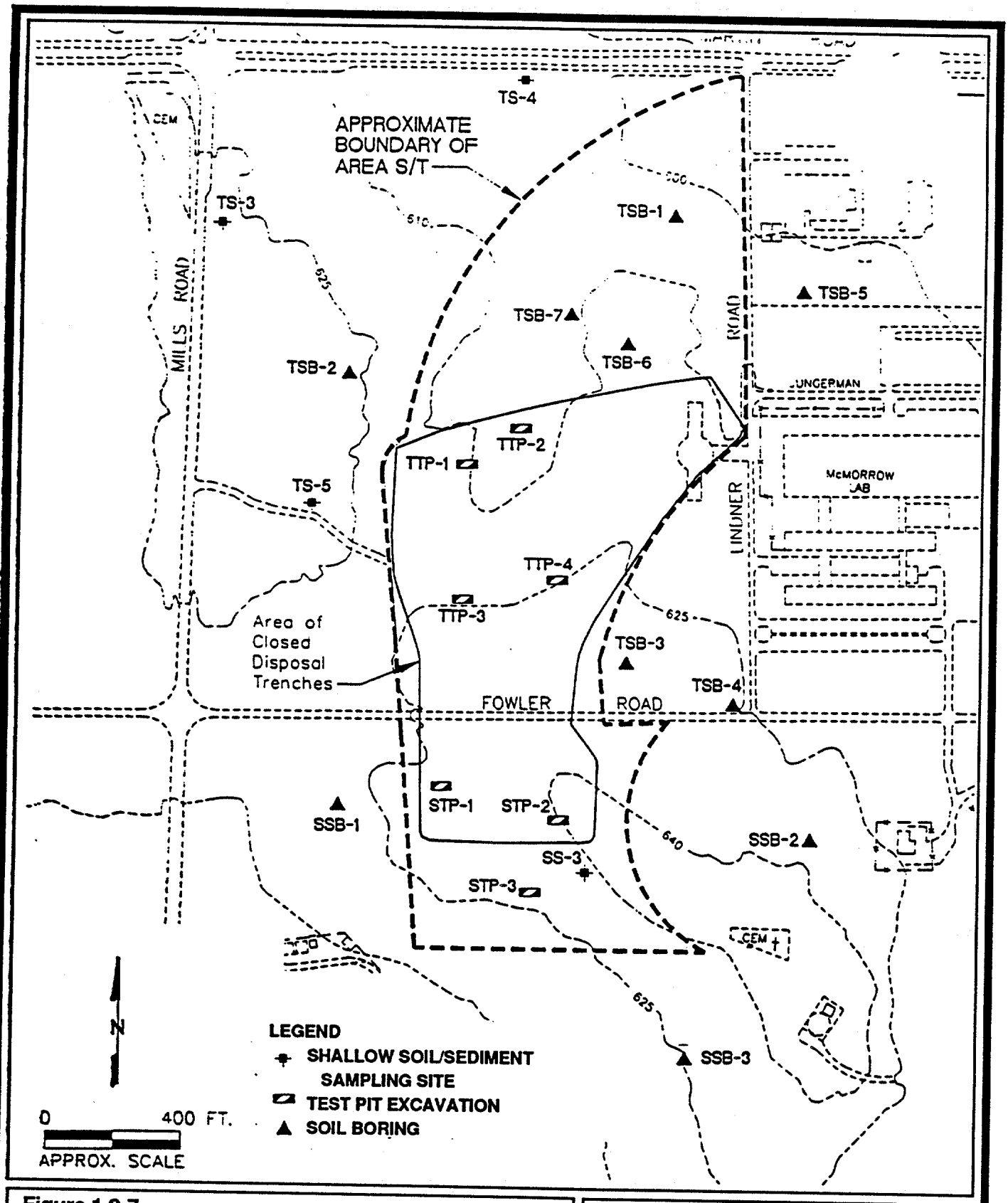


Figure 1.3-7
AREA S/T - CLOSED DISPOSAL TRENCHES - EXCAVATION AND SOIL SAMPLING LOCATIONS

SOURCES: G&M, 1992.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

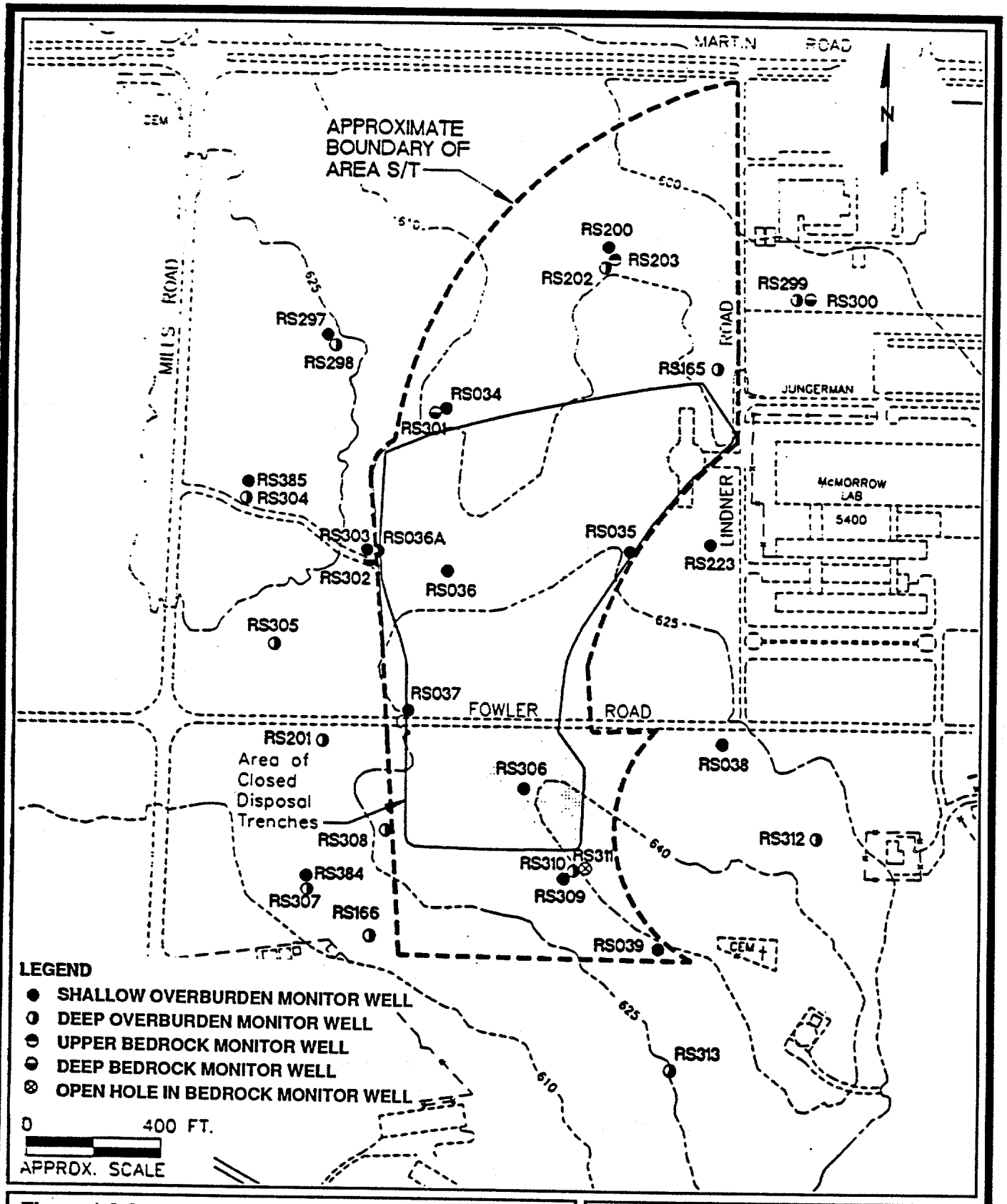


Figure 1.3-8
AREA S/T - CLOSED DISPOSAL TRENCHES - GROUNDWATER AND SURFACE WATER SAMPLING LOCATIONS

SOURCES: G&M, 1992.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS HUNTSVILLE DIVISION

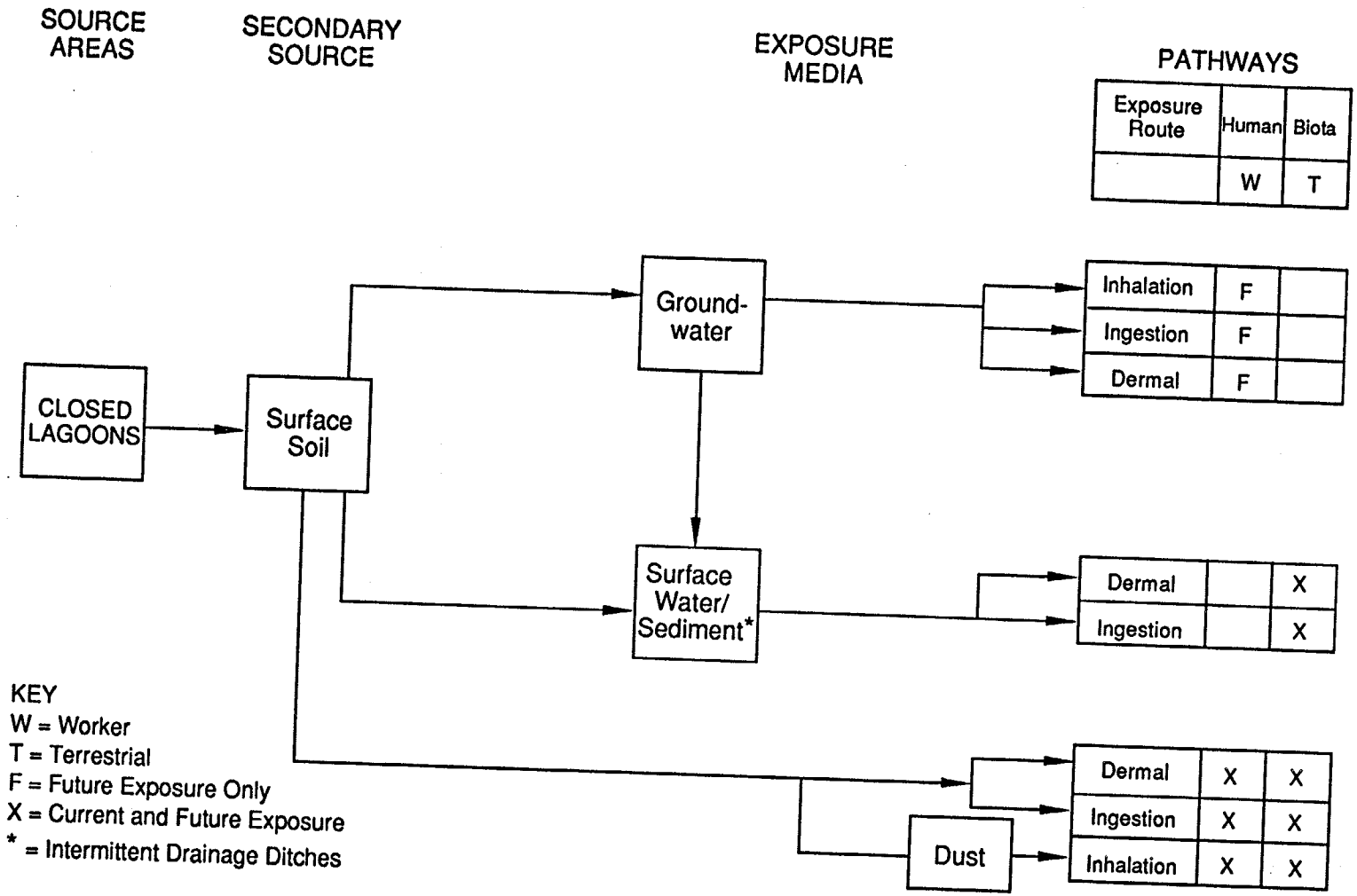


Figure 1.4-1
CONCEPTUAL SITE MODEL FOR AREA F

SOURCE: ESE.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

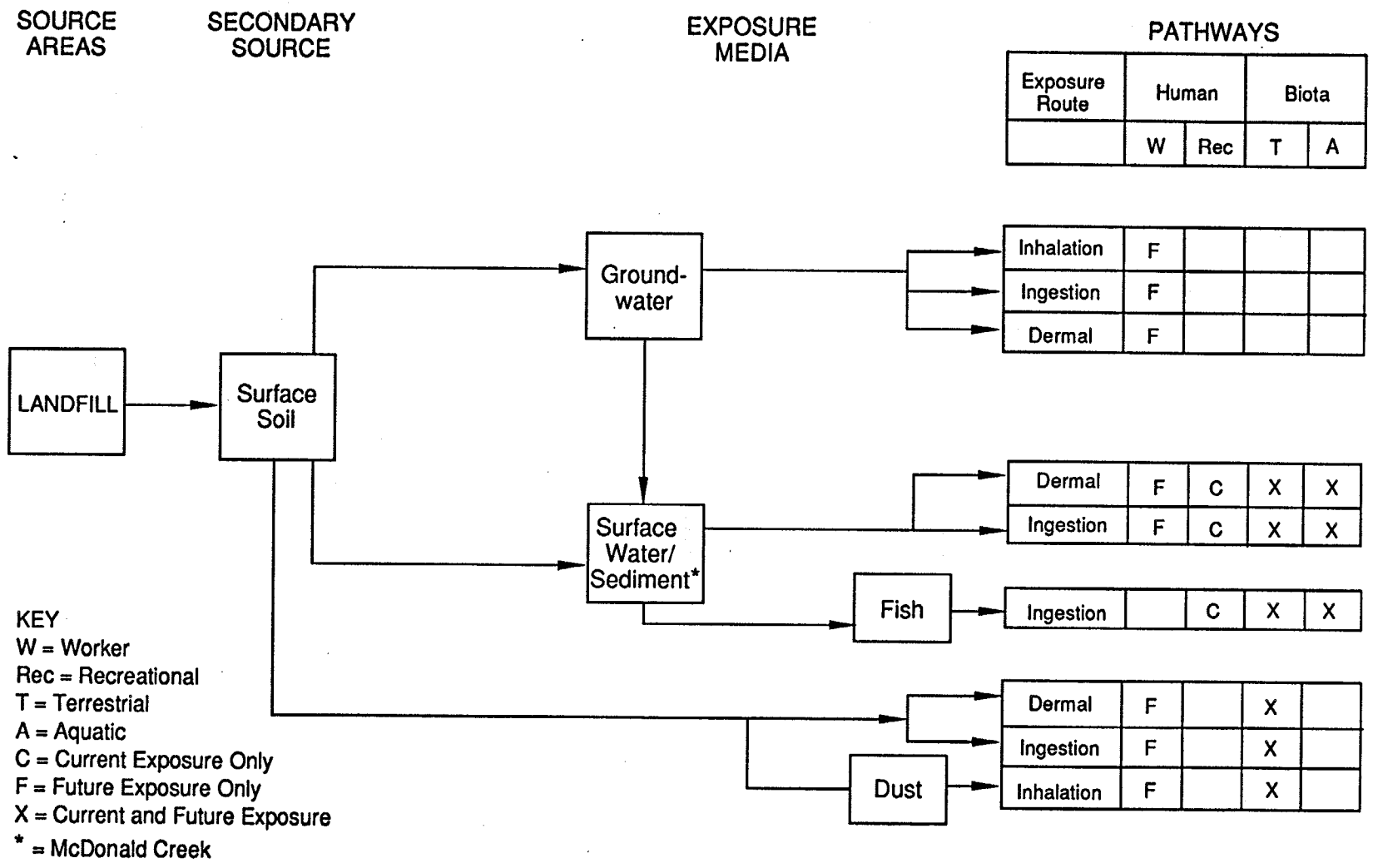


Figure 1.4-2
CONCEPTUAL SITE MODEL FOR AREA G

SOURCE: ESE.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

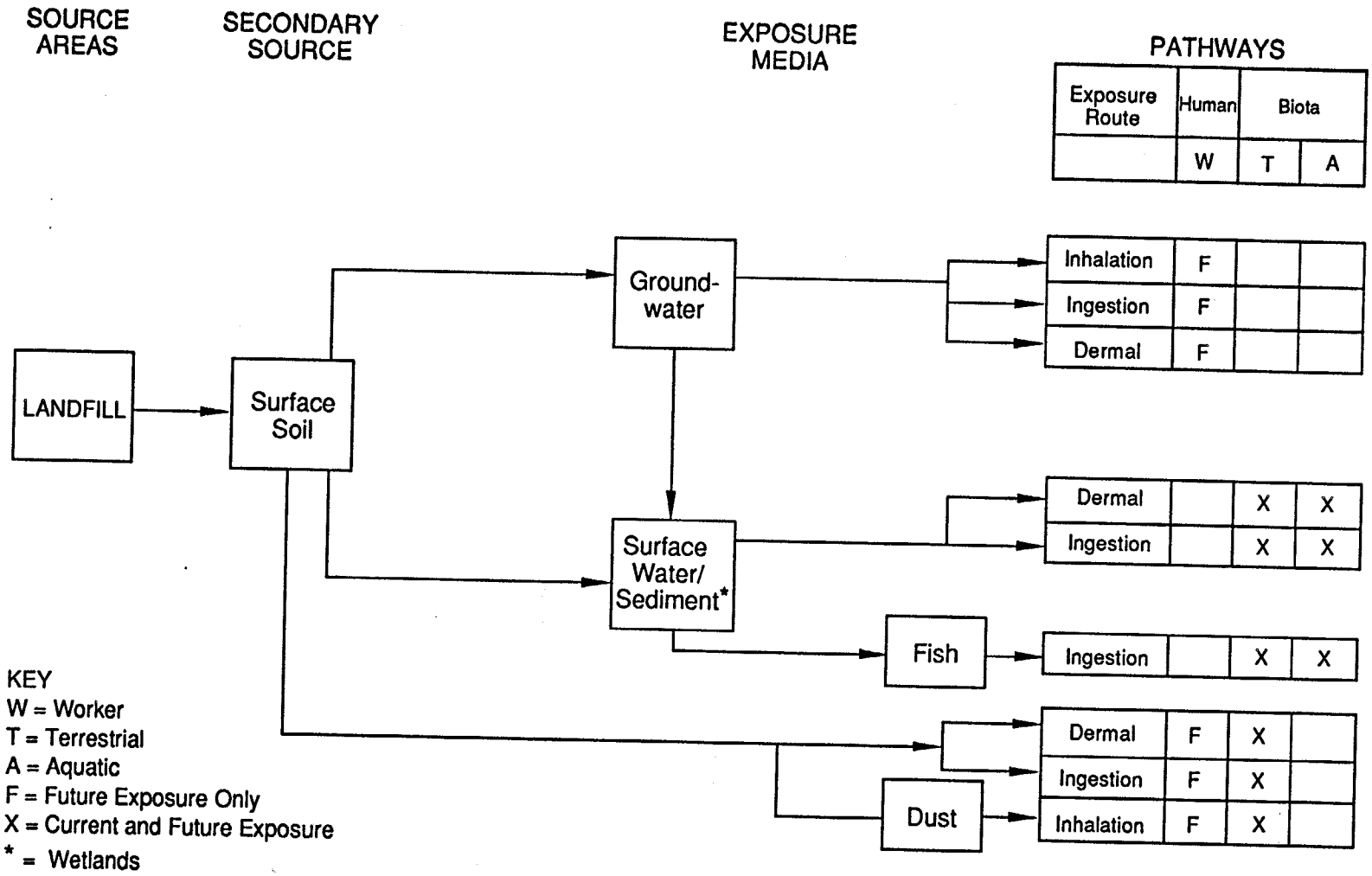


Figure 1.4-3
CONCEPTUAL SITE MODEL FOR AREA R

SOURCE: ESE.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

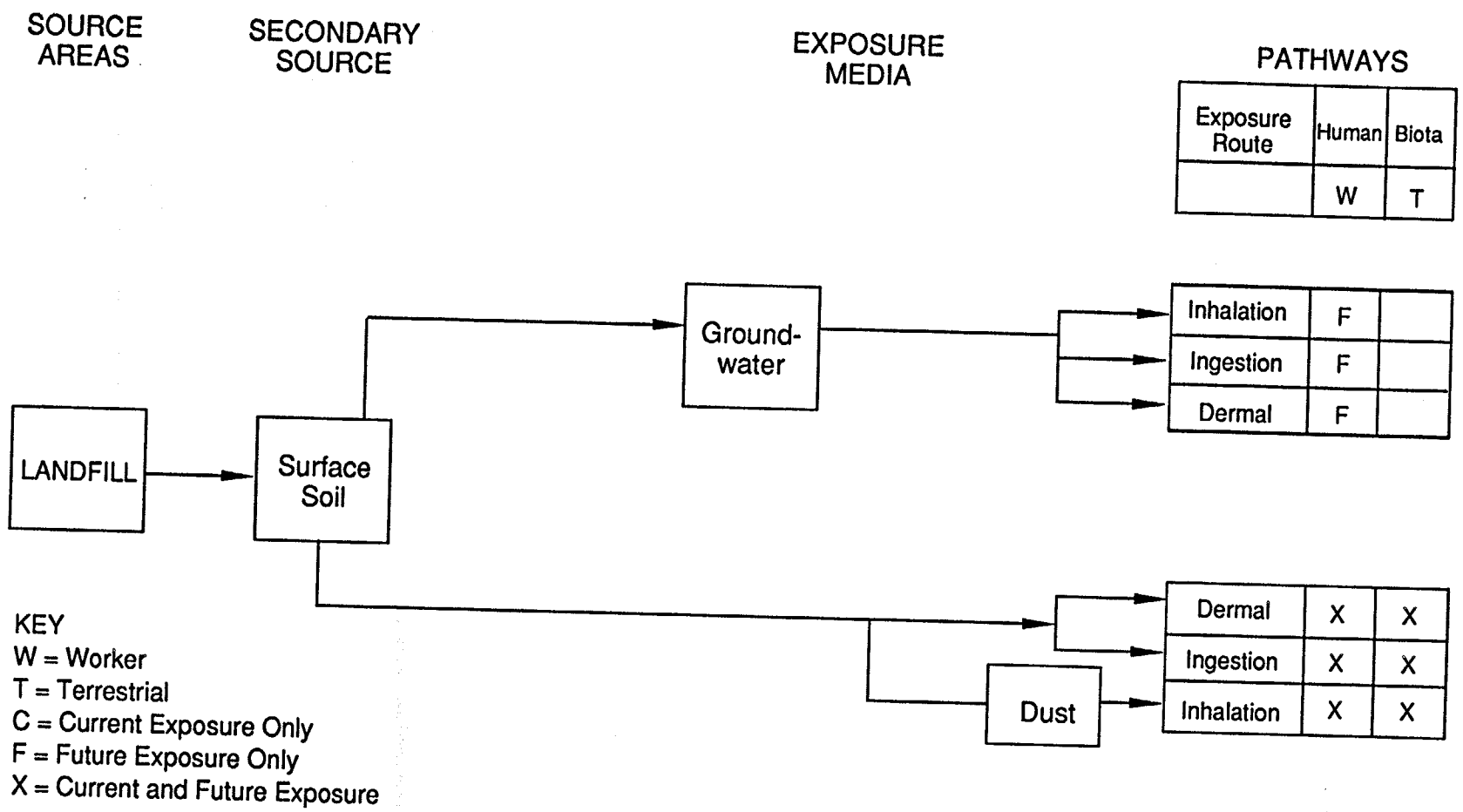


Figure 1.4-4
CONCEPTUAL SITE MODEL FOR AREA S/T

SOURCE: ESE.

REDSTONE ARSENAL

U.S. ARMY CORPS OF ENGINEERS
HUNTSVILLE DIVISION

APPENDIX A

ANALYTICAL SUMMARY TABLES
AREAS F, G, R, S/T
PHASE I AND PHASE II
(G&M, 1991; 1992)

AREA F

Summary of Analysis of Waste Samples Collected from Test Pits During the RFI at Area F, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	FTP-1 A	FTP-1 B	FTP-2 A	FTP-2 B	FTP-3 A	FTP-3 B
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg						
Acenaphthene		–	–	6500	240 J	–	–
Acenaphthylene		–	–	550 J	–	–	–
Anthracene		–	190 J	11000	340 J	–	–
Benzo(a)anthracene		570 J	520 J	28000	420 J	–	–
Benzo(a)pyrene		460 J	390 J	17000	290 J	–	–
Benzo(b)fluoranthene		810 J	730 J	30000	460 J	–	–
Benzo(ghi)perylene		–	200 J	9100	240 J	–	–
Benzo(k)fluoranthene		–	–	–	550 J	–	–
bis(2-Ethylhexyl)phthalate		3200 J	510 J	27000	–	–	89 J
Butylbenzyl phthalate		3800	240 J	–	–	–	–
Chrysene		620 J	520 J	17000	360 J	–	–
Dibenzofuran		–	–	4800	–	–	–
Dibenzo(a,h)anthracene		–	–	4600	–	–	–
Diethyl phthalate		–	–	–	–	–	120 J
Di-n-butyl phthalate		240 J	–	–	–	–	–
Fluoranthene		1400 J	1300 J	62000	1400 J	–	–
Fluorene		–	–	9900	260 J	–	–
Indeno(1,2,3-cd)pyrene		–	230 J	9700	–	–	–
2-Methylnaphthalene		–	–	1900 J	–	–	–
Naphthalene		–	–	9900	320 J	–	–
Phenanthrene		960 J	660 J	52000	1600 J	–	–
Pyrene		1000 J	820 J	24000	880 J	–	–
VOLATILE ORGANICS	ug/Kg						
Acetone		47	27	110	460	–	200
2-Butanone		–	–	32	–	–	–
Carbon tetrachloride		9	–	–	–	–	–
1,2-Dichloroethane		7	–	–	–	–	–
Ethylbenzene		–	–	9	–	–	–
Methylene Chloride		10	21	8	19	12	8
1,1,2-Trichloroethane		30	–	–	–	–	–
Xylenes (total)		–	–	160	–	–	–
METALS (TOTAL)	mg/Kg						
Arsenic		2.20 J	40000	15.70	406	15000	3.20
Barium		209	40.90 J	65.90	21.10 J	21.0 J	15.30 J
Cadmium		–	122	–	–	39.6	–
Chromium		17.10	14.80	23.70	10.80	11.0	65.90
Lead		51.70	48.90	26.90	4.30	9.10	9.60
Mercury		2.40	3.50	–	2.80	1.30	1.80

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"–" - Constituent was analyzed but not detected.

Summary of Analyses of Shallow Surface Soil Sample Collected During the RFI at Area F, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	FS-7
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg	--
VOLATILE ORGANICS	ug/Kg	
Acetone		11 J
METALS (TOTAL)	mg/Kg	
Arsenic		9.2
Barium		128
Chromium		36.4
Lead		32.1
Mercury		0.28

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Shallow Soil Samples Collected During the Phase II RFI at Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations FS-14	FS-16	FS-17	FS-18	FS-19	FS-20	FS-21	FS-22	FS-23
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/kg):									
Anthracene	- -	- -	- -	24 J	- -	- -	- -	- -	- -
Benzo (a) anthracene	39 J	- -	- -	140 J	31 J	- -	- -	17 J	- -
Benzo (b) fluoranthene	56 J	- -	- -	280 J	- -	- -	- -	- -	- -
Benzo (ghi) perlyene	- -	- -	- -	120 J	- -	- -	- -	- -	- -
Benzo (a) pyrene	- -	- -	- -	110 J	- -	- -	- -	- -	- -
Chrysene	46 J	- -	- -	140 J	- -	- -	- -	- -	- -
Fluoranthene	110 J	- -	- -	280 J	50 J	- -	- -	28 J	- -
Indeno (1,2,3-cd) pyrene	- -	- -	- -	110 J	- -	- -	- -	- -	- -
Phenanthrene	70 J	- -	- -	170 J	18 J	- -	- -	19 J	- -
Pyrene	100 J	- -	- -	290 J	57 J	- -	- -	28 J	- -
METALS (total, mg/kg):									
Arsenic	10.5	4.7	4.5	45.7	20.9	5.8	6.2	5.6	5.0
Mercury	1.0	0.25	1.3	9.1	1.7	0.37	0.46	0.26	0.53

Constituents not detected in any sample are not shown.

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

J Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

- - Constituent was analyzed but not detected.

Summary of Analyses of Waste Samples Collected From Soil Borings During the Phase II RFI at Area F, Redstone Arsenal, Huntsville, Alabama*

CONSTITUENT	Sample Locations			
	FSB-7	FSB-8		FSB-9
	A (6'-8')	A (4'-6')	C (8'-10')	C (4'-6')
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/kg):	- -	- -	NA	NA
METALS (total, mg/kg):				
Arsenic	146	6810	142	20100
Cadmium	- -	35.7	- -	107
Mercury	3.6	143	63.8	778

Constituents not detected in any sample are not shown.

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

J Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.
- - Constituent was analyzed but not detected.
NA Not analyzed

* Considered "waste" because solid debris and/or waste was visually apparent during sampling.

Summary of Analyses of Soil Samples Collected From Soil Borings During the Phase II RFI at Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations											
	FSB-3		FSB-4		FSB-5			FSB-6	FSB-7	FSB-8	FSB-9	
	A (2'-4')	B (14'-16')	A (2'-4')	B (6'-8')	A (2'-4')	DUP CC (2'-4')	B (10'-12')	B (14'-16')	B (14'-16')	B (14'-16')	A (2'-4')	B (14'-16')
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/kg):												
Acenaphthene	--	--	--	--	--	410 J	--	--	--	--	1800 J	--
Anthracene	--	--	--	--	440 J	350 J	--	--	--	--	2800 J	--
Benzo (a) anthracene	--	--	190 J	--	1400 J	1400	--	110 J	--	--	8800	--
Benzo (b) fluoranthene	--	--	170 J	--	1600 J	1500	--	75 J	--	--	8600	--
Benzo (ghi) perlyene	--	--	--	--	890 J	630 J	--	--	--	--	3300 J	--
Benzo (a) pyrene	--	--	94 J	--	1000 J	860	--	66 J	--	--	5300	--
Chrysene	--	--	130 J	--	1300 J	1100	--	97 J	--	--	6800	--
Dibenzo (a,h) anthracene	--	--	--	--	--	210 J	--	--	--	--	1300 J	--
Fluoranthene	--	--	330 J	--	2700 J	2400	--	220 J	--	--	16000	--
Fluorene	--	--	--	--	--	350 J	--	--	--	--	2100 J	--
Indeno (1,2,3-cd) pyrene	--	--	--	--	910 J	710 J	--	--	--	--	3800 J	--
Naphthalene	--	--	--	--	--	100 J	--	--	--	--	450 J	--
Phenanthrene	--	--	210 J	--	1900 J	1900	--	170 J	--	--	10000	--
Pyrene	--	--	310 J	--	2700 J	2200	--	190 J	--	--	14000	--
METALS (total, mg/kg):												
Arsenic	171	120	154	182	128	96.7	134	57.1	127	147	85.4	90.5
Mercury	0.44	0.25	9.5	21.0	0.59	1.8	4.3	0.63	0.24	5.6	5.9	1.60

Constituents not detected in any sample are not shown.

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

J Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.
-- Constituent was analyzed but not detected.

Summary of Analyses of Soil Boring Samples Collected During the RFI at Area F, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	FSB-1 S		FSB-1 D		FSB-2 S		FSB-2 D	
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg								
Bis-(2-ethylhexyl)phthalate		--		220	J	140	J	200	J
VOLATILE ORGANICS	ug/Kg								
Acetone		150		180		56		40	
Methylene Chloride		22		24		12		11	
METALS (TOTALS)	mg/Kg								
Arsenic		8540		4.5		8.8		6.1	
Barium		19.7	J	16.7	J	29	J	13.6	J
Cadmium		133		--		--		--	
Chromium		11.9		65.9		72.9		39.8	
Lead		17.1		5.7		10.5		9.6	
Mercury		133		3.7		0.57		0.18	
Selenium		--		--		0.48		--	

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Sediment Samples Collected During the RFI at Area F, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	FS-5	FS-6	FS-8	FS-9
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg				
Benzo(a)anthracene		120 J	--	--	--
Benzo(a)pyrene		60 J	--	--	--
Benzo(b)fluoranthene		150 J	150 J	--	--
Chrysene		140 J	--	--	--
Fluoranthene		280 J	--	--	--
Phenanthrene		220 J	--	--	--
Pyrene		280 J	--	--	--
VOLATILE ORGANICS	ug/Kg				
Acetone		47	54	45	35
Carbon Tetrachloride		--	--	3 J	--
Methylene Chloride		16	24	18	8
METALS (TOTAL)	mg/Kg				
Arsenic		7.9	294	7.9	10.3
Barium		179	85.2	262	44.0 J
Cadmium		--	1.2 J	--	1.7
Chromium		51.9	22.9	61.7	125
Lead		68.8	35.5	34.7	127
Mercury		--	32.9	0.13	0.14

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Sediment Samples Collected During the Phase II RFI at Area P, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations FS-10	FS-10 DUP DD	FS-11	FS-12		FS-13		FS-15
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/kg):								
Benzo (a) anthracene	- -	- -	- -	63	J	- -		- -
Benzo (b) fluoranthene	- -	- -	- -	140	J	- -		- -
Benzo (a) pyrene	- -	- -	- -	54	J	- -		- -
Chrysene	- -	- -	- -	65	J	- -		- -
Fluoranthene	- -	- -	- -	85	J	32	J	- -
Pyrene	- -	- -	- -	170	J	42	J	- -
METALS (total, mg/kg):								
Arsenic	3.7	5.4	3.7	13.1		5.8		2.1
Mercury	0.26	0.19	0.18	0.67		0.66		0.25

Constituents not detected in any sample are not shown.

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

J Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

- - Constituent was analyzed but not detected.

Summary of Analyses of Surface Water Samples Collected During the RFI at Area F, Redstone Arsenal, Huntsville, Alabama (Phase I)

CONSTITUENT	SWF-1	
VOLATILE ORGANICS (ug/L):	---	
METALS (total, ug/L):		
Barium	32.1	J

Constituents not detected in any sample are not shown.

ug/L microgram per Liter

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"---" - Constituent was analyzed but not detected.

Summary of Analyses of Ground-Water Samples Collected During the RFI at Area F, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS054	RS053	RS055	RS257	RS260	RS261	RS052	RS256	RS259	RS259 (DUP)	RS262	RS263
VOLATILE ORGANICS	ug/L												
Acetone		–	–	–	–	61	–	84	–	–	–	–	–
Chloroform		–	–	–	–	–	16	–	–	–	–	–	–
Carbon tetrachloride		–	4 J	4 J	–	4 J	17	–	–	–	–	14	7
Trichloroethene		–	–	–	–	–	3 J	–	–	–	–	5	4 J
METALS (Total)	ug/L												
Arsenic		–	7.6 J	25.2	–	–	20.2	26.5	–	–	4.6 J	5.8 J	67.9
Barium		–	27.1 J	23.8 J	21.1 J	35.8 J	–	35.5 J	23.9 J	23.4 J	22.9 J	–	18.6 J
Chromium		–	–	–	–	–	–	16.1	8.4 J	7.1 J	8.4 J	–	–
Lead		–	–	–	–	–	–	1.6 J	1.8 J	–	–	–	–
Mercury		0.30	–	–	–	1.4	–	–	–	0.10 J	–	–	–
METALS (Dissolved)	ug/L												
Arsenic		1.22 J	8.34	28.4	–	1.48 J	22.0	56.6	–	0.94 J	0.88 J	8.76	99.8
Barium		10.46 J	29.20 J	21.8 J	30.0 J	61.8	23.6 J	23.0 J	21.4 J	17.74 J	16.0 J	19.98 J	19.48 J
Cadmium		–	–	–	–	4.28	–	–	–	–	–	–	–
Chromium		2.18	3.28	2.30	6.10	4.08	2.76	2.9	1.68 J	4.12	2.12	2.6	1.86 J
Lead		2.30	–	–	–	3.3	0.32 J	–	–	–	–	0.46 J	0.48 J
Mercury		–	–	–	–	0.104	–	–	–	–	–	–	–
Selenium		–	0.76 J	0.76 J	–	–	0.68 J	–	1.82	0.98 J	1.22	–	–

Constituents not detected in any sample are not shown.

ug/L - micrograms per liter.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"–" - Constituent was analyzed but not detected.

Summary of Analyses of Ground-Water Samples Collected During the Phase II RFI at Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	RS052	RS055	RS256	RS257	RS258	RS258 DUP HH	RS259	RS261	RS263
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l)	- -	- -	- -	- -	- -	- -	- -	- -	- -
VOLATILE ORGANICS (ug/L)									
Acetone	NA	NA	NA	NA			NA	NA	NA
Chloroform					20	29			
METALS (Total, ug/L)					7.0	6.0			
Arsenic	NA	NA	NA	NA			NA	NA	NA
Barium					46.4	110			
Cadmium					240	320			
Chromium					4.8 J	7.6			
Lead					- -	5.0 J			
Mercury					13.8	52.4			
METALS (Dissolved, ug/L)					2800 J	3100 J			
Arsenic	NA	NA	NA	NA			NA	NA	NA
Barium					50	68.4			
Cadmium					115	110			
Mercury					- -	3.0			
					26 J	24			

Constituents not detected in any sample are not shown.

ug/L micrograms per Liter
mg/l milligrams per liter

J Indicates a qualitative value either below laboratory reporting limit but above instrument detection limit or a value qualified due to a control deficiency identified during the data review presented in the QCSR.

- - Constituent was analyzed but not detected.

NA Not analyzed

AREA G

Summary of Analyses of Waste Samples Collected from Test Pits During the RFI at Area G, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	GTP-1		GTP-2			GTP-3
		A	B	A	B	C	A
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg						
Benzo(a)anthracene		--	--	1800 J	--	--	--
Benzo(b)fluoranthene		--	--	1700 J	--	--	--
bis(2-Ethylhexyl)phthalate		18000 J	--	2500 J	120 J	--	460 J
2-Chloronaphthalene		--	--	--	--	--	830 J
Chrysene		--	--	1900 J	--	--	--
Di-n-butyl phthalate		8600 J	--	--	--	--	450 J
Fluoranthene		--	--	4900 J	--	--	170 J
Naphthalene		--	--	--	--	--	300 J
Phenanthrene		18000 J	--	2500 J	--	--	270 J
Pyrene		20000 J	--	2500 J	--	--	--
VOLATILE ORGANICS	ug/Kg						
Acetone		430	14 J	270	31	220	230
Benzene		--	--	5 J	--	12	--
2-Butanone		--	--	69	--	--	--
Carbon disulfide		12 J	--	7 J	--	--	--
Carbon tetrachloride		--	--	33	--	--	--
Chlorobenzene		54	--	11	--	4 J	64
1,2-Dichloroethane		--	--	51	--	--	--
1,2-Dichloroethene(total)		--	--	9 J	--	--	--
2-Hexanone		--	--	--	--	24	--
Methylene Chloride		77	14	33	7	7	9
Styrene		--	--	8 J	--	--	--
Toluene		--	--	8 J	--	--	--
1,1,2-Trichloroethane		69	--	190	--	--	--
Xylenes (total)		--	--	--	--	15	--
METALS (TOTAL)	mg/Kg						
Arsenic		5.30	3.60	8.30	7.00	2.20	3.90
Barium		73.30	47.70 J	143	102	69.0	76.0
Cadmium		1.10 J	--	--	--	--	--
Chromium		76.0	18.90	22.30	14.30	17.8	43.70
Lead		167	15.90	162	5.30	14.10	36.6
Mercury		1.40	0.80	1.80	--	0.33	1.00

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Soil Boring Samples Collected During the RFI at Area G, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	GSB-1		GSB-2		GSB-3	
		S	D	S	D	S	D
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg	--	--	--	--	--	--
VOLATILE ORGANICS	ug/Kg						
Acetone		32	31	26	29	140	92
Carbon tetrachloride		--	--	--	12	--	--
Methylene Chloride		11	--	--	--	3 J	19
METALS (TOTAL)	mg/Kg						
Arsenic		1.8	6.1	12.9	5.4	3.0	2.6
Barium		135	21.3 J	24.5 J	25.4 J	79.3	16.9
Cadmium		--	--	--	--	--	1.3
Chromium		50.2	34.6	99.5	57.0	71.1	32.7
Lead		7.5	11.4	26.7	15.0	10.9	5.2
Mercury		--	--	0.49	--	--	--
Selenium		--	0.51	--	--	--	--

Constituents not detected in any sample are not shown.
ug/Kg - micrograms per kilogram.
mg/Kg - milligrams per kilogram.
J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.
"--" - Constituent was analyzed but not detected.

ARGSB.XLS

Summary of Analyses of Sediment Samples Collected During the RFI at Area G, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	GSS-4	GSS-5	GSS-6	GSS-7	GSS-8
VOLATILE ORGANICS	ug/Kg					
Acetone		91	160	4 J	--	--
Methylene Chloride		24	16	15	19	17
METALS	mg/Kg					
Arsenic		--	--	2.9 J	5.7	2.5 J
Barium		75.8	56.1	51.6 J	22.1 J	68.0
Chromium		54.9	57.8	32.7	42.5	27.2
Lead		6.2	12.1	6.2	26.3	8.4
Mercury		0.42	0.29	--	0.15	--

Constituents not detected in any sample are not shown.

ug/kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Surface Water Samples Collected During the RFI at Area G, Redstone Arsenal, Huntsville, Alabama (Phase I)

CONSTITUENT	UNITS	SW-G-3	SW-G-4	SW-G-5
VOLATILE ORGANICS	ug/L			
Acetone		20	--	25
Methylene chloride		10	10	--
Trichloroethene		--	26	28
METALS	ug/L			
Barium		72.6 J	--	--
Lead		--	--	5.8
Mercury		--	1.6	--

Constituents not detected in any sample are not shown.

ug/L - micrograms per liter.

J - Indicates a qualitative value below laboratory reporting limit but above instrument dection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Surface Water Samples Collected During the Phase II RFI at Area G, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations SW-G-4(II)	SW-G-5(II)	SW-G-5(II) DUP FF	SW-G-6	SW-G-7	SW-G-8	SW-G-9	SW-G-10
VOLATILE ORGANICS (ug/L):								
Acetone	- -	- -	- -	23	14	7 J	- -	- -
Dichloroethene, 1,1-	- -	4 J	- -	- -	- -	- -	- -	- -
Trichloroethene	9	9	9	- -	- -	10	10	10
Xylenes (Total)	- -	6	- -	- -	- -	- -	- -	- -

Constituents not detected in any sample are not shown.

ug/L micrograms per Liter

J Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

- - Constituent was analyzed but not detected.

(II) Collected during the Phase II RFI

Summary of Analyses of Ground-Water Samples Collected During the RFI at Area G, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS111	RS113	RS114	RS114(DUP)	RS265	RS266	RS267	RS112	RS264	RS268
VOLATILE ORGANICS	ug/L										
Acetone		--	8 J	--	--	--	--	--	--	--	--
METALS (Total)	ug/L										
Arsenic		6.9 J	9.8 J	--	--	--	10.8	--	6.6 J	--	7.2 J
Barium		364	672	48.3 J	88.8 J	--	5910	146 J	930	193 J	571
Cadmium		8.5	18.7	--	--	--	79.1	--	21.1	--	21.7
Chromium		156	394	47	97.7	--	626	5.1 J	240	18.2	276
Lead		30.4	146	6.2	19.3	--	263	--	172	2.7 J	128
METALS (Dissolved)	ug/L										
Arsenic		--	--	--	--	--	--	2.92 J	--	0.64 J	--
Barium		66.2	57.8	7.5 J	9.0 J	5.68 J	36.4 J	120.2	34.6 J	160.6	3.8 J
Chromium		1.06 J	--	2 J	2.86	2.52	1.3 J	1.1 J	--	--	2.6
Lead		4.92	--	--	--	--	--	0.54 J	--	--	--

Constituents not detected in any sample are not shown.

ug/L - micrograms per liter.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

ARGGW.XLS

AREA R

Summary of Analyses of Waste Samples Collected from Test Pits During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RTP-1 A	RTP-2 A	RTP-3 A
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg			
Benzo(b)fluoranthene		--	130 J	--
VOLATILE ORGANICS	ug/Kg			
Acetone		--	--	9 J
Methylene Chloride		6	9	8
1,1,2-Trichloroethane		3 J	--	--
METALS (Total)	mg/Kg			
Arsenic		2.20 J	1.60 J	1.60 J
Barium		111	32.70 J	21.70 J
Chromium		11.30	12.90	3.10
Lead		7.80	6.70	5.30
Mercury		0.18	--	--

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Shallow Soil Samples Collected During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS-11		RS-12	
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg	--		--	
VOLATILE ORGANICS	ug/Kg				
Methylene Chloride		4	J	5	J
METALS (TOTAL)	mg/Kg				
Arsenic		7.5		5.9	
Barium		172		69.9	
Chromium		11.2		23.5	
Lead		87.4		142	
Mercury		--		0.53	

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Soil Boring Samples Collected During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RSB-1		RSB-2	
		S	D	S	D
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg				
Acenaphthene		200 J	--	--	--
Anthracene		450 J	--	--	--
Benzo(a)anthracene		1700	--	--	--
Benzo(a)pyrene		1200	--	--	--
Benzo(b)fluoranthene		2300	--	--	--
Benzo(ghi)perylene		760 J	--	--	--
Butylbenzylphthalate		910	--	--	--
Chrysene		1200	--	--	--
Bis-(2-ethylhexyl)phthalate		900	100 J	97 J	--
Fluoranthene		2800	--	--	--
Fluorene		200 J	--	--	--
Indeno(1,2,3,-cd)pyrene		760 J	--	--	--
Phenanthrene		2000	--	--	--
Pyrene		2700	--	--	--
VOLATILE ORGANICS	ug/Kg				
Acetone		200	80	53	100
Methylene Chloride		9	8	8	8
METALS (TOTAL)	mg/Kg				
Arsenic		6.9	10.6	7.6	8.5
Barium		64.0	21.1 J	45.5 J	5.7 J
Chromium		23.3	88.3	49.2	71.4
Lead		53.3	10.0	8.8	6.2
Mercury		2.9	0.19	0.17	--
Selenium		0.76 J	1.2 J	0.81 J	0.94 J

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Sediment Samples Collected During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS-7		RS-8	RS-9		RS-10	
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg							
Benzo(a)anthracene		490	J	--	--		--	
Benzo(a)pyrene		780	J	--	--		--	
Benzo(b)fluoranthene		2200	J	--	710	J	--	
Benzo(ghi)perylene		860	J	--	--		--	
Bis(2-Ethylhexyl)phthalate		530	J	--	--		--	
Chrysene		950	J	--	360	J	--	
Fluoranthene		490	J	--	310	J	--	
Indeno(1,2,3-cd)pyrene		870	J	--	--		--	
Pyrene		760	J	--	440	J	--	
VOLATILE ORGANICS	ug/Kg							
Acetone		41	J	--	12	J	8	J
Carbon Tetrachloride		--		9	--		--	
1,1-Dichloroethane		21	J	--	--		--	
Methylene Chloride		43		23	15		9	
METALS (TOTAL)	mg/Kg							
Arsenic		8.3		--	21.1		4.4	
Barium		109	J	96.2	82.4		152	
Cadmium		7.9		--	--		--	
Chromium		43.2		12.3	19.9		26.8	
Lead		58.6		29.7	30.4		54.6	
Mercury		1.2		--	0.62		--	
Silver		142		4.5	--		--	

Constituents not detected in any sample are not shown.
ug/Kg - micrograms per kilogram.
mg/Kg - milligrams per kilogram.
J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.
"--" - Constituent was analyzed but not detected.

Summary of Analyses of Surface-Water Samples Collected During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	SW-R-1		SW-R-1(DUP)		SW-R-2	
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/L						
Bis(2-ethylhexyl)phthalate		6	J	16		10	J
VOLATILE ORGANICS	ug/L						
Chloroform		—		—		8	
METALS (TOTAL)	ug/L						
Arsenic		5.1	J	5.9	J	—	
Barium		76.9	J	75.0	J	46.3	J
Silver		—		—		6.2	J

Constituents not detected in any sample are not shown.

ug/L - micrograms per Liter

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"—" - Constituent was analyzed but not detected.

Summary of Analyses of Ground-Water Samples Collected During the RFI at Area R, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS220	RS221	RS221(DUP)	RS295	RS294	RS222	RS293
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/L	NA	NA	NA			NA	
bis(2-Chloroethyl)ether					--	6 J		--
bis(2-Ethylhexyl)phthalate					--	--		6 J
VOLATILE ORGANICS	ug/L							
Acetone		--	3 J	--	15	--	40	3 J
Chlorobenzene		--	--	--	--	28	--	6
Chloroform		--	38	43	--	--	--	3 J
Methylene Chloride		--	--	--	--	--	--	3 J
METALS (Total)	ug/L							
Arsenic		--	8.8 J	8.6 J	6.4 J	--	--	--
Barium		24.4 J	115 J	117 J	1340	263	43.3 J	398
Cadmium		--	--	--	22.1	--	--	10.2
Chromium		11.5	69.3	85.7	539	155	55.1	196
Lead		1.3 J	19.1	18.4	88	4.7	3.7	46.5
METALS (Dissolved)	ug/L							
Arsenic		--	0.8 J	--	--	--	--	0.92 J
Barium		10.3 J	42.8	36 J	282	115.2	12.6 J	115.8
Cadmium		--	--	--	--	--	--	--
Chromium		1.38 J	--	1.34 J	--	1.1 J	1.22 J	--
Selenium		--	3.6 J	--	--	--	--	--

Constituents not detected in any sample are not shown.

ug/L - micrograms per liter.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

NA - Not Analyzed.

AREAS S AND T

Summary of Analyses of Waste Samples Collected from Test Pits During the RFI at Area S/T ,
Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	STP-1		STP-2		STP-3	TTP-1		TTP-2		TTP-3		TTP-4		
		A	B	A	B	A	A	B	A	B	A	B	A	A-Dup	B
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg														
Acenaphthene		–	–	–	1400 J	–	–	–	320 J	–	–	–	–	–	–
Anthracene		–	–	–	2100 J	–	–	–	280 J	–	–	–	–	–	–
Benzo(a)anthracene		–	–	–	4300	–	100 J	380 J	870 J	–	–	–	–	–	–
Benzo(a)pyrene		–	–	340 J	4000	–	150 J	–	930 J	–	–	–	–	–	–
Benzo(b)fluoranthene		–	–	500 J	6700	–	220 J	730 J	1700 J	–	–	–	–	–	–
Benzo(ghi)perylene		–	–	–	2000 J	–	120 J	–	440 J	–	–	–	–	–	–
bis(2-Ethylhexyl)phthalate		–	680 J	890 J	1200 J	–	450 J	1500 J	470 J	–	590	–	–	–	–
Chrysene		–	–	350 J	4000	–	110 J	420 J	860 J	–	–	–	–	–	–
Dibenzofuran		–	–	–	780 J	–	–	–	150 J	–	–	–	–	–	–
Dibenzo(a,h)anthracene		–	–	–	970 J	–	–	–	–	–	–	–	–	–	–
Diethyl phthalate		–	–	1900 J	–	–	–	390 J	650 J	–	–	–	–	–	–
Dimethyl phthalate		–	590 J	–	–	–	–	–	–	–	–	–	–	–	–
Di-n-butylphthlate		–	65 J	460 J	–	–	44 J	410 J	170 J	–	–	–	–	–	–
Fluoranthene		–	130 J	290 J	12000	–	110 J	960 J	2200 J	–	–	220 J	–	–	–
Fluorene		–	–	–	1700 J	–	–	–	330 J	–	–	160 J	–	–	–
Indeno(1,2,3-cd)pyrene		–	–	–	2500	–	91 J	–	450 J	–	–	–	–	–	–
2-Methylnaphthalene		–	–	–	350 J	–	–	–	–	–	–	310 J	–	–	–
Naphthalene		–	94 J	110 J	880 J	–	–	–	700 J	–	–	–	–	–	4 J
Phenanthrene		–	110 J	–	10000	–	–	480 J	1600 J	–	–	360 J	–	–	–
Pyrene		–	100 J	–	7400	–	100 J	610 J	940 J	–	–	150 J	–	–	–
VOLATILE ORGANICS	ug/Kg														
Acetone		18	220	760	130	68	32	–	82	42 J	110	1200	5 J	–	42
Carbon Disulfide		–	–	–	–	–	–	–	–	–	–	35	–	–	6 J
Chlorobenzene		–	32	160	46	–	43	76000	1200	160	5 J	800	7	3 J	110
Ethylbenzene		–	11	–	16 J	–	–	–	–	–	–	–	–	–	6 J
2-Hexanone		–	–	–	–	–	–	–	–	–	–	56 J	–	–	–
Methylene chloride		17	8	–	–	–	–	–	–	–	3 J	–	9 (13)	11	11
1,1,2-Trichloroethane		9	–	–	–	–	–	–	–	–	–	–	–	–	–
Xylenes (total)		–	11	–	–	–	–	–	–	–	–	–	–	–	8

Summary of Analyses of Waste Samples Collected from Test Pits During the RFI at Area S/T ,
Redstone Arsenal, Alabama (Phase I), page 2 of 2

CONSTITUENT	UNITS	STP-1		STP-2		STP-3	TTP-1		TTP-2		TTP-3		TTP-4		
		A	B	A	B	A	A	B	A	B	A	B	A	A-Dup	B
METALS (TOTAL)	mg/Kg														
Arsenic		1.5	11.8	5.2	1.7 J	5.9	1.5 J	4.4	5.6	--	2.6	5.3	3.6	2.2 J	5.2
Barium		77.7	59.7	38.6 J	43.3 J	10.9 J	38.4 J	104	96.7	36.1 J	10.7 J	38.6 J	229	169	128
Cadmium		--	--	--	1.0 J	--	--	--	--	--	--	--	--	1.6	--
Chromium		66.2	18.6	34.3	34.3	36.6	18.2	25.1	18.1	13.0	21.6	49.7	27.8	25.1	90.8
Lead		8	24.1	12.8	139	11.7	548	648	27.0	7.2	2.7	79.9	19.0	24.7	96.9
Mercury		1.2	0.98	0.8	0.29	0.15	0.22	4.0	--	--	0.14	0.81	0.53	0.38	2.4
Silver		--	--	--	--	--	--	19.2	5.6	--	--	--	--	5.5	2.4 J
ORGANOCHLORINE PESTICIDES	ug/Kg														
4,4'-DDE		56 J	980	4600	670	--	680	393000	2300	4700	7 J	330	480	420	440
4,4'-DDD		170 J	2600	12700	42 J	--	--	25500	2400	1500	37 J	210	800	1000	400
4,4'-DDT		201	11000	3100	1500	--	740	784000	3800	9400	--	270	1100	--	1400
Endrin		--	140 J	--	--	--	--	--	--	--	--	--	--	--	--
Methoxychlor		--	--	--	--	--	--	--	--	--	--	--	--	1400	--

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

Summary of Analyses of Shallow Surface Soil Samples Collected During the RFI at Area S/T, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	SS-3	TS-3	TS-4	TS-5
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg				
bis(2-Ethylhexyl)phthalate		710 J	NA	NA	NA
VOLATILE ORGANICS	ug/Kg				
Acetone		57	--	--	6 J
Methylene Chloride		10	30	8	--
METALS (TOTAL)	mg/Kg				
Arsenic		4.7	7.9	3.7	6.6
Barium		31.9 J	14.0 J	138	67.8
Chromium		25.1	40.9	13.0	15.6
Lead		18.3	16.8	19.6	16.3
ORGANOCHLORINE PESTICIDES	ug/Kg				
4,4'-DDE		21	NA	NA	NA
4,4'-DDT		9.1 J	NA	NA	NA

Constituents not detected in any sample are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

NA - Not Analyzed.

Summary of Analyses of Soil Boring Samples Collected During the RFI at Area S/T, Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	SSB-1		SSB-2		SSB-3		TSB-1		TSB-2		TSB-3			TSB-4		TSB-5	
		S	D	S	D	S	D	S	D	S	D	S	S-Dup	D	S	D	S	D
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS	ug/Kg																	
Bis(2-ethylhexyl)phthalate		110 J	--	--	--	96 J	--	--	140 J	--	--	--	(--)	--	--	--	--	--
VOLATILE ORGANICS	ug/Kg																	
Acetone		34	26	87	89	150	180	97	150	24	26	44	51	190	570	32	120	120
Methylene Chloride		8	7	10	12	10	10	16	11	5 J	10	11	13	15	40	11	9	7
METALS (TOTAL)	mg/Kg																	
Arsenic		8.1	8.0	4.5	8.9	7.9	5.9	4.9	2.9	7.4	8.3	4.3	3.7	8.7	8.6	3.8	6.2	--
Barium		30.5 J	10.7 J	13.5 J	7.9 J	19.5 J	12.2 J	94.8	12.0 J	30.4 J	20.9 J	91.1	81.5	9.4 J	8.0 J	11.0 J	11.0 J	9.7 J
Chromium		53.6	88.1	54.9	134	30.4	56.2	59.9	37.7	51.5	81.6	23.8	20.3	48.9	26.1	15.3	49.5	8.8
Lead		15.2	6.8	4.8	9.3	13.1	8.8	10.6	4.1	10.7	7.2	12.8	10.1	7.8	6.6	5.0	5.5	1.5
Mercury		0.17	--	--	--	--	--	--	--	1.2	--	--	--	--	--	--	--	--
ORGANOCHLORINE PESTICIDES	ug/Kg																	
alpha-BHC		--	--	--	--	--	--	--	--	--	--	--	--	--	--	0.59	--	--
4,4'-DDE		3.0	1.8	0.80 J	--	--	--	1.1 J	--	--	--	3.4 J	8.4	--	12 J	1.4	1.02 J	--
4,4'-DDT		--	--	--	--	--	--	1.8 J	--	--	--	4.5 J	--	--	95	13	0.66 J	--

Constituents not detected in any samples are not shown.

ug/Kg - micrograms per kilogram.

mg/Kg - milligrams per kilogram.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

( ) - Re-extraction data.

Summary of Analyses of Soil Boring Samples Collected During the Phase II RFI at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locatons							
	TSB-6		TSB-6A		TSB-7		TSB-7A	
	A (2' - 4')	B (14' - 16')	A (2' - 4')	B (14' - 16')	A (2' - 4')	B (14' - 16')	A (2' - 4')	B (14' - 16')
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/kg):	NA	NA	- -	- -	NA	NA	- -	- -
VOLATILE ORGANICS (ug/Kg):								
Acetone	- -	55	NA	NA	1300 J	- -	NA	NA
Methylene Chloride	3 J	- -			18 J	5 J		
PESTICIDES/PCBs (ug/kg):	- -	- -	NA	NA	- -	- -	NA	NA

Constituents not detected in any sample are not shown.

ug/kg micrograms per kilogram

J Indicates a qualitative value either below laboratory reporting limit but above instrument detection limit or a value qualified due to a control deficiency identified during the data review presented in the QCSR.

- - Constituent was analyzed but not detected.

NA Not analyzed

Summary of Analyses of Ground-Water Samples Collected During the RFI at Area S/T,
Redstone Arsenal, Alabama (Phase I)

CONSTITUENT	UNITS	RS036	RS036A	RS037	RS034	RS200	RS301	RS202	RS203	RS300	RS305	RS308	RS297	RS298	RS303	RS035
VOLATILE ORGANICS	ug/L															
Acetone		--	--	--	--	--	15	--	--	--	17	19	18	41	10 J	10 J
Benzene		31	--	19	--	--	--	--	--	--	--	7	--	--	7	--
Chlorobenzene		650	87	3000	37	--	--	--	--	--	--	190	--	--	1400	200
Chloroform		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethane		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethene(total)		--	--	--	--	--	--	--	--	--	--	36	--	--	--	--
Ethylbenzene		3 J	--	3 J	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene		--	--	--	--	--	--	--	--	--	--	13	--	--	--	--
Trichloroethane		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Vinyl chloride		--	--	--	--	--	--	--	--	--	--	46	--	--	--	--
Xylenes (total)		--	--	5	--	--	--	--	--	--	--	--	--	--	--	--
METALS (Total)	ug/L															
Arsenic		20.9	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Barium		792	259	51.3 J	59.6 J	--	50.8 J	--	--	45.5 J	42.4 J	87.7 J	--	46.0	65.8 J	60.5 J
Cadmium		4.3 J	7.8	--	--	--	--	--	--	--	--	--	--	--	--	--
Chromium		74.8	11.3	9.3 J	15.2	--	7.6 J	--	--	--	15.4	8.5 J	--	18.5	--	--
Lead		204	45.5	2.7 J	3.2	--	--	--	--	--	--	--	--	--	--	--
Mercury		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Selenium		--	--	--	--	--	2.4 J	--	--	--	2.6 J	--	2.4 J	--	2.0 J	--
Silver		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
METALS (Dissolved)	ug/L															
Arsenic		--	--	--	--	--	--	--	--	--	--	2.5	--	1.86 J	--	2.1
Barium		366	10.96 J	--	24.8 J	--	29.2 J	6.26 J	50.8	30.6 J	9.52 J	58.8	6.96 J	10.62 J	46.4	48.4
Cadmium		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Chromium		1.18 J	0.84 J	--	1.34 J	--	--	1.10 J	2.38	--	--	0.86 J	--	--	--	0.98 J
Lead		--	--	--	--	--	--	--	--	--	--	--	0.46 J	1.54	--	--
Mercury		--	--	--	--	--	--	--	--	--	--	--	--	--	--	--
Selenium		--	--	--	--	--	--	--	1.26	--	--	--	--	0.62 J	--	--
ORGANOCHLORINE PESTACIDES	ug/L	--	--	NA	--	NA	NA	NA	NA	NA	NA	NA	NA	NA	--	--

Constituents not detected in any samples are not shown.

ug/L - micrograms per liter.

J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.

"--" - Constituent was analyzed but not detected.

NA - Not Analyzed.

Summary of Analyses of Ground-Water Samples Collected During the RFI at Area S/T
Redstone Arsenal, Alabama (Phase I), page 2 of 2

CONSTITUENT	UNITS	RS309	RS310	RS039	RS313	RS038	RS299	RS312	RS201	RS165	RS302	RS306	RS306(dup)	RS311	RS166	RS223
VOLATILE ORGANICS	ug/L															
Acetone		–	23	–	–	–	–	5 J	23	9 J	6 J	81	34	6 J	–	–
Benzene		–	–	–	–	–	–	–	–	–	–	–	6	–	–	–
Chlorobenzene		–	–	–	–	–	–	–	180	–	–	210	190	–	–	–
Chloroform		2 J	20	–	6	–	–	–	–	–	–	–	–	4 J	–	5 J
1,2-Dichloroethane		–	2 J	–	–	–	–	–	–	–	–	–	–	–	–	–
1,2-Dichloroethene(total)		–	11	–	–	–	–	–	–	–	–	–	–	9	–	–
Ethylbenzene		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Tetrachloroethene		–	3 J	–	2 J	–	–	–	–	–	–	–	–	–	–	–
Trichloroethene		–	25	–	5	–	–	–	4 J	–	–	–	–	20	–	–
Trichloroethane		–	–	–	–	–	–	–	4 J	–	–	–	–	–	–	–
Vinyl chloride		–	–	–	–	–	–	–	–	–	–	–	12	–	–	–
Xylenes (total)		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
METALS (Total)	ug/L															
Arsenic		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Barium		–	52.6 J	–	–	144 J	28.9 J	57.1 J	39.1 J	43.9 J	–	118.0 J	127 J	–	–	26.4 J
Cadmium		–	–	–	–	–	–	–	–	–	–	4.9	–	–	–	–
Chromium		–	16.6	–	–	–	–	–	–	9 J	–	–	–	–	5.5 J	–
Lead		–	–	–	–	–	–	–	–	–	–	–	3.2	–	4.0	–
Mercury		–	–	–	–	–	–	–	–	–	–	–	–	–	–	0.47
Selenium		–	–	2.2 J	–	–	–	–	4.3 J	2 J	–	3 J	–	3.4 J	–	2.0 J
Silver		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
METALS (Dissolved)	ug/L															
Arsenic		–	1.9 J	–	–	–	–	1.32 J	–	–	–	1.04 J	–	–	1.26 J	–
Barium		5.2	–	11.18 J	8.7 J	91.8	15.86 J	7.68 J	27.6 J	27.4 J	14.04 J	63.8	69	7.24 J	35.20 J	11.34 J
Cadmium		–	–	–	–	–	–	–	–	–	–	1.28	1.58	–	–	–
Chromium		1.0 J	5.52	1.96 J	1.36 J	1.02 J	1.6 J	1.96 J	0.92 J	1.16 J	–	–	0.9 J	–	0.84 J	–
Lead		–	0.5 J	14.12	0.82	0.82	1.3	1.56	–	–	–	–	–	–	–	2.24
Mercury		–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Selenium		–	0.54 J	–	–	–	–	–	–	–	–	–	–	–	–	–
ORGANOCHLORINE PESTACIDES	ug/L	–	NA	–	NA	–	NA	NA	NA	–	NA	–	0.6 J	NA	NA	–

Constituents not detected in any samples are not shown.
ug/L - micrograms per liter.
J - Indicates a qualitative value below laboratory reporting limit but above instrument detection limit.
"–" - Constituent was analyzed but not detected.
NA - Not Analyzed.

Summary of Analyses of Ground-Water Samples Collected During the Phase II RFI at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	AREA S/T-A (RS036A & RS037)		AREA S/T-B (RS201 & RS305)		RS304	RS384		RS385		RS307		RS310
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l):	NA		NA		NA	NA		NA		NA		
Acenaphthene												- -
Anthracene												- -
Fluoranthene												- -
Fluorene												- -
Naphthalene												- -
Phenanthrene												- -
Pyrene												- -
VOLATILE ORGANICS (ug/l):	NA		NA									NA
Acetone					61	- -		21		440	U	
Chlorobenzene					- -	- -		- -		3	J	
1,2-Dichloroethene (total)					- -	- -		- -		5		
Methylene Chloride					- -	2	U	- -	J	2	U	
Toluene					- -	3	J	- -	J	2	J	
Trichloroethene					- -	- -		- -		3	J	
GENERAL WATER QUALITY (mg/l):					NA	NA		NA		NA		NA
Calcium	87.9		57.1									
Iron	2.76	J	83.9	J								
Magnesium	3.48		6.05									
Manganese	1.71	J	4.75	J								
Potassium	1.41		10.4									
Sodium	11.6		3.91									
Total Alkalinity as CaCO3	164		144									
BOD (5days)	3.3		2.1									
Chloride	15.4		6									
Chemical Oxygen Demand	20		20									
Hardness as CaCO3	220		185									
Oil and Grease	- -		- -									
Sulfate	1.4		- -									
Total Dissolved Solids	250		200									
Total Organic Carbon	- -		- -									
Total Suspended Solids	1000		1100									

Constituents not detected in any sample are not shown.

ug/L micrograms per Liter
mg/l milligrams per liter

J Indicates a qualitative value either below laboratory reporting limit but above instrument detection limit or a value qualified due to a control deficiency identified during the data review presented in the QCSR.

U Indicates that the constituent was reported in the sample and in the associated field or laboratory blanks. Based on the data review presented in the QCSR, the constituent is classified as undetected at the quantitation limit shown.

- - Constituent was analyzed but not detected.

NA Not analyzed

Summary of Analyses of Ground-Water Samples Collected During the Phase II RFI at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	RS036		RS035		RS034	RS303	RS036A	RS037		RS306		RS308
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l):												
Acenaphthene	6	J	18		- -	- -	- -	- -		2	J	- -
Anthracene	2	J	- -		- -	- -	- -	- -		- -		- -
Fluoranthene	3	J	- -		- -	- -	- -	- -		- -		- -
Fluorene	7	J	8	J	- -	- -	- -	- -		- -		- -
Naphthalene	60		2	J	- -	- -	- -	5	J	- -		- -
Phenanthrene	14		- -		- -	- -	- -	- -		- -		- -
Pyrene	2	J	- -		- -	- -	- -	- -		- -		- -
VOLATILE ORGANICS (ug/l):	NA		NA		NA	NA	NA	NA		NA		NA
Acetone												
Chlorobenzene												
1,2-Dichloroethene (total)												
Methylene Chloride												
Toluene												
Trichloroethene												
GENERAL WATER QUALITY (mg/l):	NA		NA		NA	NA	NA	NA		NA		NA
Calcium												
Iron												
Magnesium												
Manganese												
Potassium												
Sodium												
Total Alkalinity as CaCO3												
BOD (5days)												
Chloride												
Chemical Oxygen Demand												
Hardness as CaCO3												
Oil and Grease												
Sulfate												
Total Dissolved Solids												
Total Organic Carbon												
Total Suspended Solids												

Constituents not detected in any sample are not shown.

ug/L micrograms per Liter
mg/l milligrams per liter

J Indicates a qualitative value either below laboratory reporting limit but above instrument detection limit or a value qualified due to a control deficiency identified during the data review presented in the QCSR.

U Indicates that the constituent was reported in the sample and in the associated field or laboratory blanks. Based on the data review presented in the QCSR, the constituent is classified as undetected at the quantitation limit shown.

- - Constituent was analyzed but not detected.

NA Not analyzed

APPENDIX B

EXPOSURE CONCENTRATIONS

OBS	LISTCODE	CONTAREA	PHASE	MEDIUM	EXPCONC	ANALYTE
1	ACET	F	2	GW	3.21E-02	ACET
2	AS	F	2	GW	4.02E-02	AS
3	BA	F	2	GW	9.13E-02	BA
4	CD-A*	F	2	GW	3.70E-03	CD-A*
5	CCL4	F	2	GW	8.00E-03	CCL4
6	CHCL3	F	2	GW	6.00E-03	CHCL3
7	CR	F	2	GW	6.90E-03	CR
8	PB	F	2	GW	1.28E-02	PB
9	HG	F	2	GW	3.26E-01	HG
10	TRCLE	F	2	GW	3.30E-03	TRCLE
11	ACET	F	2	SE	5.25E-02	ACET
12	AS	F	2	SE	4.05E-02	AS
13	BA	F	2	SE	2.33E-01	BA
14	BAANTR	F	2	SE	3.29E-01	BAANTR
15	BAPYR	F	2	SE	3.40E-01	BAPYR
16	BBFANT	F	2	SE	3.03E-01	BBFANT
17	CD-S*	F	2	SE	6.80E-04	CD-S*
18	CCL4	F	2	SE	3.00E-03	CCL4
19	CR	F	2	SE	1.05E-01	CR
20	CHRY	F	2	SE	3.25E-01	CHRY
21	FANT	F	2	SE	2.92E-01	FANT
22	PB	F	2	SE	1.07E-01	PB
23	HG	F	2	SE	3.98E-03	HG
24	CH2CL2	F	2	SE	2.26E-02	CH2CL2
25	PHANTR	F	2	SE	3.63E-01	PHANTR
26	PYR	F	2	SE	3.03E-01	PYR
27	ANAPNE	F	2	SO	9.14E-01	ANAPNE
28	ACET	F	2	SO	8.00E-02	ACET
29	ANTRC	F	2	SO	8.97E-01	ANTRC
30	AS	F	2	SO	7.06E-02	AS
31	BAANTR	F	2	SO	1.95E+00	BAANTR
32	BAPYR	F	2	SO	1.36E+00	BAPYR
33	BBFANT	F	2	SO	1.98E+00	BBFANT
34	BGHIPY	F	2	SO	1.02E+00	BGHIPY
35	B2EHP	F	2	SO	3.12E+00	B2EHP
36	CD-S*	F	2	SO	4.60E-04	CD-S*
37	CHRY	F	2	SO	1.63E+00	CHRY
38	DBAHA	F	2	SO	8.54E-01	DBAHA
39	FANT	F	2	SO	3.35E+00	FANT
40	FLRENE	F	2	SO	9.54E-01	FLRENE
41	ICDPYR	F	2	SO	1.10E+00	ICDPYR
42	HG	F	2	SO	3.57E-03	HG
43	CH2CL2	F	2	SO	1.71E-02	CH2CL2
44	NAP	F	2	SO	7.83E-01	NAP
45	PHANTR	F	2	SO	2.20E+00	PHANTR
46	PYR	F	2	SO	2.99E+00	PYR
47	BA	F	1	SW	3.21E-02	BA
48	ACET	G	1	GW	5.91E-03	ACET
49	AS	G	1	GW	7.63E-03	AS
50	BA	G	1	GW	2.07E+00	BA
51	CD-A*	G	1	GW	3.19E-02	CD-A*
52	CR	G	1	GW	3.19E-01	CR
53	PB	G	1	GW	1.39E-01	PB
54	ACET	G	1	SE	1.13E-01	ACET
55	AS	G	1	SE	4.25E-03	AS
56	BA	G	1	SE	7.23E-02	BA
57	CR	G	1	SE	5.44E-02	CR
58	CH2CL2	G	1	SE	2.12E-02	CH2CL2
59	ACET	G	1	SO	1.54E-01	ACET
60	CH2CL2	G	1	SO	1.20E-02	CH2CL2
61	ACET	G	2	SW	1.59E-02	ACET
62	BA	G	2	SW	8.07E-02	BA
63	11DCLE	G	2	SW	2.90E-03	11DCLE
64	CH2CL2	G	2	SW	5.70E-03	CH2CL2
65	TRCLE	G	2	SW	1.59E-02	TRCLE

OBS	LISTCODE	CONTAREA	PHASE	MEDIUM	EXPCONC	ANALYTE
66	XYLEN	G	2	SW	3.50E-03	XYLEN
67	ACET	R	1	GW	2.17E-02	ACET
68	AS	R	1	GW	6.04E-03	AS
69	BA	R	1	GW	6.98E-01	BA
70	B2CLEE	R	1	GW	6.13E-03	B2CLEE
71	B2EHP	R	1	GW	6.13E-03	B2EHP
72	CD-A*	R	1	GW	1.24E-02	CD-A*
73	CLC6H5	R	1	GW	1.42E-02	CLC6H5
74	CHCL3	R	1	GW	1.82E-02	CHCL3
75	CR	R	1	GW	3.00E-01	CR
76	PB	R	1	GW	5.02E-02	PB
77	CH2CL2	R	1	GW	2.72E-03	CH2CL2
78	11DCLE	R	1	SE	1.67E-02	11DCLE
79	ACET	R	1	SE	3.34E-02	ACET
80	AS	R	1	SE	1.77E-02	AS
81	BA	R	1	SE	1.41E-01	BA
82	BAANTR	R	1	SE	7.75E-01	BAANTR
83	BAPYR	R	1	SE	8.80E-01	BAPYR
84	BBFANT	R	1	SE	1.81E+00	BBFANT
85	BGHIPY	R	1	SE	9.20E-01	BGHIPY
86	B2EHP	R	1	SE	7.85E-01	B2EHP
87	CD-S*	R	1	SE	6.12E-03	CD-S*
88	CCL4	R	1	SE	1.07E-02	CCL4
89	CR	R	1	SE	3.90E-02	CR
90	CHRY	R	1	SE	8.26E-01	CHRY
91	FANT	R	1	SE	5.11E-01	FANT
92	ICDPYR	R	1	SE	9.26E-01	ICDPYR
93	PB	R	1	SE	5.90E-02	PB
94	HG	R	1	SE	1.07E-03	HG
95	CH2CL2	R	1	SE	3.76E-02	CH2CL2
96	PYR	R	1	SE	7.02E-01	PYR
97	ANAPNE	R	1	SO	3.93E-01	ANAPNE
98	ACET	R	1	SO	1.60E-01	ACET
99	ANTRC	R	1	SO	4.60E-01	ANTRC
100	BAANTR	R	1	SO	1.37E+00	BAANTR
101	BAPYR	R	1	SO	9.96E-01	BAPYR
102	BBFANT	R	1	SO	1.83E+00	BBFANT
103	BGHIPY	R	1	SO	6.69E-01	BGHIPY
104	B2EHP	R	1	SO	7.80E-01	B2EHP
105	BBZP	R	1	SO	7.80E-01	BBZP
106	CHRY	R	1	SO	9.96E-01	CHRY
107	FANT	R	1	SO	2.21E+00	FANT
108	FLRENE	R	1	SO	3.93E-01	FLRENE
109	ICDPYR	R	1	SO	6.69E-01	ICDPYR
110	CH2CL2	R	1	SO	8.93E-03	CH2CL2
111	PHANTR	R	1	SO	1.60E+00	PHANTR
112	PYR	R	1	SO	2.13E+00	PYR
113	SE	R	1	SO	8.35E-04	SE
114	AS	R	1	SW	1.05E-02	AS
115	BA	R	1	SW	1.30E-01	BA
116	B2EHP	R	1	SW	1.69E-02	B2EHP
117	CHCL3	R	1	SW	1.75E-02	CHCL3
118	111TCE	ST	2	GW	4.91E-03	111TCE
119	12DCLE	ST	2	GW	2.51E-03	12DCLE
120	12DCE	ST	2	GW	5.80E-03	12DCE
121	ANAPNE	ST	2	GW	5.42E-03	ANAPNE
122	ACET	ST	2	GW	4.90E-02	ACET
123	ANTRC	ST	2	GW	5.25E-03	ANTRC
124	BA	ST	2	GW	1.20E-01	BA
125	C6H6	ST	2	GW	5.79E-03	C6H6
126	CD-A*	ST	2	GW	2.78E-03	CD-A*
127	CLC6H5	ST	2	GW	3.48E-01	CLC6H5
128	CHCL3	ST	2	GW	4.15E-03	CHCL3
129	CR	ST	2	GW	1.22E-02	CR
130	ETC6H5	ST	2	GW	2.57E-03	ETC6H5

OBS	LISTCODE	CONTAREA	PHASE	MEDIUM	EXPCONC	ANALYTE
131	FANT	ST	2	GW	5.17E-03	FANT
132	FLRENE	ST	2	GW	9.18E-03	FLRENE
133	PB	ST	2	GW	2.16E-02	PB
134	HG	ST	2	GW	1.30E-04	HG
135	CH2CL2	ST	2	GW	2.50E-03	CH2CL2
136	NAP	ST	2	GW	2.16E-02	NAP
137	PHANTR	ST	2	GW	8.11E-03	PHANTR
138	PYR	ST	2	GW	5.25E-03	PYR
139	SE	ST	2	GW	1.84E-03	SE
140	TCLEE	ST	2	GW	2.54E-03	TCLEE
141	MEC6H5	ST	2	GW	2.54E-03	MEC6H5
142	TRCLE	ST	2	GW	5.66E-03	TRCLE
143	C2H3CL	ST	2	GW	8.32E-03	C2H3CL
144	XYLEN	ST	2	GW	2.70E-03	XYLEN
145	PPDDE	ST	2	SO	4.33E-03	PPDDE
146	PPDDT	ST	2	SO	2.76E-02	PPDDT
147	ACET	ST	2	SO	4.66E-01	ACET
148	B2EHP	ST	2	SO	1.97E-01	B2EHP
149	CH2CL2	ST	2	SO	1.88E-02	CH2CL2

OBS	ANALYTE	PHASE	EXPCONC	CONTAREA	MEDIUM
1	ACET		1.10E-02	G	FS
2	11DCLE		3.80E-03	G	FS
3	CH2CL2		2.90E-02	G	FS
4	TRCLE		1.70E-01	G	FS
5	XYLEN		7.70E-03	G	FS
6	BA		8.10E-01	G	FS

Calculation of UCLs/MDCs for Constituents Detected in Shallow Surface Soil and Shallow Soil Boring Samples Collected During Phase I and Phase II at Area
Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations FSB-2-S	FSB-3-A	FSB-4-A	FSB-5-A	FS-9-A	FS-7	FS-14	FS-16	FS-17	FS-18
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/kg):										
Acenaphthene	< 205	< 210	< 395	< 2000	1800 J	< 365	< 465	< 205	< 495	< 405
Anthracene	< 205	< 210	< 395	440 J	2800 J	< 365	< 465	< 205	< 495	24
Benzo (a) anthracene	< 205	< 210	190 J	1400 J	8800	< 365	39 J	< 205	< 495	140
Benzo (b) fluoranthene	< 205	< 210	170 J	1600 J	8600	< 365	56 J	< 205	< 495	280
Benzo (ghi) perlyene	< 205	< 210	< 395	890 J	3300 J	< 365	< 465	< 205	< 495	120
Benzo (a) pyrene	< 205	< 210	94 J	1000 J	5300	< 365	< 465	< 205	< 495	110
Bis(ethylhexyl) phthalate	140	NA	NA	NA	NA	< 365	NA	NA	NA	NA
Chrysene	< 205	< 210	130 J	1300 J	6800	< 365	46 J	< 205	< 495	140
Dibenzo (a,h) anthracene	< 205	< 210	< 395	< 2000	1300 J	< 365	< 465	< 205	< 495	< 405
Fluoranthene	< 205	< 210	330 J	2700 J	16000	< 365	110 J	< 205	< 495	280
Fluorene	< 205	< 210	< 395	< 2000	2100 J	< 365	< 465	< 205	< 495	< 405
Indeno (1,2,3-cd) pyrene	< 205	< 210	< 395	910 J	3800 J	< 365	< 465	< 205	< 495	110
Naphthalene	< 205	< 210	< 395	< 2000	450 J	< 365	< 465	< 205	< 495	< 405
Phenanthrene	< 205	< 210	210 J	1900 J	10000	< 365	70 J	< 205	< 495	170
Pyrene	< 205	< 210	310 J	2700 J	14000	< 365	100 J	< 205	< 495	290
VOLATILE ORGANICS (ug/kg):		NA	NA	NA	NA		NA	NA	NA	NA
Acetone	56					11				
Methylene Chloride	12					< 2.5				
METALS (total, mg/kg):										
Arsenic	8.8	171	154	128	85.4	9.2	10.5	4.7	4.5	45.7
Barium	29	NA	NA	NA	NA	128	NA	NA	NA	NA
Cadmium	< 0.48	< 0.76	< 0.355	< 0.355	< 0.355	0.45	< 0.42	< 0.37	< 0.44	< 0.36
Chromium	72.9	NA	NA	NA	NA	36.4	NA	NA	NA	NA
Lead	10.5	NA	NA	NA	NA	32.1	NA	NA	NA	NA
Mercury	0.57	0.44	9.5	0.59	5.9	0.28	1.0	0.25	1.3	9.1
Selenium	0.48	NA	NA	NA	NA	0.225	NA	NA	NA	NA

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

Samples from top soil borings are identified by "SB" followed by the boring number and the letter "S" or "A". The corresponding deep soil boring samples are provided in separate tables.

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed
a/ Arithmetic Mean
b/ Upper Confidence Limit (95%)
c/ Maximum Detected Concentration

Calculation of UCLs/MDCs for Constituents Detected in Shallow Surface Soil and Shallow Soil Boring Samples Collected During Phase I and Phase II at Area F
Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	FS-19	FS-20	FS-21	FS-22	FS-23	$\bar{X}$ a/	UCL b/	MDC c/
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/kg):								
Acenaphthene	< 480	< 1050	< 1100	< 380	< 415	664.67	914.14	
Anthracene	< 480	< 1050	< 1100	< 380	< 415	601.93	897.48	
Benzo (a) anthracene	31	J < 1050	< 1100	17	J < 415	977.47	1946.41	
Benzo (b) fluoranthene	< 480	< 1050	< 1100	< 380	< 415	1040.73	1978.09	
Benzo (ghi) perlyene	< 480	< 1050	< 1100	< 380	< 415	671.67	1017.30	
Benzo (a) pyrene	< 480	< 1050	< 1100	< 380	< 415	791.60	1358.21	
Bis(ethylhexyl) phthalate	NA	NA	NA	NA	NA	252.50	3122.25	140
Chrysene	< 480	< 1050	< 1100	< 380	< 415	888.07	1625.96	
Dibenzo (a,h) anthracene	< 480	< 1050	< 1100	< 380	< 415	631.33	854.50	
Fluoranthene	50	J < 1050	< 1100	28	J < 415	1569.53	3348.18	
Fluorene	< 480	< 1050	< 1100	< 380	< 415	684.67	954.45	
Indeno (1,2,3-cd) pyrene	< 480	< 1050	< 1100	< 380	< 415	705.67	1104.63	
Naphthalene	< 480	< 1050	< 1100	< 380	< 415	574.67	783.07	
Phenanthrene	18	J < 1050	< 1100	19	J < 415	1095.47	2201.02	
Pyrene	57	J < 1050	< 1100	28	J < 415	1435.33	2990.93	
VOLATILE ORGANICS (ug/kg):	NA	NA	NA	NA	NA			
Acetone						33.50	79.96	56
Methylene Chloride						7.25	17.06	12
METALS (total, mg/kg):								
Arsenic	20.9	5.8	6.2	5.6	5.0	44.35	70.63	
Barium	NA	NA	NA	NA	NA	78.50	180.71	128
Cadmium	< 0.435	< 0.375	< 0.395	< 0.34	< 0.375	0.42	0.46	0.45
Chromium	NA	NA	NA	NA	NA	54.65	92.33	72.9
Lead	NA	NA	NA	NA	NA	21.30	43.60	32.1
Mercury	1.7	0.37	0.46	0.26	0.53	2.15	3.57	
Selenium	NA	NA	NA	NA	NA	0.35	0.62	0.48

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

Samples from top soil borings are identified by "SB" followed by the boring number and the letter "S" or "A". The corresponding deep soil boring samples are provided in separate tables.

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed
a Arithmetic Mean
b Upper Confidence Limit (95%)
c/ Maximum Detected Concentration

Calculation of UCLs/MDCs for Constituents Detected in Sediment Samples Collected During Phase I and Phase II at Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations FS-5	FS-6	FS-8	FS-9	FS-10	FS-11	FS-12	FS-13	FS-15	$\bar{X}$ a/	UCL b/	MDC c/
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/kg):												
Benzo (a) anthracene	120	< 405	< 195	< 200	< 430	< 600	63 J	< 485	< 210	300.89	328.79	120
Benzo (b) fluoranthene	150	150	< 195	< 200	< 430	< 600	140 J	< 485	< 210	284.44	303.04	150
Benzo (a) pyrene	60	< 405	< 195	< 200	< 430	< 600	54 J	< 485	< 210	293.22	339.72	60
Chrysene	140	< 405	< 195	< 200	< 430	< 600	65 J	< 485	< 210	303.33	325.03	140
Fluoranthene	280	< 405	< 195	< 200	< 430	< 600	85 J	32 J	< 210	270.78	292.48	280
Phenanthrene	220	< 405	< 195	< 200	< 430	< 600	< 495	< 485	< 210	360.00	363.10	220
Pyrene	280	< 405	< 195	< 200	< 430	< 600	170 J	42 J	< 210	281.33	303.03	280
VOLATILE ORGANICS (ug/kg):												
Acetone	47	54	45	35	NA	NA	NA	NA	NA	45.25	52.49	
Carbon Tetrachloride	< 3	< 3	3	< 3						3.00	3.00	
Methylene Chloride	16	24	18	8						16.50	22.60	
METALS (total, mg/kg):												
Arsenic	7.9	294	7.9	10.3	3.7	3.7	13.1	5.8	2.1	38.72	40.52	
Barium	179	85.2	262	44.0	NA	NA	NA	NA	NA	142.55	232.69	
Cadmium	< 0.48	1.2	< 0.46	1.7	< 0.39	< 0.355	< 0.445	< 0.44	< 0.375	0.65	0.68	
Chromium	51.9	22.9	61.7	125	NA	NA	NA	NA	NA	65.38	105.10	
Lead	68.8	35.5	34.7	127	NA	NA	NA	NA	NA	66.50	106.52	
Mercury	< 0.06	32.9	0.13	0.14	0.26	0.18	0.67	0.66	0.25	3.92	3.98	

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed
a/ Arithmetic Mean
b/ Upper Confidence Limit (95%)
c/ Maximum Detected Concentration

Calculations of UCLs for Constituents Detected in Surface Water Samples Collected in Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations SWF-1	$\bar{X}$ a/	UCL b/
METALS (total, ug/L):			
Barium	32.1	32.1	32.1

ug/L microgram per Liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RAFSW.XLS

Calculation of UCLs for Constituents Detected in Ground-Water Samples Collected During Phase I and Phase II at Area F, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations														
	RS262	RS263	RS052	RS256	RS259	RS054	RS053	RS055	RS257	RS260	RS261	RS258	$\bar{X}$ a	b/ UCL
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/l):	NA	ND	ND	ND	ND	NA	NA	ND	ND	NA	ND	ND		
VOLATILE ORGANICS (ug/L)														
Acetone	< 5	< 5	84	< 5	< 5	< 5	< 5	< 5	< 5	61	< 5	29	18.3	32.1
Chloroform	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	16	6	3.9	6.0
Carbon tetrachloride	14	7	< 2.5	< 2.5	< 2.5	< 2.5	4	4	< 2.5	4	17	< 2.5	5.4	8.0
Trichloroethene	5	4	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	3	< 2.5	2.9	3.3
METALS (Total, ug/L)														
Arsenic	5.8	67.9	26.5	< 2.5	< 2.5	< 2	7.6	25.2	< 2	< 2	20.2	110	22.9	40.2
Barium	< 9	18.6	35.5	23.9	23.4	< 9	27.1	23.8	21.1	35.8	< 9	320	46.4	91.3
Cadmium	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	7.6	2.9	3.7
Chromium	< 2.5	< 2.5	16.1	8.4	7.1	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	5 J	4.7	6.9
Lead	< 0.5	< 0.5	1.6	1.8	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	52.4	5.0	12.8
Mercury	< 0.1	< 0.1	< 0.1	< 0.1	0.1	0.3	< 0.1	< 0.1	< 0.1	1.4	< 0.1	2800	233.6	652.6
METALS (Dissolved, ug/L)														
Arsenic	8.76	99.8	56.6	< 0.4	0.94	1.22	8.34	28.4	< 0.4	1.48	22	68.4	24.7	41.8
Barium	18.98	19.48	23	21.4	17.74	10.46	29.2	21.8	30	61.8	23.6	110	32.3	46.6
Cadmium	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	4.28	< 0.5	3	1.0	1.7
Chromium	2.6	1.86	2.9	1.68	4.12	2.18	3.28	2.3	6.1	4.08	2.76	< 1.5	2.9	3.6
Lead	0.46	0.48	< 0.5	< 0.5	< 0.5	2.3	< 0.5	< 0.5	< 0.5	3.3	0.32	< 0.5	0.9	1.3
Mercury	< 0.02	< 0.02	< 0.02	< 0.02	< 0.02	< 0.02	< 0.02	< 0.02	< 0.02	0.104	< 0.02	26	2.2	6.1
Selenium	< 0.3	< 1	< 0.3	1.82	0.98	< 0.3	0.76	0.76	< 0.3	< 0.3	0.68	< 1.5	0.8	1.0

ug/L micrograms per Liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean
b Upper Confidence Limit (95%)
NA Not analyzed
ND Not detected

Calculations of UCLs for Constituents Detected in Shallow Soil Boring Samples Collected in Area G
Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations GSB-1-S	GSB-2-S	GSB-3-S	$\bar{X}$ a/	UCL b/
VOLATILE ORGANICS (ug/kg):					
Acetone	32	26	140	66	154
Methylene Chloride	11	< 3	3	5.7	12.0
METALS (total, mg/kg):					
Arsenic	1.8	12.9	3.0	5.9	14.3
Barium	135	24.5	79.3	79.6	155.7
Chromium	50.2	99.5	71.1	73.6	107.7
Lead	7.5	26.7	10.9	15.0	29.1
Mercury	< 0.06	0.49	< 0.06	0.20	0.55

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RAGSS.XLS

Calculations of UCLs for Constituents Detected in Sediment Samples Collected in Area G
Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations GSS-4	GSS-5	GSS-6	GSS-7	GSS-8	$\bar{X}$ a/	UCL b/
VOLATILE ORGANICS (ug/kg):							
Acetone	91	160	4	< 8.5	< 6.5	54.0	113.4
Methylene Chloride	24	16	15	19	17	18.2	21.2
METALS (total, mg/kg):							
Arsenic	< 0.39	< 0.39	2.9	5.7	2.5	2.4	4.2
Barium	75.8	56.1	51.6	22.1	68	54.7	72.3
Chromium	54.9	57.8	32.7	42.5	27.2	43.0	54.4
Lead	6.2	12.1	6.2	26.3	8.4	11.8	19.0
Mercury	0.42	0.29	< 0.08	0.15	< 0.07	0.20	0.33

ug/Kg micrograms per Kilogram
mg/Kg milligrams per Kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RAGSE.XLS

Calculation of UCLs/MDCs for Constituents Detected in Surface Water Samples Collected During Phase I and Phase II at Area G, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations SW-G-3	SW-G-4	SW-G-5	SW-G-4(II)	SW-G-5(II)	SW-G-6	SW-G-7	SW-G-8	SW-G-9	SW-G-10	$\bar{X}$ a/	UCL b/	MDC c/
VOLATILE ORGANICS (ug/L):													
Acetone	20	< 2.5	25	< 5	< 5	23	14	7 J	< 5	< 5	11.2	15.9	
Dichloroethene, 1,1-	< 2.5	< 2.5	< 2.5	< 2.5	4 J	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	2.7	2.9	
Methylene chloride	10	10	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	4.0	5.7	
Trichloroethene	< 2.5	26	28	9	9	< 2.5	< 2.5	10	10	10	11.0	15.9	
Xylenes (Total)	< 2.5	< 2.5	< 2.5	< 2.5	6	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	2.9	3.5	
METALS (total, ug/L):													
Barium	72.6	< 9.0	< 9.0	NA	NA	NA	NA	NA	NA	NA			
Lead	< 1.0	< 1.0	5.8								30.2	80.7	72.6
Mercury	< 0.1	1.6	< 0.1								2.6	6.4	5.8
											0.6	1.8	1.6

ug/L micrograms per Liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a/ Arithmetic Mean
b/ Upper Confidence Limit (95%)
c/ Maximum Detected Concentration

NA Not analyzed

(II) Collected during the Phase II RFI

Calculation of UCLs for Constituents Detected in Ground-Water Samples Collected at Area G
Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations										
	RS111	RS113	RS114	RS265	RS266	RS267	RS112	RS264	RS268	$\bar{X}$ a/	UCL b/
VOLATILE ORGANICS (ug/L)											
Acetone	< 5	8	< 5	< 5	< 5	< 5	< 5	< 5	< 5	5.33	5.91
METALS (Total,ug/L)											
Arsenic	6.9	9.8	< 2.5	< 2.5	10.8	< 2.5	6.6	< 2.5	7.2	5.70	7.63
Barium	364	672	48.3	< 9	5910	146	930	193	571	982.59	2068.45
Cadmium	8.5	18.7	< 2.5	< 2.5	79.1	< 2.5	21.1	< 2.5	21.7	17.68	31.89
Chromium	156	394	47	< 2.5	626	5.1	240	18.2	276	196.09	319.35
Lead	30.4	146	6.2	< 0.5	263	< 0.5	172	2.7	128	83.26	139.39
METALS (Dissolved, ug/L)											
Arsenic	< 0.3	< 0.3	< 0.3	< 0.3	< 0.3	2.92	< 0.3	0.64	< 0.3	0.63	1.13
Barium	66.2	57.8	7.5	5.68	36.4	120.2	34.6	160.6	3.8	54.75	86.24
Chromium	1.06	< 0.5	2	2.52	1.3	1.1	< 0.5	< 0.5	2.6	1.34	1.83
Lead	4.92	< 0.1	< 0.1	< 0.1	< 0.1	0.54	< 0.1	< 0.5	< 0.1	0.73	1.65

ug/L micrograms per Liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

FILE: ARGGWAB.XLS

Calculation of UCLs for Shallow Soil and Shallow Boring Samples Collected in Area R, Redstone Arsenal, Alabama

CONSTITUENT	Sample Locations RSB-1-S		RSB-2-S		RS-11		RS-12	$\bar{X}$ a/	UCL b/
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/Kg):									
Acenaphthene	200	<	200	<	380	<	375	289	393
Anthracene	450	<	200	<	380	<	375	351	460
√ Benzo(a)anthracene	1700	<	200	<	380	<	375	664	1373
√ Benzo(a)pyrene	1200	<	200	<	380	<	375	539	996
Benzo(b)fluoranthene	2300	<	200	<	380	<	375	814	1827
Benzo(g,h,i)perylene	760	<	200	<	380	<	375	429	669
Butylbenzyl phthalate	910	<	200	<	380	<	375	466	780
Chrysene	1200	<	200	<	380	<	375	539	996
Bis-(2-ethylhexyl)phthalate	900		97	<	380	<	375	438	780
Fluoranthene	2800	<	200	<	380	<	375	939	2206
Fluorene	200	<	200	<	380	<	375	289	393
Indeno(1,2,3-c,d)pyrene	760	<	200	<	380	<	375	429	669
Phenanthrene	2000	<	200	<	380	<	375	739	1600
Pyrene	2700	<	200	<	380	<	375	914	2130
VOLATILE ORGANICS (ug/kg):									
Acetone	200		53	<	5.5	<	5.5	66.0	159.8
Methylene Chloride	9		8		4		5	7	9

Calculation of UCLs for Shallow Soil and Shallow Boring Samples Collected in Area R, Redstone Arsenal, Alabama

CONSTITUENT	Sample Locations RSB-1-S	RSB-2-S	RS-11	RS-12	$\bar{X}$ a/	UCL b/
METALS (total, mg/kg):						
Arsenic	6.9	7.6	7.5	5.9	7.0	7.8
Barium	64.0	45.5	172.0	69.9	87.9	146.0
Chromium	23.3	49.2	11.2	23.5	26.8	43.1
Lead	53.3	8.8	87.4	142.0	72.9	130.1
Mercury	2.9	0.17	< 0.05	0.53	0.9	2.3
Selenium	0.76	0.81	< 0.22	< 0.23	0.51	0.84

ug/Kg micrograms per kilogram
mg/Kg milligrams per kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RARSS.XLS

Calculation of UCLs for Constituents Detected in Sediment Samples Collected at Area R, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations RS-7	RS-8	RS-9	RS-10	$\bar{X}$ a/	UCL b/
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/kg):						
Benzo(a)anthracene	490	< 480	< 850	< 210	508	775
Benzo(a)pyrene	780	< 480	< 850	< 210	580	880
Benzo(b)fluoranthene	2200	< 480	710	< 210	900	1807
Benzo(g,h,i)perylene	860	< 480	< 850	< 210	600	920
Bis(2-Ethylhexyl)phthalate	530	< 480	< 850	< 210	518	785
Chrysene	950	< 480	360	< 210	500	826
Fluoranthene	490	< 480	310	< 210	373	511
Indeno(1,2,3-cd)pyrene	870	< 480	< 850	< 210	603	926
Pyrene	760	< 480	440	< 210	473	702
VOLATILE ORGANICS (ug/kg):						
Acetone	41	< 7	12	8	17	33
Carbon Tetrachloride	< 11	9	< 3	< 3	7	11
1,1-Dichloroethane	21	< 3.5	< 3	< 3	7.6	16.7
Methylene Chloride	43	23	15	9	23	38

Calculation of UCLs for Constituents Detected in Sediment Samples Collected at Area R, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations RS-7	RS-8	RS-9	RS-10	$\bar{X}$ a/	UCL b/
METALS (total, mg/kg):						
Arsenic	8.3	< 0.38	21.1	4.4	8.5	17.7
Barium	109	96.2	82.4	152	109.9	140.6
Cadmium	7.9	< 0.5	< 0.5	< 0.5	2.4	6.1
Chromium	43.2	12.3	19.9	26.8	25.6	39.0
Lead	58.6	29.7	30.4	54.6	43.3	59.0
Mercury	1.2	< 0.65	0.62	< 0.65	0.8	1.1
Silver	142	4.5	< 0.5	< 0.5	36.9	108.3

ug/Kg micrograms per Kilogram
mg/Kg milligrams per Kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RARSE.XLS

Calculations of UCLs for Constituents Detected in Surface Water Samples Collected at Area R, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations SW-R-1	SW-R-2	$\bar{X}$ a/	UCL b/
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/L):				
Bis(2-ethylhexyl)phthalate	6	10	8	17
VOLATILE ORGANICS (ug/L):				
Chloroform	< 2.5	8	5.3	17.5
METALS (total, ug/L):				
Arsenic	5.1	< 2.0	3.6	10.5
Barium	76.9	46.3	61.6	129.9
Silver	< 2.5	6.2	4.4	12.6

ug/L micrograms per Liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean

b Upper Confidence Limit (95%)

RARSW.XLS

Calculation of UCLs for Constituents Detected in Ground-Water Samples Collected at Area R, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations RS220	RS221	RS295	RS294	RS222	RS293	$\bar{X}$ a/	b/ UCL
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/L)	NA	NA			NA			
Bis(2-Chloroethyl)ether			< 5	6		< 5	5	6
Bis(2-Ethylhexyl)phthalate			< 5	< 5		6	5	6
VOLATILE ORGANICS (ug/L)								
Acetone	< 5	3	15	< 5	40	3	12	22
Chlorobenzene	< 2.5	< 2.5	< 2.5	28	< 2.5	6	7.3	14.2
Chloroform	< 2.5	38	< 2.5	< 2.5	< 2.5	3	8.5	18.2
Methylene Chloride	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 3	2.6	2.7
METALS (Total, ug/L))								
Arsenic	< 2.5	8.8	6.4	< 2.5	< 2.5	< 2.5	4.2	6.0
Barium	24.4	115	1340	263	43.3	398	364.0	698.4
Cadmium	< 2.5	< 2.5	22.1	< 2.5	< 2.5	10.2	7.1	12.4
Chromium	11.5	69.3	539	155	55.1	196	171.0	300.1
Lead	1.3	19.1	88	4.7	3.7	46.5	27.2	50.2
METALS (Dissolved, ug/L)								
Arsenic	< 0.3	0.8	< 0.3	< 0.3	< 0.3	0.92	0.5	0.7
Barium	10.3	42.8	282	115.2	12.6	115.8	96.5	165.1
Chromium	1.38	< 0.5	< 0.5	1.1	1.22	< 0.5	0.87	1.14
Selenium	< 1.5	3.6	< 1.5	< 1.5	< 0.3	< 0.3	1.5	2.3

NA Not Analyzed
ug/L micrograms per Liter

Values preceded by "<" identify those consituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

a Arithmetic Mean
b Upper Confidence Limit (95%)

FILE: ARRGWA.XLS

Calculation of UCLs/MDCs for Constituents Detected in Shallow Soil Boring Samples Collected During Phase I and Phase II at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations												$\bar{X}$ a/	b/ UCL	c MDC
	SSB-1-S	SSB-2-S	SSB-3-S	TSB-1-S	TSB-2-S	TSB-3-S	TSB-4-S	TSB-5-S	TSB-6-A	TSB-6A-A	TSB-7-A	TSB-7A-A			
BASE/NEUTRAL AND ACID EXTRACTABLE ORGANICS (ug/Kg):															
Bis(2-ethylhexyl)phthalate	110	< 190	96	< 195	< 205	< 185	< 190	< 195	NA	NA	NA	NA	170.75	197.31	110
VOLATILE ORGANICS (ug/Kg):															
Acetone	34	87	150	97	24	44	570	120	< 6	NA	1300	NA	243.20	466.27	
Methylene Chloride	8	10	10	16	5	11	40	9	3 J		18 J		13.00	18.77	
METALS (total, mg/Kg):															
Arsenic	8.1	4.5	7.9	4.9	7.4	4.3	8.6	6.2	NA	NA	NA	NA	6.49	7.58	
Barium	30.5	13.5	19.5	94.8	30.4	91.1	8	11					37.35	59.48	
Chromium	53.6	54.9	30.4	59.9	51.5	23.8	26.1	49.5					43.71	52.77	
Lead	15.2	4.8	13.1	10.6	10.7	12.8	6.6	5.5					9.91	12.33	
Mercury	0.17	< 0.06	< 0.06	< 0.05	< 0.06	< 0.05	< 0.05	< 0.05					0.07	0.09	
ORGANOCHLORINE PESTICIDES (ug/Kg):															
4,4'-DDE	3	0.8	< 0.06	1.1	< 1.2	3.4	12	1.02	< 0.6	NA	< 0.6	NA	2.38	4.33	
4,4'-DDT	1.92	1.83	< 0.01	1.8	< 3.84	4.5	95	0.66	< 2.5		< 2.4		11.45	27.61	

ug/kg micrograms per kilogram
mg/kg milligrams per kilogram

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed
a/ Arithmetic Mean
b/ Upper Confidence Limit (95%)
c/ Maximum Detected Concentration

Calculation of UCLs/MDCs for Constituents Detected in Ground-Water Samples Collected During Phase I and Phase II at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations														
	RS309	RS310	RS039	RS313	RS038	RS299	RS312	RS201	RS165	RS302	RS306	RS311	RS166	RS223	RS036
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l):	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Acenaphthene															
Anthracene															
Fluoranthene															
Fluorene															
Naphthalene															
Phenanthrene															
Pyrene															
VOLATILE ORGANICS (ug/L):															
Acetone	< 5	23	< 5	< 5	< 5	< 5	5	23	9	6	81	6	< 5	< 5	< 5
Benzene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	31
Chlorobenzene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	180	< 2.5	< 2.5	210	< 2.5	< 2.5	< 2.5	650
Chloroform	2	20	< 2.5	6	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	4	< 2.5	5	< 2.5
1,2-Dichloroethane	< 2.5	2	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
1,2-Dichloroethene (total)	< 2.5	11	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	9	< 2.5	< 2.5	< 2.5
Ethylbenzene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	3
Methylene Chloride	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Tetrachloroethene	< 2.5	3	< 2.5	2	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Toluene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
1,1,1-Trichloroethane	< 5	< 5	< 5	< 5	< 5	< 5	< 5	4	< 5	< 5	< 5	< 5	< 5	< 5	< 5
Trichloroethene	< 2.5	25	< 2.5	5	< 2.5	< 2.5	< 2.5	4	< 2.5	< 2.5	< 2.5	20	< 2.5	< 2.5	< 2.5
Vinyl Chloride	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5
Xylenes	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
METALS (Total, ug/L):															
Barium	< 13	52.6	< 13	< 13	144	28.9	57.1	39.1	43.9	< 13	118	< 9	< 13	26.4	792
Cadmium	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	4.9	< 2.5	< 2	< 2	4.3
Chromium	< 2	16.6	< 2	< 2	< 2	< 2	< 2	9	< 2	< 2	< 2	< 2.5	5.5	< 2	74.8
Lead	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 0.5	4	< 1	204
Mercury	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	0.47	< 0.1
Selenium	< 1	< 1	2.2	< 1	< 1	< 1	< 1	4.3	2	< 1	3	3.4	< 1	2	< 1
METALS (Dissolved, ug/L):															
Arsenic	< 0.5	1.9	< 0.5	< 0.5	< 0.5	< 0.5	1.32	< 0.5	< 0.5	< 0.5	1.04	< 0.3	1.26	< 0.5	< 0.5
Barium	5.2	< 2.6	11.18	8.7	91.8	15.86	7.68	27.6	27.4	14.04	63.8	7.24	35.2	11.34	366
Cadmium	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	0.4	< 0.4	< 0.4	< 0.4	1.28	< 0.5	< 0.4	< 0.4	< 0.4
Chromium	1	5.52	1.96	1.36	1.02	1.6	1.96	0.92	1.16	< 0.4	< 0.4	< 0.5	0.84	< 0.4	1.18
Lead	< 0.34	0.5	14.12	0.82	0.82	1.3	1.56	< 0.2	< 0.2	< 0.2	< 0.2	< 0.1	< 0.2	2.24	< 0.2
Selenium	< 0.2	0.54	< 0.2	< 0.2	< 0.2	< 0.2	0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.3	< 0.2	< 0.2	< 1

ug/L micrograms per liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed

a/ Arithmetic Mean

b/ Upper Confidence Limit (95%)

c/ Maximum Detected Concentration

Calculation of UCLs/MDCs for Constituents Detected in Ground-Water Samples Collected During Phase I and Phase II at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations RS036A	RS037	RS034	RS200	RS301	RS202	RS203	RS300	RS305	RS308	RS297	RS298	RS303	RS035	RS385	RS304
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l):	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Acenaphthene																
Anthracene																
Fluoranthene																
Fluorene																
Naphthalene																
Phenanthrene																
Pyrene																
VOLATILE ORGANICS (ug/L):																
Acetone	< 5	< 5	< 5	< 5	15	< 5	< 5	< 5	17	19	18	41	10	10	21	61
Benzene	< 2.5	19	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	7	< 2.5	< 2.5	7	< 2.5	< 2.5	< 2.5
Chlorobenzene	87	3000	37	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	190	< 2.5	< 2.5	1400	200	< 2.5	< 2.5
Chloroform	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
1,2-Dichloroethane	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
1,2-Dichloroethene (total)	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	36	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Ethylbenzene	< 2.5	3	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Methylene Chloride	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Tetrachloroethene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Toluene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
1,1,1-Trichloroethane	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5
Trichloroethene	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	13	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
Vinyl Chloride	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5	46	< 5	< 5	< 5	< 5	< 5	< 5
Xylenes	< 2.5	5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5	< 2.5
METALS (Total, ug/L):																
Barium	259	51.3	59.6	< 2.6	50.8	< 2.6	< 2.6	45.5	42.4	87.7	< 13	46	65.8	60.5	NA	NA
Cadmium	7.8	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2	< 2		
Chromium	11.3	9.3	15.2	< 2	7.6	< 2	< 2	< 2	15.4	8.5	< 2	18.5	< 2	< 2		
Lead	45.5	2.7	3.2	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1		
Mercury	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1	< 0.1		
Selenium	< 1	< 1	< 1	< 1	2.4	< 1	< 1	< 1	2.6	< 1	2.4	< 1	2	< 1		
METALS (Dissolved, ug/L):																
Arsenic	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	2.5	< 0.5	1.86	< 0.5	2.1	NA	NA
Barium	10.96	< 2.6	24.8	< 2.6	29.2	6.26	50.8	30.6	9.52	58.8	6.96	10.62	46.4	48.4		
Cadmium	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4	< 0.4		
Chromium	0.84	< 0.4	1.34	< 0.4	< 0.4	1.1	2.38	< 0.4	< 0.4	0.86	< 0.4	< 0.4	< 0.4	0.98		
Lead	< 0.2	< 0.2	< 0.2	< 0.2	< 1	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	0.46	1.54	< 0.2	< 0.2		
Selenium	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	< 0.2	1.26	< 0.2	< 0.2	< 0.2	< 0.2	0.62	< 0.2	< 0.2		

ug/L micrograms per liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed

a/ Arithmetic Mean

b/ Upper Confidence Limit (95%)

c/ Maximum Detected Concentration

Calculation of UCLs/MDCs for Constituents Detected in Ground-Water Samples Collected During Phase I and Phase II at Area S/T, Redstone Arsenal, Huntsville, Alabama

CONSTITUENT	Sample Locations RS384	RS307	RS310	RS036	RS035	RS308	RS306	RS034	RS303	RS036A	RS037	$\bar{X}$ a/	UCL b/	MDC c/
POLYNUCLEAR AROMATIC HYDROCARBONS (ug/l):	NA	NA												
Acenaphthene			< 5	6 J	< 5	< 5	2 J	< 5	< 5	< 5	< 5	4.78	5.42	
Anthracene			< 5	2 J	< 5	< 5	< 5	< 5	< 5	< 5	< 5	4.67	5.25	2
Fluoranthene			< 5	3 J	< 5	< 5	< 5	< 5	< 5	< 5	< 5	4.78	5.17	3
Fluorene			< 5	7 J	18	< 5	< 5	< 5	< 5	< 5	< 5	6.67	9.18	
Naphthalene			< 5	60	2 J	< 5	< 5	< 5	< 5	< 5	5 J	10.78	21.58	
Phenanthrene			< 5	14	8 J	< 5	< 5	< 5	< 5	< 5	< 5	6.33	8.11	
Pyrene			< 5	2 J	< 5	< 5	< 5	< 5	< 5	< 5	< 5	4.67	5.25	2
VOLATILE ORGANICS (ug/L):			NA	NA	NA	NA	NA	NA	NA	NA	NA			
Acetone	< 5	440										26.82	48.95	
Benzene	< 2.5	< 2.5										4.14	5.79	
Chlorobenzene	< 2.5	3 J										182.26	348.48	
Chloroform	< 2.5	< 2.5										3.24	4.15	
1,2-Dichloroethane	< 2.5	< 2.5										2.48	2.51	2
1,2-Dichloroethene (total)	< 2.5	5										4.05	5.80	
Ethylbenzene	< 2.5	< 2.5										2.53	2.57	
Methylene Chloride	2 J	2 J										2.47	2.50	2
Tetrachloroethene	< 2.5	< 2.5										2.50	2.54	
Toluene	3 J	2 J										2.50	2.54	
1,1,1-Trichloroethane	< 2.5	< 2.5										4.67	4.91	4
Trichloroethene	< 2.5	3 J										4.17	5.66	
Vinyl Chloride	< 5	< 5										6.24	8.32	
Xylenes	< 2.5	< 2.5										2.58	2.70	
METALS (Total, ug/L):	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA			
Barium												74.67	120.48	
Cadmium												2.40	2.78	
Chromium												7.87	12.19	
Lead												9.76	21.63	
Mercury												0.11	0.13	
Selenium												1.56	1.84	
METALS (Dissolved, ug/L):	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA			
Arsenic												0.79	0.97	
Barium												35.66	56.53	
Cadmium												0.43	0.48	
Chromium												1.07	1.38	
Lead												0.97	1.77	
Selenium												0.29	0.37	

ug/L micrograms per liter

Values preceded by "<" identify those constituents which were not detected. Values given are equal to one-half the reported detection limit and may vary among samples due to differences in the sample matrix.

NA Not analyzed

a/ Arithmetic Mean

b/ Upper Confidence Limit (95%)

c/ Maximum Detected Concentration

HUMAN INTAKE ESTIMATION: METHODS AND EXPOSURE FACTORS

1.0 Introduction

ESE uses internally-generated software called the Automated Risk Evaluation System (ARES) to estimate the exposure of various receptors to environmental chemicals and the risks associated with those exposures. Using ARES Version 3.0, designed within the SAS/STAT Version 6.03 structure, daily chemical exposures are calculated for each completed pathway for each potential receptor using appropriate exposure formulas and factors presented in various EPA guidance documents, including the Risk Assessment Guidance for Superfund (RAGS), Human Health Evaluation Manual, Part A (EPA, 1989b), Part B (EPA, 1991a), and Supplemental Guidance (EPA, 1991b); Dermal Exposure Assessment: Principles and Applications (EPA, 1992a); and New EPA Region IV Guidance (EPA, 1992b). Where appropriate, exposure factors based on site-specific information are used in place of EPA standard default values. After determining daily exposures, ARES calculates the potential carcinogenic and noncarcinogenic risks associated with those exposures using appropriate risk reference doses (RfDs) and cancer slope factors (CSFs) available in various EPA sources, including the Integrated Risk Information System (IRIS, 1995), Region III's Risk-Based Concentration Tables (EPA, 1995), Health Effects Assessment Summary Tables (HEAST) (EPA, 1994), and Environmental Criteria and Assessment Office (ECAO) position papers; as well as Toxicological Profiles developed by the Agency for Toxic Substance and Disease Registry (ATSDR). Where no RfD is available, a provisional value is calculated using an EPA Maximum Contaminant Level (MCL) or health advisory, if available, or chronic animal data (with appropriate uncertainty factors). The following documentation provides a list of the exposure scenarios (Section 2.0), the exposure formulas (Section 3.0), and the exposure factors (Section 4.0) used to calculate the chemical intakes for RSA Sites F, G, R, and S/T, as well as the references used to develop ARES for BRA 1 (Section 5.0).

2.0 Exposure Scenarios Applicable in ARES for RSA BRA 1

Due to the presence of multiple contamination areas at RSA, exposure scenarios were developed to facilitate easier risk calculations. Each exposure scenario is unique for receptor, exposure pathways, and exposure parameters. A list of the exposure scenarios, the sites to which each scenario applies, and the exposure pathways applicable to each scenario are presented in Table C-1.

3.0 Exposure Formulas Used in ARES for RSA BRA 1

The exposure formulas incorporated in ARES are based on the formulas given in RAGS Parts A (EPA, 1989b). The lifetime soil ingestion formula is based on data provided in the RAGS Supplemental Guidance (EPA, 1991b). Identifiers have been added to the basic exposure factor abbreviations to differentiate those factors that are used in multiple formulas. The following formulas are used in the ARES for RSA Sites F, G, R, S and T:

Table C-1. Exposure Scenarios with Applicable Sites and Exposure Pathways

Scenario*	Applicable Sites	FS	GW			SE		SO			SW	
		oral	der	inh	oral	der	oral	der	inh	oral	der	oral
Current Recreational 1	G					X	X				X	X
Current Recreational 2	G	♦										
Current Worker A	F							+	+	+		
Current Worker B	S/T							#	#	#		
Future Worker 1	F, R, S/T		✡	✡	✡			X	X	X		
Future Worker 2	G		✡	✡	✡	X	X	X	X	X	X	X

Note:
FS = fish.
GW = groundwater.
SE = sediment.
SO = soil.
SW = surface water.
der = dermal.
inh = inhalation.

Table C-1. Exposure Scenarios with Applicable Sites and Exposure Pathways (Conituned, Page 2 of 2)

X = This receptor/pathway is evaluated in ARES based on measured concentrations at the contamination area.
♦ = This receptor/pathway is evaluated in ARES based on modeled concentrations of organic and inorganic contaminants in trout.
+ = Current worker exposure to Site F soil contamination is limited to potential intermittent exposure to drainage ditch sediments by workers in the buildings adjacent to the site. However, due to the scope of work, current worker exposure to all soils at Site F must be evaluated.
# = Current worker exposure to Site S/T soil contamination is limited to potential intermittent exposure to soil materials by a maintenance worker cutting the grass at Site T, only. However, due to the scope of work, current worker exposure to all soils at Site S/T must be evaluated.
✡ = Worker inhalation and dermal exposure to chemicals in groundwater during showering is evaluated in ARES to estimate the risks associated with future use of site groundwater should it ever be used as a potable water source.

*Scenarios are unique for receptor, pathways, and exposure parameters. 1,2 after a scenario indicates that while the exposure parameters are the same for like pathways, different exposure pathways are applicable. Therefore, Future Worker 1 and Future Worker 2 will have different exposure pathways, but the exposure parameters for like pathways will be the same. A,B after a scenario indicates that while the exposure pathways may be the same, different exposure parameters are applicable. Therefore, although Current Worker A and Current Worker B have the same exposure pathways, the exposure parameters are different.

Source: ESE.

3.1 Fish, Oral Exposure

$$Intake\ (mg/kg/day) = \frac{CFf * IRf * FIf * EFf * ED}{BW * AT}$$

Where:

CFf = chemical concentration in fish (mg/kg).
IRf = intake rate for fish (kg/day).
FIf = fraction of fish eaten from contaminated source (unitless).
EFf = exposure frequency for fish (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.2 Groundwater, Dermal Exposure (Theoretical Worker Exposure While Showering)

$$Intake\ (mg/kg/day) = \frac{CGW * FCw * SAgw * PC * ETgw * EFgw * ED}{BW * AT}$$

Where:

CGW = chemical concentration in groundwater (mg/L).
FCw = volumetric conversion factor for water (L/cm^3).
SAgw = skin surface area available for contact with groundwater while showering (cm^2).
PC = dermal permeability constant (cm/hour).
ETgw = exposure time for groundwater while showering (hours/day).
EFgw = exposure frequency for groundwater (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.3 Groundwater, Inhalation Exposure (Theoretical Worker Exposure to Vapors While Showering)

$$Intake\ (mg/kg/day) = \frac{CGW * FCas * IRas * ETas * EFas * ED}{BW * AT}$$

Where:

CGW = chemical concentration in groundwater (mg/L).

FCas = factor to convert groundwater concentration to shower air concentration (L/m^3).
IRas = intake rate for shower air (m^3/hour).
ETas = exposure time for shower air (hours/day).
EFas = exposure frequency for shower air (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

Site groundwater is not currently used as a water supply at the base. However, evaluation of future worker exposure to groundwater has been included as a conservative estimate of possible theoretical future exposure.

3.4 Groundwater, Oral Exposure

$$Intake\ (mg/kg/day) = \frac{CGW * IRgw * EFgw * ED}{BW * AT}$$

Where:

CGW = chemical concentration in groundwater (mg/L).
IRgw = intake rate for groundwater (L/day).
EFgw = exposure frequency for contaminated groundwater (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

Site groundwater is not currently used as a water supply on the base. However, evaluation of future worker exposure to groundwater has been included as a conservative estimate of possible theoretical future exposure.

3.5 Sediment, Dermal Exposure

$$Intake\ (mg/kg/day) = \frac{CSe * FCs * SAse * AF * ABS * EFse * ED}{BW * AT}$$

Where:

CSe = chemical concentration in sediment (mg/kg).
FCs = conversion factor for soil/sediment (kg/mg).
SAse = skin surface area available for sediment contact (cm^2/event).
AF = soil/sediment to skin adherence factor (mg/cm^2).
ABS = chemical-specific absorption factor (unitless).

EFse = exposure frequency for sediment (events/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.6 Sediment, Oral Exposure

For adult and child exposures:

$$Intake\ (mg/kg/day) = \frac{CSe * IRse * FCs * FIse * EFse * ED}{BW * AT}$$

Where:

CSe = chemical concentration in sediment (mg/kg).
IRse = sediment ingestion rate (mg/day).
FCs = conversion factor for soil/sediment (kg/mg).
FIse = fraction of sediment ingested from contaminated source (unitless).
EFse = exposure frequency for sediment (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = averaging time (days).

For lifetime exposure (derived from EPA, 1991b; Incidental Ingestion of Soil and Dust):

$$Intake\ (mg/kg/day) = Y_c * \sum_{i=1}^{2} \frac{IRse_i * ED_i}{BW_i}$$

Where:

Y_c = CSe * FCs * FIse * EFse / AT.
$IRse_i$ = sediment ingestion rate (mg/day; age-dependent; EPA, 1991b).
ED_i = exposure duration (years; age range for particular ingestion rate; EPA, 1991b).
BW_i = body weight (kg; age-dependent; EPA, 1989a).

i	IRse	ED	BW
1	200	6	15
2	100	24	70

3.7 Soil, Dermal Exposure

$$Intake\ (mg/kg/day) = \frac{CSo * FCs * SAso * AF * ABS * EFso * ED}{BW * AT}$$

Where:

CSo = chemical concentration in soil (mg/kg).
FCs = conversion factor for soil/sediment (kg/mg).
SAso = skin surface area available for soil contact (cm^2/event).
AF = soil/sediment to skin adherence factor (mg/cm^2).
ABS = chemical-specific absorption factor (unitless).
EFso = exposure frequency for soil (events/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.8 Soil, Inhalation Exposure (Suspended Particulates in Ambient Air)

$$Intake\ (mg/kg/day) = \frac{CSo * RPC * IRaa * FCs * EFaa * ED}{BW * AT}$$

Where:

CSo = chemical concentration in soil (mg/kg).
RPC = respirable particulate concentration in air (mg/m^3).
IRaa = intake rate for ambient air (m^3/day).
FCs = conversion factor for soil/sediment (kg/mg).
EFaa = exposure frequency for ambient air (events/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.9 Soil, Oral Exposure

$$Intake\ (mg/kg/day) = \frac{CSo * IRso * FCs * FIs * EFso * ED}{BW * AT}$$

Where:

CSo = chemical concentration in soil (mg/kg).
IRso = soil ingestion rate (mg/day).
FCs = conversion factor for soil/sediment (kg/mg).
FIs = fraction of soil/sediment ingested from contaminated source (unitless).

EFso = exposure frequency for soil (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = averaging time (days).

3.10 Surface Water, Dermal Exposure

$$Intake\ (mg/kg/day) = \frac{CSW * FCw * SAsw * PC * ETsw * EFsw * ED}{BW * AT}$$

Where:

CSW = chemical concentration in surface water (mg/L).
FCw = volumetric conversion factor for water (L/cm^3).
SAsw = skin surface area available for contact with surface water (cm^2).
PC = dermal permeability constant (cm/hour).
ETsw = exposure time for surface water (hours/day).
EFsw = exposure frequency for surface water (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

3.11 Surface Water, Oral Exposure

$$Intake\ (mg/kg/day) = \frac{CSW * IRsw * ETsw * EFsw * ED}{BW * AT}$$

Where:

CSW = chemical concentration in surface water (mg/L).
IRsw = intake rate for surface water (L/hour).
ETsw = exposure time for surface water (hours/day).
EFsw = exposure frequency for surface water (days/year).
ED = exposure duration (years).
BW = body weight (kg).
AT = period of time over which exposure is averaged (days).

4.0 Exposure Factors Used In the ARES for RSA BRA 1

4.1 ABS

inorganic chemicals	0.001	EPA Region IV default value	EPA, 1992b
organic chemicals	0.01	EPA Region IV default value	EPA, 1992b

---

4.2 AF

1.00 mg/cm^2 — Median of the absorption values (0.5 to 1.5 mg/cm^2) produced by Lepow et al. (1975) and Roels et al. (1980) and reported in EPA, 1984. Also, this value is the worst-case value reported by EPA (1992b).

Although EPA (1989) recommends using the value for potting soil of 1.45 mg/cm^2 (upper-bound value) when site-specific information is not available, the median adherence value was chosen because site soils are predominantly sandy in nature and sand has a lower skin adherence than potting soil.

4.3 AT

carcinogenic effects	70 years * 365 days/year	EPA, 1989b
noncarcinogenic effects	ED (years) * 365 days/year	EPA, 1989b

---

4.4 BW

Adult (Recreational, Worker)

70 kg — average (male and female) of 50th percentile values for age = 18 to 75 years. For lifetime recreational/residential sediment and soil ingestion, see Sections 3.6 and 3.9 of this documentation, respectively. — EPA, 1985

Child (Recreational)

15 kg — average (male and female) of 50th percentile values for age = 1 to 6 years. — EPA, 1985

Lifetime (Recreational)

59 kg — Assumes a child body weight of 15 kg for 6 years and an adult body weight of 70 kg for 24 years.

$$\frac{(15\ kg * 6\ years) + (70\ kg * 24\ years)}{30\ years} = 59\ kg$$

4.5 CFf

The concentrations of chemicals in fish caught in the stream adjacent to Site G are modeled values based on chemical-specific parameters (bioconcentration factor) and site-specific parameters (fish type).

4.6 CGW / CSe / CSo / CSW

The upper 95 percent confidence limit (UCL_{95}) of the mean chemical concentration was used to represent the reasonable maximum exposure (RME) concentration in each medium (i.e., sediment, soil, and surface water). If the UCL_{95} exceeded the maximum detected chemical concentration, the maximum concentration was used to represent the RME.

4.7 ED

Adult (Recreational) / Lifetime (Recreational)

30 years	national 90th percentile time at one residence	EPA, 1989b

For lifetime ingestion of sediment and soil, see Sections 3.2 and 3.5 of this documentation, respectively.

Adult (Worker)

25 years	national 95th percentile time at one workplace	EPA, 1991b

---

Child (Recreational)

6 years	assumes exposure for children age = 1 to 6 years, inclusive, in rural/residential areas

---

4.8 EFaa

Adult (Worker; Current - A)

250 days/year	assumes that a person working in the buildings adjacent to site F may be exposed to soil and indoor dust

-- --

Adult (Worker; Current - B)

16 days/year	Assumes that maintenance personnel may cut grass at Site T 2 times per month during the growing season (assumed to be March through October).

Other than incidental dermal, inhalation, and oral exposure to soil by maintenance personnel cutting grass or performing other minor duties in the potentially contaminated area, no other worker exposure to soil is expected to occur at Site T.

-- --

Adult (Worker; Future)

250 days/year	number of days spent at work	EPA, 1991b

4.9 EFas / EFgw

250 days/year	number of days spent at work	EPA, 1991b

Site groundwater is not currently used as a water supply on the base. However, evaluation of future worker exposure to groundwater has been included as a conservative estimate of possible theoretical future exposure.

4.10 EFf

350 days/year	typical residential fish intake is averaged over a year	EPA, 1991b

---

4.11 EFse / EFsw

Adult (Recreational) / Child (Recreational) / Lifetime (Recreational)

26 days/year — assumes a person may fish in McDonald Creek 1 day/week for 6 months

---

Adult (Worker; Future)

80 days/year — assumes that a worker may take a break near McDonald Creek adjacent to Site G approximately once every 3 working days

---

4.12 EFso

Adult (Worker; Current - A)

250 days/year — number of days spent at work — EPA, 1991b

Assumes that a person working in the buildings adjacent to site F may incidentally ingest soil and indoor dust.

Adult (Worker; Current - B)

16 days/year — Assumes that maintenance personnel may cut grass at Site T 2 times per month during the growing season (assumed to be March through October)

Other than incidental dermal, inhalation, and oral exposure to soil by maintenance personnel cutting grass or performing other minor duties in the potentially contaminated area, no other worker exposure to soil is expected to occur at Site T.

Adult (Worker; Future)

250 days/year — number of days spent at work — EPA, 1991b

4.13 ETas / ETgw

0.2 hour/day — 90th percentile time spent in shower — EPA, 1989b

---

4.14 ETsw

Adult (Recreational) / Child (Recreational) / Lifetime (Recreational)

2 hours/day — assumed value for persons fishing in McDonald Creek

---

Adult (Worker)

0.5 hour/day — assumed value for workers taking breaks near McDonald Creek adjacent to Site G

4.15 FCas

The concentration of VOCs in shower air is a modeled value using assumptions presented by McKone (1987) and based on the following factors and formulas. McKone values were used because they are both realistic and conservative.

18 L/m^3 — Mean ratio of chemical concentration in shower air (mg/m^3) to chemical concentration in water (mg/L) for VOCs commonly detected in groundwater.

4.16 FCs

1×10^{-6} kg/mg

4.17 FCw

0.001 L/cm^3

4.18 FIf

0.05 — Assumes that 5% of all fish consumed is caught from McDonald Creek adjacent to Site G.

4.19 FIse

0.5 — Assumes 50% of sediment exposure is from worker contact with McDonald Creek.

4.20 FIso

1.0	Assumes 100% of soil exposure is from contaminated source.	

4.21 IRaa

Adult (Worker; Current - A / Worker; Future)

20 m^3/day	reasonable upper-bound occupational inhalation rate for an 8-hour workday	EPA, 1991b

Adult (Worker; Current - B)

10 m^3/day	based on a reasonable upper-bound occupational inhalation rate for an 8-hour workday [20 m^3/day (EPA, 1991b)] and assumes that maintenance personnel may cut grass at Site T 4 hours/day.	

4.22 IRas

0.6 m^3/hour	Shower inhalation rate (all age groups)	EPA, 1989b

---

4.23 IRf

Adult (Recreational)

0.0147 kg/day	50th percentile fish ingestion rate for an adult	EPA, 1990

Child (Recreational)

0.0062 kg/day	50th percentile fish ingestion rate for a child	EPA, 1990

Lifetime (Recreational)

0.013 kg/day	Assumes a child fish ingestion rate of 0.0062 kg/day for 6 years and an adult fish ingestion rate of 0.0147 kg/day for 24 years.

$$\frac{(0.0062\ kg/day * 6\ years) + (0.0147\ kg/day * 24\ years)}{30\ years} = \mathbf{0.013\ kg/day}$$

4.24 IRgw

1.0 L/day	reasonable occupational ingestion rate	EPA, 1991b

Site groundwater is not currently used as a water supply on the base. However, evaluation of future worker exposure to groundwater has been included as a conservative estimate of possible theoretical future exposure.

4.25 IRse

Adult (Recreational)

100 mg/day	conservative value assumes adult residential IRso	EPA, 1991b

Adult (Worker; Future)

50 mg/day	conservative value assumes adult worker IRso	EPA, 1991b

Child (Recreational)

200 mg/day	conservative value assumes child residential IRso	EPA, 1991b

Lifetime (Recreational)

Sediment ingestion rates are age-dependent. For an explanation of these intake rates, see Section 3.6 of this documentation.

4.26 IRso

Adult (Worker; Current - A / Worker; Future)

50 mg/day	typical adult workplace ingestion rate	EPA, 1991b

According to EPA (1991b), "The amount of soil and dust adults contact in their houses may be similar to the amount an office or indoor maintenance worker would be expected to contact."

Adult (Worker; Current - B)

480 mg/day	soil ingestion rate during yardwork estimated by Hawley (1985)	EPA, 1991b

---

4.27 IRsw

Adult (Recreational) / Child (Recreational) / Lifetime (Recreational)

0.05 L/hour	national average for swimming (EPA, 1989a) is used as a conservative estimate for persons fishing or wading in McDonald Creek

---

Adult (Worker)

0.005 L/hour	Assumed value for incidental exposure of workers taking breaks near McDonald Creek adjacent to Site G

Although the potential may exist for future workers at Site G (should it be developed) to occasionally contact surface water during breaks, ingestion of surface water resulting from this incidental contact is highly unlikely and is included as a conservative estimate of potential exposure. IRsw for the future worker scenario is less than the current recreational scenario since persons recreating in the creek will intentionally contact surface water.

4.28 PC

PCs for inorganic chemicals are either experimentally measured values provided in Table 5-3 of EPA's Dermal Exposure Assessment document (1992a) or the default value for water. PCs for organic chemicals are either predicted K_p values provided in Table 5-7 (EPA, 1992a) or are calculated using Equation 5.11 (reproduced below from EPA, 1992a). Where a calculated value is presented, the source for log K_{ow} is provided.

$$K_p = 10^{-2.72 + (0.71 \log K_{ow}) - (0.0061 MW)}$$

Where:

K_{ow} = octanol/water partition coefficient, and
MW = molecular weight.

Arsenic	1.0×10^{-3}	default value for water
Barium	1.0×10^{-3}	default value for water
Cadmium	1.0×10^{-3}	Table 5-3 [cadmium chloride]
Chromium	2.0×10^{-3}	Table 5-3 [sodium chromate]
Lead	4.0×10^{-6}	Table 5-3 [lead acetate]
Mercury	1.0×10^{-3}	Table 5-3 [mercuric chloride]
Selenium	1.0×10^{-3}	default value for water
Silver	6.0×10^{-4}	Table 5-3 [silver nitrate]
Acenaphthene	1.5×10^{-1}	calculated (EPA, 1986)
Anthracene	2.3×10^{-1}	calculated (EPA, 1986)
Benz(a)anthracene	8.1×10^{-1}	Table 5-7
Benzo(a)pyrene	1.2×10^{0}	Table 5-7
Benzo(b)fluoranthene	1.2×10^{0}	Table 5-7
Benzo(ghi)perylene	1.7×10^{0}	calculated (EPA, 1986)
Chrysene	8.1×10^{-1}	Table 5-7
Dibenz(ah)anthracene	2.7×10^{0}	Table 5-7
Fluoranthene	3.6×10^{-1}	Table 5-7
Fluorene	3.6×10^{-1}	calculated (EPA, 1986)
Indeno(1,2,3-cd)pyrene	1.9×10^{0}	Table 5-7
Naphthalene	6.9×10^{-2}	Table 5-7
Phenanthrene	2.7×10^{-1}	Table 5-7
Pyrene	3.3×10^{-1}	calculated (EPA, 1986)
DDE, p,p'-	2.4×10^{-1}	Table 5-7
DDT, p,p'-	4.3×10^{-1}	Table 5-7
Bis(2-chloroethyl) ether	2.1×10^{-3}	Table 5-7
Bis(2-ethylhexyl) phthalate	2.3×10^{-2}	calculated (ATSDR, 1989)
Butylbenzyl phthalate	5.8×10^{-2}	calculated (HSDB, 1995)
Acetone	5.7×10^{-4}	calculated (HSDB, 1995)
Benzene	2.1×10^{-2}	Table 5-7
Carbon tetrachloride	2.2×10^{-2}	Table 5-7
Chlorobenzene	4.1×10^{-2}	Table 5-7
Chloroform	8.9×10^{-3}	Table 5-7
Dichloroethane, 1,1-	8.9×10^{-3}	Table 5-7
Dichloroethane, 1,2-	5.3×10^{-3}	Table 5-7
Dichloroethenes, 1,2-	1.0×10^{-1}	Table 5-7
Ethylbenzene	7.4×10^{-2}	Table 5-7
Methylene chloride	4.5×10^{-3}	Table 5-7
Tetrachloroethene	4.8×10^{-2}	Table 5-7
Toluene	4.5×10^{-2}	Table 5-7
Trichloroethane, 1,1,1-	1.7×10^{-2}	Table 5-7
Trichloroethene	1.6×10^{-2}	Table 5-7
Vinyl chloride	7.3×10^{-3}	Table 5-7
Xylenes, total	8.9×10^{-2}	calculated (EPA, 1986)

4.29 RPC

0.027 mg/m^3	Average 24-hour average PM_{10} concentration detected at the Childersburg, AL air sampling station for the period 1/90 through 6/91.	Steward, 1991

PM_{10} values are used to represent RPC since particles greater than 10 µm typically impact the walls of the repiratory system prior to reaching the alveoli, while particles with a diameter less than 10 µm have a significant chance of reaching the alveolar surfaces.

4.30 SAgw

All surface area values are 50th percentile values from EPA, 1985. 50th percentile values are used because surface area is related to body weight, and average body weights over the ED were used in the exposure calculations. Values based on average adult (male and female) body part surface areas (m^2) multiplied by a conversion factor of 10,000 cm^2/m^2.

total body = 18,150 cm^2 Assumes exposure while showering.

4.31 SAse / SAsw

All surface area values are 50th percentile values from EPA, 1985. 50th percentile values are used because surface area is related to body weight, and average body weights over the ED were used in the exposure calculations. Values based on average adult (male and female) body part surface areas (m^2) multiplied by a conversion factor of 10,000 cm^2/m^2. Note that SAse and SAsw do not represent the surface area of the entire body but include those body parts that may typically be in contact with sediment and surface water.

Adult (Recreational)

hands	904.	
arms	2,605.	Assumes exposure during wading or fishing.
feet	1,225.	
legs	5,930.	
	10,664. cm^2	

Adult (Worker)

904 cm^2 — It is assumed that workers at Site G may take breaks near the stream adjacent to the site, allowing for incidental contact of the hands

Although the potential may exist for future workers at Site G (should it be developed) to occasionally contact surface water and sediment during breaks, exposure to these media is highly unlikely and is included as a conservative estimate of potential exposure. Surface areas for the future worker scenario are less than the current recreational scenario since persons recreating in the creek will intentionally contact surface water and sediment.

Child (Recreational)

hands	391.	
arms	915.	Assumes exposure during wading or fishing.
feet	479.	
legs	1,735.	
	3,520. cm^2	

Lifetime (Recreational)

9,235 cm^2 — Assumes a child surface area of 3,520 cm^2 for 6 years and an adult surface area of 10,664 cm^2 for 24 years.

$$\frac{(10{,}664\ cm^2 * 24\ years) + (3{,}520\ cm^2 * 6\ years)}{30\ years} = 9{,}235\ cm^2$$

4.32 SAso

All surface area values are 50th percentile values from EPA, 1985. 50th percentile values are used because surface area is related to body weight, and average body weights over the ED were used in the exposure calculations. Values based on average adult (male and female) body part surface areas (m^2) multiplied by a conversion factor of 10,000 cm^2/m^2. It is assumed that workers at RSA will wear long pants but may have exposed arms.

hands	904.
forearms	1,173.
½ head	602.
	2,679. cm^2

5.0 References Used in Developing ARES for RSA BRA 1

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Di(2-Ethylhexyl)phthalate. Prepared by Life Systems Inc. Published by Oak Ridge National Laboratory. U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/15. NTIS No. PB89-194484.

Hawley, J.D. 1985. Assessment of health risks from exposure to contaminated soil. Risk Analysis 5(4):289-302.

Hazardous Substances Data Bank (HSDB). 1995. National Library of Medicine/Toxicology Data Network (NLM/TOXNET). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Lepow, M.L., Bruckman, L., Rubino, R.A., Markowitz, S., Gillette, M., and Kapish, J. 1975. Investigations into Sources of Lead in the Environment of Urban Children. Environ. Res. 10:415-426.

McKone, T.E. 1987. Human Exposure to Volatile Organic Compounds in Household Tap Water: The Indoor Inhalation Pathway. Envir. Sci. and Tech. 21(12):1194-1201.

Roels, H.A., Buchet, J.P., and Lauwerys, R.R. 1980. Exposure to Lead by the Oral and Pulmonary Routes of Children Living in the Vicinity of a Primary Lead Smelter. Environ. Res. 22:81-94.

Steward, A. Aug. 27, 1991. Personal Communication, Fax to Vijaya Mylavarapu, Environmental Science & Engineering, Inc., re: Air Particulate Concentrations in the Vicinity of AAAP. Alabama Department of Environmental Management (ADEM). Montgomery, AL.

U.S. Environmental Protection Agency (EPA). 1984. Risk Analysis of TCDD-Contaminated Soil. Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1985. Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments. Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC. EPA/600/8-85/010.

U.S. Environmental Protection Agency (EPA). 1986. Superfund Public Health Evaluation Manual (SPHEM). Office of Emergency and Remedial Response and Office of Solid Waste and Emergency Response, Washington, DC. EPA 540/1-86/060.

U.S. Environmental Protection Agency (EPA). 1989a. Exposure Factors Handbook. Final Report. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/043.

U.S. Environmental Protection Agency (EPA). 1989b. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Part A. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/002.

U.S. Environmental Protection Agency (EPA). 1990. Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, and Office of Research and Development, Cincinnati, OH. EPA/600/6-90/003. NTIS No. PB90-187055.

U.S. Environmental Protection Agency (EPA). 1991a. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Part B (Development of Risk-Based Preliminary Remediation Goals). Office of Emergency and Remedial Response, Washington, DC. OERR 9285.7-01B.

U.S. Environmental Protection Agency (EPA). 1991b. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Supplemental Guidance (Standard Default Exposure Factors). Interim Final. Office of Emergency and Remedial Response, Washington, DC. OSWER Directive 9285.6-03.

U.S. Environmental Protection Agency (EPA). 1992a. Dermal Exposure Assessment: Principles and Applications. Interim Report. Prepared by Versar, Inc. Office of Research and Development, Washington, DC. EPA 600/8-91/011B. NTIS No. PB92-205665.

U.S. Environmental Protection Agency (EPA). 1992b. New Interim Region IV Guidance. Bureau of Waste Cleanup, Atlanta, GA. March 20, 1992.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. NTIS No. PB94-921199.

5.0 What References Were Used in Developing ARES for RSA BRA 1?

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Di(2-Ethylhexyl)phthalate. Prepared by Life Systems Inc. Published by Oak Ridge National Laboratory. U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/15. NTIS No. PB89-194484.

Hawley, J.D. 1985. Assessment of health risks from exposure to contaminated soil. Risk Analysis 5(4):289-302.

Hazardous Substances Data Bank (HSDB). 1995. National Library of Medicine/Toxicology Data Network (NLM/TOXNET). Micromedex TOMES PLUS® System CD/ROM, Version 24, Expires 4/30/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Lepow, M.L., Bruckman, L., Rubino, R.A., Markowitz, S., Gillette, M., and Kapish, J. 1975. Investigations into Sources of Lead in the Environment of Urban Children. Environ. Res. 10:415-426.

McKone, T.E. 1987. Human Exposure to Volatile Organic Compounds in Household Tap Water: The Indoor Inhalation Pathway. Envir. Sci. and Tech. 21(12):1194-1201.

Roels, H.A., Buchet, J.P., and Lauwerys, R.R. 1980. Exposure to Lead by the Oral and Pulmonary Routes of Children Living in the Vicinity of a Primary Lead Smelter. Environ. Res. 22:81-94.

Steward, A. Aug. 27, 1991. Personal Communication, Fax to Vijaya Mylavarapu, Environmental Science & Engineering, Inc., re: Air Particulate Concentrations in the Vicinity of AAAP. Alabama Department of Environmental Management (ADEM). Montgomery, AL.

U.S. Environmental Protection Agency (EPA). 1984. Risk Analysis of TCDD-Contaminated Soil. Office of Health and Environmental Assessment, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1985. Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments. Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC. EPA/600/8-85/010.

U.S. Environmental Protection Agency (EPA). 1986. Superfund Public Health Evaluation Manual (SPHEM). Office of Emergency and Remedial Response and Office of Solid Waste and Emergency Response, Washington, DC. EPA 540/1-86/060.

U.S. Environmental Protection Agency (EPA). 1989a. Exposure Factors Handbook. Final Report. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/043.

U.S. Environmental Protection Agency (EPA). 1989b. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Part A. Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-89/002.

U.S. Environmental Protection Agency (EPA). 1990. Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, and Office of Research and Development, Cincinnati, OH. EPA/600/6-90/003. NTIS No. PB90-187055.

U.S. Environmental Protection Agency (EPA). 1991a. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Part B (Development of Risk-Based Preliminary Remediation Goals). Office of Emergency and Remedial Response, Washington, DC. OERR 9285.7-01B.

U.S. Environmental Protection Agency (EPA). 1991b. Risk Assessment Guidance for Superfund (RAGS). Volume 1: Human Health Evaluation Manual, Supplemental Guidance (Standard Default Exposure Factors). Interim Final. Office of Emergency and Remedial Response, Washington, DC. OSWER Directive 9285.6-03.

U.S. Environmental Protection Agency (EPA). 1992a. Dermal Exposure Assessment: Principles and Applications. Interim Report. Prepared by Versar, Inc. Office of Research and Development, Washington, DC. EPA 600/8-91/011B. NTIS No. PB92-205665.

U.S. Environmental Protection Agency (EPA). 1992b. New Interim Region IV Guidance. Bureau of Waste Cleanup, Atlanta, GA. March 20, 1992.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. NTIS No. PB94-921199.

APPENDIX D

DAILY INTAKES

Redstone BRA1 ARES:
Carcinogenic Intakes
Current Recreation 1 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
G	LIFREC	CURR	SE	DERM	Arsenic	2.03E-11	A	A	3.55E-11
				DERM	Methylene chloride	1.01E-09	B2	B2	7.61E-12
				ORAL	Arsenic	2.47E-10	A	A	4.32E-10
				ORAL	Methylene chloride	1.24E-09	B2	B2	9.26E-12
G	LIFREC	CURR	SW	DERM	Trichloroethene	2.43E-06	B2	B2	2.67E-08
				DERM	Methylene chloride	2.45E-07	B2	B2	1.84E-09
				DERM	Dichloroethane, 1,1-	2.47E-07	C	C	
				ORAL	Trichloroethene	8.23E-07	B2	B2	9.05E-09
				ORAL	Methylene chloride	2.95E-07	B2	B2	2.21E-09
				ORAL	Dichloroethane, 1,1-	1.50E-07	C	C	

Redstone BRA1 ARES:
Carcinogenic Intakes
Current Recreation 2 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
G	LIFREC	CURR	FS	ORAL	Trichloroethene	7.70E-07	B2	B2	8.47E-09
				ORAL	Methylene chloride	1.31E-07	B2	B2	9.85E-10
				ORAL	Dichloroethane, 1,1-	1.72E-08	C	C	

Redstone BRA1 ARES:
Carcinogenic Intakes
Current Worker A Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
F	ADWRK	CURR	SO	DERM	Benzo(a)pyrene	1.27E-07	B2	B2	9.28E-07
				DERM	Dibenz(ah)anthracene	8.00E-08	B2	B2	5.84E-07
				DERM	Benzo(b)fluoranthene	1.85E-07	B2	B2	1.35E-07
				DERM	Benz(a)anthracene	1.82E-07	B2	B2	1.33E-07
				DERM	Indeno(1,2,3-cd)pyrene	1.03E-07	B2	B2	7.55E-08
				DERM	Bis(2-ethylhexyl) phthalate	2.92E-07	B2	B2	4.09E-09
				DERM	Arsenic	6.61E-10	A	A	1.16E-09
				DERM	Chrysene	1.52E-07	B2	B2	1.11E-09
				DERM	Methylene chloride	1.60E-09	B2	B2	1.20E-11
				INH	Benzo(a)pyrene	2.56E-09	B2	B2	1.56E-08
				INH	Dibenz(ah)anthracene	1.61E-09	B2	B2	9.84E-09
				INH	Benzo(b)fluoranthene	3.73E-09	B2	B2	2.28E-09
				INH	Benz(a)anthracene	3.67E-09	B2	B2	2.24E-09
				INH	Arsenic	1.33E-10	A	A	2.00E-09
				INH	Indeno(1,2,3-cd)pyrene	2.08E-09	B2	B2	1.27E-09
				INH	Chrysene	3.07E-09	B2	B2	1.87E-11
				INH	Cadmium (solid matrix)	8.68E-13		B1	5.47E-12
				INH	Methylene chloride	3.22E-11	B2	B2	5.15E-14
				INH	Bis(2-ethylhexyl) phthalate	5.89E-09	B2	B2	
				ORAL	Benzo(a)pyrene	2.37E-07	B2	B2	1.73E-06
				ORAL	Dibenz(ah)anthracene	1.49E-07	B2	B2	1.09E-06
				ORAL	Benzo(b)fluoranthene	3.46E-07	B2	B2	2.52E-07
				ORAL	Benz(a)anthracene	3.40E-07	B2	B2	2.48E-07
				ORAL	Indeno(1,2,3-cd)pyrene	1.93E-07	B2	B2	1.41E-07
				ORAL	Arsenic	1.23E-08	A	A	2.16E-08
				ORAL	Bis(2-ethylhexyl) phthalate	5.46E-07	B2	B2	7.64E-09
				ORAL	Chrysene	2.84E-07	B2	B2	2.07E-09
				ORAL	Methylene chloride	2.98E-09	B2	B2	2.24E-11

Redstone BRA1 ARES:
Carcinogenic Intakes
Current Worker B Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
ST	ADWRK	CURR	SO	DERM	DDT, p,p'-	1.65E-10	B2	B2	5.62E-11
				DERM	Bis(2-ethylhexyl) phthalate	1.18E-09	B2	B2	1.66E-11
				DERM	DDE, p,p'-	2.59E-11	B2	B2	8.82E-12
				DERM	Methylene chloride	1.12E-10	B2	B2	8.43E-13
				INH	DDT, p,p'-	1.67E-12	B2	B2	5.67E-13
				INH	DDE, p,p'-	2.61E-13	B2	B2	8.89E-14
				INH	Methylene chloride	1.13E-12	B2	B2	1.81E-15
				INH	Bis(2-ethylhexyl) phthalate	1.19E-11	B2	B2	
				ORAL	DDT, p,p'-	2.96E-09	B2	B2	1.01E-09
				ORAL	Bis(2-ethylhexyl) phthalate	2.12E-08	B2	B2	2.97E-10
				ORAL	DDE, p,p'-	4.65E-10	B2	B2	1.58E-10
				ORAL	Methylene chloride	2.02E-09	B2	B2	1.51E-11

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
F	ADWRK	FUT	GW	DERM	Arsenic	5.10E-07	A	A	8.92E-07
				DERM	Carbon tetrachloride	2.23E-06	B2	B2	2.90E-07
				DERM	Trichloroethene	6.70E-07	B2	B2	7.37E-09
				DERM	Chloroform	6.77E-07	B2	B2	4.13E-09
				DERM	Lead	6.49E-10	B2	B2	
				INH	Chloroform	4.53E-05	B2	B2	3.67E-06
				INH	Carbon tetrachloride	6.04E-05	B2	B2	3.17E-06
				INH	Trichloroethene	2.49E-05	B2	B2	1.49E-07
				ORAL	Arsenic	1.40E-04	A	A	2.46E-04
				ORAL	Carbon tetrachloride	2.80E-05	B2	B2	3.63E-06
				ORAL	Chloroform	2.10E-05	B2	B2	1.28E-07
				ORAL	Trichloroethene	1.15E-05	B2	B2	1.27E-07
				ORAL	Lead	4.47E-05	B2	B2	
F	ADWRK	FUT	SO	DERM	Benzo(a)pyrene	1.27E-07	B2	B2	9.28E-07
				DERM	Dibenz(ah)anthracene	8.00E-08	B2	B2	5.84E-07
				DERM	Benzo(b)fluoranthene	1.85E-07	B2	B2	1.35E-07
				DERM	Benz(a)anthracene	1.82E-07	B2	B2	1.33E-07
				DERM	Indeno(1,2,3-cd)pyrene	1.03E-07	B2	B2	7.55E-08
				DERM	Bis(2-ethylhexyl) phthalate	2.92E-07	B2	B2	4.09E-09
				DERM	Arsenic	6.61E-10	A	A	1.16E-09
				DERM	Chrysene	1.52E-07	B2	B2	1.11E-09
				DERM	Methylene chloride	1.60E-09	B2	B2	1.20E-11
				INH	Benzo(a)pyrene	2.56E-09	B2	B2	1.56E-08
				INH	Dibenz(ah)anthracene	1.61E-09	B2	B2	9.84E-09
				INH	Benzo(b)fluoranthene	3.73E-09	B2	B2	2.28E-09
				INH	Benz(a)anthracene	3.67E-09	B2	B2	2.24E-09
				INH	Arsenic	1.33E-10	A	A	2.00E-09
				INH	Indeno(1,2,3-cd)pyrene	2.08E-09	B2	B2	1.27E-09
				INH	Chrysene	3.07E-09	B2	B2	1.87E-11
				INH	Cadmium (solid matrix)	8.68E-13		B1	5.47E-12
				INH	Methylene chloride	3.22E-11	B2	B2	5.15E-14
				INH	Bis(2-ethylhexyl) phthalate	5.89E-09	B2	B2	
				ORAL	Benzo(a)pyrene	2.37E-07	B2	B2	1.73E-06
				ORAL	Dibenz(ah)anthracene	1.49E-07	B2	B2	1.09E-06
				ORAL	Benzo(b)fluoranthene	3.46E-07	B2	B2	2.52E-07
				ORAL	Benz(a)anthracene	3.40E-07	B2	B2	2.48E-07
				ORAL	Indeno(1,2,3-cd)pyrene	1.93E-07	B2	B2	1.41E-07
				ORAL	Arsenic	1.23E-08	A	A	2.16E-08
				ORAL	Bis(2-ethylhexyl) phthalate	5.46E-07	B2	B2	7.64E-09
				ORAL	Chrysene	2.84E-07	B2	B2	2.07E-09
				ORAL	Methylene chloride	2.98E-09	B2	B2	2.24E-11
R	ADWRK	FUT	GW	DERM	Bis(2-chloroethyl) ether	1.63E-07	B2	B2	1.80E-07
				DERM	Arsenic	7.66E-08	A	A	1.34E-07
				DERM	Bis(2-ethylhexyl) phthalate	1.79E-06	B2	B2	2.50E-08
				DERM	Chloroform	2.05E-06	B2	B2	1.25E-08
				DERM	Methylene chloride	1.55E-07	B2	B2	1.16E-09
				DERM	Lead	2.55E-09	B2	B2	
				INH	Chloroform	1.37E-04	B2	B2	1.11E-05
				INH	Methylene chloride	2.05E-05	B2	B2	3.29E-08
				ORAL	Arsenic	2.11E-05	A	A	3.69E-05
				ORAL	Bis(2-chloroethyl) ether	2.14E-05	B2	B2	2.36E-05
				ORAL	Chloroform	6.36E-05	B2	B2	3.88E-07
				ORAL	Bis(2-ethylhexyl) phthalate	2.14E-05	B2	B2	3.00E-07
				ORAL	Methylene chloride	9.51E-06	B2	B2	7.13E-08
				ORAL	Lead	1.75E-04	B2	B2	

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
R	ADWRK	FUT	SO	DERM	Benzo(a)pyrene	9.32E-08	B2	B2	6.81E-07
				DERM	Benzo(b)fluoranthene	1.71E-07	B2	B2	1.25E-07
				DERM	Benz(a)anthracene	1.29E-07	B2	B2	9.38E-08
				DERM	Indeno(1,2,3-cd)pyrene	6.27E-08	B2	B2	4.57E-08
				DERM	Bis(2-ethylhexyl) phthalate	7.30E-08	B2	B2	1.02E-09
				DERM	Chrysene	9.32E-08	B2	B2	6.81E-10
				DERM	Methylene chloride	8.36E-10	B2	B2	6.27E-12
				DERM	Butylbenzyl phthalate	7.30E-08	C	C	
				INH	Benzo(a)pyrene	1.88E-09	B2	B2	1.15E-08
				INH	Benzo(b)fluoranthene	3.45E-09	B2	B2	2.10E-09
				INH	Benz(a)anthracene	2.59E-09	B2	B2	1.58E-09
				INH	Indeno(1,2,3-cd)pyrene	1.26E-09	B2	B2	7.71E-10
				INH	Chrysene	1.88E-09	B2	B2	1.15E-11
				INH	Methylene chloride	1.68E-11	B2	B2	2.69E-14
				INH	Bis(2-ethylhexyl) phthalate	1.47E-09	B2	B2	
				INH	Butylbenzyl phthalate	1.47E-09	C	C	
				ORAL	Benzo(a)pyrene	1.74E-07	B2	B2	1.27E-06
				ORAL	Benzo(b)fluoranthene	3.19E-07	B2	B2	2.33E-07
				ORAL	Benz(a)anthracene	2.40E-07	B2	B2	1.75E-07
				ORAL	Indeno(1,2,3-cd)pyrene	1.17E-07	B2	B2	8.54E-08
				ORAL	Bis(2-ethylhexyl) phthalate	1.36E-07	B2	B2	1.91E-09
				ORAL	Chrysene	1.74E-07	B2	B2	1.27E-09
				ORAL	Methylene chloride	1.56E-09	B2	B2	1.17E-11
				ORAL	Butylbenzyl phthalate	1.36E-07	C	C	
ST	ADWRK	FUT	GW	DERM	Vinyl chloride	7.70E-07	A	A	1.46E-06
				DERM	Tetrachloroethene	1.55E-06	B2	B2	8.04E-08
				DERM	Benzene	1.54E-06	A	A	4.47E-08
				DERM	Dichloroethane, 1,2-	1.69E-07	B2	B2	1.54E-08
				DERM	Trichloroethene	1.15E-06	B2	B2	1.26E-08
				DERM	Chloroform	4.69E-07	B2	B2	2.86E-09
				DERM	Methylene chloride	1.43E-07	B2	B2	1.07E-09
				DERM	Lead	1.10E-09	B2	B2	
				INH	Vinyl chloride	6.28E-05	A	A	1.88E-05
				INH	Chloroform	3.13E-05	B2	B2	2.54E-06
				INH	Dichloroethane, 1,2-	1.89E-05	B2	B2	1.72E-06
				INH	Benzene	4.37E-05	A	A	1.27E-06
				INH	Trichloroethene	4.27E-05	B2	B2	2.56E-07
				INH	Tetrachloroethene	1.92E-05	B2	B2	3.83E-08
				INH	Methylene chloride	1.89E-05	B2	B2	3.02E-08
				ORAL	Vinyl chloride	2.91E-05	A	A	5.52E-05
				ORAL	Dichloroethane, 1,2-	8.77E-06	B2	B2	7.98E-07
				ORAL	Benzene	2.02E-05	A	A	5.87E-07
				ORAL	Tetrachloroethene	8.88E-06	B2	B2	4.62E-07
				ORAL	Trichloroethene	1.98E-05	B2	B2	2.18E-07
				ORAL	Chloroform	1.45E-05	B2	B2	8.85E-08
				ORAL	Methylene chloride	8.74E-06	B2	B2	6.55E-08
				ORAL	Lead	7.56E-05	B2	B2	
ST	ADWRK	FUT	SO	DERM	DDT, p,p'-	2.58E-09	B2	B2	8.79E-10
				DERM	Bis(2-ethylhexyl) phthalate	1.85E-08	B2	B2	2.59E-10
				DERM	DDE, p,p'-	4.05E-10	B2	B2	1.38E-10
				DERM	Methylene chloride	1.76E-09	B2	B2	1.32E-11
				INH	DDT, p,p'-	5.21E-11	B2	B2	1.77E-11
				INH	DDE, p,p'-	8.17E-12	B2	B2	2.78E-12
				INH	Methylene chloride	3.54E-11	B2	B2	5.67E-14
				INH	Bis(2-ethylhexyl) phthalate	3.72E-10	B2	B2	
				ORAL	DDT, p,p'-	4.82E-09	B2	B2	1.64E-09
				ORAL	Bis(2-ethylhexyl) phthalate	3.45E-08	B2	B2	4.83E-10
				ORAL	DDE, p,p'-	7.57E-10	B2	B2	2.57E-10
				ORAL	Methylene chloride	3.28E-09	B2	B2	2.46E-11

Redstone BRA1 ARES:
Carcinogenic Intakes
Future Worker 2 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	ORALWOE	INHWOE	RMERISK
G	ADWRK	FUT	GW	DERM	Arsenic	9.67E-08	A	A	1.69E-07
				DERM	Lead	7.07E-09	B2	B2	
				ORAL	Arsenic	2.66E-05	A	A	4.66E-05
				ORAL	Lead	4.87E-04	B2	B2	
G	ADWRK	FUT	SE	DERM	Arsenic	4.29E-12	A	A	7.51E-12
				DERM	Methylene chloride	2.15E-10	B2	B2	1.61E-12
				ORAL	Arsenic	1.19E-10	A	A	2.08E-10
				ORAL	Methylene chloride	5.94E-10	B2	B2	4.45E-12
G	ADWRK	FUT	SO	DERM	Methylene chloride	1.13E-09	B2	B2	8.44E-12
				INH	Methylene chloride	2.27E-11	B2	B2	3.63E-14
				ORAL	Methylene chloride	2.10E-09	B2	B2	1.58E-11
G	ADWRK	FUT	SW	DERM	Trichloroethene	1.29E-07	B2	B2	1.41E-09
				DERM	Methylene chloride	1.30E-08	B2	B2	9.72E-11
				DERM	Dichloroethane, 1,1-	1.30E-08	C	C	
				ORAL	Trichloroethene	4.45E-08	B2	B2	4.89E-10
				ORAL	Methylene chloride	1.59E-08	B2	B2	1.20E-10
				ORAL	Dichloroethane, 1,1-	8.11E-09	C	C	

Redstone BRA1 ARES:
Noncarcinogenic Intakes
Current Recreation 1 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
G	ADREC	CURR	SE	DERM	Arsenic	4.61E-11	1.54E-07
				DERM	Acetone	1.23E-08	1.23E-07
				DERM	Chromium, total	5.91E-10	1.18E-07
				DERM	Methylene chloride	2.30E-09	3.84E-08
				DERM	Barium	7.84E-10	1.12E-08
				ORAL	Arsenic	2.16E-10	7.20E-07
				ORAL	Chromium, total	2.77E-09	5.54E-07
				ORAL	Acetone	5.77E-09	5.77E-08
				ORAL	Barium	3.68E-09	5.25E-08
				ORAL	Methylene chloride	1.08E-09	1.80E-08
G	ADREC	CURR	SW	DERM	Trichloroethene	5.52E-06	9.20E-04
				DERM	Barium	1.75E-06	2.50E-05
				DERM	Methylene chloride	5.57E-07	9.28E-06
				DERM	Dichloroethane, 1,1-	5.60E-07	5.60E-06
				DERM	Xylenes, total	6.76E-06	3.38E-06
				DERM	Acetone	1.97E-07	1.97E-06
				ORAL	Trichloroethene	1.62E-06	2.70E-04
				ORAL	Barium	8.21E-06	1.17E-04
				ORAL	Acetone	1.62E-06	1.62E-05
				ORAL	Methylene chloride	5.80E-07	9.67E-06
				ORAL	Dichloroethane, 1,1-	2.95E-07	2.95E-06
				ORAL	Xylenes, total	3.56E-07	1.78E-07
G	CHREC	CURR	SE	DERM	Arsenic	7.10E-11	2.37E-07
				DERM	Acetone	1.90E-08	1.90E-07
				DERM	Chromium, total	9.10E-10	1.82E-07
				DERM	Methylene chloride	3.55E-09	5.92E-08
				DERM	Barium	1.21E-09	1.73E-08
				ORAL	Arsenic	2.02E-09	6.72E-06
				ORAL	Chromium, total	2.59E-08	5.17E-06
				ORAL	Acetone	5.39E-08	5.39E-07
				ORAL	Barium	3.43E-08	4.90E-07
				ORAL	Methylene chloride	1.01E-08	1.68E-07
G	CHREC	CURR	SW	DERM	Trichloroethene	8.51E-06	1.42E-03
				DERM	Barium	2.70E-06	3.85E-05
				DERM	Methylene chloride	8.58E-07	1.43E-05
				DERM	Dichloroethane, 1,1-	8.63E-07	8.63E-06
				DERM	Xylenes, total	1.04E-05	5.21E-06
				DERM	Acetone	3.03E-07	3.03E-06
				ORAL	Trichloroethene	7.55E-06	1.26E-03
				ORAL	Barium	3.83E-05	5.47E-04
				ORAL	Acetone	7.55E-06	7.55E-05
				ORAL	Methylene chloride	2.71E-06	4.51E-05
				ORAL	Dichloroethane, 1,1-	1.38E-06	1.38E-05
				ORAL	Xylenes, total	1.66E-06	8.31E-07

Redstone BRA1 ARES:
Noncarcinogenic Intakes
Current Recreation 2 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
G	ADREC	CURR	FS	ORAL	Trichloroethene	1.71E-06	2.85E-04
				ORAL	Barium	8.16E-06	1.17E-04
				ORAL	Methylene chloride	2.92E-07	4.87E-06
				ORAL	Acetone	1.11E-07	1.11E-06
				ORAL	Dichloroethane, 1,1-	3.83E-08	3.83E-07
				ORAL	Xylenes, total	7.75E-08	3.88E-08
G	CHREC	CURR	FS	ORAL	Trichloroethene	3.37E-06	5.61E-04
				ORAL	Barium	1.61E-05	2.29E-04
				ORAL	Methylene chloride	5.75E-07	9.58E-06
				ORAL	Acetone	2.18E-07	2.18E-06
				ORAL	Dichloroethane, 1,1-	7.53E-08	7.53E-07
				ORAL	Xylenes, total	1.53E-07	7.63E-08

Redstone BRA1 ARES:
Noncarcinogenic Intakes
Current Worker A Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
F	ADWRK	CURR	SO	DERM	Bis(2-ethylhexyl) phthalate	8.18E-07	4.09E-05
				DERM	Pyrene	7.84E-07	2.61E-05
				DERM	Fluoranthene	8.78E-07	2.19E-05
				DERM	Phenanthrene	5.77E-07	1.92E-05
				DERM	Benzo(b)fluoranthene	5.19E-07	1.73E-05
				DERM	Benz(a)anthracene	5.10E-07	1.70E-05
				DERM	Chrysene	4.26E-07	1.42E-05
				DERM	Benzo(a)pyrene	3.56E-07	1.19E-05
				DERM	Indeno(1,2,3-cd)pyrene	2.90E-07	9.65E-06
				DERM	Benzo(ghi)perylene	2.67E-07	8.89E-06
				DERM	Dibenz(ah)anthracene	2.24E-07	7.47E-06
				DERM	Fluorene	2.50E-07	6.25E-06
				DERM	Arsenic	1.85E-09	6.17E-06
				DERM	Naphthalene	2.05E-07	5.13E-06
				DERM	Acenaphthene	2.40E-07	3.99E-06
				DERM	Anthracene	2.35E-07	7.84E-07
				DERM	Mercury	9.36E-11	3.12E-07
				DERM	Acetone	2.10E-08	2.10E-07
				DERM	Methylene chloride	4.47E-09	7.45E-08
				DERM	Cadmium (solid matrix)	1.21E-11	1.21E-08
				INH	Mercury	1.89E-11	2.19E-07
				INH	Methylene chloride	9.01E-11	1.05E-10
				INH	Acetone	4.22E-10	
				INH	Acenaphthene	4.83E-09	
				INH	Anthracene	4.74E-09	
				INH	Arsenic	3.73E-10	
				INH	Bis(2-ethylhexyl) phthalate	1.65E-08	
				INH	Benz(a)anthracene	1.03E-08	
				INH	Benzo(a)pyrene	7.18E-09	
				INH	Benzo(b)fluoranthene	1.05E-08	
				INH	Benzo(ghi)perylene	5.38E-09	
				INH	Cadmium (solid matrix)	2.43E-12	
				INH	Chrysene	8.59E-09	
				INH	Dibenz(ah)anthracene	4.51E-09	
				INH	Fluoranthene	1.77E-08	
				INH	Fluorene	5.04E-09	
				INH	Indeno(1,2,3-cd)pyrene	5.84E-09	
				INH	Naphthalene	4.14E-09	
				INH	Phenanthrene	1.16E-08	
				INH	Pyrene	1.58E-08	
				ORAL	Arsenic	3.46E-08	1.15E-04
				ORAL	Bis(2-ethylhexyl) phthalate	1.53E-06	7.64E-05
				ORAL	Pyrene	1.46E-06	4.88E-05
				ORAL	Fluoranthene	1.64E-06	4.10E-05
				ORAL	Phenanthrene	1.08E-06	3.59E-05
				ORAL	Benzo(b)fluoranthene	9.68E-07	3.23E-05
				ORAL	Benz(a)anthracene	9.52E-07	3.17E-05
				ORAL	Chrysene	7.95E-07	2.65E-05
				ORAL	Benzo(a)pyrene	6.64E-07	2.21E-05
				ORAL	Indeno(1,2,3-cd)pyrene	5.40E-07	1.80E-05
				ORAL	Benzo(ghi)perylene	4.98E-07	1.66E-05
				ORAL	Dibenz(ah)anthracene	4.18E-07	1.39E-05
				ORAL	Fluorene	4.67E-07	1.17E-05
				ORAL	Naphthalene	3.83E-07	9.58E-06
				ORAL	Acenaphthene	4.47E-07	7.46E-06
				ORAL	Mercury	1.75E-09	5.82E-06
				ORAL	Anthracene	4.39E-07	1.46E-06
				ORAL	Acetone	3.91E-08	3.91E-07
				ORAL	Cadmium (solid matrix)	2.25E-10	2.25E-07
				ORAL	Methylene chloride	8.35E-09	1.39E-07

Redstone BRA1 ARES:
Noncarcinogenic Intakes
Current Worker B Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
ST	ADWRK	CURR	SO	DERM	DDT, p,p'-	4.63E-10	9.26E-07
				DERM	Bis(2-ethylhexyl) phthalate	3.31E-09	1.66E-07
				DERM	DDE, p,p'-	7.26E-11	1.45E-07
				DERM	Acetone	7.82E-09	7.82E-08
				DERM	Methylene chloride	3.15E-10	5.25E-09
				INH	Methylene chloride	3.17E-12	3.69E-12
				INH	Acetone	7.88E-11	
				INH	Bis(2-ethylhexyl) phthalate	3.34E-11	
				INH	DDE, p,p'-	7.32E-13	
				INH	DDT, p,p'-	4.67E-12	
				ORAL	DDT, p,p'-	8.30E-09	1.66E-05
				ORAL	Bis(2-ethylhexyl) phthalate	5.93E-08	2.97E-06
				ORAL	DDE, p,p'-	1.30E-09	2.60E-06
				ORAL	Acetone	1.40E-07	1.40E-06
				ORAL	Methylene chloride	5.64E-09	9.40E-08

Redstone Bral ARES:
Noncarcinogenic Intakes
Future Worker 1 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
F	ADWRK	FUT	GW	DERM	Mercury	1.16E-05	3.86E-02
				DERM	Carbon tetrachloride	6.25E-06	8.93E-03
				DERM	Arsenic	1.43E-06	4.76E-03
				DERM	Trichloroethene	1.88E-06	3.13E-04
				DERM	Cadmium (aqueous matrix)	1.31E-07	2.63E-04
				DERM	Chloroform	1.90E-06	1.90E-04
				DERM	Chromium, total	4.90E-07	9.80E-05
				DERM	Barium	3.24E-06	4.63E-05
				DERM	Acetone	6.50E-07	6.50E-06
				DERM	Lead	1.82E-09	
				INH	Carbon tetrachloride	1.69E-04	2.97E-01
				INH	Acetone	6.78E-04	
				INH	Chloroform	1.27E-04	
				INH	Trichloroethene	6.97E-05	
				ORAL	Mercury	3.19E-03	1.06E+01
				ORAL	Arsenic	3.93E-04	1.31E+00
				ORAL	Carbon tetrachloride	7.83E-05	1.12E-01
				ORAL	Cadmium (aqueous matrix)	3.62E-05	7.24E-02
				ORAL	Chromium, total	6.75E-05	1.35E-02
				ORAL	Barium	8.93E-04	1.28E-02
				ORAL	Chloroform	5.87E-05	5.87E-03
				ORAL	Trichloroethene	3.23E-05	5.38E-03
				ORAL	Acetone	3.14E-04	3.14E-03
				ORAL	Lead	1.25E-04	

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
F	ADWRK	FUT	SO	DERM	Bis(2-ethylhexyl) phthalate	8.18E-07	4.09E-05
				DERM	Pyrene	7.84E-07	2.61E-05
				DERM	Fluoranthene	8.78E-07	2.19E-05
				DERM	Phenanthrene	5.77E-07	1.92E-05
				DERM	Benzo(b)fluoranthene	5.19E-07	1.73E-05
				DERM	Benz(a)anthracene	5.10E-07	1.70E-05
				DERM	Chrysene	4.26E-07	1.42E-05
				DERM	Benzo(a)pyrene	3.56E-07	1.19E-05
				DERM	Indeno(1,2,3-cd)pyrene	2.90E-07	9.65E-06
				DERM	Benzo(ghi)perylene	2.67E-07	8.89E-06
				DERM	Dibenz(ah)anthracene	2.24E-07	7.47E-06
				DERM	Fluorene	2.50E-07	6.25E-06
				DERM	Arsenic	1.85E-09	6.17E-06
				DERM	Naphthalene	2.05E-07	5.13E-06
				DERM	Acenaphthene	2.40E-07	3.99E-06
				DERM	Anthracene	2.35E-07	7.84E-07
				DERM	Mercury	9.36E-11	3.12E-07
				DERM	Acetone	2.10E-08	2.10E-07
				DERM	Methylene chloride	4.47E-09	7.45E-08
				DERM	Cadmium (solid matrix)	1.21E-11	1.21E-08
				INH	Mercury	1.89E-11	2.19E-07
				INH	Methylene chloride	9.01E-11	1.05E-10
				INH	Acetone	4.22E-10	
				INH	Acenaphthene	4.83E-09	
				INH	Anthracene	4.74E-09	
				INH	Arsenic	3.73E-10	
				INH	Bis(2-ethylhexyl) phthalate	1.65E-08	
				INH	Benz(a)anthracene	1.03E-08	
				INH	Benzo(a)pyrene	7.18E-09	
				INH	Benzo(b)fluoranthene	1.05E-08	
				INH	Benzo(ghi)perylene	5.38E-09	
				INH	Cadmium (solid matrix)	2.43E-12	
				INH	Chrysene	8.59E-09	
				INH	Dibenz(ah)anthracene	4.51E-09	
				INH	Fluoranthene	1.77E-08	
				INH	Fluorene	5.04E-09	
				INH	Indeno(1,2,3-cd)pyrene	5.84E-09	
				INH	Naphthalene	4.14E-09	
				INH	Phenanthrene	1.16E-08	
				INH	Pyrene	1.58E-08	
				ORAL	Arsenic	3.46E-08	1.15E-04
				ORAL	Bis(2-ethylhexyl) phthalate	1.53E-06	7.64E-05
				ORAL	Pyrene	1.46E-06	4.88E-05
				ORAL	Fluoranthene	1.64E-06	4.10E-05
				ORAL	Phenanthrene	1.08E-06	3.59E-05
				ORAL	Benzo(b)fluoranthene	9.68E-07	3.23E-05
				ORAL	Benz(a)anthracene	9.52E-07	3.17E-05
				ORAL	Chrysene	7.95E-07	2.65E-05
				ORAL	Benzo(a)pyrene	6.64E-07	2.21E-05
				ORAL	Indeno(1,2,3-cd)pyrene	5.40E-07	1.80E-05
				ORAL	Benzo(ghi)perylene	4.98E-07	1.66E-05
				ORAL	Dibenz(ah)anthracene	4.18E-07	1.39E-05
				ORAL	Fluorene	4.67E-07	1.17E-05
				ORAL	Naphthalene	3.83E-07	9.58E-06
				ORAL	Acenaphthene	4.47E-07	7.46E-06
				ORAL	Mercury	1.75E-09	5.82E-06
				ORAL	Anthracene	4.39E-07	1.46E-06
				ORAL	Acetone	3.91E-08	3.91E-07
				ORAL	Cadmium (solid matrix)	2.25E-10	2.25E-07
				ORAL	Methylene chloride	8.35E-09	1.39E-07

Redstone Bra1 ARES:
Noncarcinogenic Intakes
Future Worker 1 Exposure

12:13 Wednesday, June 21, 1995

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
R	ADWRK	FUT	GW	DERM	Chromium, total	2.13E-05	4.26E-03
				DERM	Chlorobenzene	2.07E-05	1.03E-03
				DERM	Cadmium (aqueous matrix)	4.41E-07	8.81E-04
				DERM	Arsenic	2.14E-07	7.15E-04
				DERM	Bis(2-chloroethyl) ether	4.57E-07	6.09E-04
				DERM	Chloroform	5.75E-06	5.75E-04
				DERM	Barium	2.48E-05	3.54E-04
				DERM	Bis(2-ethylhexyl) phthalate	5.01E-06	2.50E-04
				DERM	Methylene chloride	4.35E-07	7.25E-06
				DERM	Acetone	4.40E-07	4.40E-06
				DERM	Lead	7.13E-09	
				INH	Chlorobenzene	3.00E-04	5.26E-02
				INH	Methylene chloride	5.75E-05	6.69E-05
				INH	Acetone	4.60E-04	
				INH	Chloroform	3.84E-04	
				ORAL	Chromium, total	2.94E-03	5.87E-01
				ORAL	Cadmium (aqueous matrix)	1.21E-04	2.43E-01
				ORAL	Arsenic	5.91E-05	1.97E-01
				ORAL	Barium	6.83E-03	9.76E-02
				ORAL	Bis(2-chloroethyl) ether	6.00E-05	7.99E-02
				ORAL	Chloroform	1.78E-04	1.78E-02
				ORAL	Chlorobenzene	1.39E-04	6.94E-03
				ORAL	Bis(2-ethylhexyl) phthalate	6.00E-05	3.00E-03
				ORAL	Acetone	2.13E-04	2.13E-03
				ORAL	Methylene chloride	2.66E-05	4.44E-04
				ORAL	Lead	4.91E-04	

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
R	ADWRK	FUT	SO	DERM	Pyrene	5.58E-07	1.86E-05
				DERM	Benzo(b)fluoranthene	4.79E-07	1.60E-05
				DERM	Fluoranthene	5.78E-07	1.45E-05
				DERM	Phenanthrene	4.19E-07	1.40E-05
				DERM	Benz(a)anthracene	3.60E-07	1.20E-05
				DERM	Bis(2-ethylhexyl) phthalate	2.04E-07	1.02E-05
				DERM	Benzo(a)pyrene	2.61E-07	8.70E-06
				DERM	Chrysene	2.61E-07	8.70E-06
				DERM	Benzo(ghi)perylene	1.75E-07	5.85E-06
				DERM	Indeno(1,2,3-cd)pyrene	1.75E-07	5.85E-06
				DERM	Fluorene	1.03E-07	2.58E-06
				DERM	Acenaphthene	1.03E-07	1.72E-06
				DERM	Butylbenzyl phthalate	2.04E-07	1.02E-06
				DERM	Acetone	4.19E-08	4.19E-07
				DERM	Anthracene	1.21E-07	4.02E-07
				DERM	Methylene chloride	2.34E-09	3.90E-08
				DERM	Selenium	2.19E-11	4.38E-09
				INH	Methylene chloride	4.72E-11	5.48E-11
				INH	Acetone	8.45E-10	
				INH	Acenaphthene	2.08E-09	
				INH	Anthracene	2.43E-09	
				INH	Bis(2-ethylhexyl) phthalate	4.12E-09	
				INH	Benz(a)anthracene	7.25E-09	
				INH	Benzo(a)pyrene	5.26E-09	
				INH	Benzo(b)fluoranthene	9.65E-09	
				INH	Butylbenzyl phthalate	4.12E-09	
				INH	Benzo(ghi)perylene	3.54E-09	
				INH	Chrysene	5.26E-09	
				INH	Fluoranthene	1.17E-08	
				INH	Fluorene	2.08E-09	
				INH	Indeno(1,2,3-cd)pyrene	3.54E-09	
				INH	Phenanthrene	8.45E-09	
				INH	Pyrene	1.13E-08	
				INH	Selenium	4.41E-12	
				ORAL	Pyrene	1.04E-06	3.47E-05
				ORAL	Benzo(b)fluoranthene	8.94E-07	2.98E-05
				ORAL	Fluoranthene	1.08E-06	2.70E-05
				ORAL	Phenanthrene	7.83E-07	2.61E-05
				ORAL	Benz(a)anthracene	6.72E-07	2.24E-05
				ORAL	Bis(2-ethylhexyl) phthalate	3.81E-07	1.91E-05
				ORAL	Benzo(a)pyrene	4.87E-07	1.62E-05
				ORAL	Chrysene	4.87E-07	1.62E-05
				ORAL	Benzo(ghi)perylene	3.27E-07	1.09E-05
				ORAL	Indeno(1,2,3-cd)pyrene	3.27E-07	1.09E-05
				ORAL	Fluorene	1.92E-07	4.81E-06
				ORAL	Acenaphthene	1.92E-07	3.21E-06
				ORAL	Butylbenzyl phthalate	3.81E-07	1.91E-06
				ORAL	Acetone	7.82E-08	7.82E-07
				ORAL	Anthracene	2.25E-07	7.50E-07
				ORAL	Selenium	4.09E-10	8.17E-08
				ORAL	Methylene chloride	4.37E-09	7.28E-08

Redstone Bra1 ARES:
Noncarcinogenic Intakes
Future Worker 1 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
ST	ADWRK	FUT	GW	DERM	Chlorobenzene	5.07E-04	2.54E-02
				DERM	Benzene	4.32E-06	1.44E-02
				DERM	Fluorene	1.17E-04	2.93E-03
				DERM	Phenanthrene	7.78E-05	2.59E-03
				DERM	Pyrene	6.15E-05	2.05E-03
				DERM	Vinyl chloride	2.16E-06	1.66E-03
				DERM	Fluoranthene	6.61E-05	1.65E-03
				DERM	Naphthalene	5.29E-05	1.32E-03
				DERM	Trichloroethene	3.22E-06	5.36E-04
				DERM	Acenaphthene	2.89E-05	4.81E-04
				DERM	Tetrachloroethene	4.33E-06	4.33E-04
				DERM	Dichloroethenes, 1,2-, total	2.06E-06	2.29E-04
				DERM	Cadmium (aqueous matrix)	9.87E-08	1.97E-04
				DERM	Chromium, total	8.66E-07	1.73E-04
				DERM	Anthracene	4.29E-05	1.43E-04
				DERM	Chloroform	1.31E-06	1.31E-04
				DERM	Ethylbenzene	6.75E-06	6.75E-05
				DERM	Barium	4.28E-06	6.11E-05
				DERM	Trichloroethane, 1,1,1-	2.96E-06	3.29E-05
				DERM	Toluene	4.06E-06	2.03E-05
				DERM	Dichloroethane, 1,2-	4.73E-07	1.58E-05
				DERM	Mercury	4.62E-09	1.54E-05
				DERM	Selenium	6.54E-08	1.31E-05
				DERM	Acetone	9.91E-07	9.91E-06
				DERM	Methylene chloride	4.00E-07	6.66E-06
				DERM	Xylenes, total	8.54E-06	4.27E-06
				DERM	Lead	3.07E-09	
				INH	Chlorobenzene	7.37E-03	1.29E+00
				INH	Benzene	1.22E-04	7.20E-02
				INH	Dichloroethane, 1,2-	5.30E-05	1.85E-02
				INH	Toluene	5.37E-05	4.88E-04
				INH	Trichloroethane, 1,1,1-	1.04E-04	3.58E-04
				INH	Ethylbenzene	5.43E-05	1.87E-04
				INH	Methylene chloride	5.28E-05	6.14E-05
				INH	Dichloroethenes, 1,2-, total	1.23E-04	
				INH	Acetone	1.03E-03	
				INH	Vinyl chloride	1.76E-04	
				INH	Chloroform	8.77E-05	
				INH	Tetrachloroethene	5.37E-05	
				INH	Trichloroethene	1.20E-04	
				INH	Xylenes, total	5.71E-05	
				ORAL	Benzene	5.67E-05	1.89E-01
				ORAL	Chlorobenzene	3.41E-03	1.70E-01
				ORAL	Vinyl chloride	8.14E-05	6.26E-02
				ORAL	Cadmium (aqueous matrix)	2.72E-05	5.44E-02
				ORAL	Chromium, total	1.19E-04	2.39E-02
				ORAL	Barium	1.18E-03	1.68E-02
				ORAL	Trichloroethene	5.54E-05	9.23E-03
				ORAL	Dichloroethenes, 1,2-, total	5.68E-05	6.31E-03
				ORAL	Naphthalene	2.11E-04	5.28E-03
				ORAL	Acetone	4.79E-04	4.79E-03
				ORAL	Mercury	1.27E-06	4.24E-03
				ORAL	Chloroform	4.06E-05	4.06E-03
				ORAL	Selenium	1.80E-05	3.60E-03
				ORAL	Phenanthrene	7.94E-05	2.65E-03
				ORAL	Tetrachloroethene	2.49E-05	2.49E-03
				ORAL	Fluorene	8.98E-05	2.25E-03
				ORAL	Pyrene	5.14E-05	1.71E-03
				ORAL	Fluoranthene	5.06E-05	1.26E-03
				ORAL	Acenaphthene	5.30E-05	8.84E-04
				ORAL	Dichloroethane, 1,2-	2.46E-05	8.19E-04
				ORAL	Trichloroethane, 1,1,1-	4.80E-05	5.34E-04
				ORAL	Methylene chloride	2.45E-05	4.08E-04
				ORAL	Ethylbenzene	2.51E-05	2.51E-04
				ORAL	Anthracene	5.14E-05	1.71E-04
				ORAL	Toluene	2.49E-05	1.24E-04
				ORAL	Xylenes, total	2.64E-05	1.32E-05
				ORAL	Lead	2.12E-04	

Redstone Bra1 ARES:
Noncarcinogenic Intakes
Future Worker 1 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
ST	ADWRK	FUT	SO	DERM	DDT, p,p'-	7.24E-09	1.45E-05
				DERM	Bis(2-ethylhexyl) phthalate	5.17E-08	2.59E-06
				DERM	DDE, p,p'-	1.14E-09	2.27E-06
				DERM	Acetone	1.22E-07	1.22E-06
				DERM	Methylene chloride	4.92E-09	8.20E-08
				INH	Methylene chloride	9.92E-11	1.15E-10
				INH	Acetone	2.46E-09	
				INH	Bis(2-ethylhexyl) phthalate	1.04E-09	
				INH	DDE, p,p'-	2.29E-11	
				INH	DDT, p,p'-	1.46E-10	
				ORAL	DDT, p,p'-	1.35E-08	2.70E-05
				ORAL	Bis(2-ethylhexyl) phthalate	9.65E-08	4.83E-06
				ORAL	DDE, p,p'-	2.12E-09	4.24E-06
				ORAL	Acetone	2.28E-07	2.28E-06
				ORAL	Methylene chloride	9.18E-09	1.53E-07

Redstone Bra1 ARES:
Noncarcinogenic Intakes
Future Worker 2 Exposure

CONTAREA	RECEPTOR	CURR/FUT	MEDIUM	PATHWAY	CHEMNAME	INTAKE	RMEHI
G	ADWRK	FUT	GW	DERM	Chromium, total	2.27E-05	4.54E-03
				DERM	Cadmium (aqueous matrix)	1.13E-06	2.27E-03
				DERM	Barium	7.35E-05	1.05E-03
				DERM	Arsenic	2.71E-07	9.03E-04
				DERM	Acetone	1.20E-07	1.20E-06
				DERM	Lead	1.98E-08	
				INH	Acetone	1.25E-04	
				ORAL	Chromium, total	3.12E-03	6.25E-01
				ORAL	Cadmium (aqueous matrix)	3.12E-04	6.24E-01
				ORAL	Barium	2.02E-02	2.89E-01
				ORAL	Arsenic	7.46E-05	2.49E-01
				ORAL	Acetone	5.79E-05	5.79E-04
				ORAL	Lead	1.36E-03	
G	ADWRK	FUT	SE	DERM	Arsenic	1.20E-11	4.01E-08
				DERM	Acetone	3.21E-09	3.21E-08
				DERM	Chromium, total	1.54E-10	3.08E-08
				DERM	Methylene chloride	6.01E-10	1.00E-08
				DERM	Barium	2.05E-10	2.92E-09
				ORAL	Arsenic	3.32E-10	1.11E-06
				ORAL	Chromium, total	4.26E-09	8.52E-07
				ORAL	Acetone	8.88E-09	8.88E-08
				ORAL	Barium	5.66E-09	8.08E-08
				ORAL	Methylene chloride	1.66E-09	2.77E-08
G	ADWRK	FUT	SO	DERM	Acetone	4.04E-08	4.04E-07
				DERM	Methylene chloride	3.15E-09	5.25E-08
				INH	Methylene chloride	6.35E-11	7.39E-11
				INH	Acetone	8.15E-10	
				ORAL	Acetone	7.55E-08	7.55E-07
				ORAL	Methylene chloride	5.88E-09	9.80E-08
G	ADWRK	FUT	SW	DERM	Trichloroethene	3.60E-07	6.00E-05
				DERM	Barium	1.14E-07	1.63E-06
				DERM	Methylene chloride	3.63E-08	6.05E-07
				DERM	Dichloroethane, 1,1-	3.65E-08	3.65E-07
				DERM	Xylenes, total	4.41E-07	2.20E-07
				DERM	Acetone	1.28E-08	1.28E-07
				ORAL	Trichloroethene	1.24E-07	2.07E-05
				ORAL	Barium	6.32E-07	9.02E-06
				ORAL	Acetone	1.24E-07	1.24E-06
				ORAL	Methylene chloride	4.46E-08	7.44E-07
				ORAL	Dichloroethane, 1,1-	2.27E-08	2.27E-07
				ORAL	Xylenes, total	2.74E-08	1.37E-08

APPENDIX E

TOXICOLOGICAL PROFILES

APPENDIX E
TOXICOLOGICAL PROFILES
for
CHEMICALS OF CONCERN (COCs)

Arsenic

Barium

Benzene

Bis(2-ethylhexyl)phthalate

Cadmium

Carbon tetrachloride

Chlorobenzene

Chloroform

Chromium

Copper

1,2-Dichloroethane

Mercury

Methylene chloride

PAHs

Vinyl chloride

BARIUM
(CAS No. 7440-39-3)

INTRODUCTION

Barium is one of the alkaline earth metals. It has seven naturally occurring stable isotopes of which ^{138}Ba is the most abundant. In nature barium occurs only in a combined state, the principal mineral forms being barite (barium sulfate) and witherite (barium carbonate). It may be found as a natural component of fossil fuel and is present in air, water, and soil (WHO, 1990).

Certain barium compounds, such as acetate, nitrate, and chloride, are relatively water soluble, whereas the fluoride, carbonate, oxalate, chromate, phosphate, and sulfate salts have low solubility. With the exception of barium sulfate, the water solubility of the barium salts increases with decreasing pH.

Barite ore is the raw material from which nearly all other barium compounds are derived. Barium and its compounds are used in diverse industrial products ranging from ceramics to lubricants. Anthropogenic sources of barium are primarily industrial. Emissions may result from mining, refining, or processing of barium minerals and manufacture of barium products. It is also discharged in wastewater from metallurgical and industrial processes. Deposition on soil may result from the disposal of fly ash and sewage sludge.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) are available from EPA (IRIS, 1995; EPA, 1994) and are presented in Table 1. Chronic and subchronic inhalation reference concentrations (RfCs) and RfDs are also available from EPA and are presented in Table 1 (EPA, 1994).

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors [a] / (Modifying Factors) [b]
Oral					
Chronic	7E-2	Medium	Increased blood pressure	IRIS, 1995	3 S
Subchronic	7E-2	Medium	Increased blood pressure	EPA, 1994	3 S
Inhalation					
Chronic	1E-4 [c]	--	Fetotoxicity	EPA, 1994	1,000 C,H,S
Subchronic	1E-3 [d]	--	Fetotoxicity	EPA, 1994	100 H,S

Note: RfD = reference dose.
RfC = reference concentration.
-- = No EPA Review.

[a] Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.

[b] A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

[c] Based on an RfC of 5E-4 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

[d] Based on an RfC of 5E-3 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

EPA has determined that no single study considered alone is appropriate to calculate a lifetime RfD for barium (IRIS, 1995). Therefore, the chronic oral RfD (and interim subchronic oral RfD) of 0.07 mg/kg/day are based on a weight of evidence approach which takes into account findings of the Wones et al. (1990) and Brenniman and Levy (1984) epidemiologic studies, as well as the various rodent studies that have been conducted. Wones et al. (1990) administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers who had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study, and subjects were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for weeks 0 to 2; 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. There were no changes in systolic or diastolic blood pressures, cardiac cycle, or serum chemistry. A no-observed-adverse-effect level (NOAEL) of 10 mg/L was identified in this study, which corresponds to 0.21 mg/kg/day (based on an actual consumption rate of 1.5 L/day and a 70-kilogram body weight).

Brenniman and Levy (1984) conducted a retrospective epidemiology study which compared human mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Differences in mortality rates from all cardiovascular diseases were significantly higher in the communities with elevated barium (7.3 mg/L). However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility, or use of water softeners or medication. A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70-kg adult drinks 2 L/day).

According to EPA guidelines, an uncertainty factor (UF) of 10 is applied when a NOAEL from a subchronic human study is employed. However, this UF is not considered necessary because data are available from chronic human studies, as well as several oral chronic animal studies, which support this NOAEL (IRIS,

1995). In addition, another factor of 10 is used with a human study to protect sensitive individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique focus and the supporting studies, a UF of 3, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population. Therefore, applying a UF of 3 to the NOAEL results in a chronic (and interim subchronic) oral RfD of 0.07 mg/kg/day.

The subchronic inhalation reference concentration (RfC) is based on a subchronic animal study by Tarasenko et al. (1977) and was derived using methodology that is not current with the interim inhalation methodology used by EPA's RfD/RfC Work Group (EPA, 1994). In this study, fetotoxicity was observed in rats exposed to barium in air for 4 months, with a NOEL of 0.8 mg/m^3. Adjusting this value for human intake and applying a UF of 100 (10 for animal-to-human extrapolation and and 10 for sensitive human subpopulations) results in an interim subchronic inhalation RfC of 0.005 mg/kg/day. Applying an additional UF of 10 to extrapolate from a subchronic study to a chronic exposure results in a chronic inhalation RfC of 0.0005 mg/m^3. Assuming a healthy 70-kilogram adult inhales 20 m^3/day of air, these RfCs may be converted to subchronic and chronic inhalation RfDs of 0.001 and 0.0001 mg/kg/day, respectively.

Ingestion of high concentrations of soluble barium compounds may result in gastrointestinal effects, such as vomiting and diarrhea, and central nervous system effects, which may include violent tonic and clonic spasms and subsequent paralysis (EPA, 1986). Barium salts are considered to be muscle stimulants, expecially for the heart. This cardiostimulation, combined with vasoconstriction, is the cause of hypertension in barium-exposed individuals. Since barium is readily excreted from the body, significant accumulation of this metal in bone, muscle, renal, and other tissues is not expected (EPA, 1986).

Barium is not considered to be a significant industrial health hazard; however, occupational studies of workers exposed to dusts of barium sulfate, oxide, and carbonate have shown that workers develop a benign form of pneumoconiosis known as baritosis (HSDB, 1995; EPA, 1985). Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension. The radiologic changes in the lung are reversible upon removal from exposure (EPA, 1985). In addition, barium oxide dusts are considered potential dermal and nasal irritants (EPA, 1985).

Carcinogenicity

Barium has not been evaluated by EPA for evidence of human carcinogenic potential.

Mutagenicity

Little information was identified in the available literature regarding the mutagenic potential of barium. In a study by Nishioka (1975), barium chloride produced no increased mutation frequency in repair deficient strains of *Bacillus subtilis*. In addition, no *in vitro* viral DNA transcription errors were observed during a study by Loeb *et al*. (1978).

Teratogenicity/Reproductive Effects

Little information was identified in the available literature regarding teratogenicity or reproductive effects associated with exposure to barium. Barium chloride was found to cause minor limb abnormalities in embryonic chicks after egg injection (Ridgeway and Karnofsky, 1952), while barium fluoride was not teratogenic in rats (Schardein, 1985).

ECOTOXICITY

Aquatic Organisms

The physical and chemical properties of barium generally will preclude the existence of the toxic soluble form under usual freshwater and marine conditions (EPA, 1986). In most natural waters, there is sufficient sulfate or carbonate to precipitate the barium present in the water as a virtually insoluble, non-toxic compound (EPA, 1986). According to EPA (1986), experimental data indicate that the soluble barium concentration in fresh or marine water generally would have to exceed 50,000 μg/L before toxicity to aquatic life would be expected.

Terrestrial Organisms

In the rat, oral LD_{50} values of 118, 250, and 355 mg/kg-bw were measured from barium chloride, fluoride, and nitrate, respectively (RTECS, 1985). The acute effects of barium ingestion include saluration, nausea, diarrhea, tachycardia, hypokalaemia, twitching, flaccid paralysis of skeletal muscle, respiratory muscle paralysis, and ventricular fibrillation. Respiratory muscle paralysis and vantricular fibrillation may lead to death. The acute toxicity of various barium compounds is shown in Table 2. Doses are expressed as either LD_{50} or as LLD.

STANDARDS AND CRITERIA

Human Receptors

EPA has promulgated a nonenforceable health-based Maximum Contaminant Level Goal (MCLG) and an enforceable (for public water supplies) Maximum Contaminant Level (MCL) of 2 mg/L for barium based on the potential adverse health effects reported in epidemiological and animal studies (IRIS, 1995). In addition, EPA has set an ambient water quality criterion (AWQC) for the protection of human health from exposure to barium in water and aquatic organisms of 1 mg/L (IRIS, 1995).

No national ambient air quality standards (NAAQS) have been developed for barium under the Clean Air Act (IRIS, 1995). However, the Occupational Safety

Table 2. Toxicity of Various Barium Compounds in Laboratory Animals

Common Name	Study Type	Effect	Concentration	Units	Exposure	Duration	Comments	Reference
Rat	Acute	LD_{50}	118	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rat	Chronic	Hypertension	10 or 100	mg/L	Oral	16 months	Drinking water	RTECS, 1985
Rat	Chronic	No toxicity	250	mg/L	Oral	13 weeks	Drinking Water	RTECS, 1985
Rat	Acute	LD_{50}	418	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Rat	Acute	LD_{50}	800	mg/kg-bw	Oral		Barium carbonate	Windholz, 1983
Mouse	Acute	LD_{50}	200	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Dog	Acute	LLD	400	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Mouse	Acute	LLD	70	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Dog	Acute	LLD	90	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rabbit	Acute	LLD	170	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Guinea Pig	Acute	LLD	76	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rat	Acute	LD_{50}	250	mg/kg-bw	Oral		Barium fluoride	RTECS, 1985
Rat	Acute	LD_{50}	355	mg/kg-bw	Oral		Barium nitrate	RTECS, 1985

Source: WHO, 1990.

and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to barium (NIOSH, 1990). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1990; ACGIH, 1993). These values are presented in Table 3.

Ecological Receptors

It is generally believed that the physical and chemical properties of barium will preclude the existence of toxic soluble forms under usual marine and fresh water conditions and thus a restrictive criterion for aquatic life is considered unwarranted (IRIS, 1995).

Table 3. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	0.5 (soluble compounds)	0.5 (soluble compounds)	0.5 (soluble compounds)
STEL	--	--	--
CL	--	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.

STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.

CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.

Source: ESE.

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Brenniman, G.R. and Levy, P.S. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, NJ. p. 231-249. (cited in IRIS, 1995).

Hazardous Substances Data Bank (HSDB). 1995. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Loeb, L., Sirover, M., and Agarwal, G. 1978. Infidelity of DNA synthesis as related to mutagenesis and carcinogenesis. Adv. Exp. Med. Biol. 91:103-115. (cited in EPA, 1984).

National Institute of Occupational Safety and Health (NIOSH). 1990. Pocket Guide to Chemical Hazards. Cincinnati, OH. DHHS (NIOSH) Publication No. 90-117.

Nishioka, H. 1975. Mutagenic activities of metal compounds in bacteria. Mutat. Res. 31:185-189. (cited in EPA, 1984).

Registry of Toxic Effects of Chemical Substances (RTECS). 1985. Cumulative Supplement to the 1981-1982 Edition. National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH. NIOSH Publication No. 86-103. (Cited in WHO, 1990).

Reproductive Toxicology System (ReproTox®). 1995. Reproductive Toxicology Center, Columbia Hospital for Women Medical Center, Washington, DC. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

REFERENCES
(Continued, Page 2 of 3)

Reynolds, J.E.F. and Prasad, A.B. (eds.). 1982. Martindale-The Extra Pharmacopoeia. 28th Ed. The Pharmaceutical Press, London. (cited in HSDB, 1995).

Ridgeway, L.P. and Karnofsky, D.A. 1952. The effects of metals on the chick embryo: Toxicity and production of abnormalities in development. Ann. NY Acad. Sci. 55:203-15. (cited in ReproTox, 1995).

Schardein, J.L. 1985. Chemically Induced Birth Defects. Marcel Dekker, New York, NY. p. 633. (cited in ReproTox, 1995).

Tarasenko, M., Promin, O., and Silayev, A. 1977. Barium compounds as industrial poisons (an experimental study). Jour. Hyg. Epidemiol. Microbiol. Immunol. 21:361. (cited in EPA, 1984).

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Barium. Office of Research and Development, Office of Health and Environmental Assessment, Environmental Citeria and Assessment Office, Cincinnati, OH. EPA 540/1-86/021. NTIS No. PB86-134327.

U.S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. Prepared by Clement Associates, Inc. Arlington, VA.

U.S. Environmental Protection Agency (EPA). 1986. Quality Criteria for Water--1986. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-86-001.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. Publication No. 9200.6-303(94-1). NTIS No. PB94-921199.

Windholz, M., ed. (1983). The Merck Index, 10th Ed., Rahway, NJ, Merck and Co., Inc.

Wones, R.G., Stadler, B.L., and Frohman, L.A. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ. Health Perspect. 85:113. (cited in IRIS, 1995).

REFERENCES
(Continued, Page 3 of 3)

World Health Organization (WHO). 1990. Environmental Health Criteria 107. Barium. World Health Organization, Geneva.

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the concentration that should not be exceeded at any any time during a workday. The code "c" designates that this chemical is regulated as a potential carcinogen.

Ecological Receptors

EPA ambient water quality criteria (AWQCs) for the protection of freshwater and marine organisms against exposure to trivalent arsenic are provided by EPA (IRIS, 1994) and are presented in the following table.

AWQC (μg/L)	Freshwater	Marine
Acute	360	69
Chronic	190	36

EPA states that data are insufficient for deriving AWQC for pentavalent arsenic, but indicates that aquatic plants are the most sensitive with toxicity reported at 48 μg/L, while 850 μg/L is reported as the LOEC for the invertebrates with an acute-chronic ratio of 28 (IRIS, 1994; EPA, 1986). Dividing the LOEC by the acute-chronic ratio provides a value of 30 μg/L, which is below the aquatic plant LOEC.

REFERENCES

Adriano, D.C. 1986. Trace Elements in the Terrestrial Environment. Springer-Verlag, NY.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Braman, R.A. and Foreback, C.C. 1973. Methylated Forms of Arsenic in the Environment. Science, 182:1247-1249.

Clement, W. and Faust, S. 1981. The Release of Arsenic from Contaminated Sediments and Muds. Jour. Environ. Sci. Health, 16:87-122.

Environmental Science & Engineering, Inc. (ESE). 1989. Biota Remedial Investigation. Final Report. Prepared for Office of the Program Manager, Rocky Mountain Arsenal, Denver, CO. Contract No. DAAK11-84-D0016, Task 9. Gainesville, FL.

Gough, L.P., Shacklette, H.T., and Case, A.A. 1979. Element Concentrations Toxic to Plants, Animals, and Man. U.S. Geological Survey, Department of the Interior, Washington, DC. Survey Bulletin 1466.

Hatch, R.C. 1977. Poisons Causing Nervous Stimulation or Depression. In: Veterinary Pharmacology and Therapeutics. Jones, L.M., Booth, N.H., and McDonald, L.E., Eds. Iowa State University Press, Ames, IA.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Klaassen, C.D. 1985. Heavy Metals and Heavy Metal Antagonists. In: The Pharmacological Basis of Therapeutics. 7th Ed. Gilman, A.G., Goodman, L.S., Rall, T.W., and Murad, F., Eds. Macmillan Publishing Co., Inc., New York, NY.

Ledet, A.E., Duncan, J.R., Buck, W.B., and Ramsey, F.K. 1973. Clinical, Toxicological, and Pathological Aspects of Arsanilic Acid Poisoning in Swine. Clin. Toxicol., 6:439.

Liebig, G.F. 1965. Arsenic. In: Diagnostic Criteria for Soils and Plants. H.D. Chapman, Ed. Quality Printing Co., Inc., Abilene, TX.

REFERENCES
(Continued, Page 2 of 3)

Merry, R.H., Tiller, K.G., and Alston, A.M. 1986. The Effects of Contamination of Soil With Copper, Lead and Arsenic on the Growth and Composition of Plants. I. Effects of Season, Genotype, Soil Temperature and Fertilizers. Plant and Soil, 91:115-128.

National Academy of Sciences (NAS). 1983. Drinking Water and Health, Volume 5. National Academy of Sciences Press, Washington, DC.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Osweiler, G.D., Carson, T.L., Buck, W.B., and Van Gelder, G.A. 1985. Clinical and Diagnostic Veterinary Toxicology. 3rd Ed. Kendall/Hunt Publishing Co., Dubuque, IA.

Palmer, J.S. 1972. Toxicity of 45 Organic Herbicides to Cattle, Sheep and Chickens. USDA-ARS Prod. Res. Rep. No. 137:22-23.

Radeleff, R.D. 1970. Veterinary Toxiology. 2nd Ed. Lea and Febiger, Philadelphia, PA.

Reproductive Risk Information System (ReproText®). 1994. B.J. Dabney. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Shepard's Catalog of Teratogenic Agents. 1994. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Speer, H.L. 1973. The Effect of Arsenic and Other Inhibitors on Early Events During the Germination of Lettuce Seeds. Plant Physiology, 52:142-146.

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Arsenic. Environmental Criteria and Assessment Office. Cincinnati, OH. EPA 540/1-86/010. NTIS No. PB86-134319.

U.S. Environmental Protection Agency (EPA). 1985. Ambient Water Quality Criteria for Arsenic--1984. Office of Water Regulations and Standards, Washington, DC. NTIS No. PB85-227445.

REFERENCES
(Continued, Page 3 of 3)

U.S. Environmental Protection Agency (EPA). 1986. Standard Evaluation Procedure, Ecological Risk Assessment. Hazard Evaluation Division, Office of Pesticides Programs, Washington, DC. EPA 540/9-85/001.

U.S. Environmental Protection Agency (EPA). 1988. Special Report on Ingested Inorganic Arsenic. Skin Cancer; Nutritional Essentiality. Risk Assessment Forum, Washington, DC. EPA 625/3-87/013.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

Wauchope, R.D. and McDowell, L.L. 1984. Adsorption of Phosphate, Arsenate, Methanearsonate, and Cacodylate by Lake and Stream Sediments: Comparison with Soils. Jour. of Environ. Qual., 13:499-504.

Woolson, E.A., Axley, J.H., and Kearney, P.C. 1973. The Chemistry and Phytotoxicity of Arsenic in Soils. II. Effects of Time and Phosphorus. Soil Science Society of America Proceedings, 37:254-259.

BARIUM
(CAS No. 7440-39-3)

INTRODUCTION

Barium is one of the alkaline earth metals. It has seven naturally occurring stable isotopes of which ^{138}Ba is the most abundant. In nature barium occurs only in a combined state, the principal mineral forms being barite (barium sulfate) and witherite (barium carbonate). It may be found as a natural component of fossil fuel and is present in air, water, and soil (WHO, 1990).

Certain barium compounds, such as acetate, nitrate, and chloride, are relatively water soluble, whereas the fluoride, carbonate, oxalate, chromate, phosphate, and sulfate salts have low solubility. With the exception of barium sulfate, the water solubility of the barium salts increases with decreasing pH.

Barite ore is the raw material from which nearly all other barium compounds are derived. Barium and its compounds are used in diverse industrial products ranging from ceramics to lubricants. Anthropogenic sources of barium are primarily industrial. Emissions may result from mining, refining, or processing of barium minerals and manufacture of barium products. It is also discharged in wastewater from metallurgical and industrial processes. Deposition on soil may result from the disposal of fly ash and sewage sludge.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) are available from EPA (IRIS, 1994; EPA, 1994) and are presented in Table 1. Chronic and subchronic inhalation reference concentrations (RfCs) and RfDs are also available from EPA and are presented in Table 1 (EPA, 1994).

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors [a] / (Modifying Factors) [b]
Oral					
Chronic	7E-2	Medium	Increased blood pressure	IRIS, 1994	3 S
Subchronic	7E-2	Medium	Increased blood pressure	EPA, 1994	3 S
Inhalation					
Chronic	1E-4 [c]	--	Fetotoxicity	EPA, 1994	1,000 C,H,S
Subchronic	1E-3 [d]	--	Fetotoxicity	EPA, 1994	100 H,S

Note: RfD = reference dose.
RfC = reference concentration.
-- = No EPA Review.

[a] Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.

[b] A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

[c] Based on an RfC of 5E-4 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

[d] Based on an RfC of 5E-3 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

EPA has determined that no single study considered alone is appropriate to calculate a lifetime RfD for barium (IRIS, 1994). Therefore, the chronic oral RfD (and interim subchronic oral RfD) of 0.07 mg/kg/day are based on a weight of evidence approach which takes into account findings of the Wones et al. (1990) and Brenniman and Levy (1984) epidemiologic studies, as well as the various rodent studies that have been conducted. Wones et al. (1990) administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers who had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study, and subjects were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for weeks 0 to 2; 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. There were no changes in systolic or diastolic blood pressures, cardiac cycle, or serum chemistry. A no-observed-adverse-effect level (NOAEL) of 10 mg/L was identified in this study, which corresponds to 0.21 mg/kg/day (based on an actual consumption rate of 1.5 L/day and a 70-kilogram body weight).

Brenniman and Levy (1984) conducted a retrospective epidemiology study which compared human mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Differences in mortality rates from all cardiovascular diseases were significantly higher in the communities with elevated barium (7.3 mg/L). However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility, or use of water softeners or medication. A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70-kg adult drinks 2 L/day).

According to EPA guidelines, an uncertainty factor (UF) of 10 is applied when a NOAEL from a subchronic human study is employed. However, this UF is not considered necessary because data are available from chronic human studies, as well as several oral chronic animal studies, which support this NOAEL (IRIS, 1994). In addition, another factor of 10 is used with a human study to protect sensitive

individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique focus and the supporting studies, a UF of 3, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population. Therefore, applying a UF of 3 to the NOAEL results in a chronic (and interim subchronic) oral RfD of 0.07 mg/kg/day.

The subchronic inhalation reference concentration (RfC) is based on a subchronic animal study by Tarasenko et al. (1977) and was derived using methodology that is not current with the interim inhalation methodology used by EPA's RfD/RfC Work Group (EPA, 1994). In this study, fetotoxicity was observed in rats exposed to barium in air for 4 months, with a NOEL of 0.8 mg/m^3. Adjusting this value for human intake and applying a UF of 100 (10 for animal-to-human extrapolation and and 10 for sensitive human subpopulations) results in an interim subchronic inhalation RfC of 0.005 mg/kg/day. Applying an additional UF of 10 to extrapolate from a subchronic study to a chronic exposure results in a chronic inhalation RfC of 0.0005 mg/m^3. Assuming a healthy 70-kilogram adult inhales 20 m^3/day of air, these RfCs may be converted to subchronic and chronic inhalation RfDs of 0.001 and 0.0001 mg/kg/day, respectively.

Ingestion of high concentrations of soluble barium compounds may result in gastrointestinal effects, such as vomiting and diarrhea, and central nervous system effects, which may include violent tonic and clonic spasms and subsequent paralysis (EPA, 1986). Barium salts are considered to be muscle stimulants, expecially for the heart. This cardiostimulation, combined with vasoconstriction, is the cause of hypertension in barium-exposed individuals. Since barium is readily excreted from the body, significant accumulation of this metal in bone, muscle, renal, and other tissues is not expected (EPA, 1986).

Barium is not considered to be a significant industrial health hazard; however, occupational studies of workers exposed to dusts of barium sulfate, oxide, and carbonate have shown that workers develop a benign form of pneumoconiosis known as baritosis (HSDB, 1994; EPA, 1985). Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension. The radiologic changes in the lung are reversible upon removal from exposure (EPA, 1985). In addition, barium oxide dusts are considered potential dermal and nasal irritants (EPA, 1985).

Carcinogenicity

Barium has not been evaluated by EPA for evidence of human carcinogenic potential. In one study by Reynolds and Prasad (1982), the prolonged contact of barium-containing contraceptive devices with cervical cells presented a potential cancer risk in susceptible individuals. No further information on this study was available.

Mutagenicity

Little information was identified in the available literature regarding the mutagenic potential of barium. In a study by Nishioka (1975), barium chloride produced no increased mutation frequency in repair deficient strains of Bacillus subtilis. In addition, no *in vitro* viral DNA transcription errors were observed during a study by Loeb et al. (1978).

Teratogenicity/Reproductive Effects

Little information was identified in the available literature regarding teratogenicity or reproductive effects associated with exposure to barium. Barium chloride was found to cause minor limb abnormalities in embryonic chicks after egg injection (Ridgeway and Karnofsky, 1952), while barium fluoride was not teratogenic in rats (Schardein, 1985).

ECOTOXICITY

Aquatic Organisms

The physical and chemical properties of barium generally will preclude the existence of the toxic soluble form under usual freshwater and marine conditions (EPA, 1986). In most natural waters, there is sufficient sulfate or carbonate to precipitate the barium present in the water as a virtually insoluble, non-toxic compound (EPA, 1986). According to EPA (1986), experimental data indicate that the soluble barium concentration in fresh or marine water generally would have to exceed 50,000 μg/L before toxicity to aquatic life would be expected.

Terrestrial Organisms

In the rat, oral LD_{50} values of 118, 250, and 355 mg/kg-bw were measured from barium chloride, fluoride, and nitrate, respectively (RTECS, 1985). The acute effects of barium ingestion include saluration, nausea, diarrhea, tachycardia, hypokalaemia, twitching, flaccid paralysis of skeletal muscle, respiratory muscle paralysis, and ventricular fibrillation. Respiratory muscle paralysis and vantricular fibrillation may lead to death. The acute toxicity of various barium compounds is shown in Table 2. Doses are expressed as either LD_{50} or as LLD.

STANDARDS AND CRITERIA

Human Receptors

EPA has promulgated a nonenforceable health-based Maximum Contaminant Level Goal (MCLG) and an enforceable (for public water supplies) Maximum Contaminant Level (MCL) of 2 mg/L for barium based on the potential adverse health effects reported in epidemiological and animal studies (IRIS, 1994). In addition, EPA has set an ambient water quality criterion (AWQC) for the protection of human health from exposure to barium in water and aquatic organisms of 1 mg/L (IRIS, 1994).

No national ambient air quality standards (NAAQS) have been developed for barium under the Clean Air Act (IRIS, 1994). However, the Occupational Safety

Table 2. Toxicity of Various Barium Compounds in Laboratory Animals

Common Name	Study Type	Effect	Concentration	Units	Exposure	Duration	Comments	Reference
Rat	Acute	LD_{50}	118	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rat	Chronic	Hypertension	10 or 100	mg/L	Oral	16 months	Drinking water	RTECS, 1985
Rat	Chronic	No toxicity	250	mg/L	Oral	13 weeks	Drinking Water	RTECS, 1985
Rat	Acute	LD_{50}	418	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Rat	Acute	LD_{50}	800	mg/kg-bw	Oral		Barium carbonate	Windholz, 1983
Mouse	Acute	LD_{50}	200	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Dog	Acute	LLD	400	mg/kg-bw	Oral		Barium carbonate	RTECS, 1985
Mouse	Acute	LLD	70	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Dog	Acute	LLD	90	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rabbit	Acute	LLD	170	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Guinea Pig	Acute	LLD	76	mg/kg-bw	Oral		Barium chloride	RTECS, 1985
Rat	Acute	LD_{50}	250	mg/kg-bw	Oral		Barium fluoride	RTECS, 1985
Rat	Acute	LD_{50}	355	mg/kg-bw	Oral		Barium nitrate	RTECS, 1985

Source: WHO, 1990.

and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to barium (NIOSH, 1990). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1990; ACGIH, 1993). These values are presented in Table 3.

Ecological Receptors

It is generally believed that the physical and chemical properties of barium will preclude the existence of toxic soluble forms under usual marine and fresh water conditions and thus a restrictive criterion for aquatic life is considered unwarranted (IRIS, 1994).

Table 3. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	0.5 (soluble compounds)	0.5 (soluble compounds)	0.5 (soluble compounds)
STEL	--	--	--
CL	--	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.

STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.

CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.

Source: ESE.

REFERENCES

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Brenniman, G.R. and Levy, P.S. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, NJ. p. 231-249. (cited in IRIS, 1994).

Hazardous Substances Data Bank (HSDB). 1994. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Loeb, L., Sirover, M., and Agarwal, G. 1978. Infidelity of DNA synthesis as related to mutagenesis and carcinogenesis. Adv. Exp. Med. Biol. 91:103-115. (cited in EPA, 1984).

National Institute of Occupational Safety and Health (NIOSH). 1990. Pocket Guide to Chemical Hazards. Cincinnati, OH. DHHS (NIOSH) Publication No. 90-117.

Nishioka, H. 1975. Mutagenic activities of metal compounds in bacteria. Mutat. Res. 31:185-189. (cited in EPA, 1984).

Registry of Toxic Effects of Chemical Substances (RTECS). 1985. Cumulative Supplement to the 1981-1982 Edition. National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH. NIOSH Publication No. 86-103. (Cited in WHO, 1990).

Reproductive Toxicology System (ReproTox®). 1994. Reproductive Toxicology Center, Columbia Hospital for Women Medical Center, Washington, DC. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

REFERENCES
(Continued, Page 2 of 3)

Reynolds, J.E.F. and Prasad, A.B. (eds.). 1982. Martindale-The Extra Pharmacopoeia. 28th Ed. The Pharmaceutical Press, London. (cited in HSDB, 1994).

Ridgeway, L.P. and Karnofsky, D.A. 1952. The effects of metals on the chick embryo: Toxicity and production of abnormalities in development. Ann. NY Acad. Sci. 55:203-15. (cited in ReproTox, 1994).

Schardein, J.L. 1985. Chemically Induced Birth Defects. Marcel Dekker, New York, NY. p. 633. (cited in ReproTox, 1994).

Tarasenko, M., Promin, O., and Silayev, A. 1977. Barium compounds as industrial poisons (an experimental study). Jour. Hyg. Epidemiol. Microbiol. Immunol. 21:361. (cited in EPA, 1984).

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Barium. Office of Research and Development, Office of Health and Environmental Assessment, Environmental Citeria and Assessment Office, Cincinnati, OH. EPA 540/1-86/021. NTIS No. PB86-134327.

U.S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. Prepared by Clement Associates, Inc. Arlington, VA.

U.S. Environmental Protection Agency (EPA). 1986. Quality Criteria for Water--1986. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-86-001.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

Windholz, M., ed. (1983). The Merck Index, 10th Ed., Rahway, NJ, Merck and Co., Inc.

Wones, R.G., Stadler, B.L., and Frohman, L.A. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ. Health Perspect. 85:113. (cited in IRIS, 1994).

REFERENCES
(Continued, Page 3 of 3)

World Health Organization (WHO). 1990. Environmental Health Criteria 107. Barium. World Health Organization, Geneva.

BENZENE
(CAS No. 71-43-2)

INTRODUCTION

Benzene is used primarily as a major constituent of motor fuels, as an industrial solvent, as a solvent in the extraction of oils from seeds and nuts, and as a starting material for the production of a wide range of aromatic compounds.

With a reported solubility of 1,780 mg/L (ORNL, 1989), benzene is soluble in water. When considered in conjunction with the high solubility, the moderate soil adsorption coefficient (K_{oc}) value of 65 (ORNL, 1989) suggests that benzene will be mobile in the soil/groundwater system with little or no retardation in subsurface or sandy soils. Based on the reported vapor pressure of 76 torr at 20°C (ORNL, 1989), transport through the air-filled pores of the near-surface soils may be an important migration pathway. In saturated soils (where soil organic carbon and soil air are negligible), a large fraction of the benzene present (79 percent) is expected to be in the soil-water phase and transported with the flowing groundwater; the balance is expected to be sorbed to the soil. In surficial or near-surface soil, about 88 percent is expected to sorb to soil, with 7 percent in the soil-water phase and 5 percent in the soil-air phase (ORNL, 1989). The half-life in soil is reported to be less than 1 month, although this is dependent on the soil type (Cogley et al., 1975).

Because of its volatility, the primary fate of benzene is associated with atmospheric degradation. However, photolysis is an insignificant degradation process as benzene only absorbs light wavelengths found in the troposphere above the ozone layer (EPA, 1979). Hydrolysis is not expected to occur under ambient conditions in an aqueous environment as nucleophilic attack of the aromatic ring by water or hydroxide ion will be impeded by its negative charge-density (EPA, 1979). Although direct oxidation in water is unlikely, photooxidation may occur quickly once in the atmosphere. Assuming that

benzene depletion is solely due to attack by hydroxyl radicals, and that even the presence of high concentrations of ozone does not contribute significantly to the photooxidation process, an estimated atmospheric half-life of 2.4 to 24 hours is reported (EPA, 1979). Based on its volatility, the half-life of benzene in water is about 5 hours (Mackay and Leinonen, 1975). Benzene is oxidatively degraded by microorganisms, especially bacteria (Chapman, 1972; Tabak et al., 1981; Korte and Klein, 1982). Anaerobically, while degradation occurs, the rate is greatly reduced (Wilson et al., 1986). Benzene is reported to have a bioconcentration factor (BCF) of 65 (ORNL, 1989), indicating that it is not bioaccumulated to a great degree.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA does not provide a final oral reference dose (RfD) or inhalation reference concentration (RfC) for benzene but states that the chronic oral RfD will be reviewed by an EPA Work Group in the future and that the chronic inhalation RfD is currently being reviewed (IRIS, 1995; EPA, 1994). However, EPA's Environmental Criteria and Assessment Office (ECAO) has developed a provisional chronic oral RfD and an interim inhalation RfD that have been adopted by EPA Region III (EPA, 1995) and are presented in Table 1. Based on a subchronic oral no-observed-adverse-effect level (NOAEL) in rats of 0.7 mg/kg/day (Wolf, 1956) and applying an uncertainty factor of 3,000 (10 for sensitive human subpopulations, 10 for animal-to-human extrapolation, 10 for extrapolation from a subchronic study to a chronic exposure, and 3 to account for the inadequate database), a provisional chronic oral RfD of 0.0003 mg/kg/day is derived (EPA, 1993). In addition, an interim chronic inhalation RfD of 5.71×10^{-5} mg/kg/day has been developed by ECAO and has been recommended by EPA Region III (EPA, 1995).

Benzene has been widely studied; however, because the primary exposure route for human receptors is through occupational inhalation, most studies have

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	3×10^{-4}	Medium	Hematological effects	EPA, 1993	3,000 C,D,H,S
Subchronic	3×10^{-3}	Medium	Hematological effects	EPA, 1993	300 D,H,S
Inhalation					
Chronic	5.71×10^{-5}	--	Unknown	EPA, 1995	Unknown
Subchronic	--	--	--	--	--

Note: RfD = reference dose.
RfC = reference concentration.
-- = Not available.
NOAEL = no observed adverse effect level.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic exposure.
D = to account for deficiencies in the database.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

concentrated on the inhalation pathway. The only chronic oral study reported was a carcinogenic bioassay; no symptoms other than carcinomas were reported. No other chronic oral data for human or animal exposure were identified. Following subchronic oral exposure, rats subjected to intakes of 10 mg/kg-bw or higher for a 187-day period evidenced effects to the hemopoietic system, notably leukopenia (Wolf et al., 1956). Based on these data, an oral NOEL for the rat was set at 1 mg/kg-bw.

Chronic inhalation exposure to rats resulted in lymphocytopenia, anemia, and bone marrow hypoplasia (Snyder et al., 1978, 1980). From these studies, an inhalation LOAEL of 100 ppm may be deduced for the mouse. Following subchronic inhalation exposures, leukopenia was the most cited health effect for experimental animals (EPA, 1984). Other effects reported included growth suppression, increased organ weights, and unspecified changes to kidney, bone marrow, spleen, and testes. A subchronic NOEL of 31 ppm for leukopenia was established based on a study by Deichmann et al. (1963).

Carcinogenicity

The carcinogenic slope factor (CSF) and supporting information are summarized in Table 2. EPA has classified benzene as a Class A (known human) carcinogen (IRIS, 1995), which indicates sufficient evidence of human carcinogenicity. A CSF of 0.029 $(mg/kg/day)^{-1}$ has been determined for both inhalation and oral exposure. This classification is based on increased nonlymphocytic leukemias following occupational exposure, increased neoplasia in rats and mice following exposure through inhalation and gavage, and other supporting data. The carcinogenic effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry were compiled and reported by Aksoy et al. (1974), with a followup study subsequently completed (Aksoy, 1980). The results showed that after approximately 10 years of exposure with peak concentrations reported to be between 210 and 650 ppm, the incidence of leukemia or preleukemia was 13 per 100,000, compared to 6 per 100,000 in the

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.029	A	Leukemia	IRIS, 1995
Inhalation	0.029[a]	A	Leukemia	EPA, 1994

Note: A = known human carcinogen (adequate evidence of carcinogenicity in human epidemiological studies).

[a]Inhalation CSF is based on an inhalation unit risk (UR) of 8.3×10^{-6} $(\mu g/m^3)^{-1}$ and assumes that a healthy 70-kilogram adult inhales 20 m^3 of air per day.

Source: ESE.

general population. An additional eight leukemia cases, as well as evidence suggestive of increases in other malignancies, were reported in the followup study.

Mutagenicity

Numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC, 1982). Also, several studies have demonstrated an increased frequency of acquired chromosomal abnormalities in the white blood cells of people who have suffered benzene toxicity or have been occupationally exposed to high levels of benzene (in excess of the current maximum permissible level of exposure) for many years (TERIS, 1995). In one study, an increased frequency of acquired lymphocyte cytogenetic abnormalities was seen in a group of 14 children whose mothers had been chronically exposed to benzene and other organic solvents (TERIS, 1995).

Benzene also induced chromosomal aberrations in bone marrow cells from rabbits (Kissling and Speck, 1973), mice (Meyne and Legator, 1980) and rats (Anderson and Richardson, 1979). Several investigators have reported positive results for benzene in mouse micronucleus assays (Meyne and Legator, 1980). Benzene was not mutagenic in several bacterial and yeast systems, in the sex-linked recessive lethal mutation assay with Drosophila melanogaster or in mouse lymphoma cell forward mutation assay.

Teratogenicity/Reproductive Effects

No data regarding human teratogenicity are available (TERIS, 1995). However, the teratogenicity of benzene has been well studied in mice, rats, and rabbits (TERIS, 1995). Fetotoxicity and delayed ossification have consistently been demonstrated at doses that are maternally toxic and many times greater than the maximum permissible level of human exposure in air. No convincing evidence

exists of an increased frequency of malformations even at such high exposure levels.

ECOTOXICITY

Aquatic Organisms

A range of EC_{50} values has been reported for benzene for freshwater invertebrate and vertebrate species. Six freshwater species of fish have been tested with benzene. The resulting LC_{50} values ranged from 5,300 micrograms per liter (μg/L) for rainbow trout to 386,000 μg/L for the mosquito fish (Gambusia affinus). However, only values for the rainbow trout (5,300 μg/L) were derived using a flow-through system in which the toxicant concentration was measured (EPA, 1980). Tests involving aquatic invertebrates were inconclusive, but no adverse effects were observed at levels as high as 98,000 μg/L (EPA, 1980). Amphibians were found to be even more tolerant of benzene. The lowest LC_{50} for amphibians was a value of 190,000 μg/L reported for the clawed toad, Xenopus laevis (Slooff and Baerselman, 1980). Results of acute exposure tests with species of freshwater invertebrates and vertebrates are presented in Table 3. EPA has determined that the data are insufficient to establish any criteria for aquatic organisms, but that the value of 5.3 mg/L may be considered an acute LOEC. Applying an uncertainty factor of 100 to the acute LOEC provides a water TRV of 0.053 mg/L (53 μg/L) for aquatic organisms.

Several saltwater invertebrate and fish species have also been tested with benzene. Results indicate that the invertebrate EC_{50} values were considerably variable, with a range of values of 17,600 to 964,000 μg/L. The striped bass had 96-hour LC_{50} values of 10,900 and 5,100 μg/L (EPA, 1980). A variety of exposure studies conducted on grass shrimp (Palaemonetes pugio) by Potera (1975) revealed LC_{50} values ranging from 33,500 to 90,800 μg/L, depending upon temperature, salinity, and life-stage of the test organism.

Table 3. Acute Toxicity of Benzene to Freshwater and Saltwater Organisms

Species	LC_{50}/EC_{50} (µg/L)	Species Acute Value (µg/L)	Reference
FRESHWATER SPECIES:			
Cladoceran *Daphnia magna*	203,000	--	EPA, 1978
Cladoceran *Daphnia magna*	400,000	--	Canton and Adema, 1978
Cladoceran *Daphnia magna*	620,000	--	Canton and Adema, 1978
Cladoceran *Daphnia magna*	412,000	--	Canton and Adema, 1978
Cladoceran *Daphnia magna*	412,000	--	Canton and Adema, 1978
Cladoceran *Daphnia magna*	356,000	--	Canton and Adema, 1978
Cladoceran *Daphnia magna*	356,000	380,000	Canton and Adema, 1978
Cladoceran *Daphnia pulex*	345,000	--	Canton and Adema, 1978
Cladoceran *Daphnia pulex*	265,000	300,000	Canton and Adema, 1978
Rainbow trout (juvenile) *Oncorhynchus mykiss*	5,300	5,300	DeGraeve *et al.*, 1980
Goldfish *Carassius auratus*	34,420	34,000	Pickering and Henderson, 1966
Fathead minnow *Pimephales promelas*	33,470	--	Pickering and Henderson, 1966
Fathead minnow *Pimephales promelas*	32,000	33,000	Pickering and Henderson, 1966
Guppy *Poecilla reticulata*	36,600	36,600	Pickering and Henderson, 1966

Table 3. Acute Toxicity of Benzene to Freshwater and Saltwater Organisms (Continued, Page 2 of 2)

Species	LC_{50}/EC_{50} (µg/L)	Species Acute Value (µg/L)	Reference
FRESHWATER SPECIES: (Continued)			
Mosquitofish *Gambusia affinis*	386,000	386,000	Wallen *et al.*, 1957
Bluegill *Lepomis macrochirus*	22,490	22,000	Pickering and Henderson, 1966
SALTWATER SPECIES:			
Pacific oyster *Crassostrea gigas*	924,000	924,000	LeGore, 1974
Copepod *Tigriopus californicus*	450,000	450,000	Korn *et al.*, 1976
Bay shrimp *Crago franciscorum*	17,600	17,600	Benville and Korn, 1977
Grass shrimp *Palaemonetes pugio*	27,000	27,000	Tatem, 1975
Dungeness crab (larva) *Cancer magister*	108,000	108,000	Caldwell *et al.*, 1977
Striped bass *Morone saxatilis*	10,900	--	Meyerhoff, 1975
Striped bass *Morone saxatilis*	5,100	10,900	Benville and Korn, 1977

Note: -- = value not available.

Source: ESE.

Terrestrial Organisms

No information was found concerning the toxicity of benzene to vegetation, therefore, there is insufficient information from which to derive either soil or water TRVs for vegetation. In livestock, benzene induced bone marrow depression, leading to a leukopenia characterized by a greater decrease in lymphocytes than in granulocytes, which is a reversal of the effect in humans (Andrews and Snyder, 1986). The noncarcinogenic effects are primarily hematopoietic, although unspecified histopathologic effects were reported in kidneys and testes (Wolf et al., 1956). Prolonged or frequent dermal contact may lead to blistering; erythema; and a dry, scaly dermatitis (Gerarde, 1960). No information was found regarding benzene toxicity in fowl or in wildlife.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 4. Due to the ranking of benzene as a Group A human carcinogen, EPA has set a nonenforceable Maximum Contaminant Level Goal (MCLG) for drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 5 μg/L, which is enforceable for public water supplies (50 FR 46880). Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 6.6 to 0.066 μg/L for consumption of contaminated drinking water and aquatic organisms (IRIS, 1995). For consumption of aquatic organisms only, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 400 to 4 μg/L, respectively (IRIS, 1995). These AWQC would yield values for human consumption of water alone (W) of 6.7, 0.67, and 0.067 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on the following equation:

Table 4. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day[a]	10-Day[a]	Longterm[a]	Lifetime	Fish and Water	Fish Only	Water Only[b]
Benzene	5	0	--	235	235	--	--	0.66[c]	40[c]	0.67[c]

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is for a 10-kg child.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

No national ambient air quality standards (NAAQS) have been developed for benzene. However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1995). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1995; ACGIH, 1993). These values are presented in the following table of air quality standards.

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	3.25 c	0.325 c	32 c
STEL	16.25 c	3.25 c	--
CL	--	--	--

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the

concentration that should not be exceeded at any any time during a workday. The code "c" designates that this chemical is regulated as a potential carcinogen.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to benzene (IRIS, 1995). However, the lowest levels observed to have an effect (LOELs) on the aquatic species tested have been determined (IRIS, 1995). These LOELs, which are established when the minimum data required to derive AWQCs are not available, are presented in the following table.

LOEL (µg/L)	Freshwater	Marine
Acute	5,300	5,100
Chronic	--	700

REFERENCES

Aksoy, M., Erdem, S., and Dincol, G. 1974. Leukemia in Shoe Workers Exposed Chronically to Benzene. Blood 44:837-841 (cited in EPA, 1980).

Aksoy, M. 1980. Different Types of Malignancies due to Occupational Exposure to Benzene: A Review of Recent Observations in Turkey. Environ. Res. 23:181-190. (cited in IARC, 1982).

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Anderson, D. and Richardson, C.R. 1979. Chromosome gaps are associated with chemical mutagenesis (Abstract No. Ec-9). Environ. Mutat. 1:179.

Andrews, L.S. and Snyder, R. 1986. Toxic Effects of Solvent and Vapors. In: Toxicology: The Basic Science of Poisons. 3rd Ed. J. Doull, C.D. Klaassen, and M.O. Amdur, Eds. Macmillan Publishing Co. Inc., New York, NY.

Benville, P.E., Jr., and S. Korn. 1977. The acute toxicity of six monocyclic aromatic crude oil componenets to striped bass (Morone saxatilis) and bay shrimp (Crago franciscorum). Calif. Fish Game. 63: 204-211.

Caldwell, R.S. et al. 1977. Effects of a seawater soluble fraction of Cook Inlet crude oil and its major aromatic componeents on larval stages of the Dunegeness crab, Cancer magister. In: D.A. Wolfe (ed.), Fate and Effects of Petroleum Hydrocarbons in Marine Organisms and Ecosystems. Pergammon Press. p210.

Canton, H. and Adema, D.M.M. 1978. Reproducibility of Short-Term and Reproduction Toxicity Experiments with Daphnia magna and Comparison of the Sensitivity of Daphnia magna with Daphnia pulex and Daphnia cucullata in Short-Term Experiments. Hydrobiolgia. 59:135. (cited in EPA, 1985b).

Chapman, P.J. 1972. An Outline of Reaction Sequences Used for the Bacterial Degradation of Phenolic Compounds. Degradation Syn. Org. Mol. Biosphere, Proc. Conf., pp. 17-55.

REFERENCES
(Continued, Page 2 of 5)

Cogley, D.R., Grant, D.C., and Hoover, P.R. 1975. Report of Readily Available Data on 109 Compounds Associated with Shell Chemical Company Operations at Rocky Mountain Arsenal. Walden Research Division of Abcor, Inc., Cambridge, MA.

DeGraeve, G.M. et al. 1980. Effects of naphthalene and benzene on fathead minnows and rainbow trout. Trans. Amer. Fish. Soc. (submitted).

Deichmann, W.B., MacDonald, W.E., and Bernal, E. 1963. The Hemopoietic Tissue Toxicity of Benzene Vapors. Toxicol. Appl. Pharmacol. 5:201-224 (cited in EPA, 1982).

Gerarde, H.W. 1960. Toxicology and Biochemistry of Aromatic Hydrocarbons. Elsevier Publishing Co., New York, NY.

Hazardous Substances Data Bank (HSDB). 1995. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

International Agency for Research on Cancer (IARC). 1982. Benzene. In: Some Industrial Chemicals and Dyestuffs. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. World Health Organization, Lyon, France. 29:93-148. (cited in IRIS, 1995).

Kissling, M. and Speck, B. 1973. Chromosome aberrations in experimental benzene intoxication. Helv. Med. Acta. 36:59-66. (cited in IRIS, 1995).

Korn, S. et al., 1976. Effect of benzene on growth, gat content and calorie content of striped bass (Morone saxatilis). Fish. Bull. 74:694.

Korte, F., and Klein, W. 1982. Degradation of Benzene in the Environment. Ecotoxicology and Environmental Safety, 6:311-327.

LeGore, R.S. 1974. The effect of Alaskan crude oil and selected hydrocarbon compounds on embryonic development of the Pacific oyster, Crossostrea gigas. Ph.D. Dissertation. Univ. of Washington.

REFERENCES
(Continued, Page 3 of 5)

Mackay, D., and Leinonen, P.J. 1975. Rate of Evaporation of Low-Solubility Contaminants from Water Bodies to Atmosphere. Environmental Science and Technology, 9(13):1178-1180.

Meyerhoff, R.D. 1975. Acute toxicity of benzene, a component of crude oil, to juvenile striped bass. Jour. Fish. Res. Board Can. 32: 1864-1867.

Meyne, J. and Legator, M.S. 1980. Sex-related differences in cytogenetic effects of benzene in the bone marrow of Swiss mice. Environ. Mutat. 2:43-50. (cited in IRIS, 1995).

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1995. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

Pickering, Q.H. and C. Henderson. 1966. Acute toxicity of some important petrochemicals to fish. Journ. Water Pollution Control Fed. 38:1419.

Potera, F.T. 1975. The effects of benzene (a toxic component of petroleium) on spawning Pacific herring, (Clupea harengus passasi). Fish. Bull. 75:43-49.

Slooff, W. and Baerselman, R. 1980. Comparison of the Usefulness of the Mexican axolotl (Ambystoma Mexicanum) and the clawed toad (Xenopus laevis) in the Toxicological Bioassays. Bull. Environ. Contam. Toxicol. 24(3):439-443. (cited in EPA, 1985 and HSDB, 1995).

Snyder, C.A., Goldstein, B.D., Sellakumar, A. et al. 1978. Hematotoxicity of Inhaled Benzene to Sprague-Dawley Rats and AKR Mice at 300 ppm. Journal of Toxicology and Environmental Health, 4:605-618 (cited in Snyder et al., 1980; U.S. EPA, 1982).

Snyder, C.A., Goldstein, B.D., Sellakumar, A.R., Bromberg, I., Laskin, S., and Albert, R.E. 1980. The Inhalation Toxicology of Benzene: Incidence of Hematopoietic Neoplasms and Hematotoxicity in AKR/J and C57B1/6J Mice. Toxicol. Appl. Pharmacol. 54(2):323-331

REFERENCES
(Continued, Page 4 of 5)

Tabak, H.H., Quave, S.A., Mashni, C.I., and Barth, E.F. 1981. Biodegradability Studies with Organic Priority Pollutant Compounds. Journal Water Pollution Control Federation, 53:1503-1518.

Tatem, H.E. 1975. Toxicity and phsiological effects of oil and petroleum hydrocarbons on estuarine shrimp, Paleomonetes pugio. PhD Dissertation. Texas A.M. Univ.

Teratogen Information System (TERIS). 1995. University of Washington, Seattle, WA. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Environmental Protection Agency (EPA). 1978. In-depth studies on health and environmental impacts of selected water pollutants. U.S. Environmental Protection Agency. Contract No. 68-01-4646.

U.S. Environmental Protection Agency (EPA). 1979. Water-Related Fate of 129 Priority Pollutants. Volume II. Monitoring and Data Support Division, Washington, DC. EPA-440/4-79-029B.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Benzene. Office of Water Regulations and Standards, Criteria and Standards Division. Washington, DC. EPA 440/5-80-018.

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Benzene. Environmental Criteria and Assessment Office. EPA/540/1-86/037. NTIS No. PB86-134483.

U.S. Environmental Protection Agency (EPA). 1993. Risk Assessment Issue Paper for Derivation of a Provisional Oral RfD for Benzene (CASRN 71-43-2). Superfund Technical Support Center, Environmental Criteria and Assessment Office, Cincinnati, OH. No. 93-27.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. Publication No. 9200.6-303(94-1). NTIS No. PB94-921199.

REFERENCES
(Continued, Page 5 of 5)

U.S. Environmental Protection Agency (EPA). 1995. Risk-Based Concentration Table, January - June 1995. Technical Support Section, EPA Region III, Philadelphia, PA. March 7, 1995.

Wallen, I.E. et al. 1957. Toxicity to Gambusia affinis of certain pure chemicals in turbid waters. Sewage Industl. Wastes. 29:695. (cited in EPA, 1985).

Wilson, B.H., Smith, G.B., and Rees, J.F. 1986. Biotransformations of Selected Alkylbenzenes and Halogenated Aliphatic Hydrocarbons in Methanogenic Aquifer Material: A Microcosm Study. Environmental Science and Technology, 20:997-1002.

Wolf, M.A., Rowe, V.K., McCollister, D.D., Hollingsworth, R.L., and Oyen, F. 1956. Toxicological Studies of Certain Alkylated Benzenes and Benzene. Archives of Industrial Health, 14:387-398.

BIS(2-ETHYLHEXYL) PHTHALATE
(CAS No. 117-81-7)

INTRODUCTION

The compound bis(2-ethylhexyl) phthalate (DEHP), one of several phthalate esters, is most commonly used as a plasticizer in products manufactured for commercial, medical, and consumer uses. A small percentage of DEHP is also used as a hydraulic fluid and dielectric fluid in electrical capacitors [Agency for Toxic Substances and Disease Registry (ATSDR), 1989].

The environmental fate of DEHP is influenced strongly by its physical and chemical properties. DEHP is only slightly volatile (vapor pressure = 3.4×10^{-7} mm Hg), and the presence of this chemical in the atmosphere is due to volatilization from plastics and releases during manufacture and incineration (ATSDR, 1989). Due to its relatively high log K_{ow} (4.88), DEHP is expected to adsorb to airborne particulates and be removed from the atmosphere with percipitation, although chemical hydrolysis may occur to some small extent (ATSDR, 1989).

DEHP is not expected to volatilize from soil or surface water, even under conditions favorable to chemical evaporation. The removal of DEHP from surface water occurs primarily through absorption to suspended particulates and sediment and uptake by biota (ATSDR, 1989). Biodegradation in water under aerobic conditions exhibits a half-life of upto a month (ATSDR, 1989). In soil, DEHP is strongly bound to organic particulate matter and is not very mobile. Under aerobic conditions in soil, DEHP exhibits a half-life of several days.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

A chronic oral reference dose (RfD) is available from EPA (IRIS, 1994) and is presented in Table 1. A subchronic oral RfD and inhalation reference

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	0.02	Medium	Increased liver weight	IRIS, 1994	1,000 H,N,S
Subchronic	--	--	--	--	--
Inhalation					
Chronic	--	--	--	--	--
Subchronic	--	--	--	--	--

Note: IRIS = Integrated Risk Information System.
RfD = reference dose.
RfC = reference concentration.
-- = Not available.
LOAEL = lowest observed adverse effect level.
NOAEL = no observed adverse effect level.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
H = to extrapolate from an animal study to humans.
N = to extrapolate from a LOAEL to a NOAEL.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

concentrations (RfCs) have not been determined (IRIS, 1994; EPA, 1994).

The chronic oral RfD of 0.02 milligrams per kilogram per day (mg/kg/day) is based on an animal study by Carpenter et al. (1953) in which guinea pigs were given DEHP in their diets at doses of 0, 19, or 64 mg/kg/day for 1 year. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females, however. Because an effect was observed at the lowest dose used in the study, a lowest-observed-adverse-effect level (LOAEL) of 19 mg/kg/day was established for this study. Applying an uncertainty factor of 1,000 (10 for animal-to-human extrapolation, 10 for sensitive human subpopulations, and 10 for LOAEL to NOAEL extrapolation) results in a chronic oral RfD of 0.02 mg/kg/day.

DEHP is well absorbed from the gastrointestinal tract following oral administration (ATSDR, 1989). Animal studies show the liver and testes to be the primary endpoints for DEHP toxicity. Acute inhalation toxicity studies with rats have demonstrated that DEHP is absorbed by the lung, and dermal studies with animals indicate that DEHP is poorly absorbed by the skin (ATSDR, 1989).

Carcinogenicity

DEHP has been classified as a group B2 (probable human) carcinogen (IRIS, 1994). This classification indicates sufficient evidence of carcinogenicity in animals but inadequate evidence of human carcinogenicity. The carcinogenic slope factors (CSFs) and supporting information are presented in Table 2.

The oral CSF of 0.014 $(mg/kg/day)^{-1}$ was derived from a study by the National Toxicology Program (NTP) (1982) in which DEHP was fed to rats and mice at concentrations of 0, 6,000, or 12,000 parts per million (ppm) for 103 weeks. No clinical signs of toxicity were observed in either rats or mice. A statistically

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.014	B2	Liver tumors	IRIS, 1994
Inhalation	--	B2	--	--

Note: B2 = suspect human carcinogen (sufficient evidence of carcinogenicity in animals and inadequate data in humans).

Source: ESE.

significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenomas were observed in female rats and both sexes of mice, while a statistically significant increase in the combined incidence of neoplastic nodules and hepatocellular carcinomas was observed in the high-dose male rats. A positive dose response trend was also noted.

EPA has not developed an inhalation unit risk or CSF for DEHP (IRIS, 1994; EPA, 1994).

Mutagenicity

DEHP did not produce any mutagenic activity in assays performed with Salmonella typhimurium (Seed, 1982; Kozumbo et al., 1982) or Bacillus subtilis (Tomita et al., 1982). Monoethyl hexyl phthalate (MEHP), the monoester metabolite of DEHP, has shown positive mutagenicity in assays with Salmonella. Both DEHP and MEHP induced chromosomal aberrations and morphological transformation in cultured fetal Syrian hamster cells exposed *in utero* (Tomita et al., 1982); however, chromosomal effects were not found in Chinese hamster ovary cells (Phillips et al., 1982). According to Ganning et al. (1984), DEHP is a potent inducer of hepatic peroxisomal enzyme activity.

Teratogenicity/Reproductive Effects

Dietary levels of 0, 0.01, 0.1, and 0.3 percent DEHP (greater than 99 percent pure) were administered to mice that were examined for adverse fertility and reproductive effects using a continuous breeding protocol. DEHP was a reproductive toxicant in both sexes, significantly decreasing fertility and the proportion of live pups per litter at the 0.3 percent level and inducing damage to the seminiferous tubules. DEHP has been observed to be both fetotoxic and teratogenic (Singhe, 1972; Shiota and Nishimura, 1982).

Shiota et al. (1980) fed mice diets containing 0.05 to 1.0 percent DEHP throughout gestation. In the groups treated with 0.4 and 1.0 percent DEHP, all the implanted

ova died. At 0.2 percent, a borderline significant increase in defects was observed. The researchers estimated that the maximum no effect level was 70 mg/kg/day, which is far higher than current estimated safe human intake.

Shiota and Mima (1985) found that DEHP was teratogenic in mice if given orally but not if administered intraperitoneally. Doses of 250 to 2,000 milligrams per kilogram (mg/kg) were given on days 7 through 9. Neural tube defects and other malformations were observed at 500 mg/kg. MEHP was not teratogenic when administered orally or intraperitoneally.

ECOTOXICITY

Aquatic Organisms

As reported by Afghan and Chau (1989), evidence shows that aquatic organisms concentrate phthalate esters and are more sensitive to toxic effects than are warmblooded animals. Acute toxicity tests have been conducted on a relatively small group of freshwater fish and invertebrate species; therefore, final acute values have not been determined (EPA, 1980). Only one acute value was reported by EPA (1980) (Table 3), although additional acute toxicity data are presented in Table 3). Chronic test values ranged from <3 μg/L in Daphnia magna to 8.4 μg/L in the rainbow trout (Oncorhynchus mykiss) (Table 3) (EPA, 1980). No final chronic values were determined because of the limited database.

Bioconcentration factors for four species of aquatic animals are presented in Table 4; BCFs ranged from 14 in the sowbug (Asellus brevicaudus) to 2,680 in the scud (Gammarus pseudolimnaeus) (EPA, 1980). Additional studies show that DEHP is rapidly biomagnified by a variety of plants and animals in aquatic systems (Metcalf et al., 1973). At the 10 mg/L level, concentration values ranged from 37 mg/kg in Elodea to 11,873 mg/kg in Culex larvae; at the 0.1-mg/L level, concentration values ranged from 0.73 mg/kg in Culex pupae to

Table 3. Results of Acute and Chronic Toxicity Testing for DEHP

Species	Test Method/Effect*	Effect Level (μg/L)	Species Mean Acute or Chronic Value (μg/L)	Reference+
Acute				
**Cladoceran, Daphnia magna	LC_{50}/EC_{50} (S, U)	11,100	11,100	EPA, 1978 (EPA, 1980)
Cladoceran, Daphnia magna	48 hr	LC50	1,000-5,000	Monsanto, 1978 (EPA, 1980)
Midge, Chironomus plumosus	48 hr	LC50	>18,000	Streufert, 1977 (EPA, 1980)
Scud, Gammarus pseudolimnaeus	96 hr	LC50	>32,000	Sanders et al., 1973 (EPA, 1980)
Rainbow trout, Oncorhynchus mykiss	96 hr	LC50/EC50	540,000	Milanovich et al., 1975 (Afghan and Chau, 1989)
Bluegill, Lepomis machrochirus	96 hr	LC50	>770,000	EPA, 1978 (EPA, 1980)
Chronic				
**Cladoceran, Daphnia magna	LC	<3	<3	Mayer and Sanders, 1973 (EPA, 1980)
**Rainbow trout, Oncorhynchus mykiss	ELS	5-14	8.4	Mehrle and Mayer, 1976 (EPA, 1980)
Rainbow Trout, Oncorhynchus mykiss	24 d	Significant increase in total body protein catabolism	14-54	Mayer et al., 1977 (EPA, 1980)
Guppy, Poecilla reticulata	90 d	Increase in aborted young	100 μg/g in diet	Mayer and Sanders, 1973 (EPA, 1980)

*ELS = early life stage; LC = partial or full life cycle; S = static; U = unmeasured.
+References in parentheses are secondary sources.
**Test results accepted by EPA.

Source: ESE.

Table 4. BCFs for DEHP

Species	Tissue	Bioconcentration Factor*	Duration (days)	Reference
Scud, Gammarus pseudolimnaeus	Whole body	54-2,680+	14-21	Sanders et al., 1973
Sowbug, Asellus brevicaudus	Whole body	14-50+	21	Sanders et al., 1973
Rainbow trout, Oncorhynchus mykiss	Whole body	42-113	36	Mehrle and Mayer, 1976
Fathead minnow, Pimephales promelas	Whole body	155-886	56	Mayer, 1976
Fathead minnow, Pimephales promelas	Whole body	91-569**	56	Mayer, 1976

*Based on total ^{14}C radioactivity accumulated.
**Based on measured concentrations of bis(2-ethylhexyl) phthalate.
+Conversion from dry to wet weight.

Source: EPA, 1980.

132.02 mg/kg in Culex larvae (Table 5). EPA has calculated a weighted-average BCF for DEHP and the edible portion of all freshwater and estuarine aquatic organisms of 130 based on an average measured steady-state BCF of approximately 330 in fathead minnows (EPA, 1980).

Terrestrial organisms

Data concerning toxicity to terrestrial organisms are based on tests using laboratory animals. Results were described previously in the human health effects section.

In a study with mallard ducks reported by Afghan and Chau (1989), dietary levels of 10 mg/kg resulted in no accumulation during the 5-month study period, although low levels (0.15 mg/kg) were found in lung and breast tissue.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 6. Due to the ranking of DEHP as a Group B2 suspect human carcinogen, EPA has proposed a nonenforceable Maximum Contaminant Level Goal (MCLG) for DEHP in drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 4 μg/L, which would be enforceable for public water supplies (55 FR 30370; July 25, 1990).

EPA concluded that current information does not indicate that DEHP endangers public health at ambient concentrations (excluding emergency releases). Therefore, no regulation directed specifically at DEHP is necessary at this time under the Clean Air Act and no national ambient air quality standards (NAAQS) have been developed for DEHP (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American

Table 5. Concentration of DEHP in Organisms in Water Containing 10 and 0.1 mg/L for Various Intervals

		Concentration of B2EHP (mg/L)					
Concentration in Water (mg/L)	Exposure (hr)	Gambusia	Physa	Daphnia	Culex larvae	Culex pupae	Elodea
10	1	152	3,586	592	596	2,272	37
10	6	1,033	4,020*	532	2,634	2,578	293
10	12	1,294*	2,834	893*	5,978	3,144	1,338
10	24	145	2,350	306	11,873*	3,962	1,138
10	48	469*	487	1,551*	3,657	4,346*	290
0.1	1	0.85	12.06	42.1	23.2	0.73	1.98
0.1	6	7.23	45.08	19.61	91.5	1.51	7.72
0.1	12	5.61	45.45	15.54	132.02*	0.97	15.46
0.1	24	8.53	64.35	17.62	31.80	2.03*	27.48*
0.1	48	26.53*	85.75*	18.26*	16.37	--	23.24

*Peak value for particular organism.

Source: Metcalf et al., 1973.

Table 6. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only[a]
Chloroform	4	--	--	--	--	--	--	15,000	50,000	21,000

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is based on the following formula:

Source: ESE.

Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values are presented in the following table of air quality criteria.

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	5 c	5 c	5 c
STEL	10 c	10 c	10 c
CL	--	--	--

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the concentration that should not be exceeded at any any time during a workday. The code "c" designates that this chemical is regulated as a potential carcinogen.

Ecological Receptors

EPA AWQCs for the protection of freshwater and marine organisms are presented in the following table (IRIS, 1994). EPA is considering removing some or all of these values (IRIS, 1994).

AWQC (μg/L)	Freshwater	Marine
Acute	400	400
Chronic	360	360

REFERENCES

Afghan, B.K. and Chay, A.S.Y. (eds.). 1989. Analysis of Trace Organics in the Aquatic Environment. CRC Press, Inc. Boca Raton, FL.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Di(2-Ethylhexyl)phthalate. Prepared by Life Systems Inc. under Contract No. 68-02-4228. Published by Oak Ridge National Laboratory. U.S. Public Health Service, Atlanta, GA.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Carpenter, C.P., Weil, C.S., and Smyth, H.F. 1953. Chronic Oral Toxicity of Di(2-ethylhexyl)phthalate for Rats and Guinea Pigs. Arch. Indust. Hyg. Occup. Med. 8:219-226. (cited in IRIS, 1994).

Ganning, A.E., Brunk, V., and Dallner, G. 1984. Phthalate Esters and Their Effect on the Liver. Hepatology 4(3):541-547. (cited in IRIS, 1994).

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Kozumbo, W.J., Kroll, R., and Rubin, R.J. 1982. Assessment of the Mutagenicity of Phthalate Esters. Environ. Health Perspect. 45:103-109. (cited in IRIS, 1994).

Mayer, F.L. and Sanders, H.O. 1973. Toxiciology of phthalic acid esters in aquatic organisms. Environ. Health Perspect. 3:153.

Mayer, F.L. 1976. Residue dynamics of di-2-ethylhexylphthalate in fathead minnows (Pimephales promelas). Journ. Fish Res. Board Can. 33:2610.

Mayer, F.L. et al. 1977. Collagen Metabolism in Fish Exposed to Organic Chemicals. In: Recent Advances in Fish Toxicology, a Symposium. U.S. Environmental Protection Agency, Corvallis, OR. EPA 600/3-77-085.

Mehrle, P.M. and Mayer, F.L. 1976. Di-2-ethylhexylphthalate: Residue Dynamics and Biological Effects in Rainbow Trout and Fathead Minnows. In: Trace Substances in Environmental Health. University of Missouri Press, Columbia, MO. p.519.

REFERENCES
(Continued, Page 2 of 3)

Metcalf, R.L., Booth, G.M., Schuth, C.K., Hausen, D.J.and Lu, P. 1973. Uptake and Fate of Di-2-ethylhexylphthalate in Aquatic Organisms and in a Model Ecosystem. Environ. Health Perspect., 4:27-34.

Milanovich, F.P., Ireland, R.R., and Wilson, D.W. 1975. Dissolved Yellow Organics: Quantitativeand Qualitative Aspects of Extraction by Four Common Techniques. Environ. Letter, 8(4):337.

Monsanto Industrial Chemicals Co. (Monsanto). 1978. Acute Toxicity of Di-2-ethylhexylphthalate (DEHP) to Daphnia magna. Report No. ES-SS-78-9, St. Louis, MO.

National Toxicology Program (NTP). 1984. Di(2-ethylhexyl)phthalate: Reproduction and Fertility Assessment in CD-1 Mice When Administered by Gavage. Final Report. NTP-84-079. Research Triangle Park, NC. (cited in IRIS, 1994).

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Phillips, B.J., James, T.E.B., and Gangolli, S.D. 1982. Genotoxicity Studies of Di-(2-ethylhexyl)phthalate and its Metabolites in CHO Cells. Mutat. Res. 102:297-304. (cited in IRIS, 1994).

Sanders, H.O. et al. 1973. Toxicity, Residue Dynamics, and Reproductive Effects of Phthalate Esters in Aquatic Invertebrates. Environ. Res. 6:84.

Seed, J.L. 1982. Mutagenic Activity of Phthalate Esters in Bacterial Liquid Suspension Assays. Environ. Health Perspect. 45:111-114. (cited in IRIS, 1994).

Shepard's Catalog of Teratogenic Agents. 1994. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Shiota, K. and Mima, S. 1985. Assessment of the Teratogenicity of Di(2-Ethylhexyl)Phthalate and Mono(2-ethylhexyl)Phthalate in Mice. Arch. Toxicol. 56:263-266. (cited in Shepard, 1994).

REFERENCES
(Continued, Page 3 of 3)

Shiota, K. and Nishimura, H. 1982. Teratogenicity of Di-2-ethylhexyl Phthalate and Di-n-Butyl Phthalate in Mice. Environ. Health Perspect. 45(0):65-70. (cited in IRIS, 1994).

Shiota, K., Chou, M.J., and Nishimura, H. 1980. Embryotoxic Effects of Di-2-Ethylhexyl Phthalate (DEHP) and Di-n-Butyl Phthalate (DBP) in Mice. Environ. Res. 22:245-255. (cited in Shepard, 1994).

Singhe, A.R., Lawrence, W.H., and Autian, J. 1972. Teratogenicity of Phthalate Esters in Rats. Jour. Pharmacol. Sci. 61:51. (cited in IRIS, 1994).

Streufert, J.M. 1977. Some Effects of two phthalic acid esters on the life cycle of the midge (Chironomus plumosus). M.S. Thesis. University of Misssouri, Columbia, MO.

Tomita, I., Nakamura, Y., Aoki, N., and Inui, N. 1982. Mutagenic/Carcinogenic Potential of DEHP and MEHP. Environ. Health Perspect. 45:119-125. (cited in IRIS, 1994).

U.S. Environmental Protection Agency (EPA). 1978. In-depth studies on health and environmental impacts of selected water pollutants. U.S. Environmental Protection Agency. Contract No. 68-01-4646.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-067.

U.S. Environmental Protection Agency (EPA). 1987. Quality Criteria for Water (1986); Update Number 2. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-86-0001.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

CADMIUM

(Inorganic; CAS No. 7440-43-9)

INTRODUCTION

Cadmium, a metal found in a variety of chemical forms in the environment, may occur either naturally or as a waste product. Occurring in nature in the elemental zero valence state or with a valence of +2, cadmium has properties similar to those associated with calcium and zinc. Cadmium, which can form both organic and inorganic compounds, is often found as the salt, cadmium sulfate (Shell, 1990).

More mobile in an aqueous environment than other heavy metals, most cadmium salts may be removed by complexing with organic materials, indicating that cadmium can be adsorbed by soils, particularly clays and soils high in organic content. The overall solubility of cadmium in equilibrium with mineralized soil is primarily controlled by the carbonate and, to a lesser degree, by the hydroxyl ion, with minimum solubility occurring at pH levels of 9 or above; mobility in soils is a function of the ionic strength of nutrients in the soil (Shell, 1990). These factors indicate that, although some forms are insoluble in water, cadmium is generally considered mobile in water systems.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA provides separate chronic reference doses (RfDs) for oral exposure to cadmium in food or water (IRIS, 1994). These values are presented in Table 1. Due to the nutritional essentiality of cadmium, subchronic oral RfDs have not been established. EPA is currently reviewing the data regarding inhalation of cadmium and has not determined inhalation reference concentrations (RfCs) or RfDs (IRIS, 1994; EPA, 1994).

A chronic oral RfD of 0.001 mg/kg/day is listed as pertaining to food. Because

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral, Food					
Chronic	0.01	High	Proteinuria	IRIS, 1994	10 S
Subchronic	--	--	--	--	--
Oral, Water					
Chronic	0.001	High	Proteinuria	IRIS, 1994	10 S
Subchronic	--	--	--	--	--
Inhalation	--	--	--	--	--

Note: IRIS = Integrated Risk Information System.
RfD = reference dose.
-- = Not available.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

the expected absorption value of water is twice the 2.5-percent absorption rate expected from food, a chronic oral RfD of 0.0005 mg/kg/day, one-half the food value, is listed for exposure through water (IRIS, 1994). The inhalation RfC is under review by EPA, and no interim inhalation RfCs are available (IRIS, 1994; EPA, 1994). For the interim subchronic oral reference dose, no value is provided because of the background dietary exposure (EPA, 1994).

In terms of chronic noncarcinogenic effects following either oral or inhalation exposure, cadmium has been shown to cause renal dysfunction in animals and humans. Other chronic toxic effects in humans attributed to cadmium include anemia and pulmonary disease, edema, pneumonitis, possible effects on the endocrine system, defects in sensory function, and bone damage (Hammond and Beliles, 1980). Although still a controversial theory, cadmium has been linked to hypertension in the general human population (Hammond and Beliles, 1980). Chronic (lifetime) exposure of rats to 0.39 mg/kg-bw/day led to decreased survival, renal degeneration and necrosis, cardiac hypertrophy and neurological effects (Schroeder et al., 1963, 1964, 1965). Human oral subchronic effects are not known, but anemia was reported at 2.25 mg/kg-bw/day for rats, and renal necrosis was reported in mice exposed to 0.51 mg/kg-bw/day (EPA, 1985). Subchronic inhalation data were not identified.

Carcinogenicity

Insufficient data are available to classify cadmium as carcinogenic through ingestion. Cadmium has been classified as a B1 (probable human) carcinogen through inhalation, indicating that there is limited evidence of human carcinogenicity from epidemiologic studies and sufficient evidence of carcinogenicity in test animals (IRIS, 1994). Lung cancer was the cancer effect observed most often in human populations. In experimental animals, when exposure was via a mechanism other than direct inhalation, such as intratracheal, intramuscular, or subcutaneous, cancer growths were observed in areas removed from the exposed area, including mammaries and testes, as well as at the injection site. The inhalation cancer slope factor is listed as 6.1 $(mg/kg/day)^{-1}$ (EPA, 1994).

Mutagenicity

Mutagenicity tests are inconclusive, but positive results have occurred in mutation assays (IRIS, 1994). Chromosomal aberrations have been observed in human lymphocytes either treated in vitro or obtained from exposed workers, although the results conflict.

Teratogneicity/Reproductive Effects

No human teratogenic effects are reported, but rats exposed to 10 mg/L, equivalent to 1.7 mg/kg-bw/day, evidenced decreased fertility and increased fetal deaths. EPA (1985), therefore, reports that cadmium is teratogenic.

ECOTOXICITY

Aquatic Organisms

Various species of freshwater aquatic plants showed adverse effects at cadmium concentrations ranging from 2 μg/L for a diatom (Asterionella formosa) to 20,000 μg/L for an alga (Euglena gracilis anabaena) (EPA, 1985). The major toxic effect noted was inhibited growth. Because toxic values for aquatic plants are within or above the ranges of toxic concentrations for fishes and invertebrates, any water quality criteria for cadmium that are considered protective for freshwater animals should also protect freshwater plants.

Ambient cadmium water concentrations exceeding 10 ppb are associated with high mortality, reduced growth, inhibited reproduction, and other adverse effects in freshwater organisms (USFWS, 1985). However, several species of freshwater aquatic insects, crustaceans, and teleosts exhibited significant mortality at cadmium concentrations of 0.8 to 9.9 ppb during exposures of 4 to 33 days, with mortality generally increasing with exposure time and decreased water hardness. Carroll et al. (1979) found that calcium, not magnesium, reduced the acute toxicity of cadmium (EPA, 1985), and Giesy et al. (1977) found that dissolved organics substantially reduced the toxicity of cadmium to daphnids, but had little effect on its toxicity to fish. Different species exhibit different sensitivities to cadmium, and many other

factors might affect the results of tests of the toxicity of cadmium to aquatic organisms (EPA, 1985).

Where hardness is known, LC_{50} or EC_{50} values ranged from 9.9 to 166 μg/L for Daphnia magna, 1.75 to 10.2 μg/L for Oncorhynchus mykiss, and 11.7 to 54.2 μg/L for Pimephales promelas (fry) for various cadmium compounds (EPA, 1985) (see Table 2).

Studies conducted on 12 freshwater fish species and 5 species of freshwater invertebrates showed chronic toxicity values ranging from 0.15 μg/L for Daphnia magna to 156 μg/L for the Atlantic salmon (EPA, 1985).

Freshwater aquatic organisms accumulate cadmium from water containing cadmium concentrations not previously considered hazardous to public health or to many species of aquatic life. BCFs for cadmium in freshwater range from 3 for brook trout muscle to 12,400 for the whole body of mosquitofish (EPA, 1985). BCFs for two plants (Lemna valdiviana and Salvinia natans) were 603 and 960, respectively, for whole body (EPA, 1985). Ferard et al. (1983) demonstrated that algae (Chlorella vulgaris) held 10 days in water containing 10 ppb of cadmium contained 30 ppm dry weight, an increase of 4.5 ppm from the start of the test (BCF = 2550) (USFWS, 1985).

Terrestrial Organisms

Cadmium exposure in birds has resulted in sublethal effects, including growth retardation, anemia, and testicular damage. Mallard ducklings fed 20 ppm dietary cadmium for 12 weeks showed altered blood chemistry and mild to severe kidney lesions, and domestic pigeons developed testicular damage after only one injection of 0.5 ppm-bw of cadmium (see Table 3). In the laboratory,

Table 2. Acute Toxicity of Cadmium to Aquatic Organisms

Species	Method	Chemical	Hardness (mg/L as $CaCO_3$)	LC_{50} or EC_{50} ($\mu g/L$)	Reference*
Daphnia magna (Cladoceran)	S, U	Cadmium chloride	45	65	Biesinger and Christensen, 1972
	FT, M	Cadmium chloride	130	58	Attar and Maly, 1982
	S, M	Cadmium chloride	51	9.9	Chapman, et al., n.d.
	S, M	Cadmium chloride	104	33	Chapman, et al., n.d.
	S, M	Cadmium chloride	105	34	Chapman, et al., n.d.
	S, M	Cadmium chloride	197	63	Chapman, et al., n.d.
	S, M	Cadmium chloride	209	49	Chapman, et al., n.d.
	R, M	Cadmium chloride	100	30	Canton and Sloof, 1982
	S, M	Cadmium chloride	55 to 79	166	Spehar and Carlson, 1984a, b
Oncorhynchus mykiss (Rainbow trout)	S, M	Cadmium chloride	55 to 79	10.2	Spehar and Carlson, 1984a, b
	S, M	Cadmium chloride	39 to 48	2.3	Spehar and Carlson, 1984a, b
	FT, M	Cadmium sulfate	31	1.75	Davies, 1976
Pimephales promelas (Fathead minnow) (fry)	S, M	Cadmium chloride	40	21.5	Spehar, 1982
	S, M	Cadmium chloride	48	11.7	Spehar, 1982
	S, M	Cadmium chloride	39	19.3	Spehar, 1982
	S, M	Cadmium chloride	45	42.4	Spehar, 1982
	S, M	Cadmium chloride	47	54.2	Spehar, 1982
	S, M	Cadmium chloride	44	29.0	Spehar, 1982

Note: FT = flow through. S = static. U = unmeasured.
M = measured. R = renewal.

*References cited are from EPA, 1985.

Source: ESE.

Table 3. Sublethal Effects of Cadmium to Terrestrial Organisms

Species	Concentration	Administration	Effect	Reference*
Japanese Quail	75 ppm Cd	Diet	Bone marrow hypoplasia, anemia, hypertrophy of heart	Richardson et al., 1974
Leghorn Chickens (Adult)	2 ppm $CdSO_4$	Injection	15 to 22 days; anemia, enlarged heart myocardial infarction	Sturkie, 1973
Ring Doves	6.6 ppm-bw Cd	Injection	20 days; testicular damage	Richardson, et al., 1974
Pigeons	0.5 ppm-bw Cd	Injection	Single injection; testicular damage	Sarker and Mandal, 1973
Mallard (Ducklings)	20 ppm Cd	Diet	12 weeks; altered blood chemistry, kidney lesions	Cain et al., 1983

*References cited are from USFWS, 1985.

Source: ESE.

young mice showed altered blood chemistry when fed 1.8 ppm cadmium for 28 days (USFWS, 1985).

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 4. EPA has promulgated an enforceable (for public water supplies) Maximum Contaminant Level (MCL) of 0.005 mg/L (IRIS, 1994). Based on potential adverse effects (renal toxicity) EPA has promulgated the MCL Goal (MCLG) at this same level. The MCL/MCLG is based upon a Drinking Water Equivalent Level (DWEL) of 0.018 mg/L and an assumed drinking water contribution (including aquatic organisms) of 25 percent. An uncertainty factor of 10 was also applied (IRIS, 1994). In addition, EPA has established an ambient water quality criterion (AWQC) for human consumption of water and aquatic organisms of 10 ug/L for cadimum (IRIS, 1994). No AWQC for the consumption of aquatic organisms alone has been set.

No national ambient air quality standards (NAAQS) have been developed for cadmium under the Clean Air Act (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to cadmium (NIOSH, 1994). In addition, the American Conference of Governmental Industrial Hygienists (ACGIH) has developed nonenforceable threshold limit values (TLVs), (ACGIH, 1993). These values are presented in Table 5. The National Institute of Occupational Safety and Health (NIOSH) has not developed recommended exposure levels (RELs); however, this group does consider cadmium as a human carcinogen (NIOSH, 1994).

Ecological Receptors

AWQC for the protection of freshwater and marine organisms against exposure to copper have been established by EPA (IRIS, 1994) are presented in Table 6.

Table 4. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only
Cadmium	5	5	--	--	--	--	--	10	--	--

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

Source: ESE.

Table 5. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	0.005 (Dust) 0.1 (Fumes)	c	0.01 c (Total dusts) 0.002 c (Respirable dust)
STEL	--	--	--
CL	0.3 (Fumes)	--	0.05 c (Fumes)

Note:
TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.
STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.
CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.
c = designates that this chemical is regulated as a potential carcinogen.

Source: ESE.

Table 6. Ambient Water Quality Criteria

AWQC (μg/L)	Freshwater	Marine
Acute	3.9[a]	43
Chronic	1.1[b]	9.3

[a]The acute freshwater AWQC for cadmium is hardness-dependent. The 1-hour average concentration in μg/L of cadmium should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(1.128 * [\ln(hardness)] - 3.828)}$$

[b]The chronic freshwater AWQC for cadmium is hardness-dependent. The 4-day average concentration in μg/L of cadmium should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(0.7852 * [\ln(hardness)] - 3.49)}$$

At a hardness of 50 mg/L as $CaCO_3$, the acute and chronic criteria are 9.2 and 6.5 μg/L, respectively (IRIS, 1994).

Source: ESE.

REFERENCES

Attar, E.N. and Maly, E.J. 1982. Acute Toxicity of Cadmium, Zinc, and Cadmium-Zinc Mixtures to Daphnia magna. Arch. Environ. Contam. Toxicol. 11:291. (cited in EPA, 1985).

Biesinger, K.E. and Christensen, G.M. 1972. Effect of Various Metals on Survival, Growth, Reproduction, and Metabolism of Daphnia magna. Fish. Res. Board Can. 29:1691. (cited in EPA, 1985).

Cain, B.W., Sileo, L., Franson, J.C., and Moore, J. 1983. Effects of Dietary Cadmium on Mallard Ducklings. Environ. Res. 32:286-297. (cited in USFWS, 1985).

Carroll, J.J., Ellis, S.J., and Oliver, W.S. 1979. Influences of Hardness Constituents on the Acute Toxicity of Camium to Brook Trout (salvelinus fontinalis). Bull. Environ. Contam. Toxicol. 22:575-581. (cited in USFWS, 1985).

Chapman, G.A., Ota, S. and Recht, F. n.d. Effects of Water Hardness on the Toxicity of Metals to Daphnia magna. Available from: C.E. Stephan, U.S. EPA, Duluth, MN. Cited in EPA, 1987.

Davies, P.H. 1976. Use of Dialysis Tubing in Defining the Toxic Fractions of Heavy Metals in Natural Waters. In: R.W. Andrew, et al. (eds.), Toxicity to Biota of Metal Forms in Natural Water. International Joint Commission, Windsor, Ontario. p. 110. (cited in EPA, 1985).

Ferard, J.F., Jouany, J.M., Truhaut, R., and Vasseur, P. 1983. Accumulation of Cadmium in a Freshwater Food Chain Experimental Model. Ecotoxicol. Environ. Safety 7:43-52. (cited in USFWS, 1985).

Giesy, J.P., Leversee, G.J., and Williams, D.R. 1977. Effects of Naturally Occurring Aquatic Organic Fractions on Cadmium Toxicity to Simocephalus serrulatur (Daphnidae) and Gambusia affinis (Poeciliidae). Water Res. 11:1013-1020. (cited in USFWS, 1985).

Hammond, P.B. and Beliles, R.P. 1980. Metals. In: Toxicology, the Basic Science of Poisons. 2nd Ed. Doull, J., Klaassen, C.D., and Amdur, M.O., Eds. MacMillan Publishing Co., NY.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

REFERENCES
(Continued, Page 2 of 5)

National Toxicology Program (NTP). 1985. Fiscal Year 1985 Annual Plan. Public Health Service, U.S. Department of Health and Human Services, Washington, DC. NTP-85-055.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Richardson, M.E., Fox, M.R.S., and Fry, B.E. Jr. 1974. Pathological Changes Produced in Japanese Quail by Ingestion of Cadmium. Jour. Nutr. 104:323-338. (cited in USFWS, 1985).

Sarkar, A.K. and Mondal, R. 1973. Injurious Effect of Cadmium on Testis of Domestic Pigeon and Its Prevention by Zinc. Indian Jour. Exp. Biol. 11:108-109. (cited in USFWS, 1985).

Schroeder, H.A., Vinton, W.H., and Balassa, J.J. 1963. Effects of Chromium, Cadmium and Lead on the Growth and Survival of Rats. Jour. Nutr. 80:48-54.

Schroeder, H.A., Balassa, J.J., and Vinton, W.H., Jr. 1964. Chromium, Lead, Cadmium, Nickel and Titanium in Mice: Effect on Mortality, Tumors and Tissue Levels. Jour. Nutr. 83:239-250.

Schroeder, H.A., Balassa, J.J., and Vinton, W.H., Jr. 1965. Chromium, Cadmium and Lead in Rats: Effect on Life Span, Tumors and Tissue Levels. Jour. Nutr. 86:51-66.

Shell Chemical Company (Shell). 1990. Shell Toxicity Profile. In: Human Health Exposure Assessment for Rocky Mountain Arsenal, Toxicity Assessment, Vols. IIa and IIIa. Prepared by Ebasco Services, Inc.

Spehar, R.L. 1982. Memorandum to J.G. Eaton. USEPA, Duluth, Minnesota. February 24. (cited in EPA, 1985).

Spehar, R.L. and Carlson, A.R. 1984a. Derivation of Site-Specific Water Quality Criteria for Cadmium and the St. Louis River Basin, Duluth, Minnesota. Environ. Toxicol. Chem. 3:651. (cited in EPA, 1985).

REFERENCES
(Continued, Page 3 of 5)

Spehar, R.L. and Carlson, A.R. 1984b. Derivation of Site-Specific Water Quality Criteria for Cadmium and the St. Louis River Basin, Duluth, Minnesota. PB84-153196. (cited in EPA, 1985).

Sturkie, P.D. 1973. Effects of Cadmium on Electrocardiogram, Blood Pressure, and Hematocrit of Chickens. Avian Dis. 17:106-110. (cited in USFWS, 1985).

U.S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. Prepared by Clement Associates, Inc. Arlington, VA.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

U.S. Fish and Wildlife Service (USFWS). 1985. Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Biological Report 85(1.2). Patuxent Wildlife Research Center, Laurel, MD.

CARBON TETRACHLORIDE
(CAS No. 56-23-5)

INTRODUCTION

Widely used as an industrial solvent, dry cleaning agent, and chemical intermediate, carbon tetrachloride was banned for these purposes by FDA. Currently, it is primarily used in the synthesis of chlorofluoromethanes and as a grain fumigant and pesticide (ORNL, 1989).

Carbon tetrachloride, with a reported water solubility of 780 to 930 mg/L, may be considered soluble (Davies and Dobbs, 1984; Rodgers et al. 1980; Valvani et al., 1980). When considered in conjunction with the high solubility, the moderate soil sorption coefficient (K_{oc}) value of 439 (ORNL, 1989) suggests that carbon tetrachloride will be mobile in the soil/groundwater system, although there will be considerable retardation in subsurface or sandy soils. Based on the reported vapor pressure of 90 torr at 20°C (EPA, 1979), transport through the air-filled pores of the near-surface soils may be an important migration pathway. In surficial and near-surface unsaturated soils, an equilibrium partitioning model (ORNL, 1989) indicates that 96 percent of the carbon tetrachloride present will be sorbed to the soil with about 1 percent in the soil-water phase and 3 percent in the soil-air phase. In saturated soils (where soil organic carbon and soil air are negligible), a majority of the carbon tetrachloride (about 65 percent) is still expected to be sorbed to the soil with the balance in the soil-water phase, which can be transported with groundwater.

Because the compound is volatile, the primary fate of carbon tetrachloride is associated with the atmosphere. Photolysis is not an important degradation process in the lower troposphere as photodissociation occurs with light wavelengths shorter than 290 nm, which is found only in the stratosphere above the ozone layer; the estimated tropospheric half-life is 330 years (EPA, 1979). No reaction occurs with hydroxyl radicals, and only in the stratosphere is the compound degraded by the higher energy

ultraviolet light to form trichloromethane radicals and chlorine atoms. The former oxidize to phosgene, which is further photodissociated to form more chlorine atoms; the latter are reported to act as catalysts in the destruction of the ozone layer (EPA, 1979). The overall anticipated atmospheric lifetime is 60 to 100 years (EPA, 1984).

Carbon tetrachloride does not undergo rapid hydrolysis under normal environmental conditions; the estimated aqueous hydrolytic half-life for 1,000 mg/L at pH 7 and 25°C is 7 years, and 7,000 years for 1 mg/L (EPA, 1979). Allowing for volatilization, the half-life in a stirred solution is 29 minutes; environmental half-lives are estimated at 1.2 days in a river, 4.8 days from a lake, and 5.8 days from a pond (ORNL, 1989). Microbial degradation is not expected to occur except in treatment systems where the microbes have been acclimatized to the compound (ORNL, 1989). Although carbon tetrachloride is slightly lipophilic and tends to be found at higher concentrations in fatty tissue, no clear evidence of biomagnification exists (EPA, 1979).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) are available from EPA (IRIS, 1994; EPA, 1994) and are presented in Table 1. No inhalation reference concentrations (RfCs) or RfDs are provided for either chronic or subchronic exposure. EPA provides a chronic oral RfD of 0.0007 mg/kg/day (IRIS, 1992) and an interim subchronic oral RfD of 0.007 mg/kg-bw/day (HEAST, 1992).

The chronic oral and interim subchronic oral RfDs are based on a study by Bruckner et al. (1986) in which liver lesions were observed in rats gavaged for 12 weeks at 10 and 33 mg/kg-bw/day (IRIS, 1994). The 10 mg/kg-bw/day (converted to 7.1 mg/kg-bw/day because exposure occurred only 5 days/week) was determined to be the lowest observed adverse effect level (LOAEL), while

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	0.0007	Medium	Liver lesions	IRIS, 1994	1,000 C,H,S
Subchronic	0.007	Medium	Liver lesions	EPA, 1994	100 H,S
Inhalation					
Chronic	--	--	--	--	--
Subchronic	--	--	--	--	--

Note: RfD = reference dose.
-- = No EPA Review.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

the lowest dose of 1 mg/kg-bw/day (converted to 0.71 mg/kg-bw/day) was determined to represent the no observed adverse effect level (NOAEL). Applying an uncertainty factor of 100 (10 for animal-to-human extrapolation and and 10 for sensitive human subpopulations) results in an interim subchronic oral RfD of 0.0071 mg/kg/day. Applying an additional uncertainty factor of 10 to extrapolate from a subchronic study to chronic exposure results in a chronic oral RfD of 0.00071 mg/kg/day.

Minimal oral chronic or subchronic human exposure data are available, although reports of acute toxicity exist as a result of accidental, medicinal, or suicidal ingestion. The major pathological effects are liver and kidney damage, with death often attributable to acute renal or hepatic failure (Shell, 1990). Complete recovery of renal function from a mild case may take from 100 to 200 days with oliguria reported as the major effect; however, in a more serious poisoning, anuria may occur, leading to hypertension, acidosis, and terminal uremia if renal function is not restored (Goodman and Gilman, 1985). Concurrent CNS symptoms include dizziness, headache, confusion, and delirium (Shell, 1990).

EPA (1984) presents oral data from a carcinogenicity bioassay study involving hamsters in which a gavage dose of 12.26 mg/week for 30 weeks resulted in a 50-percent mortality rate. Because weight gain was depressed following subchronic inhalation exposure to 1 ppm for 90 days, an atmospheric concentration of 1 ppm was established as the LOAEL. At higher doses, liver damage and increased mortality were reported. Chronic human inhalation exposure led to optic nerve damage and degeneration of the myelin sheath of the sciatic nerve. No liver or kidney damage were reported. Animals evidenced hepatomegaly following chronic exposure to atmospheric levels as low as 5 ppm.

Carcinogenicity

The carcinogenic slope factors (CSFs) and supporting information are summarized in Table 2. EPA (IRIS, 1994) has classified carbon tetrachloride as a

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.13	B2	Liver tumors	IRIS, 1994
Inhalation	0.053[a]	B2	Liver tumors	EPA, 1994

Note: B2 = suspect human carcinogen (adequate evidence of carcinogenicity in animals and insufficient evidence in humans).

[a]Inhalation CSF is based on an inhalation unit risk (UR) of 1.5×10^{-5} $(\mu g/m^3)^{-1}$ (IRIS, 1994) and assumes that a healthy 70-kilogram adult inhales 20 m^3 of air per day.

Source: ESE.

group B2 (suspect human) carcinogen via ingestion and inhalation. This classification indicates adequate evidence exists to show carcinogenicity in animals based on carcinogenic responses reported in various animal studies following ingestion and inhalation of carbon tetrachloride (ATSDR, 1989; IRIS, 1994). Isolated observations of liver cancer in humans exposed to carbon tetrachloride have been reported; however, no epidemiological support is available, rendering the human data inadequate (EPA, 1984).

EPA derived an oral cancer slope factor (CSF) of 0.13 $(mg/kg/day)^{-1}$ based on the results of several animal studies (IRIS, 1994). Liver cell carcinomas were the major cancer reported in several species, with investigators theorizing that the necrotizing action on the liver was an important factor in carcinogenicity. The oral slope factor determined by EPA from the available data is 0.13 $(mg/kg/day)^{-1}$ (IRIS, 1994).

An inhalation unit risk (UR) was calculated assuming a 40 percent absorption rate in humans (EPA, 1984). This absorption coefficient was based on 30 percent absorption in monkeys, and 30 percent and 57 to 65 percent absorption in humans. A range of estimates of UR for inhalation exposures for the four studies cited in IRIS (1994) was determined, with 1.5×10^{-5} $(\mu g/m^3)^{-1}$ calculated as the geometric mean for the UR. Assuming a healthy 70-kilogram adult inhales 20 m^3/day of air, the inhalation UR may be converted to an inhalation CSF of 0.053 $(mg/kg/day)^{-1}$. The UR (or derived CSF) should not be used if the air concentration exceeds 700 $\mu g/m^3$, since above this concentration the UR may not be appropriate.

Mutagenicity

With only one positive mutagenic response elicited in a Saccharomyces cerevisiae strain (HSDB, 1994), the available information suggests that this is not a mutagenic compound; insufficient data are available to establish genotoxicity. EPA (IRIS, 1994) reports that no chromosomal or chromatid aberrations were seen in cells exposed to low concentrations, and in vivo, unscheduled DNA synthesis assays were negative. Mitotic recombination and gene conversion were reported but only at concentrations

that reduced cell viability to 10 percent. EPA (IRIS, 1994) indicates that the possibility remains that carbon tetrachloride may be metabolized to more reactive intermediate compounds that could be mutagenic. Hepatic abnormalities and retarded development were reported at birth in rats exposed in utero (HSDB, 1994). Rats given intraperitoneal injections of carbon tetrachloride at 4,800 mg/kg for 10, 15, or 20 days evidenced impairment of spermatogenesis (HSDB, 1994). Decreased sex organ weights and decreased gonadosomatic index were reported in all exposed animals, while increasing cellular damage was reported in the animals exposed for 15 and 20 days (HSDB, 1994).

Teratogenicity/Reproductive Effects

No reproductive data have been identified in the available literature. Based on numerous animal studies, the evidence indicates that embryotoxicity and fetotoxicity occur at levels that are also maternotoxic (ReproText, 1994). No teratogenic effects have been reported at levels known to be feto- or maternotoxic. Reproductively, carbon tetrachloride has been reported to prolong the estrous cycle, cause testicular atrophy, and a diminished sperm count in rats (ReproText, 1994). In rabbits, the only effect reported is limited degeneration of embryonic discs following the in vivo exposure of blastocysts to 1.01 mL/kg (Shepard, 1994).

ECOTOXICITY

Aquatic Organisms

Initially, in aquatic systems, levels as low as 35,000 μg/L were judged acutely toxic to fish in bioassays; however, the fact that the tests were static and that this is a very volatile compound suggest that carbon tetrachloride toxicity may have been underestimated and is in fact more toxic (Shell, 1990). Following the exposure of newly hatched fish, the LC_{50} at 4 days posthatching was 1,970 μg/L for rainbow trout and 1,640 μg/L for the leopard frog (Shell, 1990). In addition, it has been estimated that concentrations as low as 30 μg/L would adversely affect sensitive aquatic species (Shell, 1990). The lack of adequate data suggests that the LC_{50} value reported for the Leopard frog is more appropriate for deriving an acceptable water concentration.

Therefore, applying an uncertainty factor of 100 to the reported LC_{50} value of 1,640 μg/L, a water TRV of 0.016 mg/L (16 μg/L) is calculated.

Terrestrial Organisms

No information was available concerning the effects of carbon tetrachloride to vegetation; therefore, there is insufficient information from which to derive a vegetation TRV for either soil or water. In domestic animals, as in the human population, carbon tetrachloride was used as a trematodicide in the early 1920s. Acute toxicity was associated with CNS effects. Delayed toxicity effects are related to hepatic and, to a lesser extent, renal damage (Roberson, 1977). While effects were reported in swine exposed to levels of 320 mg/kg-bw, cattle were the most sensitive as an exposure level of 20 mg/kg-bw was reported to be acutely toxic; this represents an acute LOAEL. No information concerning wildlife was found in the available literature.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 3. Due to the ranking of carbon tetrachloride as a Group B2 suspect human carcinogen, EPA has set a nonenforceable Maximum Contaminant Level Goal (MCLG) for drinking water of zero μg/L (50 FR 46880; 13 Nov 1985) and a Maximum Contaminant Level (MCL) of 5 μg/L, which is enforceable for public water supplies (52 FR 25690; 08 Jul 1987). Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels

Table 3. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day[a]	10-Day[a]	Longterm[a]	Lifetime	Fish and Water	Fish Only	Water Only[b]
Carbon tetrachloride	5	0	--	4,000	160	71	--	0.4[c]	6.94[c]	0.42[c]

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is for a 10-kg child.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

range from 4 to 0.04 μg/L for consumption of contaminated drinking water and aquatic organisms (IRIS, 1994). For consumption of aquatic organisms only, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 69.4 to 0.694 μg/L, respectively (IRIS, 1994). These AWQC would yield values for human consumption of water alone (W) of 4.2, 0.42, and 0.042 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on the following equation:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

No national ambient air quality standards (NAAQS) have been developed for carbon tetrachloride under the Clean Air Act (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to mercury (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values are presented in the Table 4.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms. However, chronic and acute lowest observed effect levels (LOELs) have been reported by EPA and are presented in Table 5 (IRIS, 1994).

Table 4. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	12.6 c	--	31 c,s
STEL	--	12.6 c (60 min)	63 c,s
CL	--	--	--

Note:
TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.
STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.
CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.
c = designates that this chemical is regulated as a potential carcinogen.
s = signifies that dermal absorption may be a significant exposure route for this chemical.

Source: ESE.

Table 7. Ambient Water Quality Criteria

LOEL (µg/L)	Freshwater	Marine
Acute	35,200	50,000
Chronic	--	--

Source: ESE.

REFERENCES

Bruckner, J.V., MacKenzie, W.F., Muralidhara, S., Luthra, R., Kyle, G.M., and Acosta, D. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6(1):16-34. (cited in IRIS, 1994).

Davies, R.P., and Dobbs, A.J. 1984. The Prediction of Bioconcentration in Fish. Water Research, 18:1253-62.

Goodman, L.S. and Gilman, A. 1985. The Pharmacological Basis of Therapeutics. The Macmillan Co., Inc., New York, NY.

Hazardous Substances Data Bank (HSDB). 1994. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

ReproText®. 1994. B.J. Dabney. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Roberson, E.L. 1977. Antinematodal Drugs. In: Veternary Pharmacology and Theraputics. 4th Ed. Jones, L.M., Booth, N.H., and McDonald, G.E., Eds. Iowa State University Press, Ames, IA.

Rodgers, R.D., J.C. McFarlane, and A.J. Cross. 1980. Adsorption and Desorption of Bensene in Two Soils and Montmorillonite Clay. Environ. Sci. Technol. 14(4):457-460.

REFERENCES
(Continued, Page 2 of 2)

Shell Chemical Company (Shell). 1990. Shell Toxicity Profile. In: Human Health Exposure Assessment for Rocky Mountain Arsenal, Toxicity Assessment, Vols. IIa and IIIa. Prepared by Ebasco Services, Inc.

Shepard's Catalog of Teratogenic Agents. 1994. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Environmental Protection Agency (EPA). 1979. Water-Related Fate of 129 Priority Pollutants. Volume II. Monitoring and Data Support Division, Washington, DC. EPA-440/4-79-029B.

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Carbon Tetrachloride. Environmental Criteria and Assessment Office. EPA/540/1-86/039. NTIS No. PB86-134509.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

Valvani, S.C., Yalkowsky, S.H., and Roseman, T.J. 1980. Solubility and Partitioning. IV. Aqueous Solubility and Octanol-Water Partition Coefficients of Liquid Nonelectrolytes. J. Pharm. Sci. 70(5):502-505.

CHLOROBENZENE
(CAS No. 108-90-7)

INTRODUCTION

Chlorobenzene, also referred to as monochlorobenzene, is most commonly used as a solvent and as a raw material in chemical manufacturing. Although moderately volatile (vapor pressure is 8.8 torr at 20°C), it is soluble in water, with reported values ranging from 300 to 625 mg/L (Tewari et al., 1982; Valvani et al., 1980). With K_{oc} values ranging from 330 to 1,930, sorption to soil may be significant.

Although the data are not definitive, hydrolysis is not expected to occur at a rate competitive with volatilization or degradation in surface water (ORNL, 1989). Degradation can occur at a significant rate, particularly if chlorobenzene is exposed to a sludge in which the microbial population has been acclimated to benzene (ORNL, 1989).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

A chronic oral reference dose (RfD) for chlorobenzene is available from EPA (IRIS, 1994) and is presented in Table 1. EPA is re-evaluating the chronic inhalation reference concentration (RfC) for chlorobenzene and does not provide a value in IRIS (1994). However, EPA does provide an interim chronic RfC and inhalation RfD (EPA, 1994), which are listed in Table 1. Subchronic RfDs and RfCs are not available in IRIS (1994) or HEAST (EPA, 1994).

The chronic oral RfD was derived from a 13-week study in which dogs were given chlorobenzene orally by capsule at doses of 27.25, 54.5, or 272.5 mg/kg/day (IRIS, 1994). The lowest dose, 27.25 mg/kg/day, was identified as the NOAEL, and the next higher dose, 54.5 mg/kg/day, was established as the LOAEL. Effects seen at this dose included slight bile duct

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	0.02	Medium	Liver Histopathology	IRIS, 1994	1,000 C,H,S
Subchronic	--	--	--	--	--
Inhalation					
Chronic	0.005[c]	--	Liver/ Kidney	EPA, 1994	10,000 C,H,N,S
Subchronic	--	--	--	--	--

Note: IRIS = Integrated Risk Information System.
RfD = reference dose.
RfC = reference concentration.
-- = Not available.
LOAEL = lowest observed adverse effect level.
NOAEL = no observed adverse effect level.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
H = to extrapolate from an animal study to humans.
N = to extrapolate from a LOAEL to a NOAEL.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.
[c]Inhalation RfD based on an RfC of 0.02 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

proliferation, cytologic alterations, and leukocytic infiltration of the stroma, all in the liver. The results of other studies support these NOAEL and LOAEL values.

No reports of subchronic or chronic oral human exposures are available in the literature (EPA, 1984). No subchronic human inhalation data are available; however, factory workers exposed to unknown levels of chlorobenzene for 1 to 2 years experienced numbness and stiffness of the extremities as well as uncontrolled movements of the fingers (EPA, 1984).

In subchronic oral studies involving laboratory animals (IRIS, 1994), male rats and mice experienced minimal liver effects at 250 mg/kg (converted to 178.6 mg/kg/day based on exposure rates) for 13 weeks. Dogs showed no effects after exposure to 27.3 mg/day for 13 weeks (IRIS, 1994). In developing an LD_{50} value of 3,400 mg/kg-bw for rats, the reported toxic effects included liver necrosis and interference with porphyrin metabolism (USATHAMA, 1989). In a 13-week study, mice receiving dietary doses of chlorobenzene of 42.9 mg/kg/day evidenced hepatic necrosis (USATHAMA, 1989); this value represents an LOAEL. In long-term studies with rats, dietary ingestion at 50 mg/kg/day for between 90 and 93 days led to increased liver and kidney weights, and the results of other studies indicated no effects in rats at 50 mg/kg/day or in either mice or rats at 60 mg/kg/day (USATHAMA, 1989). Therefore, the value of 50 mg/kg/day represents an LOAEL for rats. Although no chronic inhalation data are available for experimental animals, dogs were the most sensitive; subchronic exposure (31 days) to a level of 31.6 mg/kg-bw led to weight loss and moribundity (EPA, 1984).

Carcinogenicity

EPA has classified chlorobenzene as a Group D carcinogen, not classifiable as to human carcinogenicity based on the lack of human data, inadequate animal data and predominantly negative genetic toxicity data in bacterial, yeast, and mouse lymphoma cells.

No human studies and only one animal study, an NTP (1985) gavage study in rats and mice have been performed. Fifty animals/sex/species/dose were administered chlorobenzene (>99% pure) by gavage 5 days/week for 103 weeks. Fifty animals/sex/species served as untreated and vehicle controls. Only the high-dose male rats (120 mg/kg/day) had a statistically significant elevation in mortality relative to their vehicle controls. No chlorobenzene-related signs of clinical toxicity were observed in the rats during the experiment; histological examination of the liver showed hepatocellular necrosis (graded as minimal to mild in all groups) caused by chlorobenzene. A statistically significant positive trend in the incidence of hepatocellular neoplastic nodules was observed in male rats. No hepatocellular tumors were observed in dosed male rats and none of the high-dose males with neoplastic nodules showed signs of hepatocellular necrosis. There was no increase in neoplastic nodules or hepatocellular carcinomas in females; however, there was a single incidence of hepatocellular carcinoma. No other significant increase in site-specific tumors or neoplastic pathology was observed in rats.

No difference in survival rate between dosed and control female mice was noted. The mortality in dosed male mice, however, was significantly increased. No chlorobenzene-related clinical toxicity and no significant increase in site-specific tumors or neoplastic pathology were observed in any mouse group.

In 1986, the Environmental Health Committee of the Science Advisory Board reviewed this study. Overall, the Committee considered the study to be of good quality, but the Comittee questioned the biological significance of the increased incidence of neoplastic liver nodules (Doull and Abrahamson, 1986). The NTP report does not specify the number of liver sections pathologically examined or which sections of the liver were examined; the location of the liver sections could cause the difference in the number and the diagnosis of lesions formed. Liver nodules are currently not considered necessarily to be progressive, and consequently lethal to the host. In this study, there was no incidence of hepatocellular carcinoma in male rats after 2 years of the study.

Mutagenicity

Mutagenicity tests indicate that chlorobenzene affects some, but not all, microorganisms. Negative results were obtained in mutagenicity studies in sex-linked recessive lethality tests and in chromosome aberration tests, while positive evidence of sister chromatid exchange was reported (NTP, 1986). When rats and rabbits were exposed to concentrations as high as 590 ppm for 6 hours daily during organogenesis, a few rabbit fetuses were found with ablepharia and forelimb flexure, but the total malformations were not increased significantly (ReproText, 1994). No adverse fetal effects were reported in rat progeny.

Teratogenicity/Reproductive Effects

No human epidemiological reproductive or developmental toxicity studies are reported in the literature (ReproText, 1994; TERIS, 1994; Shepard, 1994). A limited quantity of experimental data is available. John et al. (1984) exposed rats and rabbits for six hours/day during organogenesis to concentrations of up to 590 ppm. At the highest dose in rabbits a few fetuses with ablepharia (partial or complete absence of eyelids) and fore-limb flexure were found, but the total malformations were not increased significantly. No adverse fetal effects were identified in the rats.

ECOTOXICITY

Aquatic Organisms

In the alga, Selenastrum capricornatum, a 96-hour EC_{50} was established as 228 μg/L, as evidenced by a reduction in cell number and in chlorophyll a production (EPA, 1980). The 96-hour EC_{50} for growth inhibition was determined to be 12.5 μg/L, and the NOEL was 6.8 μg/L (USATHAMA, 1989). In a separate study, a concentration of 120 μg/L caused incipient growth inhibition of Mycrocystis aeruginosa. Chlorobenzene was toxic to embryonic largemouth bass, with a subchronic LC_{50} of 60 μg/L reported (USATHAMA, 1989). EPA (1980) reports an acute LOEC of 250 μg/L for chlorinated benzenes. Applying an uncertainty factor of 100 to the EPA-derived acute LOEC value provides a water TRV of 2.5 μg/L. This value is

expected to be protective not only of embryonic bass but other aquatic organisms as well.

Terrestrial Organisms

No data were identified describing the toxicity of chlorobenzene to vegetation or wildlife; it was, therefore, not possible to derive either a soil or water TRV for chlorobenzene.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 2. EPA has promulgated an enforceable (for public water supplies) Maximum Contaminant Level (MCL) and a nonenforceable MCL Goal (MCLG) for chlorobenzene in drinking water of 100 μg/L (56 FR 3526). These values are based on a Drinking Water Equivalent Level (DWEL) of 700 μg/L and an assumed drinking water contribution of 20 percent.

Although inhalation appears to be the primary exposure route for chlorobenzene, EPA concluded that current information does not indicate that chlorobenzene endangers public health at ambient concentrations (excluding emergency releases). Therefore, no regulation directed specifically at chlorobenzene is necessary at this time under the Clean Air Act and no national ambient air quality standards (NAAQS) have been developed for chlorobenzene (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) often developed nonenforceable recommended exposure

Table 2. Drinking Water Criteria and Standards (mg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only
Chlorobenzene	100	100	--	--	--	--	--	4.88	--	--

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.

Source: ESE.

levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values, if available, are presented in the following table of air quality standards.

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	350	--	46
STEL	--	--	--
CL	--	--	--

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the concentration that should not be exceeded at any any time during a workday.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to chlorobenzene (IRIS, 1994). However, the LOELs for the aquatic species tested have been determined (IRIS, 1994). These LOELs, which are established when the minimum data required to derive AWQCs are not available, are presented in the following table.

LOEL[a] (μg/L)	Freshwater	Marine
Acute	250	160
Chronic	50	129

[a]LOELs for chlorinated benzenes as a class of chemicals.

REFERENCES

Doull, J. and Abrahamson, S. December 16, 1986. Personal Communication, Memorandum to R.A. Griesemer, Chair Environmental Health Committee of Science Advisory Board, EPA, re: Technical comments of the Halogenated Organics Subcommittee on EPA's Draft Drinking Water Criteria Document for Monochlorobenzene. Chair and Vice-Chair of Halogenated Organics Subcommittee of Science Advisory Board, EPA. Washington, DC.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

John, J.A., Hayes, W.C., Hanley, T.R., Jr., Johnson, K.A., Gushow, T.S., and Rao, K.S. 1984. Inhalation teratology study of monochlorobenzene in rats and rabbits. Toxicol. Appl. Pharmacol. 76:365-373. (cited in Shepard, 1984).

National Toxicology Program (NTP). 1985. Toxicology and carcinogenesis studies of chlorobenzene (CAS No. 108-90-7) in F344/N rats and B6C3F1 mice (gavage studies). U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NTP No. 261. NIH Publ. No. 83-2517.

National Toxicology Program (NTP). 1986. Fiscal Year 1986 Annual Plan. U.S. Department of Health and Human Services. Public Health Service. Washington, DC. NTP-85-086.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

Reproductive Risk Information System (ReproText®). 1994. B.J. Dabney. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Shepard's Catalog of Teratogenic Agents. 1994. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

REFERENCES
(Continued, Page 2 of 2)

Teratogen Information System (TERIS). 1994. University of Washington, Seattle, WA. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Tewari, Y.B., Miller, M.M., Wasik, S.P, and Martire, D.E. 1982. Aqueous Solubility and Octanol-Water Partition Coefficient of Organic Compounds at 25°C. Jour. Chem. Eng. Data.

U.S. Army Toxic and Hazardous Materials Agency (USATHAMA). 1989. Biota Remedial Investigation. Final Report, Vol. II. Prepared for Office of the Program Manager, Rocky Mountain Arsenal Contamination Cleanup. AMXRM Aberdeen Proving Ground, MD.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Chlorobenzene. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA/440/5-80/028.

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Chlorobenzene. Environmental Criteria and Assessment Office. EPA/540/1-86/040. NTIS No. PB86-134517.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

Valvani, S.C., Yalkowsky, S.H., and Roseman, T.J. 1980. Solubility and Partitioning. IV. Aqueous Solubility and Octanol-Water Partition Coefficients of Liquid Nonelectrolytes. Jour. Pharm. Sci. 70(5):502-505.

CHLOROFORM
(CAS No. 67-66-3)

INTRODUCTION

Chloroform (trichloromethane) is produced during the chlorination of drinking water and is a common contaminant in potable water supplies. Chloroform was used for several years as an anesthetic but is now used in this capacity only in emergencies and, to some degree, in the tropics, where it is favored over some of the more volatile compounds, such as ether.

Chloroform is a volatile compound (vapor pressure is 151 torr at 20°C) and quickly moves from surface water systems to the atmosphere, where it is attacked by hydroxyl radicals, forming phosgene that is subsequently hydrolyzed to HC, CO_2 and chlorine oxide radicals that are not likely to persist (EPA, 1980). Half-lives are reported as 1.2 days in a river, 6.2 days in a pond, and 13 days in a lake; the half-life is less than 30 minutes in a stirred aqueous solution (ORNL, 1989).

Volatilization from surficial and near-surface soils is reported to be slower by about one order of magnitude (ORNL, 1989). While airborne chloroform will undergo photooxidation, it does not undergo rapid hydrolysis under normal environmental conditions. Chloroform is not believed to undergo microbial degradation except in acclimated water treatment systems and in active landfills (ORNL, 1989). While chloroform on the soil surface is likely to volatilize, based on the water solubility of 8,200 mg/L at 20°C and the soil sorption coefficient (K_{oc}) value of 44, any remaining portion will most likely be leached to groundwater (ORNL, 1989).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) are available from EPA (IRIS, 1995; EPA, 1994) and are presented in Table 1. Inhalation reference concentrations (RfCs) are currently under review by an EPA Work Group and no inhalation RfCs or RfDs are provided in IRIS (1995) or HEAST (EPA, 1994).

The chronic oral RfD (and interim subchronic oral RfD) of 0.01 mg/kg/day are based on a chronic animal study be Heywood et al. (1979) in which dogs were exposed to chloroform at either 15 or 30 mg/kg/day for 6 days/week for 7.5 years. Fatty cysts and altered hepatocytes, considered to be treatment-related, were observed in livers of some dogs in both treatment groups. Also, a dose-related increase in hepatic enzyme levels was noted in the high-dose animals. Therefore, the LOAEL was determined to be 15 mg/kg/day. Adjusting this value for 7 day/week exposure period and applying an uncertainty factor of 1,000 (10 for use of a LOAEL instead of a NOAEL, 10 for animal-to-human extrapolation, and 10 for sensitive human subpopulations) results in a chronic oral RfD of 0.01 mg/kg/day.

Chronic oral exposure in humans adversely affects the central nervous system (CNS) as well as the liver, kidneys, and heart (NIOSH, 1974). Chronic effects reported following oral exposure of rats to levels of 60 mg/kg/day or greater include decreased liver weights and serum cholinesterase levels, an increased incidence of a noncancerous respiratory disease, and gonadal atrophy (EPA, 1984). Chloroform readily passes the cell membrane, and effects include CNS disturbances, liver glutathione depletion, and gonadal and bone marrow abnormalities (USATHAMA, 1989). Animals on high-fat or protein-poor diets appear more susceptible to hepatotoxicity, while high-carbohydrate and high-protein diets appear to have a protective effect (USATHAMA, 1989). Liver necrosis and gonad dysfunction were reported in rats at 150 mg/kg/day (Palmer

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	0.01	Medium	Fatty liver cysts	IRIS, 1995	1,000 H,N,S
Subchronic	0.01	--	Fatty liver cysts	EPA, 1994	1,000 H,N,S
Inhalation					
Chronic	--	--	--	--	--
Subchronic	--	--	--	--	--

Note: IRIS = Integrated Risk Information System.
RfD = reference dose.
RfC = reference concentration.
-- = Not available.
LOAEL = lowest observed adverse effect level.
NOAEL = no observed adverse effect level.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
H = to extrapolate from an animal study to humans.
N = to extrapolate from a LOAEL to a NOAEL.
S = to protect sensitive human subpopulations.

[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

et al., 1979). No effects in humans following subchronic oral exposure at 2.5 mg/kg/day were reported by EPA (1984) or in rats exposed to 30 mg/kg/day (Palmer et al., 1979), but necrosis of the liver and dysfunction of the gonads were reported in rats at 150 mg/kg/day (Palmer et al., 1979).

Occupational human exposure via inhalation in the workplace at levels between 22 and 237 ppm is reported to result in depression, gastrointestinal disturbances, headache, and frequent and scalding urination (EPA, 1984). Other reported effects include cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdose is attributed to ventricular fibrillation. In rats, inhalation exposure to as little as 25 ppm produced histopathological changes in the liver and kidney (EPA, 1984). Similar effects were present in guinea pigs and rabbits, although the data are questionable as results were observed in the lowest and highest doses, but none were observed at the middle dose.

Carcinogenicity

Chloroform has been classified as a group B2 (probable human) carcinogen (IRIS, 1995). This classification indicates sufficient evidence of carcinogenicity in animals but inadequate evidence of human carcinogenicity. The carcinogenic slope factors (CSFs) and supporting information are presented in Table 2.

The oral CSF of 0.0061 $(mg/kg/day)^{-1}$ was derived from a drinking water bioassay by Jorgenson et al. (1985) in which chloroform was administered in drinking water to rats and mice at concentrations of 200, 400, 900, and 1800 mg/L for 104 weeks. These concentrations were reported by the author to correspond to 19, 38, 81, and 160 mg/kg/day for rats and 34, 65, 130, and 263 mg/kg/day for mice. A significant increase in renal tumors in rats was observed in the highest dose group and considered dose-related. The liver tumor incidence in mice was not significantly increased. This study was specifically designed to measure the effects of exposure to low doses of chloroform.

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.0061	B2	Kidney tumors	IRIS, 1995
Inhalation	0.081[a]	B2	Liver tumors	EPA, 1994

Note: B2 = suspect human carcinogen (sufficient evidence of carcinogenicity in animals and inadequate data in humans).
UR = unit risk.

[a]Inhalation CSF based on an inhalation UR of 2.3×10^{-5} $(\mu g/m^3)^{-1}$ (IRIS, 1995) and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

The inhalation unit risk (UR) of 2.3×10^{-5} $(\mu g/m^3)^{-1}$ is based on a gavage study by NCI (1976) in which mice were dosed with chloroform at time-weighted average levels of 138 and 277 mg/kg/day (males) and 238 and 477 mg/kg/day (females) for 78 weeks. Highly significant increases in hepatocellular carcinomas were observed in mice of both sexes, while hepatic nodular hyperplasia was observed in those animals not developing hepatocellular carcinoma. Mice exposed to 90 mg/kg/day of chloroform developed kidney tumors. Limited data suggest that oral human exposure to chloroform leads to increased risk of bladder, colon, and rectal cancer (EPA, 1984). Assuming that a healthy 70-kg adult inhales 20 m^3 of air per day, an inhalation CSF of 0.081 $(mg/kg/day)^{-1}$ is derived (EPA, 1994).

Mutagenicity

Although not independently confirmed, significantly higher frequencies of acquired chromosomal aberrations were noted in the lymphocytes of women occupationally exposed to chloroform and other organic solvents (Funes-Cravioto et al., 1977). Similar findings were observed in the children of these women. The relevance of acquired somatic chromosomal aberrations to the risk of malformations or any other disease in the offspring is unknown.

The majority of tests for genotoxicity of chloroform have been negative (IRIS, 1995). One study, however, demonstrated binding of radiolabeled chloroform to calf thymus DNA following metabolism by rat liver microsomes (IRIS, 1995). Chloroform caused mitotic recombination in Saccharomyces (Callen et al., 1980) and sister chromatid exchange in cultured human lymphocytes and in mouse bone marrow cells exposed in vivo (Morimoto and Koizumi, 1983). A host-mediated assay using mice indicated that chloroform was metabolized in vivo to a form mutagenic to a strain of Salmonella. Likewise urine extracts from chloroform-treated mice were mutagenic (Agustin and Lim-Sylianco, 1978).

Teratogenicity/Reproductive Effects

Chloroform crosses the human placenta and can be detected in fetal blood (ReproText, 1995). The frequency of congenital anomalies was no greater than expected among 492 children of laboratory workers occupationally exposed to organic solvents during the first trimester of pregnancy; 128 of these mothers reported first trimester exposure to chloroform (Axelsson et al., 1984). The only cases where chloroform was suspected of human reproductive effects were two cases of eclamptic toxemia of pregnancy in women working in the same laboratory where chloroform was used (ReproText, 1995). Eclampsia in pregnancy follows high blood pressure and retention of fluid and is marked by headache, visual disturbances, and either convulsions, coma, or both.

In general, chloroform has been highly embryotoxic and somewhat teratogenic in animal studies with fetal toxicity generally occurring at exposure levels associated with maternal toxicity (Shepard, 1995). Teratogenic effects were reported in rats and mice exposed to 30 ppm or higher via inhalation on days 6 to 15 of gestation (EPA, 1984). Following the inhalation of chloroform, rats experienced increased post-implantation deaths, decreased fetal weight gain, reduced conception rate, increased resorptions, and retarded fetal growth (TERIS, 1995). In mice, chloroform impaired pregnancy, increased pre-implantation losses, retarded fetal growth, and caused cleft palates (TERIS, 1995). The frequency of cleft palate was increased among the offspring of mice exposed chronically during pregnancy to chloroform vapors at a concentration 50 times the NIOSH occupational standard of 2 ppm (about 1/100 of the human anesthetic dose) (TERIS, 1995). When male mice were exposed to chloroform through inhalation, structural abnormalities in sperm were reported; this effect was not observed following the intraperitoneal injection of chloroform (ReproText, 1995). Anal atresia was observed with increased frequency among the offspring of pregnant rats after similar exposure, but not after exposure to 15 times the NIOSH occupational standard (TERIS, 1995). In both studies, considerable maternal toxicity occurred.

In contrast, no malformations were observed in the offspring of rats or rabbits given chloroform orally during pregnancy at doses up to 400 mg or 50 mg/kg/day, respectively, although there was evidence of maternal toxicity (ReproText, 1995). Oral doses greater than 100 mg/kg/day in female rabbits were toxic to dam and fetus (USATHAMA, 1989), intimating that 100 mg/kg/day represents a LOAEL for the rabbit (TERIS, 1995).

ECOTOXICITY

Aquatic Organisms

Toxic concentrations reported in the literature for chloroform in aquatic systems cover a wide range of values. Acute toxicity tests conducted on rainbow trout, bluegill, and a daphnia species evidenced median effect concentrations of 28,900 to 115,000 μg/L (EPA, 1980). Birge et al. (1980) reported 96-hour LC_{50} values of 270 to 35,100 μg/L in toads and frogs exposed from egg stage to hatchlings; fish LC_{50} values were reported from 2,030 to 75,000 μg/L (Anderson and Lusty, 1980). Chronic (27-day) LC_{50} values of 2,030 and 1,240 μg/L were reported for rainbow trout larvae at water hardness values of 50 and 200 mg/L, respectively (EPA, 1980). The 96-hour LC_{50} value of 270 μg/L reported by Birge et al. (1980) for toads and frogs may be considered the acute LOAEL. Applying an uncertainty factor of 100 yields a water TRV of 0.0027 mg/L (2.7 μg/L) for aquatic organisms.

Terrestrial Organisms

No information was identified regarding the toxicity of chloroform to vegetation; however, some general observations may be made regarding potential interactions. With an log octanol-water partition coefficient (K_{ow}) of 1.94, chloroform is partially miscible with water based on a regression analysis by Briggs et al. (1982, 1983). This suggests that chloroform will enter the plant and be translocated within the plant structure. As it is lipophilic, it may pass through the cuticle of the leaf, if in contact for a sufficient length of time; conversely, the volatility of the compound would limit the time available.

No oral or inhalant toxic levels are provided for livestock. Booth (1977) states that only a 2- to 4-percent chloroform concentration in air is necessary to induce anesthesia in an animal in a reasonable timeframe of 10 to 12 minutes and that this concentration should be lowered to 1.5 percent for the duration of anesthesia. Dogs fasted for 24 hours and then anesthetized for 1.5 hours evidenced central necrosis of one-third to one-half of the liver lobules. No information regarding the effects in terrestrial wildlife was identified.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 3. EPA has not established a health-based Maximum Contaminant Level Goal (MCLG) for chloroform in drinking water due to insufficient information (IRIS, 1995). However, an interim MCL of 100 μg/L for total trihalomethanes (chloroform, bromodichloromethane, dibromochloromethane, and bromoform) is proposed based on chronic toxicity data for chloroform and existing technology and treatment methods (44 FR 68624). Chloroform produced CNS depression, as well as hepatic, renal, teratogenic and carcinogenic effects at dose levels from 30 to 350 mg/kg. This MCL applies only to community water systems which serve a population of 10,000 or more individuals and add a disinfectant (oxidant) to the water in any part of the drinking water treatment process.

Although inhalation appears to be the primary exposure route for chloroform, EPA concluded that current information does not indicate that chloroform endangers public health at ambient concentrations (excluding emergency releases). Therefore, no regulation directed specifically at chloroform is necessary at this time under the Clean Air Act and no national ambient air quality standards (NAAQS) have been developed for chloroform (IRIS, 1995). EPA indicated that it intends to add chloroform to the list of hazardous air pollutants for which it intends to establish emission standards under section

Table 3. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only[a]
Chloroform	100[b]	--	--	--	--	--	--	0.19[c]	15.7[c]	0.19[c]

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[b]National Interim Primary Drinking Water Regulation (NIPDWR) (45 FR 57332) for total trihalomethanes (chloroform, bromodichloromethane, dibromochloromethane, and bromoform).
[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

112(b)(1)(A) of the Clean Air Act. The EPA will decide whether to add chloroform to the list only after studying possible techniques that might be used to control emissions of chloroform and further assessing the public health risks. The EPA will add chloroform to the list if emission standards are warranted.

The Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1995). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1995; ACGIH, 1993). These values are presented in the following table of air quality criteria.

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	9.78 c	--	49 c
STEL	--	9.78 (60 min) c	--
CL	--	--	--

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the

concentration that should not be exceeded at any any time during a workday. The code "c" designates that this chemical is regulated as a potential carcinogen.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to chloroform (IRIS, 1995). However, the LOELs for the freshwater aquatic species tested have been determined (IRIS, 1995). These LOELs, which are established when the minimum data required to derive AWQCs are not available, are presented in the following table.

LOEL (µg/L)	Freshwater	Marine
Acute	2,890	--
Chronic	1,240	--

REFERENCES

Agustin, J.S. and Lim-Sylianco, C.Y. 1978. Mutagenic and clastogenic effects of chloroform. Bull. Phil. Biochem. Soc. 1:17-23. (cited in IRIS, 1995).

Anderson, D.R., and Lusty, E.B. 1980. Acute Toxicity and Bioaccumulation of Chloroform to Four Species of Freshwater Fish. U.S. NTIS NUREG/CR-0893.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Axelsson, G., Lutz, C., and Rylande, R. 1984. Exposure to solvents and outcome of pregnancy in university laboratory employees. Brit. Jour. Ind. Med. 41:305-312, 1984. (cited in TERIS, 1995).

Birge, W.J., Black, J.A., and Kuehne, A.E. 1980. Effects of Organic Compounds on Amphibian Reproduction. University of Kentucky Water Resources Research Institute, Lexington, KY. PB80-147523. Research Report No. 121.

Booth, N.H. 1977. Drugs Acting on the Central Nervous System; Inhalant Anesthetics. In: Veterinary Pharmacology and Therapeutics. 4th Ed. Jones, L.M., Booth, N.H., and McDonald, L.E., Eds. Iowa State University Press, Ames, IA.

Briggs, G.G., Bromilow, R.H., and Evans, A.A. 1982. Relationships Between Lipophilicity and Root Uptake and Translocation of Non-Ionized Chemicals by Barley. Pesticide Science, 13:495-504.

Briggs, G.G., Bromilow, R.H., Evans, A.A., and Williams, M. 1983. Relationships Between Lipophilicity and the Distribution of Non-Ionized Chemicals in Barley Shoots Following Uptake by the Roots. Pesticide Science, 14:492-500.

Callen, D.F., Wolf, C.R., and Philpot, R.M. 1980. Cytochrome P-450 mediated genetic activity and cytotoxicity of seven halogenated aliphatic hydrocarbons in Saccharomyces cerevisiae. Mutat. Res. 77:55-63. (cited in IRIS, 1995).

REFERENCES
(Continued, Page 2 of 3)

Funes-Cravioto, F., Kolmodin-Hedman, B., Lindsten, J., and Nordenskjold, M. 1977. Chromosome aberrations and sister-chromatid exchange in workers in chemical laboratories and a rotoprinting factory and in children of women laboratory workers. Lancet. 2:322-325. (cited in TERIS, 1995).

Heywood, R., Sortwell, R.J., and Noel, P.R.B. 1979. Safety evaluation of toothpaste containing chloroform; long-term study in beagle dogs. Jour. Environ. Pathol. Toxicol. 2:835-851. (cited in IRIS, 1995).

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Jorgenson, T.A., Meierhenry, E.F., and Rushbrook, C.J. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. Fund. Appl. Toxicol. 5(4):760-769. (cited in IRIS, 1995).

Morimoto, K. and Koizumi, A. 1983. Trihalomethanes-induced sister chromatid exchanges in human lymphocytes in vitro and mouse bone marrow cells in vivo. Environ. Res. 32(1):72-79. (cited in IRIS, 1995).

National Cancer Institute (NCI). 1976. Report on Carcinogenesis Bioassay of Chloroform. National Cancer Institute, Washington, DC. NTIS PB 264018. (cited in IRIS, 1995).

National Institute of Occupational Safety and Health (NIOSH). 1974. Recommended Standard Criteria for an Occupational Exposure to Chloroform. U.S. DHEW, PHS, CDC, Rockville, MD. NTIS PB 246-695.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1995. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

REFERENCES
(Continued, Page 3 of 3)

Palmer, A.K., Street, A.E., Roc, F.J.C., Worden, A.N., and Van Abbe, N.J. 1979. Safety Evaluation of Toothpaste Containing Chloroform. II. Long-term Studies in Rats. Journal of Environmental Pathology, Toxicology, and Oncology, 2:821-833.

Reproductive Risk Information System (ReproText®). 1995. B.J. Dabney. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Shepard's Catalog of Teratogenic Agents. 1995. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Teratogen Information System (TERIS). 1995. University of Washington, Seattle, WA. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Army Toxic and Hazardous Materials Agency (USATHAMA). 1989. Biota Remedial Investigation. Final Report, Vol. II. Prepared for Office of the Program Manager, Rocky Mountain Arsenal Contamination Cleanup. AMXRM Aberdeen Proving Ground, MD.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Chloroform. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-80-033. NTIS No. PB81-117442

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Chloroform. Environmental Citeria and Assessment Office, Office of Research and Development, Cincinnati, OH. EPA 540/1-86/010. NTIS No. PB86-134210.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. Publication No. 9200.6-303(94-1). NTIS No. PB94-921199.

CHROMIUM

(Chromium metal: CAS No. 7440-47-3)
(Chromium III: CAS No. 16065-83-1)
(Chromium VI: CAS No. 18540-29-9)

INTRODUCTION

Chromium is found throughout the environment and is normally present in one of two valence states, trivalent [Cr(III)] or hexavalent [Cr(VI)]. Cr(III) is considered to be less toxic and more stable than is Cr(VI). Because Cr(III) is readily hydrolyzed and precipitated as chromium hydroxide, it is less mobile in the environment (EPA, 1985). Cr(VI), conversely, is soluble in water and, thus, is more mobile in the environment. Cr(III) is considered an essential element in human nutrition.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) for both Cr(III) and Cr(VI) are available from EPA (IRIS, 1994; EPA, 1994) and are presented in Table 1. Inhalation reference concentrations (RfCs) for both Cr(III) and Cr(VI) are currently under review by an EPA RfD/RfC work group (IRIS, 1994; EPA, 1994).

The chronic (and interim subchronic) oral RfD for Cr(III) was developed from two studies by Ivankovic and Preussmann (1975) in which rats were exposed to dietary levels of chromic acid (Cr_2O_3) up to 5 percent of total diet for either 90 days or 2 years (IRIS, 1994). No effects were observed in the 2-year study, but in the 90-day study, the absolute weights of livers and spleens were reduced in animals exposed to the highest dose of 1,400 mg/kg-bw/day. Applying an uncertainty factor of 100 (10 for animal-to-human extrapolation and 10 for sensitive human subpopulations) and a modifying factor of 10 (to reflect uncertainty in the study NOEL) results in a chronic (and interim subchronic) oral RfD of 1 mg/kg/day for Cr(III). The modifying factor was incorporated for the following reasons: 1) the effects observed in the 90-day study were not

Table 1. Noncarcinogenic Oral Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Cr(III)					
Chronic	1	Low	None observed	IRIS, 1994	100 H,S / (10 X)
Subchronic	1	Low	None observed	EPA, 1994	100 H,S / (10 X)
Cr(VI)					
Chronic	0.005	Low	None observed	IRIS, 1994	500 F,H,S
Subchronic	0.02	Low	None observed	EPA, 1994	100 H,S

Note: RfD = reference dose.

[a]Uncertainty factors are typically, but not limited to, multiples of 10 and are provided using the following codes:

F = to account for the less-than-lifetime exposure duration of the principal study.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.

[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

X = to account for uncertainties in the NOEL when no LOAEL is derived from the study.

Source: ESE.

explicitly addressed in the 2-year study and, thus, the highest NOAEL in the 2-year study may be a LOAEL; 2) the absorption of chromium is low (<1%) and is influenced by a number of factors; thus, a considerable potential variation in absorption exists; and 3) animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed (IRIS, 1994). This RfD is limited to metallic Cr(III) of insoluble salts.

The chronic and subchronic oral RfDs for Cr(VI) were derived from a study by MacKenzie et al. (1958) in which no effects were observed in rats exposed to drinking water containing 25 mg/L, which is equivalent to 2.4 mg/kg-bw/day of Cr(VI). Applying an uncertainty factor of 100 (10 for animal-to-human extrapolation and 10 for sensitive human subpopulations) results in a subchronic oral RfD of 0.02 mg/kg/day for Cr(VI). Applying an additional uncertainty factor of 5 to account for the less-than-lifetime study duration results in a chronic oral RfD of 0.005 mg/kg/day. These RfDs are limited to metallic Cr(VI) of soluble salts.

Carcinogenicity

Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both Cr(III) and Cr(VI) compounds; however, since only Cr(VI) has been found to be carcinogenic in animal studies, EPA concluded that only Cr(VI) should be classified as a human carcinogen. The potential carcinogenicity of Cr(III) is currently being reviewed by an EPA inter-office work group.

EPA has classified Cr(VI) as a Group A known human carcinogen via inhalation, only. This classification is based on a number of epidemiological studies reported by EPA (IRIS, 1994) from which a correlation between lung cancer and inhalation of chromium, primarily Cr(VI), was established. Cr(VI) compounds have not produced lung tumors in animals by inhalation. Based on the epidemiologic studies, EPA has derived an inhalation unit risk (UR) for Cr(VI) of 0.012 $(\mu g/m^3)^{-1}$ (IRIS, 1994).

Assuming that a healthy 70-kilogram adult inhales 20 m^3/day of air, the UR may be converted to an inhalation cancer slope factor (CSF) of 41 $(mg/kg/day)^{-1}$ (EPA, 1994). The UR should not be used if the air concentration exceeds 0.8 $\mu g/m^3$, since above this concentration the UR may not be appropriate.

There were no long-term studies of ingested Cr(VI) identified in the literature; therefore, EPA has not classified ingested Cr(VI) with respect to carcinogenic potential.

Mutagenicity

A large number of chromium compounds have been assayed in *in vitro* genetic toxicology assays (IRIS, 1994). In general, Cr(VI) is mutagenic in bacterial assays whereas Cr(III) is not (IRIS, 1994). Likewise Cr(VI) but not Cr(III) was mutagenic in yeasts and in V79 cells (IRIS, 1994). Both Cr(III) and Cr(VI) compounds decrease the fidelity of DNA synthesis *in vitro*, while Cr(VI) compounds inhibit replicative DNA synthesis in mammalian cells and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage (IRIS, 1994). Chromate has been shown to transform both primary cells and cell lines (IRIS, 1994). Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr(VI) and Cr(III) salts were clastogenic for cultured human leukocytes (IRIS, 1994).

Teratogenicity/Reproductive Effects

No reproductive data were found for chromium or its compounds in humans. Although there is a concern that chromium may affect male fertility, based on animal studies, semen quality was not correlated with concentrations of chromium in welders (Bonde and Ernst, 1992).

Cr(VI) impaired reproductive function and caused sterility in rats at levels above 0.125% in the diet (ReproText, 1994). Chromium rapidly accumulated in the testes,

and Cr(VI) injected intreperitoneally at a dose of 0.5 mg/kg, 5 days a week for 8 weeks, produced reduction in sperm motility in the epididymis of Wistar rats. A decrease in serum testosterone and increase in FSH were also seen. These results suggest that chromium may be affecting male fertility in rats by both direct action on the testes and indirectly through alterations in hormone levels (Ernst and Bonde, 1992).

Chromium trioxide administered by injection caused birth defects and resorptions in hamsters (ReproText, 1994). Chromium chloride and trioxide were teratogenic in mice; while sodium dichromate was mildly teratogenic in chickens (ReproText, 1994). Transplacental transfer of chromium chloride has been shown in mice (Friberg et al., 1986), and chromium levels in the human fetus are ten times those of adults (ReproText, 1994). The embryonic and fetal uptake of chromate was ten times that of Cr(III) in rats (Friberg et al., 1986).

No malformations were noted among the offspring of rats fed diets containing 2 to 5 percent Cr_2O_3 (trivalent) during pregnancy (TERIS, 1994). Increased frequencies of fetal death, as well as cleft palate and other malformations, were observed among the offspring of hamsters treated with 5 to 15 mg/kg of CrO_3 (hexavalent) during pregnancy (TERIS, 1994). This teratogenic effect exhibited the typical dose and time of gestation dependence. Similarly, increased frequencies of malformations, including cleft palates and neural tube defects, were observed among the offspring of pregnant mice treated with 100 mg/kg/day of $CrCl_3$ or 20 mg/kg/day of CrO_3 (TERIS, 1994).

ECOTOXICITY

Aquatic Organisms

Cr(VI)--Data indicate that Cr(VI) is more toxic to freshwater biota in comparatively soft and acidic waters and that younger life stages are more sensitive than older organisms (USFWS, 1986). Acute toxicity tests have been conducted with a wide variety of freshwater plants, and LC_{50} values range from 2,500 to 25,000 μg/L (USFWS, 1986) (see Table 2). In chronic studies on plants, reduced growth of the

alga Chlamydomonas reinhardi was found at Cr(VI) concentrations of 10 μg/L (USFWS, 1986) (see Table 3). In a study reported by EPA (1980), Eurasian watermilfoil (Myriophyllum spicatum) showed root weight inhibition at concentrations of 1,900 μg/L Cr(VI) (USFWS, 1986).

Based on LC_{50} values from acute toxicity studies, aquatic organisms most sensitive to Cr(VI) are freshwater crustaceans and rotifers, with values of 435 and 3,100 μg/L, respectively (USFWS, 1986) (see Table 2). EPA (1980) reported that 10 μg/L Cr(VI) reduced survival and fecundity in the cladoceran Daphnia magna (USFWS, 1986). Chronic values for freshwater animals species range from 73 μg/L in the rainbow trout (Oncorhyncus mykiss) to 1,990 μg/L in the fathead minnow (Pimephales promelas) (USFWS, 1986) (see Table 4). Mangi et al. (1978) conducted bioconcentration studies on the chlorophytes Hydrodictyon reticulatum and Oedogonium sp. growing in a medium containing 10 μg/L Cr(VI); BCFs ranged from 200 to 600 in 14 days (USFWS, 1987). Accumulation of Cr by living and dead plant tissue was reportedly extensive, with uptake linearly approximating concentration on a logarithmic basis. In 1982, Jouany reported that the green alga Chlorella vulgaris biomagnified Cr(VI) from the growth medium about 1,000 times in 28 days at ambient concentrations of 300 μg/L (USFWS, 1986). BCF data are available from two studies with rainbow trout, and the BCF is about 1 (USFWS, 1986).

Cr(III)--No data for the acute toxicity of Cr(III) to plants were found. In a chronic toxicity test reported by EPA (1980), root weight inhibition was found in Eurasian watermilfoil (M. spicatum) at 9,900 μg/L Cr(III) (USFWS, 1986). Results of acute toxicity tests of Cr(III) to freshwater aquatic animals show that, in soft water, values range from 2,000 μg/L for Daphnia magna to 5,070 μg/L for the fathead minnow (Pimephales promelas) (see Table 2). In hard water, the

Table 2. Acute Toxicities of Cr(VI) and Cr(III) to Aquatic Life (Page 1 of 3)

Chemical Species, Ecosystem, Taxonomic Group, Organism, Modifiers	Acute Toxicity			
	Concentration (μg/L)	Percent dead	Duration of test	Reference*
Cr(VI)				
Freshwater				
Plants	2,500 to 25,000	50	96 h	Mangi et al., 1978
Rotifers				
Philodena acuticornis				
Water hardness, in mg/L as $CaCO_3$				
25	3,100	50	96 h	Buikema et al., 1974
81	15,000	50	96 h	Buikema et al., 1974
Mulluscs				
Snail (Physa heterostropha)				
Water hardness, in mg $CaCO_3$/L				
45	17,300	50	96 h	EPA, 1980
171	31,600 to 40,600	50	96 h	EPA, 1980
Crustaceans				
Amphipod (Gammarus pseudolimnaeus)	67,000	50	96 h	EPA, 1980
Freshwater prawn (Machrobrachium lamarrei)	1,840	50	96 h	Murti et al., 1983
Cladoceran (Daphnia magna)	435	50	24 h	Jouany et al., 1982
Fish				
Mud skipper (Boleophthalmus dussumieri)	30,500	50	96 h	Krishnaja and Rege, 1982
Rainbow trout (Oncorhyncus mykiss)				
Weight 0.2 g				
Water pH 7.8	12,200	50	96 h	Van der Putte, 1981
Water pH 7.0	7,600	50	96 h	Van der Putte, 1981
Water pH 6.5	3,400	50	96 h	Van der Putte, 1981
Weight 25 g				
Water pH 7.8	65,500	50	96 h	Van der Putte, 1981
Water pH 7.0	45,000	50	96 h	Van der Putte, 1981
Water pH 6.5	20,200	50	96 h	Van der Putte, 1981

Table 2. Acute Toxicities of Cr(VI) and Cr(III) to Aquatic Life (Continued, Page 2 of 3)

Chemical Species, Ecosystem, Taxonomic Group, Organism, Modifiers	Acute Toxicity Concentration (µg/L)	Percent dead	Duration of test	Reference*
Fish (Cont.)				
2 spp.	17,600 to 118,000	50	96 h	Negilski, 1976
3 spp.				
Softwater	<18,000	50	96 h	Stevens et al., 1976
Hardwater	>133,000	50	96 h	Stevens et al., 1976
Salmon fingerlings (Oncorhyncus sp.)	200	53	12 w	Stevens et al., 1976
Goldfish (Carassius auratus)	110,000	50	96 h	Riva et al., 1981
Water hardness, in mg $CaCO_3$/L				
20	37,500	50	96 h	EPA, 1980
220	>90,000	50	96 h	EPA, 1980
Bluegill (Lepomis macrochirus)				
Water hardness, in mg $CaCO_3$/L				
20	118,000	50	96 h	EPA, 1980
44	113,000	50	96 h	EPA, 1980
45	110,000 to 170,000	50	96 h	EPA, 1980
120	213,000	50	96 h	EPA, 1980
171	130,000 to 135,000	50	96 h	EPA, 1980
360	133,000	50	96 h	EPA, 1980
Striped bass (Morone saxatilis)	30,400	50	96 h	EPA, 1980
Cr(III)				
Freshwater				
Molluscs				
Snail (Amnicola sp.)	8,400	50	96 h	EPA, 1980
Annelids				
Worm (Nais sp.)	9,300	50	96 h	EPA, 1980
Arthropods				
Ampipod (Gammarus sp.)	3,200	50	96 h	EPA, 1980
Insects, 4 spp.	2,000 to 64,000	50	96 h	EPA, 1980

Table 2. Acute Toxicities of Cr(VI) and Cr(III) to Aquatic Life (Continued, Page 3 of 3)

Chemical Species, Ecosystem, Taxonomic Group, Organism, Modifiers	Acute Toxicity			
	Concentration (μg/L)	Percent dead	Duration of test	Reference*
Arthropods (Cont.)				
Cladoceran (Daphnia magna)				
Water hardness, in mg $CaCO_3$/L				
48	2,000	50	96 h	EPA, 1980
52	16,800	50	96 h	EPA, 1980
99	27,400	50	96 h	EPA, 1980
110	26,300	50	96 h	EPA, 1980
195	51,400	50	96 h	EPA, 1980
215	58,700	50	96 h	EPA, 1980
Fish				
9 spp.	3,300 to 71,900	50	96 h	EPA, 1980
3 spp.				
Soft water	<3,000	50	96 h	Stevens et al., 1976
Hard water	>72,000	50	96 h	Stevens et al., 1976
Salmon fingerlings	200	0	12 w	Stevens et al., 1976
Rainbow trout				
Juveniles	4,400	50	96 h	Steven and Chapman, 1984
Eggs	495	100	30 d	Steven and Chapman, 1984
Fathead minnow				
Water hardness, in mg $CaCO_3$/L				
20	5,070	50	96 h	EPA, 1980
203	7,000 to 29,000	50	96 h	EPA, 1980
360	67,400	50	96 h	EPA, 1980

Note: d = day.
g = gram.
h = hour.
w = week.

*References other than EPA (1980) were cited from USFWS (1986).

Source: ESE.

Table 3. Chronic Toxicities of Cr(VI) and Cr(III) to Aquatic Plants (Page 1 of 2)

Species	Chemical	Hardness (mg/L as CaCO3)	Effect	Concentration (μg/L)	Reference*
Cr(VI) Freshwater Species					
Chlamydomonas reinhardi (Green alga)	Potassium dichromate	--	Reduction in growth	10	Zarafonetis and Hampton, 1974
Selenastrum capricornutum (Green alga)	Sodium chromate	--	Inhibition in growth	45	Garton, 1972
Scenedesmus sp. (Green alga)	Potassium dichromate	--	Inhibition in growth	500	Staub et al., 1973
Navicula seminulum (Diatom)	Potassium dichromate	45	50 percent growth reduction	187 to 308	Academy of Natural Sciences, 1960
Navicula seminulum (Diatom)	Potassium dichromate	171	50 percent growth reduction	254 to 442	Academy of Natural Sciences, 1960
Myriophyllum spicatum (Eurasian watermilfoil)	Dichromate+	--	50 percent root weight inhibition	1,900	Stanley, 1974
Cr(III) Freshwater Species					
Myriophyllum spicatum (Eurasian watermilfoil)	--	--	50 percent root weight inhibition	9,900	Stanley, 1974

Table 3. Chronic Toxicities of Cr(VI) and Cr(III) to Aquatic Plants (Continued, Page 2 of 2)

Note: -- = not measured.

*References cited are from EPA (1980b).
+Salt not given.

Source: USFWS, 1986.

Table 4. Acute Toxicities of Cr(VI) and Cr(III) to Aquatic Plants

Species	Test	Chemical	Hardness (mg/L as CaCO3)	Limits (μg/L)*	Chronic Value (μg/L)*	Reference+
Cr(VI) Freshwater Species						
Oncorhynchus mykiss (Rainbow trout)	ELS	Chromium trioxide	34	51 to 105	73	Sauter et al., 1976
Oncorhynchus mykiss (Rainbow trout)	ELS	Sodium dichromate	45	200 to 350	265	Benoit, 1976
Salvelinus fontinalis (Brook trout)	LC	Sodium dichromate	45	200 to 350	265	Benoit, 1976
Pimephales promelas (Fathead minnow)	LC	Potassium dichromate	209	1,000 to 3,950	1,990	Pickering, 1980
Cr(III) Freshwater Species						
Daphnia magna (Cladoceran)	LC	Chromic nitrate	52	47 to 93	66	Chapman, Manuscript
Pimephales promelas (Fathead minnow)	LC	Chromium potassium sulfate	203	750 to 1,400	1,020	Pickering, 1980

*Results are expressed as chromium, not as the compound.
+References cited are from EPA (1980a).

Source: ESE.

corresponding toxicity values are 58,000 μg/L for D. magna and 67,400 μg/L for the fathead minnow. Comparative data indicate that Cr(III) is more toxic in soft water than in hard water (USFWS, 1986). Few chronic toxicity study data were found in the literature. In one chronic study reported by EPA (1980), fecundity of Daphnia magna was reduced at 44 μg/L Cr(III) (USFWS, 1986).

In bioaccumulation studies reported by Neff et al. (1978), annelid worms (Tubifex sp.) accumulated about 1 ppm Cr during a 2-week exposure in sediments containing 175 ppm Cr(III) (USFWS, 1986). No BCF data are available for Cr(III).

Terrestrial Organisms

Cr(VI)--The LD_{50} value for mice, as reported by Steven et al. in 1976, was 5 mg/kg body weight for Cr(VI) (USFWS, 1986). In chronic toxicity studies, Cr(VI) was fatal to laboratory dogs in 3 months at dietary levels of 100 ppm; however, no measurable effects were observed in chickens at dietary levels of 100 ppm over a 32-day period (USFWS, 1986). Most mammalian experimental animals died at injected doses of 1 to 5 mg/kg-bw.

Cr(III)--Cr(III) compounds were generally less toxic than Cr(VI) compounds; however, survival may be affected by differences in uptake of anionic and cationic Cr(III) species (USFWS, 1986). The LD_{50} value for mice, as reported by Steven et al. in 1976, was 260 mg/kg-bw. The LC_{50} value for chicken embryos is 22.9 mg/kg-bw of Cr(III) (Haseltine et al., 1985). Water-soluble Cr(III) has been shown to produce toxic effects in rabbits (Hatherill, 1981; Tandon et al., 1978).

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 5. EPA has promulgated a nonenforceable health-based Maximum Contaminant Level

Table 5. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only
Chromium, total	100	100	--	--	--	--	--	--	--	--
Chromium (III)	--	--	--	1,400	1,400	240	120	170,000	3,433,000	179,000
Chromium (VI)	--	--	--	1,400	1,400	240	120	50	--	--

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

Source: ESE.

Goal (MCLG) and an enforceable (for public water supplies) Maximum Contaminant Level (MCL) of 0.1 mg/L for total chromium [Cr(III) and Cr(VI)] based on the RfD methodology for Cr(VI), the more toxic chromium species (IRIS, 1994). The MCLG is based upon a DWEL of 0.17 mg/L calculated from available human and animal data and an assumed drinking water contribution of 20 percent. An uncertainty factor of 500 was applied. The MCLG also falls into the safe and adequate daily dietary intake range of 50 to 200 mg/day for Cr(III) established by the National Research Council in the National Academy of Sciences (NAS, 1989).

No national ambient air quality standards (NAAQS) have been developed for chromium metal or individual species of chromium under the Clean Air Act (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to chromium metal and individual species/salts (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively, for chromium as a metal and salts (NIOSH, 1994; ACGIH, 1993). These values are presented in the Table 6.

Ecological Receptors

AWQC for the protection of freshwater and marine organisms against exposure to Cr(III) and Cr(VI) have been established by EPA (IRIS, 1994) are presented in Table 7.

Table 6. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	0.5 (soluble chromic and chromous salts) 1 (metal and insoluble salts)	0.5 [8-hour; metal and Cr(II) and Cr(III) compounds] 0.001 c [10-hour; Cr(VI) compounds]	0.5 [metal and Cr(II) and Cr(III) compounds] 0.05 c [Cr(VI) compounds and chromate]
STEL	--	--	--
CL	0.1 s (chromic acid and chromates)	--	0.05 c (Fumes)

Note:
TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.
STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.
CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.
c = designates that this chemical is regulated as a potential carcinogen.
s = signifies that dermal absorption may be a significant exposure route for this chemical.

Source: ESE.

Table 7. Ambient Water Quality Criteria

AWQC (μg/L)	Freshwater	Marine
Cr(III)		
Acute	980 [a]	10,300 [c]
Chronic	120 [b]	--
Cr(VI)		
Acute	16	1,100
Chronic	11	50

[a] The acute freshwater AWQC for Cr(III) is hardness-dependent. The 1-hour average concentration in μg/L of Cr(III) should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(8.19 * [\ln(hardness)] + 3.688)}$$

[b] The chronic freshwater AWQC for Cr(III) is hardness-dependent. The 4-day average concentration in μg/L of Cr(III) should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(8.19 * [\ln(hardness)] + 1.561)}$$

[c] Insufficient data is available to derive an AWQC. The indicated value is a lowest-observed-effect level (LOEL).

At a hardness of 50 mg/L as $CaCO_3$, the acute and chronic criteria for Cr(III) are 980 and 120 μg/L, respectively (IRIS, 1994).

Source: ESE.

REFERENCES

Academy of Natural Sciences of Philadelphia. 1960. The Sensitivity of Aquatic Life to Certain Chemicals Commonly Found in Industrial Wastes. Final Report: U.S. PHS Grant RG-3965 (CZR1). (cited in EPA, 1980).

Benoit, D.A. 1976. Chronic Effects of Hexavalent Chromium on Brook Trout (Salvelinus fontinalis) and Rainbow Trout (Salmo gairdneri). Water Res. 10:497. (cited in EPA, 1980).

Bonde, J.P. and Ernst, E. 1992. Sex hormones and semen quality in welders exposed to hexavalent chromium. Human Exp. Toxicol. 11:259-263. (cited in ReproText, 1994).

Buikema, Jr., A.L., Cairns, Jr., J., and Sullivan, G.W. 1974. Evaluation of Philodena acuticornis (Rotifera) as a Bioassay Organism for Heavy Metals. Water Resour. Bull. 10:648-661. (cited in USFWS, 1986).

Chapman, G.A., Ota, S. and Recht, F. n.d. Effects of Water Hardness on the Toxicity of Metals to Daphnia magna. Available from: C.E. Stephan, U.S. EPA, Duluth, MN. (cited in EPA, 1987).

Ernst, E. and Bonde, J.P. 1992. Sex hormones and epididymal sperm parameters in rats following sub-chronic treatment with hexavalent chromium. Human Exp. Toxicol. 11:255-258. (cited in ReproText, 1994).

Friberg, L., Nordberg, G.F., and Vouk, V. (Eds.). 1986. Handbook on the Toxicology of Metals. Vols. I and II. Elsevier Science Publishers, Amsterdam, Holland. (cited in ReproText, 1994).

Garton, R.R. 1972. Biological Effect of Cooling Tower Blowdown. 71st Natl. Meet. Am. Inst. Chem. Eng. Jour., Dallas, TX. (cited in EPA, 1980).

Haseltine, S.D., Sileo, L., Hoffman, D.J., and Mulhern, B.M. 1985. Effects of Chromium on Reproduction and Growth of Black Ducks. Manuscript. (cited in USFWS, 1986).

Hatherill, J.R. 1981. A Review of the Mutagenicity of Chromium. Drug Chem. Toxicol. 4:185-195. (cited in USFWS, 1986).

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

REFERENCES
(Continued, Page 2 of 4)

Ivankovic, S. and Preussmann, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Food Cosmet. Toxicol. 13:347-351. (cited in IRIS, 1994).

Jouany, J.M., Vasseur, P., Ferard, J.F. 1982. Ecotoxicite Directe et Integree du Chrome Hexavalent sur Deux Niveaux Trophiques Associes: Chlorella vulgaris et Daphnia magna. Environ. Pollut. 27A:207-221. (cited USFWS, 1986).

Krishnaja, A.P. and Rege, M.S. 1982. Induction of Chromosomal Aberrations in Fish Boleophthalmus dussumieri after Exposure in vivo to Mitomycin C and Heavy Metals Mercury, Selenium and Chromium. Mutat. Res. 102:71-82. (cited in USFWS, 1986).

MacKenzie, R.D., Byerrum, R.U., Decker, C.F., Hoppert, C.A., and Langham, R.F. 1958. Chronic toxicity studies. II. Hexavalent and trivalent chromium administered in drinking water to rats. Amer. Med. Assoc. Arch. Ind. Health. 18:232-234. (cited in IRIS, 1994).

Mangi, J., Schmidt, K., Pankow, J., Gaines, L., and Turner, P. 1978. Effects of Chromium on Some Aquatic Plants. Environ. Pollut. 16:285-291. (cited in USFWS, 1986).

Murti, R., Omkar, O., and Shukla, G.S. 1983. Chromium Toxicity to a Freshwater Prawn Macrobrachium lamarrei (H.M. Edwards). Toxicol. Lett. 18:257-261. (cited in USFWS, 1986).

National Academy of Sciences (NAS). 1989. Recommended Dietary Allowances. 10th Ed. Food and Nutrition Board, National Research Council, Washington, DC.

Neff, J.M., Foster, R.S., and Slowey, J.F. 1978. Availability of Sediment-Absorbed Heavy Metals to Benthos with Particular Emphasis on Deposit-Feeding Infauna. Tech. Rep. D-78-42, U.S. Army Waterways Exp. Sta., Vicksburg, Mississippi. 286 pp. (cited in USFWS, 1986).

Negilski, D.S. 1976. Acute Toxicity of Zinc, Cadmium and Chromium to the Marine Fishes, Yellow-eye Mullet (Aldrichetta forsteri C. & V.) and Small-Mouthed Hardyhead (Atherinasoma microstoma Whitley). Aust. J. Mar. Freshwater Res. 27:137-149. (cited in USFWS, 1986).

REFERENCES
(Continued, Page 3 of 4)

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Pickering, O.H. 1980. Chronic Toxicity of Hexavalent Trivalent Chromium to the Fathead Minnow (Pimephales promelas). Arch. Environ. Contam. Toxicol. 9:405. (cited in EPA, 1980).

ReproText®. 1994. B.J. Dabney. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Riva, M.C., Flos, R., Crespi, M., and Balasch, J. 1981. Letal Potassium Dichromate and Whitening (blankophor) Exposure of Goldfish (Carassius auratus): Chromium Levels in Gills. Comp. Biochem. Physiol. 68C:161-165. (cited in USFWS, 1986).

Sauter, S., et al. 1976. Effects of Exposure to Heavy Metals on Selected Freshwater Fish. Toxicity of Copper, Cadmium, Chromium and Lead to Eggs and Fry of Seven Fish Species. EPA 600/3-76-105. Duluth, MN. (cited in EPA, 1980).

Stanley, R.A. 1974. Toxicity of Heavy Metals and Salts to Eurasian Water-milfoil (Myriophyllum spicatum L.) Arch. Environ. Contam. Toxicol. 2: 331. (cited in EPA, 1980).

Staub, R.J., et al. 1973. Effects of Industrial Effluents on Primary Phytoplankton Indicators. Tenn. Water Resources Res. Center. Res. Rep. 26. (cited in EPA, 1980).

Stevens, D.G. and Chapman, G.A. 1984. Toxicity of Trivalent Chromium to Early Life Stages of Steelhead Trout. Environ. Toxicol. Chem. 3:125-133. (cited in USFWS, 1986).

Steven, J.D., Davies, L.J., Stanley, E.K., Abbott, R.A., Ihnat, M., Bidstrup, L., and Jaworski, J.F. 1976. Effects of Chromium in the Canadian Environment. Nat. Res. Coun. Canada, NRCC No. 15017. 168 pp. Avail. from Publications, NRCC/CNRC, Ottawa, Canada, K1A OR6. (cited in USFWS, 1986).

REFERENCES
(Continued, Page 4 of 4)

Tandon, S.K., Saxena, D.K., Gaur, J.S., and Chandra, S.V. 1978. Comparative Toxicity of Trivalent and Hexavalent Chromium. Alterations in Blood and Liver. Environ. Res. 15:90-99. (cited in USFWS, 1986).

Teratogen Information System (TERIS). 1994. University of Washington, Seattle, WA. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality for Chromium. Office of Water Regulations and Standards. EPA 440/5-80-035. Washington, DC.

U.S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. Prepared by Clement Associates, Inc. Arlington, VA.

U.S. Environmental Protection Agency (EPA). 1992. Health Effects Assessment Summary Tables (HEAST). Annual FY 1992. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. OHEA ECAO-CIN-821.

U.S. Fish and Wildlife Service (USFWS). 1986. Polychlorinated Biphenyl Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Patuxent Wildlife Research Center, Laurel, MD. Biological Report 85(1.7).

U.S. Fish and Wildlife Service (USFWS). 1987. Habitat Suitability Index. Models: Burred Owl. Patuxent Wildlife Research Center, Laurel, MD. Biological Report 82(10.143).

Van der Putte, I., Brinkhorst, M.A., and Koeman, J.H. 1981. Effect of pH on the Acute Toxicity of Hexavalent Chromium to Rainbow Trout (Salmo gairdneri). Aquatic Toxicol. 1:129-142. (cited in USFWS, 1986).

Zarafonetis, J.H. and Hampton, R.E. 1974. Some Effects of Small Concentrations of Chromium on Growth and Phytosynthesis in Algae. Mich. Acad. 6:417. (cited in EPA, 1980).

Copper
(CAS No. 7440-50-8)

INTRODUCTION

Copper occurs naturally in many animals and plants and is an essential micronutrient for all living organisms, including humans (ATSDR, 1989). Copper is one of the most important industrial and commercial metals because of its durability, ductility, malleability, and electrical and thermal conductivity (ATSDR, 1989). In industry, copper is used primarily for the production of wire, brass, and bronze alloys, with the electrical industry being the primary users. It is also extensively used in agriculture in fertilizers and as bactericides and fungicides. Other agricultural uses include use as a feed additive, growth promoter, and an agent for disease control in livestock and poultry.

Copper may exist in two oxidation states: +1 or +2. Copper(+1) is unstable in aerated water over the pH range of most natural waters (6 to 8), oxidizing to the +2 state. In the aquatic environment, the fate of copper is determined by the formation of complexes, especially with humic substances, and sorption to hydrous metal oxides, clays, and organic materials. Sorption to these materials, both suspended in the water column and in the sediments, results in relative enrichment of the solid phase and reduction in the dissolved level (Shell, 1990). The amount of copper able to remain in solution is directly dependent on water chemistry, especially pH; temperature; and concentrations of other chemical species (Shell, 1990).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA does not provide any oral reference doses (RfDs), stating that available toxicity data were inadequate to calculate an RfD (EPA, 1993). EPA lists the current drinking water standard of 1.3 mg/L in place of the RfD (EPA, 1993). Assuming that a 70-kg individual drinks 2 liters per day (L/day) of water, this value is equivalent to 0.037 mg/kg/day. No inhalation reference concentrations (RfCs) or RfDs are provided in IRIS (1994) or HEAST (EPA, 1993).

EPA (1984) reported symptoms of dizziness, nausea, vomiting, and abdominal pain in humans exposed acutely (single exposure) to levels up to approximately 0.5 mg/kg-bw. No toxic effects were reported in rats exposed to copper for 16 months at levels up to approximately 80 mg/kg-bw/day. Chronically, the most studied human toxicity is Wilson's disease, which is an inherited autosomal recessive disorder of copper metabolism that causes increased copper deposition in the liver, kidneys, brain, and cornea (EPA, 1984), with copper levels in the liver of affected individuals reaching 20 times those of healthy individuals. Toxic effects associated with this disorder are damage to erythrocytes, kidneys, corneas, and the CNS, and may lead to death.

Subchronic inhalation data were not identified, but chronic inhalation exposure in humans is documented (IRIS, 1991). Known as vineyard sprayer's lung, exposure results from the use of copper sulfate on grapes to control mildew. Reported toxic effects included dyspnea, weakness, anorexia, weight loss, and the occurrence of radiographic opacities. These pulmonary opacities eventually regressed and then evidenced calcification.

Carcinogenicity

EPA lists copper as a group D (not classifiable) carcinogen (IRIS, 1994). The basis for this designation is a lack of human data, inadequate animal data from assays, and equivocal mutagenicity data.

Mutagenicity

Dose-related mutagenic effects were observed in Escherichia coli cultures at levels as low as 2 ppm copper sulfate (IRIS, 1994). Errors in DNA synthesis have been induced in viruses incubated with unreported concentrations of copper chloride or copper acetate (IRIS, 1994). Chromosomal aberrations were induced in isolated rat hepatocytes when incubated with copper sulfate (Sina et al., 1983). Casto et al. (1979) showed enhanced cell transformation in Syrian hamster embryo cells infected with simian adenovirus with the addition of cuprous sulfide and copper sulfate.

Teratogenicity/Reproductive Effects

Excessive systemic copper in human beings with untreated Wilson's disease appears to be associated with infertility or miscarriage although there have been reports of successful pregnancies in treated patients (Piussah and Mathieu, 1985; Biller et al., 1985). There is also a case report in which untreated Wilson's disease in a pregnant woman was associated with placental deposition of copper, neonatal hepatomegaly, and persistent elevation of hepatic enzymes at 1 year of age (Oga et al., 1993). It should be remembered that D-penicillamine, the treatment for Wilson's disease, has itself been associated with birth defects such as cutis laxa and mesenchymal abnormalities.

In normal pregnancies, maternal serum copper levels increase to 1.5-2.0 mg/L (ReproTox, 1994). Cord blood concentrations are lower than those in maternal blood, although the absolute amount of copper present varies with geographic location. Copper levels in the fetus increase with gestational age and most studies agree that maternal serum copper concentrations at term correlate with the weight of the fetus. It has been suggested that dietary sources of copper during pregnancy are inadequate and that supplemental copper is advisable. Elevations in placental and cord serum copper levels have been found in preeclampsia (ReproTox, 1994). A decrease in cord blood copper has been found in pregnancies complicated by pre-term premature rupture of the membranes. An increased maternal serum copper has been found to be a prognostic sign of high risk pregnancy, although one report found a low maternal copper level to be a predictor of high risk pregnancy. It is not clear from any of these reports whether copper may be a cause of the described adverse outcomes rather than a reflection of related factors such as nutrition. The maternal dose of copper at which metal levels will be toxic to the conceptus has not been established.

Copper has been used as a component for intrauterine contraceptive devices (IUDs), which may release about 70 ug copper per day into the uterus and surrounding tissues (Zipper et al., 1972). In vitro experiments with human cells have suggested that the

copper released from a copper containing IUD can inhibit cell growth (Jones et al., 1973). Conceptions have occurred with copper-containing IUDs in place (Barash et al., 1990; Edelman et al., 1979). In reports on the small number of pregnancies that occurred in the presence of copper-bearing IUDs, investigators have found elevated ceruloplasmin in the cord blood, spontaneous abortion of an anencephalic fetus, cleft palate, and other abnormalities, including limb defects (ReproTox, 1994). A recent clinical study analyzed the morphology and histology of 11 human embryos that were conceived in the presence of copper-releasing IUDs, and voluntarily aborted between 7 and 12 weeks. No signs of malformations or excessive copper uptake were detected in this group of conceptuses (Barash et al., 1990). Although the findings from this small sample are reassuring, they are not adequate to prove that intrauterine copper does not have adverse developmental effects. In the event of pregnancy with an IUD in place, the risk of spontaneous abortion is significantly increased, particularly if the IUD cannot be removed when pregnancy is confirmed (Edelman et al., 1979).

Copper is excreted in breast milk, but its concentration in milk is not altered by increasing the maternal intake of copper or by giving copper intravenously (ReproTox, 1994). Typically, levels in breast milk decline rapidly during the first week postpartum and more slowly after that, until stabilizing at about 0.2 ug/mL of milk after about 6 months of lactation (Casey et al., 1989).

Reproductive effects of copper in animals depend on the species studied. Incubation of fish eggs with copper gives rise to a marked increase in abnormalities of growth and development in the offspring (Ozoh, 1980; Birge and Black, 1979). Copper was not found to be teratogenic in chicks although it decreased collagen synthesis in embryonic chick bone in culture (Ridgeway and Karnofsky, 1952; Miyahara et al., 1984). Hamsters developed cardiac abnormalities when treated in utero with copper salts (DiCarlo, 1980; Ferm and Hanlon, 1974). This effect was more regularly seen with copper citrate than with copper sulfate. Copper treatment was embryolethal and teratogenic in mice (O'Shea and Kaufman, 1979). In rats, copper potentiated the

teratogenic effect of zinc deficiency while not being teratogenic itself (Reinstein et al., 1984).

ECOTOXICITY

Aquatic Organisms

In aquatic animals, mean acute toxicity values have been reported at concentrations as low as 6.5 μg/L for Daphnia magna in hard water to 10,200 μg/L for bluegill (Lepomis macrochirus) in hard water (Table 1) (EPA, 1985). Toxicity tends to decrease as hardness, alkalinity, and total organic carbon increase (EPA, 1985). Chronic toxicity values range from 3.9 μg/L for brook trout (Salvelinus tontinalis) to 60.4 μg/L for northern pike (Esox lucius) (Table 2). Water hardness has no apparent effect on chronic toxicity; however, acute-chronic ratios appear to be affected by hardness (EPA, 1985). Therefore, water hardness is incorporated into AWQC determination.

Although copper is known to inhibit photosynthesis and plant growth, toxicity data on individual species are not numerous. Copper concentrations from 1 to 8,000 μg/L have been shown to affect aquatic plant growth (Table 3). Aquatic plant sensitivity to copper toxicity appears to be similar to that of aquatic animals; therefore, water quality criteria for animal species appear to be adequate for the protection of aquatic plants (EPA, 1985).

Table 1. Acute Toxicity of Copper to Aquatic Animals

Species	Method	Chemical	Hardness (mg/L as $CaCO_3$)	LC50 or EC50 (μg/L)*	Species Mean Acute Value (μg/L)+	Reference#
Worm, Lumbriculus variegatus	S, U	Copper sulfate	30	150	242.7	Bailey and Liu, 1980
Tubificid worm, Limnodrilus hoffmeisteri	S, U	Copper sulfate	100	102	53.08	Wurtz and Bridges, 1961
Snail, Campeloma decisum	FT, M	Copper sulfate	35-55	1,700	1,877	Arthur and Leonard, 1970
Snail, Goniobasis livescens	S, M	Copper sulfate	154	390	166.2	Paulson et al., 1983
Cladoceran, Daphnia magna	S, U	Copper sulfate	226	200	--	Cabejszek and Stasiak, 1960
Cladoceran, Daphnia magna	S, U	Copper chloride	99	50	--	Adema and deGroot van Zijl, 1972
Cladoceran, Daphnia magna	S, U	Copper sulfate	45	10	--	Cairns et al., 1978
Amphipod, Gammarus pseudolimnaeus	FT, M	Copper sulfate	45	20	22.09	Arthur and Leonard, 1970
Amphipod, Gammarus pulex	R, U	Copper chloride	104	41	--	Stephenson, 1983

Table 1. Acute Toxicity of Copper to Aquatic Animals (Continued, Page 2 of 4)

Species	Method	Chemical	Hardness (mg/L as $CaCO_3$)	LC50 or EC50 (μg/L)*	Species Mean Acute Value (μg/L)+	Reference#
Crayfish, Orconectes rusticus	FT, M	Copper sulfate	100-125	3,000	1,397	Hubschman, 1967
Crayfish (larva), Procambarus clarkii	FT, M	--	17	720	1,990	Rice and Harrison, 1983
Damselfly, Unidentified	S, M	--	50	4,600	4,600	Rehwoldt et al., 1973
Stonefly, Acroneuria lycorias	S, M	Copper sulfate	40	8,300	10,240	Warnick and Bell, 1969
Caddisfly, Unidentified	S, M	--	50	6,200	6,200	Rehwoldt et al., 1973
Midge, Chironomus sp.	S, M	Copper sulfate	50	30	30.00	Rehwoldt et al., 1973
Rainbow Trout, Oncorhynchus mykiss	FT, M	Copper sulfate	30	19.9	--	Howarth and Sprague, 1978
Rainbow Trout, Oncorhynchus mykiss	FT, M	Copper sulfate	101	176	--	Howarth and Sprague, 1978
Rainbow Trout (smolt), Oncorhynchus mykiss	FT, M	Copper chloride	23	29	--	Chapman, 1975, 1978

Table 1. Acute Toxicity of Copper to Aquatic Animals (Continued, Page 3 of 4)

Species	Method	Chemical	Hardness (mg/L as $CaCO_3$)	LC50 or EC50 (μg/L)*	Species Mean Acute Value (μg/L)+	Reference#
Rainbow Trout (adult), Oncorhynchus mykiss	FT, M	Copper chloride	42	57	--	Chapman, 1975; Chapman and Stevens, 1978
Rainbow Trout (fry), Oncorhynchus mykiss	FT, M	Copper nitrate	--	253	--	Hale, 1977
Rainbow Trout (fry), Oncorhynchus mykiss	FT, M	Copper nitrate	125	200	--	Spear, 1977; Anderson and Spear, 1980a
Fathead minnow, Pimephales promelas	S, U	Copper sulfate	20	50	--	Tarzwell and Henderson, 1960
Fathead minnow, Pimephales promelas	FT, M	Copper sulfate	202	460	--	Pickering et al., 1977
Fathead minnow, Pimephales promelas	FT, M	--	45	200	--	Andrew, 1976
Fathead minnow, Pimephales promelas	S,U	Copper sulfate	360	1,760	--	Pickering and Henderson, 1966
Brown bullhead, Ictalurus nebulosus	FT, M	Copper sulfate	202	170	--	Brungs et al., 1973
Striped bass, Morone saxatillis	S, U	Copper sulfate	35	620	--	Wellborn, 1969

Table 1. Acute Toxicity of Copper to Aquatic Animals (Continued, Page 4 of 4)

Species	Method	Chemical	Hardness (mg/L as $CaCO_3$)	LC50 or EC50 (μg/L)*	Species Mean Acute Value (μg/L)+	Reference#
Striped bass (larva), Morone saxatillis	S, U	Copper sulfate	34.5	25	--	Hughes, 1973
Pumpkinseed, Lepomis gibbosus	FT, M	Copper sulfate	125	1,940	--	Spear, 1977; Anderson and Spear, 1980b
Bluegill Lepomis macrochirus	FT, M	Copper sulfate	45	1,100	--	Benoit, 1975
Bluegill Lepomis macrochirus	S, U	Copper sulfate	20	200	--	Tarzwell and Henderson, 1960
Bluegill Lepomis macrochirus	FT, M	Copper chloride	200	320	86.67	Geckler et al., 1976
Orangethroat darter, Etheostoma spectabile	FT, M	Copper sulfate	200	850	230.2	Geckler et al., 1976

*Results are expressed as copper, not as the chemical.
+Freshwater Species Mean Acute Values are calculated at a hardness of 50 mg/L using the pooled slope.
#All references are cited in EPA, 1985.

Source: ESE.

Table 2. Chronic Toxicity of Copper to Aquatic Animals

Species	Test*	Chemical	Hardness (mg/L as $CaCO_3$)	Limits (μg/L)†	Chronic Value (μg/L)**	Reference#
Snail, Campeloma decisum	LC	Copper sulfate	35-55	8-14.8	10.88	Arthur and Leonard, 1970
Cladoceran, Daphnia magna	LC	Copper chloride	51	11.4-16.3	13.63	Chapman et al., Manuscript
Cladoceran, Daphnia magna	LC	Copper chloride	104	20-43	29.33	Chapman et al., Manuscript
Cladoceran, Daphnia magna	LC	Copper chloride	211	7.2-12.6	9.525	Chapman et al., Manuscript
Amphipod, Gammarus pseudolimnaeus	LC	Copper sulfate	45	4.6-8	6.066	Arthur and Leonard, 1970
Caddisfly, Clistornia magnifica	LC	Copper chloride	26	8.3-13	10.39	Nebeker et al., 1984
Rainbow Trout Oncorhynchus mykiss	ELS	Copper sulfate	45.4	11.4-31.7	19.01	McKim et al., 1976
Brook Trout Salvelinus tontinalis	LC	Copper sulfate	45	9.5-17.4	12.86	McKim and Benoit, 1971
Brook Trout Salvelinus tontinalis	ELS	Copper sulfate	45.4	22.3-43.5	31.15	McKim et al., 1978

Table 2. Chronic Toxicity of Copper to Aquatic Animals (Continued, Page 2 of 3)

Species	Test*	Chemical	Hardness (mg/L as $CaCO_3$)	Limits (μg/L)†	Chronic Value (μg/L)**	Reference#
Brook Trout Salvelinus tontinalis	ELS	Copper sulfate	37.5	3-5	3.873	Sauter et al., 1976
Brook Trout Salvelinus tontinalis	ELS	Copper sulfate	37.5	3-5	3.873	Sauter et al., 1976
Northern pike, Esox lucius	ELS	Copper sulfate	45.4	34.9-104.4	60.36	McKim et al., 1978
Bluntnose minnow, Pimephales notatus	LC	Copper sulfate	194	4.3-18	8.798	Horning and Neiheisel, 1979
Fathead minnow, Pimephales promelas	LC	Copper sulfate	198	14.5-33	21.87	Mount, 1968
Fathead minnow, Pimephales promelas	LC	Copper sulfate	30	10.6-18.4	13.97	Mount and Stephan, 1969
Fathead minnow, Pimephales promelas	ELS	--	45	13.1-26.2	18.53	Lind et al., Manuscript
White sucker, Catostomus commersoni	ELS	Copper sulfate	45.4	12.9-33.8	20.88	McKim et al., 1978
Bluegill, Lepomis macrochirus	LC	Copper sulfate	45	21-40	28.98	Benoit, 1975

Table 2. Chronic Toxicity of Copper to Aquatic Animals (Continued, Page 3 of 3)

---

*LC = life cycle or partial life cycle; ELS = early life stage.
†Results are expressed as copper, not as the chemical.
#All references are cited in EPA, 1985.

Source: ESE.

Table 3. Toxicity of Copper to Aquatic Plants

Species	Effect	Result (µg/L)	Reference#
Alga, Anabaena flos-aqua	75-percent growth inhibition	200	Young and Lisk, 1972
Alga, Anabaena strain 7120	Lag in growth	64	Laube et al., 1980
Alga, Ankistrodesmus braunil	Growth reduction	640	Laube et al., 1980
Alga, Chlamydomonas sp.	Growth reduction	8,000	Cairns et al., 1978
Alga, Chlorella pyrenoidosa	Lag in growth	1	Steeman-Nielsen and Wium-Andersen, 1970
Alga, Chlorella regularis	Lag in growth	20	Sakaguchi et al., 1977
Alga, Chlorella saccharophila	96-hour EC50	550	Rachlin et al., 1982
Alga, Chlorella sp.	Photosynthesis inhibited	6.3	Gachter et al., 1973
Alga, Chlorella vulgaris	96-hour IC50	62	Ferard et al., 1983
Alga, Chlorella vulgaris	33-day EC50 (growth)	180	Rosko and Rachlin, 1977
Alga, Scenedesmus acuminatus	40-percent growth reduction	300	Stokes and Hutchinson, 1976
Algae, Mixed culture	Significant reduction in photosynthesis	5	Elder and Horne, 1978
Blue green algae, Mixed culture	50-percent reduction in photosynthesis	25	Steeman-Nielsen and Bruun-Laursen, 1976
Diatom, Navicula incerta	4-day EC50	10,450	Rachlin et al., 1983
Diatom, Nitzschia linearis	5-day EC50	795-815	Academy of Natural Sciences, 1960; Patrick et al., 1968

Table 3. Toxicity of Copper to Aquatic Plants (Continued, Page 2 of 2)

Species	Effect	Result (μg/L)	Reference#
Diatom, Nitzschia palea	Complete growth inhibition	5	Steeman-Nielsen and Wium-Anderson, 1970
Duckweed, Lemna minor	7-day EC50	119	Walbridge, 1977
Macrophyte, Elodea canadensis	50-percent reduction in photosynthesis	150	Brown and Rattigan, 1979
Eruasian watermilfoil, Myriophyllum spicatum	32-day EC50 (root weight)	250	Stanley, 1974
Green alga, Selenastrum capricornutum	14-day EC50 (cell volume)	85	Christensen and Scherfig, 1979
Blue alga, Microcystis aeruginosa	Incipient inhibition	30	Bringmann, 1975; Bringmann and Kuhn, 1976, 1978a, 1978b

#All references are cited in EPA, 1985.

Source: ESE.

Copper does not appear to bioaccumulate in the edible portion of freshwater aquatic organisms. Bioconcentration factors (BCFs) range from 1 for the bluegill (Lepomis macrochirus) to 2,000 for the alga Chlorella regularis (Table 4).

Terrestrial Organisms

In livestock, two species are associated with potential copper toxicity. Sheep are susceptible to acute and chronic copper toxicosis. Acute toxicity, often the result of improper administration of anthelminthic compounds, has been reported at doses of approximately 200 mg/kg. Symptoms include vomiting, excessive salivation, abdominal pain, and diarrhea (a greenish-tinged, fluid feces); collapse and death may follow within 24 to 48 hours (Osweiler et al., 1985). Fatalities were reported in lambs exposed to feed with 27 mg/kg copper (Tait et al., 1971), and a milk substitute with 9 mg/L was fatal to lambs (Nilson, 1968). Chronic toxicity results from the rapid metabolism and storage of copper in the liver until a level sufficient to disrupt storage is reached, usually by the inhibition of vital enzyme systems. At that point, large quantities of copper are released back into the bloodstream, potentially leading to a hemolytic crisis. This, in turn, causes kidney failure and may lead to liver lesions (Clarke and Clarke, 1975). Applying an uncertainty factor of 500 to the acute lethal value of 27 mg/kg in the diet provides a reference daily intake of 0.054 mg/kg/day. Assuming that a 30-kg lamb drinks 3 L/day of water, a water value of 0.025 mg/L (25 μg/L) may be calculated that is expected to be protective of livestock. Assuming that a lamb would ingest soil at a rate of 0.16 percent of its body weight, a soil concentration of 35 mg/kg may be calculated.

Swine evidence a higher incidence of copper toxicosis due to the practice of providing young pigs diets high in copper to increase daily weight gains (Osweiler et al., 1985). Swine exposed to dietary levels of 250 mg/kg copper sulfate (2.6 mg/kg-bw/day) for 79 days suffered liver damage, as evidenced by elevated serum liver enzyme and jaundice (ESE, 1989). Using an uncertainty value of 50 for a subchronic LOAEL and the weight and consumption values for a market pig, this is equivalent to a water

Table 4. Bioaccumulation of Copper by Aquatic Organisms

Species	Tissue	Duration (days)	Bioconcentration Factor	Reference#
Alga, Chlorella regularis	--	20 hours	2,000	Sakaguchi et al., 1977
Alga, Chroococcus paris	--	10 min.	up to 4,000	Les and Walker, 1984
Asiatic clam, Corbicula fluminea	Soft tissue	28	17,700-22,600	Graney et al., 1983
Cladoceran, Daphnia magna	Whole body	7	471*	Winner, 1984
Stonefly, Pteronarcys californica	--	14	203	Nehring, 1976
Fathead minnow (larva), Pimephales promelas	--	30	290	Lind et al., Manuscript
Bluegill, Lepomis macrochirus	Muscle	660	1.0	Benoit, 1975

*Bioconcentration factor was converted from dry weight to wet weight basis.
#All references are cited in EPA, 1985.

Source: ESE.

concentration of 0.4 mg/L (400 μg/L). Because the values derived for the lamb are more protective, they will be used for the livestock and wildlife.

Because copper is an essential micronutrient for plant nutrition, most adverse effects result from copper deficiency (Adriano, 1986). Toxicity has been associated with soil copper concentrations of 150 to 400 mg/kg. The most common toxic effects are reduced growth vigor, poorly developed and discolored root system, and leaf chlorosis. In addition, copper toxicity causes stunting, reduced branching, thickening, and unusually dark coloration in the rootlets of many plants.

STANDARDS AND CRITERIA

Human Receptors

EPA has established a nonenforceable Maximum Contaminant Level (MCL) Goal (MCLG) of 1.3 mg/L for copper is based on potential adverse effects (gastrointestinal) reported in human studies (IRIS, 1994). EPA has concluded that setting an MCL for copper is not feasible and believes that the treatment approach contained in the final rule (corrosion control, source water reduction, public education and copper service line replacement) will achieve the public health goals of the SDWA without the problems associated with establishing MCLs (IRIS, 1994). A secondary MCL of 1 mg/L has been proposed for copper based on organoleptic considerations (IRIS, 1994). No ambient water quality criteria (AWQC) for the protection of human health from the consumption of water and aquatic organisms contaminated with copper have been established (IRIS, 1994).

No national ambient air quality standards (NAAQS) have been developed for copper under the Clean Air Act (CAA). However, the Occupational Safety and Health Administration (OSHA) has promulgated an enforceable permissible exposure limit (PEL) for worker exposure (NIOSH, 1994), while the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable

recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values are presented in Table 5.

Ecological Receptors

AWQC for the protection of freshwater and marine organisms against exposure to copper have been established by EPA (IRIS, 1994) are presented in Table 6.

Table 5. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	1 (Dusts, mists, and copper compounds, except fumes)	1 (Dusts, mists, and copper compounds, except fumes)	1 (Dusts, mists, and copper compounds, except fumes)
STEL	--	--	--
CL	--	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.

STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.

CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.

c = designates that this chemical is regulated as a potential carcinogen.

s = signifies that dermal absorption may be a significant exposure route for this chemical.

Source: ESE.

Table 6. Ambient Water Quality Criteria

AWQC (μg/L)	Freshwater	Marine
Acute	9.2[a]	2.9
Chronic	6.5[b]	--

[a]The acute freshwater AWQC for copper is hardness-dependent. The 1-hour average concentration in μg/L of copper should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(0.9422 * [\ln(hardness)] - 1.464)}$$

[b]The chronic freshwater AWQC for copper is hardness-dependent. The 4-day average concentration in μg/L of copper should not exceed more than once every 3 years on the average the numerical value given by:

$$e^{(0.8545 * [\ln(hardness)] - 1.465)}$$

At a hardness of 50 mg/L as $CaCO_3$, the acute and chronic criteria are 9.2 and 6.5 μg/L, respectively (IRIS, 1994).

Source: ESE.

REFERENCES

Adriano, D.C. 1986. Trace Elements in the Terrestrial Environment. Springer-Verlag, NY.

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Draft Toxicological Profile for Copper. Prepared by Syracuse Research Corporation. U.S. Public Health Service, Atlanta, GA.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Barash, A. et al. 1990. Development of human embryos in the presence of a copper intrauterine device. Gynecol. Obstet. Invest. 29:203-6. (cited in ReproTox, 1994).

Biller, J. et al. 1985. Successful pregnancy in Wilson's disease: a case report and review of the literature. Europ. Neurol. 24:306-9. (cited in ReproTox, 1994).

Birge, W.J. and Black, W.A. 1979. Effects of copper on embryonic and juvenile stages of aquatic animals. Copper Environ. 2:373-99. (cited in ReproTox, 1994).

Casey, C.E. et al. 1989. Studies in human lactation: secretion of zinc, copper and manganese in human milk. Amer. Jour. Clin. Nutr. 49:773-85. (cited in ReproTox, 1994).

Casto, B.C., Meyers, J., and DiPaolo, J.A. 1979. Enhancement of viral transformation for evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. Cancer Res. 39:193-198. (cited in IRIS, 1994).

Clarke, E.G.C., and Clarke, M.L. 1975. Veterinary Toxicology. 1st Ed. Balliere Tindall, London and MacMillan Publishing Co., Inc., New York, NY.

DiCarlo, F.J. 1980. Syndromes of cardiovascular malformations induced by copper citrate in hamsters. Teratol. 21:89-101. (cited in ReproTox, 1994).

Edelman, D.A. et al. 1979. Intrauterine devices and their complications. G.K. Hall & Co., Boston, MA. (cited in ReproTox, 1994).

REFERENCES
(Continued, Page 2 of 4)

Environmental Science and Engineering, Inc. (ESE). 1989. Biota Remedial Investigation. Final Report, Vol. II. Prepared for: Office of the Program Manager, Rocky Mountain Arsenal Contamination Cleanup. Gainesville, FL.

Ferm, V.H. and Hanlon, D.P. 1974 Toxicity of copper salts in hamster embryonic development. Biol. Reprod. 11:97-101. (cited in ReproTox, 1994).

Integrated Risk Information System (IRIS). 1991. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 9, Expires 7/31/91. Managed by Micromedex, Inc.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 22, Expires 10/31/94. Managed by Micromedex, Inc.

Jones, R.W. et al. 1973. Effect of copper-containing intrauterine devices on human cell in culture. Brit. Med. Jour. ii:520-22. (cited in ReproTox, 1994).

Miyahara, T. et al. 1984. Interaction between cadmium and copper in relation to the collagen metabolism of embryonic chick bone in tissue culture. Toxicol. Appl. Pharmacol. 75:479-84. (cited in ReproTox, 1994).

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 22, Expires 10/31/94. Managed by Micromedex, Inc., Denver, CO.

Oga, M., Matsui, N., Anai, T., Yoshimatsu, J., Inoue, I., Miyakawa, I. 1993. Copper disposition of the fetus and placenta in a patient with untreated Wilson's disease. Amer. Jour. Obstet. Gynecol. 169:196-8. (cited in ReproTox, 1994).

O'Shea, K.S. and Kaufman, M.H. 1979. Influence of copper on the early post-implantation mouse embryo: an *in vivo* and *in vitro* study. Arch. Devel. Biol. 186:297-308. (cited in ReproTox, 1994).

REFERENCES
(Continued, Page 3 of 4)

Osweiler, G.D., Carson, T.L., Buck, W.B., and Van Gelder, G.A. 1985. Clinical and Diagnostic Veterinary Toxicology. 3rd Ed. Kendall/Hunt Publishing Co., Dubuque, IA.

Ozoh, P.T. 1980. Effects of reversible incubations of zebrafish eggs inn copper and lead ions with or without shell membranes. Bull. Environ. Contam. Toxicol. 24:270-5. (cited in ReproTox, 1994).

Piussah, C. and Mathieu, M. 1985. Teratogenic risk during treatment of Wilson's disease. Jour. Genet. Hum. 33:357-62. (cited in ReproTox, 1994).

Reinstein, N.H. et al. 1984. Zinc-copper interactions in the pregnant rat: fetal outcome and maternal and fetal zinc, copper and iron. Jour. Nutr. 114:1266-79. (cited in ReproTox, 1994).

Reproductive Toxicology System (ReproTox®). 1994. Reproductive Toxicology Center, Columbia Hospital for Women Medical Center, Washington, DC. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 22, Expires 10/31/94. Managed by Micromedex, Inc., Denver, CO.

Ridgeway, L.P. and Karnofsky, D.A. 1952. The effect of metals on the chick embryo: toxicity and production of abnormalities in development. Ann. NY Acad. Sci. 55:203-15. (cited in ReproTox, 1994).

Shell Chemical Company (Shell). 1990. Shell Toxicity Profile. In: Human Health Exposure Assessment for Rocky Mountain Arsenal, Toxicity Assessment, Vols. IIa and IIIa. Prepared by Ebasco Services, Inc.

Sina, J.F., Bean, C.L., Dysart, G.R., Taylor, V.I., and Bradley, M.O. 1983. Evaluation of the alkaline elution/rat hepatocyte assay as a predictor of carcinogenic/mutagenic potential. Mutat. Res. 113(5):357-391. (cited in IRIS, 1994).

Tait, R.M., Krishnamurti, C.R., Gilchrist, E.W., and MacDonald, K. 1971. Chronic Copper Poisoning in Feeder Lambs. Can. Vet. Jour., 12:73-75.

U.S. Environmental Protection Agency (EPA). 1979. Environmental Fate of 129 Priority Pollutants, Volume II. Washington D.C. EPA 440/4-79-029b.

REFERENCES
(Continued, Page 4 of 4)

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Copper. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 540/1-86.

U.S. Environmental Protection Agency (EPA). 1985. Ambient Water Quality Criteria for Copper--1984. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-84-031.

U.S. Environmental Protection Agency (EPA). 1993. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1993. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540-R-93-058. NTIS No. PB93-921199.

Zipper, J. et al. 1972. Development of a new type of intrauterine contraceptive. In: New Concepts in Contraception. W. Potts, Ed. Medical and Technical Publishing Co. Ltd., Oxford, England. (cited in ReproTox, 1994).

1,2-DICHLOROETHANE
(CAS No. 107-06-2)

INTRODUCTION

The compound 1,2-dichloroethane, which represents the largest volume of chlorinated VOC produced in the United States, is used primarily as a starting material in the manufacture of vinyl chloride and other chlorinated solvents such as tetrachloroethene; trichloroethene; 1,1,1-trichloroethane; and 1,1-dichloroethene (ORNL, 1989). Minor applications include use as a cleaning solvent and degreaser, a grain fumigant, a wetting agent, and a lead-scavenging agent in gasoline, as well as uses in upholstery and paints (ORNL, 1989).

1,2-Dichloroethane is expected to be highly mobile in the soil/groundwater system because it is soluble in water (8,690 mg/L at 20°C) and has a K_{oc} of 14 L/kg (ORNL, 1989). The estimated K_{oc} value of 14 indicates that it will not be strongly bound to soils (ORNL, 1989). Based on a vapor pressure of 63.7 torr at 20°C, transport of 1,2-dichloroethane vapors through the air-filled pores of unsaturated soils followed by photochemical oxidation in the atmosphere is an important loss mechanism for near-surface contaminated soils (ORNL, 1989). In saturated subsurface soils (where soil organic carbon and soil air are negligible), a large fraction of the 1,2-dichloroethane (approximately 94 percent) is expected to be present in the soil-water phase and transported with flowing groundwater (ORNL, 1989). The compound 1,2-dichloroethane is a chlorinated aliphatic; therefore, it is not rapidly metabolized in the environment, although it can be degraded by acclimated microbial populations (ORNL, 1989). Under normal environmental conditions, 1,2-dichloroethane is not expected to undergo rapid hydrolysis (ORNL, 1989). Thus, groundwater underlying 1,2-dichloroethane-contaminated soils with low organic content may be highly vulnerable to contamination.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA has not derived any chronic or interim subchronic oral reference doses (RfDs) or inhalation reference concentrations (RfCs) for 1,2-dichloroethane (IRIS, 1995; EPA, 1994). However, EPA's Environmental Criteria and Assessment Office (ECAO) has developed a provisional chronic oral RfD and an interim inhalation RfD that have been adopted by EPA Region III (EPA, 1995) and are presented in Table 1. Based on a subchronic oral no-observed-adverse-effect level (NOAEL) in rats of 26 mg/kg/day (NTP, 1991) and applying an uncertainty factor of 1,000 (10 for sensitive human subpopulations, 10 for animal-to-human extrapolation, and 10 for extrapolation from a subchronic study to a chronic endpoint), a provisional chronic oral RfD of 0.03 mg/kg/day is derived (EPA, 1993). In addition, an interim chronic inhalation RfD of 2.86×10^{-3} mg/kg/day has been developed by ECAO and has been recommended by EPA Region III (EPA, 1995).

Short-term human inhalation exposure to 1,2-dichloroethane has resulted in headaches, dizziness, nausea, vomiting, abdominal pain, irritation of the mucous membranes, and systemic injury to the liver, kidney, and lungs (ORNL, 1989). Several human poisonings by ingestion of 1,2-dichloroethane are reported in the literature. Most of these cases were fatal, with death attributed to respiratory and circulatory failure (ORNL, 1989). Prolonged skin contact may result in dermatitis and, in severe cases, moderate edema and necrosis can develop (ORNL, 1989).

Short-term inhalation studies of rats and mice exposed to 1,2-dichloroethane have resulted in CNS depression (i.e., inactivity or stupor) and organic and histopathological changes in the liver and kidneys (ORNL, 1989). Oral LD_{50} values of 670, 860, and 5,700 mg/kg have been reported for rats, rabbits, and dogs, respectively (RTECS, 1995). Exposure to 1,2-dichloroethane vapors has

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	0.03	Low	Kidney lesions	EPA, 1993	1,000 C,H,S
Subchronic	0.3	Low	Kidney lesions	EPA, 1993	100 H,S
Inhalation					
Chronic	0.00286	--	Unknown	EPA, 1995	Unknown
Subchronic	--	--	--	--	--

Note: RfD = reference dose.
RfC = reference concentration.
-- = Not available.
NOAEL = no observed adverse effect level.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic exposure.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

resulted in reversible clouding of the corneas of dogs and foxes but not of other species (ORNL, 1989). Although 1,2-dichloroethane is absorbed through the skin, large doses are required to elicit acute systemic poisoning (i.e., the dermal LD_{50} in rabbits was calculated to be 2,800 mg/kg) (ORNL, 1989).

Chronic exposures to 1,2-dichloroethane in an occupational environment have been associated with effects similar to those observed for short-term exposures (ORNL, 1989). Although fatal cases occur less frequently than from acute exposures, chronic effects can progress unless the exposures are reduced (ORNL, 1989).

Carcinogenicity

Carcinogenic slope factors (CSFs) and supporting information are summarized in Table 2. EPA has classified 1,2-dichloroethane as a group B2 (probable human) carcinogen for the oral and inhalation exposure routes (IRIS, 1995). This classification was selected because sufficient evidence exists to show carcinogenicity in animals, but no data concerning the carcinogenicity in humans exist (IRIS, 1995). An oral cancer slope factor (CSF) of 0.091 $(mg/kg/day)^{-1}$ was determined based on the results of an oral animal study by NCI (1978) in which rats and mice were gavaged for 78 weeks with either 47 and 95 mg/kg/day (rats), 97 and 195 mg/kg/day (male mice), and 149 and 299 mg/kg/day (female mice). 1,2-Dichloroethane produced carcinomas of the forestomach and hemangiosarcomas of the circulatory system (male rats), mammary adenocarcinomas (female rats), lung adenomas, and hepatocellular carcinomas (mice). Using route-to-route extrapolation, EPA (IRIS, 1995) has adopted the oral CSF for the inhalation route as well.

Mutagenicity

The compound 1,2-dichloroethane has been found to be genotoxic in the Ames assay and has also induced sex-linked recessive lethal mutations in *Drosophila melanogaster* (ORNL, 1989). However, 1,2-dichloroethane administered in

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.091	B2	Stomach, Circulatory System, Liver	IRIS, 1995
Inhalation	0.091	B2	Stomach, Circulatory System, Liver, Lung	EPA, 1994

Note: B2 = probable human carcinogen (adequate evidence of carcinogenicity in animals and insuffucuent evidence in humans).

Source: ESE.

drinking water to mice failed to produce dominant lethal mutations at concentrations of 50 mg/kg (ORNL, 1989). In testing with human cell lines, 1,2-dichloroethane was identified as a direct-acting mutagen, the observed differential in sensitivity was attributed to varying levels of glutathione S-transferase levels (HSDB, 1995). Unactivated 1,2-dichloroethane has been identified as a weak mutagen, but when activated with glutathione, it becomes a powerful mutagen for several life forms, showing both DNA repair mutations and sex chromosome loss and nondisjunction (OHM/TADS, 1995). Some tester strains of Salmonella typhimurium evidenced mutagenic activity with and without an activator, however, no transformations were reported in mouse hepatocytes exposed to levels as high as 250 mg/L (HSDB, 1995).

Teratogenicity/Reproductive Effects

No embryotoxicity, fetotoxicity, or reproductive effects were observed or attributed to 1,2-dichloroethane in chronic inhalation studies with pregnant rats and rabbits, however, maternal toxicity was observed in both species (ORNL, 1989). Although parental exposure of rats indicated no adverse effects on the reproductive capacity of the adults or on growth and survival of the offspring (ORNL, 1989), a reduction was observed in the size of the litter, the number of live births, fetal weight, and vitality of the pups (OHM/TADS, 1995). Surviving first generation females, when bred, demonstrated prolonged estrus periods and high perinatal mortality (OHM/TADS, 1995).

ECOTOXICITY

Aquatic Organisms

Aquatic species most sensitive to 1,2-dichloroethane exposure are members of the class crustacea. The 24- and 48-hour LC_{50} values for Daphnia magna are 250,000 and 220,000 μg/L, respectively (EPA, 1985), and the NOEL is reported to be 68,000 μg/L (LeBlanc, 1980). The 96-hour LC_{50} values for the bluegill and the fathead minnow are 430,000 and 116,000 μg/L, respectively, and the 7-day LC_{50} value for the guppy (Poccilia reticulata) is 106,000 μg/L under static

conditions (EPA, 1980). As a chronic test, a concentration of 29,000 to 59,000 μg/L was estimated as the acceptable concentration in a 32-day early life stage test with fathead minnows (EPA, 1980). Body weight of the juveniles was reduced at concentrations of 59 mg/L in water. Survival of juveniles, percentage of normal larvae at hatch, and the hatchability of the embryos were not affected by the exposure concentrations (EPA, 1985). EPA (1987) reports acute and chronic LOEC values of 118,000 and 20,000 μg/L, respectively. Applying an uncertainty factor of 10 to the chronic LOEC provides a water TRV of 2,000 μg/L for aquatic organisms.

Bioconcentration of 1,2-dichloroethane in aquatic species is unlikely, due to its physical and chemical properties. In a tracer study, a BCF of 2 was established for bluegill in flowing water (WHO, 1987). The half-life for the elimination of 1,2-dichloroethane from fish tissues was 1 to 2 days (WHO, 1987).

Terrestrial Organisms

Little information is available concerning the toxicity of 1,2-dichloroethane to vegetation. It has been reported to retard the growth and development of plants and to interfere with seedling development. A vapor concentration of 3 milligrams per cubic meter (mg/m^3) was lethal and mutagenic for barley seeds over a 24-hour exposure period (Ehrenberg et al., 1974). In other cases, it has induced morphological and chlorophyll mutations, resulting in necrosis and atrophy of the plant, however, exposure levels were not presented (HSDB, 1995). The compound 1,2-dichloroethane is nontoxic to many species of economically important plants (Gast and Early, 1956) but is toxic to invertebrates when used as a fumigant (EPA, 1985). Data were unavailable for invertebrate toxicity in relation to soil concentrations. Because of inadequate data, no soil or water TRVs for vegetation can be determined.

No information was found regarding the toxicity of 1,2-dichloroethane to ruminant livestock. One study using pigs identified an air concentration of

3,000 ppm as the lowest concentration at which a lethal effect was reported (LC_{LO}) following a 7-hour exposure (HSDB, 1995). Chickens provided a diet containing 250 or 500 mg/kg 1,2-dichloroethane exhibited decreased egg weights at both levels and decreased numbers of eggs and feed intake at the higher level (Alumot et al., 1976, WHO, 1987). No other information was identified regarding toxicity to livestock or terrestrial wildlife.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 3. Due to the ranking of 1,2-dichloroethane as a Group B2 human carcinogen, EPA has set a nonenforceable Maximum Contaminant Level Goal (MCLG) for drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 5 μg/L, which is enforceable for public water supplies (50 FR 46880). Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 9.4 to 0.094 μg/L for consumption of contaminated drinking water and aquatic organisms (IRIS, 1995). For consumption of aquatic organisms only, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 2,430 to 24.3 μg/L, respectively (IRIS, 1995). These AWQC would yield values for human consumption of water alone (W) of 0.094, 0.0094, and 0.00094 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

Although no official reference doses are available for 1,2-dichloroethane, health-based drinking water advisory levels are available. For children, a long-term health advisory value of 0.74 mg/L was developed by applying an uncertainty

Table 3. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day[a]	10-Day[a]	Longterm[a]	Lifetime	Fish and Water	Fish Only	Water Only[b]
1,2-Dichlroethane	5	0	--	740	740	740	--	0.94[c]	243[c]	0.94[c]

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is for a 10-kg child.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

factor of 100 to the NOAEL of 7.4 mg/kg/day derived from a combination of three inhalation studies involving rats, guinea pigs, rabbits, and monkeys that were exposed to concentrations ranging from 100 to 500 ppm. At the higher levels, symptoms included pulmonary congestion, myocarditis, and fatty degeneration of the liver, kidney, heart, and adrenal gland (IRIS, 1995).

No national ambient air quality standards (NAAQS) have been developed for 1,2-dichloroethane. However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1995). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1995; ACGIH, 1993). These values are presented in Table 4.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to 1,2-dichloroethane (IRIS, 1995). However, the lowest levels observed to have an effect (LOELs) on the aquatic species tested have been determined (IRIS, 1995; EPA, 1987). These LOELs, which are established when the minimum data required to derive AWQCs are not available, are presented in Table 5.

Table 4. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	4 c	4 c	40 c
STEL	8	8	--
CL	--	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.

STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.

CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.

c = designates that this chemical is regulated as a potential carcinogen.

Source: ESE.

Table 5. Ambient Water Quality Criteria

LOEL (µg/L)	Freshwater	Marine
Acute	118,000	113,000
Chronic	20,000	--

Source: ESE.

REFERENCES

Alumot, E., Meidler, M., Holstein, P., and Herzberg, M. 1976. Tolerance and daily intake of ethylene dichloride in the chicken diet. Food and Cosmet. Toxicol. 14:111-114. (cited in WHO, 1987).

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Ehrenberg, Z., Osterman-Golkar, S., Singh, D., and Lundqvist, U. 1974. On the reaction kinetics and mutagenic activity of methylating and beta-halogenoethylating gasoline additives. Radiation Bot. 15:184-194. (cited in WHO, 1987).

Gast, R. and Early, J. 1956. Phytotoxicity of solvents and emulsifiers used in insecticide formulations. Agric. Chem. 11:42-45, 136-139. (cited in EPA, 1985).

Hazardous Substances Data Bank (HSDB). 1995. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

LeBlanc, G.A. 1980. Acute toxicity of priority pollutants to water flea (Daphnia magna). Bull. of Environ. Contam. and Toxicol. 24:684-691. (cited in EPA, 1985).

National Cancer Institute (NCI). 1978. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 55. Washington, DC. DHEW Publ. No. (NIH) 78-1361.

National Toxicology Program (NTP). 1991. Toxicity studies of 1,2-dichloroethane (ethylene dichloride) (CAS No. 107-06-2) in F344/N rats, Sprague-Dawley rats, Osborne-Mendel rats, and B6C3F1 mice (drinking water and gavage studies). NTP TOX 4. DHHS Publication No. (NIH) 91-3123. NTIS No. PB91-185363.

REFERENCES
(Continued, Page 2 of 3)

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1995. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

Oil and Hazardous Materials/Technical Assistance Data System (OHM/TADS). 1995. U.S. Environmental Protection Agency. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Registry of Toxic Effects of Chemical Substances (RTECS). 1995. National Institute of Occupational Safety and Health (NIOSH). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-80-029. NTIS No. PB81-117400

U.S. Environmental Protection Agency (EPA). 1985. Health Assessment Document for 1,2-Dichloroethane (Ethylene Dichloride). Office of Health and Environmental Assessment. Washington, D.C. EPA 600/8-84-006F.

U.S. Environmental Protection Agency (EPA). 1987. Quality Criteria for Water (1986); Update Number 2. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-86-0001.

U.S. Environmental Protection Agency (EPA). 1993. Risk Assessment Issue Paper for Derivation of a Provisional Oral RfD for 1,2-Dichloroethane (CASRN 107-06-2). Superfund Technical Support Center, Environmental Criteria and Assessment Office, Cincinnati, OH. No. 93-22.

REFERENCES
(Continued, Page 3 of 3)

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. EPA 540/R-94/020. Publication No. 9200.6-303(94-1). NTIS No. PB94-921199.

U.S. Environmental Protection Agency (EPA). 1995. Risk-Based Concentration Table, January - June 1995. Technical Support Section, EPA Region III, Philadelphia, PA. March 7, 1995.

World Health Organization (WHO). 1987. Environmental Health Criteria No. 62. 1,2-Dichloroethane. World Health Organization, Geneva, Switzerland.

MERCURY

(Inorganic; CAS No. 7439-97-6)
(Methylmercury; CAS No. 22967-92-6)

INTRODUCTION

Although mercury occurs naturally in the environment, it is also found in several manufactured products, including pharmaceuticals (Shell, 1990). It is used in agriculture as a fungicide and seed dressing for grains and flower bulbs and as a foliar spray against plant diseases (Adriano, 1986).

Mercury is found in most soil types, existing as a large variety of minerals. The most common are the sulfides cinnabar and meta-cinnabar, and the majority of mercury recovered from the earth comes from meta-cinnabar (Adriano, 1986). Mercury is found in one of three stable oxidation states: 0, +1, and +2. Elemental mercury is rare; however, the other forms may be transformed to elemental mercury. Mercury also exists in nature in the form of inorganic or organic complexes or as stable mercuric sulfides.

Mercury can migrate by the volatilization from aquatic and terrestrial sources through the reduction of metallic mercury to complexed species and by the deposition of HgS in reducing sediments. Atmospheric transport is a major environmental distribution pathway. Wet and dry precipitation are both important mechanisms for the removal of mercury from the atmosphere (EPA, 1985). Once mercury is in the atmosphere, photolysis is important in the breakdown of airborne mercurials and may be important in some aquatic systems.

Sorption to suspended and bed sediments is one of the most important processes determining the fate of mercury in aquatic systems; sorption is the strongest for the Hg^{+2} species onto organic materials. As a result, mercury is generally complexed to organic compounds and is not readily leached from either organic-

rich or mineral-rich soils (Rosenblatt et al., 1975). Most mercury compounds can be remobilized in aquatic systems by microbial conversion to methyl and dimethyl forms. Conditions reported to enhance microbial conversion include large amounts of available mercury, large numbers of bacteria, absence of strong complexing agents, near neutral pH, high temperatures, and moderately aerobic conditions.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA is reviewing final chronic RfDs for mercury and does not provide values in IRIS (1994). Interim oral chronic and subchronic RfDs for elemental mercury are available from EPA (1994) and are presented in Table 1. In addition, EPA (1994) provides interim chronic and subchronic inhalation reference concentrations (RfCs), which are also listed in Table 1.

Acute oral toxicity is dependent on the form of the absorbed compound. Inorganic monovalent salts are relatively nontoxic due to poor absorption. Generally, divalent salts are more toxic than monovalent, and ionizable salts are more toxic than those that do not ionize. Symptoms associated with ingestion of ionizable salts include pharyngitis, loosening of the teeth, abdominal pain, nausea, vomiting, diarrhea, and shock, followed by nephritis, anuria, and hepatitis (Shell, 1990). CNS effects are also associated with mercury exposure. The effects of (elemental) mercury vapor are neuropsychiatric in nature, with exposed individuals evidencing extreme shyness, insomnia, and emotional instability; tremors induced through voluntary movement are also routinely observed (Hammond and Beliles, 1980). Methyl mercury toxicity is more sensory, with vision and motor reflexes the primary areas of effect (Hammond and Beliles, 1980). Symptoms reported for exposed animals include the onset of ataxia and incoordination, hypersensitivity, salivation, tremors, electrocardiogram changes, tonic-clonic convulsions, followed by coma and death (Hatch, 1977).

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	3E-4	--	Renal	EPA, 1994	1,000 C,H,S
Subchronic	3E-4	--	--	EPA, 1994	--
Inhalation					
Chronic	8.6E-5[c]	--	CNS	EPA, 1994	10 (3) S
Subchronic	8.6E-5[c]	--	--	EPA, 1994	--

Note:
CNS = central nervous system.
RfD = reference dose.
RfC = reference concentration.
-- = No EPA Review.

[a]Uncertainty factors are multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.

[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various data deficiencies.

[c]Based on an RfC of 3E-4 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

No chronic human ingestion data are available; however, rats exposed to dietary levels of mercury acetate resulting in intake levels starting at 2 mg/kg/day evidenced morphological changes in the kidney (Shell, 1990).

Acute effects resulting from inhalation of mercury vapor include acute pneumonitis and lung tissue damage as well as abdominal pain, diarrhea, headache, gingivitis, and lip and cheek ulceration (Shell, 1990). Occupational studies indicate that chronic inhalation exposure to mercury vapor primarily affects the CNS and the kidneys (proteinuria); the latter effect has also been reported by the NRCC (1977). At high exposure levels, extreme irritability, excitability, delirium, hallucinations, anxiety, and manic depressive psychosis have been reported (Shell, 1990).

Carcinogenicity

EPA currently classifies mercury as a group D (not classifiable) carcinogen (IRIS, 1994). The basis for this classification is the lack of human data available and the insufficiency of animal and other supporting data. Rats were injected intraperitoneally over a 2-week period with metallic mercury and then observed for their lifetimes. Sarcomas were only seen in those tissues that had come into direct contact with the metal (IRIS, 1994). Mice provided dietary levels of 0, 15, or 30 mg/kg feed for up to 78 weeks evidenced mortality at the highest exposure level. At the 15-mg/kg level, 13 of 16 males evidenced renal tumors, while one adenoma was identified among 37 control animals surviving past 53 weeks, and none was found in either control or exposed females (IRIS, 1994).

Mutagenicity

The mutagenic responses of nonmammalian cell cultures to mercury salts in vitro were inconclusive (Shell, 1990). Chromosomal aberrations have been observed in the lymphocytes of humans occupationally exposed to mercury vapors.

Teratogenicity/Reproductive Effects

Reproductive and developmental effects in humans are not well known, although case studies indicate menstrual disturbances as well as spontaneous abortions and infant mortality among women exposed to mercury vapors during pregnancy (Shell, 1990). Fetal fatalities and severe neurological effects among surviving neonates were observed following prenatal exposure (Shell, 1990). The evaluation of teratogenic effects of mercury vapor is inconclusive, although parental administration of inorganic mercury salts in experimental animals has produced fetal abnormalities (ATSDR, 1987).

ECOTOXICITY

Aquatic Organisms

Toxicity data are available for three classes of mercury: divalent inorganic mercury, methylmercury, and other compounds (chiefly organic). The data suggest that freshwater plants appear to be more sensitive to methylmercury than to other forms of mercury (see Table 2). For the species shown, the alga Chlorella vulgaris was the most sensitive at a concentration range of 0.8 to 4.0 μg/L in a 15-day EC_{50} growth test.

USFWS (1987) reports that certain species of macrophytes can strongly affect the cycling of mercury. For a freshwater ecosystem (Florida Everglades), Tripp and Harris (1976) indicate that mangrove vegetation plays an important role in mercury cycling. More research is needed on the role of higher plants in the mercury cycle.

When toxicity data for selected species of mercury were compared (EPA, 1985), the cladocerans Daphnia magna and Daphnia pulex were the most sensitive organisms when exposed to concentrations of divalent inorganic mercury (see Table 3). Invertebrates such as the mayfly, stonefly, caddishly, and damselfly were almost three orders of magnitude more resistant. Fishes were sensitive (compared with invertebrates) at 30 μg/L (guppy), 160 μg/L (bluegill), and 275 μg/L (rainbow trout) as presented in Table 4.

Table 2. Toxicity of Mercury to Freshwater Aquatic Plants

Species	Chemical	Effect	Result (µg/L)*	Reference+
MERCURY (II)				
Chlorella vulgaris (Alga)	Mercuric chloride	LC_{50}	100 to 1,000	Gipps and Biro, 1978
Chlorella vulgaris (Alga)	Mercuric chloride	LC_{50}	148 to 296	Rai, 1979; Rai et al., 1981
Microcystis aeruginosa (Blue Alga)	Mercuric chloride	8-day incipient inhibition	5	Bringmann, 1975; Bringmann and Kuhn, 1976, 1978a, b
Myriophylium spicatum (Eurasian watermilfoil)	Mercuric chloride	32-day EC_{50} (root weight)	3,400	Stanley, 1974
METHYLMERCURY				
Anabaena flos-aquae (Alga)	Methylmercuric chloride	EC_{50} (growth)	6.0	Thomas and Montes, 1978
Chlorella vulgaris (Alga)	Methylmercuric chloride	15-day EC_{50} (growth)	0.8 to 4.0	Rai et al., 1981
OTHER MERCURY COMPOUNDS				
Anabaena flos-aquae (Alga)	Phenylmercuric acetate	EC_{50} (growth)	2.8	Thomas and Montes, 1978

*Results are expressed as mercury, not as the chemical.
+References cited are from EPA, 1985.

Source: ESE.

Table 3. Acute Toxicity of Mercury to Freshwater Aquatic Organisms (Page 1 of 3)

Species	Method	Chemical	LC_{50} or EC_{50} (µg/L)*	References+
MERCURY (II)				
Daphnia magna (Cladoceran)	S, U	Mercuric chloride	2.217	Canton and Adema, 1978
Daphnia pulex (Cladoceran)	S, U	Mercuric chloride	2.217	Canton and Adema, 1978
Gammarus sp. (Amphipod)	S, M	Mercuric nitrate	10	Rehwoldt et al., 1973
Branchlura sowerbyl (Tubificid worm)	R, U	Mercuric chloride	80	Chapman et al., 1982
Chironomus sp. (Midge)	S, M	Mercuric nitrate	20	Rehwoldt et al., 1973
Ephemerella subvaria (Mayfly)	S, U	Mercuric chloride	2,000	Warnick and Bell, 1969
(Unidentified) Damselfly	S, M	Mercuric nitrate	1,200	Rehwoldt et al., 1973
Acroneuria lycorias (Stonefly)	S, U	Mercuric chloride	2,000	Warnick and Bell, 1969
Hydropsyche betteni (Caddisfly)	S, U	Mercuric chloride	2,000	Warnick and Bell, 1969
Amnicola sp. [Snail (adult)]	S, M	Mercuric nitrate	80	Rehwoldt et al., 1973
Faxonella clypeatus (Crayfish) (male, mixed ages)	R, M	Mercuric chloride	20	Heit and Fingerman, 1977; Heit, 1981
Pimephales promelas (Fathead minnow)	FT, M	Mercuric chloride	150	Call et al., 1983

Table 3. Acute Toxicity of Mercury to Freshwater Aquatic Organisms (Continued, Page 2 of 3)

Species	Method	Chemical	LC_{50} or EC_{50} (µg/L)*	References+
MERCURY (II), cont.				
Oncorhynchus mykiss [Rainbow trout (juvenile)]	FT, M	Mercuric chloride	275	Lock and vanOverbeeke, 1981
Gambusia affinis [Mosquitofish (female)]	S, U	Mercuric chloride	180	Joshi and Rege, 1980
Poecilla reticulata [Guppy (116 - 157 mg)]	R, U	Mercuric chloride	30	Deshmukh and Marathe, 1980
Lepomis macrochirus [Bluegill (juvenile)]	S, U	Mercuric chloride	160	Holcombe et al., 1983
METHYLMERCURY				
Salvelinus fontinalis [Brook trout (yearling)]	FT, M	Methylmercuric chloride	65	McKim et al., 1976
Salvelinus fontinalis [Brook trout (juvenile)]	FT, M	Methylmercuric chloride	84	McKim et al., 1976
Oncorhynchus mykiss [Rainbow trout (juvenile)]	R, U	Methylmercuric chloride	25	Matida et al., 1971

Table 3. Acute Toxicity of Mercury to Freshwater Aquatic Organisms (Continued, Page 3 of 3)

Species	Method	Chemical	LC_{50} or EC_{50} (µg/L)*	References+
METHYLMERCURY, cont.				
Oncorhynchus mykiss [Rainbow trout (larva)]	R, U	Methylmercuric chloride	24	Wobeser, 1973
OTHER MERCURY COMPOUNDS				
Oncorhynchus mykiss [Rainbow trout (juvenile)]	R, U	Phenylmercuric acetate	5	Matida et al., 1971
Pimpehales promelas (Fathead minnow)	S, M	Mercuric acetate	40	Curtis et al., 1979; Curtis and Ward, 1981
Carassius auratus (Goldfish)	S, U	Phenylmercuric lactate	82	Ellis, 1947
Cyprinus carplo (Common carp)	R, U	2-methhoxy ethyl mercuric choride	139	Das and Misra, 1982

Note: FT = flow through. S = static. R = renewal.
U = unmeasured. M = measured.

*Results are expressed as mercury, not as the chemical.
+References cited are from EPA, 1985.

Source: ESE.

Table 4. Chronic Toxicity of Mercury to Freshwater Aquatic Organisms

Species	Test	Chemical	Limits (µg/L)	Chronic Value (µg/L)	Reference*
DIVALENT INORGANIC MERCURY					
Daphnia magna (Cladoceran)	LC+	Mercuric chloride	0.72 to 1.28	0.96	Biesinger et al., 1982
Pimephales promelas (Fathead minnow)	ELS	Mercuric chloride	<0.23	<0.23	Call et al., 1983
METHYL MERCURY					
Daphnia magna (Cladoceran)	LC+	Methylmercuric chloride	0.52 to 0.87	0.6726	Biesinger et al., 1982
Salvelinas fontinalis (Brook trout)	LC	Methylmercuric chloride	0.19 to 0.93	0.5193	McKim et al., 1976
OTHER MERCURY COMPOUNDS					
Daphnia magna (Cladoceran)	LC+	Phenylmercuric acetate	1.12 to 1.90	1.459	Biesinger et al., 1982

*References cited are from EPA (1985).
+Renewal.

Source: ESE.

Only two species (rainbow trout and brook trout) were tested for methylmercury toxicity. Rainbow trout (larvae and juveniles) were the most sensitive species to acute toxicity (24 and 25 μg/L, respectively). Juvenile and yearling brook trout were slightly more resistant, having acute values of 84 and 64 μg/L, respectively. For selected other mercury compounds (see Table 3), the rainbow trout again was the most sensitive species tested (LC_{50} value of 5 μg/L).

Results of chronic tests performed on selected invertebrate and vertebrate species with mercury compounds are presented in Table 4. The brook trout was the most sensitive species (value of 0.5193 μg/L) to chronic methyl mercury toxicity. The cladoceran Daphnia magna was slightly more resistant, having a chronic value of 0.67 μg/L. Only one species, Daphnia magna, was used in chronic tests for other mercury compounds. The value was 1.46 μg/L (see Table 4).

The biological half-life of mercury in fish is approximately 2 to 3 years, which results in a high BCF for fish (EPA, 1985). Depuration of mercury is slow, and long-term reduction in the concentration of mercury in fish tissue is due largely to dilution by the growth of additional tissue. In animals of higher trophic levels, the transfer efficiency of mercury is greater than at lower levels (USFWS, 1987). Accumulation patterns were enhanced or modified by numerous biological and abiotic factors. In general, mercury accumulation was enhanced by elevated water temperatures; reduced water salinity or hardness; reduced water pH; increased age of the organism; reduced organic matter content of the medium; and the presence of zinc, cadmium, or selenium in solution.

Table 5 presents mercury bioaccumulation data for selected freshwater animal species. In whole-body tests, the rainbow trout and fathead minnow had BCFs of 11,000 and 44,130 to 81,670, respectively, for methylmercuric chloride. Brook trout tested with the same compound for muscle tissue produced a BCF of 11,000 to 33,000. Consistent with acute and chronic toxicity results, methylmercury is more toxic to subject organisms than inorganic mercury and

Table 5. Bioaccumulation of Mercury by Freshwater Aquatic Organisms

Species	Tissue	Chemical	Duration (days)	BCF	Reference*
Pimephales promelas (Fathead minnow)	Whole body	Mercuric chloride	287	4,994	Snarski and Olson, 1982
Oncorhyncus mykiss (Rainbow trout)	Whole body	chloride	60	11,000	Boudou and Ribeyre, 1984
Salvelinus fontinalis (Brook trout)	Muscle	Methylmercuric chloride	273	11,000 to 33,000	McKim et al., 1976
Pimephales promelas (Fathead minnow)	Whole body	Methylmercuric chloride	336	44,130 to 81,670	Olson et al., 1975

*References cited are from EPA (1985).

Source: ESE.

other mercury compounds.

Table 6 contains toxicity data for selected amphibian species in 7-day EC_{50} tests, a 5-day EC_{50} test, and two chronic tests (17 days and 4 months, respectively). For 7-day EC_{50} tests on frogs/toads, the narrow-mouthed toad was the most sensitive at 1 to 1.3 μg/L. The southern gray treefrog and the squirrel treefrog were slightly more resistant at 2.4 μg/L, and Fowler's toad and the pig frog (65.9 and 67.2 μg/L, respectively) were the most resistant. In general, the data suggest that treefrogs are the most susceptible specimens to exposure to mercuric chloride.

Terrestrial Organisms

Plants are relatively insensitive to the toxic effects of mercury and may accumulate high levels in tissues before any effects are seen (ESE, 1989). Soil levels as low as 50 mg/kg may lead to severe pre-emergence loss of new shoots, especially among sensitive species such as lettuce and carrots and the dicotyledons, which include the bean family and other legumes. Conversely, the monocotyledons, including most of the grains and grasses, are much less sensitive, evidencing little or no effect at soil concentrations of 50 to 100 mg/kg. Toxic effects reported include growth inhibition, yield reduction, and reduction of chlorophyll content (Adriano, 1986). Suggested mechanisms of action include the inactivation of biochemical and physiological processes. Mercury is reported to inhibit protein synthesis in plant leaves and may affect the water-absorbing and transporting mechanisms in plants. Treatment of plants with mercuric chloride disrupted and reduced photosynthetic activity in plants. Metabolic activity may be affected as a result of structural changes in enzymes resulting from the strong affinity mercury has for sulfur-containing thiol and sulfhydryl groups (Adriano, 1986).

Grains appear to translocate mercury to straw and foliage at levels similar to soil levels; however, grain levels are lower. Generally, vegetable crops may take up

Table 6. Toxicity of Mercury Compounds to Selected Amphibians (Page 1 of 3)

Species	Chemical	Duration	Effect	Result (µg/L)*	Reference+
Rana grylio (Pig frog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	67.2	Birge et al., 1979
Rana heckscheri) (River frog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	59.9	Birge et al., 1979
Rana pipiens (Leopard frog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	7.3	Birge et al., 1979
Gastrophryne carolinensis (Narrow-mouthed toad) (embryo,larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	1.0 to 1.3	Birge et al., 1978, 1979
Bufo debilis (Green toad) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	40.0	Birge et al., 1979
Bufo fowleri (Fowler's toad) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	65.9	Birge et al., 1979
Bufo punctatus (Red-spotted toad) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	36.8	Birge et al., 1979
Acris crepitans (Northern cricket frog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	10.4	Birge et al., 1979

Table 6. Toxicity of Mercury Compounds to Selected Amphibians (Continued, Page 2 of 3)

Species	Chemical	Duration	Effect	Result (µg/L)*	Reference+
Hyla chrysoscelis (Southern gray treefrog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	2.4	Birge et al., 1979
Hyla crucifer (Spring peeper) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	2.8	Birge et al., 1979
Hyla gratlosa (Barking treefrog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	2.5	Birge et al., 1979
Hyla squirella (Squirrel treefrog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	2.4	Birge et al., 1979
Hyla versicolor (Gray treefrog) (embryo, larva)	Mercuric chloride	7 days	EC_{50} (death and deformity)	2.6	Birge et al., 1979
Xenopus laevis (African clawed frog)	Mercuric chloride	11 months	Substantial mortality	0.16 to 0.2	Birge et al., 1978
Xenopus laevis (African clawed frog)	Mercuric chloride	48 hours	LC_{50}	74	Sloof and Baerselman, 1980; Slooff et al., 1983
Rana pipiens (Leopard frog)	Methylmercuric chloride	4 months	Failure to metamorphose	1 to 10	Chang et al., 1974
Rana pipiens (Leopard frog) (blastula embryo)	Methylmercuric chloride	5 days	LC_{50}	12 to 16	Dial, 1976

Table 6. Toxicity of Mercury Compounds to Selected Amphibians (Continued, Page 3 of 3)

Species	Chemical	Duration	Effect	Result (µg/L)*	Reference+
Rana pipiens (Leopard frog) (gastrula embryo)	Methylmercuric chloride	5 days	LC_{50}	8 to 12	Dial, 1976
Rana pipiens (Leopard frog) (neural plate embryo)	Methylmercuric chloride	5 days	LC_{50}	12 - 16	Dial, 1976
Rana pipiens (Leopard frog) (gastrula embryo)	Methylmercuric chloride	96 hours	EC_{50} (teratogenesis)	8 - 12	Dial, 1976
Rana pipiens (Leopard frog) (neural plate embryo)	Methylmercuric chloride	96 hours	EC_{50} (teratogenesis)	12	Dial, 1976
Triturus viridescens (Newt)	Methylmercuric chloride	>2 days	Delayed limb regeneration	8	Chang et al., 1976
Triturus viridescens (Newt)	Methylmercuric chloride	17 days	Death	300	Chang et al., 1976
Triturus viridescens (Newt)	Methylmercuric chloride	8 days	Death	1,000	Chang et al., 1976

*Results are expressed in mercury, not in the chemical.
+References cited are from EPA, 1985.

Source: ESE.

mercury into the roots, but there is little evidence of translocation to aerial portions of the plant; certain soil types such as gravelly loam soils are exceptions, and certain plants may accumulate mercury (Adriano, 1986). Considering the severe pre-emergence loss to be equivalent to a 50-percent growth reduction, an uncertainty factor of 10 may be applied to the 50-mg/kg soil value to derive a soil concentration of 5 mg/kg that is expected to be protective of vegetation.

Table 7 presents chronic toxicity data for two species of worms (tiger worms and earthworms) exposed to methylmercury compounds. Earthworms were most sensitive, with 50-percent mortality at a compound concentration in soil of 0.79 mg/kg and 100-percent mortality at 5.0 mg/kg in 60-day exposure tests (Abbasi and Soni, 1983). Tiger worms were more resistant, with 21-percent mortality at 5.0 mg/kg and 100-percent mortality at 25.0 mg/kg.

USFWS (1987) provides toxicity data for bird species following exposure to various compounds of mercury (see Table 8). In mallards, deaths occurred between 4 and 48 hours; in pheasants, deaths occurred between 4 and 6 days.

Hill (1981) found that methylmercury was always more toxic than inorganic mercury in tests with coturnix and that young birds were usually more sensitive than older birds. As with aquatic organisms, mercury toxicity to birds varies with the form of the compound, dose, route of administration, species, sex, age, physiological condition, and abiotic conditions. For example, in northern bobwhite chicks fed diets containing methylmercury chloride, mortality was significantly lower than when the solvent was acetone than when it was another carrier such as propylene glycol or corn oil (Spann et al., 1986). Organomercury compounds interact with higher temperatures and pesticides, such as DDE and parathion, to produce additive or greater than additive toxicity and with selenium to produce less than additive toxicity (Fimreite, 1979).

Effects of organomercury compounds to selected mammalian species provided by

Table 7. Toxicity of Mercury Compounds to Selected Invertebrates

Species	Compound	Concentration in Soil (mg/kg)	Exposure	Effect	Reference*
Eisenia foetida (Tiger worm)	Methylmercury	25.0	12 weeks	100 percent dead	Beyer et al., 1985
Eisenia foetida (Tiger worm)	Methylmercury	5.0	~12 weeks	21 percent dead	Beyer et al., 1985
Octochaetus pattoni (Earthworm)	Methylmercury	0.79	60 days	50 percent dead	Abbasi and Soni, 1983
Octochaetus pattoni (Earthworm)	Methylmercury	5.0	60 days	100 percent dead	Abbasi and Soni, 1983

*References cited are from USFWS, 1987.

Source: ESE.

Table 8. Toxicity of Mercury Compounds to Birds (Page 1 of 3)

Species	Treatment	Compound	Concentration	Exposure	Effect	Reference*
Anas platyrhynchos (Mallard)	Acute oral	Methyl	2.2 to 23.5 mg/kg-bw	14 days	LD50	Hudson et al., 1984
	Acute oral	Ethyl	75.7 mg/kg-bw	14 days	LD50	Hudson et al., 1984
	Acute oral	Phenyl	524.7 mg/kg-bw	14 days	LD50	Hudson et al., 1984
Colinus virginianus (Northern bobwhite)	Acute oral	Methyl	23.8 mg/kg-bw	14 days	LD50	Hudson et al., 1984
Coturnix coturnix coturnix (Coturnix)	Acute oral	Methyl	11.0 to 27.0 mg/kg-bw	14 days	LD50	Hill, 1981
	Acute oral	Inorganic	26.0 to 54.0 mg/kg-bw	14 days	LD50	Hill, 1981
Coturnix japonica (Japanese quail)	Acute oral	Methyl	14.4 to 33.7 mg/kg-bw	14 days	LD50	Hill and Soares, 1984
	Acute oral	Ethyl	21.4 mg/kg-bw	14 days	LD50	Hudson et al., 1984
	Acute oral	Inorganic	31.1 mg/kg-bw	14 days	LD50	Hill and Soares, 1984
Columba livia (Rock dove)	Acute oral	Ethyl	22.8 mg/kg-bw	14 days	LD50	Hudson et al., 1984
Gallus domesticus (Domestic chicken)	Acute oral	Phenyl	60.0 mg/kg-bw	14 days	LD50	Mullins et al., 1977
Passer domesticus (House sparrow)	Acute oral	Methyl	12.6 to 37.8 mg/kg-bw	14 days	LD50	Hudson et al., 1984
Perdix perdix (Gray partridge)	Acute oral	Ethyl	17.6 mg/kg-bw	14 days	LD50	Hudson et al., 1984

Table 8. Toxicity of Mercury Compounds to Birds (Continued, Page 2 of 3)

Species	Treatment	Compound	Concentration	Exposure	Effect	Reference*
Phasianus colchicus (Ring-necked pheasant)	Acute oral	Ethyl	11.5 mg/kg-bw	14 days	LD50	Hudson et al., 1984
	Acute oral	Methyl	11.5 to 26.8 mg/kg-bw	14 days	LD50	Hudson et al., 1984
	Acute oral	Phenyl	65.0 to 101.0 mg/kg-day	14 days	LD50	Mullins et al., 1977 Hudson et al., 1984
Coturnix	Dietary	Inorganic	32.0 mg/kg	9 weeks	LD-0	Hill, 1981
	Dietary	Inorganic	2,956 to 5,086 mg/kg	5 to 7 days	LD50	Hill, 1981
	Dietary	Methyl	4.0 mg/kg	9 weeks	LD-0	Hill, 1981
	Dietary	Methyl	8.0 mg/kg	5 days	Some deaths	Hill and Soares, 1984
Ring-necked pheasant	Dietary	Ethyl	4.2 mg/kg	70 days	LD-0+	Spann et al., 1972
	Dietary	Ethyl	12.5 mg/kg	70 days	LD50	Spann et al., 1972
	Dietary	Ethyl	37.4 mg/kg	28 days	LD50	Spann et al., 1972
	Dietary	Ethyl	112.0 mg/kg	15 days	LD50	Spann et al., 1972
Japanese quail	Dietary	In ethanol, methanol, or water	500 mg/kg-bw	28 days	LD-55	El-Begearmi et al., 1980
Mallard	Applied to egg surface	Methyl	9.0 µg/egg	Single dose	Some deaths	Hoffman and Moore, 1979

Table 8. Toxicity of Mercury Compounds to Birds (Continued, Page 3 of 3)

Species	Treatment	Compound	Concentration	Exposure	Effect	Reference*
Chicken	In drinking water	Inorganic	500.0 mg/L	3 days	Some deaths	Grissom and Thaxton, 1985

*References cited are fom USFWS, 1987.

Source: ESE.

USFWS (1987) are shown in Table 9. Larger animals such as mule deer appear to be more resistant to mercury than smaller mammals such as mink, cats, and dogs; the reasons for this difference are unknown but are thought to be related to differences in metabolism and rates of detoxification.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 10. EPA has promulgated an enforceable (for public water supplies) Maximum Contaminant Level (MCL) of 0.002 mg/L (IRIS, 1994). Based on potential adverse effects (renal toxicity) EPA has promulgated the MCL Goal (MCLG) at this same level. This MCLG is based upon a DWEL of 0.01 mg/L and an assumed drinking water contribution of 20 percent (IRIS, 1994).

No national ambient air quality standards (NAAQS) have been developed for mercury under the Clean Air Act (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to mercury (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values are presented in the Table 11.

TWA is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect. The short-term exposure limit, or STEL, is the 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any any time during a workday even if the 8-hour TWA is not exceeded. The ceiling level, or CL, unless otherwise specified is the concentration that should not be exceeded at any any time during a workday. The "s" after the criteria

Table 9. Toxicity of Organomercury Compounds to Selected Mammalian Species (Page 1 of 2)

Species	Dose	Administration	Effects	Reference*
Canis familiaris (Dog)	0.1 to 0.25 mg/kg-bw during entire pregnancy	Oral	High incidence of stillbirths	Khera, 1979
Felis domesticus (Cat)	0.25 mg/kg-bw daily for 90 days (total 80 to 90 mgHg)	Dietary	Mean survival time 78 days. Convulsions starting at day 68; all with signs by day 90. Liver residues of survivors were 40.2 and 18.1 mg/kg fresh weight for total mercury and inorganic mercury, respectively	Eaton et al., 1980
Sus spp. (Pig)	0.5 mg/kg-bw during pregnancy	Oral	High incidence of stillbirths	Khera, 1979
Mustela vison (Mink)	1.0 mg/kg	Dietary	Fatal to 100 percent in about 2 months	Sheffy and St. Amant, 1982
Lutra canadensis (River otter)	>2.0 mg/kg	Dietary	Fatal	Kucera, 1983

Table 9. Toxicity of Organomercury Compounds to Selected Mammalian Species (Continued, Page 2 of 2)

Species	Dose	Administration	Effects	Reference*
Mink	5.0 mg/kg	Dietary	All dead in 30 to 37 days. Elevated residues in kidney (37.7 mg/kg fresh weight) and liver (55.6) prior to death	Sheffy and St. Amant, 1982
Odocoileus hemionus hemionus (Mule deer)	17.88 mg/kg body weight	Single oral dose	LD50	Hudson et al., 1984

*References cited are from USFWS, 1987.

Source: ESE.

Table 10. Drinking Water Criteria and Standards (mg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day[a]	10-Day[a]	Longterm[a]	Lifetime	Fish and Water	Fish Only	Water Only[b]
Mercury (Inorganic)	0.002	0.002	--	--	--	--	--	0.000144	0.000146	0.0105

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is for a 10-kg child.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

Table 11. Air Quality Criteria

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA (s)	0.01 (alkyl) 0.05 (vapor)	0.01 (alkyl) 0.05 (vapor)	0.1 (inorganic) 0.01 (alkyl)
STEL (s)	0.03 (alkyl)	0.03 (alkyl)	0.03 (alkyl)
CL	--	--	--

ACGIH has posted the following TLVs (mg/m^3) for mercury in their 1993 Notice of Intended Changes:

alkylmercury componds: TWA, 0.01; STEL, 0.03;
arylmercury compounds: TWA, 0.1; and
inorganic compounds (including vapors): TWA, 0.025.

values designate that the listed agencies consider exposure through the skin, mucous membranes, and/or eyes to be a potential significant exposure route.

Ecological Receptors

EPA ambient water quality criteria (AWQCs) for the protection of freshwater and marine organisms against exposure to mercury are provided by EPA (IRIS, 1994) and are presented in the following table.

AWQC (µg/L)	Freshwater	Marine
Acute	2.4	2.1
Chronic	0.012	0.025

REFERENCES

Abbasi, S.A., and Soni, R. 1983. Stress-Induced Enhancement of Reproduction in Earthworms Octochaetus pattoni Exposed to Chromium (VI) and Mercury III)--Implications in Environmental Management. Int. Jour. Env. Stud., 22:43-47. Cited in USFWS, 1987a.

Adriano, D.C. 1986. Trace Elements in the Terrestrial Environment. Springer-Verlag, NY.

Agency for Toxic Substances and Disease Registry (ATSDR). 1987. Draft Toxicity Profile on Mercury. In: Superfund Chemical Profiles. Vol. I. U.S. Public Health Service. Washington, DC.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 0-936712-99-6.

Beyer, W.N., Cromartie, E., and Moment, G.B. 1985. Accumulation of Methymercury in the Earthworm, Eisenia fcetida, and Its Effect on Regeneration. Bull. Env. Contam. Toxicol. 35:157-162. Cited in EPA, 1987a.

Biesinger, K.E., et al. 1982. Chronic Effects of Inorganic and Organic Mercury on Daphnia magna: Toxicity, Accumulation, and Loss. Arch. Env. Contam. Toxicol. 11:769. Cited in EPA, 1985b.

Birge, W.J., et al. 1978. Embryo-Larval Bioassays on Inorganic Coal Elements and in situ Biomonitoring of Coal-Waste Effluents. In: D.E. Samuel, et al. (eds), Surface Mining and Fish/Wildlife Needs in the Eastern United States. PB 298353. Cited as EPA, 1985b.

Birge, W.J., et al. 1979. The Effects of Mercury on Reproduction of Fish and Amphibians. In: J.O. Nriagu (ed), The Biogeochemistry of Mercury in the Environment. Elsevier, New York. Cited in EPA, 1985b.

Boudou, A. and Ribeyre, F. 1984. Influence of Exposure Length on the Direct Bioaccumulation of Two Mercury Compounds by Salmo gairdneri (fry) and the Relationship Between Organism Weight and Mercury Concentration. Water Res. 18:81. Cited in EPA, 1985b.

Bringmann, G. 1975. Determination of the Biologically Harmful Effect of Water Pollutants by Means of the Retardation of Cell Proliferation of the blue Algae Microcystis. Gesundheits-Ing. 96:238. Cited in EPA, 1985b.

REFERENCES
(Continued, Page 2 of 7)

Bringmann, G. and Kuhn, R. 1976. Comparative Results of the Damaging Effects of Water Pollutants Against Bacteria (Pseudomonas putida) and Blue Algae (Microcystis aeruginosa). Gas-Wasserfach, Wasser-Abwasser 117:410. Cited in EPA, 1985b.

Bringmann, G. and Kuhn, R. 1978. Testing of Substances for their Toxicity Threshold: Model Organisms Microcystis (Diplocystis) aeruginosa and Scenedesmus quadricauda. Mitt. Int. Ver. Theor. Angew. Limnol. 21:275. Cited in EPA, 1985b and EPA 1986g.

Call, D.J., et al. 1983. Toxicity and Metabolism Studies with EPA Priority Pollutants and Related Chemicals in Freshwater Organisms. PB83-263665. Washington, DC. Cited in EPA, 1985a and b.

Canton, H. and Adema, D.M.M. 1978. Reproducibility of Short-Term and Reproduction Toxicity Experiments with Daphnia magna and Comparison of the Sensitivity of Daphnia magna with Daphnia publex and Daphnia cucullata in Short-Term Experiments. Hydrobiologia 59:135. Cited in EPA, 1985b amd HSDB, 1990.

Chang, L.W., et al. 1974. Effects of Methylmercury Chloride on Rana pipiens Tadpoles. Env. Res. 8:82. Cited in EPA, 1985b.

Chang, L.W., et al. 1976. Dose-Dependent Effects of Methylmercury on Limb Regeneration of Newts (Triturus viridescens). Env. Res. 11:305. Cited in EPA, 1985b.

Chapman, P.M., et al. 1982. Relative Tolerances of Selected Aquatic Oligochaetes to Individual Pollutants and Environmental Factors. Aquat. Toxicol. 2:47. Cited in EPA, 1985b.

Curtis, M.W., et al. 1979. Acute Toxicity of 12 Industrial Chemicals to Freshwater and Saltwater Organisms. Water Res. 13:137. Cited in EPA, 1985b.

Curtis, M.W. and Ward, C.H. 1981. Aquatic Toxicity of Forty Industrial Chemicals: Testing in Support of Hazardous Substance Spill Prevention Regulation. Jour. Hydrol. 51:359. Cited in EPA, 1985b.

REFERENCES
(Continued, Page 3 of 7)

Das, A.K. and Misra, B.N. 1982. Toxicity of MEMC (2-Methoxy ethyl mercuric chloride), an Organomercurial Fungicide to Fish, Cyprinus carpio (L.). Jour. Env. Biol. 3:19. Cited in EPA, 1985b.

Deshmukh, S.S. and Marathe, V.B. 1980. Size Related Toxicity of Copper and Mercury to Lebistes reticulatus (Peter), Labeo rohita (Ham.) and Cyprinus carpio (Linn.). Indian Jour. Exp. Biol. 18:421. Cited in EPA, 1985b.

Dial, N.A. 1976. Methylmercury: Teratogenic and Lethal Effects in Frog Embryos. Teratol. 13:327. Cited in EPA, 1985b.

Eaton, R.D.P., Secord, D.C. and Hewitt, P. 1980. An Experimental Assessment of the Toxic Potential of Mercury in Ringed Seal Liver for Adult Laboratory Cats. Toxicol. Appl. Pharmacol. 55:514-521. Cited in USFWS, 1987a.

El-Begearmi, M.M., Ganther, H.E., and Sunde, M.L. 1980. Toxicity of Mercuric Chloride in Japanese Quail as Affected by Methods of Incorporation into the Diet. Poult. Sci. 59:2216-2220. Cited in USFWS, 1987a.

Ellis, M.M. 1947. Toxicity of Phenyl-mercuric Lactate for Fish. Special Scientific Report No. 42. U.S. Fish and Wildlife Service. Cited in EPA, 1985b.

Environmental Science and Engineering, Inc. (ESE). 1989. Biota Remedial Investigation. Final Report, Vol. II. Prepared for: Office of the Program Manager, Rocky Mountain Arsenal Contamination Cleanup. Gainesville, FL.

Fimreite, N. 1979. Accumulation and Effects of Mercury on Birds. In: The Biogeochemistry of Mercury in the Environment, J.O. Nriagu, Editor. Elsevier/North-Holland Biomedical Press, NY. pp. 601-627. Cited from USFWS, 1987a.

Gipps, J.F. and Biro, P. 1978. The Use of Chlorella vulgaris in a Simple Demonstration of Heavy Metal Toxicity. Jour. Biol. Educ. 12:207. Cited in EPA, 1985b.

Grissom, R.E., Jr., and Thaxton, J.P. 1985. Onset of Mercury Toxicity in Young Chickens. Arch. Env. Contam. Toxicol. 14:193-196. Cited in USFWS, 1987a.

REFERENCES
(Continued, Page 4 of 7)

Hammond, P.B. and Beliles, R.P. 1980. Metals. In: Toxicology, the Basic Science of Poisons. 2nd Ed. Doull, J., Klaassen, C.D., and Amdur, M.O., Eds. MacMillan Publishing Co., NY.

Hatch, R.C. 1977. Poisons Causing Nervous Stimulation or Depression. In: Veterinary Pharmacology and Therapeutics. Jones, L.M., Booth, N.H., and McDonald, L.E., Eds. Iowa State University Press, Ames, IA.

Heil, D.M. and Barbarick, K.A. 1989. Water Treatment Sludge Influence on the Growth of Sorghum-Sudangrass. Jour. of Env. Qual., 18(3): 292-298.

Heit, M. and Fingerman, M. 1977. The Influences of Size, Sex and Temperature on the Toxicity of Mercury to Two Species of Crayfishes. Bull. Env. Contam. Toxicol. 18:572. Cited in EPA, 1985b.

Hill, E.F. 1981. Inorganic and Organic Mercury Chloride Toxicity to Coturnix: Sensitivity Related to Age and Quantal Assessment of Physiological Responses. Ph.D. Thesis, Univ. Maryland, College Park. Cited in USFWS, 1987a.

Hill, E.F., and J.H.Soares, Jr. 1984. Subchronic mercury exposure in Coturnix and a method of hazard evaluation. Env. Toxicol. Chem. 3:489-502.

Hoffman, D.J., and Moore, J.M. 1979. Teratogenic Effects of External Egg Applications of Methyl Mercury in the Mallard, Anas platyrhynchos. Teratology 20:453-462. Cited in USFWS, 1987a.

Holcombe, G.W., et al. 1983. Toxicity of Selected Priority Pollutants to Various Aquatic Organisms. Ecotoxicol. Env. Safety 7:400. Cited in EPA, 1985b.

Hudson, R.H., Tucker, R.K., and Haegele, M.A. 1984. Handbook of Toxicity of Pesticides to Wildlife. Second Edition. U.S. Fish Wildl. Serv. Resour. Publ. 153. Cited in USFWS, 1986b and 1987a, and 1988.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Joshi, A.G. and Rege, M.S. 1980. Acute Toxicity of Some Pesticides and a Few Inorganic Salts to the Mosquito Fish Gambusia affinis (Baird and Girard). Indian Jour. Exp. Biol. 18:435. Cited in EPA, 1985b.

REFERENCES
(Continued, Page 5 of 7)

Khera, K.S. 1979. Teratogenic and Genetic Effects of Mercury Toxicity. Pages 501-518 in J.O. Nriagu (ed.). The Biogeochemistry of Mercury in the Environment. Elsevier/North-Holland Biomedical Press, New York. Cited in USFWS, 1987a.

Kucera, E. 1983. Mink and Otter as Indicators of Mercury in Manitoba Waters. Canad. Jour. Zool. 61:2250-2256. Cited in USFWS, 1987a.

Lock, R.A.C. and van Overbeeke, A.P. 1981. Effects of Mercuric Chloride and Methylmercuric Chloride on Mucus Secretion in Rainbow Trout, Salmo gairdneri. Richardson Comp. Biochem. Physiol. 69C:67. Cited in EPA, 1985b.

Matida, Y., et al. 1971. Toxicity of Mercury Compounds to Aquatic Organisms and Accumulation of the Compounds by the Organisms. Bull. Freshwater Fish. Res. Lab. 21:197. Cited in EPA, 1985b.

McKim, J.M., et al. 1976. Long-term Effects of Methylmercuric Chloride on Three Generations of Brook Trout (Salvelinus fontinalis): Toxicity, Accumulation, Distribution, and Elimination. Jour. Fish. Res. Board Can. 33:2726. Cited in EPA, 1985b.

Mullins, W.H., Bizeau, E.G., and Benson, W.W. 1977. Effects of Phenyl Mercury on Captive Game Farm Pheasants. Jour. Wildl. Manage. 41:302-308. Cited in USFWS, 1987a.

National Research Council of Canada (NRCC). 1977. Effects of Alkali Halides in the Canadian Environment. National Research Council of Canada Associate Committee on Scientific Criteria for Environmental Quality. Ottawa, Canada.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Olson, G.F., et al. 1975. Mercury Residues in Fathead Minnows, Pimephales promelas Rafinesque, Chronically Exposed to Methylmercury in Water. Bull. Env. Contam. Toxicol. 14:129. Cited in EPA, 1985b.

Rai, L.C. 1979. Mercuric Chloride Effect on Chlorella. Phykos 18:105. Cited in EPA, 1985b.

REFERENCES
(Continued, Page 6 of 7)

Rai, L.C., Gaur, J.P. and Kumar, H.D. 1981. Phycology and Heavy Metal Pollution. Biol. Rev. 56:99-151.

Rehwoldt, R., Lawrence, L., Shaw, C., and Wirhowski, E. 1973. The Acute Toxicity of Some Heavy Metal Ions Toward Benthic Organisms. Bull. Env. Contam. Toxicol. 10:291-294. Cited in EPA, 1987, 1985b.

Rosenblatt, D.H., Miller, T.A., Dacre, J.C., Muul, I., and Cogley, D.R. 1975. Problem Definition Studies on Potential Environmental Pollutants. II. Physical, Chemical, Toxicological, and Biological Properties of 16 Substances. Technical Report 7509. U.S. Army Medical Bioengineering Research and Development Laboratory, Fort Detrick, Frederick, MD.

Sheffy, T.B., and St. Amant, J.R. 1982. Mercury Burdens in Furbearers in Wisconsin. Jour. Wild. Manage. 46:1117-1120. Cited in USFWS, 1987a.

Shell Chemical Company (Shell). 1990. Shell Toxicity Profile. In: Human Health Exposure Assessment for Rocky Mountain Arsenal, Toxicity Assessment, Vols. IIa and IIIa. Prepared by Ebasco Services, Inc.

Slooff, W. and Baerselman, R. 1980. Comparison of the Usefulness of the Mexican axolotl (Ambystoma Mexicanum) and the Clawed Toad (Xenopus laevis) in the Toxicological Bioassays. Bull. Env. Contam. Toxicol. 24(3):439-443. Cited in EPA, 1985b and HSDB, 1990.

Slooff, W., et al. 1983. Comparison of the Susceptibility of 22 Freshwater Species to 15 Chemical Compounds. I. (Sub)acute Toxicity Tests. Aquat. Toxicol. 4:113. Cited in EPA, 1985b.

Snarski, V.M. and Olson, G.F. 1982. Chronic Toxicity and Bioaccumulation of Mercuric Chloride in the Fathead Minnow (Pimephales promelas). Aquat. Toxicol. 2:143. Cited in EPA, 1985b.

Spann, J.W., Heath, R.G., Kreitzer, J.F., and Locke, L.N. 1972. Ethyl Mercury P-toluene Sulfonanilide: Lethal and Reproductive Effects on Pheasants. Science 175:328-331. Cited in USFWS, 1987a.

Spann, J.W., Heinz, G.H., Camardese, Hill, E.F., Moore, J.F., and Murray, H.C. 1986. Differences in Mortality Among Bobwhite Fed Methylmercury Chloride Dissolved in Various Carriers. Env. Toxicol. Chem., 5:721-724. Cited in USFWS, 1987a.

REFERENCES
(Continued, Page 7 of 7)

Stanley, R.A. 1974. Toxicity of Heavy Metals and Salts to Eurasian Water-milfoil (Myriophyllum spicatum L.) Arch. Env. Contam. Toxicol. 2: 331. Cited in EPA, 1980b and 1985b.

Thomas, D.L. and Montes, J.G. 1978. Spectrophotometrically Assayed Inhibitory Effects of Mercuric Compounds on Anabaena flos-aquae and Anacystis nidulans (Cyanophyceae). Jour. Phycol. 14:494. Cited in EPA, 1985b.

Tripp, M., and Harris, R.C. 1976. Role of Mangrove Vegetation in Mercury Cycling in the Florida Everglades. Pages 489-497 in J.O. Nriagu (ed.). Environmental Biogeochemistry, Vol. 2. Metals Transfer and Ecological Mass Balances. Ann Arbor Sci. Publ., Ann Arbor, MI. Cited in USFWS, 1987a.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Criteria for Mercury. Office of Water Regulations and Standards. EPA 440/5-80-058. Washington, DC. Cited in USFWS, 1987a.

U.S. Environmental Protection Agency (EPA). 1985. Ambient Water Quality Criteria for Mercury--1984. Office of Water Regulations and Standards. Washington, DC. EPA 440/5-84-026.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

U.S. Fish and Wildlife Service (USFWS). 1987. Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Patuxent Wildlife Research Center, Laurel, MD. Biological Report 85(1.10).

Warnick, S.L. and Bell, H.L. 1969. The Acute Toxicity of Some Heavy Metals on Different Species of Aquatic Insects. Jour. Water Pollut. Control Fed. 41:280. Cited in EPA, 1985b.

Wobester, G.A. 1973. Aquatic Mercury Pollution. Studies of Its Occurrence and Pathologic Effects on Fish and Mink. Ph.D. Thesis. University of Saskatchewan, Canada. Cited in EPA, 1985b.

METHYLENE CHLORIDE
(CAS No. 75-09-2)

INTRODUCTION

Methylene chloride, also known as dichloromethane, is widely used in a variety of industrial and commercial applications, including metal degreasing, paint stripping, as a cleaning solvent, as a thinner, in photographic film and synthetic manufacturing, as an aerosol propellant, and as an insecticidal fumigant (ORNL, 1989).

Based on its high water solubility of 13,400 mg/L at 20°C and its low soil sorption coefficient (K_{oc}) value of 8.8, methylene chloride is expected to be highly mobile in the soil/groundwater system with little or no retardation in subsurface or sandy soils (ORNL, 1989). Transport of methylene chloride vapors through the air-filled pores of unsaturated soils followed by photochemical oxidation is an important loss mechanism for near-surface contaminated soils (ORNL, 1989). In saturated subsurface soils (where soil organic carbon and soil air is negligible), a large fraction of the methylene chloride (approximately 96 percent) is expected to be present in the soil-water phase and transported with flowing groundwater (ORNL, 1989). Because methylene chloride is a low molecular weight chloroaliphatic, it is not rapidly metabolized in the environment, although it can be degraded by acclimated microbial populations (ORNL, 1989). Under normal environmental conditions, methylene chloride is not expected to undergo rapid hydrolysis (ORNL, 1989).

Atmospheric transport and dispersion are also important migration pathways; however, photolysis, degradation by hydroxyl radicals, and intermedia transfer via rainout prevent methylene chloride from accumulating in the atmosphere (ATSDR, 1989).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) are available from EPA (IRIS, 1994; EPA, 1994) and are presented in Table 1. Chronic and subchronic inhalation reference concentrations (RfCs) and RfDs are also available from EPA and are presented in Table 1 (IRIS, 1994; EPA, 1994).

The chronic oral RfD (and interim subchronic oral RfD) of 0.06 mg/kg/day are based on a chronic animal study by the National Coffee Association (1982) in which rats were exposed to methylene chloride in drinking water at either 5, 50, 125, or 250 mg/kg/day for 2 years (IRIS, 1994). Histological alterations of the liver were observed, with a NOAEL of 5 mg/kg/day. Adjusting this value for human intake and applying an uncertainty factor of 100 (10 for animal-to-human extrapolation and and 10 for sensitive human subpopulations) results in a chronic (and interim subchronic) oral RfD of 0.06 mg/kg/day.

The chronic inhalation RfC (and interim subchronic inhalation RfC) of 3 mg/m^3 are based on a chronic animal study by Nitschke et al. (1988) in which rats were exposed to methylene chloride in air for 2 years (EPA, 1994). Histological alterations of the liver were observed, with a NOAEL of 695 mg/m^3. Adjusting this value for human intake and applying an uncertainty factor of 100 (10 for animal-to-human extrapolation and and 10 for sensitive human subpopulations) results in a chronic (and interim subchronic) inhalation RfC of 3 mg/kg/day. Assuming a healthy 70-kilogram adult inhales 20 m^3/day of air, the RfC may be converted to a chronic and subchronic inhalation RfD of 0.86 mg/kg/day.

In humans, short-term, high-level exposures to methylene chloride vapors (1,000 to 20,000 ppm) can result in lightheadedness, sustained levels of carboxyhemoglobin and subsequent oxygen deprivation, weakness, nausea, and drunken behavior (ORNL, 1989). More severe poisonings generally involve correspondingly greater disturbances of the central and peripheral nervous

Table 1. Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Oral					
Chronic	6E-2	Medium	Hepato-toxicity	IRIS, 1994	100 H,S
Subchronic	6E-2	--	Hepato-toxicity	EPA, 1994	100 H,S
Inhalation					
Chronic	8.6E-1[c]	--	Hepato-toxicity	EPA, 1994	100 H,S
Subchronic	8.6E-1[c]	--	Hepato-toxicity	EPA, 1994	100 H,S

Note: RfD = reference dose.
RfC = reference concentration.
-- = No EPA Review.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.
[c]Based on an RfC of 3E+0 mg/m^3 and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

systems, such as decreased blood pressure and increased heart rate, depression, anesthesia, coma, and death (ATSDR, 1989). Fatalities reportedly due to methylene chloride exposure have been attributed to cardiac injury and heart failure (EPA, 1985). No deleterious effects on the health or performance of adults could be detected following repeated exposures to vapor levels of 250 ppm or less for 7.5 hours per day for 10 days (ORNL, 1989). Direct contact with liquid methylene chloride can be irritating to the skin and, when splashed in the eye, is painfully irritating but not likely to cause serious injury (ORNL, 1989).

A broad range of noncarcinogenic effects has been observed in experimental animals following oral and inhalation exposures to methylene chloride. The compound may cause death when animals are administered large doses orally, intraperitoneally, subcutaneously, or by inhalation; kidney and liver damage have been observed following large doses via inhalation exposure (ATSDR, 1989). Subchronic and chronic oral and inhalation exposures of animals to methylene chloride result in liver and CNS toxicity (ORNL, 1989).

Carcinogenicity

The carcinogenic slope factor (CSF) and supporting information are summarized in Table 2. EPA (IRIS, 1994) has classified methylene chloride as a group B2 (suspect human) carcinogen via the oral exposure route. This classification indicates sufficient evidence exists to show carcinogenicity in animals based on carcinogenic responses reported in various animal studies following inhalation and ingestion of methylene chloride (ATSDR, 1989), but inadequate evidence of human carcinogenicity as two studies of chemical factory workers failed to demonstrate an excess of cancers (IRIS, 1994).

EPA derived an oral cancer slope factor (CSF) of 0.0075 $(mg/kg/day)^{-1}$ based on a 24-month study by the National Coffee Association (1982, 1983) in which rats were provided drinking water with methylene chloride at levels from 5 to 250 mg/kg/day and mice were provided drinking water with methylene chloride at levels from 0 to 250 mg/kg/day (IRIS, 1994). The incidence of combined

Table 2. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	0.0075	B2	Liver tumors	IRIS, 1994
Inhalation	0.0016[a]	B2	Liver tumors	IRIS, 1994

Note: B2 = suspect human carcinogen (adequate evidence of carcinogenicity in animals and insufficient evidence in humans).

[a]Inhalation CSF is based on an inhalation unit risk (UR) of 4.7×10^{-7} $(\mu g/m^3)^{-1}$ and assumes that a healthy 70-kilogram adult inhales 20 m^3 of air per day.

Source: ESE.

hepatocellular carcinoma and neoplastic nodules in female rats was significantly higher in the 50 and 250 mg/kg/day dose groups than in the matched control groups. As supporting data, lung and liver tumors were observed in mice following a lifetime inhalation exposure to methylene chloride (ATSDR, 1989).

An inhalation unit risk (UR) of 4.7×10^{-7} $(\mu g/m^3)^{-1}$ was derived from a 2-year inhalation study in which mice and rats were exposed to levels as high as 4,000 ppm (IRIS, 1994). Assuming a healthy 70-kilogram adult inhales 20 m^3/day of air, the inhalation UR may be converted to an inhalation CSF of 0.0016 $(mg/kg/day)^{-1}$.

Mutagenicity

Methylene chloride was found to be mutagenic as well as effective in inducing recombination and gene conversion during bacterial assays; however, *in vivo* experiments have shown negative results for genotoxicity (ORNL, 1989). Methylene chloride has been genotoxic in a variety of test systems. It was mutagenic in the Salmonella assay both with and without activation (ReproTox, 1994). It has been tested in at least two batteries of genetic assays. Methylene chloride was not active in inducing mutations in mammalian cells in the HGPRTase assay; and it did not induce DNA repair synthesis in rat cells (ReproText, 1994). In addition, methylene chloride was weakly positive for sister chromatid exchanges in vitro; induced chromosome aberrations in hamster cells in vitro but not in vivo (ReproText, 1994). While methylene chloride is clearly a genotoxin in bacterial systems and in some assays using mammalian cells, its genetic risk for man, if any, is not known (ReproText, 1994).

Teratogenicity/Reproductive Effects

Methylene chloride has also been studied for possible reproductive effects in man and animals. It did not cause structural malformations in rats at 4,500 ppm, but did produce lower birth weights (Hardin & Manson, 1980). The offspring of the maternally-exposed rats lagged in behavioral development and did not adapt well to new situations. The high level of methylene chloride used in this study produced

some toxicity in the mothers; therefore it is not clear if methylene chloride would have been selectively toxic to the fetuses at lower doses.

In another study methylene chloride given at 1,250 ppm to pregnant rats and mice from day 6 to 15 of gestation also retarded the development of the offspring but did not produce structural malformations (ReproText, 1994). When given orally to rats at 4% in the diet, it produced maternal toxicity but was not teratogenic (ReproText, 1994). It did produce some teratogenic effect in mice when given orally or injected at 0.1 mL (ReproText, 1994).

Animals studies have indicated that methylene chloride can cross the placenta. Methylene chloride has also been found in human fetuses and breast milk Russian women exposed to methylene chloride and gasoline reported an incidence of gynecological complaints (ReproText, 1994). CNS defects were reported in the offspring of women exposed to methylene chloride and other solvents (ReproText, 1994). The EPA regards methylene chloride as having "minimal" teratogenic potential (ReproText, 1994).

ECOTOXICITY

Aquatic Organisms

In static tests with Daphnia magna, EPA (1982) determined a 48-hour LC_{50} value of 224,000 μg/L. In an acute test with the fathead minnow Pimephales promelas, Verschueren (1983) determined an LC_{10} value of 51,000 μg/L and LC_{50} values ranging from 193,000 to 232,000 μg/L for 96-hour and 72-hour tests, respectively. An acute 96-hour LC_{50} value of 220,000 μg/L was reported for bluegill Lepomis macrochirus (Buccafusco et al., 1981). These values are summarized in Table 3.

No information regarding the toxicity of methylene chloride to aquatic vegetation was identified in the available literature.

Table 3. Toxicity of Methylene Chloride to Aquatic Organisms

Scientific Name	Test and Condition	Concentration	Exposure Duration	Effects	Reference
Daphnia magna	LC50	224000	48-H		EPA, 1982
Lepomis marcrochirus	LC50	220000	96-H		Buccafusco et al., 1981
Pimephales promelas	LC50	193000	96-H		Verschueren, 1983
Pimephales promelas-rafinesqe	EC10	66000	48-H		Verschueren, 1983
Pimephales promelas-rafinesqe	EC10	69000	24-H		Verschueren, 1983
Pimephales promelas-rafinesqe	EC50	99000	48-H		Verschueren, 1983
Pimephales promelas-rafinesqe	EC50	99000	96-H		Verschueren, 1983
Pimephales promelas-rafinesqe	LC10	51000	96-H		Verschueren, 1983
Pimephales promelas-rafinesqe	LC50	232000	72-H		Verschueren, 1983
Pimephales promelas	EC50	330000	96-H	Increased respiration, darkly colored and lost equilibrium prior to death	Geiger et al., 1986

Source: ESE.

Although experimental data are lacking, methylene chloride would not be expected to bioconcentrate due to its low octanol/water partition coefficient (log K_{ow} is 1.25), from which an estimated BCF of 5 can be estimated using the recommended regression equation (HSDB, 1994).

Terrestrial Organisms

Several studies on the toxic effects of methylene chloride to mammals have been reported. Kirschman et al. (1986) determined 90-day lowest effect levels (LELs) for Mus spp. and Rattus rattus at 580 mg/kg and 190 mg/kg, respectively. In other studies using Rattus rattus where mortality was the test effect investigated, Verschueren (1983) and Burek et al. (1984) determined LC_{50} values ranging from 1,600 to 3,500 mg/kg, respectively. Mortality was the end point for 75% of rabbits (Sylvilagus spp.) tested with methylene chloride at a reported concentration of 10,000 mg/kg (Browning, 1965). Mackinson et al. (1981) reported fatty liver, icterus, pneumonia, and splenic atrophy effects in dogs (Canis familaris) at 1,000 mg/kg. These data are summarized in Table 4.

No information regarding the toxicity of methylene chloride to vegetation was identified in the available literature.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 5. Due to the ranking of methylene chloride as a Group B2 suspect human carcinogen, EPA has set a nonenforceable Maximum Contaminant Level Goal (MCLG) for drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 5 μg/L, which is enforceable for public water supplies (55 FR 30370; 25 Jul 1990). Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from

Table 4. Toxicity of Methylene Chloride to Terrestrial Organisms

Scientific Name	Test and Condition	Concentration	Exposure Duration	Effects	Reference
Mus spp.	LEL	580 MG/KG	90-D		Kirschman et al., 1986
Rattus rattus	LD50	1600 MG/KG	ORAL		Verschueren, 1983
Rattus rattus	LEL	190 MG/KG	90-D		Kirschman et al., 1986
Sylvilasus spp.	75% Dead	10000 PPM			Browning, 1965
Rattus rattus		4500 PPM	3 weeks pregestational or 17 days gestation	Increased liver weight; decreased fetal weight	IARC, 1979
Rattus rattus	CHRONIC	3500 PPM		Increased mortality rate	Burek et al., 1984
Canis familiaris	CHRONIC	1000 PPM	5-WKS	Fatty liver, icterus, pneumonia, splenic atrophy	Mackison et al., 1981

Source: ESE.

Table 5. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day[a]	10-Day[a]	Longterm[a]	Lifetime	Fish and Water	Fish Only	Water Only[b]
Methylene chloride	5	0	--	13,300	1,500	500	--	0.19[c]	15.7[c]	0.19[c]

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is for a 10-kg child.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[c]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

1.9 to 0.019 μg/L for consumption of contaminated drinking water and aquatic organisms (IRIS, 1994). For consumption of aquatic organisms only, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 157 to 1.57 μg/L, respectively (IRIS, 1994). These AWQC would yield values for human consumption of water alone (W) of 1.9, 0.19, and 0.019 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on the following equation:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

No national ambient air quality standards (NAAQS) have been developed for mercury under the Clean Air Act (IRIS, 1994). However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure to mercury (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs)
and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). These values are presented in the Table 6.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms. However, chronic and acute lowest observed effect levels (LOELs) have been reported by EPA and are presented in Table 5 (IRIS, 1994).

Table 6. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	1,760 c	--	174 c
STEL	--	--	--
CL	3,530 c 7,060 c (5-min max peak in any 2-hour period)	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.

STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.

CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.

c = designates that this chemical is regulated as a potential carcinogen.

Source: ESE.

Table 7. Ambient Water Quality Criteria

LOEL (μg/L)	Freshwater	Marine
Acute	11,000	12,000
Chronic	--	6,400

Source: ESE.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Methylene Chloride. Prepared by Life Systems, Inc. U.S. Public Health Service, Atlanta, GA. ATSDR/TP-88/18. NTIS No. PB89-194468.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Browning, E. 1965. Toxicity and Metabolism of Industrial Solvants. American Elsevier, New York, NY.

Burek, J.D., et al. 1984. Fund. Appl. Toxicol. 4(1):30-47.

Hardin, B.D. and Manson, J.M. 1980. Absence of dichloromethane teratogenicity with inhalation exposure in rats. Toxicol. Appl. Pharmacol. 52:22-28. (cited in ReproText, 1994).

Hazardous Substances Data Bank (HSDB). 1994. National Library of Medicine (NLM). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

International Agency for Research on Cancer (IARC). 1979. Monographs on the evalution of the Carcinogenic Risk of Chemicals to Man.

Kirschman, J.C., et al. 1986. Food. Chem. Toxicol. 24(9):943-949

Mackison, F.W., Stricoff, R.S., and Partridge, L.J., Jr. 1981. Occupational Health Guidelines for Chemical Hazards. Washington, DC. DHHS(NIOSH) Publication No. 81-123.

National Coffee Association. 1982. 24-Month chronic toxicity and oncogenicity study of methylene chloride in rats. Final Report. Prepared by Hazleton Laboratories America, Inc., Vienna, VA. (Unpublished). (cited in IRIS, 1994).

REFERENCES
(Continued, Page 2 of 2)

National Coffee Association. 1983. 24-Month oncogenicity study of methylene chloride in mice. Prepared by Hazleton Laboratories America, Inc., Vienna, VA. (cited in IRIS, 1994).

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Nitschke, K.D., Burek, J.D., Bell, T.J., Kociba, R.J., Rampy, R.W., and McKenna, M.J. 1988. Methylene chloride: A 2-year inhalation toxicity and oncogenicity study in rats. Fund. Appl. Toxicol. 10:1-12. (cited in EPA, 1994).

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

ReproText®. 1994. B.J. Dabney. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Reproductive Toxicology System (ReproTox®). 1994. Reproductive Toxicology Center, Columbia Hospital for Women Medical Center, Washington, DC. Part of the Reproductive Risk Information System (ReproRisk®), Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

U.S. Environmental Protection Agency (EPA). 1982. Intermedia Priority Pollutant Guidance Documents. R. Kayser, D. Sterling, and Viviani, Eds. Washington, DC.

U.S. Environmental Protection Agency (EPA). 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. Prepared by Clement Associates, Inc. Arlington, VA.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

PAHs

(Acenaphthene; CAS No. 67-66-3
(Anthracene; CAS No. 120-12-7
(Benzo(a)pyrene; CAS No. 50-32-8
(Benzo(ghi)perylene; CAS No. 191-24-2
(Chrysene; CAS No. 218-01-9
(Fluoranthene; CAS No. 206-44-0
(Indeno(1,2,3-cd)pyrene; CAS No. 193-39-5
(Naphthalene; CAS No. 91-20-3
Acenaphthylene; CAS No. 208-96-8)
Benz(a)anthracene; CAS No. 56-55-3)
Benzo(b)fluoranthene; CAS No. 205-99-2)
Benzo(k)fluoranthene; CAS No. 207-08-9)
Dibenz(ah)anthracene; CAS No. 53-70-3)
Fluorene; CAS No. 86-73-7)
2-Methylnaphthalene; CAS No. 91-57-6)
Pyrene; CAS No. 129-00-0)

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a class of compounds consisting of substituted and unsubstituted polycyclic and heterocyclic aromatic rings. PAHs are formed by the incomplete combustion of organic compounds in the presence of insufficient oxygen. PAHs occur from both natural and anthropogenic sources. As a group, they are widely distributed in the environment and found in animal and plant tissue, sediments, air, and surface water (Radding et al., 1976). Formed during the incomplete combustion of organic matter, PAHs are common constituents of tar, soot, petroleum products, engine lubricant wastes, tobacco smoke, automotive exhaust, smoked meats, fried foods, and creosote-treated wood (EPA, 1980).

Anthracene group compounds (see Table 1) are 3- and 4-ring PAHs. On a commercial basis, these compounds have been imported in small quantities for special uses and are common constituents of coal tars, typically used in creosotes. Anthracene group compounds have possible carcinogenicity that might require careful reviews. They have moderately low volatility and water solubility. Due to their relatively low water solubilities and fairly high K_{ow} values, adsorption unto both organic and inorganic matter is a primary removal pathway for these compounds in the water column. The fraction of these PAHs that remains in the water column is expected to undergo photolytic degradation, the extent of which is affected by turbidity and light penetration (Zepp and Schlotzhauer, 1979). Volatilization from the aqueous phase is not expected to be a major fate process.

Table 1. Physical and Chemical Properties of PAHs

PAH	Molecular Weight (g/mole)	Water Solubility (mg/L, 25°C)	Octanol/Water Partition Coefficient (log K_{ow})	Vapor Pressure (torr)	Henry's Law Constant (atm · m^3/mole)	Organic Carbon Partition Coefficient, K_{oc}^* (mL/g)
Naphthalene Group						
Naphthalene	128.19	31.7	3.29	0.09	4.8×10^{-4}	8.51×10^{2}
Anthracene Group						
Anthracene	178.24	0.045	4.45	2.4×10^{-4}	1.25×10^{-3}	1.4×10^{4}
Acenaphthene	154.21	3.42	4.33	1.6×10^{-3}	9.33×10^{-5}	4.6×10^{3}
Fluoranthene	202.26	0.26	5.33	5×10^{-6}	5.12×10^{-6}	3.8×10^{4}
Fluorene	166.23	1.69	4.18	1×10^{-2}	1.29×10^{-3}	7.3×10^{3}
Phenanthrene	178.24	1.00	4.46	9.6×10^{-4}	2.25×10^{-4}	1.4×10^{4}
Pyrene	202.26	0.14	5.32	2.5×10^{-6}	4.75×10^{-6}	3.8×10^{4}
BaP Group						
BaP	252.32	0.0038	6.08	5.6×10^{-9}	4.89×10^{-7}	5.5×10^{6}
Acenaphthylene	152.21	3.93	3.72	0.030	1.52×10^{-3}	2.5×10^{3}

Table 1. Physical and Chemical Properties of PAHs (Continued, Page 2 of 2)

PAH	Molecular Weight (g/mole)	Water Solubility (mg/L, 25°C)	Octanol/Water Partition Coefficient (log K_{ow})	Vapor Pressure (torr)	Henry's Law Constant (atm · m^3/mole)	Organic Carbon Partition Coefficient, K_{oc}* (mL/g)
Benz(a) anthracene	228.28	0.009	5.61	2.2×10^{-8}	7.34×10^{-7}	1.38×10^{6}
Benzo(b) fluoranthene	252.32	0.001	6.08	5.0×10^{-7}	1.66×10^{-4}	5.5×10^{-5}
Benzo(k) fluoranthene	252.32	0.00055	6.08	5.0×10^{-7}	3.02×10^{-4}	5.5×10^{5}
Benzo perylene	276.34	0.0003	6.51	1.0×10^{-10}	1.21×10^{-7}	1.6×10^{6}
Chrysene	228.28	0.0018	5.61	6.3×10^{-9}	1.05×10^{-6}	2.00×10^{5}
Dibenzo(a,h)-anthracene	278.36	0.0005	6.84	1.0×10^{-10}	7.3×10^{-8}	3.3×10^{6}
Indeno(1,2,3,-cd)pyrene	276.34	0.0005	6.51	1.0×10^{-10}	6×10^{-10}	1.6×10^{6}

Note: atm · m^3/mole = atmospheres per cubic meter per mole.
g/mole = grams per mole.
mL/g = milliliters per gram.

*All K_{oc} values from Superfund Public Health Evaluation Manual except naphthalene, which was a measured value reported by Karickhoff et al., 1981 (EPA, 1986).

Source: ESE.

Benzo(a)pyrene (BaP) group chemicals (see Table 1), in pure forms, are typically used as research laboratory standards. They have no commercial production or use other than as constituents of coal tars and coal tar containing creosotes. They generally have low vapor pressures and water solubilities, with the exception of acenaphthylene, which is soluble and volatile. Some of the PAHs in this group have been identified as carcinogens.

The extent of migration of PAHs in the environment is a function of the log octanol-water and organic carbon partition coefficients (K_{ow} and K_{oc}). With comparably high K_{ow} and K_{oc} values and low water solubilities, PAHs are expected to strongly adsorb to particulate matter, particularly those high in organic content (EPA, 1982). The low vapor pressures and Henry's law constants for these PAHs suggest that volatilization is not a primary removal mechanism from either soil or aquatic systems. In general, the volatilization rate decreases as the vapor pressure decreases, both of which are inversely related to the number of aromatic rings. Southworth (1979) concluded that the rate of volatilization of PAHs with four or more rings is insignificant under all conditions.

In aquatic systems, the removal mechanisms for most PAHs are photochemical reactions, sorption onto particulate matter, and subsequent sedimentation and microbial degradation (EPA, 1984). PAHs do not contain groups amenable to hydrolysis; however, direct photolysis may be an important fate process for PAHs dissolved in an aqueous environment (EPA, 1976).

The mobility of naphthalene in soil and groundwater is strongly affected by the extent of soil sorption, which is a reversible process for this particular PAH. The potential for soil desorption and the relatively high water solubility of naphthalene suggest that migration from soil into groundwater and surface water may be an important transport process for naphthalene (EPA, 1982). In addition, volatilization of naphthalene from aqueous solutions is reported as a significant removal process. Volatilization from soils may also be an important transport mechanism, although sorption of naphthalene

vapors onto soil materials may slow vapor phase transport (EPA, 1982). Once in the atmosphere, naphthalene is photooxidized; however, under normal soil and aqueous conditions, naphthalene is not expected to undergo hydrolysis or oxidation/reduction reactions. Although some aerobic microbial degradation is likely, this process is not expected to be a predominant fate mechanism due to the low concentration of microorganisms (at depth) and the low dissolved oxygen (EPA, 1982).

Bacteria have been shown to use some PAHs as a sole source of carbon for growth (Radding et al., 1976). Evidence for bacterial degradation is limited, and no compound-specific information is available. Because no organisms have been isolated that are capable of using 4- or 5-ringed compounds as a sole carbon source, it is assumed that they are co-metabolized with simpler compounds.

HUMAN HEALTH EFFECTS

PAHs may be separated into two specific groups: the potentially carcinogenic PAHs, which include benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, BaP, chrysene, dibenz(ah)anthracene, and indeno(1,2,3-cd)pyrene; and the noncarcinogenic PAHs, which include the remaining PAHs. Potential noncarcinogenic effects of exposure to carcinogenic PAHs will also be discussed.

Noncarcinogenic Effects

Chronic and subchronic oral reference doses (RfDs) for several PAHs are available from EPA (IRIS, 1994; EPA, 1994) and are presented in Table 2. No inhalation RfCs or RfDs are provided in IRIS (1994) or the Health Effects Assessment Summary Tables (HEAST) (EPA, 1994).

Table 2. Oral Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects

Chemical	Oral RfD (mg/kg/day)	Confidence Level	Critical Effect	RfD Basis/RfD Source	Uncertainty Factors[a] / (Modifying Factors)[b]
Acenaphthene					
Chronic	0.06	Low	Hepato-toxicity	IRIS, 1994	3,000 C,D,H,S
Subchronic	0.6	--		EPA, 1994	300 D,H,S
Anthracene					
Chronic	0.3	Low	None	IRIS, 1994	3,000 C,D,H,S
Subchronic	3.0	--		EPA, 1994	300 D,H,S
Fluoranthene					
Chronic	0.04	Low	Nephro-pathy	IRIS, 1994	3,000 C,D,H,S
Subchronic	0.4	--		EPA, 1994	300 D,H,S
Fluorene					
Chronic	0.04	Low	Decreased erythrocyte count	IRIS, 1994	3,000 C,D,H,S
Subchronic	0.4	--		EPA, 1994	300 D,H,S
Naphthalene					
Chronic	0.04	--	Decreased body weight	EPA, 1992b	1,000 D,H,S
Subchronic	0.04	--		EPA, 1992b	1,000 D,H,S
Pyrene					
Chronic	0.03	Low	Nephro-toxicity	IRIS, 1994	3,000 C,D,H,S
Subchronic	0.3	--		EPA, 1994	300 D,H,S

Table 2. Oral Noncarcinogenic Reference Doses and Potential Noncarcinogenic Effects (Continued, Page 2 of 2)

Note: IRIS = Integrated Risk Information System.
RfD = reference dose.
-- = No EPA Review.

[a]Uncertainty factors are typically multiples of 10 and are provided using the following codes:
C = to extrapolate from a subchronic study to a chronic endpoint.
D = to account for any data deficiencies.
H = to extrapolate from an animal study to humans.
S = to protect sensitive human subpopulations.
[b]A modifying factor is an extra safety factor (ranging from 1 to 10) incorporated in the RfD development to account for various other data deficiencies.

Source: ESE.

Except for naphthalene, the RfDs were based on critical oral mouse studies; the oral RfD for naphthalene is based on a rat study. For purposes of deriving a conservative baseline risk assessment, the lowest RfD other than naphthalene (0.03 mg/kg/day for pyrene) may be used for deriving the noncarcinogenic risks associated with exposure to all PAHs, carcinogenic and noncarcinogenic, that do not have an oral RfD. It is not appropriate to apply the RfD for naphthalene to other PAHs because of the differences in the physicochemical and biological properties.

Animal studies demonstrate that PAHs tend to affect proliferating tissues, such as bone marrow, lymph organs, gonads, and intestinal epithelium (ATSDR, 1990). Although PAHs are distributed throughout the body, the hemopoietic and lymphoid systems appear to be their major target organs in animals. For example, humans that consumed laxatives containing anthracene for prolonged periods demonstrated an increased incidence of colon and rectum melanosis (deposition of black pigments), while hemopoietic toxicity was observed in patients with liver or breast cancer treated with anthracene-containing chemotherapeutic agents.

Other nonmalignant effects associated with exposure to PAHs include liver and kidney damage, various skin disorders, and immunosuppressive effects (ATSDR, 1990; Clement, 1985). Several dermal studies indicate that some PAHs can cause hemolymphatic changes in the lymph nodes in rats, and human dermal exposure to high concentrations of PAHs has resulted in burning, itching, dermatitis, hyperkeratosis, and papular and vesicular eruptions, as well as phototoxic and photoallergic effects [Clement, 1985; Clayton and Clayton, 1981; International Agency for Research on Cancer (IARC), 1983].

Carcinogenicity

EPA has classified the carcinogenic PAHs as group B2 (probable human) carcinogens (IRIS, 1994), which indicates that insufficient human data are available to determine the potential carcinogenicity of the PAHs but that sufficient experimental animal data are available. Oral and inhalation CSFs for BaP have been developed by EPA (IRIS,

1994; EPA, 1992b) and are presented in Table 3. In addition, a relative potency approach, which uses the CSF for BaP and a toxicity equivalent factor (TEF), is used by EPA Region IV (1992a) to produce appropriate CSFs for the other carcinogenic PAHs. Therefore, applying a TEF of 1.0 to dibenz(ah)anthracene; 0.1 to benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene; and 0.01 to chrysene results in oral/inhalation CSFs of 7.3/6.1 for dibenz(ah)anthracene; 0.73/0.61 for benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene; and 0.073/0.061 for chrysene.

The potential for carcinogenic PAHs to induce malignant tumors, both at the site of application and systemically, dominates the consideration of health hazards resulting from exposure (ATSDR, 1990). BaP is a moderately potent experimental carcinogen in many species by various routes of exposure (ATSDR, 1990). No reports directly correlate human BaP exposures and tumor development, although humans are likely to be exposed via numerous routes (ATSDR, 1990). Oral administration of carcinogenic PAHs produced tumors of the forestomach in mice, and laryngeal and tracheal tumors were observed in laboratory animals following inhalation, intratracheal, and intravenous administration (ATSDR, 1990; Clement, 1985). Dermal studies with mice indicate that carcinogenic PAHs produce skin carcinomas and lung adenomas (ATSDR, 1990; Clement, 1985).

Mutagenicity

Generally, carcinogenic PAHs are active in mutagenic assays. In most cases, some mammalian metabolic activation system is necessary prior to genotoxic activity. BaP has been shown to cause various genotoxic effects in a broad range of mammalian and prokaryotic cell assay systems (EPA, 1991). In mammalian

Table 3. Carcinogenic Slope Factors and Potential Carcinogenic Effects for BaP

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	7.3	B2	Forestomach	IRIS, 1994
Inhalation	6.1[a]	B2	Respiratory Tract	EPA, 1992b

Note: B2 = suspect human carcinogen (sufficient evidence of carcinogenicity in animals and inadequate data in humans).

[a]Inhalation CSF based on an inhalation unit risk of 1.7×10^{-3} $(\mu g/m^3)^{-1}$ (EPA, 1992b) and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

cell cultures, BAP tested positive in assays for DNA damage, forward mutation, chromosomal effects, and cell transformation. In prokaryotes, BAP tested positive in DNA damage assays and in both reverse and forward mutation assays.

Benz(a)anthracene and dibenz(ah)anthracene were mutagenic and produced DNA damage in cultured human cells and the majority of non-human systems tested (ATSDR, 1990). There is also some evidence that benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(ghi)perylene, chrysene, fluoranthene, and indeno(1,2,3-cd)pyrene are genotoxic (ATSDR, 1990). Of these PAHs, only chrysene and fluoranthene have shown positive genotoxic effects in human cell cultures. The majority of test results for anthracene, phenanthrene, and pyrene are negative, although positive results were reported for each chemical in at least one in vitro test (ATSDR, 1990). There is no evidence suggesting that acenaphthene, acenaphthylene, or fluorene are genotoxic.

Teratogenicity/Reproductive Effects

Pertinent data regarding the reproductive and developmental toxicity of carcinogenic PAHs in humans and experimental animals following inhalation, oral, or dermal exposure could not be located in the available literature.

Available data from oral animal studies indicates that exposure of pregnant dams to BaP resulted in fetal resorptions and malformations and sterility in first generation mouse progeny (ATSDR, 1990). Pre- and post-implantation losses and decreased fetal weight and survival were reported in rats. Anthracene, benz(a)anthracene, chrysene,and dibenz(ah)anthracene have also been tested. Daily subcutaneous administration of dibenz(ah)anthracene in rats from the first day of pregnancy resulted in fetal death and resorption and may have affected the fertility of the dams (ATSDR, 1990). Perenteral exposure to chrysene resulted in liver tumors in mouse progeny.

ECOTOXICITY

Aquatic Organisms

Little aquatic testing has been conducted with fluoranthene. Freshwater acute toxicity for fish and invertebrates is estimated to range from an LC_{50} value of 3.98 mg/L for bluegill sunfish (Lepomis macrochirus) to 325 mg/L for the cladoceran (Daphnia magna). The freshwater alga Selenastrum capricornutum was exposed for 96 hours to fluoranthene, and an EC_{50} value of approximately 54.5 mg/L was determined based on the reported reduction in cell numbers and chlorophyll a (EPA, 1980).

The saltwater sandworm (Neanthes arenaceodentata) was the representative invertebrate tested and was most sensitive to phenanthrene (LC_{50} of 370 μg/L) (see Table 4). The LC_{50} values for benzo(a)anthracene and chrysene were both >1000 μg/L.

A variety of species were tested using fluorene, and the data indicate that the fathead minnow (Pimephales promelas) is least sensitive (LC_{50}>100,000 μg/L) and the bluegill (Lepomis macrochirus) was most sensitive (LC_{50} of 500 μg/L).

No chronic toxicity data for fluoranthene were found for freshwater fish or invertebrates. Existing data on a saltwater invertebrate, Mysidopsis bahia, report an acute-to-chronic ratio of 2.5 (EPA, 1980). Based on this ratio, the estimated chronic value for the most sensitive freshwater species tested would be 1.6 mg/L.

Only phenanthrene and fluorene were used in chronic tests, and the midge (Chironomus plumosus) was more resistant to fluorene toxicity than Daphnia magna (see Table 5). Two species were used for phenanthrene tests, and Daphnia magna (95 percent chronic index of 590 to 840 μg/L) was more resistant than the rainbow trout (95 percent chronic index of 10 to 90 μg/L).

Table 4. Acute Toxicity of PAHs to Freshwater Aquatic Organisms

Species and Chemical	Concentration (μg/L)	Effect	Reference
Lepomis macrochirus (Bluegill)			
Benz(a)anthracene	1,000	LC_{87} (6 months)	EPA, 1980
Fluorene	500	LC_{12} (30 days)	Finger et al., 1985
Fluorene	910	LC_{50} (96 hours)	Finger et al., 1985
Gammarus pseudolimnaeus (Amphipod)			
Fluorene	600	LC_{50} (96 hours)	Finger et al., 1985
Oncorhynchus mykiss (Rainbow trout)			
Fluorene	820	LC_{50} (96 hours)	Finger et al., 1985
Pimephales promelas (Fathead minnow)			
Fluorene	>100,000	LC_{50} (96 hours)	Finger et al., 1985

Source: ESE.

Table 5. Chronic Toxicity of PAHs to Freshwater Aquatic Organisms

Species and Chemical	95 Percent Chronic Index (μg/L)	Reference
Chironomus plumosus (Midge)		
Fluorene	1,900 to 3,000	Mayer and Ellersieck, 1986
Daphnia magna (Cladoceran)		
Fluorene	330 to 550	Mayer and Ellersieck, 1986
Phenanthrene	590 to 840	Millman et al., 1984
Oncorhyncus mykiss (Rainbow trout)		
Phenanthrene	10 to 90	Millman et al., 1984

Source: ESE.

Terrestrial Organisms

Plants can adsorb PAHs from soils through their roots and translocate them to other plant parts, such as developing shoots. Uptake rates were governed, in part, by PAH concentrations, PAH water solubility, soil type, and PAH physicochemical state (vapor and particulate). Lower molecular weight PAHs were absorbed by plants more readily than higher molecular weight PAHs [U.S. Fish and Wildlife Service (USFWS), 1987].

Above-ground parts of vegetables contain more PAHs than underground parts, which is attributed to airborne deposition and subsequent adsorption. The limited information available on PAH-induced phytotoxic effects indicates that these responses are rare (USFWS, 1987).

Data on the biological effects of BaP, 3-methylcholanthrene, and perylene to reptiles and amphibians are limited, but are summarized in Table 6. Data are even more limited on the effects of PAHs on avian wildlife (see Table 7).

Measured BCFs for naphthalene in rainbow trout and bluegill sunfish range from 40 to 300 (Rogers et al., 1983). Studies in fish have shown that uptake and depuration of naphthalene is rapid; therefore, once external contamination is removed, accumulated burdens would be cleared rapidly (Rogers et al., 1983). Studies with the freshwater invertebrate D. pulex report a rapid and greater accumulation from food than from water, which suggests the potential for bioaccumulation of naphthalene in secondary consumers (e.g., fish) through ingestion of zooplankton (Trucco et al., 1983). Available data indicate that BaP will bioaccumulate. In Daphnia magna the BCF is 134,248 (see Table 8).

Table 6. Induced Mixed-Function Oxidation Systems

Species and Chemical	Affected Area	Reference*
Ambystoma tigrinum (Tiger Salamander)		
Perylene	Liver and Skin	Couch et al., 1983
3-Methyl-Cholanthrene	Liver and Skin	Anderson et al.,1982
Rana pipiens (Leopard Frog)		
BaP	Liver	Stegeman, 1981; Schwen and Mannering, 1982a, 1982b
Thamnophis sp. (Garter Snake)		
3-Methyl-Cholanthrene	Liver	Stegeman, 1981; Schwen and Mannering, 1982a, 1982b

*References cited are from USFWS, 1987.

Source: ESE.

Table 7. Toxic Effects of Selected PAHs on Adult and Embryo Mallard Ducks

Compound	Treatment	Concentration	Duration	Effect	Reference*
Naphthalenes, Naphthenes and Phenanthrene	Diet	4,000 mg/kg	7 months	No mortality; no visible signs of toxicity; 25-percent increase in liver weight; 30-percent increase in blood flow to liver	Patton and Dieter, 1980
7,12-Dimethylbenz(a) anthracene	Topical application to eggs	0.002 μg/egg	18 days	26-percent mortality; survivors exhibited reduced embryonic growth, increased anomolies	Hoffman and Gay, 1981
7,12-Dimethylbenz(a) anthracene	Topical application to eggs	0.01 μg/egg	18 days	90-percent mortality	Hoffman and Gay, 1981
Chrysene	Topical application to eggs	0.015 μg/egg	18 days	90-percent mortality	Hoffman and Gay, 1981
BaP	Topical application to eggs	0.002 μg/egg	18 days	0-percent mortality; increased anomolies in survivors	Hoffman and Gay, 1981
BaP	Topical application to eggs	0.01 μg/egg	18 days	60-percent mortality	Hoffman and Gay, 1981
BaP	Topical application to eggs	0.05 μg/egg	3 days	75-percent mortality	Hoffman and Gay, 1981

*References cited are from USFWS (1987).

Source: ESE.

Table 8. BCFs for PAHs

Species and Chemical	Duration	BCF	Reference*
Daphnia magna (Cladoceran)			
BaP	3 days	134,248	Lu et al., 1977
Daphnia pulex (Cladoceran)			
Anthracene	60 minutes	200	EPA, 1980a
Anthracene	24 hours	760 to 1,200	Southworth et al., 1978
Benz(a)anthracene	24 hours	10,109	Southworth et al., 1978
9-Methylanthracene	24 hours	4,583	Neff, 1985
BaP	3 days	134,248	Lu et al, 1977
Naphthalene	24 hours	131	Neff, 1985
Pyrene	24 hours	2,702	Neff, 1985
Crassostrea virginica (Eastern oyster)			
BaP	14 days	242	Couch et al., 1983
Pimephales promelas (Fathead minnow)			
Anthracene	2 to 3 days	485	Southworth, 1979
Hexagenia sp. (Mayfly)			
Anthracene	28 hours	3,500	EPA, 1980
Oncorhyncus mykiss (Rainbow trout)			
Anthracene	72 hours	4,400 to 9,200	Linder et al., 1985
BaP	10 days	182 to 920	Gerhart and Carlson, 1978
Fluoranthene	21 days	379	Gerhart and Carlson, 1978
Pyrene	21 days	69	Gerhart and Carlson, 1978
Physa p. (Snail)			
BaP	3 days	82,231	Lu et al., 1977
Lepomis macrochirus (Bluegill)			
Fluorene	30 days	200 to 1,800	Finger et al., 1985
Naphthalene	24 hours	310	McCarthy and Jimenez, 1985

*References cited are from USFWS (1987).

Sources: ESE.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 9. Due to the ranking of BaP as a Group B2 suspect human carcinogen, EPA has proposed a nonenforceable Maximum Contaminant Level Goal (MCLG) for BaP in drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 0.2 μg/L, which would be enforceable for public water supplies (57 FR 31776; July 17, 1992). EPA has also proposed the following MCLs for carcinogenic PAHs: 0.1 μg/L for benz(a)anthracene; 0.2 μg/L for benzo(b)fluoranthene, benzo(k)fluoranthene, and chrysene; 0.3 μg/L for dibenz(ah)anthracene; and 0.4 μg/L for indeno(1,2,3-cd)pyrene.

Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L for carcinogenic PAHs. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 0.028 to 0.00028 μg/L for consumption of drinking water and aquatic organisms (W + F) contaminated with PAHs (IRIS, 1994). For consumption of aquatic organisms only (F), the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 0.31 to 0.0031 μg/L, respectively (IRIS, 1994). These AWQC would yield values for human consumption of PAHs in water alone (W) of 0.031, 0.0031, and 0.00031 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on Equation 1 as follows:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F} \qquad \text{(Eq. 1)}$$

In addition, EPA has developed individual values for fluoranthene as follows: W + F = 42 μg/L, F = 54 μg/L, and W = 190 μg/L (IRIS, 1994).

Table 9. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only
PAHs, as a class, unless otherwise specified	--	--	--	--	--	--	--	0.0028[b]	0.031[b]	0.0031[b]
Benz(a)anthracene	0.1 p	--	--	--	--	--	--	--	--	--
Benzo(b)fluoranthene	0.2 p	--	--	--	--	--	--	--	--	--
Benzo(k)fluoranthene	0.2 p	--	--	--	--	--	--	--	--	--
Benzo(a)pyrene	0.2 p	0 p	--	--	--	--	--	--	--	--
Chrysene	0.2 p	--	--	--	--	--	--	--	--	--
Dibenz(ah)anthracene	0.3 p	--	--	--	--	--	--	--	--	--
Fluoranthene	--	--	--	--	--	--	--	42	54	190
Indeno(1,2,3-cd)pyrene	0.4 p	--	--	--	--	--	--	--	--	--

Table 9. Human Regulatory Criteria (μg/L) (Continued, Page 2 of 2)

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

[b]Carcinogenic; value represents 10^{-6} cancer risk.

Source: ESE.

No national ambient air quality standards (NAAQS) have been developed for PAHs individually or as a class of chemicals. However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1994). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1994; ACGIH, 1993). Available air quality standards are presented in Table 10.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to PAHs (IRIS, 1994). However, the lowest levels observed to have an effect (LOELs) on the aquatic species tested have been determined for several PAHs as well as for PAHs as a class of chemicals (IRIS, 1994). These LOELs, which are established when the minimum data required to derive AWQCs are not available, are presented in Table 11.

Table 10. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
Coal Tar Pitch Volatiles[a]			
TWA	0.2 c	0.1 c	0.2 c
STEL	--	--	--
CL	--	--	--
Asphalt Fumes[b]			
TWA	--	--	5
STEL	--	--	15
CL	--	--	25
Naphthalene			
TWA	50	50	52
STEL	75	75	79
CL	--	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.
STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.
CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.
c = designates that this chemical is regulated as a potential carcinogen.

[a]Benzene soluble fraction for OSHA PELs, cyclohexane extractable fraction for NIOSH RELs; includes acenaphthene, acenaphthylene, anthracene, benzo(a)pyrene, chrysene, fluoranthene, fluorene, phenanthrene, and pyrene.
[b]Includes fluoranthene.

Source: ESE.

Table 11. Ambient Water Quality Criteria (μg/L)*

PAH/LOEL	Freshwater	Marine
Acenaphthene#		
Acute	1,700	970
Chronic	520	710
Fluoranthene		
Acute	3,980	40
Chronic	--	16
Naphthalene		
Acute	2,300	2,350
Chronic	620	--
Phenanthrene		
Acute	30	77
Chronic	6.3	4.6
PAHs as a class, unless otherwise specified		
Acute	--	300
Chronic	--	--

Note: -- = not available.

*All values from IRIS, 1994 unless otherwise noted.
#Values from EPA, 1987.

Source: ESE.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Prepared by Clement International Corporation. U.S. Department of Health and Human Services, Washington, DC. TP-90-20. NTIS No. PB91-181537.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Clayton, G.D. and Clayton, F.E., eds. 1981. Patty's Industrial Hygiene and Toxicology. Volumes 2A, 2B, and 2C. Third Revised Ed. John Wiley and Sons, NY.

Clement Associates, Inc. 1985. Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites. Final Report. For U.S. Environmental Protection Agency. Arlington, VA.

Integrated Risk Information System (IRIS). 1994. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

International Agency for Research on Cancer (IARC). 1983. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 32: Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental Data. World Health Organization, Lyon, France.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1994. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 23, Expires 1/31/95. Managed by Micromedex, Inc., Denver, CO.

Radding, S.B., Mill, T., Gould, C.W., Liu, P.H., Johnson, H.L., Bomberger, D.C., Fojo, C.V. 1976. The Environmental Fate of Selected Polynuclear Aromatic Hydrocarbons. For U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, DC. (Cited in EPA, 1980).

Rodgers, J.H., Jr., Dickson, K.L., Saleh, F.Y. and Staples, C.A. 1983. Use of Microcosms to Study Transport, Transformation, and Fate of Organics in Aquifer Systems. Environ. Toxicol. Chem., 2(2):155-167.

REFERENCES
(Continued, Page 2 of 3)

Southworth, G.R. 1979. Transport and Transformation of Anthracene in Natural Waters. In: Aquatic Toxicology. L.L. Marking and R.A. Kimerle (eds.). ASTM STP 667. American Society for Testing and Materials, Philadelphia, PA. (Cited in USFWS, 1987).

Trucco, R.G., Engelhardt, F.R. and Stacey, B. 1983. Toxicity, Accumulation and Clearance of Aromatic Hydrocarbons in Daphnia pulex. Environ. Pollut., 31A(3) 191-202.

U.S. Environmental Protection Agency (EPA). 1976. The Environmental Fate of Selected Polynuclear Aromatic Hydrocarbons. Office of Toxic Substances, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC. EPA 440/5-80-069.

U.S. Environmental Protection Agency (EPA). 1982. An Exposure and Risk Assessment for Benzo(a)pyrene and Other Polycyclic Aromatic Hydrocarbons. Final Draft Report. Volumes I-IV. Office of Water Regulations and Standards, Monitoring and Data Support Division, Washington, DC. Contract Number 68-01-6160.

U.S. Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Office of Health and Environmental Assessment and Environmental Criteria and Assessment Office, Cincinnati, OH for Office of Emergency and Remedial Response, Washington, DC. EPA 540/1-86/013.

U.S. Environmental Protection Agency (EPA). 1987. Quality Criteria for Water 1986. Update No. 2. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1991. Drinking Water Criteria Document for PAH. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Office of Water Regulations and Standards, Washington, DC.

REFERENCES
(Continued, Page 3 of 3)

U.S. Environmental Protection Agency (EPA). 1992a. Addendum to the Region IV Supplement to the Risk Assessment Guidance for Superfund (RAGS). Region IV, Atlanta, GA. February 10, 1992.

U.S. Environmental Protection Agency (EPA). 1992b. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1992. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC. OERR 9200.6-303 (92-1). NTIS No. PB92-921199.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Research and Development and Office of Emergency and Remedial Response, Washington, DC.

U.S. Fish and Wildlife Service (USFWS). 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Biological Report 85(1.11). Washington, DC.

Zepp, R.G. and Schlotzhauer, P.F. 1979. Photoreactivity of Selected Aromatic Hydrocarbons in Water. In: Polynuclear Aromatic Hydrocarbons, P.W. Jones and P. Leber, Eds. Ann Arbor Science Publications, Ann Arbor, MI.

VINYL CHLORIDE
(CAS No. 75-01-4)

INTRODUCTION

Approximately 96 percent of the vinyl chloride (also known as chloroethene) produced in the U.S. is used in the production of polyvinyl chloride (PVC) and other vinyl polymers (ORNL, 1989). The remaining 4 percent is used in the synthesis of 1,1,1-trichloroethane. While PVC is used throughout most industries, the plumbing and electrical industries account for the largest portion of the PVC used today. Another large user of PVC is the food industry where PVC is used in plasticized film, bottles, and bottle-cap liners and gaskets; it has been banned from use with alcoholic beverages because of the migration of the vinyl chloride monomer into the alcohol (ORNL, 1989).

Based on its high water solubility of 1,100 mg/L at 20°C and its low soil sorption coefficient (K_{oc}) value of 8.2, vinyl chloride is expected to be highly mobile in the soil/groundwater system with little or no retardation in subsurface or sandy soil (ORNL, 1989). With a reported vapor pressure of 2,300 torr at 20°C, vinyl chloride is extremely volatile, and movement through the air-filled pores of the near-surface soils is a major transport pathway (ORNL, 1989). Based on an equilibrium partitioning model, approximately 97 percent of the compound present in the unsaturated near-surface soil at 2°C will be partitioned to the soil-air compartment, with about 2 percent adsorbed to the soil and about 1 percent present in the soil-water compartment (ORNL, 1989). In the saturated deep soil where soil organic carbon and soil air are negligible, about 97 percent is expected to be present in the soil-water compartment, with the remaining 3 percent sorbed to the soil (ORNL, 1989). Soil half-lives of 0.2 and 0.5 day were reported when vinyl chloride was incorporated to a depth of 1 and 10 cm, respectively (ORNL, 1989).

Degradation in the soil is limited; vinyl chloride is resistant to biodegradation in aerobic systems. Following exposure to methanogenic microorganisms in sand, vinyl chloride was 50- and 100-percent degraded within 4 and 11 weeks, respectively, under laboratory conditions; these degradation rates were reduced to 20 and 55 percent, respectively, in the absence of sand (ORNL, 1989). In water, a half-life of 0.805 hour is estimated for a river 1 m deep flowing at 3 m/sec and with a wind velocity of 3 m/sec in the air immediately above the river. In water, no photodegradation was observed after 90 hours, but in the presence of free radicals or of sensitizers that are found in sediments, degradation may occur rapidly (ORNL, 1989). In the atmosphere, vinyl chloride exists primarily in the vapor phase. Any gas-phase vinyl chloride will degrade rapidly as a result of reacting with photochemically produced hydroxyl radicals; the half-life is estimated to be 1.5 days (HSDB, 1991). In the presence of nitrogen oxides, its reactivity is even greater, leading to an estimated half-life of 3 to 7 hours (HSDB, 1991).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

EPA has not yet reviewed vinyl chloride with the purpose of deriving oral and inhalation reference doses (RfDs) (IRIS, 1995; EPA, 1994). However, the Agency for Toxic Substances and Disease Registry (ATSDR) does provide a chronic oral minimal risk level (MRL) of 0.0013 mg/kg/day (1989). This MRL is based on a lifetime dietary study in rats by Til et al. (1983) in which hepatotoxicity was observed with a NOAEL of 0.13 mg/kg/day and incorporates an uncertainty factor of 100 (10 for animal-to-human extrapolation and 10 for sensitive human subpopulations).

Following acute inhalation exposure, vinyl chloride causes CNS depression (HSDB, 1995). An acute exposure level of 1,000 ppm represents the minimum exposure level at which symptoms, including drowsiness, slight visual disturbances, faltering gait, numbness, and a tingling of the extremities, are

reported (HSDB, 1995). No clinical disturbances in humans were reported following a 7-hour exposure to a concentration of 500 ppm (ORNL, 1989).

Little acute exposure data are available regarding experimental animals; the 2-hour LC_{50} for mice and rabbits is 113,000 ppm, 150,000 ppm for rats, and 230,000 ppm for guinea pigs (ORNL, 1989). All three species died as a result of narcosis within 30 minutes of exposure to 300,000 ppm; pulmonary edema was noted, but liver and kidney injuries were low (ORNL, 1989).

In addition to the carcinogenic effects of chronic exposure, a decreased number of platelets in the blood, an enlarged spleen, and decreased pulmonary function were reported (ORNL, 1989). Another less frequent effect in humans is a condition known as acroosteolysis, a disease of the hand identified with workers in the vinyl chloride industry. In this disease, growths are found on the hand, the fingers swell and shorten to become club-like, and the individual develops Raynaud's phenomenon, which is a vasomotor disturbance often characterized by acute pain in the fingers that is aggravated by cold (HSDB, 1995). Other long-term effects include hepatic damage as a result of interference with essential pathways, potentially leading to cytotoxic (necrosis or steatosis) and/or cholestatic (biliary stasis) injuries (HSDB, 1995).

Carcinogenicity

Carcinogenic slope factors (CSFs) and supporting information are summarized in Table 1. Having reviewed the carcinogenicity of vinyl chloride, EPA has classified it as a group A (known human) carcinogen by both the inhalation and ingestion routes (EPA, 1994). This classification indicates that sufficient evidence exists of carcinogenicity in humans. An oral CSF of 1.9 $(mg/kg/day)^{-1}$ has been derived from a study in which rats exposed to dietary levels of 10 to 50 mg/kg evidenced lung carcinomas (EPA, 1994). An inhalation CSF of 0.3 $(mg/kg/day)^{-1}$ was derived from a 1-year study in which experimental animals, including several rat strains, were exposed 5 days/week for 1 year to

Table 1. Carcinogenic Slope Factors and Potential Carcinogenic Effects

Chemical	Carcinogenic Slope Factor (CSF) $(mg/kg/day)^{-1}$	Weight-of-Evidence Classification	Type or Site of Cancer	CSF Basis/ CSF Source
Oral	1.9	A	Liver, Lung	EPA, 1994
Inhalation	0.3[a]	A	Liver	EPA, 1994

Note: A = known human carcinogen (adequate evidence of carcinogenicity in human epidemiological studies).

[a]Inhalation CSF based on an inhalation unit risk of 8.4×10^{-5} $(\mu g/m^3)^{-1}$ (EPA, 1994) and assumes that a healthy 70-kilogram adult inhales 20 m^3/day of air.

Source: ESE.

airborne vinyl chloride concentrations that ranged from 1 to 30,000 ppm and to oral doses of 0.03 to 50 mg/kg/day (USAF, 1990; EPA, 1994). The primary effect following inhalation exposure was the formation of liver angiosarcomas, although a wide range of neoplasms were also identified in the kidneys, brain, skin, and the zymbal gland (ORNL, 1989).

Within a group of humans who worked for at least a year in positions involving probable exposure to vinyl chloride, a significant excess of malignant neoplasms of the brain and other areas of the CNS was reported (HSDB, 1995). While hepatic carcinomas have also been associated with exposure to vinyl chloride, the evidence of respiratory tract and digestive tract carcinomas is inconclusive but suggests that vinyl chloride is the causative agent (HSDB, 1995). Carcinogenic effects have also been observed in human fetuses following maternal exposure (ORNL, 1989). In mice and rats, air concentrations as low as 50 ppm have led to liver angiosarcomas as well as other forms of cancer (HSDB, 1995).

With respect to the listed CSFs, EPA (1994) states the following:

> "The most recently reviewed quantitative toxicity values listed here (Table 1) appear in EPA documents published in 1984 and 1985. Use of the values on an interim basis was validated by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) (04/05/90). The Agency is aware that these values do not incorporate considerable information that is now available. The Office of Health and Environmental Assessment's position is that these toxicity values do not reflect state-of-the-art science for vinyl chloride. EPA now has individual animal data, not available when the oral unit risk was calculated, that may influence this value. Additional information that may be factored into a revised quantitative toxicity value includes data on increased sensitivity observed in young animals and data on metabolism/ pharmacokinetics. A unit risk for air that considers information on young age exposure increases the risk (i.e., lowers the risk-specific dose) by at least 3-fold. The consideration of metabolism/ pharmacokinetics will further increase the risk. One unpublished physiologically-based pharmacokinetic model prediction results in a 100-fold increased risk."

Mutagenicity

Chromosome aberrations have been reported in workers occupationally exposed to vinyl chloride, with the most significant abnormalities associated with the highest exposures (HSDB, 1995; ORNL, 1989). Exposures to concentrations below 15 ppm are not expected to result in aberrations (ORNL, 1989).

No mutagenic effects were observed in male mice exposed for 5 days to levels as high as 30,000 ppm. Cells evidenced some mutations (HSDB, 1995) when incubated with liver supernatant from phenobarbital-pretreated rats, which acts as an activator. Mutations were observed in several Salmonella strains and other bacteria, including Escherichia coli strains (ORNL, 1989). It was also mutagenic in the recessive lethal test using Drosophila melanogaster but not in tests of translocations or sex-chromosome loss.

Teratogenicity/Reproductive Effects

While an increased number of birth defects, including CNS defects, deformities of the upper alimentary and genital tracts, and clubfoot, are associated with exposure to vinyl chloride, the evidence is still unable to show a definitive cause-and-effect relationship (ORNL, 1989; HSDB, 1995). In experimental animals, female mice, rats, and rabbits exposed to levels as high as 2,500 ppm during gestation did not evidence gross teratogenic effects; however, an increase occurred in minor skeletal abnormalities at 500 ppm and higher levels (ORNL, 1989). In a Russian study, pregnant rats exposed continuously to a mean daily concentration of 6.15 mg/m^3 (2.40 ppm) during gestation evidenced embryotoxic and teratogenic effects as manifested by elevated total embryonal mortality, lowered fetal weight, and the induction of external and internal anomalies in fetal development (HSDB, 1995). A value of 10 mg/m^3 (3.9 ppm) was determined to be the threshold value.

Ungvary et al. (1978) exposed rats to 1500 ppm vinyl chloride during pregnancy and observed increased fetal mortality but no malformations. Salnikova and

Kitsovskaya (1980) exposed Wistar rats to 4.8 mg/m^3 vinyl chloride during the entire gestation by inhalation. An alteration of blood vessel permeability, nervous system functional disturbance, and other abnormalities in offspring were found. A dose of 35.3 mg/m^3 produced a slight embryotoxic effect.

ECOTOXICITY

Aquatic Organisms

Information concerning toxicity of vinyl chloride to aquatic organisms is sparse. When bacterial populations were exposed to levels as high as 900,000 μg/L, no effect was reported, indicating that vinyl chloride is not toxic to bacteria at that concentration (EPA, 1980).

Terrestrial Organisms

No information was identified in the available literature regarding the toxicity of vinyl chloride to vegetation, livestock, or terrestrial wildlife.

STANDARDS AND CRITERIA

Human Receptors

A summary of drinking water standards and criteria is presented as Table 2. Due to the ranking of vinyl chloride as a Group A human carcinogen, EPA has set a nonenforceable Maximum Contaminant Level Goal (MCLG) for drinking water of zero μg/L and a Maximum Contaminant Level (MCL) of 2 μg/L, which is enforceable for public water supplies. Also due to its carcinogenic ranking, the EPA ambient water quality criterion (AWQC) for the protection of human health should be zero μg/L. However, zero may not be attainable with present technology; therefore, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 20 to 0.2 μg/L for consumption of contaminated drinking water and aquatic organisms (IRIS, 1995). For consumption of aquatic organisms only, the recommended AWQC for the 10^{-5} to the 10^{-7} risk levels range from 525 to 5.25 μg/L, respectively (IRIS, 1995). These AWQC would yield values for

Table 2. Human Regulatory Criteria (μg/L)

Compound	SDWA			Drinking Water Health Advisories				AWQC[a]		
	Primary MCL	MCL Goal	Secondary MCL	1-Day	10-Day	Longterm	Lifetime	Fish and Water	Fish Only	Water Only[a]
Vinyl chloride	2	0	--	--	--	--	--	2	525	2

Note: SDWA = EPA Safe Drinking Water Act.
MCL = maximum contaminant level.
MCLG = maximum contaminant level goal.
AWQC = EPA ambient water quality criteria.
-- = no value available.

[a]Carcinogenic; value represents 10^{-6} cancer risk.
[b]Value is based on the following formula:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}.$$

Source: ESE.

human consumption of water alone (W) of 20, 2, and 0.2 μg/L for 10^{-5}, 10^{-6}, and 10^{-7} risks, respectively, based on the following equation:

$$\frac{1}{W+F} = \frac{1}{W} + \frac{1}{F}$$

Although vinyl chloride has been designated as a hazardous air pollutant under Section 112 of the Clean Air Act (40 CFR 61.01; (7/1/88), no national ambient air quality standards (NAAQS) have been developed for this chemical. However, the Occupational Safety and Health Administration (OSHA) has promulgated enforceable permissible exposure limits (PELs) for worker exposure (NIOSH, 1995). In addition, the National Institute of Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH) have developed nonenforceable recommended exposure levels (RELs) and threshold limit values (TLVs), respectively (NIOSH, 1995; ACGIH, 1993). These values are presented in Table 3.

Ecological Receptors

No AWQCs have been developed for the protection of freshwater or marine organisms against exposure to vinyl chloride, and the lowest levels observed to have an effect (LOELs) on aquatic species have not been determined (EPA, 1987).

Table 3. Air Quality Standards

Standard (mg/m^3)	OSHA PEL	NIOSH REL	ACGIH TLV
TWA	2.6 c	c	13 c
STEL	--	--	--
CL	13 (15 min.)	--	--

Note: TWA = time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed on a daily basis without adverse effect.
STEL = short-term exposure limit; 15-minute (unless otherwise specified) TWA exposure that should not be exceeded at any time during a workday even if the 8-hour TWA is not exceeded.
CL = ceiling level; unless otherwise specified, the concentration that should not be exceeded at any time during a workday.
c = designates that this chemical is regulated as a potential carcinogen.

Source: ESE.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Vinyl Chloride. Prepared by Syracuse Research Corporation. U.S. Department of Health and Human Services, Washington, DC. ATSDR/TP-88/25. NTIS No. PB90-103870.

American Conference of Governmental Industrial Hygienists (ACGIH). 1993. 1993 - 1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices. Technical Information Office, Cincinnati, OH. ISBN: 1-882417-03-8.

Hazardous Substances Data Bank (HSDB). 1995. U.S. Department of Health and Human Services. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Integrated Risk Information System (IRIS). 1995. U.S. Environmental Protection Agency (EPA). Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

NIOSH Pocket Guide to Chemical Hazards (NIOSH). 1995. National Institute of Occupational Safety and Health. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Oak Ridge National Laboratory (ORNL). 1989. Installation Restoration Program Toxicology Guide. Biomedical and Environmental Information Analysis, Health and Safety Research Division. Prepared for U.S. Air Force Systems Command, Wright-Patterson Air Force Base. Oak Ridge, TN.

Salnikova, L.S. and Kitsovskaya, I.A.. 1980. Effect of vinyl chloride on embryogenesis in the rat. Gig. Tr. Prof. Zabol., 3:46-47. (cited in Shepard, 1995).

Shepard's Catalog of Teratogenic Agents. 1995. T.H. Shepard. Micromedex TOMES PLUS® System CD/ROM, Version 25, Expires 7/31/95. Managed by Micromedex, Inc., Denver, CO.

Til, H.P., Immel, H.R., and Feron, V.J. 1983. Lifespan oral carcinogenicity study of vinyl chloride in rats. Final Report. Civo Institues, TNO. Report No. V 93.285/291099. (cited in ATSDR, 1989).

REFERENCES
(Continued, Page 2 of 2)

Ungvary, G., Hudak, A., Tetrai, E., Lorincz, M., and Folly, G. 1978. Effects of vinyl chloride exposure alone and in combination with trypan blue, applied systematically during all thirds of pregnancy on the fetuses of CFY rats. Toxicol., 11:45-54. (cited in Shepard, 1995).

U.S. Air Force (USAF). 1990. The Installation Restoration Program; Remedial Investigation/Feasibility Study; Stage 2 for Dobbins Air Force Base, GA; Statement of Work (SOW), Modification 3. Contract No. F33615-84-D-4401/0018.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Vinyl Chloride. Office of Water Regulations and Standards. Washington, DC. EPA 440/5-80/078.

U.S. Environmental Protection Agency (EPA). 1987. Quality Criteria for Water; Update No. 2. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC.

U.S. Environmental Protection Agency (EPA). 1994. Health Effects Assessment Summary Tables (HEAST). Annual Update FY 1994. Office of Solid Waste and Emergency Response, Washington, DC. EPA 540/R-94/020. Publication No. 9200.6-303(94-1). NTIS No. PB94-921199.

APPENDIX F

HUMAN RISKS AND HAZARD INDICES

REDSTONE BRA1 ARES
NONCARCINOGENIC HAZARD INDICES

Area F; Current Adult Worker; Chronic

ANALYTE	MEDIUM				GRAND TOTAL
	Soil				
	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		
Acenaphthene	4.0E-06		7.5E-06	1.1E-05	1.1E-05
Acetone	4.2E-07		7.8E-07	1.2E-06	1.2E-06
Anthracene	7.8E-07		1.5E-06	2.2E-06	2.2E-06
Arsenic	6.2E-06		1.2E-04	1.2E-04	1.2E-04
Benz(a)anthracene	1.7E-05		3.2E-05	4.9E-05	4.9E-05
Benzo(a)pyrene	1.2E-05		2.2E-05	3.4E-05	3.4E-05
Benzo(b)fluoranthene	1.7E-05		3.2E-05	5.0E-05	5.0E-05
Benzo(ghi)perylene	8.9E-06		1.7E-05	2.5E-05	2.5E-05
Bis(2-ethylhexyl) phthalate	4.1E-05		7.6E-05	1.2E-04	1.2E-04
Cadmium (solid matrix)	1.2E-08		2.3E-07	2.4E-07	2.4E-07
Chrysene	1.4E-05		2.7E-05	4.1E-05	4.1E-05
Dibenz(ah)anthracene	7.5E-06		1.4E-05	2.1E-05	2.1E-05
Fluoranthene	2.2E-05		4.1E-05	6.3E-05	6.3E-05
Fluorene	6.3E-06		1.2E-05	1.8E-05	1.8E-05
Indeno(1,2,3-cd)pyrene	9.7E-06		1.8E-05	2.8E-05	2.8E-05
Mercury	3.1E-07	2.2E-07	5.8E-06	6.4E-06	6.4E-06
Methylene chloride	7.5E-08	1.0E-10	1.4E-07	2.1E-07	2.1E-07
Naphthalene	5.1E-06		9.6E-06	1.5E-05	1.5E-05
Phenanthrene	1.9E-05		3.6E-05	5.5E-05	5.5E-05
Pyrene	2.6E-05		4.9E-05	7.5E-05	7.5E-05
TOTAL	2.2E-04	2.2E-07	5.2E-04	7.3E-04	7.3E-04

Area F; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Acenaphthene					4.0E-06		7.5E-06	1.1E-05	1.1E-05
Acetone	1.3E-05		6.3E-03	6.3E-03	4.2E-07		7.8E-07	1.2E-06	6.3E-03
Anthracene					7.8E-07		1.5E-06	2.2E-06	2.2E-06
Arsenic	4.8E-03		1.3E+00	1.3E+00	6.2E-06		1.2E-04	1.2E-04	1.3E+00
Barium	4.6E-05		1.3E-02	1.3E-02					1.3E-02
Benz(a)anthracene					1.7E-05		3.2E-05	4.9E-05	4.9E-05
Benzo(a)pyrene					1.2E-05		2.2E-05	3.4E-05	3.4E-05
Benzo(b)fluoranthene					1.7E-05		3.2E-05	5.0E-05	5.0E-05
Benzo(ghi)perylene					8.9E-06		1.7E-05	2.5E-05	2.5E-05
Bis(2-ethylhexyl) phthalate					4.1E-05		7.6E-05	1.2E-04	1.2E-04
Cadmium (aqueous matrix)	2.6E-04		7.2E-02	7.3E-02					7.3E-02
Cadmium (solid matrix)					1.2E-08		2.3E-07	2.4E-07	2.4E-07
Carbon tetrachloride	8.9E-03	3.0E-01	1.1E-01	4.2E-01					4.2E-01
Chloroform	3.8E-04		1.2E-02	1.2E-02					1.2E-02
Chromium, total	9.8E-05		1.4E-02	1.4E-02					1.4E-02
Chrysene					1.4E-05		2.7E-05	4.1E-05	4.1E-05
Dibenz(ah)anthracene					7.5E-06		1.4E-05	2.1E-05	2.1E-05
Fluoranthene					2.2E-05		4.1E-05	6.3E-05	6.3E-05
Fluorene					6.3E-06		1.2E-05	1.8E-05	1.8E-05
Indeno(1,2,3-cd)pyrene					9.7E-06		1.8E-05	2.8E-05	2.8E-05
Lead									
Mercury	3.9E-02		1.1E+01	1.1E+01	3.1E-07	2.2E-07	5.8E-06	6.4E-06	1.1E+01
Methylene chloride					7.5E-08	1.0E-10	1.4E-07	2.1E-07	2.1E-07
Naphthalene					5.1E-06		9.6E-06	1.5E-05	1.5E-05
Phenanthrene					1.9E-05		3.6E-05	5.5E-05	5.5E-05

(CONTINUED)

Area F; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Pyrene					2.6E-05		4.9E-05	7.5E-05	7.5E-05
Trichloroethene	6.3E-04		5.4E-03	6.0E-03					6.0E-03
TOTAL	5.4E-02	3.0E-01	1.2E+01	1.3E+01	2.2E-04	2.2E-07	5.2E-04	7.3E-04	1.3E+01

Area G; Current Adult Recreation; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	FS		Sediment			Surface Water			
	PATHWAY		PATHWAY			PATHWAY			
	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	
Acetone	2.2E-06	2.2E-06	2.5E-07	1.2E-07	3.6E-07	3.9E-06	3.2E-05	3.6E-05	3.9E-05
Arsenic			1.5E-07	7.2E-07	8.7E-07				8.7E-07
Barium	1.2E-04	1.2E-04	1.1E-08	5.3E-08	6.4E-08	2.5E-05	1.2E-04	1.4E-04	2.6E-04
Chromium, total			1.2E-07	5.5E-07	6.7E-07				6.7E-07
Dichloroethane, 1,1-	3.8E-07	3.8E-07				5.6E-06	3.0E-06	8.6E-06	8.9E-06
Methylene chloride	4.9E-06	4.9E-06	3.8E-08	1.8E-08	5.6E-08	9.3E-06	9.7E-06	1.9E-05	2.4E-05
Trichloroethene	2.9E-04	2.9E-04				1.8E-03	2.7E-04	2.1E-03	2.4E-03
Xylenes, total	3.9E-08	3.9E-08				3.4E-06	1.8E-07	3.6E-06	3.6E-06
TOTAL	4.1E-04	4.1E-04	5.7E-07	1.5E-06	2.0E-06	1.9E-03	4.3E-04	2.3E-03	2.7E-03

REDSTONE BRA1 ARES
NONCARCINOGENIC HAZARD INDICES

Area G; Current Child Recreation; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	FS		Sediment			Surface Water			
	PATHWAY		PATHWAY			PATHWAY			
	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	
Acetone	4.4E-06	4.4E-06	3.8E-07	1.1E-06	1.5E-06	6.1E-06	1.5E-04	1.6E-04	1.6E-04
Arsenic			2.4E-07	6.7E-06	7.0E-06				7.0E-06
Barium	2.3E-04	2.3E-04	1.7E-08	4.9E-07	5.1E-07	3.9E-05	5.5E-04	5.9E-04	8.2E-04
Chromium, total			1.8E-07	5.2E-06	5.4E-06				5.4E-06
Dichloroethane, 1,1-	7.5E-07	7.5E-07				8.6E-06	1.4E-05	2.2E-05	2.3E-05
Methylene chloride	9.6E-06	9.6E-06	5.9E-08	1.7E-07	2.3E-07	1.4E-05	4.5E-05	5.9E-05	6.9E-05
Trichloroethene	5.6E-04	5.6E-04				2.8E-03	1.3E-03	4.1E-03	4.7E-03
Xylenes, total	7.6E-08	7.6E-08				5.2E-06	8.3E-07	6.0E-06	6.1E-06
TOTAL	8.1E-04	8.1E-04	8.7E-07	1.4E-05	1.5E-05	2.9E-03	2.0E-03	4.9E-03	5.7E-03

Area G; Future Adult Worker; Chronic

ANALYTE	MEDIUM														GRAND TOTAL
	Ground Water				Sediment			Soil				Surface Water			
	PATHWAY				PATHWAY			PATHWAY				PATHWAY			
	Dermal	Inhala-tion	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	Dermal	Oral	TOTAL	
Acetone	2.4E-06		1.2E-03	1.2E-03	6.4E-08	1.8E-07	2.4E-07	8.1E-07		1.5E-06	2.3E-06	2.6E-07	2.5E-06	2.7E-06	1.2E-03
Arsenic	9.0E-04		2.5E-01	2.5E-01	4.0E-08	1.1E-06	1.1E-06								2.5E-01
Barium	1.0E-03		2.9E-01	2.9E-01	2.9E-09	8.1E-08	8.4E-08					1.6E-06	9.0E-06	1.1E-05	2.9E-01
Cadmium (aqueous matrix)	2.3E-03		6.2E-01	6.3E-01											6.3E-01
Chromium, total	4.5E-03		6.2E-01	6.3E-01	3.1E-08	8.5E-07	8.8E-07								6.3E-01
Dichloroethane, 1,1-												3.7E-07	2.3E-07	5.9E-07	5.9E-07
Lead															
Methylene chloride					1.0E-08	2.8E-08	3.8E-08	5.3E-08	7.4E-11	9.8E-08	1.5E-07	6.1E-07	7.4E-07	1.3E-06	1.5E-06
Trichloroethene												1.2E-04	2.1E-05	1.4E-04	1.4E-04
Xylenes, total												2.2E-07	1.4E-08	2.3E-07	2.3E-07
TOTAL	8.8E-03		1.8E+00	1.8E+00	1.5E-07	2.2E-06	2.4E-06	8.6E-07	7.4E-11	1.6E-06	2.5E-06	1.2E-04	3.3E-05	1.6E-04	1.8E+00

Area R; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY				PATHWAY				
	Dermal	Inhala-tion	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	
Acenaphthene					1.7E-06		3.2E-06	4.9E-06	4.9E-06
Acetone	8.8E-06		4.3E-03	4.3E-03	8.4E-07		1.6E-06	2.4E-06	4.3E-03
Anthracene					4.0E-07		7.5E-07	1.2E-06	1.2E-06
Arsenic	7.1E-04		2.0E-01	2.0E-01					2.0E-01
Barium	3.5E-04		9.8E-02	9.8E-02					9.8E-02
Benz(a)anthracene					1.2E-05		2.2E-05	3.4E-05	3.4E-05
Benzo(a)pyrene					8.7E-06		1.6E-05	2.5E-05	2.5E-05
Benzo(b)fluoranthene					1.6E-05		3.0E-05	4.6E-05	4.6E-05
Benzo(ghi)perylene					5.8E-06		1.1E-05	1.7E-05	1.7E-05
Bis(2-chloroethyl) ether	6.1E-04		8.0E-02	8.1E-02					8.1E-02
Bis(2-ethylhexyl) phthalate	2.5E-04		3.0E-03	3.2E-03	1.0E-05		1.9E-05	2.9E-05	3.3E-03
Butylbenzyl phthalate					1.0E-06		1.9E-06	2.9E-06	2.9E-06
Cadmium (aqueous matrix)	8.8E-04		2.4E-01	2.4E-01					2.4E-01
Chlorobenzene	1.0E-03	5.3E-02	6.9E-03	6.1E-02					6.1E-02
Chloroform	1.1E-03		3.6E-02	3.7E-02					3.7E-02
Chromium, total	4.3E-03		5.9E-01	5.9E-01					5.9E-01
Chrysene					8.7E-06		1.6E-05	2.5E-05	2.5E-05
Fluoranthene					1.4E-05		2.7E-05	4.1E-05	4.1E-05
Fluorene					2.6E-06		4.8E-06	7.4E-06	7.4E-06
Indeno(1,2,3-cd)pyrene					5.8E-06		1.1E-05	1.7E-05	1.7E-05
Lead									
Methylene chloride	7.2E-06	6.7E-05	4.4E-04	5.2E-04	3.9E-08	5.5E-11	7.3E-08	1.1E-07	5.2E-04
Phenanthrene					1.4E-05		2.6E-05	4.0E-05	4.0E-05
Pyrene					1.9E-05		3.5E-05	5.3E-05	5.3E-05
Selenium					4.4E-09		1.6E-07	1.7E-07	1.7E-07
TOTAL	9.3E-03	5.3E-02	1.3E+00	1.3E+00	1.2E-04	5.5E-11	2.3E-04	3.5E-04	1.3E+00

REDSTONE BRA1 ARES
NONCARCINOGENIC HAZARD INDICES

Area F; Current Adult Worker; Chronic

ANALYTE	MEDIUM				GRAND TOTAL
	Soil				
	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		
Acenaphthene	9.6E-07		8.9E-07	1.9E-06	1.9E-06
Acetone	1.0E-07		9.4E-08	1.9E-07	1.9E-07
Anthracene	1.9E-07		1.8E-07	3.6E-07	3.6E-07
Arsenic	1.5E-06		1.4E-05	1.5E-05	1.5E-05
Benz(a)anthracene	4.1E-06		3.8E-06	7.9E-06	7.9E-06
Benzo(a)pyrene	2.8E-06		2.7E-06	5.5E-06	5.5E-06
Benzo(b)fluoranthene	4.1E-06		3.9E-06	8.0E-06	8.0E-06
Benzo(ghi)perylene	2.1E-06		2.0E-06	4.1E-06	4.1E-06
Bis(2-ethylhexyl) phthalate	9.8E-06		9.2E-06	1.9E-05	1.9E-05
Cadmium (solid matrix)	2.9E-09		2.7E-08	3.0E-08	3.0E-08
Chrysene	3.4E-06		3.2E-06	6.6E-06	6.6E-06
Dibenz(ah)anthracene	1.8E-06		1.7E-06	3.5E-06	3.5E-06
Fluoranthene	5.3E-06		4.9E-06	1.0E-05	1.0E-05
Fluorene	1.5E-06		1.4E-06	2.9E-06	2.9E-06
Indeno(1,2,3-cd)pyrene	2.3E-06		2.2E-06	4.5E-06	4.5E-06
Mercury	7.5E-08	2.2E-07	7.0E-07	9.9E-07	9.9E-07
Methylene chloride	1.8E-08	1.0E-10	1.7E-08	3.5E-08	3.5E-08
TOTAL	4.0E-05	2.2E-07	5.1E-05	9.1E-05	9.1E-05

REDSTONE BRA1 ARES
NONCARCINOGENIC HAZARD INDICES

Area F; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Acenaphthene					4.0E-06		7.5E-06	1.1E-05	1.1E-05
Acetone	1.3E-05		6.3E-03	6.3E-03	4.2E-07		7.8E-07	1.2E-06	6.3E-03
Anthracene					7.8E-07		1.5E-06	2.2E-06	2.2E-06
Arsenic	4.8E-03		1.3E+00	1.3E+00	6.2E-06		1.2E-04	1.2E-04	1.3E+00
Barium	4.6E-05		1.3E-02	1.3E-02					1.3E-02
Benz(a)anthracene					1.7E-05		3.2E-05	4.9E-05	4.9E-05
Benzo(a)pyrene					1.2E-05		2.2E-05	3.4E-05	3.4E-05
Benzo(b)fluoranthene					1.7E-05		3.2E-05	5.0E-05	5.0E-05
Benzo(ghi)perylene					8.9E-06		1.7E-05	2.5E-05	2.5E-05
Bis(2-ethylhexyl) phthalate					4.1E-05		7.6E-05	1.2E-04	1.2E-04
Cadmium (aqueous matrix)	2.6E-04		7.2E-02	7.3E-02					7.3E-02
Cadmium (solid matrix)					1.2E-08		2.3E-07	2.4E-07	2.4E-07
Carbon tetrachloride	8.9E-03		1.1E-01	1.2E-01					1.2E-01
Chloroform	3.8E-04		1.2E-02	1.2E-02					1.2E-02
Chromium, total	9.8E-05		1.4E-02	1.4E-02					1.4E-02
Chrysene					1.4E-05		2.7E-05	4.1E-05	4.1E-05
Dibenz(ah)anthracene					7.5E-06		1.4E-05	2.1E-05	2.1E-05
Fluoranthene					2.2E-05		4.1E-05	6.3E-05	6.3E-05
Fluorene					6.3E-06		1.2E-05	1.8E-05	1.8E-05
Indeno(1,2,3-cd)pyrene					9.7E-06		1.8E-05	2.8E-05	2.8E-05
Lead									
Mercury	3.9E-02		1.1E+01	1.1E+01	3.1E-07	2.2E-07	5.8E-06	6.4E-06	1.1E+01
Methylene chloride					7.5E-08	1.0E-10	1.4E-07	2.1E-07	2.1E-07
Trichloroethene	6.3E-04		5.4E-03	6.0E-03					6.0E-03
TOTAL	5.4E-02		1.2E+01	1.2E+01	1.7E-04	2.2E-07	4.2E-04	5.9E-04	1.2E+01

Area G; Current Adult Recreation; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	FS		Sediment			Surface Water			
	PATHWAY		PATHWAY			PATHWAY			
	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	
Acetone	2.2E-06	2.2E-06	2.5E-07	2.3E-07	4.8E-07	3.9E-06	3.2E-05	3.6E-05	3.9E-05
Arsenic			1.5E-07	1.4E-06	1.6E-06				1.6E-06
Barium	1.2E-04	1.2E-04	1.1E-08	1.1E-07	1.2E-07	2.5E-05	1.2E-04	1.4E-04	2.6E-04
Chromium, total			1.2E-07	1.1E-06	1.2E-06				1.2E-06
Dichloroethane, 1,1-	3.8E-07	3.8E-07				5.6E-06	3.0E-06	8.6E-06	8.9E-06
Methylene chloride	4.9E-06	4.9E-06	3.8E-08	3.6E-08	7.4E-08	9.3E-06	9.7E-06	1.9E-05	2.4E-05
Trichloroethene	2.9E-04	2.9E-04				1.8E-03	2.7E-04	2.1E-03	2.4E-03
Xylenes, total	3.9E-08	3.9E-08				3.4E-06	1.8E-07	3.6E-06	3.6E-06
TOTAL	4.1E-04	4.1E-04	5.7E-07	2.9E-06	3.5E-06	1.9E-03	4.3E-04	2.3E-03	2.7E-03

Area G; Current Child Recreation; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	FS		Sediment			Surface Water			
	PATHWAY		PATHWAY			PATHWAY			
	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	
Acetone	4.4E-06	4.4E-06	3.8E-07	2.2E-06	2.5E-06	6.1E-06	1.5E-04	1.6E-04	1.6E-04
Arsenic			2.4E-07	1.3E-05	1.4E-05				1.4E-05
Barium	2.3E-04	2.3E-04	1.7E-08	9.8E-07	1.0E-06	3.9E-05	5.5E-04	5.9E-04	8.2E-04
Chromium, total			1.8E-07	1.0E-05	1.1E-05				1.1E-05
Dichloroethane, 1,1-	7.5E-07	7.5E-07				8.6E-06	1.4E-05	2.2E-05	2.3E-05
Methylene chloride	9.6E-06	9.6E-06	5.9E-08	3.4E-07	4.0E-07	1.4E-05	4.5E-05	5.9E-05	6.9E-05
Trichloroethene	5.6E-04	5.6E-04				2.8E-03	1.3E-03	4.1E-03	4.7E-03
Xylenes, total	7.6E-08	7.6E-08				5.2E-06	8.3E-07	6.0E-06	6.1E-06
TOTAL	8.1E-04	8.1E-04	8.7E-07	2.7E-05	2.8E-05	2.9E-03	2.0E-03	4.9E-03	5.8E-03

Area G; Future Adult Worker; Chronic

ANALYTE	MEDIUM														GRAND TOTAL
	Ground Water				Sediment			Soil				Surface Water			
	PATHWAY			TOTAL	PATHWAY		TOTAL	PATHWAY			TOTAL	PATHWAY		TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Oral		Dermal	Inhala-tion	Oral		Dermal	Oral		
Acetone	2.4E-06		1.2E-03	1.2E-03	6.4E-08	1.8E-07	2.4E-07	8.1E-07		1.5E-06	2.3E-06	2.6E-07	2.5E-06	2.7E-06	1.2E-03
Arsenic	9.0E-04		2.5E-01	2.5E-01	4.0E-08	1.1E-06	1.1E-06								2.5E-01
Barium	1.0E-03		2.9E-01	2.9E-01	2.9E-09	8.1E-08	8.4E-08					1.6E-06	9.0E-06	1.1E-05	2.9E-01
Cadmium (aqueous matrix)	2.3E-03		6.2E-01	6.3E-01											6.3E-01
Chromium, total	4.5E-03		6.2E-01	6.3E-01	3.1E-08	8.5E-07	8.8E-07								6.3E-01
Dichloroethane, 1,1-												3.7E-07	2.3E-07	5.9E-07	5.9E-07
Lead															
Methylene chloride					1.0E-08	2.8E-08	3.8E-08	5.3E-08	7.4E-11	9.8E-08	1.5E-07	6.1E-07	7.4E-07	1.3E-06	1.5E-06
Trichloroethene												1.2E-04	2.1E-05	1.4E-04	1.4E-04
Xylenes, total												2.2E-07	1.4E-08	2.3E-07	2.3E-07
TOTAL	8.8E-03		1.8E+00	1.8E+00	1.5E-07	2.2E-06	2.4E-06	8.6E-07	7.4E-11	1.6E-06	2.5E-06	1.2E-04	3.3E-05	1.6E-04	1.8E+00

Area R; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY				PATHWAY				
	Dermal	Inhala-tion	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	
Acenaphthene					1.7E-06		3.2E-06	4.9E-06	4.9E-06
Acetone	8.8E-06		4.3E-03	4.3E-03	8.4E-07		1.6E-06	2.4E-06	4.3E-03
Anthracene					4.0E-07		7.5E-07	1.2E-06	1.2E-06
Arsenic	7.1E-04		2.0E-01	2.0E-01					2.0E-01
Barium	3.5E-04		9.8E-02	9.8E-02					9.8E-02
Benz(a)anthracene					1.2E-05		2.2E-05	3.4E-05	3.4E-05
Benzo(a)pyrene					8.7E-06		1.6E-05	2.5E-05	2.5E-05
Benzo(b)fluoranthene					1.6E-05		3.0E-05	4.6E-05	4.6E-05
Benzo(ghi)perylene					5.8E-06		1.1E-05	1.7E-05	1.7E-05
Bis(2-chloroethyl) ether	6.1E-04		8.0E-02	8.1E-02					8.1E-02
Bis(2-ethylhexyl) phthalate	2.5E-04		3.0E-03	3.2E-03	1.0E-05		1.9E-05	2.9E-05	3.3E-03
Butylbenzyl phthalate					1.0E-06		1.9E-06	2.9E-06	2.9E-06
Cadmium (aqueous matrix)	8.8E-04		2.4E-01	2.4E-01					2.4E-01
Chlorobenzene	1.0E-03	5.3E-02	6.9E-03	6.1E-02					6.1E-02
Chloroform	1.1E-03		3.6E-02	3.7E-02					3.7E-02
Chromium, total	4.3E-03		5.9E-01	5.9E-01					5.9E-01
Chrysene					8.7E-06		1.6E-05	2.5E-05	2.5E-05
Fluoranthene					1.4E-05		2.7E-05	4.1E-05	4.1E-05
Fluorene					2.6E-06		4.8E-06	7.4E-06	7.4E-06
Indeno(1,2,3-cd)pyrene					5.8E-06		1.1E-05	1.7E-05	1.7E-05
Lead									
Methylene chloride	7.2E-06	6.7E-05	4.4E-04	5.2E-04	3.9E-08	5.5E-11	7.3E-08	1.1E-07	5.2E-04
Phenanthrene					1.4E-05		2.6E-05	4.0E-05	4.0E-05
Pyrene					1.9E-05		3.5E-05	5.3E-05	5.3E-05
Selenium					4.4E-09		1.6E-07	1.7E-07	1.7E-07

(CONTINUED)

Area R; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
TOTAL	9.3E-03	5.3E-02	1.3E+00	1.3E+00	1.2E-04	5.5E-11	2.3E-04	3.5E-04	1.3E+00

Area ST; Current Adult Worker; Chronic

ANALYTE	MEDIUM				
	Soil				
	PATHWAY			TOTAL	GRAND TOTAL
	Dermal	Inhala-tion	Oral		
Acetone	2.3E-07		1.7E-07	4.0E-07	4.0E-07
Bis(2-ethylhexyl) phthalate	2.5E-07		1.8E-07	4.2E-07	4.2E-07
DDE, p,p'-	2.2E-07		1.5E-07	3.7E-07	3.7E-07
DDT, p,p'-	1.4E-06		9.9E-07	2.4E-06	2.4E-06
Methylene chloride	7.9E-09	4.2E-12	5.6E-09	1.3E-08	1.3E-08
TOTAL	2.1E-06	4.2E-12	1.5E-06	3.6E-06	3.6E-06

REDSTONE BRA1 ARES
NONCARCINOGENIC HAZARD INDICES

Area ST; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Acenaphthene	4.8E-04		8.8E-04	1.4E-03					1.4E-03
Acetone	2.0E-05		9.6E-03	9.6E-03	2.4E-06		4.6E-06	7.0E-06	9.6E-03
Anthracene	1.4E-04		1.7E-04	3.1E-04					3.1E-04
Barium	6.1E-05		1.7E-02	1.7E-02					1.7E-02
Benzene	2.2E-04	2.1E+00	2.8E-03	2.1E+00					2.1E+00
Bis(2-ethylhexyl) phthalate					2.6E-06		4.8E-06	7.4E-06	7.4E-06
Cadmium (aqueous matrix)	2.0E-04		5.4E-02	5.5E-02					5.5E-02
Chlorobenzene	2.5E-02	1.3E+00	1.7E-01	1.5E+00					1.5E+00
Chloroform	2.6E-04		8.1E-03	8.4E-03					8.4E-03
Chromium, total	1.7E-04		2.4E-02	2.4E-02					2.4E-02
DDE, p,p'-					2.3E-06		4.2E-06	6.5E-06	6.5E-06
DDT, p,p'-					1.4E-05		2.7E-05	4.1E-05	4.1E-05
Dichloroethane, 1,2-	1.4E-05	3.7E-02	3.5E-04	3.7E-02					3.7E-02
Dichloroethenes, 1,2-, total	4.6E-04		1.3E-02	1.3E-02					1.3E-02
Ethylbenzene	6.8E-05	1.9E-04	2.5E-04	5.1E-04					5.1E-04
Fluoranthene	1.7E-03		1.3E-03	2.9E-03					2.9E-03
Methylene chloride	6.7E-06	6.1E-05	4.1E-04	4.8E-04	8.2E-08	1.2E-10	1.5E-07	2.4E-07	4.8E-04
Trichloroethane, 1,1,1-	7.1E-05	7.7E-04	1.1E-03	2.0E-03					2.0E-03
Vinyl chloride	1.7E-03		6.3E-02	6.4E-02					6.4E-02
TOTAL	3.1E-02	3.5E+00	3.7E-01	3.9E+00	2.2E-05	1.2E-10	4.1E-05	6.3E-05	3.9E+00

REDSTONE BRA1 ARES
CARCINOGENIC RISKS

Area F; Current Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)		MEDIUM				
		Soil				
		PATHWAY				
		Dermal	Inhala-tion	Oral	TOTAL	GRAND TOTAL
Arsenic	(A A A)	1.2E-09	2.0E-09	2.2E-08	2.5E-08	2.5E-08
Benz(a)anthracene	(B2 B2 B2)	1.3E-07	2.2E-09	2.5E-07	3.8E-07	3.8E-07
Benzo(a)pyrene	(B2 B2 B2)	9.3E-07	1.6E-08	1.7E-06	2.7E-06	2.7E-06
Benzo(b)fluoranthene	(B2 B2 B2)	1.4E-07	2.3E-09	2.5E-07	3.9E-07	3.9E-07
Bis(2-ethylhexyl) phthalate	(B2 B2 B2)	4.1E-09		7.6E-09	1.2E-08	1.2E-08
Cadmium (solid matrix)	(B1)		5.5E-12		5.5E-12	5.5E-12
Chrysene	(B2 B2 B2)	1.1E-09	1.9E-11	2.1E-09	3.2E-09	3.2E-09
Dibenz(ah)anthracene	(B2 B2 B2)	5.8E-07	9.8E-09	1.1E-06	1.7E-06	1.7E-06
Indeno(1,2,3-cd)pyrene	(B2 B2 B2)	7.5E-08	1.3E-09	1.4E-07	2.2E-07	2.2E-07
Methylene chloride	(B2 B2 B2)	1.2E-11	5.2E-14	2.2E-11	3.4E-11	3.4E-11
TOTAL		1.9E-06	3.3E-08	3.5E-06	5.4E-06	5.4E-06

Area F; Future Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)		MEDIUM								
		Ground Water				Soil				
		PATHWAY				PATHWAY				
		Dermal	Inhala-tion	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	GRAND TOTAL
Arsenic	(A A A)	8.9E-07		2.5E-04	2.5E-04	1.2E-09	2.0E-09	2.2E-08	2.5E-08	2.5E-04
Benz(a)anthracene	(B2 B2 B2)					1.3E-07	2.2E-09	2.5E-07	3.8E-07	3.8E-07
Benzo(a)pyrene	(B2 B2 B2)					9.3E-07	1.6E-08	1.7E-06	2.7E-06	2.7E-06
Benzo(b)fluoranthene	(B2 B2 B2)					1.4E-07	2.3E-09	2.5E-07	3.9E-07	3.9E-07
Bis(2-ethylhexyl) phthalate	(B2 B2 B2)					4.1E-09		7.6E-09	1.2E-08	1.2E-08
Cadmium (solid matrix)	(B1)						5.5E-12		5.5E-12	5.5E-12
Carbon tetrachloride	(B2 B2 B2)	2.9E-07	3.2E-06	3.6E-06	7.1E-06					7.1E-06
Chloroform	(B2 B2 B2)	4.1E-09	3.7E-06	1.3E-07	3.8E-06					3.8E-06
Chrysene	(B2 B2 B2)					1.1E-09	1.9E-11	2.1E-09	3.2E-09	3.2E-09
Dibenz(ah)anthracene	(B2 B2 B2)					5.8E-07	9.8E-09	1.1E-06	1.7E-06	1.7E-06
Indeno(1,2,3-cd)pyrene	(B2 B2 B2)					7.5E-08	1.3E-09	1.4E-07	2.2E-07	2.2E-07
Lead	(B2 B2 B2)									
Methylene chloride	(B2 B2 B2)					1.2E-11	5.2E-14	2.2E-11	3.4E-11	3.4E-11
Trichloroethene	(B2 B2 B2)	7.4E-09	1.5E-07	1.3E-07	2.8E-07					2.8E-07
TOTAL		1.2E-06	7.0E-06	2.5E-04	2.6E-04	1.9E-06	3.3E-08	3.5E-06	5.4E-06	2.6E-04

Area G; Current Lifetime Recreation; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)		MEDIUM								GRAND TOTAL
		FS		Sediment			Surface Water			
		PATHWAY		PATHWAY			PATHWAY			
		Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	
Arsenic	(A A A)			3.6E-11	4.3E-10	4.7E-10				4.7E-10
Dichloroethane, 1,1-	(C C C)									
Methylene chloride	(B2 B2 B2)	9.8E-10	9.8E-10	7.6E-12	9.3E-12	1.7E-11	1.8E-09	2.2E-09	4.1E-09	5.1E-09
Trichloroethene	(B2 B2 B2)	8.5E-09	8.5E-09				2.7E-08	9.0E-09	3.6E-08	4.4E-08
TOTAL		9.5E-09	9.5E-09	4.3E-11	4.4E-10	4.8E-10	2.9E-08	1.1E-08	4.0E-08	5.0E-08

Area G; Future Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)		MEDIUM														GRAND TOTAL
		Ground Water			Sediment			Soil				Surface Water				
		PATHWAY			PATHWAY			PATHWAY				PATHWAY				
		Dermal	Oral	TOTAL	Dermal	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	Dermal	Oral	TOTAL		
Arsenic	(A A A)	1.7E-07	4.7E-05	4.7E-05	7.5E-12	2.1E-10	2.2E-10									4.7E-05
Dichloroethane, 1,1-	(C C C)															
Lead	(B2 B2 B2)															
Methylene chloride	(B2 B2 B2)				1.6E-12	4.5E-12	6.1E-12	8.4E-12	3.6E-14	1.6E-11	2.4E-11	9.7E-11	1.2E-10	2.2E-10		2.5E-10
Trichloroethene	(B2 B2 B2)											1.4E-09	4.9E-10	1.9E-09		1.9E-09
TOTAL		1.7E-07	4.7E-05	4.7E-05	9.1E-12	2.1E-10	2.2E-10	8.4E-12	3.6E-14	1.6E-11	2.4E-11	1.5E-09	6.1E-10	2.1E-09		4.7E-05

Area R; Future Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)		MEDIUM								
		Ground Water				Soil				
		PATHWAY				PATHWAY				
		Dermal	Inhala-tion	Oral	TOTAL	Dermal	Inhala-tion	Oral	TOTAL	GRAND TOTAL
Arsenic	(A A A)	1.3E-07		3.7E-05	3.7E-05					3.7E-05
Benz(a)anthracene	(B2 B2 B2)					9.4E-08	1.6E-09	1.8E-07	2.7E-07	2.7E-07
Benzo(a)pyrene	(B2 B2 B2)					6.8E-07	1.1E-08	1.3E-06	2.0E-06	2.0E-06
Benzo(b)fluoranthene	(B2 B2 B2)					1.2E-07	2.1E-09	2.3E-07	3.6E-07	3.6E-07
Bis(2-chloroethyl) ether	(B2 B2 B2)	1.8E-07		2.4E-05	2.4E-05					2.4E-05
Bis(2-ethylhexyl) phthalate	(B2 B2 B2)	2.5E-08		3.0E-07	3.2E-07	1.0E-09		1.9E-09	2.9E-09	3.3E-07
Butylbenzyl phthalate	(C C C)									
Chloroform	(B2 B2 B2)	1.3E-08	1.1E-05	3.9E-07	1.2E-05					1.2E-05
Chrysene	(B2 B2 B2)					6.8E-10	1.1E-11	1.3E-09	2.0E-09	2.0E-09
Indeno(1,2,3-cd)pyrene	(B2 B2 B2)					4.6E-08	7.7E-10	8.5E-08	1.3E-07	1.3E-07
Lead	(B2 B2 B2)									
Methylene chloride	(B2 B2 B2)	1.2E-09	3.3E-08	7.1E-08	1.1E-07	6.3E-12	2.7E-14	1.2E-11	1.8E-11	1.1E-07
TOTAL		3.5E-07	1.1E-05	6.1E-05	7.3E-05	9.5E-07	1.6E-08	1.8E-06	2.7E-06	7.5E-05

Area ST; Current Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)	MEDIUM				GRAND TOTAL
	Soil				
	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		
Bis(2-ethylhexyl) phthalate (B2 B2 B2)	1.7E-11		3.0E-10	3.1E-10	3.1E-10
DDE, p,p'- (B2 B2 B2)	8.8E-12	8.9E-14	1.6E-10	1.7E-10	1.7E-10
DDT, p,p'- (B2 B2 B2)	5.6E-11	5.7E-13	1.0E-09	1.1E-09	1.1E-09
Methylene chloride (B2 B2 B2)	8.4E-13	1.8E-15	1.5E-11	1.6E-11	1.6E-11
TOTAL	8.2E-11	6.6E-13	1.5E-09	1.6E-09	1.6E-09

Area ST; Future Adult Worker; Chronic

ANALYTE (WOEs: Dermal Oral Inhalation)	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Benzene (A A A)	4.5E-08	1.3E-06	5.9E-07	1.9E-06					1.9E-06
Bis(2-ethylhexyl) phthalate (B2 B2 B2)					2.6E-10		4.8E-10	7.4E-10	7.4E-10
Chloroform (B2 B2 B2)	2.9E-09	2.5E-06	8.8E-08	2.6E-06					2.6E-06
DDE, p,p'- (B2 B2 B2)					1.4E-10	2.8E-12	2.6E-10	4.0E-10	4.0E-10
DDT, p,p'- (B2 B2 B2)					8.8E-10	1.8E-11	1.6E-09	2.5E-09	2.5E-09
Dichloroethane, 1,2- (B2 B2 B2)	1.5E-08	1.7E-06	8.0E-07	2.5E-06					2.5E-06
Lead (B2 B2 B2)									
Methylene chloride (B2 B2 B2)	1.1E-09	3.0E-08	6.6E-08	9.7E-08	1.3E-11	5.7E-14	2.5E-11	3.8E-11	9.7E-08
Tetrachloroethene (B2 B2 B2)	8.0E-08	3.8E-08	4.6E-07	5.8E-07					5.8E-07
Trichloroethene (B2 B2 B2)	1.3E-08	2.6E-07	2.2E-07	4.9E-07					4.9E-07
Vinyl chloride (A A A)	1.5E-06	1.9E-05	5.5E-05	7.6E-05					7.6E-05
TOTAL	1.6E-06	2.5E-05	5.7E-05	8.4E-05	1.3E-09	2.1E-11	2.4E-09	3.7E-09	8.4E-05

Area ST; Current Adult Worker; Chronic

ANALYTE	MEDIUM				GRAND TOTAL
	Soil				
	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		
Acetone	1.6E-07		2.8E-06	3.0E-06	3.0E-06
Bis(2-ethylhexyl) phthalate	1.7E-07		3.0E-06	3.1E-06	3.1E-06
DDE, p,p'-	1.5E-07		2.6E-06	2.7E-06	2.7E-06
DDT, p,p'-	9.3E-07		1.7E-05	1.8E-05	1.8E-05
Methylene chloride	5.2E-09	3.7E-12	9.4E-08	9.9E-08	9.9E-08
TOTAL	1.4E-06	3.7E-12	2.5E-05	2.6E-05	2.6E-05

Area ST; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Acenaphthene	4.8E-04		8.8E-04	1.4E-03					1.4E-03
Acetone	2.0E-05		9.6E-03	9.6E-03	2.4E-06		4.6E-06	7.0E-06	9.6E-03
Anthracene	1.4E-04		1.7E-04	3.1E-04					3.1E-04
Barium	6.1E-05		1.7E-02	1.7E-02					1.7E-02
Benzene	1.4E-02	7.2E-02	1.9E-01	2.8E-01					2.8E-01
Bis(2-ethylhexyl) phthalate					2.6E-06		4.8E-06	7.4E-06	7.4E-06
Cadmium (aqueous matrix)	2.0E-04		5.4E-02	5.5E-02					5.5E-02
Chlorobenzene	2.5E-02	1.3E+00	1.7E-01	1.5E+00					1.5E+00
Chloroform	2.6E-04		8.1E-03	8.4E-03					8.4E-03
Chromium, total	1.7E-04		2.4E-02	2.4E-02					2.4E-02
DDE, p,p'-					2.3E-06		4.2E-06	6.5E-06	6.5E-06
DDT, p,p'-					1.4E-05		2.7E-05	4.1E-05	4.1E-05

(CONTINUED)

Area ST; Future Adult Worker; Chronic

ANALYTE	MEDIUM								GRAND TOTAL
	Ground Water				Soil				
	PATHWAY			TOTAL	PATHWAY			TOTAL	
	Dermal	Inhala-tion	Oral		Dermal	Inhala-tion	Oral		
Dichloroethane, 1,2-	3.2E-05	3.7E-02	8.2E-04	3.8E-02					3.8E-02
Dichloroethenes, 1,2-, total	4.6E-04		1.3E-02	1.3E-02					1.3E-02
Ethylbenzene	6.8E-05	1.9E-04	2.5E-04	5.1E-04					5.1E-04
Fluoranthene	1.7E-03		1.3E-03	2.9E-03					2.9E-03
Fluorene	2.9E-03		2.2E-03	5.2E-03					5.2E-03
Lead									
Mercury	1.5E-05		4.2E-03	4.3E-03					4.3E-03
Methylene chloride	6.7E-06	6.1E-05	4.1E-04	4.8E-04	8.2E-08	1.2E-10	1.5E-07	2.4E-07	4.8E-04
Naphthalene	1.3E-03		5.3E-03	6.6E-03					6.6E-03
Phenanthrene	2.6E-03		2.6E-03	5.2E-03					5.2E-03
Pyrene	2.1E-03		1.7E-03	3.8E-03					3.8E-03
Selenium	1.3E-05		7.2E-03	7.2E-03					7.2E-03
Tetrachloroethene	8.7E-04		2.5E-03	3.4E-03					3.4E-03
Toluene	2.0E-05	1.5E-03	2.5E-04	1.7E-03					1.7E-03
Trichloroethane, 1,1,1-	3.3E-05	3.6E-04	5.3E-04	9.2E-04					9.2E-04
Trichloroethene	1.1E-03		9.2E-03	1.0E-02					1.0E-02
Vinyl chloride	1.7E-03		6.3E-02	6.4E-02					6.4E-02
Xylenes, total	4.3E-06		1.3E-05	1.7E-05					1.7E-05
TOTAL	5.6E-02	1.4E+00	5.9E-01	2.0E+00	2.2E-05	1.2E-10	4.1E-05	6.3E-05	2.0E+00

APPENDIX G

SPECIES LISTS FOR ENDANGERED, THREATENED, AND SPECIAL CONCERN BIOTA AT REDSTONE ARSENAL (NATURAL RESOURCE MANAGEMENT PLAN, JUNE 1992)

Table G-1. Endangered, Threatened, and Special Concern Vertebrates Whose Distributions Include the Project Area (Page 1 of 2)

Scientific Name	Common Name	Alabama Listing	Federal Listing
FISH			
Etheostoma tuscumbia	Tuscumbia darter	T	NL
Hemitremia flammea	Flame Chub	SSC	NL
Typhlichthys subterraneus	Southern cavefish	SSC	NL
Fundulus albolineatus	Whiteline topminnow	SSC	NL
AMPHIBIANS			
Cryptobranchus a. alleganiensis	Hellbender	T	NL
Gyrinophilus palleucus	Tennessee cave salamander	SSC	NL
REPTILES			
Alligator mississippiensis	American Alligator	T	E
Trionyx spiniferus spiniferus	Eastern spiny softshell	SSC	NL
BIRDS			
Aquila chrysaetos	Golden eagle	E	NL
Haliaeetus leucocephalus	Bald eagle	E	E
Pandion haliaetus	Osprey	E	NL
Falco peregrinus anatum	American peregrine falcon	E	T
Falco peregrinus tundrius	Arctic peregrine falcon	E	E
Dendrocopos borealis	Red-cockaded woodpecker	E	E
Egretta caerulea	Little blue heron	SSC	NL
Mycteria americana	Wood stork	SSC	NL
Nycticorax nycticorax	Black-crowned night heron	SSC	NL
Accipiter striatus	Sharp-shinned hawk	SSC	NL
Accipiter cooperi	Cooper's Hawk	SSC	NL
Buteo lineatus	Red-shouldered hawk	SSC	NL
Falco columbarius	Merlin	SSC	NL
Thryomanes bewickii	Bewick's wren	SSC	NL
Limnothlypis swainsonii	Swainsons warbler	SSC	NL
Aimophila aestivalis	Bachman's sparrow	SSC	NL

Table G-1. Endangered, Threatened, and Special Concern Vertebrates Whose Distributions Include the Project Area (Page 2 of 2)

Scientific Name	Common Name	Alabama Listing	Federal Listing
MAMMALS			
Myotis grisescens	Gray bat	E	E
Myotis sodalis	Indiana bat	E	E
Ursus a. americanus	Northern black bear	E	NL
Felis concolor coryi	Florida Panther	E	E
Sorex l. longirostris	Southeastern Shrew	SSC	NL
Myotis a. austroriparius	Southeastern myotis	SSC	NL
Myotis l. lucifugus	Little brown myotis	SSC	NL
Myotis keenii septentrionalis	Keen's myotis	SSC	NL
Plecotus rafinesquii	Rafinesque's big-eared bat	SSC	NL
Microtus o. ochragaster	Prairie vole	SSC	NL

Note: E = Endangered.
T = Threatened.
SSC = Species of Special Concern.
NL = Not listed.

Source: USACE, 1986.

Table G-2. Endangered, Threatened, and Special Concern Invertebrates Whose Distributions Include the Project Area

Scientific Name	Alabama Status	Federal Status
Arthropoda: Crustracea		
Palaemonias alabamae	SSC	NL
Mollusca: Gastropoda		
Marstonia olivacea	PE	NL
Mollusca: Pelecypoda		
Pegias fabula	E, PE	NL
Quadrula c. cylindrica	E	NL
Fusconaia cuneolus	E	E
Fusconaia cor	E	NL
Fusconaia barnesiana	E	NL
Lexingtonia dolabelloides	E	NL
Plethobasus cicatricosus	E, PE	E
Plethobasus cooperianus	E, PE	E
Pleurobema clava	E, PE	NL
Pleurobema oviforme	E	NL
Pleurobema plenum	E	E
Hemistena lata	E	NL
Ptychobranchus subtentum	E	NL
Dromus dromas	E	E
Actinonaias l. ligamentina	E, PE	NL
Actinonaias pectorosa	E	NL
Oboraria subrotunda	E	NL
Oboraria retusa	E, PE	NL
Potamilus laevissimus	E	NL
Toxolasma l. lividus	E	NL
Toxolasma cylindrellus	E	E
Villosa fabalis	E	NL
Villosa t. taeniata	E	NL
Lampsilis orbiculata	SSC	E
Mollusca: Pelecypoda		
Lampsilis ovata	E	NL
Epioblasma triquetra	E	NL
Truncilla truncata	T	NL
Epioblasma brevidens	T	NL
Cumberlandia monodonta	SSC	NL
Plethobasus cyphyus	SSC	NL
Ptychobranchus fasciolaris	SSC	NL

Note: E = Endangered.
T = Threatened.
SSC = Species of Special Concern.
NL = Not listed.

Source: USACE, 1986

Table G-3. Endangered, Threatened, and Special Concern Plants Whose Distributions Include the Project Area

Scientific Name	Common Name	Alabama Listing	Federal Listing
Trillium pusillum	Liliaceae	E	NL
Panax quinquefolius	Araliaceae	E	NL
Nevusia alabamensis	Rosaceae	E	NL
Carex purpurifera	Cyperaceae	T	NL
Trillium erectum var. sulcatum	Liliaceae	T	NL
Leavenworthia torulosa	Brassicaceae	T	NL
Stylophorum diphyllum	Papaveraceae	T	NL
Athyrium pycnocarpon	Aspidiaceae	SSC	NL
Lycopodium flabelliforme	Lycopodiaceae	SSC	NL
Ophioglossum engelmannii	Ophioglossaceae	SSC	NL
Orchis spectabilis	Orchidaceae	SSC	NL
Platanthera peramoena	Orchidaceae	SSC	NL
Continus obovatus	Anacardiaceae	SSC	NL
Jeffersonia diphylla	Berberidaceae	SSC	NL
Gymnocladius dioica	Fabaceae	SSC	NL
Oxalis grandis	Oxalidaceae	SSC	NL
Actaea pachypoda	Ranunculaceae	SSC	NL
Anemone caroliniana	Ranunculaceae	SSC	NL
Veronica anagallis - aquatica	Scrophulariaceae	SSC	NL
Valeriana pauciflora	Valerianaceae	SSC	NL

Note: E = Endangered.
T = Threatened.
SSC = Species of Special Concern.
NL = Not listed.

Source: USACE, 1986

APPENDIX H

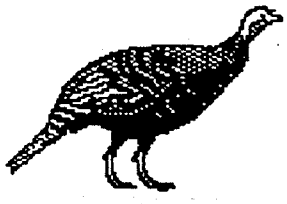
NRMP FLORA AND FAUNA SURVEY

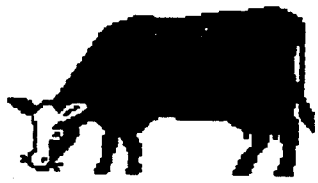
APPENDIX H NOTES

Please note that flora and fauna surveys have not been specifically conducted at Areas F, G, R, and S/T of RSA.

The lists provided in this appendix contain names of species that are potentially on or in the vicinity of RSA and Wheeler National Wildlife Refuge as cited in U.S. Fish and Wildlife Service brochures.

It is probable, due to lack of habitat and other reasons, that many of the organisms on these lists are not found at Areas F, G, R, or S/T of RSA.





NATURAL RESOURCES MANAGEMENT PLAN

FOR

REDSTONE ARSENAL

PARTS I, II, III, IV, V

JUNE 1992

Some Principle Plant Species at Redstone Arsenal, Alabama

Trees and Shrubs

Loblolly Pine - *Pinus taeda*
Shortleaf Pine - *Pinus echinata*
Slash Pine - *Pinus elliottii*
Virginia Pine - *Pinus virginiana*
Eastern Redcedar - *Juniperus virginiana*
Black Walnut - *Juglans nigra*
Shagbark Hickory - *Carya ovata*
Pignut Hickory - *Carya glabra*
Mockernut Hickory - *Carya tomentosa*
Bitternut Hickory - *Carya cordiformis*
Eastern Cottonwood - *Populus deltoides*
Black Willow - *Salix nigra*
Blue Beech - *Carpinus caroliniana*
Ironwood - *Ostrya virginiana*
River Birch - *Betula nigra*
Speckled Alder - *Alnus rugosa*
American Beech - *Fagus grandifolia*
Northern Red Oak - *Quercus rubra*
Scarlet Oak - *Quercus coccinea*
Southern Red Oak - *Quercus falcata*
Cherrybark Oak - *Quercus falcata* var. *pagodaefolia*
Blackjack Oak - *Quercus marilandica*
Water Oak - *Quercus nigra*
Willow Oak - *Quercus phellos*
Nuttal's Oak - *Quercus nuttallii*
Overcup Oak - *Quercus lyrata*
Post Oak - *Quercus stellata*
White Oak - *Quercus alba*
Swamp Chestnut Oak - *Quercus michauxii*
Black Oak - *Quercus velutina*
Chestnut Oak - *Quercus prinus*
Shumard Oak - *Quercus shumardii*
American Elm - *Ulmus americana*
Winged Elm - *Ulmus alata*
Slippery Elm - *Ulmus rubra*
Hackberry - *Celtis occidentalis*
Sugarberry - *Celtis laevigata*
Red Mulberry - *Morus rubra*
Osage Orange - *Maclura pomifera*
Yellow Poplar - *Liriodendron tulipifera*
Pawpaw - *Asimina triloba*
Sassafras - *Sassafras albidum*
Sweet Gum - *Liquidambar styraciflua*
Sycamore - *Platanus occidentalis*

Black Cherry - Prunus serotina
Eastern Redbud - Cercis canadensis
Honey Locust- Gleditsia triacanthos
Black Locust - Robinia pseudoacacia
Silver Maple - Acer saccharinum
Red Maple - Acer rubrum
Box Elder - Acer negundo
Black Gum - Nyssa sylvatica
Water Tupelo - Nyssa aquatica
Flowering Dogwood - Cornus florida
Sourwood - Oxydendrum arboreum
Persimmon - Diospyros virginiana
Green Ash - Fraxinus pennsylvanica
White Ash - Fraxinus americana
Wild Rose - Rosa spp.
Hawthorn - Crataegus spp.
Wild Plum - Prunus spp.
Smooth Sumac - Rhus glabra
Winged Sumac - Rhus copallina
Possumhaw - Ilex decidua
Winterberry - Ilex laevigata
Strawberry Bush - Euonymus americanus
Red Buckeye - Aesculus pavia
Devil's Walking Stick - Aralia spinosa
Wild Azalea - Rhododendron nudiflorum
Mountain Laurel - Kalmia latifolia
Huckleberry - Vaccinium spp.
Privet - Ligustrum sp.
French Mullberry, Beauty Berry - Callicarpa americana
Buttonbush - Cephalanthus occidentalis
Elderberry - Sambucus canadensis
Haw - Viburnum spp.
Fringetree, Graybeard - Chionanthus virginica

Vines

Greenbrier - Smilax spp.
Kudzu - Pueraria lobata
Poison Ivy - Rhus radicans
Rattan Vine - Berchemia scandens
Wild Grape - Vitis spp.
Virginia Creeper - Parthenocissus quinquefolia
Crossvine - Anisostichus capreolata
Trumpet Creeper - Campsis radicans
Blackberry - Rubus argutus
Dewberry - Rubus trivialis
Japanese Honeysuckle - Lonicera japonica
Passion Flower, Maypops - Passiflora incarnata
Morning Glory - Ipomoea spp.

Forbs

Pokeweed - *Phytolacca americana*
Ragweed - *Ambrosia artemisiifolia*
Gaint Ragweed - *Ambrosia trifida*
Stick Tights, Spanish needles - *Bidens* sp.
Goldenrod - *Solidago* spp.
Beggarweed - *Desmodium* spp.
Partridge Pea - *Cassia* spp.
Sericea Lespedeza - *Lespedeza cuneata*
Common Lespedeza - *Lespedeza striata*
Native Lespedezas - *Lespedeza* spp.
Sicklepod - *Cassia obtusifolia*
Vetch - *Vicia* spp.
Bitterweed - *Helenium amarum*
Cocklebur- *Xanthium strumarium*
PoorJoe - *Diodia* sp.
Thoroughwort - *Eupatorium* sp.
Horseweed - *Erigeron canadensis*
Fireweed - *Erechtites hieracifolia*

Grasses

Broomsedge - *Andropogon* spp.
Cane - *Arundinaria gigantea*
Barnyard Grass, Wild Millet - *Echinochloa crusgalli*
Foxtail Grass - *Setaria* sp.
Paspalum Grass - *Paspalum* spp.
Johnson Grass - *Sorghum halepense*
Bermuda Grass - *Cynodon dactylon*
Crab Grass - *Digitaria* spp.
Panic Grass - *Panicum* spp.
Fescue - *Festuca* sp.
Plume Grass - *Erianthus* sp.

Aquatic and Marsh Plants

Smartweed - *Polygonum* spp.
American Lotus - *Nelumbo lutea*
Cattail - *Typha* sp.
Duckweed- *Lemna* sp. and *spirodela* sp.
Coontail - *Ceratophyllum* sp.
Jewel-Weed - *Impatiens capensis*
Parrot Feather - *Myriophyllum brasiliense*
Water Primrose - *Ludwigia* sp.
Sedge - *Carex* spp.
Umbrella Sedge - *Cyperus* spp.
Lizard's Tail - *Saururus* spp.
Bulrush - *Scirpus* spp.
Rush - *Juncus* spp.

Plants Used in Food Plots

Bicolor Lespedeza - *Lespedeza bicolor*
Wheat - *Triticum* sp.
Crimson Clover - *Trifolium incarnatum*
Brown Top Millet - *Panicum ramosum*
Japanese Millet - *Echinochloa crusgalli* var. *frumentacea*

FISH AND WILDLIFE RESOURCES

Mammal Species Inventory

This list is taken from the U.S Fish and Wildlife Service brochure, Mammals of Wheeler National Wildlife Refuge.

Shorttail Shrew - Blarina brevicauda - common
Least Shrew - Cryptotis parva - uncommon
Eastern Mole - Scalopus aquaticus - common
Little Brown Bat (Brown Myotis) Myotis lucifugus - fairly common throughout warm weather
Gray Bat - Myotis grisescens - endangered species, but common locally
Keens Bat or Myotis - Myotis keeni - occasional during warm weather
Indiana Bat - Myotis sodalis - endangered species but common locally
Silver-haired Bat - Lasionycteris noctivagans - uncommon
Eastern Pipistrel - Pipistrellus subflavus - fairly common
Red Bat - Lasiurus borealis - common
Seminole Bat - Lasiurus seminolus - occasional in warm weather
Hoary Bat - Lasiurus cinereus - hibernates in local caves
Big Brown Bat - Eptesicus fuscus - fairly common
Evening Bat- Nycticeius humeralis - fairly common
Big-Eared Bat - Plecotus rafinesquei - occasional in warm weather
Opossum - Didelphis marsupialis - common
Eastern Cottontail - Sylvilagus floridanus - common on uplands
Swamp Rabbit - Sylvilagus aquaticus - fairly common in low woodland
Eastern Gray Squirrel - Sciurus carolinensis - common
Eastern Fox Squirrel - Sciurus niger - fairly common
Southern Flying Squirrel - Glaucomys volans - fairly common
Eastern Chipmunk - Tamias striatus - common
Groundhog - Marmota monax - common
Beaver - Castor canadensis - common along all streams
Muskrat - Ondatra zibethica - common around water
Eastern Harvest Mouse - Reithrodontomys humulis - common
White-footed Mouse - Peromyscus leucopus - common
Cotton Mouse - Peromyscus gossypinus - fairly common
Golden Mouse - Ochrotomys nuttalli - fairly common
Rice Rat - Orzomys palustris - fairly common
Cotton Rat - Sigmodon hispidus - common
Eastern Woodrat - Neotoma floridana - fairly common
Prarie Vole - Microtus ochrogaster
Pine Vole - Pitymys pinetorum
Norway Rat - Rattus norvegicus - introduced, common near buildings
Housemouse - Mus musculus - introduced, common near buildings
Coyote - Canis latrans - common
Red Fox - Vulpes fulva - common
Gray Fox - Urocyon cinereoargenteus - common in woodland
Racoon - Procyon lotor - common
Longtail Weasel - Mustela frenata - rare
Mink - Mustela vison - fairly common around water
Spotted Skunk or Civet Cat - Spilogale putorius - rare
Striped Skunk - Mephitis mephitis - common
River Otter - Lutra canadensis - uncommon
Bobcat - Lynx rufus - uncommon
White-tailed Deer - Odocoileus virginianus - common

Bird Species Inventory

This list is taken from U.S Fish and Wildlife Service brochure, Birds of Wheeler National Wildlife Refuge.

SEASON

SP	- Spring.	March-May
S	- Summer.	June-August
F	- Fall	September-November
W	- Winter.	December-February

STATUS

a	- abundant	
c	- common	certain to be seen in suitable habitat.
u	- uncommon	present, but not always found.
o	- occasional	occurs erratically.
r	- rare	seen at intervals of 2 to 5 years.
x	- accidental	has been seen fewer than 5 times.
*	-	nest locally.

	SP	S	F	W
BLUEBIRDS and THRUSHES				
Eastern Bluebird*	c	c	c	u
Veery	u		u	
Grey-checked Thrush	u		u	
Swainson's Thrush (Olive-backed Thrush)	u		u	
Hermit Thrush	u		c	c
Wood Thrush*	c	c	c	
American Robin*	c	c	c	u
MIMICS				
Grey Catbird*	c	c	c	
Northern Mockingbird*	c	c	c	c
Brown Thrasher*	c	c	c	c
PIPITS				
Water Pipit	r		c	c
Sprague's Pipit				x
WAXWINGS, SHRIKES and STARLINGS				
Cedar Waxwing	c	r	c	c
Loggerhead Shrike*	c	c	c	c
European Starling*	a	a	a	a
VIREOS				
White-eyed Vireo*	c	c	c	
Yellow-throated Vireo*	c	c	c	
Warbling Vireo	o		o	
Philadelphia Vireo	o		u	
Red-eyed Vireo*	c	c	c	
Solitary Vireo	u		u	
WOOD WARBLERS				
Blue-winged Warbler	u		u	
Golden-winged Warbler	u		u	
Tennessee Warbler	c		c	
Orange-crowned Warbler	u		u	o
Nashville Warbler	u		c	
Northern Parula	c	u	c	
Yellow Warbler*	c	u	c	r
Chestnut-sided Warbler	u		u	
Magnolia Warbler	c		c	
Cape May Warbler	u		u	
Black-throated Blue Warbler	x			
Yellow-rumped Warbler (Myrtle Warbler)*	c		c	c
Black-throated Green Warbler	u		u	
Blackburnian Warbler	u		u	
Yellow-throated Warbler	c	r	c	
Pine Warbler*	c	c	c	c
Prairie Warbler	c	u	c	
Palm Warbler	c		c	u
Bay-breasted Warbler	u		u	
Blackpoll Warbler	c		r	
Cerulean Warbler	u		u	
Black-and-white Warbler*	c	c	c	
American Redstart*	c	u	c	
Prothonotary Warbler*	c	c	c	
Worm-eating Warbler	u	u	u	
Swainson's Warbler	u		u	
Ovenbird	c		c	
Northern Waterthrush	u		u	
Louisiana Waterthrush*	u	u	u	
Kentucky Warbler	c		c	
Connecticut Warbler	o		o	
Mourning Warbler	r		r	
Common Yellowthroat*	c	c	c	o
Hooded Warbler*	c	c	c	
Wilson's Warbler	u		o	
Canada Warbler	u		u	
Yellow-breasted Chat*	c	c	c	
TANAGERS				
Summer Tanager*	c	c	u	
Scarlet Tanager	u		u	
CARDINALS, GROSBEAKS and BUNTING				
Northern Cardinal*	c	c	c	c
Rose-breasted Grosbeak	c		c	
Blue Grosbeak*	c	c	c	
Indigo Bunting*	c	c	c	
Dickcissel*	c	c	u	
SPARROWS				
Rufous-sided Towhee*	c	c	c	c
Bachman's Sparrow	r	r		r
American Tree Sparrow (Townsend's Sparrow)	o		o	o
Chipping Sparrow	u	u	u	u
Clay-colored Sparrow	x			
Field Sparrow*	c	c	c	c
Vesper Sparrow	o		o	c
Lark Sparrow	r	r	r	
Savannah Sparrow	u		c	c
Grasshopper Sparrow*	u	u	u	r
Henslow's Sparrow	r		r	r
Le Conte's Sparrow	r		r	r
Sharp-tailed Sparrow (Nelson's Sparrow)	x			
Fox Sparrow	u		u	u
Song Sparrow	u		c	c
Lincoln's Sparrow	o		o	
Swamp Sparrow	c		c	c
White-throated Sparrow	c		c	a
White-crowned Sparrow			u	u
Harris' Sparrow			x	
Dark-eyed Junco (Slate-colored Junco)	c	o	c	c
Lapland Longspur			u	u
BLACKBIRDS, MEADOWLARKS and ORIOLES				
Bobolink	c		u	
Red-winged Blackbird*	c	c	a	a
Eastern Meadowlark*	c	c	a	c
Western Meadowlark			x	x
Yellow-headed Blackbird	x		x	x
Rusty Blackbird	u		c	c
Brewer's Blackbird	o		u	u
Common Grackle*	c	c	a	a
Brown-headed Cowbird*	c	c	a	a
Orchard Oriole*	c	c		
Northern Oriole (Baltimore Oriole)	u	o	u	
FINCHES				
Purple Finch	u		c	c
House Finch	u		u	u
Red Crossbill				x
Pine Siskin			u	u
American Goldfinch*	c	u	c	c
Evening Grosbeak	u		u	u
WEAVER FINCHES				
House Sparrow*	c	c	c	c

	SP	S	F	W
OONS AND GREBES				
Red-throated Loon				x
Common Loon	c	r	c	c
Pied-billed Grebe*	c	u	c	c
Horned Grebe	u		c	c
Red-necked Grebe			o	o
Eared Grebe	o		o	
PELICANS AND CORMORANTS				
American White Pelican			o	
Double-crested Cormorant	u		u	u
ANHINGA				
Anhinga	r	r	r	
BITTERNS, EGRETS and HERONS				
American Bittern	u		u	
Least Bittern	u	u		
Great Blue Heron	c	c	a	a
Great Egret	u	u	u	r
Snowy Egret	u	u		
Little Blue Heron	c	c	u	
Tricolored Heron (Louisiana Heron)	o	o		
Cattle Egret	c	c	u	
Green-backed Heron (Green Heron)	c	c		
Black-crowned Night-Heron	u	u	u	r
Yellow-crowned Night-Heron*	c	c	u	
IBISES and STORKS				
White Ibis	o	o	o	
Glossy Ibis	r	r	r	
Wood Stork (Wood Ibis)		r	r	
ATERFOWL, DUCKS, EESE and SWANS				
Fulvous Whistling-Duck (Tree Duck)				x
Tundra Swan (Whistling Duck)			o	o
Mute Swan		x	x	
Greater White-fronted Goose	o		a	o
Snow Goose	u		c	c
Ross' Goose				x
Brant				x
Barnacle Goose			x	x
Canada Goose*	u	u	a	a
Egyptian Goose	x		x	
Wood Duck*	c	c	a	c
Green-winged Teal	u		c	c
American Black Duck*	u	u	c	c
Mallard*	u	u	a	a
Northern Pintail	u	r	c	c
Blue-winged Teal	c	r	c	c
Cinnamon Teal				x
Northern Shoveler	c		c	u
Gadwall	u	r	c	c
Eurasian Wigeon			x	x
American Wigeon	u		a	a
Canvasback	c		c	c
Redhead	u		u	u
Ring-necked Duck	c		c	c
Greater Scaup			o	o
Lesser Scaup	c		c	c
Common Eider				x
King Eider				x
Harlequin Duck				x
Oldsquaw	r		u	u
Black Scoter (Common Scoter)			r	r
Surf Scoter				r
White-winged Scoter				u
Common Goldeneye	r			c
Bufflehead	r		r	c
Hooded Merganser*	u	r	c	c
Common Merganser (American Merganser)	r		u	u
Red-breasted Merganser	c		u	u
Ruddy Duck	u		c	c
Masked Duck	x			
Ruddy Shelduck			x	x
VULTURES				
Black Vulture	u	u	u	r
Turkey Vulture	u	u	u	r
OSPREYS, KITES, EAGLES and HAWKS				
Osprey	u		u	
Mississippi Kite	o	o		
Bald Eagle	r		u	u
Northern Harrier (Marsh Hawk)	u		c	c
Sharp-shinned Hawk*	u	u	c	u
Cooper's Hawk*	u	u	c	u
Red-shouldered Hawk*	u	u	u	u
Broad-winged Hawk	u		c	
Swainson's Hawk			x	x
Red-tailed Hawk*	u	u	c	c
Rough-legged Hawk			o	o
Golden Eagle			o	o
American Kestrel (Sparrow Hawk)*	c	c	c	c
Merlin (Pigeon Hawk)	u	r	u	u
Peregrine Falcon (Duck Hawk)	r		r	r
GALLINACEOUS BIRDS				
Ring-necked Pheasant	r	r	r	r
Wild Turkey*	u	u	u	u
Northern Bobwhite (Quail)*	c	c	c	c
RAILS, COOTS and GALLINULES				
Yellow Rail				x
Black Rail		x		
King Rail	c	c	c	u
Virginia Rail	c		c	
Sora	c		c	
Purple Gallinule	o	o		
Common Moorhen (Common Gallinule)	u	u	u	
American Coot*	a	u	a	c
CRANES				
Sandhill Crane			x	
PLOVERS				
Black-bellied Plover	u		u	
Greater Golden Plover	u		u	
Wilson's Plover	x			
Semipalmated Plover	c		c	
Piping Plover	u		u	
Killdeer*	c	c	a	c
STILTS and AVOCETS				
Black-necked Stilt	x		x	
American Avocet			u	

	SP	S	F	W
SHOREBIRDS				
Greater Yellowlegs	a	u	a	u
Lesser Yellowlegs	a	u	a	u
Solitary Sandpiper	c	u	c	
Willet	u	u	u	
Spotted Sandpiper*	c	c	c	r
Upland Sandpiper (Upland Plover)	u		u	
Whimbrel (Hudsonian Curlew)	r		r	
Hudsonian Godwit	r		r	
Marbled Godwit	r			
Ruddy Turnstone	u		u	
Red Knot			r	
Sanderling	u		c	
Semipalmated Sandpiper	c	u	c	r
Western Sandpiper	u		c	
Least Sandpiper	a	u	a	u
White-rumped Sandpiper	u		c	
Baird's Sandpiper	o	r	o	
Pectoral Sandpiper	a	u	a	r
Dunlin (Red-backed Sandpiper)	u		c	u
Stilt Sandpiper	u		u	
Buff-breasted Sandpiper	o		u	
Short-billed Dowitcher	u		u	
Long-billed Dowitcher	c	r	u	
Common Snipe	c	u	c	c
American Woodcock	u	u	c	c
Wilson's Phalarope	u	r	u	
Red-necked Phalarope (Northern Phalarope)	r		o	
GULLS and TERNS				
Laughing Gull	u		o	u
Franklin's Gull	r	r	r	r
Bonaparte's Gull	u		u	u
Ring-billed Gull	u	r	a	a
Herring Gull	u	r	c	c
Glaucous Gull				r
Great Black-backed Gull				r
Caspian Tern	u	u	u	
Common Tern	o	o	o	
Forster's Tern	u	u	u	
Least Tern	o	o		
Black Tern	u	u	c	
PIGEONS and DOVES				
Rock Dove (Domestic Pigeon)	c	c	c	c
White-winged Dove			x	
Mourning Dove*	c	c	a	c
Common Ground Dove	o	o	o	
CUCKOOS				
Black-billed Cuckoo	u		u	
Yellow-billed Cuckoo*	c	c	c	
OWLS				
Common Barn Owl*	c	c	c	c
Eastern Screech-Owl*	c	c	c	c
Great Horned Owl*	u	u	c	c
Barred Owl*	u	u	u	u
Short-eared Owl			u	u
Northern Saw-whet Owl				x
GOATSUCKERS				
Common Nighthawk*	c	c	c	
Chuck-will's widow*	c	c	c	
Whip-poor-will	u		u	
SWIFTS				
Chimney Swift*	c	c	c	

	SP	S	F	W
HUMMINGBIRDS				
Ruby-throated Hummingbird*	c	c	c	
KINGFISHERS				
Belted Kingfisher*	c	c	c	c
WOODPECKERS				
Red-headed Woodpecker*	c	c	c	u
Red-bellied Woodpecker*	c	c	c	c
Yellow-bellied Sapsucker	c		c	c
Downy Woodpecker*	c	c	c	c
Hairy Woodpecker*	c	c	c	c
Northern Flicker (Common Flicker)*	c	c	c	c
Pileated Woodpecker*	c	c	c	c
FLYCATCHERS				
Olive-sided Flycatcher	r		r	
Eastern Wood-Pewee*	c	c	c	
Yellow-bellied Flycatcher			o	
Acadian Flycatcher*	c	c	c	
Least Flycatcher	u		u	
Ash-throated Flycatcher			x	
Great Crested Flycatcher*	c	c	c	
Eastern Kingbird*	c	c	c	
Scissor-tailed Flycatcher			x	
LARKS				
Horned Lark*	c	o	c	c
MARTINS and SWALLOWS				
Purple Martin*	c	c	c	
Tree Swallow	c		c	
Northern Rough-winged Swallow*	c	c	c	
Bank Swallow	c		c	
Cliff Swallow*	c	c	c	
Barn Swallow*	c	c	c	
BLUE JAYS and CROWS				
Blue Jay*	c	c	c	c
American Crow (Common Crow)*	c	u	a	a
CHICKADEES and TITMICE				
Carolina Chickadee*	c	c	c	c
Tufted Titmouse*	c	c	c	c
NUTHATCHES				
Red-breasted Nuthatch	u		u	c
White-breasted Nuthatch*	u	u	u	u
Brown-headed Nuthatch	r		r	
CREEPERS				
Brown Creeper			c	c
WRENS				
Carolina Wren*	c	c	c	c
Bewick's Wren*	u	u	r	r
House Wren			u	u
Winter Wren	u		c	c
Sedge Wren (Short-billed Marsh Wren)	r		r	
Marsh Wren (Long-billed Marsh Wren)	o		u	r
KINGLETS and GNATCATCHERS				
Golden-crowned Kinglet	u		c	c
Ruby-crowned Kinglet	c		c	c
Blue-grey Gnatcatcher*	c	u	c	

Reptile and Amphibian Species Inventory

This list is taken from the U.S. Fish and Wildlife Service brochure on amphibians and reptiles of Wheeler National Wildlife Refuge by Drs. Dan W. Speake and Robert Mount.

Amphibians

Spadefoot Toad - Scaphiopus holbrooki holbrooki - Common
American Toad - Bufo americanus - Common
Fowlers Toad - Bufo woodhousei fowleri - Common
Northern Cricket Frog - Acris crepitans crepitans - Common
Northern Spring Peeper - Hyla crucifer crucifer - Common
Eastern Gray Treefrog - Hyla versicolor versicolor - Common
Upland Chorus Frog - Pseudacris triseriata feriarum - Common
Mountain Chorus Frog - Pseudacris brachyphona - Common
Eastern Narrow - mouthed Toad - Gastrophryne carolinensis - Common
Bullfrog - Rana catesbeiana - Uncommon
Southern Leopard Frog - Rana utricularia - Fairly Common
Green Frog - Rana clamitans melanota - Common
Hellbender - Cryptobranchus alleganiensis alleganiensis-Rare
Mudpuppy - Necturus maculosus maculosus - Common
Red-spotted Newt - Notophthalmus viridescens viridescens - Fairly Common
Spotted Salamander- Ambystoma maculatum - Common
Marbled Salamander - Ambystoma opacum - Common
Small-mouthed Salamander - Ambystoma texanum - Rare
Eastern Tiger Salamander - Ambystoma tigrinum tigrinum - Fairly Common
Dusky Salamander - Desmognathus fuscus fuscus - Common
Zigzag Salamander - Plethodon dorsalis dorsalis - Fairly Common
Slimy Salamander - Plethodon glutinosus glutinosus - Fairly Common
Northern Red Salamander - Pseudotriton ruber ruber - Fairly Common
Long-tailed Salamander - Eurycea longicauda longicauda - Fairly Common
Two-lined Salamander - Eurycea bislineata ssp. - Fairly Common
Cave Salamander - Eurycea lucifuga - Uncommon-occurs in local caves

Reptiles

Common Snapping Turtle - Chelydra serpentina serpentina - Common
Common Musk Turtle - Sternotherus odoratus - Common
Eastern Mud Turtle - Kinosternon subrubrum subrubrum - Common
Eastern Box Turtle - Terrapene carolina carolina - Common
Map Turtle - Graptemys geographica - Common
Ouachita Map Turtle - Graptemys pseudogeographica ouachitenis - Common
Southern Painted Turtle - Chrysemys picta dorsalis - Common
Midland Painted Turtle - Chrysemys picta marginata - Common
Cumberland Turtle - Chrysemys scripta troosti - Common
Red-eared Turtle - Chrysemys scripta elegans - Common
River Cooter - Chrysemys concinna concinna - Common
Midland Smooth Softshell Turtle - Trionyx muticus muticus - Uncommon

Eastern Spiny Softshell Turtle - Trionyx spinifer spinifer - Uncommon
Green Anole - Anolis carolinensis carolinensis - Rare
Northern Fence Lizard - Sceloporus undulatus hyacinthimus - Fairly Common
Six-lined Race Runner - Cnemidophorus sexlineatus sexlineatus - Uncommon
Slender Glass Lizard - Ophisaurus attenuatus longicaudus - Fairly Common
Ground Skink - Scincella lateralis - Common
Five-lined Skink - Eumeces fasciatus - Common
Southeastern Five-lined Skink - Eumeces inexpectatus - Uncommon
Broad-headed Skink - Eumeces laticeps - Common
Eastern Smooth Earth Snake - Virginia valeriae valeriae - Fairly common though seldom seen
Rough Earth Snake - Virginia striatula
Northern Red-bellied Snake - Storeria occipitomaculata occipitomaculata - Common
Midland Brown Snake - Storeria dekayi wrightorum - Common
Yellow-bellied Water Snake - Nerodia erythrogaster flavigaster - Common
Midland Water Snake - Nerodia sipedon pleuralis - Common
Queen Snake - Natrix septemvittata - Common
Eastern Garter Snake - Thamnophis sirtalis sirtalis - Common
Eastern Ribbon Snake - Thamnophis sauritus sauritus - Common
Western Mud Snake - Farancia abacura reinwardti - Uncommon
Hognose Snake - Heterodon platyrhinos - Common
Northern Ringneck Snake - Diadophis punctatus edwardsi - Common
Mississippi Ringneck Snake - Diadophis punctatus stictogenys - Fairly Common
Eastern Worm Snake - Carphophis amoenus amoenus - Common though seldom seen
Northern Black Racer - Coluber constrictor constrictor - Common
Eastern Coachwhip - Masticophis flagellum flagellum - Uncommon
Rough Green Snake - Opheodrys aestivus - Fairly Common
Gray Rat Snake - Elaphe obsoleta spiloides - Uncommon
Black Rat Snake - Elaphe obsoleta obsoleta - Common
Corn Snake - Elaphe guttata guttata - Common
Scarlet Snake - Cemophora coccinea - Rare
Scarlet Kingsnake - Lampropeltis triangulum elapsoidese - Rare
Eastern Milk Snake - Lampropeltis triangulum triangulum - Uncommon
Mole Snake - Lampropeltis calligaster rhombomaculata - Rare
Black Kingsnake - Lampropeltis getulus niger - Fairly Common
Crowned Snake - Tantilla coronata - Fairly Common
Northern Copperhead - Agkistrodon contortrix mokasen - Uncommon
Eastern Cottonmouth - Agkistrodon piscivorus piscivorus - Uncommon
Canebreak Rattlesnake - Crotalus horridus atricaudatus - Rare
Carolina Pigmy Rattlesnake - Sistrurus miliarius miliarius - Rare
American Alligator - Alligator mississippiensis
Timber Rattlesnake - Cratalus horridus horridus - Rare

Fish Species Inventory

This list is summarized from Fishes of the Wheeler National Wildlife Refuge by William C. Reeves. It includes species found in the Tennessee River.

Status:

The status, with reference to relative abundance, of the fish species known to occur in the waters of Wheeler National Wildlife Refuge is presented in the following five major categories:

Abundant - occurring in large numbers in many locations.

Common - widely distributed, may be quite numerous at certain times of the year or at particular locations.

Uncommon - observed only occasionally, because of restricted habitat or small numbers present.

Rare - present in very small numbers or, where abundant, found in very restricted habitat in only a few locations.

Undetermined - status unknown, these species are expected to occur in the waters of Wheeler Refuge, but their presence and/or numbers have not been validated. Many of the species were placed in the category because of a lack of basic information on them within the refuge boundaries.

Ohio lamprey - *Ichthyomyzon bdellium* - rare
Chestnut lamprey - *Ichthyomyzon castaneus* - common
Least brook lamprey - *Lampetra aepyptera* - rare
Paddlefish, spoonbill cat - *Polyodon spathula* - uncommon
Spotted gar - *Lepisosteus oculatus* - common
Longnose gar - *Lepisosteus osseus* - common
Shortnose gar - *Lepisosteus platostomus* - undetermined
Bowfin - *Amia calva* - uncommon
American eel - *Anguilla rostrata* - uncommon
Skipjack herring - *Alosa chrysochloris* - abundant
Gizzard shad - *Dorosoma cepedianum* - abundant
Threadfin shad - *Dorosoma petenense* - abundant
Goldeye - *Hiodon alosoides* - rare
Mooneye - *Hiodon tergisus* - uncommon
Redfin pickerel - *Esox americanus* - uncommon
Chain pickerel - *Esox niger* - common
Grass pickerel - *Esox vermiculatus*
Stoneroller - *Campostoma anomalum* - common
Goldfish - *Carassius auratus* - uncommon
Rosyside dace - *Clinostomus funduloides* - undetermined
Common carp - *Cyprinus carpio* - abundant
Flame chub - *Hemitrema flammea* - rare
Cypress minnow - *Hybognathus hayi* - undetermined

Silvery Minnow - Hybognathus nuchalis - undetermined
Bigeye Chub - Hybopsis amblops - undetermined
Blotched Chub - Hybopsis insignis - undetermined
Silver Chub - Hybopsis storeriana - undetermined
River Chub - Nocomis micropogon - undetermined
Golden Shiner - Notemigonus crysoleucas - common
Rosefin Shiner - Notropis ardens - undetermined
Emerald Shiner - Notropis atherinoides - common
Ghost Shiner - Notropis buchanani - uncommon
Striped Shiner - Notropis chrysocephalus - common
Pugnose Minnow - Opsopoeodus emiliae - undetermined
Ribbon Shiner - Notropis fumeus - undetermined
Whitetail Shiner - Notropis galacturus - undetermined
Tennessee Shiner - Notropis leuciodus - undetermined
Mountain Shiner - Notropis lirus - undetermined
Rosyface Shiner - Notropis rubellus micropteryx - undetermined
Spotfin Shiner - Notropis spilopterus - undetermined
Telescope Shiner - Notropis telescopus - undetermined
Mimic Shiner - Notropis volucellus - undetermined
Steelcolor Shiner - Notropis whipplei - common
Bluntnose Minnow - Pimephales notatus - common
Fathead Minnow - Pimephales promelas - uncommon
Bullhead Minnow - Pimephales vigilax - common
Blacknose Dace - Rhinichthys atratulus - uncommon
Creek Chub - Semotilus atromaculatus - common
River Carpsucker - Carpiodes carpio - uncommon
Quillback - Carpiodes cyprinus - uncommon
Highfin Carpsucker - Carpiodes velifer - uncommon
White Sucker - Catostomus commersoni - uncommon
Creek Chubsucker - Erimyzon oblongus - undetermined
Northern Hogsucker - Hypentelium nigricans - undetermined
Smallmouth Buffalo - Ictiobus bubalus - common
Bigmouth Buffalo - Ictiobus cyprinellus - uncommon
Black Buffalo - Ictiobus niger - uncommon
Spotted Sucker - Minytrema melanops - abundant
Silver Redhorse - Moxostoma anisurum - common
River Redhorse - Moxostoma carinatum - uncommon
Black Redhorse - Moxostoma duquesnii - common
Golden Redhorse - Moxostoma erythrurum - common
Shorthead Redhorse - Moxostoma breviceps - common
Blue Catfish - Ictalurus furcatus - common
Black Bullhead - Ictalurus melas - common
Yellow Bullhead - Ictalurus natalis - uncommon
Brown Bullhead - Ictalurus marmoratus - uncommon
Channel Catfish - Ictalurus punctatus - common
Slender Madtom - Noturus exilis - undetermined
Flathead Catfish - Pylodictis olivaris - common
Tadpole Madtom - Noturus gyrinus
Southern Cavefish - Typhlichthys subterraneus - undetermined
Pirate Perch - Aphredoderus sayanus - undetermined
Blackspotted Topminnow - Fundulus olivaceus - common

Blackstripe Topminnow - Fundulus notatus
Mosquitofish - Gambusia affinis - common
Brook Silverside - Labidesthes sicculus - common
White Bass - Morone chrysops - common
Yellow Bass - Morone mississippiensis - common
Striped Bass - Morone saxatilis - undetermined
Rock Bass - Ambloplites rupestris - uncommon
Green Sunfish - Lepomis cyanellus - abundant
Warmouth - Chaenobryttus gulosus - common
Orangespotted Sunfish - Lepomis humilis - common
Bluegill - Lepomis macrochirus - abundant
Longear Sunfish - Lepomis megalotis - common
Redear Sunfish - Lepomis microlophus - common
Smallmouth Bass - Micropterus dolomieui - uncommon
Spotted Bass - Micropterus punctulatus - common
Largemouth Bass - Micropterus salmoides - common
White Crappie - Pomoxis annularis - common
Black Crappie - Pomoxis nigromaculatus - uncommon
Redbreast Sunfish - Lepomis auritus
Greenside Darter - Etheostoma blennioides - common
Blenny Darter - Etheostoma blennius - undetermined
Rainbow Darter - Etheostoma caeruleum - undetermined
Blackside Snubnose Darter - Etheostoma duryi - uncommon
Fantail Darter - Etheostoma flabellare - undetermined
Blueside Darter - Etheostoma jessiae - undetermined
Stripetail Darter - Etheostoma kennicotti - undetermined
Johnny Darter - Etheostoma nigrum - undetermined
Redline Darter - Etheostoma rufilineatum - undetermined
Tennessee Snubnose Darter - Etheostoma simoterum - undetermined
Spottail Darter - Etheostoma squamiceps - undetermined
Speckled Darter - Etheostoma stigmaeum - undetermined
Tuscumbia Darter - Etheostoma tuscumbia - rare
Banded Darter - Etheostoma zonale - undetermined
Logperch - Percina caprodes - common
Blackside Darter - Percina maculata - undetermined
Dusky Darter - Percina sciera - undetermined
River Darter - Percina shumardi - undetermined
Stargazing Darter - Percina uranidea - undetermined
Sauger - Stizostedion canadense - common
Walleye - Stizostedion vitreum - uncommon
Yellow Perch - Perea flavescens
Freshwater Drum - Aplodinotus grunniens - abundant
Banded Sculpin - Cottus carolinae - uncommon

Threatened and Endangered Species

These species are federally listed as threatened or endangered as of September 1987*, and are potentially on or in the vicinity of Redstone Arsenal.

Mammals

Gray Bat - *Myotis grisescens* - endangered
Indiana Bat - *Myotis sodalis* - endangered
Florida Panther - *Felis concolor coryi* - endangered

Birds

Bald Eagle - *Haliaeetus leucocephalus* - endangered
American Peregrine Falcon - *Falco peregrinus anatum* - endangered
Arctic Peregrine Falcon - *Falco peregrinus tundrius* - threatened
Bachman's Warbler - *Vermivora bachmanii* - endangered
Red-cockaded Woodpecker - *Dendrocopos borealis* - endangered

Reptiles

American Alligator - *Alligator mississippiensis* - threatened due to similarity of appearance

Fish

Slackwater darter - *Etheostoma boschungi* - threatened
Snail darter - *Percina tanasi* - threatened

Mollusks

Orange-footed Pearly Mussel - *Plethobasus cooperianus* - endangered
Pink Mucket Pearly Mussel - *Lampsilis orbiculata obiculata* - endangered
Rough Pigtoe Pearly Mussel - *Pleurobema plenum* - endangered

Crustaceans

Alabama Cave Shrimp - *Palaemonias alabamae* - endangered

* The Alabama Cave Shrimp was first sighted in a cave on Redstone Arsenal in 1974. Since the species was federally listed in October 1988, a contract has been established with the U.S. Fish and Wildlife Service to determine the geological aspects of the species habitat and make recommendations for actions to improve species habitat and encourage species population increase. Presently, all activities near the cave site are being closely monitored and will be curtailed or halted should a determination be made by the U.S. Fish and Wildlife Service that critical habitat requires such.

Environmental
Science &
Engineering, Inc.
A CILCORP Company

December 15, 1995

Commander
U.S. Army Missile Command
AMSMI-EQ (Hewitt)
Building 112
Environmental Management Planning
Redstone Arsenal, AL 35898-5349

Dear Mr. Hewitt:

Re: Replacement Pages for Baseline Risk Assessment for Group 1 Sites Addressing EPA Comments Dated August 21, 1995; Delivery Order No. 0010; ESE No. 3942025G-0300-3100-CM-95-12-12-1

Enclosed are the replacement pages for the Final BRA1 for you to review. To expedite your review, we have included the previous text from the Draft Final BRA1 marked with strikeout, along with the revised text. These pages address the two specific EPA comments (dated August 21, 1995 from RPM, James Barksdale) as stated below:

1. Page 3-15. Unit-specific information concerning the Alabama Cave Shrimp is not provided.

   Response: Unit-specific information concerning the Alabama Cave Shrimp has been included as revised text beginning at paragraph 3.1.3.2.9

2. Page 3-43, Table 3.2-6. Unit-specific information concerning future residential land use is not provided.

   Response: Unit-specific information concerning future residential land use from the Redstone Arsenal Master Plan has been included as revised text for Sections 3.1.2.3, 3.2.4.6, and page 2 of Table 3.2-6.

Upon receipt of the cover letter, we will distribute the remaining copies to the appropriate regulatory and agency reviewers. Please call me if you have any questions or comments related to this submittal.

Sincerely,

ENVIRONMENTAL SCIENCE & ENGINEERING, INC.

Claire E. Marcussen
Project Manager

p/rsa/pm/memos/12-15-95

P.O. Box 1703 Gainesville, FL 32602-1703 Phone (904) 332-3318 (800) 874-7872 Fax (904) 333-6622

~~3.2.4.6 Future Resident~~

~~The potential for a future resident living directly on one of the study areas is unlikely. Future land use will be in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA in which there will be development in accordance with RSA remaining as an Army based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo Base Realignment and Closure (BRAC), which further supports that the current mission will continue into the future. Thus, this information supports the conclusion that a future residential scenario is unrealistic. In addition, landfill sites would have limited future uses as they would not be foundationally sound to support residential development.~~

~~3.2.4.6.1 If the study areas were to be scheduled for such land use in the future, the risks associated with such activity would need to be evaluated at that time. It is not useful to evaluate the risks for a future resident at this time because chemical concentrations in various media are expected to change considerably over time.~~

3.2.5 SUMMARY OF COMPLETED HUMAN EXPOSURE PATHWAYS TO BE QUANTIFIED IN BRA

A summary of the human exposure pathways quantified in this BRA are presented in Table 3.2-6 and are discussed in the following sections.

3.2.5.1 Groundwater Ingestion Pathway

Domestic/industrial consumption of groundwater is a pathway of concern when humans use wells that tap into groundwater flowing beneath a potentially contaminated site. Exposure will occur as a result of direct ingestion of water and beverages and foods made with water, as well as incidental ingestion during various activities, such as showering or washing cars. This pathway is not complete for current residents or workers because there are currently no drinking water wells near Areas F, G, R, or S/T. Although there are private wells located in Huntsville, none of these wells are close enough to the study areas that groundwater contamination could be attributed to the subject areas.

3.2.5.1.1 The groundwater consumption pathway is considered complete only for future workers and is evaluated for this receptor at all five of the study areas. The scenario of having a potable water well on one of the disposal sites is unlikely, but the scenario is

included in the BRA so that the potential risks associated with groundwater contamination at the study areas may be quantitatively evaluated. As per EPA region-wide (1989b) and Region IV (1992c) guidance, the quantitative evaluation of groundwater exposure is based only on unfiltered samples. Filtered samples may be discussed qualitatively, where appropriate.

3.2.5.2 Groundwater Dermal Contact Pathway

Domestic direct contact with groundwater may occur during various activities, including showering, cooking, and washing cars, dishes, and laundry. Dermal exposure to groundwater in an industrial setting may result from certain work-related processes, as well as showering. As with the groundwater ingestion pathway, this pathway is not complete for current residents or workers because there are currently no wells near Areas F, G, R, or S/T.

3.2.5.2.1 The groundwater dermal contact pathway is considered complete only for future workers and is evaluated for this receptor at all five of the areas. The future worker scenario assumes that a worker will have dermal contact with the water during showering. The scenario of having a potable water well on one of the disposal sites is unlikely, but as with groundwater ingestion, the scenario is included in the BRA so that the potential risks associated with groundwater contamination at the study areas may be quantitatively evaluated.

3.2.5.3 Groundwater Inhalation Pathway

Inhalation exposure to groundwater VOCs may occur as these chemicals volatilize from groundwater to indoor air. Similar to direct contact, domestic inhalation of groundwater chemicals may occur during various activities, including showering, cooking, and washing cars, dishes, and laundry. Inhalation exposure to groundwater VOCs in an industrial setting may result from certain work-related processes, as well as showering. The groundwater inhalation pathway is not complete for current residents or workers because there are currently no wells near Areas F, G, R, or S/T.

3.2.5.3.1 As with groundwater ingestion and direct contact, the groundwater inhalation pathway is considered complete only for future workers and is evaluated for this receptor at all five of the areas. The future worker scenario assumes that a worker will inhale groundwater VOCs while showering. Although unlikely, this scenario is included in the BRA

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Continued, Page 2 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Future Land Use, Continued			
Residential	Use of the site for future residential purposes	No	For this BRA, the analytical data and the exposure assumptions were evaluated based on an industrial land use. This projected future land use is in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA to be developed in accordance with RSA remaining as an Army-based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo BRAC, which further supports that the current mission will continue into the future. Thus, this information supports that the future residential scenario is unrealistic. RSA-49 (Area F), RSA-48 (Area G), RSA-59 (Area R), and RSA-55/54 (Area S/T) are located within the north central portion of the arsenal that is designated as an "unbuildable area" in the Master Plan (USACE, 1986) because of the following reasons: (1) RSA-48, -49, and -55 are designated as contaminated areas on the "Planning Constraints" map in the Master Plan (USACE, 1986); (2) RSA-54 is designated as an inactive landfill on the "Planning Constraints" map in the Master Plan (USACE, 1986); and (3) the eastern edge of RSA-48 as well as the eastern half of RSA-59 are located in the 100-year floodplain (NRMP, 1992; USACE, 1986) with elevations less than 572.5 ft-msl.

Source: ESE.

Table 3.3-1. Soil Intake Values for Terrestrial Indicator Species

Species	Soil Intake (kg soil/kg-BW/day)	Intake Factors			Source / Comments
Mouse	4E-03	IR	0.0038	kg/day	(0.2 g/g-day * 19 grams)
		EF	365	days/year	
		ED	1	year	EPA Factor
		%soil	0.02		EPA factor
		BW	0.019	kg	average of six mean values from EPA doc. (Millar, Fordham, Dewsbury)
		AT	365	days	
Raccoon	6E-03	IR	0.35	kg/day	based on Spector (1956)
		EF	365	days/year	
		ED	3	years	
		%soil	0.094		EPA factor
		BW	5.7	kg	Adult weight based on EPA factors (Sanderson, Nagel, Johnson)
		AT	1095	days	EPA factor (Johnson)
Deer	1E-03	IR	0.972	kg/day	based on g/kg BW value for cattle from LAAP report
		EF	365	days/year	
		ED	5	years	
		%soil	0.063		factor from jackrabbit EPA factor
		BW	54	kg	RSA Natural Resources document
		AT	1825	days	RSA Natural Resources Document - NUJ estimation
Bobwhite	7E-04	IR	0.014	kg/day	based on EPA factor of .07775 x BW (avg. from Koerth&Guthery data)
		EF	300	days/year	total lifespan - days
		ED	0.8333333333	years	EPA Factor
		%soil	0.01		based on EPA factor value for American woodcock
		BW	0.174	kg	EPA Factor (avg. from Robel, Roseberry & Klimstra, Guthery)
		AT	304.16666667	days	

Source: ESE.

3.2.4.6 Future Resident

The potential for a future resident living directly on one of the study areas is unlikely. Future land use will be in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA in which there will be development in accordance with RSA remaining as an Army-based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo Base Realignment and Closure (BRAC), which further supports that the current mission will continue into the future. Thus, this information supports the conclusion that a future residential scenario is unrealistic. In addition, landfill sites would have limited future uses as they would not be foundationally sound to support residential development.

3.2.4.6.1 RSA-49 (Area F), RSA-48 (Area G), RSA-59 (Area R), and RSA-55/54 (Area S/T) are located within the north central portion of the arsenal that is designated as an "unbuildable area" in the Master Plan (USACE, 1986) because of the following reasons:

- RSA-48, -49, and -55 are designated as contaminated areas on the "Planning Constraints" map in the Master Plan (USACE, 1986);
- RSA-54 is designated as an inactive landfill on the "Planning Constraints" map in the Master Plan (USACE, 1986); and
- The eastern edge of RSA-48 as well as the eastern half of RSA-59 are located in the 100-year floodplain (NRMP, 1992; USACE, 1986) with elevations less than 572.5 ft-msl.

3.2.4.6.2 If the study areas were to be scheduled for such land use in the future, the risks associated with such activity would need to be evaluated at that time. It is not useful to evaluate the risks for a future resident at this time because chemical concentrations in various media are expected to change considerably over time.

3.1.2.2.1 The five study areas discussed in this BRA are closed or abandoned disposal sites. The study areas are all heavily vegetated and are used for containment of waste or contaminated materials.

3.1.2.2.2 Outside the boundaries of RSA, much of the adjacent area is metropolitan with single-and multi-family residential, shopping, and industrialized areas. Because the City of Huntsville borders RSA on three sides, the more rural areas are those that border RSA to the southwest and southeast and across the Tennessee River to the south (Advanced Sciences, 1992). In these areas, as well as a few of the other outlying areas of Madison County, agriculture (e.g., soybeans, cotton, livestock) remains an economic mainstay.

~~3.1.2.3 Potential Alternate Future Uses~~

~~Future uses of RSA are anticipated to be the same as the current use (industrial), because the Army does not have plans to change the overall current mission of RSA. The Army has no plans to develop or use the five study areas under discussion for any purpose. These areas of abandoned or closed landfills will remain unused for the foreseeable future.~~

~~3.1.2.3.1 Based on the planning constraints and physical features of RSA, an analysis of the buildable areas was conducted (MICOM, 1992). Buildable areas are those where proposed major facilities with concentrations of personnel can be located. All land within one or more of the areas in the following list was considered unbuildable (MICOM, 1992).~~

~~1. Steep slopes (20 percent or more);~~

~~2. Flood plains (100 year floodline);~~

~~3. Explosive clearance zones;~~

~~4. Test areas;~~

~~5. Contaminated areas (i.e., toxic substance manufacturing, munitions drop areas, demolition areas, mustard gas or lewisite dumping and/or storage, DDT dumping areas, arsenic ponds);~~

~~6. Sanitary landfill areas (past and current);~~

~~7. Airfield clear zones; and~~

~~8. Potential airfield accident zones.~~

~~3.1.2.3.2 These development constraints indicate that only about 30 percent of the more than 38,000 acres at the installation are buildable (MICOM, 1992).~~

3.1.2.4 RSA and City of Huntsville Groundwater Use

Local municipal water supplies rely on groundwater and surface water resources from wells, springs, and surface waters that surround RSA near the installation boundaries (G&M, 1992). Figure 3.1-1 provides the locations of the withdrawal points of these water resources. Specific data for springs, wells, and surface water resources in and around RSA are provided in Table 3.1-2. Water resource withdrawal points labeled on Figure 3-1.1, and described in the following text, refer to the identification numbers on this table.

3.1.2.4.1 There are a number of private wells located east and west of RSA and to the south, across the Tennessee River (see Figure 3.1-1 and Table 3.1-2). The City of Huntsville is supplied by four wells upgradient of RSA and by two plants drawing from Wheeler Reservoir. As shown in Table 3.1-3, the four wells include the following:

1. Brahan Spring at Huntsville (Location 2 on Table 3.1-2)--Natural spring with well casings set below the surface of spring intake. Services approximately 60,000 customers, in network with other water sources, for potable water supply in Huntsville.
2. Well X-44 (Location 12 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
3. Well P-68 (Location 18 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
4. Well Q-79 (Location 23 on Table 3.1-2)--Well is used, in network, to supply potable water for approximately 60,000 customers in the City of Huntsville.
5. Lincoln/Dallas wells (Location 39 on Table 3.1-2)--Wells used to supply, in network with other sources, potable water for approximately 60,000 customers in the City of Huntsville.

3.1.2.4.2 In addition to the four well or well clusters providing potable water to the City of Huntsville, there are two surface water intakes that also supply potable water. As shown in Table 3.1-2, the surface water intakes for the City of Huntsville include:

3.1.2.3 Potential Alternate Future Uses

Future uses of RSA are anticipated to be the same as the current use (industrial) because the Army does not plan to change the overall current mission of RSA. The Army has no plans to develop or use the areas under discussion in this BRA for any purpose. These areas of abandoned or closed landfills and former ponds will remain unused for the foreseeable future.

3.1.2.3.1 Based on the planning constraints and physical features of RSA, a report entitled *Analysis of Existing Facilities and Environmental Assessment Report* (USACE, 1986) was prepared as part of the Master Plan for RSA to address the economic, social, environmental, and land use implications of the future development of RSA. Buildable areas are those where proposed major facilities with concentrations of personnel can be located. All land within one or more of the areas in the following list was considered unbuildable (USACE, 1986).

- Steep slopes (20 percent or more),
- Floodplain (100-year floodline encompassing elevations less than 572.5 ft-msl),
- Explosive clearance zones,
- Test areas,
- Contaminated areas,
- Sanitary landfill areas (past and current),
- Airfield clear zones, and
- Airfield accident potential zones.

3.1.2.3.2 Although the installation consists of more than 38,000 acres, only about 30 percent is buildable based on the previously listed development constraints (USACE, 1986). RSA-49 (Area F), RSA-48 (Area G), RSA-59 (Area R), and RSA-55/54 (Area S/T) are located within the north central portion of the arsenal that is designated as an "unbuildable area" in the Master Plan (USACE, 1986) because of the following reasons:

- RSA-48, -49, and -55 are designated as contaminated areas on the "Planning Constraints" map in the Master Plan (USACE, 1986);
- RSA-54 is designated as an inactive landfill on the "Planning Constraints" map in the Master Plan (USACE, 1986); and
- The eastern edge of RSA-48 as well as the eastern half of RSA-59 are located in the 100-year floodplain (NRMP, 1992; USACE, 1986).

Because RSA-48, -49, and -55/54 are designated as unbuildable, these areas cannot be used for future residential development. According to the Master Plan (USACE, 1986), future landuse for these areas will be either operational facilities (i.e., RSA-48 and -55), administrative buildings (i.e., RSA-49), or research and development facilities (i.e., RSA-54) as designated on the "Reservation Plan" map in the Master Plan for RSA (USACE, 1986).

widely used by wildlife species. In addition to this excellent trophic support of wildlife, these forests provide much cover needed for nesting and resting.

3.1.3.2.7 The pine plantations are the least diverse of the forest types. Older plantations have a low potential for wildlife support due to a scant understory of blackberry and bracken fern, which provide suboptimal food and cover. Younger plantations, however, are somewhat more diverse and have a more dense understory yielding a moderate value to wildlife.

Wetlands

3.1.3.2.8 Wetlands on RSA occur in the form of beaver ponds, sloughs, marshes, tupelo swamps, backwater, and overflow hardwood bottomlands. Wetlands were once considered wastelands, but are now known to serve many valuable functions. Among these, wetlands filter excess nutrients and reduce sedimentation, which helps to maintain the quality of downgradient water systems. Wetlands help to keep erosion under control and promote groundwater storage. They also provide critical habitat for many species of fish and wildlife. Of the five areas discussed in this BRA, the most significant wetland is the system located adjacent to Area R.

~~**Caves and Water-Filled Void Spaces**~~

~~**3.1.3.2.9** Two caves that are not in Areas F, G, R, or S/T have been identified at RSA: Mathews Cave and Ravine, which is on the northwest border of RSA (USACE, 1992); and Bobcat Cave, which is 3 miles north of the confluence of Indian Creek and Huntsville Spring Branch (USACE, 1986). However, to date, it has not been determined that caves and water-filled void spaces are present under Areas F, G, R, or S/T. Until such a determination is made, groundwater will not be considered to be a direct exposure pathway for ecological receptors. MICOM is currently working with the U.S. Fish and Wildlife Service (USFWS) to determine if cave and water filled void space habitats exist under Areas F, G, R, and S/T. Results of the MICOM/USFWS efforts should be available by the end of this year. If such habitats are found near the five areas of concern, then risks to aquatic life will be addressed. The results of the cave and water filled void space habitat assessments will be presented in an addendum to the RSA BRA1.~~

3.1.3.3 Aquatic and Terrestrial Communities

Aquatic communities in the surface water near Area G include aquatic invertebrates, and all trophic levels of fish, turtles, and amphibians. The wetlands near Area R had only shallow water at the time of the ESE visit (May and June 1994). These wetland surface waters are not expected to sustain fish much larger than *Gambusia* (mosquitofish).

3.1.3.3.1 Small animals expected to be found at the study areas include mice, rabbits, skunks, and raccoons. Several game species that have been introduced to RSA since 1954 include quail, wild turkey, and white-tailed deer. These populations are quite prosperous at RSA. Some of the successful terrestrial communities on RSA that have been documented as being nuisance populations include blackbird flocks, beavers, ground hogs, coyotes, and skunks (MICOM, 1992). In addition to these species, bluebird and wood duck populations are being actively managed so that appropriate nesting habitat is available at RSA. A complete list of species believed to inhabit RSA can be found in the NRMP (MICOM, 1992) in Appendix H.

3.1.3.4 Subpopulations of Potential Concern

As with the identification of human populations, a similar evaluation of release and exposure pathways and study area characteristics was performed to identify the primary ecological populations of concern. Potential receptors were identified by considering the types of chemicals released, the physical characteristics of the study area that may affect chemical migration, current and expected land uses, and habitat type (i.e., wetland, 100-year floodplain, proximity to surface water body).

3.1.3.5 Endangered or Threatened Fauna

A complete list of potential endangered, threatened, or species of special concern is provided in Appendix G. Table 3.1-3 lists endangered or threatened fauna.

3.1.3.6 Nonhuman Representative Receptors

Due to the number and diversity of nonhuman receptors present at the study areas, it is not feasible to evaluate each species present. EPA guidance indicates that biological receptors (ecosystem components expected to reflect adverse effects of pollutant stress) and endpoints (type of actual or potential impact due to contaminant exposure by a receptor) be selected to

Caves and Water-Filled Void Spaces

3.1.3.2.9 Literature searches thus far have indicated that seven caves are known to exist on RSA. However, none of the caves are located within the borders of the areas of concern for BRA1 (Areas F, G, R, or S/T). The seven caves are Bobcat Cave, Matthews Cave, Jasper's Folly Cave, Adam's Cave, Five and One-Half Cave, Formation Pit, and Fox Den Cave (Jones *et al.*, 1968; Moser *et al.*, 1992; USACE, 1992; Hartfield, 1995). The following paragraphs provide brief descriptions of each of the seven caves.

3.1.3.2.9.1 The Alabama cave shrimp is known to occur in two caves in Madison County, Alabama, one of which is Bobcat Cave (Hartfield, 1995). Bobcat Cave is approximately 1,000 feet east of the western boundary in the west-central part of RSA and about 3 miles north of the confluence of Indian Creek and Huntsville Spring Branch (Moser *et al.*, 1992; USACE, 1986). The Speleological Society Cave Number for Bobcat Cave is AL1283 (Moser *et al.*, 1992).

3.1.3.2.9.2 Matthews Cave is located on the northwest border of RSA (USACE, 1992). Its Speleological Society Cave Number is A23 and its location is the southeast quarter of the southeast quarter of Section 12, Township 4 South, Range 2 West (SESE 12-4S-2W) (Jones *et al.*, 1968). Figure 1.1-1 provides a boundary of RSA as well as section numbers.

3.1.3.2.9.3 The location of Jasper's Folly Cave (National Speleological Society Cave Number A181) is SENE 17-4S-1W (Jones *et al.*, 1968). Jasper's Folly Cave is on the east side of Madkin Mountain in the northern part of RSA (GSA, 1968).

3.1.3.2.9.4 Adam's Cave (National Speleological Society Cave Number A412) is located at SWNW 27-5S-1W (Jones *et al.*, 1968). Adams Cave is lcoated near Woodward Cemetery (GSA, 1968), which is approximately 2 miles east of Unit 2 (RSA-12, -13, -14, -131, -132, and -133).

3.1.3.2.9.5 Five and One-Half Cave is located near Hatton Mountain in the southwestern part of the Arsenal (GSA, 1968) at NENE 14-5S-2W (Jones *et al.*, 1968). Its National Speleological Society Cave Number is A421 (Jones *et al.*, 1968).

3.1.3.2.9.6 Formation Pit is located near Jasper's Folly (GSA, 1968) at NESE 17-4S-1W (Jones *et al.*, 1968). Its National Speleological Society Cave Number is A431 (Jones *et al.*, 1968).

Fox Den Cave is located on Madkin Mountain in the northern part of the Arsenal (GSA, 1968). Its National Speleological Society Cave Number is A600 and its location is SWNE 20-4S-1W (Jones *et al.*, 1968).

3.1.3.2.9.7 Six of the seven caves are distributed in Gasper Formation and Warsaw Limestone (GSA, 1968), which is distinct from the Tuscumbia Limestone thought to underlie the sites of concern in this BRA (G&M, 1991a). Bobcat Cave is formed in the Tuscumbia Limestone (Moser *et al.*, 1992). In addition to Bobcat Cave, numerous caves in Madison County are within the Tuscumbia Limestone formation (GSA, 1968).

3.1.3.2.9.8 It has not yet been determined if caves or water-filled void spaces are present under Areas F, G, R, or S/T. If further studies indicate that habitats suitable for cave life exist beneath the areas addressed in this BRA, then risks to aquatic life will be evaluated. The results of the cave and water-filled void space habitat assessments will be presented in an addendum to the RSA BRA1 when further information is available.

Table 3.2-6. Summary of Exposure Pathways to be Analyzed for RSA Areas F, G, R, S, and T (Continued, Page 2 of 2)

Potentially Exposed Population	Exposure Route, Medium, and Exposure Point	Pathway Selected for Evaluation?	Reason for Selection or Exclusion
Future Land Use, Cont.			
Residential	Use of the site for future residential purposes	No	~~For this BRA, the analytical data and the exposure assumptions were evaluated based on an industrial land use. This projected future land use is in accordance with the RSA Master Plan (USACE, 1989), which outlines a program for RSA to be developed in accordance with RSA remaining as an Army based facility. The Master Plan states that the mission of the facility is to remain an active military installation. RSA has not been identified as a site that will undergo BRAC, which further supports that the current mission will continue into the future. Thus, this information supports that the future residential scenario is unrealistic.~~

Source: ESE.

Table 3.3-1. Soil Intake Values for Terrestrial Indicator Species

Species	Soil Intake (kg soil/kg-BW/day)	Intake Factors			Source / Comments
Mouse	4E-03	IR	0.0038	kg/day	(0.2 g/g-day * 19 grams)
		EF	365	days/year	
		ED	1	year	EPA Factor
		%soil	0.02		EPA factor
		BW	0.019	kg	average of six mean values from EPA doc. (Millar, Fordham, Dewsbury)
		AT	365	days	
Raccoon	6E-03	IR	0.35	kg/day	based on Spector (1956)
		EF	365	days/year	
		ED	3	years	
		%soil	0.094		EPA factor
		BW	5.7	kg	Adult weight based on EPA factors (Sanderson, Nagel, Johnson)
		AT	1095	days	EPA factor (Johnson)
Deer	1E-03	IR	0.972	kg/day	based on g/kg BW value for cattle from LAAP report
		EF	365	days/year	
		ED	5	years	
		%soil	0.063		factor from jackrabbit EPA factor
		BW	54	kg	RSA Natural Resources document
		AT	1825	days	RSA Natural Resources Document - NUJ estimation
Bobwhite	7E-04	IR	0.014	kg/day	based on EPA factor of .07775 x BW (avg. from Koerth&Guthery data)
		EF	300	days/year	total lifespan - days
		ED	0.8333333333	years	EPA Factor
		%soil	0.01		based on EPA factor value for American woodcock
		BW	0.174	kg	EPA Factor (avg. from Robel, Roseberry & Klimstra, Guthery)
		AT	304.16666667	days	

Source: ESE.

Additional References

Hartfield, P. 1995. Alabama Cave Shrimp (*Paleamonaias alabamae*) Recovery Plan. Technical Draft. Prepared by the U.S. Fish and Wildlife Service, Jackson, Mississippi. Prepared for the Southeast Region of the U.S. Fish and Wildlife Service, Atlanta, Georgia.

Jones, W.B. and Varnedoe, W.W., Jr. 1968. Caves of Madison County, Alabama. Geological Survey of Alabama. Division of Economic Geology. Circular 52.

Moser, P.H. and Rheams, K.F. 1992. *Hydrogeologic Investigations of Shelta and Bobcat Caves and Adjoining Areas, Madison County, Alabama*. Geological Survey of Alabama. E.A. Mancini, State Geologist. Water Resources Division. Prepared in cooperation with the U.S. Department of the Interior Fish and Wildlife Service. Cooperative Agreement No. 14-16-0004-90-935. Tuscaloosa, AL.